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Trauma Surgery

Edited by Ozgur Karcioglu and Hakan Topacoglu

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Notice

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Meet the editors

Dr. Ozgur Karcioglu commenced his residency in Dokuz Eylul University Medical School, Department of Emergency Medicine, and graduated in 1998. He served as the chairman of the department from 2005 to 2007. He completed the "International Emergency Medicine" Fellowship in PSU in 2005.
Dr. Karcioglu served as a founder and board member of the Emergency Medical Association of Turkey after 1995. He has had more than 100 articles published in scientific journals, contributed to 3 books as editor, and authored 33 chapters. He is a member of the editorial and review boards of a number of journals and an instructor of the Advanced Cardiac Life Support Course. His research interests are resuscitation, advanced life support, pain, disasters, trauma, and toxicology.

Prof. Dr. Hakan Topacoglu completed the emergency medicine residency program at Dokuz Eylul University Medical School in 2002, where he served as an assistant professor from 2002 to 2007 and an associate professor from 2007 to 2010. During this period, he worked as a member of many boards of the university. In addition, he served as a manager of the Centre of the Emergency Medicine and Disaster Training from 2008 to 2010. He is currently working as a professor doctor at the university hospital in Duzce. He has published more than 40 peer-reviewed articles in international journals, which have been cited over 400 times and he presented more than 80 abstracts at national and international symposia. His H index is currently 11, i10 index is 13, and RG score is 27,3. He is a Board member of the Emergency Medical Association of Turkey and he contributed as an editor in two books.

Contents

Preface

The twenty-first century is witnessing a growing threat to human beings imposed by many sources, namely natural disasters, terrorism and other conflicts, warfare, and transportation accidents; all of which ignite the rise of major trauma incidents worldwide. This phenomenon mandates physicians involved in trauma management to be prepared to evaluate, diagnose, treat, and stabilize patients who have been exposed to some type of traumatic injuries. These injuries comprise a wide array of patients, from those ready to be discharged after a brief evaluation to those hardly viable after resuscitative attempts and life-saving operations. The successful management of trauma depends on a working collaboration of emergency medicine, surgical disciplines, intensive care medicine, and virtually all ancillary services of a hospital to achieve and maintain homeostasis. Although technological advances render many resuscitative techniques more easily available in most parts of the world, there is still space to improve trauma care, especially in exsanguination, hemorrhagic shock, heart and major vessel injuries, pelvic and long bone fractures, ultrasound use, blood products transfusion, and resuscitation in general, which comprise the theme of the book.

This book is intended to increase awareness of the increased toll of major and multiple traumas and to help individual physicians be prepared for every situation in this context.

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Section 1

Fractures and Related injuries

Chapter 1

Fracture Repair: Its Pathomechanism and Disturbances

Grzegorz Szczęsny

Additional information is available at the end of the chapter

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Abstract

Healing of the bone fracture is a biological process that is based on various cell lineages recruited, activated and regulated by molecular mediators, namely chemokines, growth factors, and cytokines, cooperating in a cascade of events aimed to fill the fracture gap with callus. Remodeling of the callus rebuilds the microarchitecture to the mature bone—cancellous or compact, depending on the type of the bone that was primarily at the fracture gap. Restitution of the bone continuity requires activation of mesenchymal stem cells that transform into osteoblasts and mature into osteocytes. It is activated and regulated by molecules released from blood platelets from posttraumatic hematoma, traumatized tissues, nerve endings, and inflowing inflammatory cells. The significance of the inflammatory cells in this process is inappreciable, as they eradicate pathogens, remove wound debris, and supply the fracture gap with molecules regulating forthcoming cellular events. They also provide immune regulation of the healing. To proceed uneventfully, healing requires an adequate bone contact and biomechanical environment, proper oxygenation, and nutrition. Unfortunately, up to 15% of bone fractures show some kinds of disturbances that may result in cessation of reparative processes leading to non-union. Factors, responsible for that, are brought to date based on current literature and clinical observations.

Keywords: fracture repair, bone fracture, non-union, mechanical, infection, iatrogenic, mesenchymal stem cells (MSCs), immune control, pharmaceuticals, nutrition

1. Background

Healing of the bone fracture is a biological process that restores its continuity, mechanical properties, and structure. It bases on various cell lineages recruited, activated and regulated by molecular mediators, namely chemokines, growth factors, and cytokines, cooperating in a cascade of events aimed to fill the fracture gap with callus, which later on is remodeled into

mature bone. Thus, this process is, in fact, a regeneration, not healing, as its goal is to restore not only the bone's continuity but also its structure.

Clinically, healing manifests with remission of pain corresponding with gradually increasing stiffness enabling transduction of mechanical loads. Radiographically-with formation and remodeling of the callus in-between its gap. Monitoring of this process indicates the advance of the reparative processes.

2. Cellular aspects of fracture healing

From the histological point of view, restitution of the bone continuity proceeds due to accumulation and activation of mesenchymal stem cells (MSCs) that, transforming into osteoblasts and maturating into osteocytes, synthesize and release proteins forming the extracellular matrix (ECM).

In the vast majority of cases, MSCs that settle hematoma differentiate into chondrocytes. But revascularization, due to the ingrowth of blood vessels from the vasculature of the adjacent tissues, improves local oxygenation enabling the transformation of newly inflowing progenitors into osteoblasts that, maturating into osteocytes, initiate ossification forming bone cuff around the fracture gap. Starting from its periphery, it moves toward the center replacing the soft callus with woven bone [1, 2]. Later on, it's remodeling rebuilds the microarchitecture to the mature bone-cancellous or compact, depending on the type of the bone that was primarily at the fracture gap.

The described above process, the endochondral ossification, proceeds in ca. 97–98% of all fractures, whereas remaining 2–3% heal due to the direct osteonal growth in the process called primary bone healing basing on the intramembranous ossification [3]. The latter one is possible, when the volume of the fracture's gap is minimal, thus in non-displaced or impacted fractures only. In those cases, the short distance between bone fragments enables osteonal remodeling toward the fracture gap restoring its vascularization and mineralizing it.

MSCs residue several tissues, including bone marrow, endosteum, and periosteum. They are abundantly represented in adipose tissue surrounding the extremity with subcutaneous fat and form a subpopulation of its leukocytes in peripheral blood as well [4]. Thus, extravasated into posttraumatic hematoma and recruited from adjacent tissues they form a population of precursors for reparative processes.

Their accumulation proceeds due to chemotactic stimulation. Stromal-derived factor-1 (SDF-1; also known as CXC-motif chemokine 12: CXCL12) is one of the most potent attractants of MSCs. Widely distributed in bone marrow, it splits in-between neighboring tissues, when bone continuity is broken, recruiting progenitors that accumulate at the sites of its highest concentration, the fracture gap. Inflowing cells multiply under the mitogenic stimulation of platelet-derived growth factor (PDGF) [5].

MSCs are precursors of various cells of mesenchymal origin, including chondrocytes, fibroblasts, adipocytes, neurons, and myocytes. The direction of their differentiation depends upon molecular regulation and local physicochemical conditions. When stimulated improperly or

under unfavorable conditions, they may differentiate into, unwanted from the point of view of the fracture healing, cellular population forming cartilaginous or fibrous pseudoarthrosis.

Hypoxia, hypercapnia, and acidosis that characterize deprived of vasculature posttraumatic hematoma, promote their differentiation into chondrocytes, whereas higher oxygen tension and reduced acidosis-into osteoblasts [6].

3. Molecular stimulators of fracture repair

Four sources of molecular stimulators and regulators of bone healing could be distinguished:

1. Extravagated blood forming the posttraumatic hematoma
2. Traumatized bone and tissues neighboring it
3. Nerve endings at the adjacent tissues
4. Inflowing inflammatory cells

Platelets are the abundant source of molecular substances of blood origin. Released from granules into a posttraumatic hematoma, those substances activate, together with mediators released from nerve endings, and cellular events proceeding in the fracture gap.

Platelets participate in various reparative processes, being involved in the restoration of traumatized mucous and epithelia, healing various soft tissues (i.e. muscle) and the bone, and restoration of the vascularity in the process of angiogenesis. An influence of other hematoma products, including fibrin clot and activated clotting factors, hemoglobin, complement cascade and subcellular structures such as subcellular fragments of blood cells increases, giving an insight into a complex role of several hematoma compounds in the healing [7].

Traumatized tissues provide molecular stimuli that are released in response to injury. Damage - associated molecular pattern molecules (DAMPs) are the most potent activators of the sterile, traumatic inflammation ("first hit"), whereas the later one ("second hit") mostly dependents on molecules provided by the inflowing immune cells. Those molecules activate immune system directly through toll-like receptors (TLRs) [8]. So far, several DAMPs have been distinguished, including heat-shock proteins (HSPs), high-mobility group box 1 (HMGB-1), monosodium urate, heparan sulfate, adenosine triphosphate (ATP), polysaccharides, proteoglycan, phospholipids, and deoxyribonucleic acid (DNA). Similar capabilities possess hyaluronian fragments released from disintegrated ECM [9, 10].

Nerve endings provide neuromediators that participate in fracture repair, including calcitonin gene - related peptide and neuropeptide-Y [11]. Released in response to mechanical (injury) and physicochemical (hypoxia, acidosis) stimuli, they participate in the molecular regulation of cellular events during the reparative phase and callus mineralization [12]. However, they were also found to control remodeling [13].

The later abundant source of molecular stimulators are leukocytes originating from the blood-forming hematoma and inflowing from the peripheral circulation. Granulocytes are the first

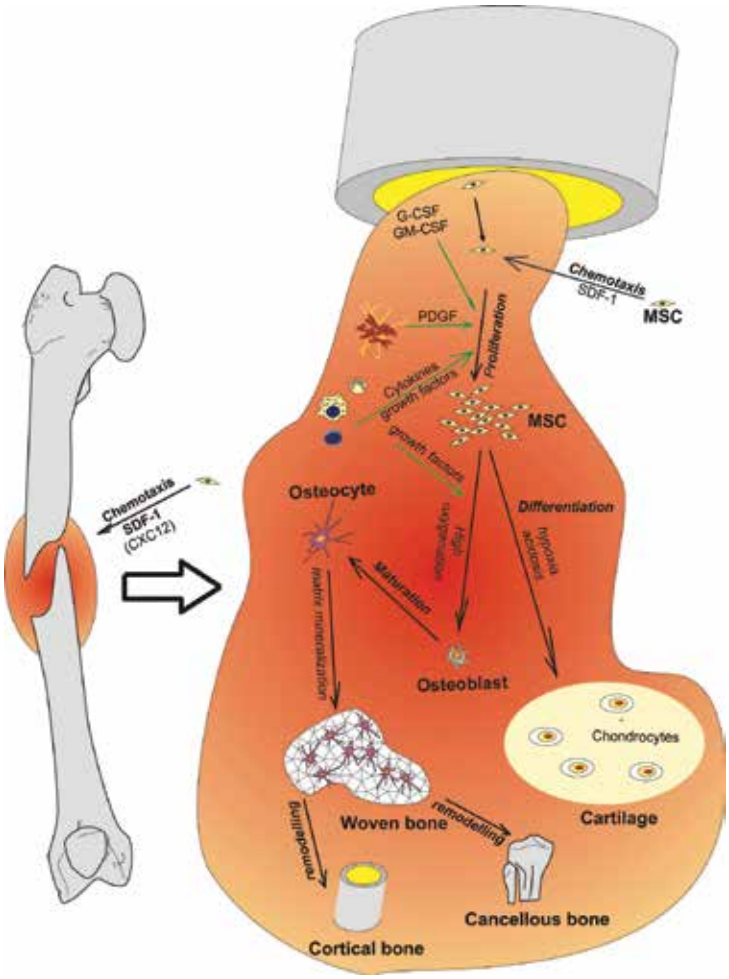

Figure 1. Schematic representation of recruitment, proliferation, multiplication, and differentiation of MSCs into osteoblasts or chondroblasts.

cellular population that actively populates the fracture gap. Those cells infiltrate the wound as early as at the sixth hour after injury providing its innate immune protection against pathogens, but also participating in reparative processes [14]. Being followed by lymphocytes and monocytes/macrophages they form an inflammatory phase of the healing cascade.

The significance of the inflammatory cells for the reparative processes is inappreciable, as they eradicate pathogens, remove wound debris and, partially, foreign bodies, but also supply the fracture gap with molecules regulating forthcoming cellular events. They also provide immune regulation of the healing, as the response of the lymph node draining the fracture gap was shown to reflect its cellular and molecular processes [35]. This mechanism seems to depend on regulatory B and T lymphocytes (B_{regs} and T_{regs}), as they were shown to participate in fracture healing. B_{regs} were presented to suppress the inflammatory phase secreting anti-inflammatory cytokines IL-10 (interleukin-10) and TGF-β (transforming growth factor-β), and enhancing maturation of T_{regs} [15]. At the early phase of the reparative processes, they

probably prevent from auto aggression against infiltrating progenitors, thus enabling them to proliferate and differentiate into bone forming cells. Depletion of B_{reg} cells, analogically to splenectomy, results in the delay of the fracture healing [16, 17]. Moreover, T cells were shown to promote maturation of the osteoblasts [18].

Immune cells are an ample source of several molecular substances, including cytokines (i.e. IL-6 and IL-8) and growth factors (PDGF, fibroblast growth factor; FGF, TGF-β, and bone morphogenetic proteins—BMPs) [19]. Together with molecules released from nerve endings and bone marrow, they regulate cellular events stimulating proliferation and differentiation of MSCs.

The most effective MSCs stimulators are the granulocyte colony-stimulating factor (G-CSF) and granulocyte-macrophage-colony-stimulating factor (GM-CSF), cytokines (IL-1, IL-3, IL-7, IL-8, and IL-12), stem cell factor (SCF), Flt3 (fms like tyrosine kinase 3) ligand, macrophage inflammatory protein-1 (MIP-1) and the chemokines GROβ (growth-regulated oncogene β; also known as CXC-2) and SDF-1 [20, 21]. Each of them evokes the unique effect promoting migration, division, activating synthesis, and release of molecules forming an appropriate environment or fulfilling the specialized biological function. They enable migration, multiplication, and differentiation of progenitors into desired cell lineage. The most potent stimulators of MSCs differentiation into, according to the local physicochemical environment, chondrocytes, or osteoblasts are TGF-β, several BMPs (2, 4, 6, 7, 13, and 14), IGF-1 (insulin-like growth factor), and FGF [22, 23]. The final outcome in the form of fracture healing results from the convergent actions of numerous factors influencing the target cells in an appropriate time sequence and place (**Figure 1**).

4. Extracellular matrix mineralization

Mineralization of the ECM restores mechanical properties of the gap bringing back its ability to carry body weight. Briefly, it consists in the deposition of calcium and phosphate precipitates, hydroxyapatite, around the mesh of ECM proteins, namely collagens [24]. The process takes place in matrix vesicles; subcellular structures of approx. 20–200 nm in diameter that contains a number of compounds, including annexins (annexin V), alkaline phosphatase, calbindin-D9k, pyrophosphatases, carbonic anhydrase, AMP-ases, bone sialoprotein-1 (BSP-1), osteonectin, osteocalcin, and several growth factors [25].

Matrix vesicles concentrate inorganic substrates for mineralization due to annexin-formed calcium channels and Na/Pi phosphate transporters (NPT3/Pit1; natrium-phosphate transporter 3/POU domain class 1 transcription factor 1). High concentration of those ions results in their spontaneous precipitation to amorphous octa-Ca/Pi crystals that later on, when released from the vesicles, are converted by osteonectin, osteocalcin, and bone sialoprotein-1 (BSP-1) into hydroxyapatite. Hydroxyapatite crystals are deposited into the ECM at the outer and inner surface of the collagen fibrils [26]. In consequence, the collagen forming ECM being responsible for bone elasticity also serves as a scaffold for inorganic substances [27]. Their remodeling by matrix metalloproteinases sets the direction of trabecular bone remodeling, creating it is three-dimensional structure according to the direction of mechanical loads, and thus optimizing its microarchitecture for the most effective resistance [28, 29]. Finally, remodeling restores the structure of the primary callus to the mature bone identical to that primarily present at the fracture gap.

5. Remodeling

Remodeling proceeds in consequence of osteolysis and forthcoming osteogenesis. In the beginning, a group of activated osteoclasts, acidifying ECM, dissolve the osteoid and enzymatically (MMPs) digest its proteins. In consequence, resorptive (Howship) lacuna is formed.

Released molecules that are stored in the latent form bound to ECM heparan sulfate (BMPs, Vascular endothelial growth factor (VEGF), FGF, and EGF) activate proliferation and folding into three-dimensional structures of endothelial cells originating from neighboring blood vessels [30]. Those form vascular loops (sprouts) in-growing into the lacunae, providing its blood supply. Inflowing MSCs differentiate into osteoblasts that repopulate lacunae as osteocytes excreting ECM proteins and mineralizing them.

Osteoclasts at the top (cutting cone) gradually move across the bone as far as they reach its borderline (osteoclastic tunneling; remaining as Haversian canal), and finally undergo apoptosis. Passing across the fracture, they restore bone continuity (osteonal fracture healing), but only when the distance between bone fragments does not exceed 1 mm [31]. If the distance is higher, each bone fragment is remodeled alone and the fracture gap remains intact, that is not healed.

6. Uneventful and disturbed fracture healing

From the clinical point of view, the fracture is considered to be healed, when its mechanical properties are restored allowing carrying the body weight. Thus, the main indicators of successful healing are lack of the pathological mobility corresponding with a resolution of pain and restored the ability to carry mechanical loads confirmed by radiographic images showing callus mineralization and remodeling.

It was estimated that up to 15% of fractures display some kind of healing disturbances [32]. Depending on the severity of the pathological changes, it ranges from slow fracture healing (slow union), delayed union or non-union, if complete inhibition of the reparative processes occurs. The lack of union and the resultant non-union (pseudoarthrosis) are diagnosed when callus was not formed in-between bone fragments in an assumed period of time and all regenerative processes have stopped.

According to the recommendations of the food and drug administration, a non-union could be diagnosed, when the fracture is not healed in the 9th post-injury month, or any evidence of the healing progress could be observed on X-rays during the three consecutive months. However, the number of orthopedists that diagnose non-union as early as at the 6th post-fracture month implementing procedures that improve reparative processes increases. However, it is also believed that the time of the healing of a given bone should be determined arbitrarily, based on the clinical experience [33].

The varying opinions that concern the definition of disturbed fracture healing come from the lack of diagnostic tools that could demonstrate the moment of cessation of regenerative processes. The very important flaw of radiographic monitoring is the possibility to assess the status of the healing after a sufficiently long period of follow-up. Moreover, it does not allow predicting the final result.

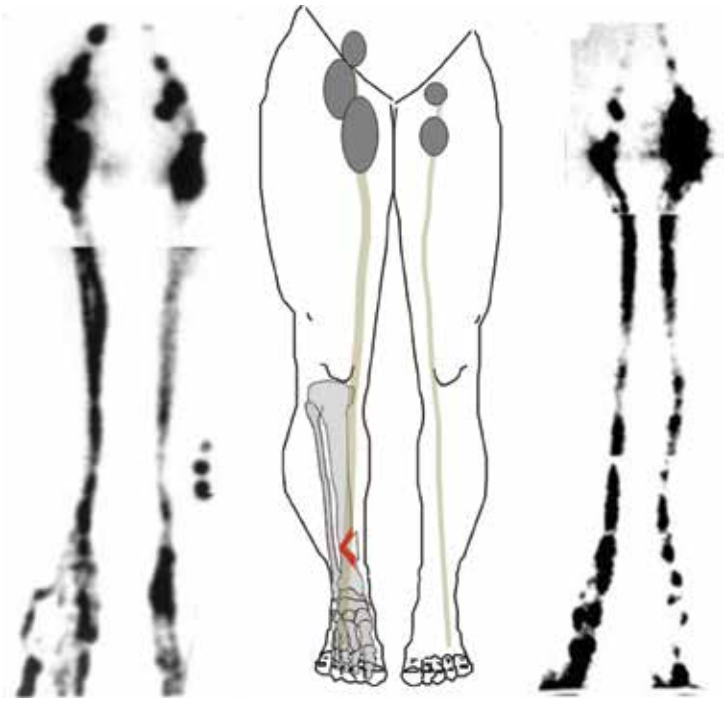

Figure 2. Lymphoscintigrams of the uneventful (left) and disturbed (right) healings of the fractures of the right extremities. Uneventful healing is characterized by enlarged regional lymph nodes and lymphatic outflow, when compared with contralateral limb. If the healing is disturbed, decreased lymph outflow, and regional lymph nodes, are observed.

The only examination that may be useful in the monitoring of the healing process and predicting its outcome is limb lymphoscintigraphy [34]. Observation of the lymphatic system showed that regional lymph node draining the fracture is a subject of molecules released from its gap. Thus, increased lymph drainage and enlargement of the lymph node (accumulation of cells) reflect molecular and cellular events taking place at the fracture gap that may be used as an indicator of the quality of reparative processes (**Figure 2**).

Uneventful healing may be divided into three phases:

1. Reactive- colonization of posttraumatic hematoma by inflammatory cells,
2. Reparative-replacement of hematoma with cartilaginous tissue and its endochondral ossification (primary callus),
3. Remodeling-formation of mature bone with the structure analogous to that prior to the fracture.

The first phase lasts up to several (3–7) days, the second up to 4 weeks and the third may last up to 2 (or even more) years after the fracture. However, the advance of the healing may differ even in adjacent areas; especially, when proceeds in comminuted fractures. In consequence, remodeling already proceeding in between some bone fragments may coexist with early, reactive phase between others.

Histologically, uneventful healing is characterized by soft callus filling the fracture gap in the 2nd week after injury (soft callus). In the 4th week, the callus should already be replaced by spongy bone (hard callus), and in the 8th-be a subject of remodeling. Non-union is characterized by the lack of ossification at the 4th post-fracture week, despite the fact that similarly to uneventful healing, the fracture's gap is filled with an excess of cartilage "flowing" out of it. In the 8th week, young fibrous tissue with scarce and loose foci of cartilaginous tissue is observed and finally, the pseudoarthrosis is formed [35].

On a molecular level, there are no differences in the expression of PDGF, TGF-β, and FGF-2 in the 1st week after the fracture in both uneventfully and healing with delay fracture gaps. But in the 8th week, in contrary to uneventful healing, whose osteocytes express all these factors, none of them is expressed [36]. It was proved that lack of the mentioned above molecular stimuli leading to non-union could also be produced surgically removing tissues from the fracture gap that may result from repeated debridement or rinsing drainage [37].

7. Factors disturbing healing of the fracture

As the goal of reparative events is to fill the fracture gap with cells possessing osteogenic potential, the participation of their precursors, the MSCs, is crucial. As MSCs are widely distributed in the body, the risk of their deficiency is rather not feasible. Nevertheless, those cells exert some specific features that may reduce their number and activity.

First of all, they are very sensitive to unfavorable conditions, distinctly responding to inordinate mechanical stimuli, hypoxia, and malnutrition [38]. They are also very prone to injury, regardless of its mechanism: mechanical, thermal (burns, frostbites), chemical (acids, bases, toxins), electric, or radiative. Thus, massive traumatization of tissues neighboring the fracture deprives them of progenitors resulting in cessation of reparative processes. Moreover, their loss, exposing the bone to the outer environment, favor its drying that promotes intravascular coagulation depriving the fracture of blood supply. Also, iatrogenic injuries, including vast surgical approach, wide periosteal stripping, excessive cauterization, or just brutal operative technique, superimpose traumatic changes impairing the healing.

Second of all, removal or drainage of hematoma or cellular infiltrates from the fracture, especially, when performed repeatedly, deprives it of molecular regulators [7].

Third of all, under hypoxia MSCs have been shown to differentiate into chondrocytes, instead of osteoblasts. This process, being natural at early stages of reparative processes, when prolongs, results in the formation of cartilaginous pseudoarthrosis. The problem usually occurs, when the fracture is immobilized inadequately or is not immobilized at all, as excessive movements between bone fragments disrupt newly formed vasculature depriving it of blood supply [39].

An especially unappreciated is the contribution of the shock in the cessation of fracture gap perfusion. Centralizing the circulation to protect the circulation of vital organs, it deprives the perfusion of peripheral tissues, including the fractured gap and surrounding it tissues [40]. When prolongs, shock aggravates tissue injury, impairs the healing, and increases the risk of

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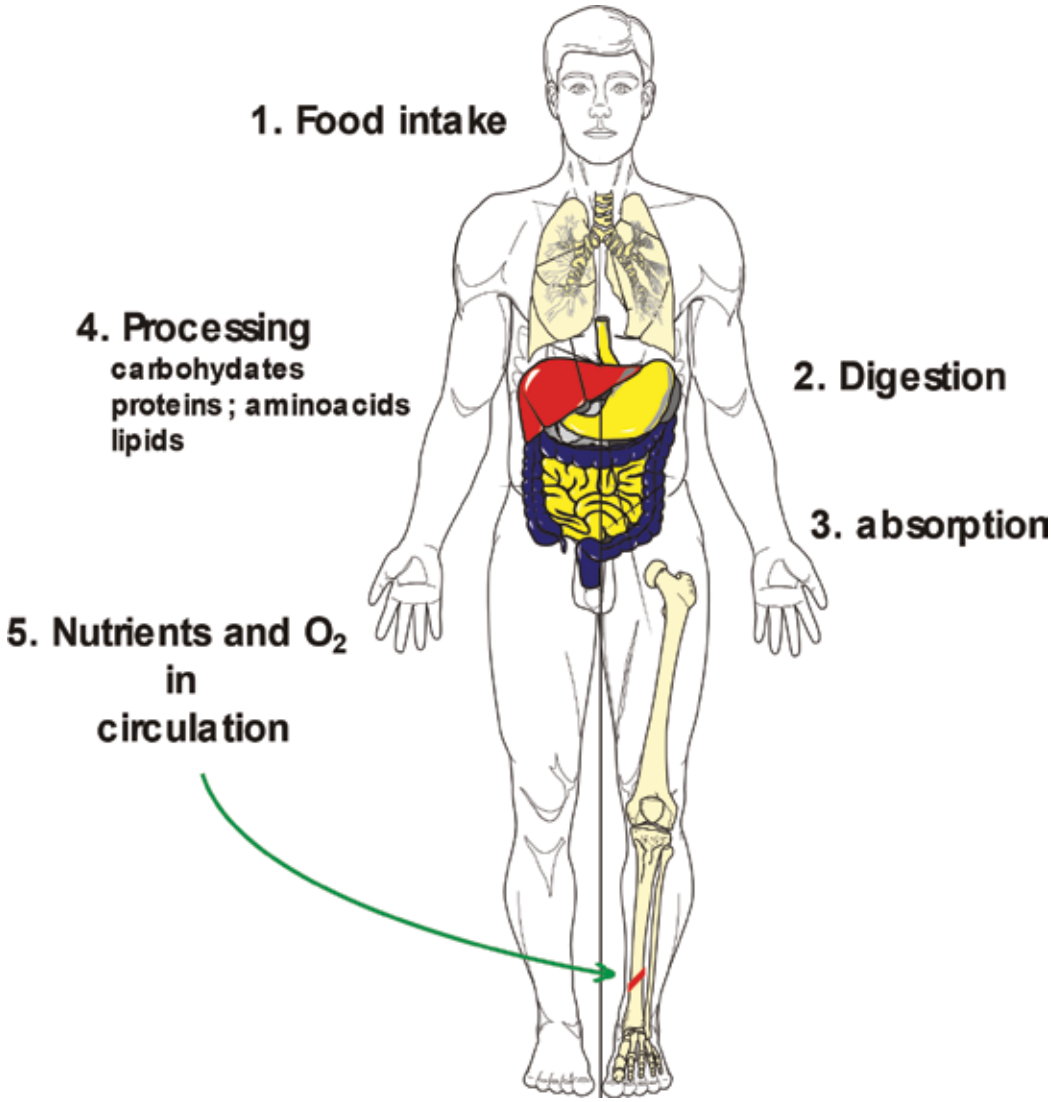

Figure 3. Oxygenation and nourishment of the fracture gap.

infection [41, 42]. An inadequate blood supply may also originate from central cardiovascular (stroke, cardiac arrest) insufficiency and peripheral vascular (i.e. atherosclerosis, venous thrombosis) diseases [43] (**Figure 3**).

Satisfactory healing requires an appropriate oxygenation and nutrition. Bone fracture corresponds with the disruption of its vasculature leading to the necrosis of 5 to 10 mm-width bone fragments adjacent to the fracture gap [44]. The area of necrosis may spread on the damage of the neighboring soft tissues, cardiovascular insufficiency (decompensated heart failure, arterial damage, or occlusion, venous thrombosis), anemia, or infection [45, 46]. Hypothermia also exerts an impact on local circulation constricting blood vessels [47].

Properly balanced diet provides all the nutrients, vitamins, and minerals that are necessary for healing. In case of bone fracture, an attention has to be paid over calcium, phosphates (osteoid formation), proteins (source of amino acids for collagen synthesis), and vitamin D_3 that may, in some cases, require supplementation.

Starvation is nowadays relatively seldom in developed societies, whose overweight and obese population alarmingly increases. Nevertheless, it could not be forgotten that it pertains only ca one-fourth of the Earth's population, whereas the next three-fourth suffers from hunger. Moreover, starvation and malnutrition may result from other than just a food shortage, reasons.

At the risk are especially elder, handicapped (also mentally) persons, drugs or alcohol abused, patients suffering from anorexia, and all others suffering from disturbed food intake, digestion, absorption, or processing. Thus, at risk are all those suffering from various digestive disorders, including short bowel syndrome, Hirschsprung's or Crohn's diseases, liver cirrhosis, pancreatitis and many others. Diabetes also leads to some type of starvation, as intracellular hypoglycemia deprives cells of glucose, the most important source of energy [48].

An increasing number of population implementing restrictive diet to reduce the body weight may present various nutritional deficiencies. Nevertheless, so far any religious (i.e. exclusion from the diet some kind of a meat) nor ideological (i.e. growing population of vegetarians and vegans) dietary restrictions nor customs were reported to influence bone healing. However, their negative insult, especially on young individuals, should be considered [49].

7.1. Habits disturbing healing of the fracture

Several habits affect the healing. Entering hundreds of detrimental substances, including highly toxic and carcinogenic ones, smoking impairs the function of progenitor cells, impairs local circulation, and reduces hemoglobin oxygenation disturbing reparative processes [50]. Alcohol was also shown to evoke its negative impact, but in small quantities may be beneficial supporting the fracture energetically and improving its perfusion [51, 52].

Several other addictions, including opioids, cannabinoids, and psychostimulants, indirectly influence the healing trajectories degrading the patient's psychosomatic status, and thus resulting in poverty, homelessness, malnourishment, and increased susceptibility to infections and additional injuries. Addicted persons have also limited access to health services, both due to social and economic reasons, and their irrational behavior. Moreover, some of them are not interested in successful treatment at all, as complications, when occur ease them to obtain social support.

7.2. Mechanical aspects of fracture healing

Immobilization and stabilization of bone fragments after their anatomical repositioning provide an optimal mechanical environment for the healing. Thus, casts splints, and orthopedic implants are the most effective methods of treatment.

They provide an optimal, biomechanical environment to the fracture gap, as excessive interfragmentary movements disrupt callus' vasculature. Newly formed blood vessels, built with the single layer of endothelial cells only, are very fragile. Irrespective of the direction (by side,

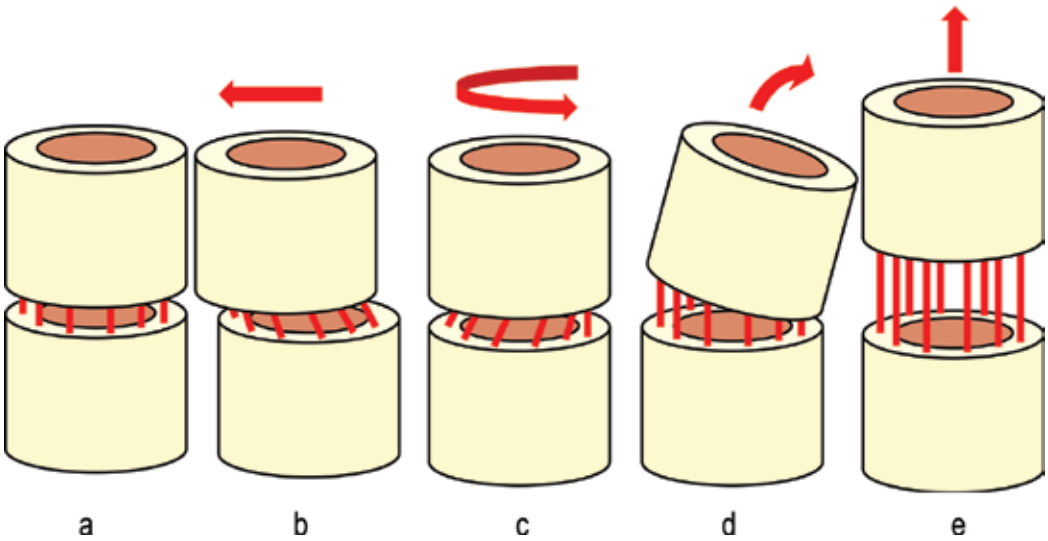

Figure 4. Fracture gap and its newly formed vasculature (red lines; a) dislocated by-side (b), rotation (c), angulation (d) and controlled axial movements (e).

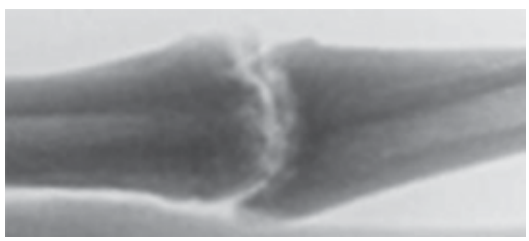

Figure 5. Hypertrophic non-union of the non-stabilized fracture.

angular, rotation or distraction), displacements disrupt the microvasculature that deprives the fracture of blood supply arresting the healing at the phase of cartilage ossification (**Figure 4**). In consequence, the cartilaginous pseudoarthrosis is formed presenting an abundant callus formation, the hypertrophic non-union [53]. It is usually observed in not immobilized fractures, but may also occur in stabilized ones due to implant's destruction (**Figure 5**).

However, rigid fixation precluding movements between bone fragments deprives them of mechanical stimuli that promote osteoblastogenesis [54]. Optimal amplitude of axial movements is below 1 mm, as those are beneficial for osteogenesis, but do not disrupt the blood supply. Other dislocations are detrimental.

It was shown that an excessive distance between bone fragments leads to the cessation of reparative processes leading to non-union [55]. The contact between bone fragments is reduced by a half when translocation reaches 6% of the bone's diameter or five degrees of angulation. Moreover, decreasing cortical thickness that characterizes osteoporotic bone aggravates the loss of interfragmentary contact (**Figure 6a-c**). That leads to the conclusion that fractures require accurate repositioning, especially osteoporotic ones [56].

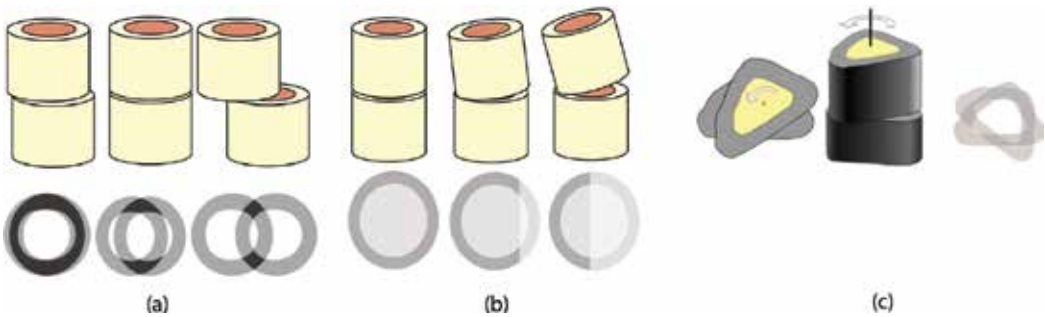

Figure 6. (a) An influence of by-side dislocations between bone fragments on bone contact. (b) An influence of angular dislocations between bone fragments on bone contact. (c) An influence of rotation between bone fragments on bone contact.

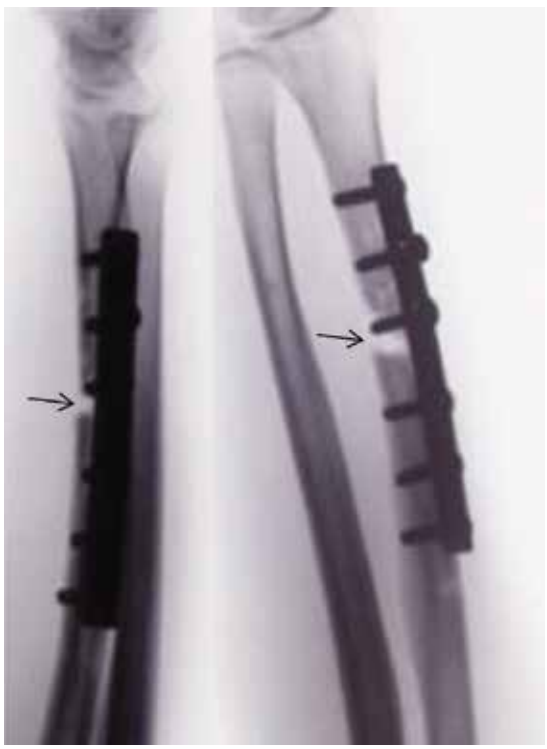

Figure 7. An inappropriate reduction of the fracture; Lack of the contact between bone fragments and screw situated in-between the fracture gap (arrows) disturb the healing.

To enable healing, the maximal distance between bone fragments should not exceed 1 mm, although the minimal is the best (**Figure 7**). Compression, shortening the distance between bone fragments, is nowadays implemented under several treatment modalities including

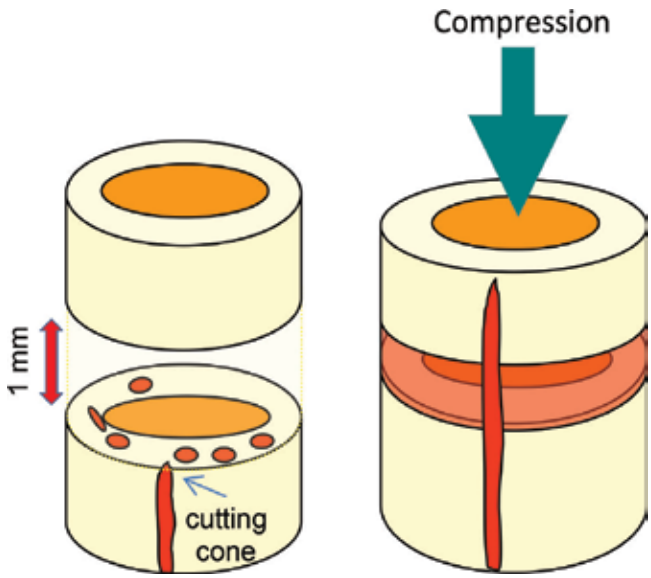

Figure 8. Schematic representations showing the role of compression of the fracture gap in bone healing.

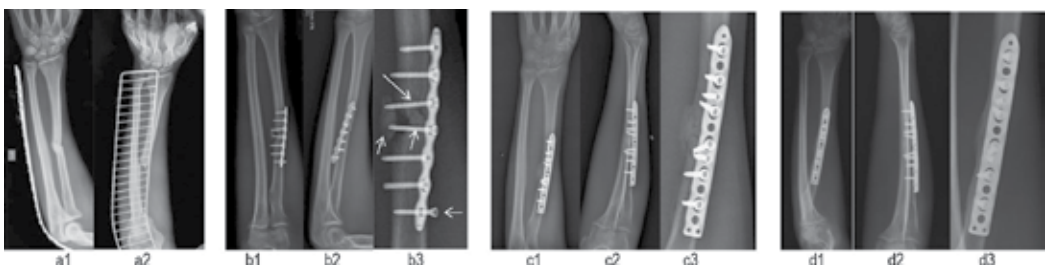

Figure 9. Disturbed healing due to an inadequate stabilization of the comminuted fracture of the left radius shaft in 26-years-old male. The fracture (a1 and a2) was primarily stabilized with the interlocking plate (b1 and b2), but lack of the contact between one of its intermediate fragments precluded the healing. Moreover, mechanical stability was not obtained, as both proximal and distal fragments were stabilized practically with two screws only. The third and the fourth screws situated in-between the fracture gap, were mechanically inefficient and foreclosing the healing; the seventh' was not screwed in at all (b3). Five months later, due to the lack of the progress of the healing (b), the fracture was revised, the fibrous scar was removed and marrow cavity was refreshed, bone fragments were reduced anatomically and stabilized with interlocking plate using four screws for each main fragment. Uneventful healing was observed on X-rays made on the 7th (c) and 12th (d) months after reoperation. Progressive healing (c3) and satisfactory remodeling (d3) were being observed.

compression screws and plates, pre-bending of Arbeitsgemeinschaft für Osteosynthesefragen (AO) plates, intramedullary and external stabilizations and so on [57]. Moreover, it mechanically stimulates osteogenesis (**Figure 8**).

Interposition of soft tissues or foreign material in-between bone fragments forms the barrier that precludes restoration of bone's continuity (**Figure 9**).

7.3. Pharmacotherapy

The negative impact of several pharmaceutics on healing processes was reported, including chemotherapeutics, antimicrobial drugs, steroids, heparins and antiresorptive drugs.

Chemotherapeutics are toxic to MSCs, reducing their number and activity and thus, depriving the fracture of osteoblastic progenitors. Their influence is aggravated by radiotherapy that is regularly used to treat neoplasms. Together with changed metabolism evoked by the tumor itself, those impair the healing.

Antibiotics were reported to affect the healing despite their beneficial capabilities to control infection. Tetracyclines were shown to impair ossification, thus arresting skeletal growth and fracture healing. Moreover, their negative impact prolongs for years, as bound with osteoid they impair bone remodeling decreasing its mechanical strength and thus, increase the risk of forthcoming fractures. Beta-lactams and cephalosporins, as well as ciprofloxacin, clindamycin, rifampicin, macrolides, and many others, and also evoke their negative impact. Their usage is justified as far as the positive antimicrobial effect is rationalized, that is weighed against negative influence on the reparative processes [58].

Corticosteroids, used in asthma, rheumatoid and dermatologic diseases, demineralize skeleton resulting in steroid-induced osteoporosis. In consequence, increased susceptibility to fractures, but also their impaired healing and remodeling, occur. Moreover, their chronic use threatens the bone viability bringing the risk of steroid-induced osteonecrosis [59]. Nevertheless, those unwanted side effects could easily be controlled modifying the route of administration and reducing their doses [60]. Analogically, nonsteroidal, anti-inflammatory drugs (NSAIDs), widely used analgesics, disturb reparative processes, usually expressing their negative influence, when chronically used at high doses [51]. Antihistamines were also reported to affect the healing [61].

Heparins, regularly used in trauma surgery for antithrombotic prophylaxis, are known to bind several growth factors including TGF-β and BMPs, FGF and EGF, decreasing their bioavailability for reparative processes [30]. Bisphosphonates, antiresorptive drugs dedicated for treatment of osteoporosis and prevention of fragility fractures, impair bone remodeling and healing, but also bring the risk of atypical fractures [62].

Other drugs were also shown to evoke negative impact on the bone union, including those used in the treatment of hypertension. Captopril, for instance, hinders angiogenesis and collagen deposition [63] and beta blockers affect wound healing through disturbed fibroblast proliferation [64].

7.4. Infection

Pathogens, colonizing the fracture gap, compete with its cells for nutrients, oxygen, and growth regulators depriving them of substances that are necessary for reparative processes. Moreover, hypoxia turns progenitor's differentiation into chondroblastic cell lineage, and pathogen-associated molecular patterns activate immune response aggravating the risk of non-union [65].

Unfortunately, eradication of microbes from the fracture gap is very hard, at least due to limited blood perfusion and poor antibiotic penetration. Moreover, they produce biofilms that protect them from recognition and counteraction by the immune system and antibiotics [66].

8. Final remarks

Healing of the bone fracture is a biological process that proceeds due to the cooperation of various cell lineages under the control of the molecular regulators. Since, it bases on mechanisms that were validated during skeletogenesis, everyone, who developed the skeleton properly, possess the mechanisms that enable him to heal the fracture. Thus, our role is just to provide optimal conditions for those natural mechanisms (**Figure 10**).

From the clinical point of view, an adequate supply of oxygen, nutrients, minerals, and vitamins under an appropriate biomechanical environment are the most important, as they enable those natural, biological mechanisms, to proceed uneventfully. Thus, fracture immobilizations or stabilizations, rational nourishment, improving circulation and local blood perfusion, withholding smoking, reducing alcohol intake, and rationalizing pharmaceutical medication are among the most effective activities that improve the healing. Factors that positively and negatively affect it were discussed above giving the clear suggestions for effective treatment. Unfortunately, several of them could not be corrected or just are above our limits. Nevertheless, in the vast majority of cases, one can introduce the treatment that could reduce of the risk non-union.

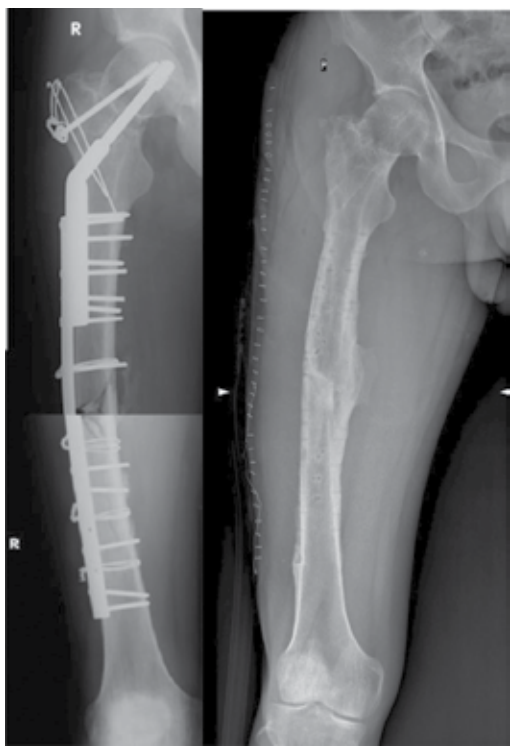

Figure 10. Comminuted, multiple-level fractures of the right femur in 41-years-old male (femoral neck, trochanteric, and the shaft) anatomically reduced and stabilized operatively. The final result (36 months) showing satisfactory bone union at all fractures after implants removal.

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Chapter 2

Management of Open Fracture

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Additional information is available at the end of the chapter

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Abstract

Open fractures are common and their prevalence is increasing in elderly people. The burden of open fractures is high because of economic and social costs. Most open fractures occur in lower limbs. The use of validated protocols, will optimize our outcomes when treating open fractures. The first step began with the proper identification of the fracture characteristics and the hidden soft tissue injury. The use of an adequate and early antibiotic prophylaxis is mandatory and then, we have to perform adequate irrigation and debridement. Finally, we have to decide to temporally fix the fracture or proceed with the definitive fixation method. Recently, the creation of dedicated "orthoplastic" units has increased the outcomes in high-energy tibial fractures. These fractures should be managed in adequate trauma centers that should be used to face all the complications that will appear during the reconstruction procedure because complications can be as high as 50% in high-energy open fractures.

Keywords: open, fracture, antibiotic, reconstruction, management, trauma

1. Introduction

Open fractures are common with an incidence of 30 open fractures for every 100,000 people every year, with an average age of 45 years [1]. Depending on the gender, we can distinguish two peaks: in males between 15 and 19 years and in females in patients older than 90 years [1]. Road traffic accidents are the main mechanisms of injury in these fractures, but we have seen an epidemiologic change in the last years because the incidence of open fractures related to motor vehicle accidents have decreased in the twenty-first century [2]. This lower incidence is

due to a high decrease in the number of open fractures in the occupants of motor vehicles probably because of the improvement of vehicles and traffic road security. This situation is in contrast with an increase, in the last years, in the incidence of open fractures in cyclist, motorcyclists and pedestrian accidents [2]. There is also a trend to see an increased incidence of open fractures in the elderly due to all mechanisms (high- and low-energy trauma) [1, 3, 4].

The presence of an open fracture is challenging because of several reasons. First of all, it is a complicated situation because of the generation of a bone defect or the presence of complex fracture patterns; second, we have to solve soft tissue coverage, and in some cases, we also have to recover the blood flow to the extremity.

The management of open fracture has evolved during the last few years, with the introduction of algorithms and the integration of the "orthoplastic" management, in several trauma units in hospitals all over the world [5, 6]. We can see in countries, like United Kingdom, the presence of national protocols to favor an early transfer of patients with these injuries to a trauma center, in order to improve the final outcomes (British Orthopedic Association Standards for Trauma 4 [BOAST 4]: The management of severe lower limb fractures).

In the case of multiple traumatized patients, open fractures should be individually addressed in order to minimize the general complications of a prolonged reconstructive procedure, minimizing the second-hit phenomenon in unstable patients [7–9]. The decision of limb salvage can be difficult to achieve, but in these situations, if we follow a validated protocol, we can optimize the chances of a favorable outcome.

In this chapter, we present the most recent evidence associated with the management of open fractures, with the objective of optimizing the management in these injuries, applying validated protocols in order to maximize the final outcomes obtained in patients with an open fracture.

2. Classification

Several classifications have been used to classify open fractures. We have chosen two from all of them because of the utility and spread through the orthopaedic community. The first one was described by Gustilo [10–12]. He distinguished three scales according to the energy of the mechanism of injury. The full description is reviewed in **Table 1**.

We can see in this classification that grade I injuries are simple fractures, usually with the skin disrupted from the inside because of the spike of the fracture, with limited contamination and good soft tissue coverage. Grade II injuries are usually the effect of a moderate trauma, with more soft tissue contusion and a more complex fracture pattern. Grade III injuries are the consequence of a high-energy trauma; we can find comminution and contamination in the fracture and an extensive soft tissue injury associated with periosteal striping. If the wound can be adequately covered and has no vascular injury, it is sub-classified as A. If the fracture cannot be covered by a soft tissue envelope, and we perform a rotational or free flap procedure to achieve coverage, we are talking about grade B injuries (**Figure 1**). It is important to underline

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	I	II	III-A	III-B	III-C
Energy of mechanism	Low	Moderate	High	High	High
Wound size	<1 cm	>1 cm	Usually >10 cm	Usually >10 cm	Usually >10 cm
Soft tissue injury	Low	Moderate	Extensive	Extensive	Extensive
Contamination	NO	Low	Severe	Variable	Variable
Conminution/ Fracture pattern	No/ Simple	Some/ Simple	Severe/ Complex	Severe/ Complex	Severe/ Complex
Soft tissue coverage	Yes	Yes	Yes	No, requires reconstructive procedure	Variable
Vacular injury injury	No	No	No	No	Yes, require reparation

Table 1. Summary of the Gustilo and Anderson classification, with the division of grade III fractures (red) in grade IIIA, IIIB and IIIC.

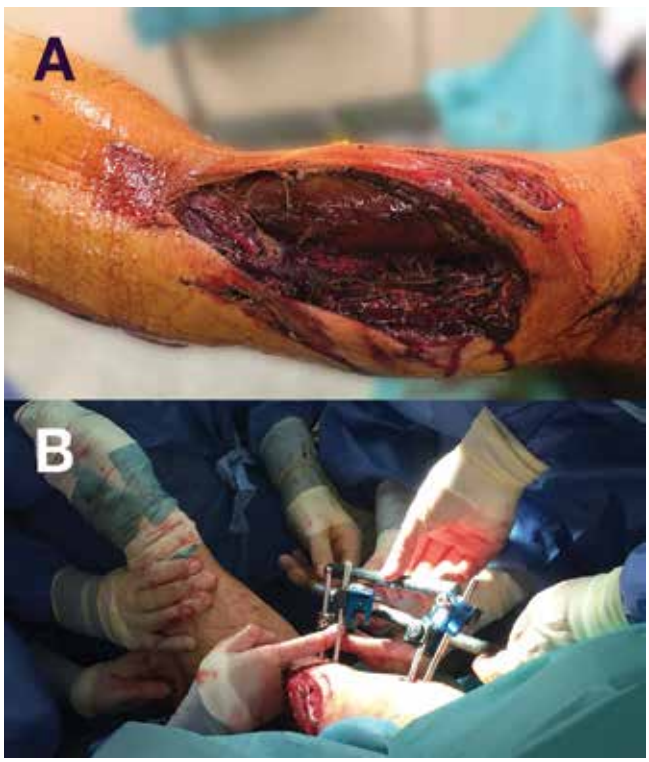

Figure 1. Image A: Clinical picture of an open IIIC fracture of the humerus. Image B: Photograph in the operative theater of the early and initial management, by temporary fixation with an external fixator, to facilitate vascular reconstruction and protection of the repair.

Skin	
1	Can be approximated.
2	Cannot be approximated.
3	Extensive degloving.

Contamination	
1	None or minimal contamination.
2	Surface contamination (easily removed not embedded in bone or deep soft tissues).
3a	Imbedded in bone or deep soft tissues.
3b	High risk environmental conditions (barnyard, fecal, dirty water etc).

Muscle	
1	No muscle in area, no appreciable muscle necrosis, some muscle injury with intact muscle function.
2	Loss of muscle but the muscle remains functional, some localized necrosis in the zone of injury that requires excision, intact muscle-tendon unit.
3	Dead muscle, loss of muscle function, partial or complete compartment excision, complete disruption of a muscle- tendon unit, muscle defect does not approximate.

Bone Loss	
1	None.
2	Bone missing or devascularized but still some contact between proximal and distal fragments .
3	Segmental bone loss.

Arterial	
1	No injury.
2	Arterial injury without ischemia.
3	Arterial injury with distal ischemia .

Table 2. Summary of the different conditions included in the classification of the Orthopedic Trauma Association.

that a partial skin or total skin graft is not considered as criteria to classify the fracture as grade IIIB. If we have to face a vascular injury that needs to be repaired, we are talking about grade IIIC. This classification has several utilities: first, it has a prognostic meaning [13], that is, the higher you move in the scale, the chances of infection and complication increase. The second is widespread and used worldwide, and it is clinically useful because it can guide the initial therapy when facing an open fracture.

In 2010, in the Journal of Orthopedic Trauma, an article was published proposing a new classification for open fractures based on a meticulous review of the literature made by the Classification Committee of the Orthopedic Trauma Association (OTA) [14]. This classification is useful to classify open fractures of the upper extremity, lower extremity and pelvis in adults and children in a clinically relevant way. This classification proposed five parameters to be measured: skin, muscle cover, contamination of the wound, arterial injury and bone loss (**Table 2**) [14].

3. Initial management

We have to be aware of several difficulties when initially treating an open fracture, the antibiotic treatment, the time until debridement and the decision of temporally external fixation versus definitive fixation. Most of the evidence of the management of open fractures in long bones is based on open tibial management because it is the most frequent bone involved in open fractures due to its location and characteristics [1].

3.1. Antibiotic prophylaxis

Antibiotic prophylaxis is one of the mainstays of open fracture management. From previous reports, we know that the most common pathogens involved in the colonization of open

fractures are Coagulase Negative Staphylococci [15], but depending on the geographic situation, the resistances of these bacteria may change, and orthopedic surgeons should identify the local resistances of the bacteria in their respective area. It is imperative to prescribe antibiotic prophylaxis as soon as possible [16, 17] because early antibiotics diminish infection rates in open fractures [17–19]. This is one of the easiest factors to improve in order to optimize the open fracture management in our clinical practice [20]. The British Orthopedic Association recommends to administering antibiotics within 3 h from the injury. There is also controversy about the perfect antibiotic prophylaxis in the treatment of open fractures. Local or national protocols are of high value if they are adequate to current evidence and population antibiotic resistance. The British Orthopedic Association (BOAST 4) suggests the use of Co-amoxiclav (1.2 g) or Cefuroxime (1.5 g) every 8 h and continue until wound debridement. We should choose clindamycin 600 mg every 6 h if there is a penicillin allergy. Other validated recommendations are the use of cefazolin and gentamicin [21] or piperacillin/tazobactam for 24 h after debridement [22]. Although the use of vancomycin is safe, it is still controversial except for patients allergic to penicillin because it seems that it does not have any benefit in patients with open fractures added to cefazolin [23]. A recent publication suggests a benefit in the use of early vancomycin powder in the wound (locally) to prevent biofilm formation [24]. Other strategies for antibiotic elution in the fracture site are being studied, for example, gentamicin-coated nails are promising, with low infection rates [25], or the use of gentamicin sponges [26].

3.2. Time of debridement

Time of debridement is also a constant controversy [16]. There was a "6-hour rule" in open fracture for early debridement, but recent publications have put this postulate in doubt. There is enough evidence that supports that time for debridement is not a main factor that conditions infection rates or outcomes [16, 27, 28]. This debridement can be safely performed in the first 24 h, and there is consensus to wait within this 24 h for the best conditions, ideally with an orthoplastic team to plan the reconstruction [6, 29]. Primary early closure of open fractures will improve outcomes and diminish septic complication [30].

3.3. Negative pressure wound therapy

The use of negative pressure wound therapy in open fractures that cannot be closed, in the first debridement, is an option that should be considered individually because despite there being evidence that favors its use as a temporary cover until definitive plastic reconstruction [31, 32], there is also a concern about its effect in bacterial growth and local antibiotic effectiveness [33]. Negative wound therapy is an alternative for temporary wound closure in those patients whose condition contraindicates the reconstruction (e.g. polytrauma patients who are not suitable for surgery). In those cases, we should maintain the dressings and change them in short periods of time [32]. A defined limit period of time to use negative pressure wound therapy is not clear, and despite its complications, it is reasonable to extend its use in cases of impossibility of soft tissue coverage because in these situations, it seems to decrease the complications when compared to wet dressings [31]. It is safe to proceed with the conversion

from external fixation to internal fixation, in the presence of negative pressure wound therapy, if we hold on the safe interval accepted for conversion from external fixation to internal fixation (less than 2 weeks) [34].

3.4. Initial fixation

Another point of conflict is the initial fixation method for open fractures, particularly, in femur and tibia. It is important to obtain an adequate fixation, in order to minimize pain, optimize wound and facilitate patient manipulation. It seems that grades I and II open fractures can be managed in a similar way like close fractures, with the adequate antibiotic prophylaxis and wound debridement and closure [18], and in the case of tibial fractures, the use of reamed intramedullary nails seems to be reasonable [35] and the use of temporary plating can be a trick to achieve anatomic reduction [36]. More controversies exist in grade III open fracture management. In grade IIIA, the use of non-reamed intramedullary nails seems to be a good and safe option (superior), compared to temporary external fixation in fractures with minimum bone defect, with minimized complications and good union rates [37, 38]. Recent reviews suggest that reamed nailing is not inferior to unreamed nailing in terms of function [38, 39] in grade IIIB open tibial fractures. In these fractures, there is evidence that supports the use of similar treatment options than those used for grade IIIA, and in those situations, early wound coverage is done and minimum bone defect is present [5]. If we are in the presence of a bone defect, the use of a protocoled treatment with temporary external fixation may be useful for definitive treatment. In the case of using an external fixator to temporary or definitive management, we should have in mind that future interventions should avoid damaging essential structures or compromise future reconstructive procedures. New modular devices allow us to achieve adequate fixation with different configurations and prevent inadvertent injuries or compromise future approaches.

Early management of IIIC and some IIIB fractures should be first guided by the need of amputation versus limb salvage, and this topic will be shown in a dedicated chapter. In the case of a IIIC open fracture, vascular repair is mandatory, and our efforts should be focused in obtaining a quick and stable fixation to protect the vascular repair (**Figure 2**). In these cases, we should also consider early preventive fasciotomies to prevent compartment syndrome caused by a revascularized ischemic limb.

3.5. Compartment syndrome

The suspicion of a compartment syndrome should always be present in high-energy trauma, especially in non-conscious patients. Compartment pressure should be measured in case of doubt in these patients, and if there is an increase, or high clinical suspicion, a fasciotomy should be performed [40, 41]. Compartment syndrome is more frequent in young patients with closed fractures, managed by external fixation and intramedullary nailing, but it can also develop in an open fracture, particularly if we closed the fascial compartment with tension [42]. The use of drains and lax closure (or non-closure) of the fascia will help to prevent the increase of the fascial compartment pressure. The use of continuous pressure monitoring

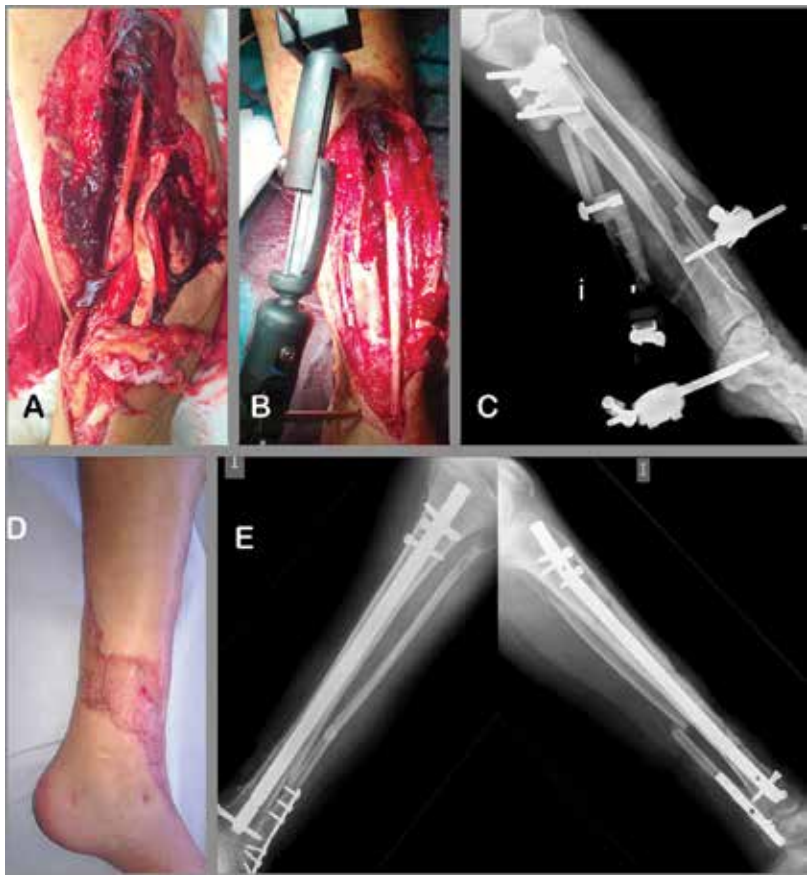

Figure 2. A complete reconstructive procedure in an open fracture grade IIIB tibial fracture, in a 43-year-old female. Image A: photograph in the emergency department of a grade IIIB open fracture in the emergency department. Image B: photograph in the operative theater, temporary fixation of the fracture with an external fixation. Image C: X-ray of the temporarily stabilized fracture with an external fixator, after soft-tissue and bone debridement. Image D: the final soft tissue coverage of the injury. Image E: an anteroposterior and lateral X-ray, after definitive fixation of the fracture with a nail.

seems to be an option for selected patients, but it is not efficient to use routinely in all patients [43, 44].

3.6. Referral to a trauma center

If we have to treat a complex open fracture, in a center without the resources to make the reconstruction, it would be a good option to complete antibiotic prophylaxis, immobilize the injury in a proper way after an initial clinical assessment and refer the patient to a trauma center as soon as possible, especially in the presence of a vascular injury that we would not be able to repair. In the case that the patient needs a long transfer to the definitive centre, or the initial debridement surgery would not be performed within 24 h, it is considered a good option to temporary apply an external fixation, perform the irrigation and debridement and then transfer the patient to a trauma centre to perform the reconstructive procedure.

4. Definitive management

There are several options to finally address an open fracture, and we should select the method appropriate for the clinical situation. In the case of polytrauma patients and high-energy trauma, most of the patients are treated with temporally external fixation and later with definitive conversion to internal fixation (**Figure 2**).

Depending on the situation, we should choose the appropriate moment to perform this conversion to internal fixation. In the case of unstable patients, the best moment for internal conversion is given by patient situation and systemic status, but it is safe to perform this conversion in an interval of time lesser than 14 days [45]. There are several articles that calculate the infection rate of the conversion from external to internal fixation, with percentages that move between 0 and 40, depending on the interval between the injury and the definitive internal fixation with a nail, in long bones of the lower limb (**Table 3**).

In the case of upper extremity, the use of plates is more common, and in humeral open fractures, it is safe to perform the conversion from external fixation to a plate during the first 2 weeks after the injury, with a reasonable complication rate [56].

In some circumstances, we may have to treat a fracture fixed with an external fixator for a long time (more than 4 weeks). In this situation, it is reasonable to retire the external fixator, use an orthosis or a cast, and wait for 2–4 weeks to perform the definitive internal fixation, if there are no septic complications. Another option is to use an external fixator as the definitive device to treat the fracture; this is a good option in patients who are not amenable for internal fixation.

Author	Year	N	Open fractures (%)	Days	Infection (%)
Gill	2016	84	100	12 (7-14)	7
Galvin	2015	125	0	7	2,5
Moody	2009	58	88	9	26
Harwood	2006	98	29	14	5,4
Nowotarski	2000	59	32	7	1,7
Scalea	2000	35	Unknown	7	2,3
Paderni	2001	5	83	50	40
Winkler	1998	43	30	12	2,3
Hontzsch	1993	62	74	15	0
Marshall	1991	4	33	Unknown	0
Wu	1991	15	100	5	13

Table 3. A review of articles is given, which focuses on the complication rate, after conversion from external fixation to internal fixation [34, 46–55].

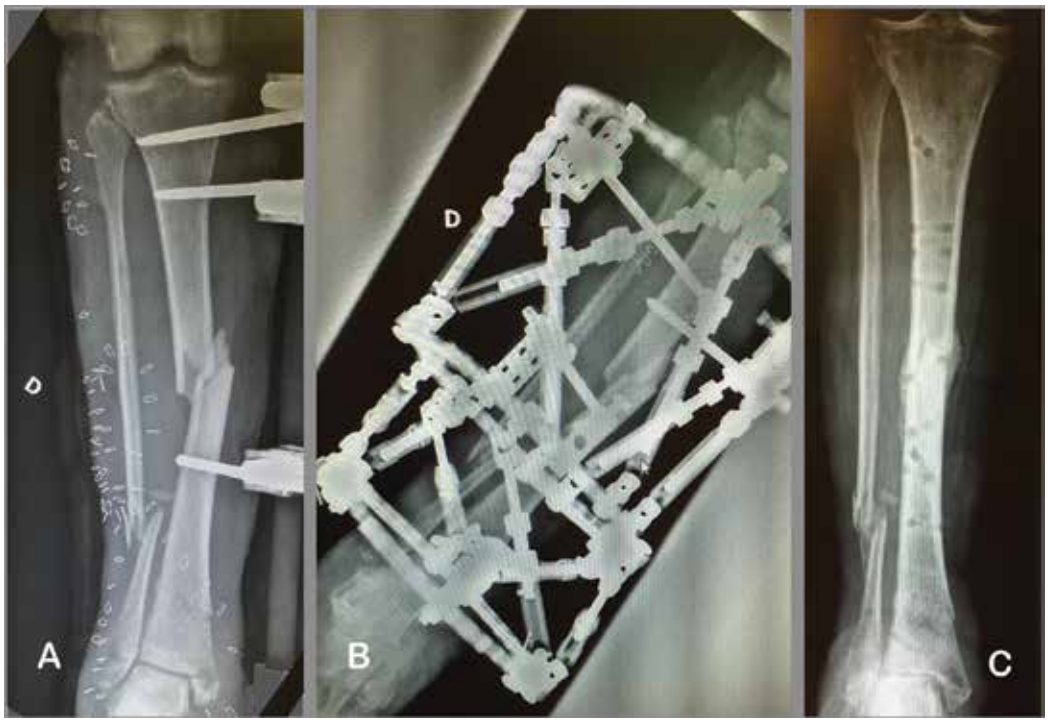

Figure 3. The treatment of an open grade bifocal IIIB fracture in an 82-year-old female. Image A: initial X-ray of the patient who was treated with an external fixation for 3 months, with no callus formation and development of a malunion. The patient required multiple coverage procedures during this period of time. Image B: initial X-ray after the patient was treated with a Taylor Spatial Frame, correction begun. Image C: X-ray after 6 months since the implantation of the TSF. The fracture is healed, good alignment and full soft-tissue coverage is achieved.

The use of computer-assisted orthopaedic devices will help to correct and treat sequelae caused by temporally external fixation (**Figure 3**).

5. Limb salvage or amputation

Most evidence about the decision between limb salvage and amputation is obtained from the lower extremity assessment project (LEAP) study and war-related trauma studies. Several rating scores have been proposed to facilitate the decision, for example, the OTA classification for open fractures, the Mangled extremity severity score or the Ganga Hospital Score [57–60]. Recent publications have demonstrated that these scores should be reviewed to include the new therapeutic advances to prevent amputation and improve the sensitivity and specificity of these scores [61], so these scores alone should not be the only criteria to make our decision.

Because of legal reasons, it is important to include in the clinical records, the anamnesis and graphical documents of the injury, especially in those cases we have decided to perform amputation.

From previous studies, we know that in case of limb conservation, we will face a secondary amputation rate of 3.9%, a complication rate near 40% (10% infections), a 24% non-union rate and an 8% of long-term osteomyelitis. In the case of amputation, we have to consider a reamputation rate of 5.4% and a 25% complication rate in the first 3 months (1/3 infections). At 7 years post-injury, patients treated with amputation or limb salvage procedure were found to have similarly poor outcomes [62], but costs were higher for amputee patients because of the cost of the prostheses [63].

The factors that can modify outcomes, in patients with mangled extremities, are numerous: tobacco consumption is one of the most important, with an increase of 37% in the non-union rate, an increase higher than two times in infection and almost four times in osteomyelitis. Ceasing smoking will improve the union rate, the infection rate and the risk of osteomyelitis, but patients will never have the same risk as a non-smoker. Personal status, education level, gender, age, economic status and patient self-esteem are pre-lesional factors, and worker compensations, depression, SIP score, walking speed, pain and aggressive physiotherapy are post-lesional factor that will modify the outcomes [64–68].

6. Complications

Complication rates in open fractures are dependent on the type of injury. Commonly, we can say that complication rates increase with the Gustilo classification [13, 69]. We can expect a high complication rate for most grade III open fractures, but only a small increase in complications for grade I and II open fractures [69].

For grade I fractures, we can assume a complication rate near close fractures, if they are managed in a proper way, and patients will recover fast [70].

In grade II fractures, the complication rate would be slightly higher than seen in close fractures or grade I [13]. We will appreciate a low increase in the infection rate, and patients would experience a delay in the time until full recovery [70, 71]. In these situations, the use of a plate, if indicated, would not increase the infection rate [71].

In the case of tibial open fractures, from grades IA to IIIA, if they can be managed initially by a nail, with a proper protocol, the complication rate would be similar to close fractures in the first 30 days after the injury [72].

In limb-threatening injuries, if we choose a salvage procedure, we can expect a high complication rate. If we look for the lower extremity assessment project (LEAP) study group outcomes, we will find wound infections in 23% of patients, 31% of non-unions, 5% of malunions and 9% of osteomyelitis [73] in these injuries. These complications would decrease functions in these patients.

Complications would also favor the development of secondary complications, for example, if a patient suffers an infection, he/she will also increase the non-union and delay healing rate (with an odds ratio higher than 4) [74].

7. Conclusions

Open fractures can produce a huge disability in patients. The use of evidence-based protocols and treatments will help us to optimize patient's outcomes. Centers used to manage high-energy trauma with an "orthoplastic" team will achieve the best results in open fractures, specially grade III, and will be prepared to manage the devastating complications that will appear during the reconstructive steps of these fractures.

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Conflict of interest

Authors declare that they do not have conflict of interest.

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Chapter 3

Clinical and Radiological Assessment of Acetabular Fracture

Balaji Zacharia, Dhiyaneswaran Subramaniyam and Muhammed Ayyub

Additional information is available at the end of the chapter

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Abstract

Acetabular fractures are one of the rare injuries. They are usually occurring following a high energy trauma. The type of acetabular fractures is mainly depend on the position of the femoral head at the time of injury. The acetabular fracture are usually associated with visceral and neurovascular injuries. There can be anterior, posterior or central fracture dislocation associated with this injury. A closed degloving injury of the subcutaneous tissue which is detached from the underlying fascia-Morel-Lavelle lesion is also common feature. The sciatic nerve injury can be associated with posterior wall or column injuries. The most commonly used investigation are anteroposterior, Judet views and 2D/3D computerized tomography. Most commonly used classification is modified Judet and Letournel classification. They divided acetabular fractures into five simple fracture patterns and five associated fracture pattern. The Orthopedic trauma association modified Letournel classification and gave computerized coding. In this chapter, we are describing the clinical features and classification of acetabular fracture in a simple and vivid manner.

Keywords: acetabular fractures, Judet and Letournel classification, Morel-Lavelle lesion, Judet views, spur sign

1. Introduction

Acetabular fractures are one of the rare injuries. The incidence of acetabular fracture is about 3 per 1 lakh population. There are bimodular pattern of occurrence of acetabular fractures. In elderly patients, it is usually produced by low energy trauma and in younger patients it is produced by high energy trauma. The pattern of fracture is mainly determined by the

position of femoral head at the time of injury, force vector and quality of bone .This fracture are usually associated visceral and neurovascular injuries. In this chapter, we are going to describe the clinical feature and classification of acetabular fractures.

2. Clinical features

The most common cause of injury in acetabular fractures is road traffic accidents and fall from a height [1, 2]. Usually acetabular fractures are associated with other major visceral injuries. There are very few literature available on the relationship of non-orthopedic injuries associated with acetabular fractures. Even though most of the acetabular fractures are caused due to axial compression along the femur, driving the femoral head into the acetabulum [3]. In a study on patients with combined pelvic and acetabular fractures, Dalal et al. reported 27% with traumatic brain injuries, even though there was relatively low rate of abdominal injuries [4]. According to him, some of the causative energy of the injury pattern is distributed to long bones before it reaches pelvis and torso, thus producing lower extremity fractures. Most of the posterior wall or column fractures are caused by axial forces transmitted to the acetabulum by knee and femur with the hip in adducted and flexed position—Dashboard injury. The other fracture pattern is caused by direct lateral compression force transmitted through the trochanter or indirect axial force along the femur in extended position of hip joint [5, 6]. In yet another study, it was noted that axial load pattern of injuries were associated with lung injury, retroperitoneal haematoma, traumatic brain injury and lower extremity fractures whereas incidence of genito-urinary injury, hepatic and splenis injury, pelvic vascular injury are more associated with lateral compressive type of acetabular fracture [7].

Life threatening injuries like head injury, abdominal and other visceral injury must get priority in the initial evaluation of acetabular fracture. Other skeletal injuries, like fractures of patella-ipsilateral shaft of femur, tibial plateau fractures and knee ligamentous injury are also looked into. The lower limb will be flexed, adducted and internally rotated in posterior dislocation of hip which is usually associated with posterior wall or column fracture whereas it will be in abducted, extended and externally rotated in anterior dislocation. Lateral displacement of the anterior superior iliac spine on the affected side might give clue regarding central fracture dislocation of hip. Local injuries including skin, open acetabular fractures and perineum and scrotal injury must be excluded. A closed degloving injury of the subcutaneous tissue which is detached from the underlying fascia—Morel-Lavele lesion must be looked into because of the risk of high infection and wound healing in post-operative period. These lesions are not apparent initially but become evident later [8]. A careful neurological examination to rule out sciatic and common peroneal nerve injury should be done. There are isolated reports of injury to iliofemoral artery associated with high anterior column fracture and superior gluteal artery injury in displaced fracture into the greater sciatic notch. Hence, high index of suspicion should be there to rule out vascular injury of the ipsilateral lower limb [9, 10].

Sometimes acetabular fracture can produce profuse bleeding and shock. According to Letournel and Judet "the cause of shock relates to severity of trauma and to hemorrhage from the fracture site and often from other visceral lesions. The respective part played by the various elements producing the shock may be difficult to apportion" [4]. According to Dalal et al. patients with

acetabular fractures needed lower transfusion rate than with pelvic fractures. But in yet another study out of 16 acetabular fracture, 2 fracture required embolization compared to 9 of the 100 pelvic fractures. The transverse and posterior wall fractures often require blood transfusion. Both column fractures, T-shaped fracture, fracture with extension into the sciatic notch and those fracture associated with pelvic injury are more likely to have greater blood loss and may need blood transfusion [11].

3. Radiological anatomy

Accurate classification of acetabular injury is important for its proper treatment. Various classification system have been suggested because of the complex anatomy of the acetabulum. Judet and Letournel classification is the most widely accepted and commonly used classification. Radiograph provides most of the essential information for classifying acetabular fractures. Computerized tomography with 3D reconstruction imaging is also useful in classifying acetabular fractures [12–16].

The acetabulum is an incomplete hemispherical socket with an inverted horseshoe shaped articular surface surrounding the non-articular cotyloid fossa. The articular socket is composed of and supported by two lamda [λ] shaped columns as described by Judet and Letournel (**Figure 1**). The anterior and posterior walls are extensions of the respective column and forms the cup of the acetabulum [16–18]. The anterior column is longer and larger and composed of

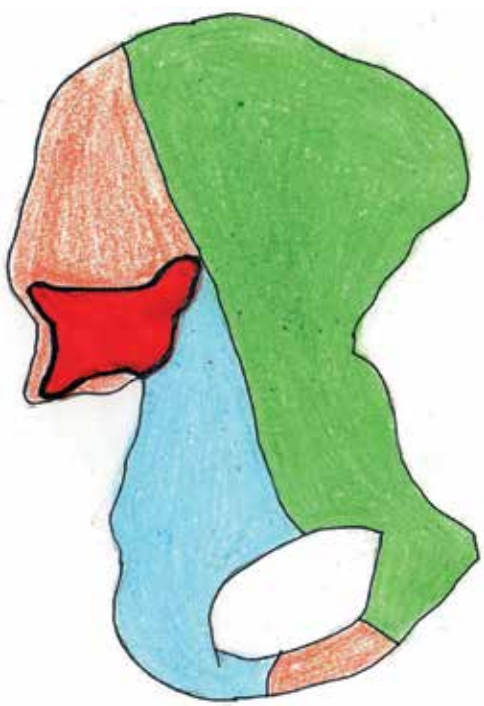

Figure 1. Columns of acetabulum. Blue—Posterior column. Green—Anterior column. Red—Sciatic buttress.

iliac crest, iliac spine, anterior half of the acetabulum and pubis. The posterior column extends superiorly from the ischiopubic ramus and consists of ischim, ischial spine and the posterior half of the acetabulum and the dense bone forming the sciatic notch. The anterior and the posterior columns meet at the sciatic buttress. The sciatic buttress extends posteriorly from anterior and posterior column to become the articular surface of the sacroiliac joint thus connecting the columns with the axial skeleton. The roof (dome) of the acetabulum is the weight bearing area that supports the femoral head. The quadrilateral plate is flat thin bone which is forming the medial wall of acetabulum. The iliopectineal eminence is the prominent part in the anterior column that lies directly over the femoral head.

4. Radiological evaluation

The radiographic evaluation of the acetabulum includes an anterio-posterior (AP) view and Oblique (Judet) views [19]. Minimal individual variations of the normal articular area can be visualized by comparing the anatomical landmarks of both hips. For example, comparison of the medial clear space between the head and tear drop will give a clue regarding femoral head subluxation. The iliopectineal line represents the anterior column and ilioischial line represents the posterior column. A line joining the lateral edge of sourcil to lateral edge of the inferior tear drop represents the anterior wall and a line representing the lateral edge of superior acetabulum to the lateral sclerotic area in the inferior margin of acetabulum represents the posterior wall [20].

In the AP radiograph, we have to look into certain radiological landmarks.

1. Iliopectineal line.
2. Ilioischial line.
3. Tear drop (formed laterally by inner wall of acetabulum and medially by quadrilateral plate).
4. Roof.
5. Anterior wall.
6. Posterior wall.

The iliac oblique view will clearly show anterior wall and posterior column, quadrilateral surface and posterosuperior roof arc. The obturator oblique view will show posterior wall, anterior column and anteriosuperior roof arc. The obturator ring represents parts of both columns, and fracture of the obturator ring may be associated with other pelvic injuries like lateral pelvic compression injury or sacral fracture.

Evaluation of the acetabular fracture is made easier by both 2D and 3D CT scan [21]. The entire pelvis is usually included in the tomogram to avoid missing a fracture and comparison of the opposite side. The newer spiral CT scan of 1.5 mm and continuous bone section through the pelvis and acetabulum can be obtained. The 3D reconstruction view will help the surgeon to understand the fracture pattern and its anatomical location inter-operatively. Fracture lines in sagittal and coronal planes, marginal impaction, intra-articular loose fragments, concentricity

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of the reduction, rotation of the articular fragment and other associated pelvic injuries can be very well visualized in CT scan. Fracture lines in the sagittal plane represent transverse and wall fractures. Fracture lines in the coronal plane represent column fracture and the vertical limb of T-fracture. Magnetic resonance imaging (MRI) has no value in the immediate evaluation or classification, but is useful in determining the vascularity of femoral head at a later date [22].

5. Classification

The universally accepted classification of acetabular fracture was described by Judet and Letournel in 1964 and later modified by Letournel [12, 14]. This classification helps the surgeon to approach acetabular fracture in a more anatomical and fracture biomechanics. The Orthopaedic Trauma Association (OTA) modified Letournel classification and gave computerized coding according to AO comprehensive classification of fractures of long bones [23]. According to Judet and Letournel classification, acetabular fractures are divided into two basic groups: simple fracture type and complex fracture type. Each group consists of five types. The simple fracture pattern includes isolated anterior and posterior wall fractures, isolated anterior and posterior column fractures and transverse fracture (**Figure 2**). The Associated fracture pattern includes T-type fracture, combined fractures of posterior column and wall, transverse fracture with posterior wall fracture, anterior column with posterior hemitransverse fracture and bicolumnar

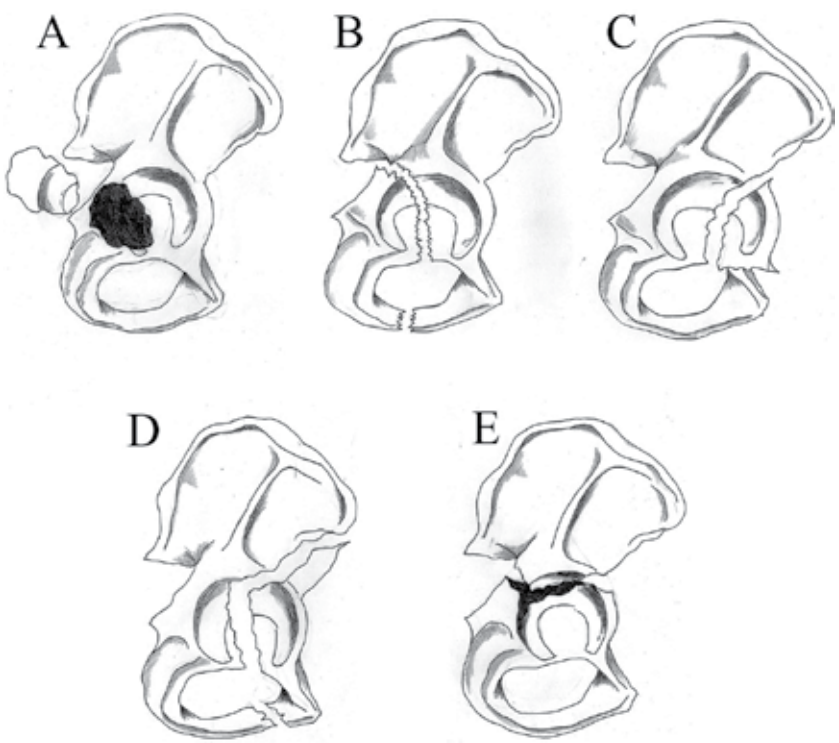

Figure 2. Simple acetabular fractures. (A) Posterior wall fracture (B) Posterior column fracture (C) Anterior wall fracture (D) Anterior column fracture (E) Transverse fracture.

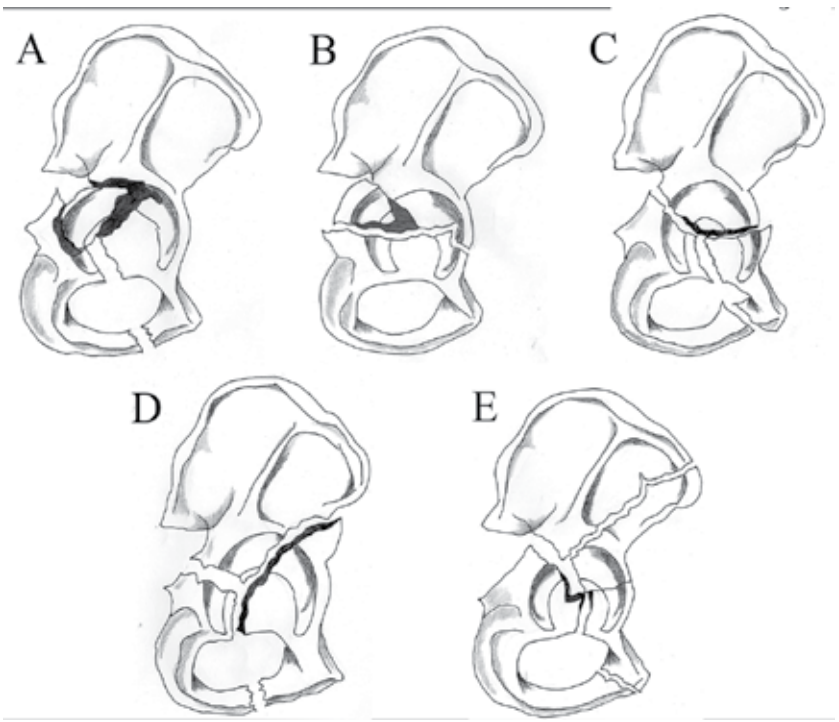

Figure 3. Associated fractures. (A) Posterior wall with posterior column fracture (B) Transverse with posterior wall fracture (C) T shaped fracture.(D) Anterior column with posterior hemitransverse fracture.(E) Bicolumn fracture.

fracture (**Figure 3**). There is one exception in each group. The transverse fracture in the simple group involves both columns and the anterior column with posterior hemitransverse fracture involves only one column in complex group [24].

6. Simple fractures

6.1. Posterior wall fracture

This is the most common type of acetabular fracture. It involves disruption of the posterior wall which can be single or multifragmentary. There can be marginal impaction or acetabular depression fracture commonly associated with the posterior dislocation of the hip joint and radiologically identifiable disruption of the posterior wall with break in the sagittal plane of CT scan.

6.2. Posterior column fracture

Characterized by disruption of the ischial portion of the pelvis and fracture line usually extends through the sciatic buttress. The fracture line usually extends from obturator foramen to greater sciatic notch through weight bearing dome of the acetabulum. The iliac oblique view shows

break in the ilioischial line and displaced articular cartilage along with posterior segment with an intact portion of roof, creating an image resembling a flying bird (Gull sign) [11]. In CT scan, fracture line will be seen in the posterior column in the coronal plane of axial section.

6.3. Anterior wall fractures

It involves disruption of anterior wall. The radiograph demonstrate disruption in the iliopectineal line which is best seen in iliac oblique view. CT scan demonstrates fracture in sagittal plane of axial cut.

6.4. Anterior column fracture

The fracture disrupts iliopectineal line. The fracture line extends from anterior iliac crest to superior pubic ramus disrupting the obturator formen. Obturator oblique view will show fracture line disrupting the anterior column. In high or intermediate anterior column fractures, the roof segment usually displaces medially. CT scan shows fracture in coronal plane in the anterior column of axial section.

6.5. Transverse fracture

This fracture separates acetabulum into two segments transversely. It involves both columns with intact obturator foramen. This fracture is subdivided into three types according to the level of fracture, namely infratectal, juxtatectal and supratectal [11] (**Figure 4**). Radiographically, both iliopectineal and ilioischial lines will be disrupted. In CT scan, it is characterized by sagittal-oriented fracture line extending through both columns.

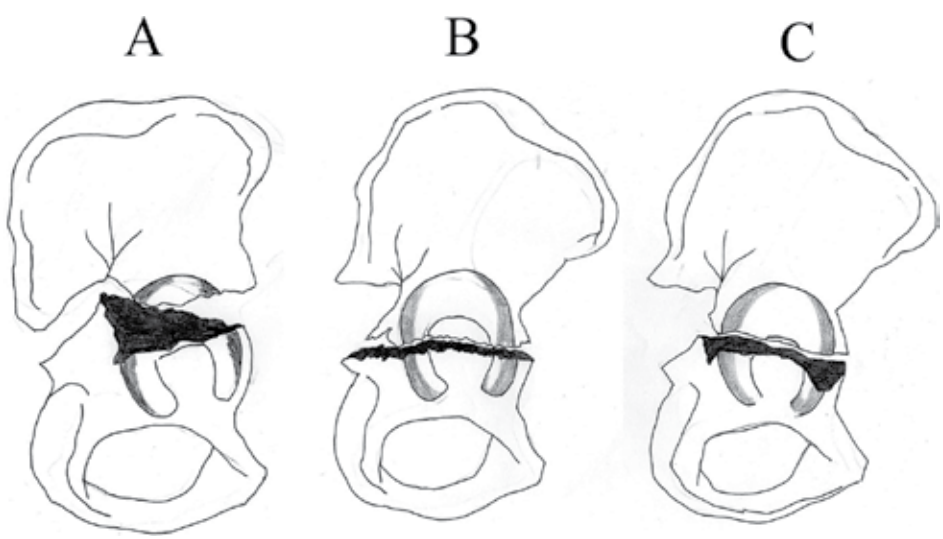

Figure 4. Types of transverse fractures. (A) Supratectal fracture (B) Transtectal fracture (C) Infratectal fracture.

7. Associated Fracture Patterns

7.1. T-shaped acetabular fractures

T-shaped fracture is a combination of transverse acetabular fracture with vertical extension into obturator ring distrupting it. Sometimes there is extension of fracture into pubic ramus and ischim. The vertical fracture component differentiates this fracture from transverse fracture. But the superior extension of the fracture does not involve the iliac wing which is an important point to differentiate this fracture from both column fracture. On the radiograph, both iliopectineal and ilioischial lines will be disrupted *in* both Judet views. On the CT scan, the transverse component will be seen in sagittal plane of axial image.

7.2. Both column fracture

There are many fracture patterns which involves both column like transverse, T-shaped fracture, transverse with posterior wall but all these fractures have to be differentiated from both column fracture. It is one of the commonest acetabular fracture pattern [13].This fracture is differentiated from all other fracture patterns in that articular surface is completely detached from the remaining portion of the iliac wing which articulate to the sacrum. The anterior and posterior columns separate from each other. It is actually a fracture involving anterior and posterior columns with fracture line extension into the obturator ring and iliac wing. Obturator ring and iliac wing involvement is a must for diagnosing this fracture pattern. Sometimes, CT scan is the only investigation to identify this fracture type. The pathognomonic sign of both column fracture is the presence of spur sign in obturator oblique view, which means the remaining portion of the ilium attached to the sacrum with sciatic buttress is seen projected lateral to the medially displaced acetabulum. Central dislocation of the femoral head is usually seen in the AP view. CT scan shows fracture involving both the columns with disruption of obturator ring and inferior pubic ramus. The main fracture line extending superiorly from the acetabulum into the iliac wing is seen in the coronal plane.

7.3. Transverse fracture with posterior wall

This fracture pattern has the characteristics of both transverse and posterior wall fractures described above. It will not disrupt the obturator foramen. In the radiograph both iliopectineal and ilioischial lines will be disrupted. Additional fracture in the posterior wall will also be seen. Obturator oblique views and CT scan will help to identified the posterior wall fracture.

7.4. Anterior column with posterior hemitransverse fracture

The fracture line usually extends from antero-inferior iliac spine and passes inferiorly through the cotyloid fossa exiting at the superior ramus disrupting the obturator foramen. A pure transverse fracture of the posterior column alone is seen with it. It is sometimes referred as reverse T-fracture. Usually the transverse component will be undisplaced. The ilipectineal line will be always disrupted. Obturator oblique view is the most ideal view to pick up this fracture in radiograph. CT scan helps to distinguish it from transverse and T- fractures.

7.5. Posterior column with posterior wall fracture

The fracture line will extend from the sciatic notch to the obturator ring through the cotyloid fossa. The AP view shows disruption of ilioischial line. The obturator view shows posterior wall fracture and iliac oblique view shows posterior column fracture.

Comprehensive classification has been developed to standardize the nomenclature world-wide. An alpha-numeric classification system was developed by AO group for acetabular fractures based on its severity.

Type A-Fracture involving single wall or column.

Type B-Fracture involving both anterior and posterior columns (T-type and transverse fracture).

Type C-Fracture involving both anterior and posterior columns, but all articular segments including the roof are detached from *the* remaining segment of intact ilium (Both column fracture).

The evaluation and classification of acetabular fractures based on Judet and Letournel can be concluded like this:

A. Acetabular fracture with intact obturator ring. The possibilities are:

1. Anterior wall fracture (iliopectineal line alone disrupted with anterior wall disruption).
2. Posterior wall fracture (posterior wall disruption).
3. Transverse fracture (Both iliopectineal and ilioischial lines disrupted).
4. Transverse with posterior wall fracture (both iliopectineal and ilioischial lines with posterior wall disruption).

B. Acetabular fractures with obturator ring disrupted are:

1. Anterior column fracture (iliopectineal line disruption).
2. Anterior column with posterior hemitransverse (both iliopectineal and ilioischial line disruption).
3. Posterior column fracture (Ilioischial line disruption).
4. Posterior column with posterior wall fracture (Ilioischial and posterior wall disruption).
5. T-shaped fracture (both iliopectineal and ilioischial line disruption).

C. Acetabular fracture with obturator foramen disruption with fracture line extending to the iliac wing.

1. Bicolumn Fracture (Spur Sign).

AO comprehensive classification of the fractures acetabulum.

Type A: Partial articular, one column involved.

A_1-Posterior wall fracture.

A_2-Posterior column fracture.

A_3-Anterior wall or anterior column fracture.

Type B: Partial articular fracture.

B_1-Transverse fracture.

B_2-T shaped fracture.

B_3-Anterior column + posterior hemitransverse fracture.

Type C: Complete articular fracture.

C_1-Both column fracture, high variety.

C_2-Both column fracture, low variety.

C_3-Both column fractures involving the sacroiliac joint.

In a review of 229 patients with acetabular fractures by Herman et al. found certain fracture patterns, which cannot be included in any one of the Judet and Letournel classification. They have proposed a newer classification system based combination between the displacement vector of the fractures and the specific fracture architectural structures. But further studies are required to evaluate the utility of the proposed classification [25].

8. Principles of treatment

The aim of treatment of any acetabular fracture is to get anatomical reduction and stable fixation and early mobilization and delayed weight bearing. This can be achieved either by operative or non-operative treatment. In displaced fracture more than 2 mm, the roof arc angle <30°, Failure to achieve and maintain concentric reduction by closed methods, incarcerated or impacted intra-articular fragments are indication for open reduction. If there is vascular or sciatic nerve injury developing following a closed manipulation such situation warrants emergency ORIF. In patients with poor bone quality, late presentation, medical contraindication, severely communited fracture, secondary congruency can be treated non operatively. Most of the fractures are reduced using the principle of ligamentotaxsis. Interfragmentary screws are used for rigid fixation which should be protected using reconstruction plate in the neutralization mode [26].

Contributions

All authors helps in collecting data, analysis, statistics, writing and editing the manuscript. We have no conflict of interest for this manuscript and we have not accepted any financial assistance from within or outside of our institution for collecting data,writing manuscript and for its publications

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Chapter 4

Maxillofacial Fractures: From Diagnosis to Treatment

Mohammad Esmaeelinejad

Additional information is available at the end of the chapter

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Abstract

Oral and maxillofacial fractures are common injuries among multiple trauma patients. Mid-face fractures are considered serious medical problems rather than all other maxillofacial injuries due to their complexity of management. An appropriate treatment plan is essential to reconstruct the mid-face aperture esthetically and functionally. Favorable results are provided by interdisciplinary approaches and appropriate surgical treatments. The authors believe in that a complete and universal book about trauma surgery should contain a chapter about this issue which includes all aspects of mid-face fractures. So we aim to provide a comprehensive chapter about diagnosis and treatment of mid-face fractures which may be a complete and useful guideline for trauma surgeons.

Keywords: Le Fort fractures, orbital wall fracture, NOE complex

1. Introduction

Mid-face fractures are common in different populations [1, 2]. Facial fractures are detected in almost 5–10% of trauma patients [3]. Motor vehicle accidents seem to be the first cause of mid-face fractures all around the word [4]. The other causes of facial fractures including mid-face trauma indicated in the literature are assaults, falls, sport injuries, and anima attacks [5, 6].

The importance of mid-face is clear in function and esthetics. The mid-face skeleton is important in providing a functional unit for respiratory, olfactory, vision, and digestive systems. The mid-face consists of vertical, horizontal, and sagittal pillars. Understanding the principles of mid-facial repair is the key to optimize the outcome.

Diagnosing mid-face fractures is sometimes very difficult in emergency cases. Diagnosis of the types of mid-face fractures is the first and basic step in management of mid-face trauma.

The treatment of mid-face fractures is complex due to the physiology and anatomy of mid-facial subunits. Quality of life of the patients is influenced following unsuccessful management of mid-face fractures which lead to permanent functional problems. Esthetic disfiguring trauma changes the whole mid-facial compartments.

This chapter aims to present a comprehensive review of mid-face fractures types' diagnosis and management.

2. Examination of trauma patients

Advanced trauma life support (ATLS) is the first step that should be applied in emergency cases. Airway obstruction should be evaluated as soon as possible since the mid-face is the beginning of the respiratory pathway. Hemorrhage and secretions may obstruct the oropharynx and nasopharynx. Removal of fractured teeth, clots, and loose dental crowns or dentures is important to open the oral airway. Packing should be used to control acute bleeding. Intubation to secure the airway in instable mid-face fractures is the next step that should be considered in emergency patients [7, 8]. It is important to keep the airway open in mid-face fractures because there is always the potential of airway obstruction due to displacement of bones or severe bleeding in such cases.

Cervical spine injuries are common in facial fractures. The incidence rate of cervical spine trauma in pediatric facial fracture cases is almost 3.5% [9] whilst this number is much higher in adult trauma patients [10]. According to the possibility of spinal injuries in facial trauma patients stabilizing the cervical spine by a rigid collar is necessary until the spinal injury is ruled out.

After providing a secure airway, ATLS protocol can continued. When the patient is stable, facial examination to detect the mid-face fractures is executed as follow.

3. Maxillofacial fractures

3.1. Le Fort fractures

3.1.1. Classification

Le Fort fractures are classified as three types. Le Fort I injury is defined as separation of maxilla from the mid-face (**Figure 1**A). Nasal septum, lateral nasal walls, lateral maxillary sinus wall, and pterygoid plates are involved in these kinds of fractures (**Figure 2**). Le Fort II fracture is also called the pyramidal fracture pattern which is identified by the separation of nasomaxillary complex (**Figure 1**B). Nasal and lacrimal bones, nasofrontal suture, infraorbital rims, and pterygoid plates are involved in this fracture pattern. Le Fort III also known as craniofacial dissociation is detected by the separation of the whole mid-face from the skull (**Figure 1**C). This fracture occurs in nasofrontal and zygomaticomaxillary sutures, zygomatic arch, and pterygoid plates.

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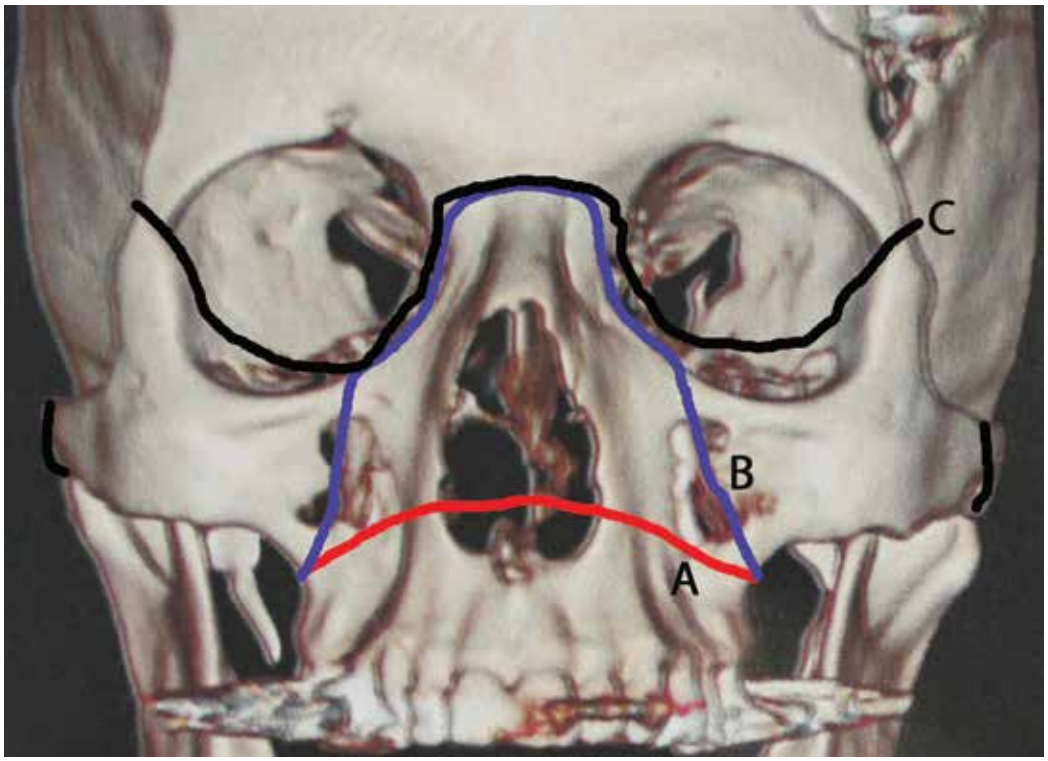

Figure 1. Le Fort I (A), II (B), III (C) fracture patterns on a three-dimensional model.

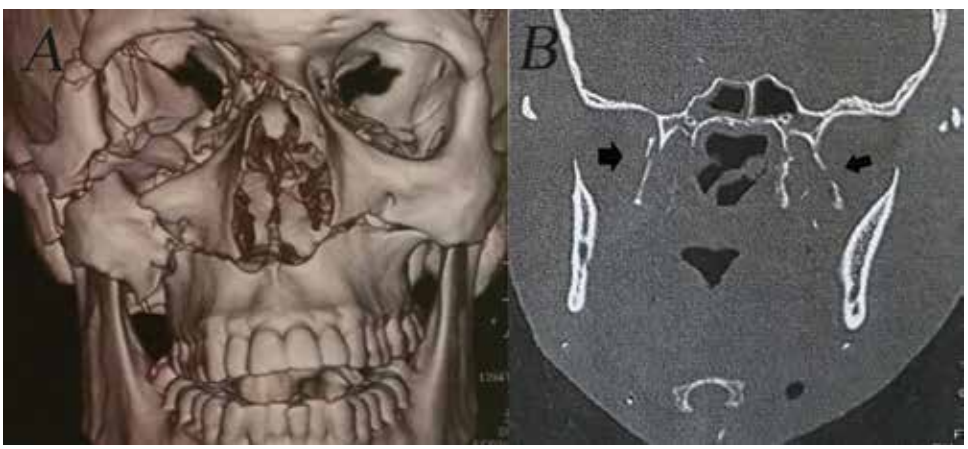

Figure 2. A, a three-dimensional view of Le Fort I fracture and B, pterygoid plate involvement in Le Fort I fracture (arrow head).

3.1.2. Signs and symptoms

As an initial examination mobility of maxilla is evaluated. The maxillary arch is grasped by thumb and pointing fingers of one hand and the mobility is checked by the other hand on pyriform

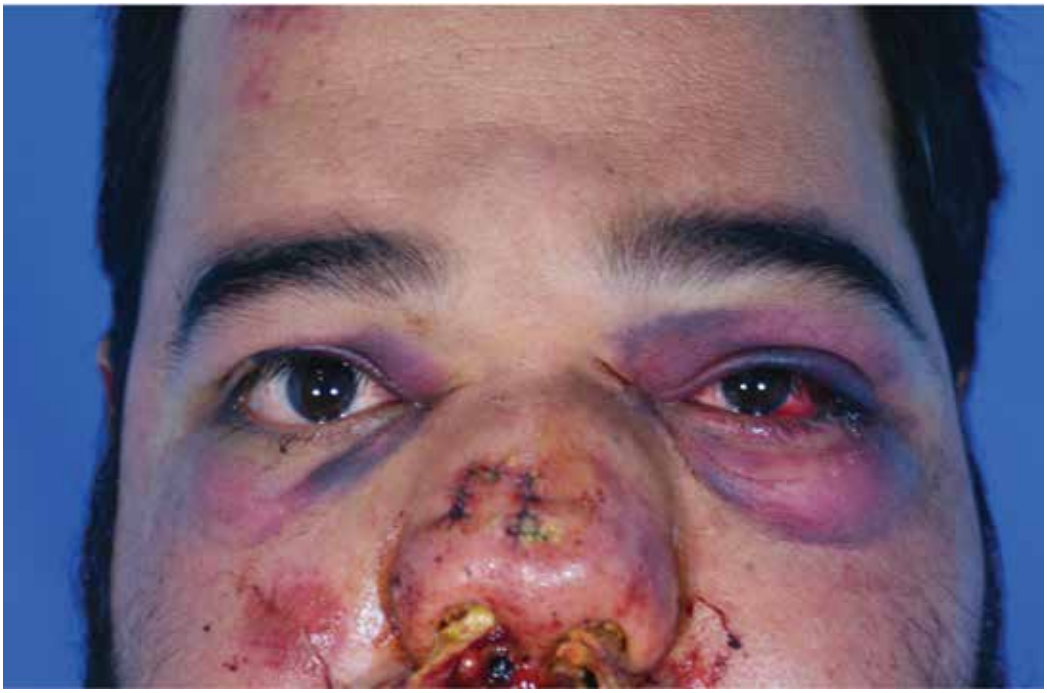

Figure 3. A classic raccoon eye is a sign of Le Fort II fracture.

aperture, nasofrontal suture, and zygomaticofrontal suture. In Le Fort fractures, lateral and medial pterygoid muscles pull the fracture segment posteriorly and inferiorly lead to an anterior open bite deformity. So malocclusion is an important sign in diagnosing the Le Fort fractures. Epistaxis is a common sign in all three patterns of Le Fort fractures. Hypoesthesia of the infraorbital nerve is seen in types I and II of Le Fort fractures. Bilateral periorbital ecchymosis which is called raccoon eyes is a classic sign of Le Fort II and III fractures (**Figure 3**). The clinician should be aware of the possibility of cerebrospinal fluid (CSF) leak in Le Fort II and III fractures.

3.1.3. Management

The decision to choose whether the open or closed technique in Le Fort fractures is dependent on the mobility of the maxilla and severity of maxillary displacement results in malocclusion. Minor maxillary displacement and malocclusion and low mobility of fractured segment are the indications of closed treatment. Closed technique could be performed by either maxillomandibular fixation (MMF) or skeletal suspension (**Figure 4**). The method of choice in the treatment of mobile maxilla with severe malocclusion is open reduction and internal fixation (ORIF). In the Le Fort I pattern lateral nasal walls and zygomatic buttresses are used to provide stability by four plates. Displaced Le Fort II fracture is treated by ORIF of bilateral infraorbital rims and zygomatic buttresses simultaneously using a miniplate to fix the nasofrontal suture. Mobile mid-face and esthetic problems following Le Fort III fracture (dish-face deformity) are the main indications of ORIF treatment. The number of fixations is dependent on

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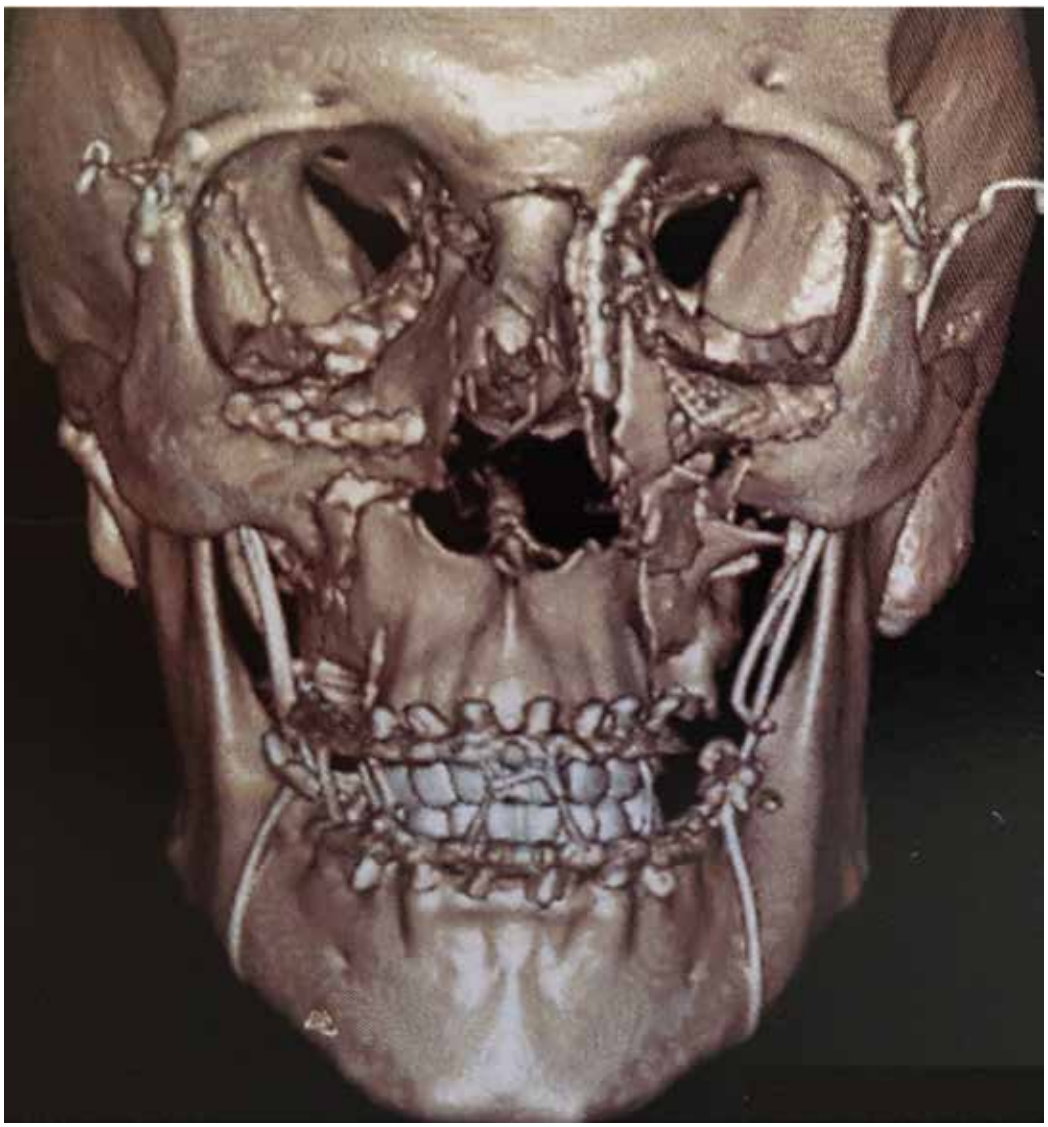

Figure 4. Suspension and closed treatment of comminuted Le Fort fractures.

the extent of comminution and dislocation. Bilateral zygomatic arches and zygomaticofrontal sutures and nasofrontal sutures should be fixed in severely displaced cases.

3.2. Palatal fractures

3.2.1. Classification

Hendrickson et al. [11] classified the palatal fracture into six patterns anatomically (**Figure 5**). Computed tomographies (CTs) in coronal and axial views are helpful in detecting the palatal fractures. Alveolar fracture is classified as type I palatal fracture in which it is categorized

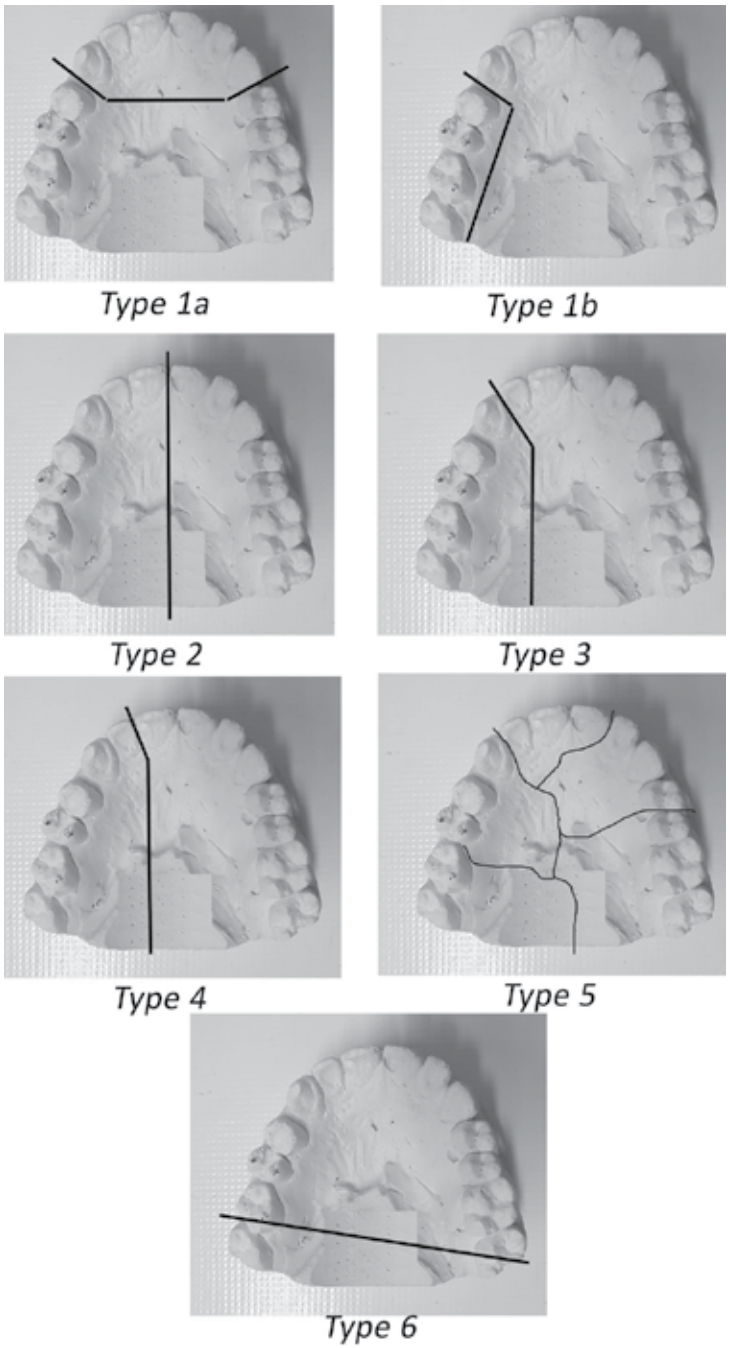

Figure 5. Classification of palatal fractures.

into two subcategories of anterior and posterolateral fractures. Anterior type I palatal fracture involves the incisor teeth and involving the posterior teeth it is defined as type 1b palatal fracture. Type II palatal fracture is defined as sagittal fracture which is less common in adults. Type III and IV fractures are the most common palatal fractures in adults [11]. Type III is also

called para-sagittal fracture which occurs in the thin part of the palate lateral to the attachment of vomer bone to the maxilla. The anterior limit of the fracture is between canine teeth which extend to the pyriform aperture. Type III fracture pattern extends posteriorly to the tuberosity or track approximate to the midline. Type IV fracture also known as para-alveolar fracture is a variant of the type III pattern. The fracture line in this pattern tracks medial to the alveolar bone of maxilla. The type V pattern is a complex fracture with comminution fragments. The transverse palatal fracture is classified as the type VI pattern which is the least common palatal fracture type.

3.2.2. Signs and symptoms

Mobility of alveolar segments should be checked for the entire maxillary arch. Displacement of fractured segments results in malocclusion which is an important sign to the clinician in diagnosing the palatal fracture. Ecchymosis of the palate may also indicate the line of fracture.

3.2.3. Management

When the occlusion is good enough and the fractured segment is either minimally displaced or not displaced at all the surgeon may decide to follow the patient and choose no intervention. MMF is the treatment of choice in minimally displaced palatal fractures unless there is a contraindication for MMF. Gunning and palatal splints are other amenable methods for closed treatment of palatal fracture (**Figure 6**). ORIF of palatal fracture is indicated in severely mobile and displaced patterns to prevent splaying of the fragments.

3.3. Orbital fractures

3.3.1. Classification

According to the involved orbital walls there are five fracture patterns. The most common fracture of the orbit is the orbital floor fracture mostly detected as a blow-out fracture [6] (**Figure 7**).

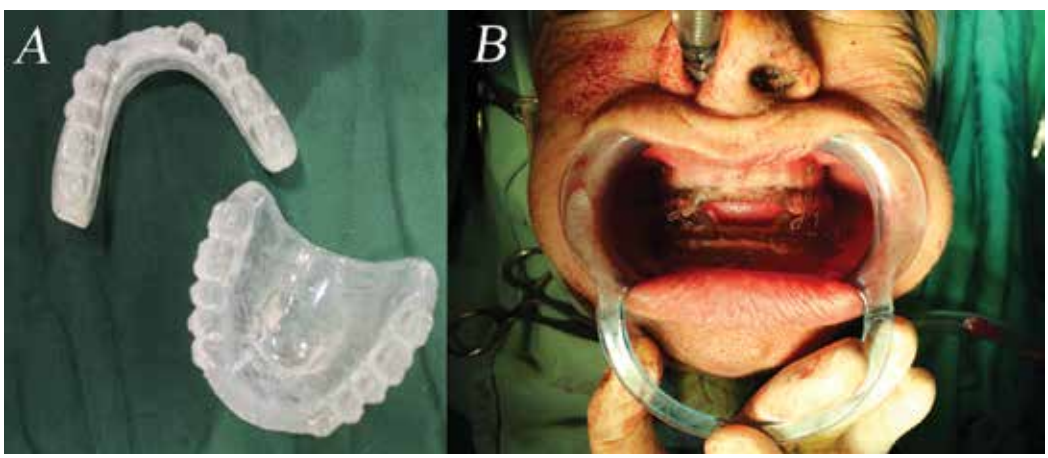

Figure 6. (A) Gunning for closed treatment of simultaneous mandibular and palatal fractures in an edentulous patient and (B) Maxillomandibular fixation for closed treatment of the patient.

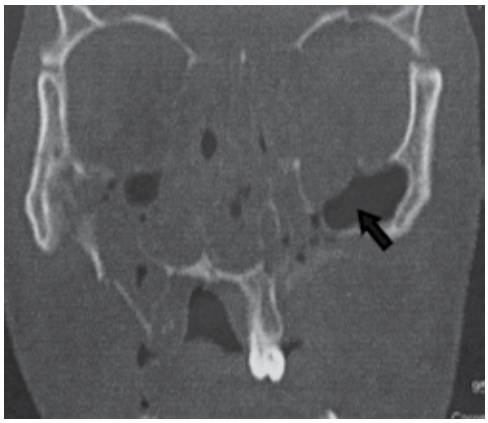

Figure 7. Coronal CT view indicating orbital floor (blow out) fracture.

Orbital roof fracture is the most common fracture in pediatric population [12]. Other less common orbital fractures involve medial or lateral wall. Combined orbital fracture especially involving all four orbital walls are the least common orbital fracture [6] patterns whilst the leading functional and esthetic problems of this pattern are much more serious than former fracture types.

3.3.2. Signs and symptoms

Entrapment of extraocular muscles should be assessed when there is suspected orbital wall fracture (**Figure 8**). Forced duction test is helpful in distancing between muscle entrapment and neurologic disturbance although this test is sometimes falsely negative due to post-injury edema. Diplopia is a common sign of orbital fracture, especially medial fracture pattern due to rectus muscle entrapment [13]. Infraorbital nerve hypoesthesia is a symptom of orbital fracture especially when the infraorbital rim is involved [14]. Subconjunctival hemorrhage and periorbital ecchymosis are useful signs of an underlying orbital fracture [15] (**Figure 9**). Enophthalmus is an important sign of orbital fracture and also a significant indication of orbital reconstruction [16]. Enophthalmus usually occurs as a result of increased orbital volume or loss of orbital content especially orbital fat.

3.3.3. Management

Orbital fracture cases are non- or minimally displaced should just observe. No intervention is needed when Orbital fractures do not result in any ocular problems including diplopia or enophthalmus. Orbital fracture treatment is a controversial issue among maxillofacial and oculoplastic surgeons. Fracture size, timing of the reconstruction, and biomaterials for reconstructions are all important issues which should be considered in orbital fracture repair. The debate still is present in deciding on whether to treat an orbital fracture or not. The investigations are insufficient with high heterogeneity in this field. As a general rule it is almost acceptable that defects more than 50% of the orbital wall or 2 cm length should be treated [17]. Enophthalmus and positive-forced duction tests are two indications for management of orbital wall fractures.

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Figure 8. The patient is not able to look upward concurrently by both eyes due to left orbital floor fracture lead to inferior rectus muscle entrapment.

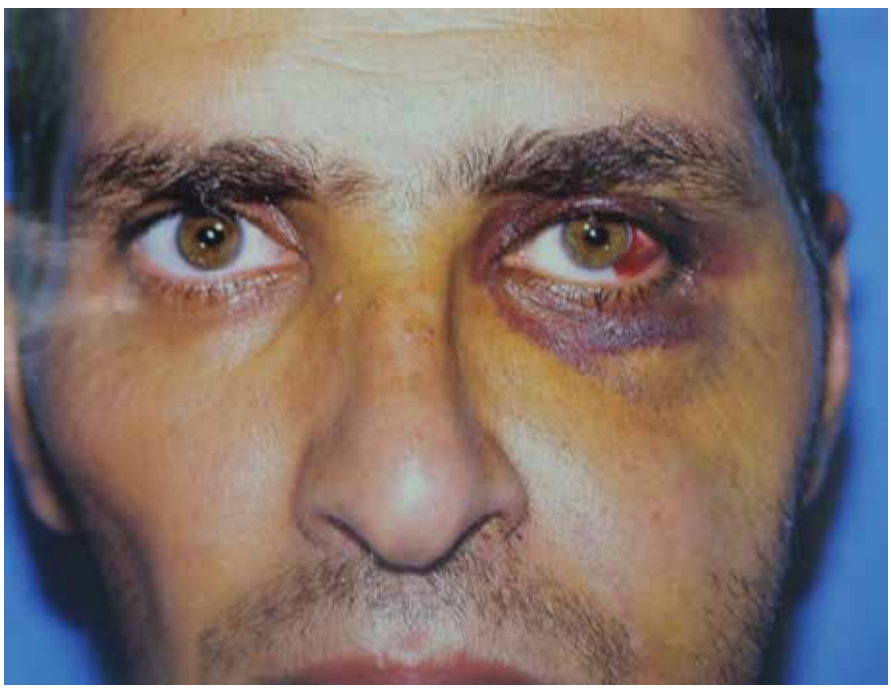

Figure 9. Periorbital ecchymosis and subconjuctival hemorrhage following orbital fracture.

Timing of orbital reconstruction is categorized into three groups of immediate categories: within 24 h, early (between first and day 14), and delayed (after 2 weeks) [18]. When the reason of diplopia is muscle entrapment immediate reconstruction of the orbit is advocated by the investigators. Blow-out fracture in young patients is the other indication for immediate repair. Early orbital reconstruction is advocated by some surgeons in cases of early enophthalmus and symptomatic diplopia with positive forced duction test. Early reconstruction should also be considered in cases with large orbital wall defects (more than 50% defects). Symptomatic diplopia with negative force duction test and late-onset enophthalmus are indications for delayed orbital reconstructions [18].

Decision-making on the ideal material for orbital reconstruction is based on the surgeon's experience, cost, defect size, and medical history (**Figure 10**) [19]. The available material and their pros and cons are categorized in **Table 1**.

3.4. Naso-orbital-ethmoid (NOE) fractures

3.4.1. Classification

According to Markowitz's classification naso-orbital-ethmoid (NOE) fracture is defined as three patterns [20] (**Figure 11**). Type I NOE fracture is defined as single-segment central fragment. This pattern could be in a uni- or bilateral form. The medial tendon is attached to the fractures segment in this pattern. Type II NOE fracture consists of comminuted central fragments external to the medial canthal tendon insertion. In type III fracture the fracture line

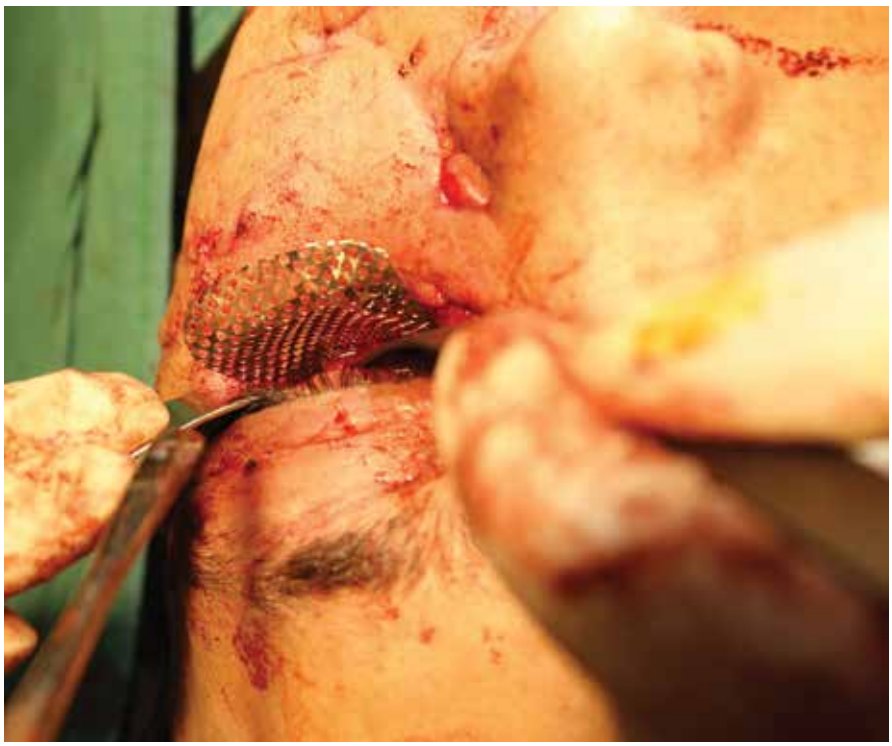

Figure 10. Titanium meshwork plate is used to reconstruct the orbital floor defect.

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Materials	Examples	Advantages	Disadvantages	Indications
Autogenous bone grafts	Iliac bone graft, caldaria grafts	Biocompatibility, cost effective, variability in thickness, radio opacity	Donor site morbidity, difficult to shape, high resorption rate	Large defects, immature orbits, secondary defect reconstruction
Resorbable materials	poly-l-lactic acid (PLLA),	Replacement with bone formation	High cost, radiolucency, low stability	Small defects
Non-resorbable materials	Titanium mesh, Porous polyethylene sheets	High stability, easy fixation, availability, no donor site defect	High cost, increased infection rate	Medium size defects with medium complexity

Table 1. Materials available for orbital reconstruction.

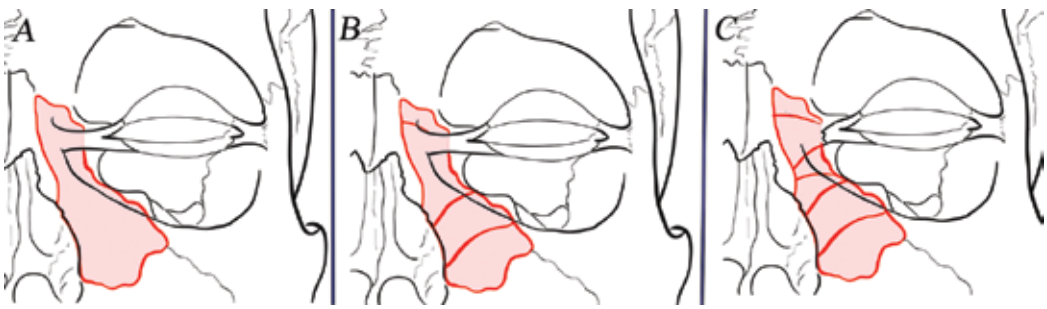

Figure 11. Naso-orbital-ethmoid fracture types. A, type I Naso-orbital-ethmoid fracture. B, type II naso-orbital-ethmoid fracture. C, type III naso-orbital-ethmoid fracture.

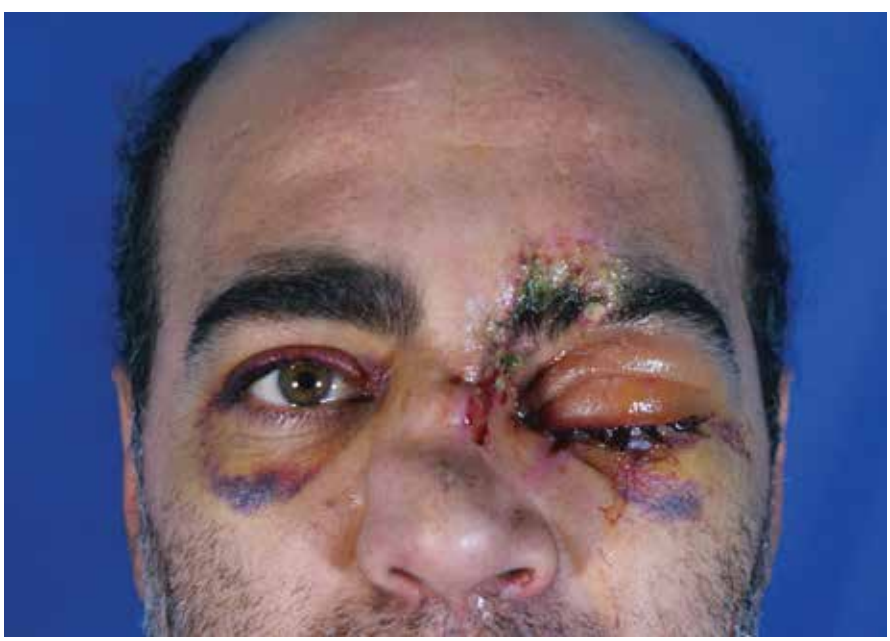

Figure 12. Signs in a patient with naso-orbital-ethmoid fracture. Rounding of the left medial canthus (arrow) and traumatic telecanthus is obvious in this patient.

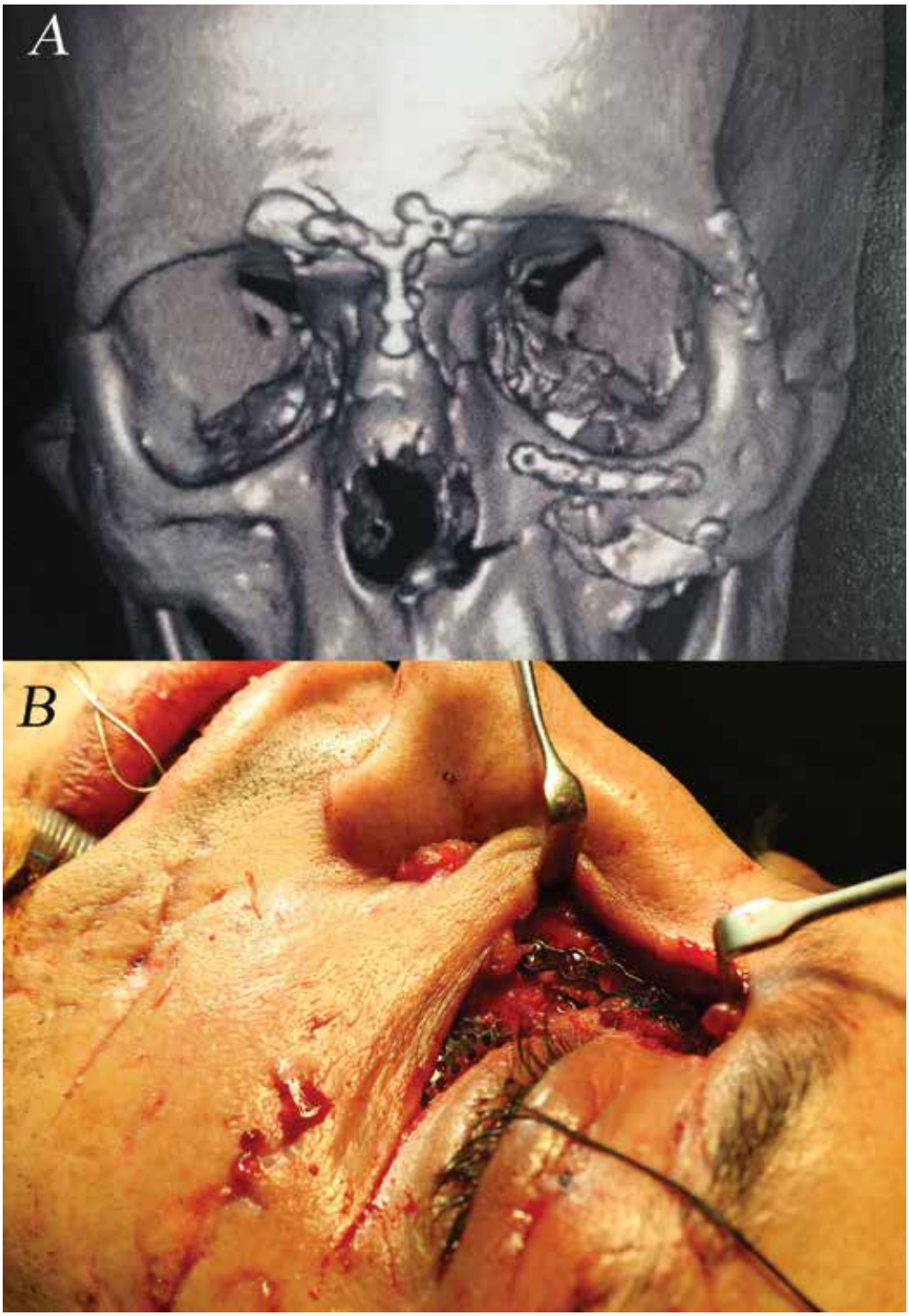

Figure 13. A, fixation of the type I naso-orbital-ethmoid fracture. B, reconstruction of left type II naso-orbital-ethmoid fracture.

extends into the medial canthal insertion segment. The medial canthal tendon either remains attached to the central segment or does not.

3.4.2. Signs and symptoms

Epistaxis is a common sign of NOE fracture. Involving the NOE complex in trauma patients results in splayed nasal complex and widened the nasal bridge. In the case of medial canthal tendon detachment or disruption of traumatic telecanthus and medial canthus rounding occurs (**Figure 12**). The intercanthal distance is usually half of the interpupillary distance (average of 28–35 mm in white adults). So when this measure is more than 40 mm or half of the interpupillary distance, the traumatic telecanthus is defined [21]. Bimanual test is a useful method in detecting the instability of NOE fracture [22].

3.4.3. Management

Stabilization of the fractures segment is the only intervention advocated in NOE type I fracture (**Figure 13**A). Stabilizing the central fragment in which the medial canthal tendon is inserted is the treatment of choice in type II fracture (**Figure 13**B). Transnasal wiring simultaneously with orbital medial wall reconstruction is considered in type III pattern.

4. Surgical approaches in treatment of mid-face fractures

4.1. Intraoral approaches

Intraoral approach and vestibular incision is the most common technique used in treatment of Le Fort fractures (**Figure 14**). Circum-vestibular incision mesial to the second premolar is used to reach the nasal lateral walls and zygomatic buttresses. As mentioned earlier these buttresses are stable enough to maintain the maxilla at the right position following rigid fixation. Cinch suture and V-Y plasty is necessary when the incision involves the nasalis muscles.

4.2. Extraoral approaches

After decision-making of rigid fixation of the Le Fort III fracture, extraoral approaches to the zygomaticofrontal and nasofrontal sutures are applied (**Figure 15**). Bicoronal flap is the common approach to achieve all three sutures by one sing incision. Also this is a good approach in repairing the NOE fracture. The incision is made several centimeters behind the hair line between the upper origins of the temporal muscles from one superior temporal line to the other. Dissection of the flap is performed in the subgaleal plane up to 2 cm above the superior orbital rims. The periosteum is incised at this level and subperiosteal dissection is continued to expose the zygomaticofrontal and nasofrontal sutures. Using a suction drain is optional during closure.

When there is no displacement of nasofrontal suture, fixation of zygomaticofrontal sutures is applicable by lateral brow approach. The incision is made almost 2 cm parallel to the hair follicles

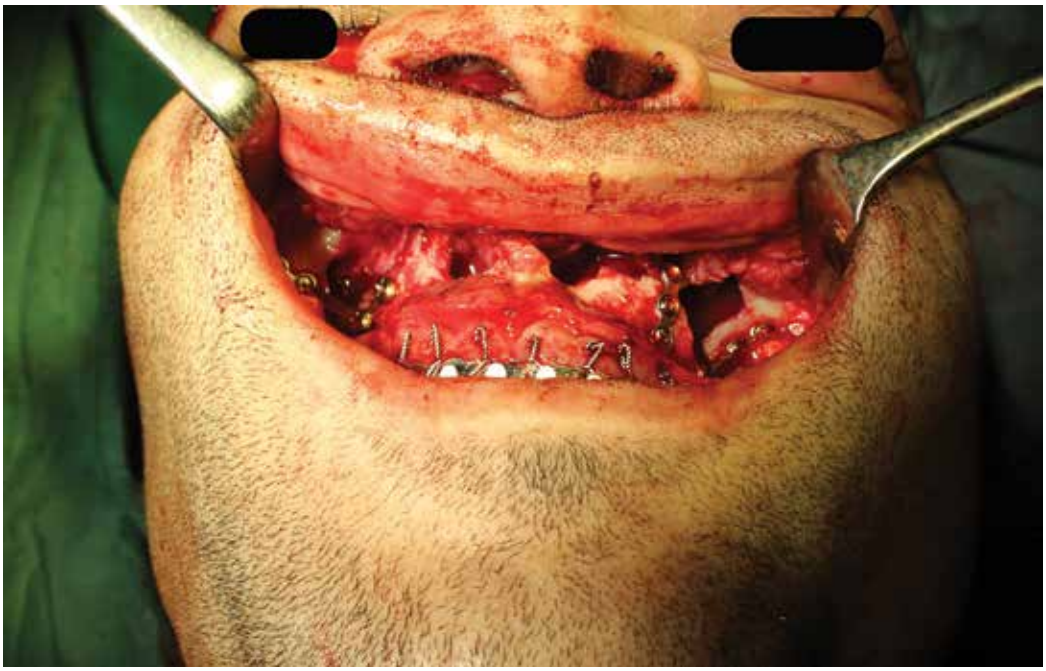

Figure 14. Intraoral approach to expose the Le Fort I fracture line.

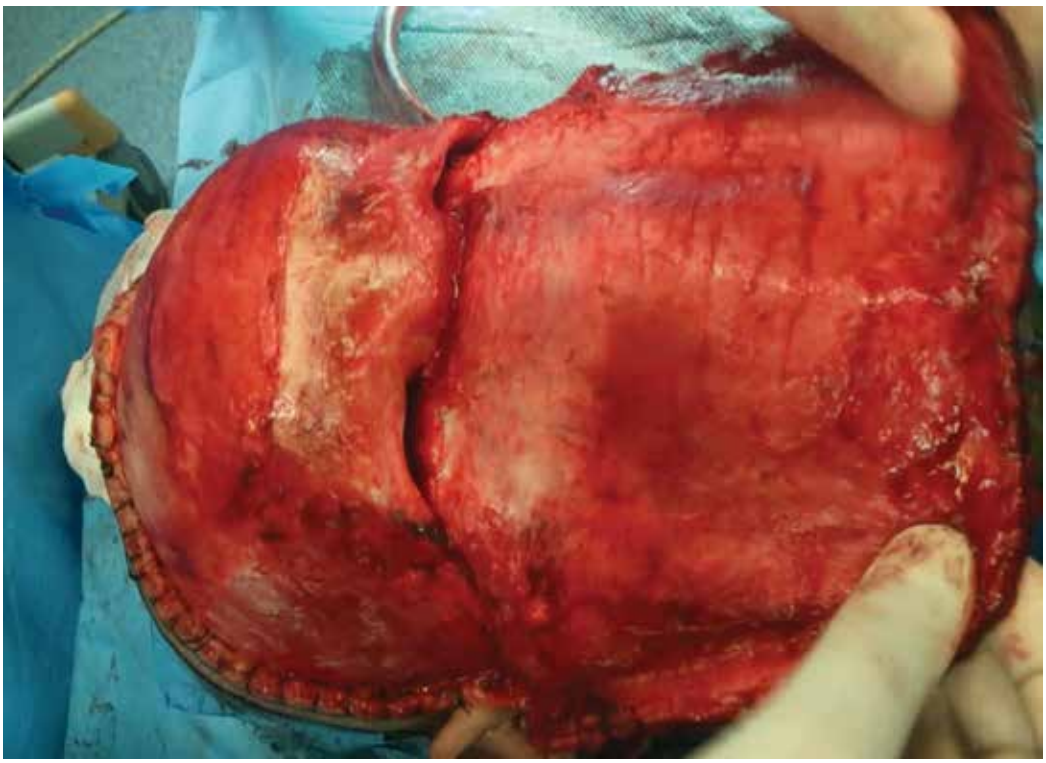

Figure 15. Coronal approach for management of Le Fort III fracture (courtesy of Dr. Fereydoun Pourdanesh).

of the lateral eyebrow (**Figure 16**). The advantages of this technique are least noticeable scar and no adjacent anatomical structure.

Glabellar and ethmoidal (known as Lynch approach) approaches are used in solitary NOE fracture. The latter technique is not recommended by AOCMF due to visible scar band (web) [23]. Glabellar incision is made in old patients in the glabellar furrows through the skin, subcutaneous layer, and the periosteum.

4.3. Periorbital approaches

Four kinds of periorbital approaches are represented in the literature for reconstruction of orbital fractures and Le Fort II fracture. The incisions on the lower lid are classified into three types based on the distance from the gray line (**Figure 17**). The periorbital approach is called subciliary incision when this distance is about 2–3 mm. When this distance is almost 3–4 mm to the gray line the incision is known as mid-lower lid or subtarsal approach. The dissection of these two techniques is in three fashions. The best dissection technique is to start a few millimeters subcutaneously followed by orbicularis oculi muscle dissection. Skin only or pre-orbicularis oculi muscle incision is not advocated by authors due to high possibility of ectropion rate. The third incision is called skin-muscle flap which involves both skin and orbicularis oculi muscle.

Another popular periorbital approach because of its invisible scar is the transconjunctival technique (**Figure 18**). The incision is made parallel to the gray line through the conjunctive. This approach is divided into preseptal and retroseptal techniques based on the dissection plane. Lateral canthotomy and inferior cantholysis are used in some cases when the surgeon needs more access to the orbit.

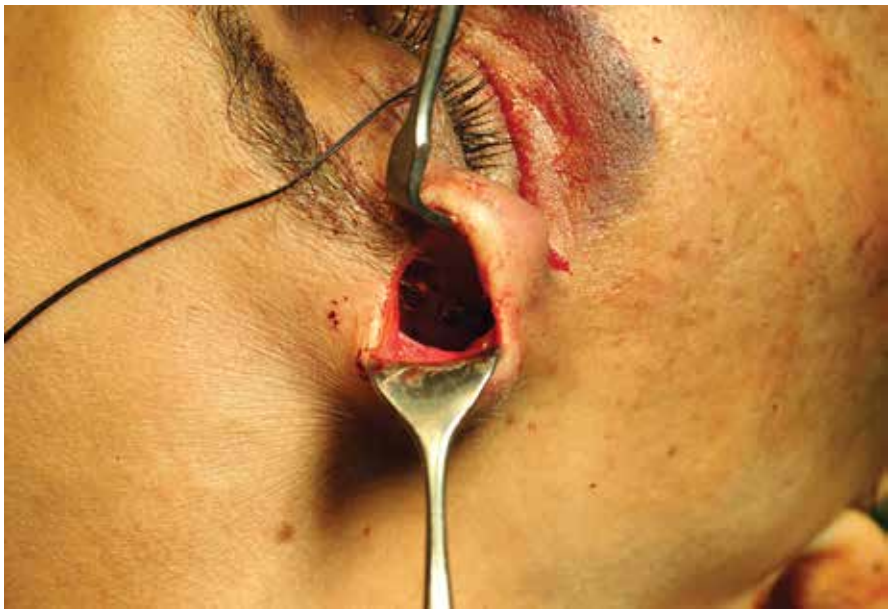

Figure 16. Lateral brow approach.

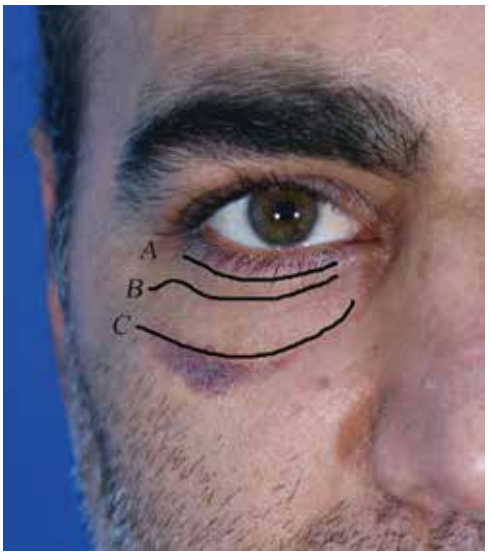

Figure 17. Periorbital incisions. Subciliary (A), subtarsal (B), and infraorbital (C) approaches are shown in this picture.

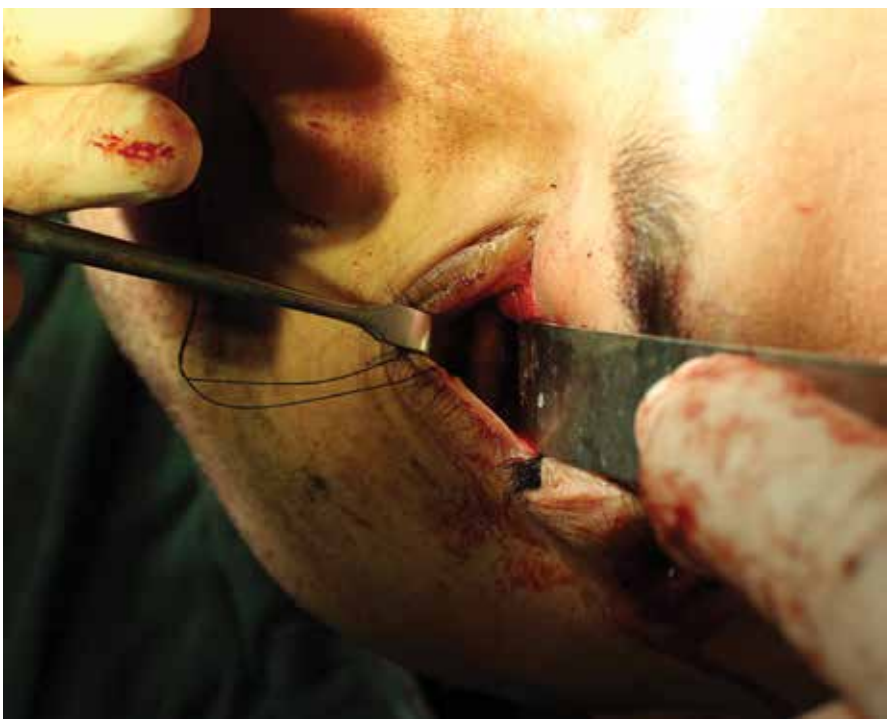

Figure 18. Transconjunctival approach is used to expose the orbital floor fracture.

5. Conclusions

The mid-face is esthetically and functionally very important which makes repairing the deformities of this facial part very difficult. Diagnosing the exact injuries on the facial bones is the

key step of deciding the treatment plan. The surgeon should have enough knowledge of facial anatomy and physiology to be able to reconstruct the fractured segments. Deformity following facial trauma is hard to repair on the second surgery. So the importance of managing almost all problems of mid-face fractures in the first surgery is pretty clear to all traumatologists.

Conflict of interest

The authors declare that they have no conflict of interest. The photos not referenced in the text belong to the author.

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Thoracoabdominal Injuries

Chapter 5

Abdominal Trauma

Göksu Afacan

Additional information is available at the end of the chapter

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Abstract

Abdominal injuries may be life threatening and should be approached cautiously. After trauma, the abdomen may be sanctuary for occult bleeding that, if not discovered and corrected expeditiously, may lead to deleterious consequences. Patients with abdominal trauma should have rapid assessment, stabilization, and early surgical consultation to maximize the chances of a successful outcome. Deaths from abdominal trauma result principally from hemorrhage or sepsis. Most deaths from abdominal trauma are preventable. Patients at risk of abdominal injury should undergo prompt and thorough evaluation. In some cases, dramatic physical findings may be due to abdominal wall injury in the absence of intraperitoneal injury. If the results of diagnostic studies are equivocal, diagnostic laparoscopy or exploratory laparotomy should be considered, since they may be lifesaving if serious injuries are identified early.

Keywords: abdomen, trauma, surgery

Abdominal injuries may be life threatening and should be approached cautiously. After trauma, the abdomen may be sanctuary for occult bleeding that, if not discovered and corrected expeditiously, may lead to harmful consequences. Patients with abdominal trauma should have rapid assessment, stabilization, and early surgical consultation to maximize the chances of a successful outcome.

1. General evaluation

Initial management of patients with abdominal trauma is the same as for all other trauma patients. Begin with a rapid primary survey, including evaluation of the airway, breathing, circulation, disability, and exposure.

If the abdomen is the probable source of exsanguinating hemorrhage, the patient should be transferred to the operating room for immediate laparotomy. The hemodynamically stable patient can be more meticulously assessed within the framework of the secondary survey. Evaluation always includes comprehensive physical examination with pelvic and rectal examinations and may require specific laboratory and radiologic tests.

1.1. Airway

Administer high flow oxygen and intubate the patient if necessary. Maintain cervical spine immobilization until potential injury is ruled out.

1.2. Breathing

Auscultate for breathe sounds. Inspect for asymmetry of chest wall movement, open wounds, or flail segments. Palpate the chest wall carefully as palpable crepitus may indicate a pneumothorax or rib fractures. Pulse oximetry and capnography may be useful.

1.3. Circulation

Stop gross external hemorrhage with direct pressure. Assess pulses, capillary refill, and blood pressure. Obtain intravenous access with at least two large bore (≥16 gauge) catheters. If peripheral intravenous access is inadequate, place a central venous catheter.

1.4. Disability

Complete a brief and focused neurologic examination to document the patient's baseline. The examination should include an assessment of pupillary size and reactivity, a determination of the patient's Glasgow coma scale (GCS) score, and notation of any focal neurologic deficits such as unilateral weakness or poor muscle tone. Ideally, perform the examination before administering pain medications, sedatives, or paralytics.

1.5. Exposure

Completely undress the patient, although be careful to prevent or recognize and correct associated hypothermia. Begin a more thorough secondary survey, including examining all skin folds, the back, and axillae for occult penetrating injuries.

Do not remove impaled foreign bodies because they may be providing hemostasis from a vascular injury. Foreign body removal should be performed with surgical consultation in a more controlled setting.

Any penetrating injury below the nipple line warrants evaluation for intra-abdominal injury. In patients in motor vehicle collisions, look for ecchymosis or erythema in the area of clavicles

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or across the abdomen. The classic "seat belt sign" or linear bruising across the lower abdomen is a marker for intra-abdominal injury.

2. General examination

Examine the abdomen for tenderness, distention, rigidity, or guarding.

Evaluate the pelvis for anteroposterior or lateral instability with gentle pressure; this does not require much force and should not be repeatedly performed. Examine the genitalia and look for blood at the urethral meatus, especially in males. Perform digital rectal examination in any patient with abdominal trauma. Look for gross blood, assess sphincter tone, and note any other evidence of trauma. If blood at the urethral meatus or a high riding prostate is present, placement of a urinary catheter is contraindicated, and a retrograde urethrogram is required to evaluate for potential urethral injury.

2.1. Laboratory evaluation

Initial laboratory evaluation should include hemoglobin and hematocrit and platelet count to establish a baseline, and a blood type and screen in case transfusion of packed red cells are needed. A lactate level may be obtained and, if elevated, is an excellent indicator of shock. Base deficit is another indicator of shock. The role of amylase in abdominal trauma is uncertain. Examination of the urine may reveal gross hematuria, which suggests significant injury to the urogenital tract.

2.2. Plain radiography

Almost all major trauma patients require plain X-rays of the chest, pelvis, and cervical spine. Although rarely used today because of the ubiquity of computed tomography (CT) scanning, a one-shot intravenous pyelogram may be useful in patients with flank wounds or gross hematuria who are unable to undergo further diagnostic testing prior to operative intervention.

2.3. Ultrasonography

Ultrasonography has emerged as the primary initial diagnostic examination of the abdomen in multisystem injured blunt trauma patients. Emergency ultrasonography has been studied extensively and is rapid and accurate in the identification of intraperitoneal free fluid. Also, it is safe in special patient populations (e.g., pediatrics, obstetrics). Focused assessment with sonography for trauma (FAST) examination is a bedside test that has demonstrated good accuracy with relatively minimal operator experience. In the standard FAST examination, four areas are scanned: the right upper quadrant, the subxiphoid area, the left upper quadrant, and the pelvis. Unstable patients with a positive FAST examination should undergo urgent exploratory laparotomy [1]. Unlike CT, a FAST examination is rapid, can be performed bedside in the emergency department, and is easily repeatable [2] (**Figure 1**).

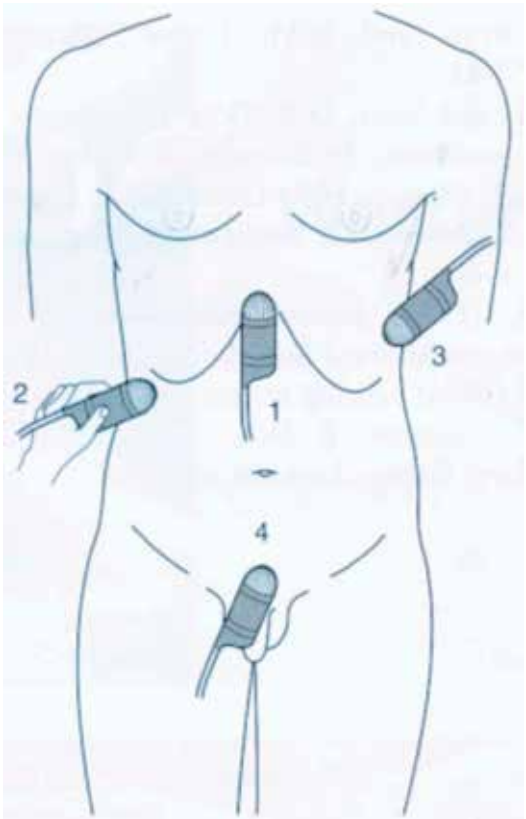

Figure 1. Transducer positions for FAST: pericardial area, right and left upper quadrants, and pelvis.

2.4. CT scanning

CT is noninvasive, qualitative, sensitive, and accurate for the diagnosis of intra-abdominal injury. Modern spiral scanners have greatly decreased the time required for obtaining high quality images. However, CT scanning remains expensive. CT scanning requires transport from the acute care area and should not be attempted in the unstable patient.

CT scanning has a primary role in defining the location and magnitude of intra-abdominal injuries related to blunt trauma. It has the advantage of detecting most retroperitoneal injuries, but it may not identify some gastrointestinal injuries. The formation provided on the magnitude of injury allows for nonoperative management of patients with solid organ injuries.

In the hemodynamically stable patient, CT scanning is an excellent diagnostic modality that is easy to perform. No diagnostic modality outperforms CT in the evaluation of intraperitoneal as well as retroperitoneal injuries (**Figure 2**).

2.5. Diagnostic peritoneal lavage

Diagnostic peritoneal lavage (DPL) is designated to detect the presence of intraperitoneal blood, although its use has decreased significantly at many centers with the use of the FAST

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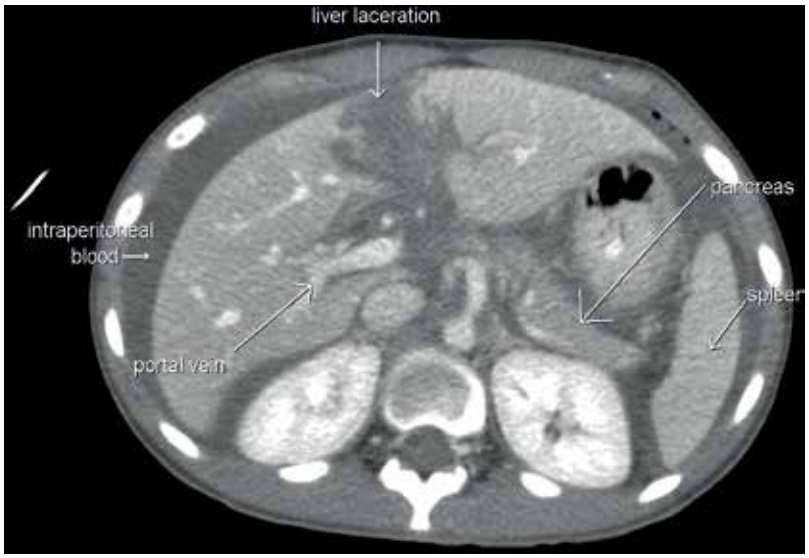

Figure 2. A CT image in blunt abdominal trauma (liver laceration and intraperitoneal blood was shown with the landmarks such as the pancreas, spleen, and portal vein).

examination. Determinations of leucocytes, particulate matter, or amylase in the lavage fluid may indicate the presence of a bowel injury. Drainage of lavage fluid from a chest tube or urinary catheter may indicate a lacerated diaphragm or bladder. Lavage can be performed easily and rapidly, with minimal cost and morbidity. It is an invasive procedure that will affect the findings on physical examination, and it should be performed by a surgeon [3].

The procedure is neither qualitative nor quantitative. It cannot identify the source of hemorrhage, and relatively small amounts of intraperitoneal bleeding may result in a positive study.

Although DPL has largely been replaced by ultrasonography, it is still used occasionally. The main concern regarding DPL is that it is overly sensitive for intra-abdominal blood, which has led to a high rate of negative or nontherapeutic laparotomies [4].

If DPL is considered, it should be performed only after consultation with the trauma surgeon, who should perform this diagnostic study in most cases (**Table 1**).

2.6. Laparoscopy

Laparoscopy has an important role in stable patients with penetrating abdominal trauma. It can quickly establish whether peritoneal penetration has occurred and thus reduce the number of negative and nontherapeutic trauma laparotomies performed [5]. Laparoscopy has also been applied safely and effectively as a screening tool in stable patients with blunt abdominal trauma [6].

The use of laparoscopy, with or without CT scanning or DPL, is being studied. It is less invasive than traditional laparotomy and may shorten hospital stays and decrease patient costs, although it requires surgical consultation [7].

Positive
20 mL gross blood on free aspiration (10 mL in children)
≥100,000 red cells/μL
≥500 white cells/μL (if obtained 3 h or more after injury)
≥175 units amylase/dL
Bacteria on Gram-stained smear
Bile (by inspection or chemical determination of bilirubin content)
Food particles
Intermediate
Pink fluid on free aspiration
50,000–100,000 red cells/μL in blunt trauma
100–500 white cells/μL
75–175 units amylase/dL
Negative
Clear aspirate
≤100 white cells/μL
≤75 units amylase/dL

Table 1. Criteria for evaluation of peritoneal lavage fluid.

2.7. Emergency (exploratory) laparotomy

Most patients with penetrating abdominal injuries will also require laparotomy given the high incidence of intra-abdominal injury once the fascia has been violated. Hemodynamically unstable patients sustaining blunt or penetrating trauma with a positive screening test [such as focused assessment with sonography for trauma (FAST) examination or diagnostic peritoneal lavage (DPL)] require laparotomy to control hemorrhage and evaluate for intra-abdominal injuries. Also patients with obvious diaphragmatic injury noted on chest X-ray require emergency laparotomy [8].

The tree main indications for exploration of the abdomen following blunt trauma are peritonitis, unexplained hypovolemia, and the presence of other injuries known to be frequently associated with intra-abdominal injuries. Peritonitis after blunt abdominal trauma is rare but always requires exploration. Signs of peritonitis can arise from rupture of a hollow organ, such as the duodenum, bladder, intestine, or gallbladder from pancreatic injury, or occasionally from the presence of retroperitoneal blood.

Emergency abdominal exploration should be considered for patients with profound hypovolemic shock and a normal chest X-ray unless extra-abdominal blood loss is sufficient to account for the hypovolemia. In most cases a rapidly performed FAST examination or peritoneal lavage will confirm the diagnosis of intraperitoneal hemorrhage. Patients with blunt trauma and hypovolemia should be examined first for intra-abdominal bleeding even if there is no overt evidence of abdominal trauma. Hemoperitoneum may present with no

Method	Time/cost	Advantage/disadvantage
Physical examination	Quick/no cost	Useful for serial examinations, very limited by other injuries, coma, drug intoxication, poor sensitivity and specificity
Diagnostic peritoneal lavage (DPL)	Quick/inexpensive	Rapid results in unstable patient but invasive and may be overly sensitive for blood and not specific for site of injury, requires experience and may be limited if previous surgery
Focused assessment with sonography for trauma (FAST)	Quick/inexpensive	Rapid detection of intra-abdominal fluid and pericardial tamponade, may be limited by operator experience, large body habitus, subcutaneous air, poor for detection of bowel injury. Fairly sensitive but not highly specific
Helical computerized abdominal tomography (CT)	Slower/expensive	Most specific for site of injury and can evaluate retroperitoneum, very good sensitivity but may miss bowel injury, risk of reaction to contrast dye

Table 2. Comparison of diagnostic methods for abdominal trauma.

signs except hypovolemia. The abdomen may be flat and nontender. Patients whose extra-abdominal bleeding has been controlled should respond to initial fluid resuscitation with an adequate urine output and stabilization of vital signs. If hypovolemia recurs, intra-abdominal bleeding must be considered to be the cause.

Injuries frequently associated with abdominal injuries are rib fractures, pelvic fractures, abdominal wall injuries, and fractures of the thoracolumbar spine (**Table 2**).

3. Surgical consultation

Seek surgical consultation early in the management of patients with abdominal trauma, especially if the patient is hemodynamically unstable [9].

4. Fluid resuscitation

Rapid infusion of large amounts of crystalloids may disrupt the formation of the soft clot and dilute the clotting factors, leading to increased bleeding. Attempts to make the patient normotensive are not recommended. A more reasonable goal may be to obtain systolic blood pressure of 80–90 mmHg, or a mean arterial pressure of 70 mmHg. Crystalloids remain first-line fluids, followed by infusions of packed red blood cells [10].

5. Types of injuries

The distribution of blunt and penetrating injury in a given population is highly dependent upon geographic location. Blunt injuries predominate in rural areas, while penetrating injuries

are more common in urban areas. The specific type of injury varies according to whether the trauma is penetrating or blunt. The mechanism of injury in blunt trauma is rapid deceleration, and noncompliant organs such as the liver, spleen, pancreas, and kidneys are at greater risk of injury due to parenchymal fracture.

Deaths from abdominal trauma result principally from hemorrhage or sepsis. Most deaths from abdominal trauma are preventable. Patients at risk of abdominal injury should undergo prompt and thorough evaluation. In some cases, dramatic physical findings may be due to abdominal wall injury in the absence of intraperitoneal injury. If the results of diagnostic studies are equivocal, diagnostic laparoscopy or exploratory laparotomy should be considered, since they may be lifesaving if serious injuries are identified early.

5.1. Penetrating trauma

Penetrating injuries may cause sepsis if they perforate a hollow viscous. Increasing abdominal tenderness demands surgical exploration. White blood cell count elevations and fever appearing several hours following injury are keys to early diagnosis.

Penetrating injuries can cause severe and early shock if they involve a major vessel or the liver. Penetrating injuries of the spleen, pancreas, or kidneys usually do not bleed massively unless a major vessel to the organ (e.g., renal artery) is damaged. Bleeding must be controlled promptly. A patient in shock with a penetrating injury of the abdomen who does not respond to 2 L of fluid resuscitation should be operated on immediately following chest X-ray [11].

The treatment of hemodynamically stable patients with penetrating injuries to the lower chest or abdomen varies. All surgeons agree that patients with signs of peritonitis or hypovolemia should undergo surgical exploration, but treatment is less certain for patients with no signs of peritonitis or sepsis who are hemodynamically stable [12].

Most stab wounds of the lower chest or abdomen should be explored, since a delay in treatment of hollow viscous perforation can result in severe sepsis. Some surgeons have recommended a selective policy in the management of these patients. When the depth of injury is in doubt, local wound exploration may rule out peritoneal penetration. Laparoscopy may ultimately have a role in the evaluation of penetrating injuries. All gunshot wounds of the lower chest and abdomen should be explored because the incidence of injury to major intra-abdominal structures is 90% in such cases [13].

5.2. Blunt trauma

Blunt abdominal trauma (BAT) comprises 75% of all blunt trauma and is the most common example of this injury. The majority occurs in motor vehicle accidents, in which rapid deceleration may propel the driver into the steering wheel, dashboard, or seatbelt causing contusions in less serious cases, or rupture of internal organs from briefly increased intraluminal pressure in the more serious, dependent on the force applied. It is important to note that initially there may be little in the way of overt clinical signs to indicate that serious internal abdominal injury has occurred, making assessment more challenging and requiring a high degree of clinical suspicion [14].

There are two basic physical mechanisms at play with the potential of injury to intra-abdominal organs: compression and deceleration. The former occurs from a direct blow, such as a punch, or compression against a non-yielding object such as a seatbelt or steering column. This force may deform a hollow organ, thereby increasing its intraluminal or internal pressure, leading to rupture [15]. Deceleration, on the other hand, causes stretching and shearing at the points at which mobile structures, such as the bowel, are anchored. This can cause tearing of the mesentery of the bowel, and injury to the blood vessels that travel within the mesentery. Classic examples of these mechanisms are a hepatic tear along the ligamentum teres and injuries to the renal arteries [16].

When blunt abdominal trauma is complicated by "internal injury," the liver and spleen are most frequently involved, followed by the small intestine [17].

In rare cases, this injury has been attributed to medical techniques such as the Heimlich maneuver, attempts at cardiopulmonary resuscitation and manual thrusts to clear an airway. Although these are rare examples, it has been suggested that they are caused by applying unnecessary pressure when administering such techniques. Finally, the occurrence of splenic rupture with mild blunt abdominal trauma in those convalescing from infectious mononucleosis is well reported.

A major addition in management of blunt trauma has been the focused assessment with sonography for trauma (FAST) examination. Ultrasound has proved to be an ideal modality in the immediate evaluation of the trauma patient because it is rapid and accurate for the detection of intra-abdominal fluid or blood and is readily repeatable.

The goal of the FAST examination is the identification of abnormal collections of blood or fluid. In this regard, it obviates the need for diagnostic peritoneal cavity, but attention is directed also to the pericardium and to the pleural space.

6. Specific organ injuries

6.1. Liver injuries

Numerous methods for the definitive control of hepatic hemorrhage have been developed. Minor lacerations may be controlled by direct compression to the injury site. For similar injuries which do not respond to compression, topical hemostatic techniques have been successful. Small bleeding vessels may be controlled electrocautery. Microcrystalline collagen can be used. The powder is placed on a clean sponge and applied directly to the site. Pressure is maintained for 5–10 min. Fibrin glue has been used for both superficial and deep lacerations and appears to be an effective topical agent [18].

Suturing of the hepatic parenchyma remains an effective hemostatic technique. Although this treatment has been maligned as a cause of hepatic necrosis, hepatic sutures are often used for persistently bleeding lacerations less than 3 cm in depth. It is also an appropriate alternative for deeper lacerations if the patient will not tolerate further hemorrhage. The preferred suture is 2–0 or 0 chromic attached to a large and curved blunt needle. The large diameter of the suture helps prevent it from pulling through Glisson's capsule [19].

Most sources of venous hemorrhage within the liver can be managed with parenchymal sutures, and even injuries of the retrohepatic vena cava and hepatic veins have been successfully tamponaded by closing the hepatic parenchyma over the bleeding vessel [20].

Venous hemorrhage due to penetrating wounds that transverse the central portion of the liver can be managed by suturing the entrance and exit wounds with horizontal mattress sutures. Although intrahepatic hematomas may form that can become infected, this may be preferable to an intracaval shunt or deep hepatotomy. Suturing of the hepatic parenchyma is not always successful in controlling the hemorrhage particularly if it is of arterial origin [21].

Hepatic arterial ligation may be appropriate for patients with recalcitrant arterial hemorrhage from deep within the liver. However, its utility is limited since hemorrhage from the portal and hepatic venous systems will continue. Arterial ligation is a reasonable alternative to a deep hepatotomy particularly in unstable patients [22]. While ligation of the right or left hepatic artery is well tolerated in humans, ligation of the proper hepatic artery is not necessarily associated with survival. The fate of the dearterialized lobe is unpredictable [23].

An uncommon but perplexing hepatic injury is the subcapsular hematoma. This lesion occurs when the parenchyma of the liver is disrupted by blunt trauma, but Glisson's capsule remains intact. The hematoma may be recognized either at the time of the surgery or preoperatively if a CT is performed. Regardless of how the lesion is diagnosed, subsequent decision making is often difficult.

Resectional debridement is indicated for the removal of peripheral portions of nonviable hepatic parenchyma. The mass of tissue removed should rarely exceed 25% of the liver. Since additional blood loss may occur, it should be reserved for patients who are in good metabolic condition and who will tolerate additional blood loss.

Omentum has been used to fill large defects in the liver. The rationale for this is that it provides an excellent source of macrophages and that it fills a potential dead space with a viable tissue. The omentum can also provide a little additional support for parenchymal sutures and is often strong enough to prevent them from cutting through Glisson's capsule [24].

Since hemorrhage from hepatic injuries is often treated without identifying and controlling each individual bleeding vessel, arterial pseudoaneurysm may develop (**Table 3**). If the pseudoaneurysm enlarges, it will eventually rupture into the parenchyma of the liver, a bile duct, or into adjacent portal venous branch (**Figure 3**).

6.2. Gallbladder and extrahepatic bile ducts injuries

Injuries of the gallbladder are treated by lateral suture or cholecystectomy, whichever is easier.

Injuries of the extrahepatic bile ducts are challenge. Because of the proximity of the portal vein, hepatic artery, and vena cava associated vascular injuries are common and the patient's physiologic status is often poor. Sometimes laparoscopic injuries may occur (**Table 4**).

Injuries of the hepatic ducts are almost impossible to satisfactorily repair under emergency circumstances. One approach is to intubate the duct for external drainage and attempt a repair when the patient recovers. Alternatively, the duct can be ligated if the opposite lobe is normal and uninjured (see also **Figure 4** for gallbladder injury).

Grade	Injury description
I	**Hematoma:** subcapsular, <10% of the surface area
	Laceration: capsular tear, <1 cm in parenchymal depth
II	**Hematoma:** subcapsular, 10–50% surface area, intraparenchymal, 10 cm in diameter
	Laceration: 1–3 cm in parenchymal depth, <10 cm in length
III	**Hematoma:** subcapsular, >50% of surface area or expanding or ruptured subcapsular hematoma with active bleeding; intraparenchymal, >10 cm or expanding or ruptured
	Laceration: >3 cm in parenchymal depth
IV	**Hematoma:** ruptured intraparenchymal hematoma with active bleeding
	Laceration: parenchymal disruption involving 25–75% of a hepatic lobe or one to three Couinaud segments within a single lobe
V	**Laceration:** parenchymal disruption involving >75% of a hepatic lobe or more than three Couinaud segments within a single lobe
	Vascular: juxtahepatic venous injuries (i.e., retrohepatic vena cava or central major hepatic veins)
VI	**Vascular:** hepatic avulsion

Table 3. American Association for the Surgery of trauma liver injury scale.

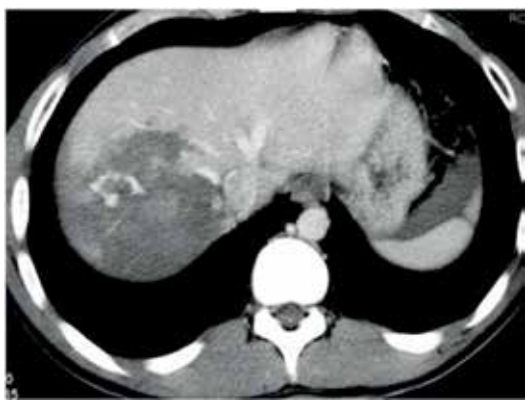

Figure 3. Grade III AAST liver injury, contrast enhanced CT image.

6.3. Spleen injuries

Splenic injuries are treated nonoperatively, by splenic repair, partial splenectomy, or resection, depending on the extent of the injury and the condition of the patient [25]. Enthusiasm for splenic salvage has been driven by the evolving trend toward nonoperative management of solid organ injuries, and the rare but often fatal complication of overwhelming postsplenectomy infection which is caused by encapsulated bacteria (e.g., *Haemophilus influenzae*, *Streptococcus pneumoniae*, *Neisseria meningitidis*) [26]. For this reason attempts to salvage the spleen are more vigorous in children [27].

Hilar injuries or pulverized splenic parenchyma are usually treated by splenectomy.

Type	Criteria
A	Cystic duct leak or leak from small ducts in the liver bed
B	Occlusion of an aberrant right hepatic duct
C	Transection without ligation of an aberrant right hepatic duct
D	Lateral injury to a major bile duct
E1	Transection >2 cm from the hilum
E2	Transection <2 cm from the hilum
E3	Transection in the hilum
E4	Separation of major ducts in the hilum
E5	Type C injury plus injury in the hilum

Table 4. Strasberg classification of laparoscopic bile duct injury.

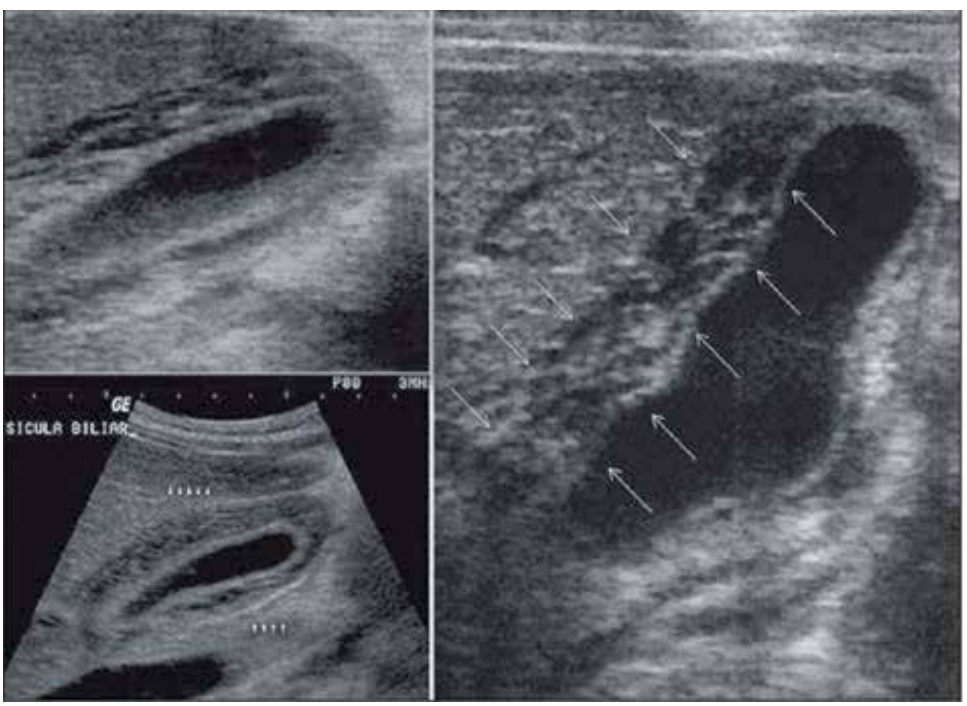

Figure 4. Longitudinal section of gallbladder (ultrasound view), parietal thickening with multiple echogenic layers intermingled with fluid.

Splenectomy also indicates for lesser splenic injuries in patients who have developed a coagulopathy and have multiple abdominal injuries, and it is usually necessary in patients with failed splenic salvage attempts [28] (**Table 5**).

If splenectomy is performed, vaccines against the encapsulated bacteria are administered (**Figure 5**).

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Grade	Injury description
I	**Hematoma:** subcapsular, <10% of the surface area
	Laceration: Capsular tear, <1 cm parenchymal depth
II	**Hematoma:** subcapsular, 10–50% surface area, intraparenchymal, 5 cm in diameter
	Laceration: 1–3 cm parenchymal depth, trabecular vessels not involved
III	**Hematoma:** subcapsular, >50% surface area or expanding, ruptured subcapsular or parenchymal hematoma, intraparenchymal hematoma >5 cm or expanding
	Laceration: >3 cm parenchymal depth or involving trabecular vessels
IV	**Laceration:** Involves segmental or hilar vessels producing major devascularization (>25% of spleen)
V	**Laceration:** Completely shattered spleen
	Vascular: Hilar vascular injury that devascularizes spleen

Table 5. American Association for the Surgery of trauma spleen injury scale.

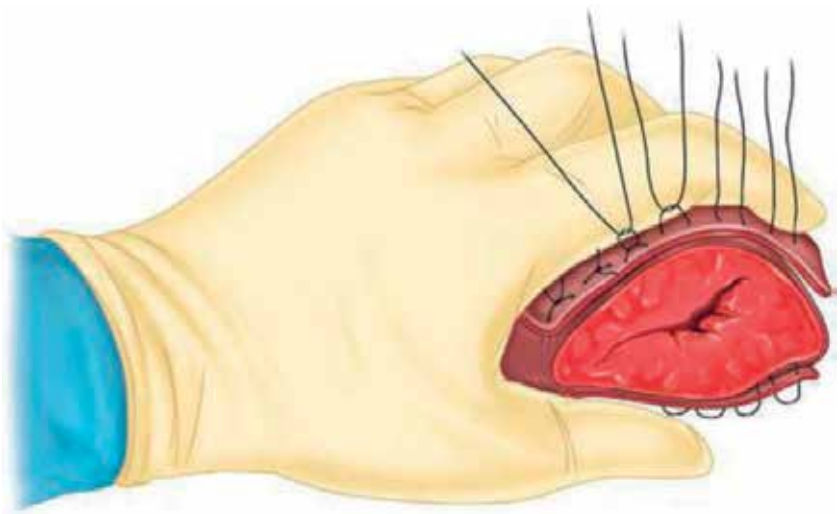

Figure 5. Interrupted pledgeted sutures may effectively control hemorrhage from the cut edge of the spleen.

The failure rate of nonoperative management of splenic injuries in adults increases with grade of splenic injury; Grade I, 5%; Grade II, 10%; Grade III, 20%; Grade IV, 33%; and Grade V, 75% in adults but not in children. Most failures occur within 72 h of injury [29]. Patients with significant splenic injuries treated nonoperatively should be observed in a monitored unit and have immediate access to a CT scanner, a surgeon, and operating room [30]. Changes in physical examination, hemodynamic stability, ongoing blood, or fluid requirements indicate the need for laparotomy. Arteriography with embolization has been reported to increase the success rate [31].

6.4. Diaphragm injuries

Diaphragmatic injuries are frequently difficult to detect initially.

The presence of abdominal contents in the thorax may not be obvious on initial chest X-ray. Insertion of a nasogastric tube may facilitate the diagnosis. However, diaphragmatic injuries may be missed even on initial CT scan (**Figures 6** and **7**).

Laparoscopy has also been used to evaluate potential diaphragmatic injuries [32].

Undiagnosed diaphragmatic injuries are a significant cause of morbidity and mortality.

6.5. Duodenum injuries

Duodenal hematomas are caused by a direct blow to the abdomen and occur more often in children than adults. Blood accumulates between the seromuscular and submucosal layers, eventually causing obstruction.

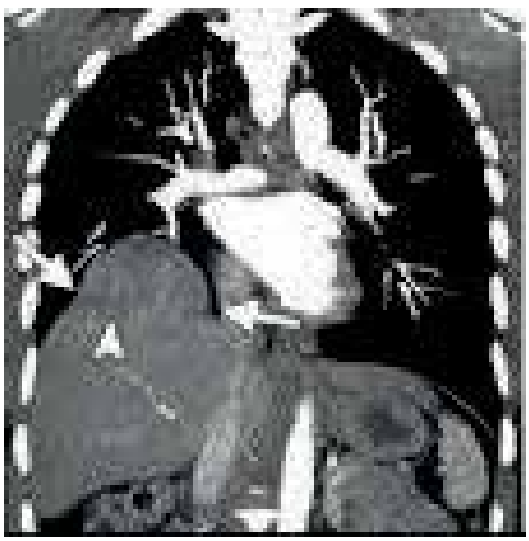

Figure 6. Hump sign of the diaphragmatic injury in CT scan (showed by arrows).

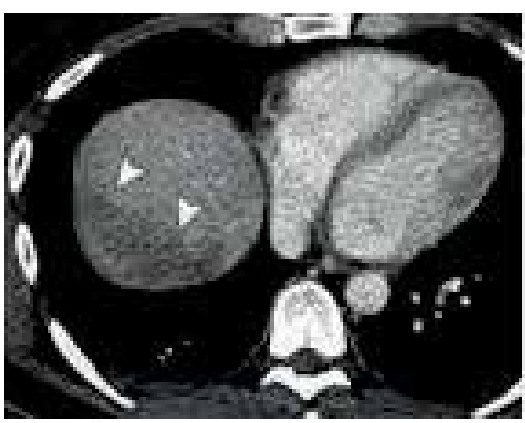

Figure 7. Band sign in diaphragmatic injury.

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Most duodenal hematomas in children can be managed nonoperatively with nasogastric suction and parenteral nutrition.

Duodenal perforations can be caused by both blunt and penetrating trauma. Blunt injuries are difficult to diagnose because the contents of the duodenum have a neutral pH, few bacteria, and are often contained by the retroperitoneum. Mortality may exceed 30% if the lesion is not identified and treated within 24 h.

Grade	Pancreatic injury
I	Hematoma with minor contusion/laceration but without duct injury
II	Major contusion/laceration but without duct injury
III	Distal laceration or parenchymal injury with duct injury
IV	Proximal laceration or parenchymal injury with injury to bile duct/ampulla
V	Massive disruption to pancreatic head

Table 6. American Association for Surgery in trauma pancreatic trauma grading system.

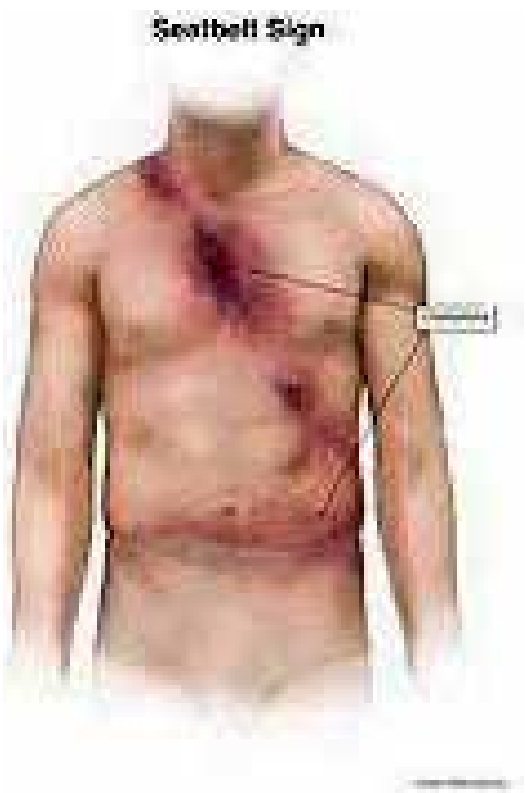

Figure 8. "Seat belt sign".

6.6. Pancreas injuries

Blunt pancreatic transection at the neck of the pancreas can occur with a direct blow to the abdomen. As an isolated injury, it is more difficult to detect than blunt duodenal rupture; however, a missed pancreatic injury is more benign [33]. Since the main pancreatic duct is transected, the patient will develop a pseudocyst or pancreatic ascites, but there is little inflammation since the pancreatic enzymes remain unactivated [34].

It is apparent that no ideal method exists for identifying pancreatic ductal injuries that cannot be ruled out by direct exploration [35].

Fortunately, majority of pancreatic fistulas will close spontaneously with only supportive care [36] (**Table 6**).

6.7. Gastrointestinal tract injuries

Both penetrating and blunt injuries can cause gastrointestinal tract (GIT) injuries. Injuries to the GIT may be clinically difficult to detect and are more common with penetrating than blunt trauma. GIT injuries occur in 30% of stab wounds and in 80% of gunshot wounds to the abdomen [37].

In blunt trauma, an abdominal wall bruise or "seat belt sign" should raise the level of suspicion since the finding is associated with a GIT injury [38] (**Figure 8**).

GIT injuries may be missed on FAST examination or CT scan. The finding of free fluid in the abdomen on CT scan without a specific solid organ injury is highly suspicious of a hollow viscus injury [39].

Such injury may be present even if the patient can tolerate a trial of fluids by mouth in hospital care. Patients have been able to walk out of the hospital and return later with fever and a rigid abdomen [40].

7. Conclusion

Blunt abdominal trauma comprises 75% of all abdominal injury and penetrating injuries may cause sepsis if they perforate a hollow viscous. Careful examination and close follow-up and early surgical consultation may reduce mortality and mobidity of these patients.

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Chapter 6

Emergency and Current Approaches to Thoracic Traumas

Turkan Dubus

Additional information is available at the end of the chapter

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Abstract

Trauma is one of the main causes of death in the young population, and trauma-related deaths usually occur in the initial hours following the trauma and are frequently associated with bleeding. In elderly populations, physiological changes and concomitant conditions alleviate the negative consequences of trauma. In injuries to the head or spine, thoracic trauma is the trauma category that is mostly associated with a serious risk of mortality, being generally penetrating or blunt thoracic traumas. Of these, blunt thoracic traumas are more frequent and fatal than penetrating traumas and are caused most often by traffic accidents. The most common causes of death related to blunt thoracic trauma include injuries to the heart and main vessels, and in such cases, emergency resuscitation, early diagnosis, and fast and effective treatment could be life-saving. Penetrating thoracic traumas may result in intrathoracic organ injuries and develop following stab wounds, firearm injuries, and explosions and are likely to require very urgent interventions. Emergency medical interventions could be life-saving in the presence of penetrating thoracic trauma, while mortality in these cases is mostly due to respiratory problems and heart or lung pathologies.

Keywords: trauma, thorax, emergency, surgery

1. Introduction

Traumas are one of the main causes of mortality and morbidity worldwide, particularly among young people, and is the leading cause of death in those below 45 years of age and the fourth leading cause of death in all age groups combined [1]. Based on 2012 data from the World Health Organization (WHO), traffic accidents are the 9th leading cause of death worldwide, with more than 1.2 million people dying from traffic accidents every year [2]. Trauma-related

death occurs most frequently in the initial hours following the trauma, and is often associated with bleeding. It is, therefore, of vital importance that patients with general body trauma are urgently, accurately and reliably evaluated in emergency units and the source of bleeding identified [3].

Among all forms of trauma, thoracic traumas are the leading cause of mortality after cranial and vertebral traumas. In Turkey, thoracic traumas account for almost 10–15% of all trauma cases seen in emergency units, and approximately 70% of all of these are blunt traumas. The leading cause of blunt thoracic traumas is traffic accidents, while other causes include assault and falls from height, among others. Of all blunt thoracic traumas, almost 15% occur as isolated thoracic traumas, while the rest involve multi-organ traumas, which, in the line of frequency, are traumas of the extremities, head, abdomen, pelvis and vertebra. The rate of mortality is about 2–5% in all thoracic traumas, while this rate may increase to 35% if accompanied by multi-system injuries [4–7].

The outcome of a trauma is determined by the cause and severity of the trauma, as well as the physical status of the exposed individual. Hemodynamics and respiratory parameters gain importance in the presence of thoracic traumas, and possible outcomes may include bleeding, pneumothorax, contusions, heart failure and intrathoracic pressure changes, and such functional abnormalities as hypoxia and hypotension, which may occur due to mediastinal dislocations [4, 8]. The consequences of blunt thoracic traumas may range from simple rib fractures to more severe conditions, such as multiple displaced rib fractures, causing flail chest, tracheal bronchial ruptures and cardiovascular ruptures. The most frequent site affected by thoracic trauma is the thoracic wall, followed in order of frequency by the pleural membranes and lung parenchyma [9].

The most significant cause of mortality, on the other hand, is cardiac and major vessel injuries. Emergency resuscitation, preferential diagnosis, basic interventions (such as thoracentesis or catheter/tube thoracotomy) and effective treatment are the necessary interventions for patients presenting with a trauma. The first assessment should involve checking the circulation and airways. Sternotomies or and thoracotomies are required in 10% of all blunt injuries, while emergency thoracotomies are required in 1–2% of cases [8]. Around one-third of all deaths occur at the time of trauma, while the remaining two-thirds occur after presentation to the emergency unit and medical interventions. The period from the trauma until the first hospital admission is considered as the “golden time” [5], as mortalities could be decreased by almost 30% with the timely transfer of patients from the trauma site, the effective implemented emergency resuscitation, and the emergency diagnosis and interventions at the hospital. Due to the high probability of mortality and morbidity, patients with thoracic traumas should be routinely monitored by Chest Surgeons, Emergency Medicine Specialists, specialists from the other relevant fields, nurses and other healthcare providers, all of whom play significant roles in both the diagnosis and treatment of such patients [2].

The presence of other injuries, such as large bone fractures, head traumas, intoxication, brain hypoxia or shock findings, as identified during the physical examination of patients presenting

with blunt thoracic trauma, can lead to confusion among healthcare professionals, and so radiological imaging may become necessary [4–6]. In this respect, it is important to retain devices such as ultrasound and X-ray, electrocardiogram (ECG) and echocardiography (ECHO) for the monitoring of trauma patients in emergency clinics. Previous studies have shown that bedside ultrasonography provides more specific and reliable data than physical examinations in assessments of pleural fluid and pneumothorax at the time of initial evaluation when a patient presents to the emergency unit with thoracic trauma [10–13].

When compared with blunt thoracic traumas, penetrating thoracic traumas are less common. Gunshot injuries account for 5% of all thoracic traumas, while sharp object injuries account for almost 37% of cases [14, 15]. Penetrating traumas may occur in isolated regions that requires sudden and mechanical power, at the trauma site, the object may cause tension and contusion in the body, while very severe traumas, on the other hand, may result in organ rupture.

Currently, the probability of survival after a penetrating trauma will be higher when the transfer from the scene of the accident to hospital is quick, and as a result of improvements in the comfort of patient care. In conclusion, the urgent assessment of trauma patients and timely emergency interventions could be life-saving. Moreover, the type of sharp object involved and the time of trauma are crucial in penetrating thoracic injuries. A prospective study has shown that a thoracotomy may be required in 14% of stabbings and 15–20% of gunshot injuries [16]. Not every patient requires a thoracotomy, and so thoracotomy decisions should be based on clinical and radiological evaluations.

The present study aims to describe in detail the steps to be followed from the first presentation until medical intervention for the effective management of patients being referred with any thoracic trauma, and to discuss the current concepts related to the various types of thoracic traumas.

2. History

From Ancient Greece up to the modern era, most records of thoracic traumas are related to deadly penetrating thoracic injuries. The Edwin Smith Papyrus (3000BC) from the Egyptian era provided information on three patients with penetrating thoracic traumas, two of whom were treated conservatively, while an esophagus suture was used on a cervical esophageal injury in the other [17]. In the thirteenth century, Theodoric defined two forms of rib fracture, based on whether the end of the fracture was turned inward or outward. Ribs that were turned outward were reduced and connected to each other after the application of local medication. In 1767, Larrey spoke about the importance of occlusive dressing and tube drainage in patients with an open hemothorax, although the drainage system used in 1867 by Hillier has been reported to be the most similar to the one being used today [18]. World War II was a turning point in the history of trauma, with the importance of immediately closing the defect in the chest wall following a penetrating thoracic trauma being noted by everyone in the war [19].

3. Primary approaches in patients with thoracic trauma

Trauma patients in particular should be followed with simple and systematic interventions. In recent years, trauma patients in emergency clinics have usually been treated in line with the American College of Surgeons' advanced life support (ATLS = Advanced Trauma Life Support) protocol, which is classified into primary and secondary care. Primary care consists of approaches for the identification and emergency treatment of life-threatening problems in patients exposed to sudden trauma. The individual or individuals responsible for primary care play significant roles in any intervention carried out related to the survival of trauma patients. Primary care should follow the following stages, the order of which should never be changed: ensuring airway flow and fixation of the neck vertebra (A), evaluation of the respiratory system (B), circulatory system (C), consciousness (D) and total body evaluation (E), known as "ABCDE" [5, 20].

3.1. Initial evaluation of life-threatening thoracic trauma

Patients with thoracic traumas are evaluated according to the ATLS protocol. There are six potentially morbid conditions that may occur following thoracic trauma: massive hemothorax, tension pneumothorax, open pneumothorax, flail chest, cardiac tamponade, air embolism and respiratory obstruction. Respiratory obstructions may result in the development of stridor, apnea, cyanosis and subcutaneous emphysema. Broken teeth following trauma, secretions, the development of hematoma due to cervical bleeding, and injuries to the larynx or trachea may result in obstructions of the airways, and these generally represent an indication for emergency intubation [21].

3.2. Secondary evaluation of life-threatening thoracic trauma

Secondary care, on the other hand, comprises the urgent identification of potentially life-threatening conditions and their treatment. Even hemodynamically stable trauma patients should undergo a detailed total body evaluation, and advanced investigations and examinations should be performed by relevant specialists. Detailed investigations are crucial at this stage, as it is possible that some traumas may be overlooked during primary care. It is also important to obtain a detailed anamnesis during secondary care. In the following stage, all body parts of the trauma patients should be evaluated with a physical examination, ultrasonography and/or radiological investigations (such as direct radiographs of the lungs, vertebra, pelvis, extremities, computerized tomography and MRI, if needed), as required [4, 20].

4. Traumatic pneumothorax

Traumatic pneumothorax develops when air from the atmosphere or lung parenchyma infiltrates the pleural space following blunt or penetrating trauma. The most commonly encountered etiologic cause is injury of the pleura or the lungs due to rib fractures. In cases of blunt thoracic

trauma, the bulla or blebs that may already be present in the lungs may rupture, or tracheal bronchi injury may develop [22, 23]. Traumatic pneumothoraxes are classified into three groups as follows: simple, open and tension pneumothorax, which are detailed below.

4.1. Simple pneumothorax

Simple pneumothorax frequently develops secondary to rib fractures, but may in rare cases develop following barotrauma. The patient presents with major symptoms of pain and dyspnea, and respiratory sounds are decreased at the side of pneumothorax. Diagnosis is based on the visualization of the pleural line on a chest radiography. The air may be spontaneously resorbed in patients with mild pneumothorax, though it may be sufficient to monitor such cases under nasal oxygen therapy. A tube thoracostomy must be performed in moderate or advanced cases of pneumothorax. Considering that the pneumothorax may alleviate in patients connected to mechanical ventilators, a tube thoracostomy should not be delayed in these patients [6, 7].

4.2. Open pneumothorax

Open pneumothorax is defined as the deposition of air between the parietal and visceral pleural membranes. In cases of penetrating thoracic trauma, an open pneumothorax develops due following the infiltration of positive pressure atmospheric air into the pleural space after an injury to the thoracic wall and parietal pleura, which is a life-threatening condition that requires emergency intervention [24].

Pneumothorax may also develop as a result of injuries to the parietal pleura or small airways, even if there is no penetrating injury. With each inspiration of the patient, air enters into the pleural space through the open region on the thoracic wall, as the defect in the thoracic wall is shorter than the trachea and has a lower resistance. In the event of the defect being larger than 0.75-times the tracheal diameter, air enters through the defect instead of the trachea [25], and pushes the heart and major vessels, and the mediastinum to the opposite side. As the capacity of the thoracic space decreases during expiration, the air moves out, and the heart and other mediastinal structures relocate back. This is called “mediastinal flutter”. The patient develops hypoxia, asphyxia, respiratory acidosis and decreased cardiac output. Torsion of the vena cava inferior and superior also occurs. Cardiac output decreases upon the decrease in cardiac venous return, and the patient may go into cardiac arrest.

The first intervention for open pneumothorax should be the closure of the terminal end of the open defect on the thoracic wall in such a way to that the entry and exit of air is prevented. Alternatively, the pneumothorax could be totally closed, and the patient could be monitored following a tube thoracostomy (**Figure 1**) [22, 26].

4.3. Tension pneumothorax

Tension pneumothorax develops as a result of injury between the parietal and visceral pleural sheets, or injury to the trachea or bronchi. It may develop spontaneously or be iatrogenic, other

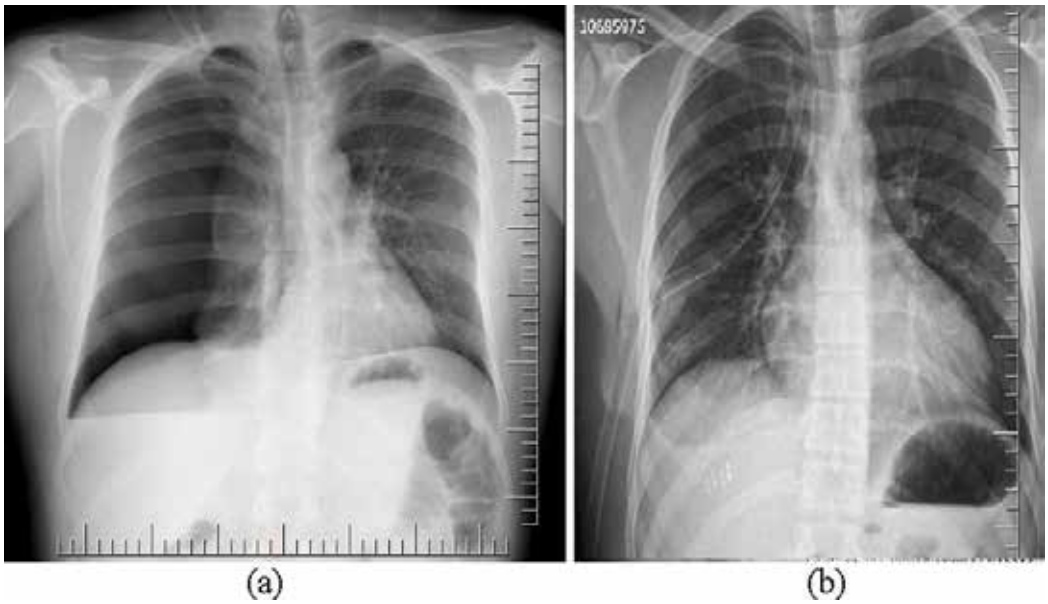

Figure 1. PA chest radiography of a case operated on with a right tube thoracostomy due to a developed traumatic right pneumothorax.

than being cause by trauma and is frequently encountered in closed pneumothorax. In tension pneumothorax, there is one-way air entry into the pleural space, and the increased air pressure within the pleural space puts pressure on the lung and pushes the mediastinum to the opposite side, which ultimately applies pressure on the other lung. Pushing the mediastinum along with the heart and other vascular structures towards the opposite thoracic space is called "mediastinal shift".

Increased mediastinal shift impairs cardiac venous filling and presents a life-threatening condition, with the patient developing dyspnea, tachypnea, hypoxia, tachycardia, hypotension and agitation. Radiological imaging shows increased air pressure in the pleural space, the total collapse of the lung on the affected side, the widening of the costal intervals, the detrusion of the diaphragm and the translocation of the mediastinal structures to the opposite side. In the absence of an emergency diagnosis and tube thoracostomy, the patient may experience a dramatic course, including hypoxemia, metabolic acidosis, decreased cardiac output, cardiac arrest or even death.

As an emergency intervention, to empty the air in the pleural space, a thoracentesis can be performed at the point of interception between the midclavicular line and the 2nd intercostal space to empty the air from the intrapleural space, thus reducing the pressure on the lungs and vital organs. After this, a tube thoracotomy should be performed as soon as possible. Of all cases of tension pneumothorax resulting from penetrating thoracic traumas, 75% can be treated with a tube thoracostomy [27, 28].

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5. Traumatic hemothorax

Hemothorax is the deposition of blood between the pleural membranes, and is most frequently caused by trauma. Traumatic hemothorax may originate from the thoracic wall, lungs, blood vessels, mediastinum or diaphragm. In blunt thoracic injuries, hemothorax frequently develops as a result of the bleeding of the pleura or the lung parenchyma, secondary to rib fractures [23]. While patients may remain asymptomatic, they may also present with hypovolemia or even shock, depending on the amount of bleeding. The development of hemothorax in structures with high blood flow, such as the heart, aorta, pulmonary artery, and vena cava inferior and superior, may very quickly become mortal [29, 30].

Hemothorax also has negative effects on the hemodynamics and respiratory system. Bleeding limits the expansion of the lung at the concerned region, and the mediastinum may shift to the opposite side if the bleeding continues. In an adult, the thorax may unilaterally be infiltrated by up to 6 L of blood. While bleeding of up to 500–750 mL can be tolerated, bleeding of 750–1500 mL can result in the development of tachycardia and hypotension, and signs of shock start to be seen in the presence of bleeding above 1500 mL (6). Diagnosis is made based on a direct lung radiography and thoracentesis, although thoracic CT is more specific for the diagnosis of hemothorax. Recently, in emergency clinics, bedside USI has also frequently been used for the diagnosis of hemothorax [6, 26, 29, 31].

A diagnosis of hemothorax can also be made when the hematocrit level in the sample obtained from the pleural space by thoracentesis is more than 50% of the hematocrit level measured in a spontaneously obtained blood sample. In the presence of penetrating traumas, hemothorax is most frequently caused by intercostal artery injuries, which, along with internal mammarian artery injuries, are the most common injuries causing persistent bleeding, and therefore require thoracotomies. For patients who are taken for emergency thoracotomies due to hemorrhagic shock, even applying finger pressure to the bleeding artery can rapidly improve the patient's vital signs [32].

Treatment approaches to traumatic hemothorax vary, depending on whether the injury is blunt or penetrating, and on the amount of bleeding. In such cases, the first procedure to be performed should be a tube thoracostomy, as this can serve as an important guide for monitoring the amount of hemothorax and for the prevention of intrathoracic hematoma deposition. Vascular access should be established and appropriate fluid and blood product replacements, such as erythrocyte infusion, should be performed as necessary. The drainage of 1500 mL or more blood following a tube thoracostomy is considered as a massive hemothorax and represents an indication for a thoracotomy. Emergency thoracotomy indications in hemothorax: (**Table 1**) [23, 26, 31].

Major complications of traumatic hemothorax include thrombus/hematoma in the pleural space, pleural infection, pleural effusion and chylothorax. Clotting blood should be replaced within 1 week following the trauma, or else the hemothorax starts to be organized and the risk of infection increases (**Figure 2**) [7, 22].

1. Drainage ≥1500 mL after initial tube thoracostomy,
2. 200 mL/h drainage during the first 2–4 h of follow-up,
3. 100 mL/h drainage during the first 6–8 h of follow-up,
4. ≥1500 mL/day drainage during the first 24 h,
5. Progression of shock despite treatment.

Table 1. Emergency thoracotomy indications in hemothorax.

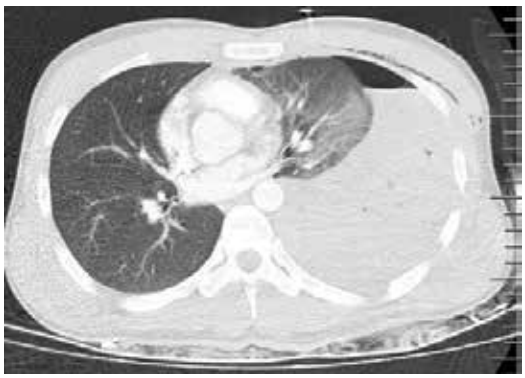

Figure 2. Thorax CT: Hemothorax following thoracic trauma.

6. Tube thoracostomy

A tube thoracostomy is performed for the treatment of traumatic pneumothorax and/or hemothorax, and it is an essential surgical intervention in cases requiring drainage of the pleural cavity in patients suffering a chest trauma. A tube thoracostomy is indicated for all open thoracic injuries, for pneumothorax of more than 10% and for radiologically-confirmed hemothorax. Even if there is no apparent pneumothorax and/or hemothorax, a tube thoracostomy could still be performed in intensive care patients with severe chest traumas who require mechanical ventilation. If a patient with penetrating or blunt chest trauma is transferred to the emergency unit when the vitals are completely lost, or about to be lost, it would not be erroneous to perform an emergency bilateral tube thoracostomy [9, 13, 33].

A tube thoracostomy is generally performed from the anterior axillary line, at the level of the 5th intercostal space from the lateral edge of the pectoralis major muscle. This is the region where the chest wall is at its thinnest, and is ideal for a tube thoracostomy. After local anesthesia, a skin incision is made 1–2 cm below the space where the chest tube is to be inserted, and using a clamp, the intercostal muscles are separated from the lower ribs to allow entry to the pleural space. Generally, a 28 or 32 F chest tube is inserted through this obliquely formed tunnel, and a closed underwater drainage system is formed by connecting the tube to the chest bottle. The chest tube should be directed as far as possible towards the apex for

pneumothorax, and towards the posterior and lateral for hemothorax. After the tube thoracostomy is complete, the position of the chest tube and the status of the air and/or fluid in the pleural space should be evaluated through a direct lung radiography. The chest tube should never be clamped for any reason while the patient is being transported or transferred outside the emergency unit. The tube thoracostomy is removed under the control of a lung radiography after the air leakage stops or drainage drops below 100 mL/day [22, 31, 34].

7. Emergency care thoracotomy

Emergency care thoracotomies are considered to be life-saving procedures for a limited patient group. Nowadays, the already effective and still developing emergency transport methods, and the fact that resuscitative interventions are performed before the patients arrive at the hospital, mean that a higher number of almost-morbid patients actually arrive at the hospital. The rate of survival following an emergency care thoracotomy has been reported to vary between 0 and 64%, with the best outcomes achieved for isolated penetrating cardiac injuries. On the other hand, this rate varies between 1 and 3% in cases of blunt and multiple trauma, for which the time of transfer to a hospital is longer. Emergency care thoracotomies are performed on almost-morbid patients in the emergency unit by making a left anterolateral incision between the 4th and 5th intercostal space. Emergency care thoracotomies should not be confused with thoracotomies performed in an operating room or in intensive care during the first hours following the initial injury [35, 36]. The reasons for an emergency care thoracotomy include the drainage of the pericardial tamponade, the control of intrathoracic or cardiac bleeding, the control of massive bronchovenous air embolisms, or bronchopleural fistula, open cardiopulmonary resuscitation or temporary occlusion of the descending thoracic aorta (cross-clamp placement) (**Table 2**) [36].

Contraindications for emergency care thoracotomies include cardiopulmonary resuscitation lasting longer than 15 minutes for penetrating chest traumas, cardiopulmonary resuscitation lasting longer than 5 minutes for blunt chest traumas, non-traumatic arrest, severe head trauma, severe multi-system injuries, the absence of appropriately trained staff and insufficient equipment. The preferable incision for emergency care thoracotomy is an anterolateral thoracotomy, which is carried out on the side with the predicted injury following a physical examination. The incision is performed transversely, the chest cavity is entered through the

Rescuable cardiac arrest after injury
• Prehospital cardiopulmonary resuscitation that lasts less than 15 min in patients exposed to penetrating trauma
• Prehospital cardiopulmonary resuscitation that lasts less than 5 min in patients exposed to blunt trauma
Serious continuous systolic hypotension after injury (≤60 mmHg) caused by:
• Cardiac tamponade
• Bleeding (intrathoracic, intraabdominal, extremity, cervical)
• Air embolism

Table 2. Indications of emergency care thoracotomy.

4th or 5th intercostal space and a chest retractor is inserted. After the bleeding is controlled and a steady heartbeat has been achieved, the patient must be transferred to the operating room as soon as possible [36].

8. Subcutaneous emphysema

Subcutaneous emphysema develops as a result of the entry of air into the subcutaneous soft tissue of the thoracic wall. Characteristic crepitations are felt during palpation, while diagnosis can be made through a visualization of air in the subcutaneous tissue and between the muscles in a lung radiography. The amount of skin emphysema depends on the amount of air leakage from the lungs. While only skin crepitations are present in mild cases, advanced subcutaneous emphysema can be seen when there is intense air leakage and a large defect in the parietal pleura. These cases may present with swelling of the head, neck and face, and while there is no specific treatment for subcutaneous emphysema, the underlying factor should be eliminated. To reduce subcutaneous emphysema, air drainage can be performed by injecting a few large-lumen wide-diameter granules subcutaneously and between the muscles of the anterior thoracic wall (**Figure 3**) [31, 32].

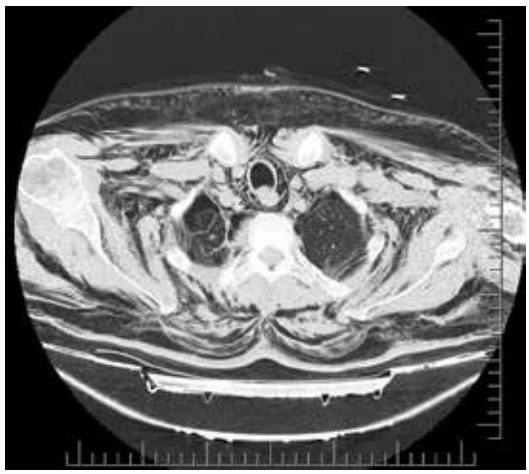

Figure 3. Thorax CT image of a patient with common subcutaneous emphysema that results from a blunt thorax trauma.

9. Contusion and hematoma of the thoracic wall

The thoracic wall is supplied by the internal thoracic artery, which originates directly from the aorta and branches to the intercostal arteries before spreading to all ribs along both sides of the

sternum. Bleeding may occur following rib fractures and the tearing of the thoracic wall muscles. As the thoracic wall has a large vascular supply, subcutaneous bleeding develops frequently, and this is more common in the elderly. Conservative treatment methods and blood transfusions, if required, are often sufficient for its management [26, 31].

10. Rib fractures

The most common type of injury, and one encountered in approximately 35–40% of all thoracic trauma cases, is rib fracture. Rib fractures are more frequent in the elderly due to the decreased elasticity of the thoracic wall. An anterior trauma to the thoracic wall generally results in rib fracture from the outer surface, while lateral traumas cause internal rib fractures. Rib fractures are mostly encountered along 4–9 and the middle axillary line, while fractures of the first and second ribs are generally rare, as these are supported by the clavicle, scapula and shoulders. That said, these ribs may be broken due to very high-energy trauma, and fractures of this kind may well be accompanied by subclavian vessel and brachial plexus injuries. It should be highlighted that rib fractures may be accompanied by lung, bronchus or cardiac injuries. Additionally, abdominal organ injuries, such as the liver and spleen, may occur in the presence of 9th–12th rib fractures, while trauma to the anterior thoracic wall may result in costochondral detachment, which is a more painful condition that requires a longer duration of treatment [22, 26, 31].

Painful tenderness is the most important symptom in the event of rib fracture, and the symptom generally increases with coughing, deep breathing and movement. Friction between the broken rib ends may be felt during a physical examination. Almost half of all rib fractures go unnoticed in lung radiographies, while thoracic CT is more specific for their diagnosis [22, 26]. The treatment of rib fractures is based on pain control and respiration exercises. Pulmonary rehabilitation, including respiration exercises, is crucial in preventing pain-induced secretions, lack of expectoration, atelectasis and pneumonia. Early-term complications of rib fractures include pneumothorax and hemothorax, and late-term complications include atelectasis and pneumonia. A tube thoracostomy is inevitable in the presence of hemothorax and pneumothorax, and surgical fixation becomes necessary when the fractured tips are displaced, and when there are fractures to more than one consecutive rib. Morbidity and mortality in rib fractures depend on the age of the patient, the number and localization of the fractured ribs, and the degree of the concomitant trauma (**Figure 4**) [6, 31, 37].

11. Flail chest

Flail chest may develop in the event of a fracture of three or more consecutive ribs, and can result in the paradoxical respiration of the thoracic wall in at least two places, preventing the formation of negative inspiratory pressure and lung expansion on the affected side of the thorax. Expiration, on the other hand, is not sufficient, due to the lack of adequate positive airway pressure as the concerned region moves outwards during expiration. This impairs

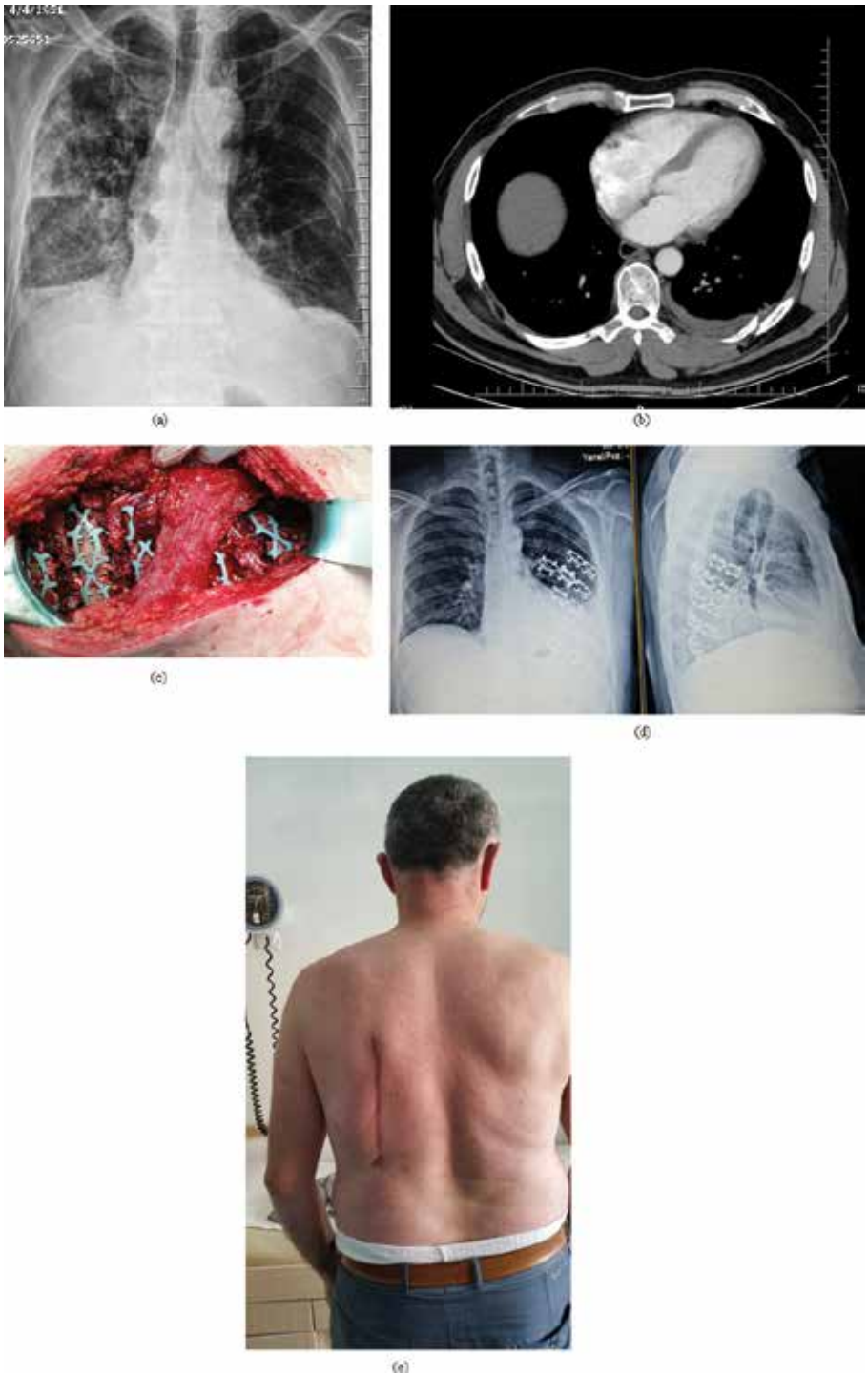

Figure 4. PA radiography, thorax CT and surgical fixation of the ribs of a patient with left multiple rib fractures developed following blunt thoracic trauma, and the post-operative appearance of the patient's skin incision scar.

hemodynamics, and there is always a risk of developing mediastinal shift, decreased cardiac output, hypotension, syncope and sudden cardiac arrest [7, 22].

Decreased respiratory sounds, as heard on auscultation, suggest hemothorax, pneumothorax and/or a lung contusion. A lung radiography and thoracic CT will show rib fractures accompanied by injuries [4, 37]. The treatment of flail chest is based on the use of strong analgesics (intercostal blockage, epidural analgesia and patient-controlled analgesia) and respiration physiotherapy, and while mechanical ventilation may become necessary, it is currently used less frequently. A bronchoscopy is also very important in preventing secretions. The rate of mortality associated with flail chest varies between 10 and 15%, and the most common causes of mortality are massive hemothorax, lung contusion and ARSD [28]. Nowadays, rib fixation (with MRI-compatible nitinol/titanium plates) is preferred in patients who stay in intensive care for long periods of time, who cannot tolerate other interventions or who need thoracotomies due to morbidity.

12. Sternum fractures

Sternum fractures mostly occur during in-vehicle traffic accidents, particularly in the elderly and in front-seat passengers. They are generally transverse fractures, and most commonly develop at the point of junction between the manubrium and corpus sterni, or at the corpus sterni. An accurate diagnosis can be made through a lateral radiography and thoracic CT, and patients should be hospitalized and closely monitored with ECHO and ECG assessments [26, 31, 38].

13. Clavicle fractures

Clavicle fractures have become more common since the use of seatbelts in vehicles became mandatory, and are seen most often in the 1/3rd middle part of the clavicle. A figure of eight bandage is often sufficient for the treatment of a clavicle fracture, and recovery is usually within 3 weeks with conservative therapy, although surgery may be required in rare cases (**Figure 5**) [22, 26, 31].

14. Scapula fractures

As the scapula is a thick bone and is well-protected by the muscles in the chest wall, scapula fractures only develop as a result of high-energy trauma. Scapula fractures can be diagnosed with a direct lung radiography or thoracic CT, and may be accompanied by brachial plexus injuries. For treatment, the shoulder is strapped and immobilized. Scapula fractures rarely require surgical treatment [26, 31].

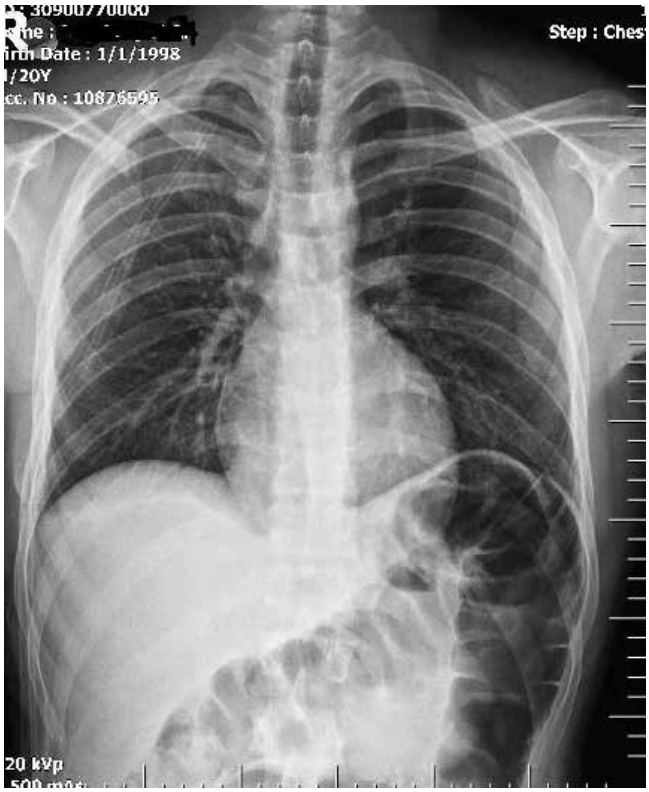

Figure 5. PA chest radiography of a case operated on with a right tube thoracostomy due to a developed traumatic right pneumothorax and right clavicle fracture.

15. Traumatic diaphragm injuries

Traumatic diaphragm injuries may occur due to blunt or penetrating traumas of the thorax and abdomen. Of all diaphragm injuries, 75% are associated with blunt traumas, and 25% are due to penetrating traumas. Their incidence varies between 1 and 5%. The right diaphragm is protected against injuries by the liver, and so diaphragm ruptures are five times more common on the left side than on the right side [9, 39, 40].

Conventional radiological investigations that can be performed when the patient is stabilized are the most important diagnostic methods. Diaphragm elevations, basal atelectasis, loss and/or irregularity of diaphragm borders, blunting of the costophrenic sinus and abnormal nasogastric tube positioning are among the key findings in a direct lung radiography. Furthermore, a fluoroscopy can indicate whether or not the diaphragm is immobile, or can display paradoxical movements [41, 42].

CT is also important for the identification of concomitant injuries, such as those to the liver, spleen or kidneys. The CT findings of a diaphragm injury include the interruption of diaphragm continuity, visualization of a defect in the diaphragm, herniation of the abdominal

organs into thoracic cavity, abnormal positioning of the nasogastric tube, direct contact of the posterior of the ribs with such organs as the liver and stomach, and injuries that progress from one side of the diaphragm towards the other side [9, 41, 42]. In cases where, despite all investigations, there is still suspicion, a thoracoscopy and/or laparoscopy can be performed during the same session.

16. Thoracoscopy

A thoracoscopy is performed to evaluate intrathoracic structures in elective conditions, and is not recommended for emergency situations [22]. It is a minimally invasive method used to clean clots from inside the thorax, to visualize diaphragm injuries, to examine the pericardium, and to remove foreign objects, to control bleeding and for the insertion of a chest tube. The patient must be stable enough to tolerate a double-lumen intubation [42, 43].

17. Conclusion

Patients with thoracic trauma should be evaluated quickly. Life-saving interventions should be implemented by the emergency physician and/or ambulance physician in the event of primary life-threatening injuries. In addition, a thoracotomy should be carried out by a thorax surgeon on site, and possible complications arising out of the specific situation should be considered. It is important to keep equipment ready and available in the event of chest surgery, and to take precautions based on the cause and severity of the trauma without losing time to distinguish between multiple traumas and isolated thoracic traumas.

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Section 3

Injuries in the Extreme Ages

Chapter 7

Geriatric Trauma

Banu Arslan

Additional information is available at the end of the chapter

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Abstract

Worldwide, the proportion of elderly people is constantly increasing. The aging of the baby boomers (people born between 1946 and 1964) and longer life spans (the maximum number of years that a human can live) result in a substantial increase in the number and proportion of older adults (whose age is ≥65). The older population is projected to more than double from 40.3 million in 2010 to 83.7 million in 2050 and, by 2050, it is estimated that older adults will represent 20.9% of the US population. In the early twentieth century, the average life expectancy at birth was 47.3 whereas it was 76.9 in 2000. With the increase in life expectancy due to improvement in quality of medical care, additionally, the oldest old age (age ≥ 85) forms a rapidly growing group within the older population. The rapid growth of these populations has many significant impacts on public health, emergency room visits, and economy.

Keywords: geriatric trauma, ATLS, trauma

1. Introduction and epidemiology

Worldwide, the number and proportion of elderly people is constantly increasing. The aging of the baby boomers (people born between 1946 and 1964) and longer life spans (the maximum number of years that a human can live) result in a substantial increase in the number and proportion of older adults (who is age ≥ 65).The elderly population is projected to reach to 83, 7 million in the year of 2050 and, by 2050, it is estimated that older adults will represent 20.9% of the U.S. population [1]. With the surge of the elderly population, there will be an increasing number of geriatric trauma patients admit to the emergency departments. Additionally, the rapid growth of these populations will have many significant impacts on public health and economy.

Geriatric trauma patients are less likely to be injured than younger people; however, they are more likely to have fatal outcomes. Death rates for Americans have decreased in the last century. Although there is a dramatic decline in deaths from cardiovascular diseseas, heart diseases remain the leading cause of deaths in the elderly. Also, trauma became the more common cause of death. According to the National Center for Health Statistics 2015 report, unintentional injuries became the seventh common cause of death in the elderly [2].

2. Pathophysiology

2.1. What is aging?

Aging is characterized by a progressive loss of physiological integrity, leading to impaired function and increased vulnerability to death [3]. This multifactorial and extremely complex process results in significant anatomic and functional changes in all major organ systems. Most important systems which are affected are seen in **Table 1**.

2.2. Age-related alterations and clinical consequences

Airway: The anatomy and physiology of the airway are affected with the aging process. Tooth decay which is common in elderly may cause loose, dislodged and subsequently aspiration of the teeth during emergency procedures such as endotracheal intubation (ET). Esthetic operations and loss of teeth interfere with achieving a good face-mask seal. Pharynx becomes more dry and fragile and care must be taken to prevent profuse bleeding while using laryngoscope. Oral cavity tumors and macroglossia may limit visualization of the vocal cords. Usage of Miller blade can be considered [4]. Also, cervical osteoarthritis increases the risk for spinal cord injury. Excessive movement of the neck should be avoided.

Age-related alterations: See **Table 1.**

2.3. Common mechanisms of injury

The common causes of geriatric trauma include falls, motor vehicle collisions, pedestrian injuries and thermal injuries and elder abuse (**Figure 1**).

Falls remain the leading cause of geriatric trauma and affect approximately 30% of persons aged ≥65 years each year [17]. Approximately 50% of people living in long-term care institutions fall each year, and 40% of them experienced recurrent falls [18]. Women experience significantly more fall-related injuries than men (35.7 vs.24.6%, respectively) [19]. Falls account for 40% of all injury-associated deaths [20]. Predisposing risk factors include age-related changes in muscle strength, gait and balance, poor vision and home hazards. İn addition, drugs and alcohol may contribute to falls. Anticoagulants usage are frequent in elderly and it may cause potentially lethal injuries even with minor traumas. Osteoporosis and the tendency to fall increase the risk of hip fractures. Also, falls are the most common cause of traumatic brain injury in the elderly. Even when those injuries are minor, they seriously affect older

Organ system	Age-related alterations	Clinical consequences
Respiratory system	Elastin component of the lung matrix ↑	Risk for pneumonia↑ [5]
	type III collagen ↓ [5].	Poor tolerance to rib fractures
	Pulmonary compliance ↓	Work of breathing ↑
	Osteoporosis	Risk for respiratory failure ↑ [8].
	Stiffness of the thoracic cage	Forced expiratory volume in one second (FEV1) ↓
	Outward recoil ↑	Forced vital capacity (FVC) ↓
	Kyphosis [6].	FEV1/FVC ↓[9].
	Chest wall compliance ↓	Functional residual capacity (FRC) ↓
	Thickening of the alveolar basement membrane [7].	Residual volume (RV) ↑
	Diffusion capacity ↓	Vital capacity (VC) ↓
	Gas change ↓	
	Muscle atrophy	
	Respiratory muscle weakness [8].	
Circulatory system	Vascular stiffness	Elevated baseline blood pressure
	Left ventricle (LV) wall thickness ↑	Atherosclerosis of coronary vessel
	[10].	Risk for cardiac ischemia ↑
	Retarded early diastolic cardiac filling and	Increased risk of dysrhythmias
	LV diastolic function ↓	Impaired cardiac reserve
	Afterload ↑	Cardiac index ↓
	Left atrial size ↑	Lack of classic response to hypovolemia
	myocyte mass with ↓	
	deposition of amyloid and collagen	
	Deterioration of the cardiac conduction	
	Decreased sensitivity to catecholamines	
	Maximal heart rate ↓	
	Maximum tachycardia response ↓ [11].	

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Organ system	Age-related alterations	Clinical consequences
Musculoskeletal system	Stiffening of structural instruments (tendons, ligaments, cartilage)	Risk of injury ↑
	Spontaneous rupture	Risk of fracture ↑
	Joint stability ↓	Difficulty for oral intubation
	ineffective repair of cartilage tissue	Risk of falls ↑
	Osteoarthritis (cervical, temporomandibular)	Mobility ↓
	Bone volume-mass ↓	
	Muscle size-number ↓ [12].	
	Osteoporosis	
	Sarcopenia	
Nutrition and metabolism	Taste acuity, smell and appetite decrease	Food intake ↓ [13].
	Poor dentition	
	Inability to eat independently	
Central nervous system	Brain volume decreases	Less contusions
	Replaced by cerebrospinal fluid [14].	Clinical signs may manifest late
	Protection against contusions	More subdural hematoma
	Blood can be collected	Vision and auditory functions ↓ [16].
	Parasagittal bridging veins stretch	Reaction time ↑
	More prone to tear injury	Attention span ↓
	Demyelination ↑	Less epidural hematoma
	Peripheral conduction velocity slows	Risk for spine and spinal cord injury
	Dura adheres to the skull more tightly	
	Cerebral blood flow	
	Cerebral oxygen consumption [15].	
	Degeneration of vertebras, intervertebral disks and facet joints.	

Table 1. Age-related alterations and their clinical consequences.

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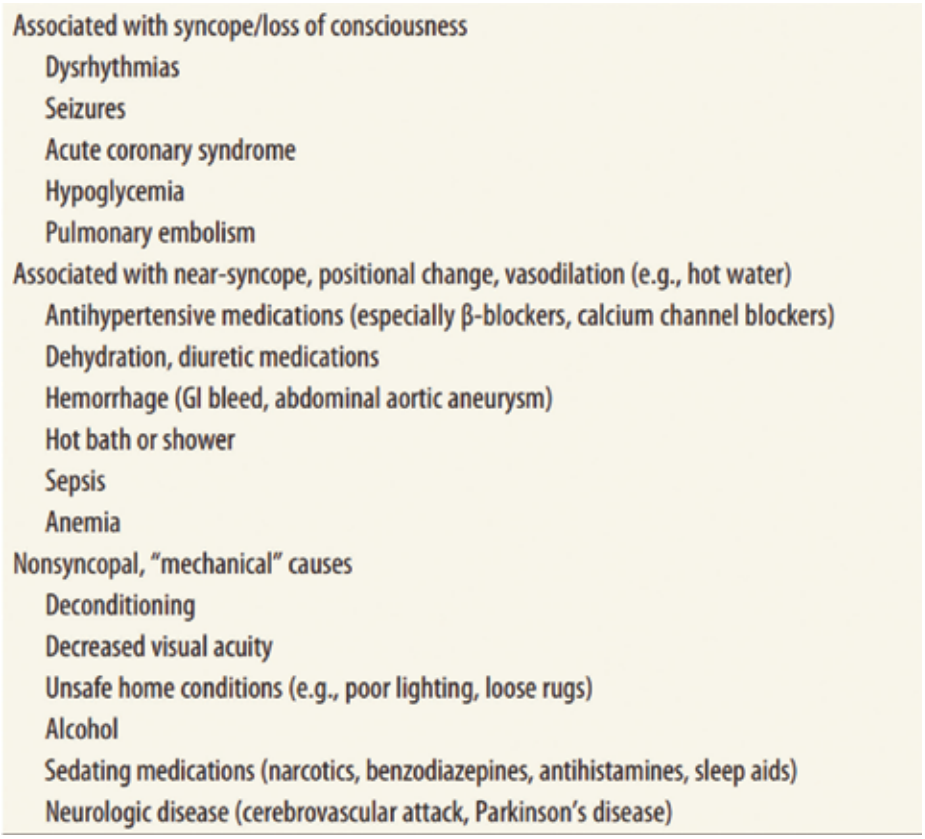

Figure 1. Common causes of falls in the elderly [64].

adults' quality of life by inducing a fear of falling, which can lead to self-imposed activity restrictions, anxiety, social withdraw and depression [21].

Motor vehicle collision involving elderly continue to increase. Age-related changes that include vision and hearing impairment, decreased night vision and glare resistance are the prominent factors on the incidence of injury and death. Additionally, medical conditions and medications may distort the reaction time, attention and judgment which increase the risk for the collision.

Pedestrian injuries: according to the 2015 pedestrian data, 19% of all pedestrian fatalities and an estimated 13% of all pedestrians injured were people aged 65 and older in the United States, and pedestrian-motor vehicle collisions are one of the most lethal mechanisms of injury in this age group with a 53% case fatality rate [22].

Thermal injuries: There is a direct relationship between age and burn mortality, as evidenced by the traditionally taught BauxScore. The empiric formula is clearly the simplest, whereby the sum of the patient's age and burn size predict mortality. Based on the data from the American Burn Association (ABA) National Burn Repository (NBR) from 2000 to 2009, overall

mortality was 4% in all age groups and 17% in seniors [23]. Moreover, for seniors there is a greater increase in mortality risk for every 1% increase in burn size and 1-year increase in age than among adults [23].

3. Clinical features and the management of injured elderly patients

The management of injured elderly requires the rapid assessment and rapid intervention of life-threatening situations. The assessment sequence should be same as in adults and pediatric population and includes the following elements:

3.1. Prehospital management and triage

The triage decision can be made through "field triage decision scheme" which was published by the American College of Surgeons Committee on Trauma (ACS-COT) to provide a guidance for the field triage process (**Figure 2**). Under triage, inaccurate triage which results in an assignment of lower triage level is more common among the elderly patients [24]. In order to avoid high under-triage rates in elderly, two important statements were added to Step Four of the scheme:

- SBP <110 might represent shock after age 65.
- Low impact mechanisms (e.g., ground-level falls) might result in severe injury.

Furthermore, we recommend that the injured elderly who met Step Four criteria should be transported to the trauma center [25]. Moreover, elderly seem to benefit more from triage to trauma center with improved outcomes [26]. Also, it is important that the transferring and receiving facilities develop transfer agreements in advance.

3.2. Primary survey

Primary survey of geriatric trauma patients includes rapid and efficient assessment of vital functions, assessment of the ABCDs, and identification and therapeutic intervention of life-threatening conditions as those for adults. Establishing and maintaining a patent airway to provide adequate oxygenation within-line cervical stabilization is the first objective. Avoiding excessive movement of the neck is crucial to prevent spinal cord injury. Because geriatric patients have limited respiratory reserve, early administration of supplemental oxygen is crucial. Early intubation should be considered if geriatric trauma patients present shock or chest wall injury/altered level of consciousness. For geriatric trauma patients, it is more challenging to recognize the early symptoms of shock. The aging process diminishes the physiologic reserve and chronic diseases can impair their ability to respond to injury; a tachycardic response may be absent or blunted. Also, medications such as β-blockers may mask tachycardia. Blood pressures are also misleading in the elderly patients. Due to increased incidence of underlying hypertension, the clinician must use a higher cutoff for hypotension than in younger patients [27]. In addition, frequently repeated measurement and interpreting the results according to baseline and previous ones may help the clinicians. Early and close monitoring must be instituted. Resuscitation of the elderly warrants special attention. Fluid

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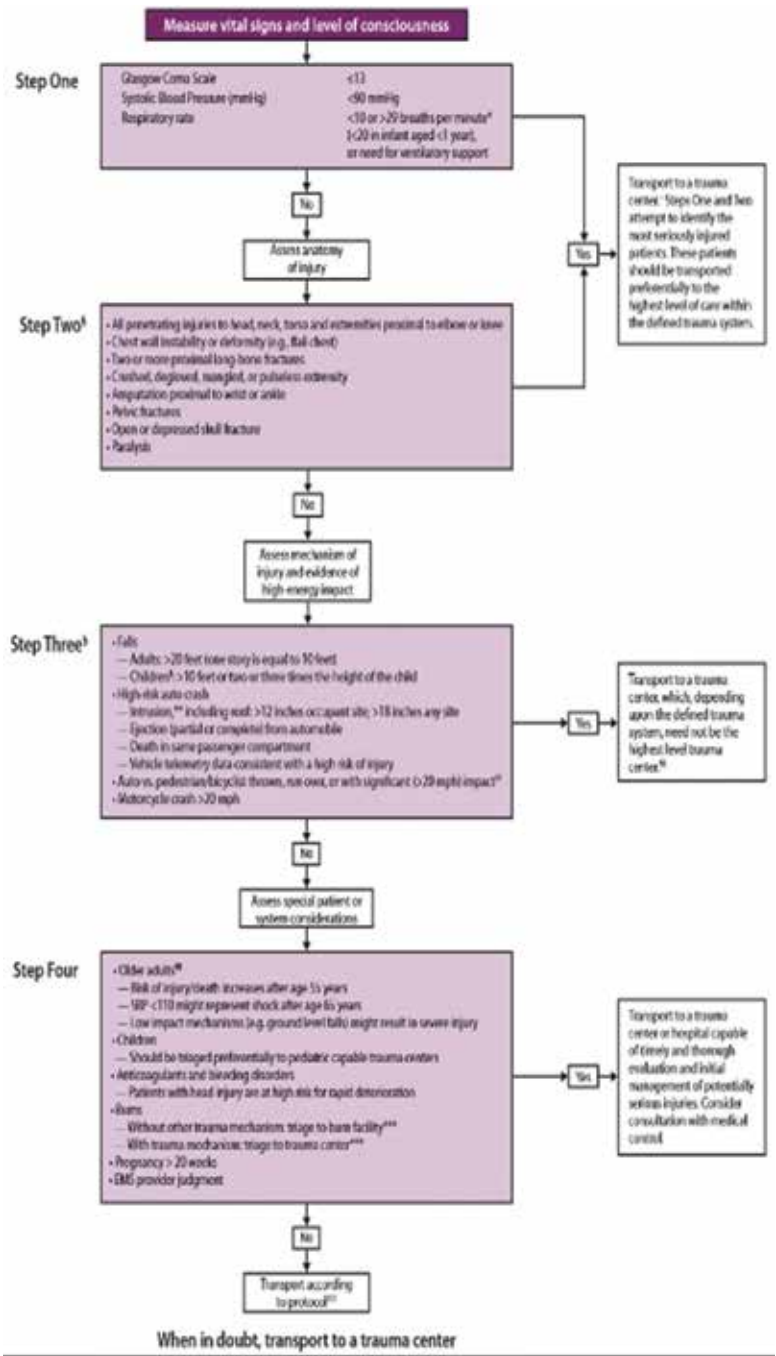

Figure 2. Guidelines for field triage of injured patients-United States, 2011 [65].

resuscitation is often challenging in geriatric trauma patients because of underlying cardiac dysfunction and concerns about precipitating heart failure. Primary survey also includes urinary and gastric catheters, arterial blood gas levels and X-rays (e.g., chest and pelvis). In the

elderly, due to alterations with aging, anticoagulant usage may increase the chance of profuse bleeding during the catheter procedure. Special care should be taken during this procedure.

3.3. Secondary survey

Secondary survey includes head-to-toe evaluation, reassessment of all vital signs, diagnostic tests and expanded history of the geriatric trauma patients. A detailed description of the secondary survey is provided separately; special circumstances in geriatric trauma patients are discussed here. Clinicians should focus on identifying and treating injuries which were not discovered during the primary survey. Geriatric trauma patients often present with significant occult injury mostly caused by minor mechanism such as ground-level falls. It is demonstrated that the elderly with blunt head trauma are more likely to present in occult fashion than youngsters, even if they have significant intracranial injury. Moreover, persistent vomiting and headache were less likely to occur in elderly with any intracranial injury [28]. Also, initially stable geriatric trauma patients may deteriorate rapidly and without warning. During the secondary survey it is essential to assess the alterations in mental status, especially compared to presentation.

3.4. High-risk injuries

The risk of complications increases with the severity of the *trauma*; however, even minor traumas such as ground-level falls or slipping while walking off a curb may seem relatively harmless in elderly patients, they can lead to severe injury and death [29].

3.5. Head injury

Traumatic brain injury (TBI) is a significant problem among the elderly. For the age of 65 years and older, falls are the primary mechanisms of TBI-related ED visits (81.8%) and TBI-related deaths (54.4%) [30]. In the review of the literature, it is recognized that older adults with moderate–severe TBI have poor outcomes with high rates of significant disability and mortality. Two major factors put geriatric trauma patients at a greater risk for increased incidence of TBI: age-related structural changes and preinjury anticoagulant-antiplatelet usage. First, with aging, parasagittal bridging veins stretch and make the elderly more susceptible to traumatic tears. Thus, the elderly have a higher incidence of subdural hematoma. Also, cerebral atrophy leads to a significant amount of blood accumulating in subdural area before clinical signs manifest. Rapid neurologic decline should be considered in these patients. Second, an increased incidence of the anticoagulant and antiplatelet therapy in the elderly may have detrimental consequences. It is suggested that taking anticoagulant therapy at the time of the injury increases the risk of intracranial hemorrhage [31] and is related with worse outcomes [32, 33]. One of the most frequently prescribed anticoagulant medications is warfarin. Also, Franko et al. concluded that warfarin use at the time of injury also makes mortality significantly higher after the age 70 [32]. Thus, immediate noncontrast head computed tomography (CT) is recommended for the elderly patients who take anticoagulant or antiplatelet therapy, even if their trauma seems minor. Additionally, rapid screening for anticoagulant use, INR value and subsequent correction with blood component therapy may improve outcomes.

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3.6. Spine injury

Cervical spine injuries are more common in the elderly and the incidence appears to be increasing [34, 35]. The most commonly seen injury site is upper cervical spine (UCS) especially the odontoid process [36] and caused by falls. The UCS injuries are associated with a high rate of mortality and morbidity. Elderly patients tend to sustain more C-spine fracture following simple falls such as ground-level falls [37]. It is attributed to increased frequency of preexisting cervical spine pathology such as osteoporosis and osteoarthritis [36]. It may also result in occult presentation, delayed diagnosis, increased risk for spinal cord injuries and difficulty in interpreting plain radiographs. Moreover, mild extension injuries followed by fall or rear-end motor vehicle crushes may cause central cord syndrome in the presence of preexisting spinal canal stenosis [37].

Thoracolumbar spine fractures in the elderly are usually associated with osteoporosis. Osteoporosis affects almost 50% of these individuals and contributes to the occurrence of spontaneous vertebral compression fractures. The majority of the osteoporotic vertebral fractures are situated in thoracolumbar spine, and the anterior wedge compression fractures are the most common site.

Treatment of diagnosed vertebral fractures in these individuals is still controversial. Two options are avaliable: conservative therapy and surgery. Unstable fractures, flexion distraction injuries and severe burst fractures causing neurologic deficit mostly indicate surgical intervention. However, in the patient who is neurologically intact, conservative treatment including bad-rest and bracing seems a more viable option depending on the type of fracture [38]. Consequently, we recommended that apparently low-energy level injuries should be considered as a high-risk for spine injury and investigated elaborately. CT scan is the preferred initial modality for assessing the geriatric cervical spine because The Canadian Cervical-Spine Rule, but not the National Emergency X-Radiography Utilization Study criteria, excludes patients aged ≥65 years from being considered low risk for cervical spine injury.

3.7. Chest trauma

Chest traumas account for ~796,000 emergency department (ED) visits annually in the USA [39]. For blunt chest trauma, the most prominent factors in etiology are falls and motor vehicle collisions. The elderly are more prone to incur chest injuries following blunt chest trauma, and this is associated with a high risk of mortality and morbidity [40]. Rib fractures and pulmonary contusions are more common in this population due to preexisting osteoporosis, loss of muscle mass and comorbidities [41]. The mortality and risk for pneumonia following blunt chest trauma significantly increase after 65 years [40, 41] and it is correlated with the increased number of rib fractures [40, 42]. In the presence of pulmonary contusion, clinicians should consider early ventilatory support because these patients are highly vulnerable to respiratory compromise. Given these risks, detailed physical examination, close observation and early administration of supplemental oxygen with adequate pain medication are highly recommended for elderly patients with even one rib fracture. Also, advanced imaging is warranted in older patients with multiple rib fractures. CT may be necessary to assess the extent of

injuries that might not be seen on plain radiographs. Simple pneumothorax and hemothorax are poorly tolerated by elderly patients. Thus, geriatric patients with life-threatening chest trauma should be considered for intensive care unit (ICU) observation.

3.8. Abdominal trauma

Abdominal examination can be less reliable and more difficult because of decreased pain sensation and increased laxity of abdominal wall musculature. Also, guarding and rigidity may be lacking in the elderly. Tachycardia response to hemorrhagic shock may not be seen even in the setting of significant blood loss. High index of suspicion and close observation must be continued to be avoided under-diagnosis. The Focused Assessment with Sonography for Trauma (FAST) can be used to detect intraperitoneal fluid in patients who sustain blunt abdominal trauma. CT remains the gold standard to diagnose intra-abdominal injuries. Retroperitoneum is an occult source of bleeding. Also, the risk of occult retroperitoneal bleeding is higher with chronic anticoagulant usage. Therefore, CT with contrast should be considered to evaluate hemorrhage, especially for the elderly patients who have pelvis or hip fracture.

3.9. Musculoskeletal trauma

Fractures are frequent in the elderly and can cause severe pain, disability and loss of independence. The increased risk of fracture with age may attribute to increased risk of fall, osteoporosis, sarcopenia and frailty.

Pelvis fractures: In the elderly, low-energy traumas such as ground level falls may result in pelvic fractures [43]. Although patients with pelvic fractures due to minor trauma generally do not present complications, mortality and morbidity increase with accompanied hemorrhage and other associated injuries. The portable AP pelvic X-ray should be obtained as a part of the primary survey. However, posterior ring fractures can be missed. Patients who have pelvic tenderness following pelvic trauma must be assessed regarding pelvic fracture. CT of the pelvis can be obtained in stable patients. If an active bleeding is suspected, pelvic contrast CT is recommended considering the risk of contrast-induced nephropathy. If an active bleeding is identified, arteriography and embolization can be performed for the patients in danger of life. Consequently, expeditious hemorrhage control with simultaneous emergency skeletal stabilization and resuscitation is crucial for the management of pelvic fractures in the elderly.

Proximal femur fractures: In elderly patients, hip fractures should be considered as a serious injury. They may lead to immobility, permanent dependence and death. According to several epidemiological studies, the incidence of proximal femoral fractures increases with age, starting at 40 years, with a steep increase after 75 years of age. The average age of patients with hip fracture is over 80, and nearly 80% are women [44]. Although isolated hip fractures do not usually cause class III or class IV shock, long-term prognosis mostly depends on age, comorbidities, anticoagulant therapy and frailty [45]. Hip fractures are the most common cause of accident-related deaths in older people accounting for 18% deaths within 4 months of a hip fracture and 30% within a year [46]. The risk of fracture increases with the number of falls [47] and backward fall mechanism and low bone mineral density (BMD) [48]. Most hip fractures can be diagnosed by typical history and clinical presentations. The first choice

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for diagnose is plain radiographs. However, it is estimated that 2–9% of fractures may be radiographically occult [49], and further imaging such as CT and MRI is required to make a definitive diagnosis. MRI has higher sensitivity than CT for detecting occult hip fractures. Additionally, nuclear medicine scintigraphy may be another choice for diagnosis due to high sensitivity. However, access to the scintigraphy usually is difficult and, it has limited capability to delineate the full nature of the fracture.

4. Special circumstances

4.1. Preexisting medical conditions

Elderly individuals are more likely to have preexisting comorbidities. The presence of a preexisting medical condition was associated with increase in mortality of elderly patients who sustained low or moderate severity trauma [50]. The most frequent preinjury comorbidities are hypertension (HT), diabetes mellitus (DM), coronary artery disease (CAD) and use of anticoagulants/antiplatelets [51]. Preinjury medical conditions usually make the management of geriatric trauma patients challenging; preexisting HT can hide the early signs of shock and cause delay or under-treatment and the presence of heart failure may cause volume overload and pulmonary edema during IV fluid therapy. ET intubation also would be challenging in the patient who has cervical or temporomandibular arthritis. Thus, early detection of preexisting medical conditions, appropriate treatment and follow-up care may improve outcomes following trauma in elderly.

4.2. Pre-injury medication usage

As the population ages, increasing numbers of elderly are being prescribed a medication for chronic medical conditions. It was shown that medications (especially sedatives and hypnotics, antidepressants, and benzodiazepines [52]) are particularly complex risk factors for falls and the risk of falling increases with the number of medications taken [18]. Also, polypharmacy is associated with occurrence of drug–drug interactions and adverse drug reactions which are frequently encountered in the elderly [53]. B-adrenergic blocking agents may limit the tachycardia response which can result in undesirable decreased cardiac output and reduced tissue perfusion. Calcium-channel blockers may prevent peripheral vasoconstriction and contribute to produce hypotension. Chronic diuretic use may lead to elderly patients being chronically hypovolemic, hyponatremic and hypokalemic. Additionally, declines in renal and hepatic function may alter the metabolism and clearance of these drugs. The side effects, drug interactions should always be considered and potentially nephrotoxic drugs must be given in adjusted doses based on calculated creatinine clearance.

4.3. Risk of bleeding

In the elderly population, both age-related structural changes and usage of some chronic medications may increase the risk of bleeding. Chronic anticoagulant therapy can increase the risk of hemorrhage, especially intracranial hemorrhage (ICH) [31]. The usage of warfarin at the time of injury also makes mortality significantly higher after the age 70 [32]. Recent

data show that Apixaban, dabigatran and rivaroxaban have lower risk of intracranial bleeding compared to warfarin [54]. However, they may potentially carry more risk of major bleeding than warfarin [55]. The influence of preinjury aspirin therapy on bleeding and the mortality is still uncertain [56]. However, the increased risk of subdural hematoma following head trauma was shown in the patients who are under preinjury aspirin plus clopidogrel therapy [57]. Hemorrhage cannot be tolerated appropriately. Therefore, the management of elderly trauma patients who are under anticoagulant therapy requires special care. Early diagnosis, close monitoring and maintaining optimal hemoglobin level are crucial. The optimal hemoglobin level for injured elderly patients is still controversial. A general suggestion is that hemoglobin concentration should be maintained over 10 g/Dl in order to maximize oxygen carrying capacity and delivery. Also, correction of coagulation defects is very important. According to the Eastern Association for the Surgery Trauma, all elderly patients with evidence of posttraumatic ICH on CT with Warfarin should have their INR be corrected toward a normal range within 2 h of admission [58]. Moreover, tranexamic acid, an antifibrinolytic agent, may reduce blood loss after traumatic injury. According to the recent data, tranexamic acid may reduce mortality without significant adverse side effects when given within 1–3 h [59]. The dose is 1 g of tranexamic acid IV bolus over 10 min, followed by 1 g IV over 8 h.

Pain management altered physiology changes the way analgesic drugs are distributed and metabolized therefore the pain management of geriatric trauma patient requires extra caution. The main approach should provide optimal treatment of pain while minimizing the risk of medication-related adverse effects. The standardized tools to assess the pain may be beneficial [60] (**Table 2**).

Pain type or source	Nonopioids	Opioids	Adjuvant analgesics	Other	Comments
Major trauma generalized pain	Acetaminophen, NSAIDs during posttrauma healing phase	Bolus or continuous IV opioids* during emergency phase; PO or IV opioids during healing phase	IV ketamine (very rare)	Inhaled NO	Use of ketamine is restricted to pain refractory to other treatments due to severe CNS side effects Inhaled NO is used for incident pain
Major trauma (regionalized pain)	NSAIDs (parenteral, oral) during posttrauma healing phase	Bolus or continuous IV opioids during emergency phase plus regional anesthesia	IV ketamine (very rare)	Inhaled NO	Use of ketamine is restricted to pain refractory to other treatments due to severe CNS side effects. Inhaled NO is used for incident pain

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Pain type or source	Nonopioids	Opioids	Adjuvant analgesics	Other	Comments
Burns	Acetaminophen, NSAIDs, during rehabilitative phase	High dose of IV opioids ± PCA for NPO patients; oral opioids (e.g., morphine, hydromorphone) when taking PO	Parenteral ketamine (very rare) IV lidocaine (very rare)	BNZ Inhaled NO	Use of ketamine is restricted to pain refractory to other treatments due to severe CNS side effects. Inhaled NO is used for incident pain Infusion of low-dose lidocaine is restricted to burn pain refractory to opioids.
Minor trauma	Acetaminophen, NSAIDs	Opioids for mild-to-moderate pain			
Procedural pain	NSAIDs for preemptive analgesia and postprocedural pain	IV opioids (e.g., morphine, hydromorphone, fentanyl) unless contraindicated**	Local anesthetics (e.g., EMLA®, lidocaine, bupivacaine, ropivacaine) IV ketamine	BNZ (e.g., diazepam, lorazepam, midazolam) Inhaled NO Propofol***	Local anesthetics may be applied topically (e.g., EMLA®), injected into tissue, or used for nerve blocks Use of ketamine limited by severe CNS side effects

BNZ: benzodiazepines; CNS: central nervous system; EMLA®: Eutectic Mixture of Local Anesthetics (lidocaine and prilocaine); IV: intravenous; LAs: local anesthetics; NO: nitrous oxide; NPO: nothing per os (by mouth); NSAIDs: nonsteroidal anti-inflammatory drugs, including aspirin: PO: per os (oral); PCA: patient-controlled analgesia; PRN: as needed; TD: transdermal.

Modified from American Pain Society, Section IV: Management of Acute Pain and Chronic Noncancer Pain. http://americanpainsociety.org/uploads/education/section_4.pdf.

*Titrate opioids carefully to maintain stable cardiovascular and respiratory status. Monitor neurological and neurovascular status continuously in patients with head injury or limb injury, respectively.

**Contraindications to opioid analgesia include altered sensorium, full-term pregnancy, lung disease or inability to monitor and manage certain side effects (e.g., respiratory depression).

***Hypnotic general anesthetic that produces good sedation.

Table 2. Systemic medications for acute pain management.

The search of literature mostly suggests that paracetamol should be considered as a first-line treatment for both acute and chronic pain due to its efficacy and good safety profile. NSAIDs are one of the most widely used painkillers. Clinicians must be concerned about the

Type of Abuse	Clinical Markers Indicating Abuse or Neglect
Physical abuse	Abrasion and laceration in sites other than the arms and legs or multiple ones should raise suspicion. Bruising on face, neck, the chest wall, the abdomen, the buttocks, the palms and soles. Tramline bruising. Fractured, subluxed, or avulsed teeth Fractures of the zygomatic arc, mandible and maxilla Fractures not involving the hip, humerus, or vertebra A spiral fracture of a large bone with no history of gross injury or atypical site
Verbal or psychological abuse	Subtle signs of intimidation, such as deferring questions to a caregiver or potential abuser Evidence of isolation of victim from both previously trusted friends and family members
Sexual abuse	The majority of victims have cognitive impairment or have functional limitations. Bruising of the uvula and the palate may indicate forced oral copulation Bleeding, abrasions, lacerations in the anogenital area as well as difficulty in sitting New diagnosis of sexually transmitted diseases, especially in nursing home residents (and especially in cluster outbreaks)
Financial exploitation	Inability to pay for medicine, medical care, food, rent, or other necessities Failure to renew prescriptions or keep medical appointments Unexplained worsening of chronic medical problems that were previously controlled Malnutrition, weight loss, or both, without an obvious medical Firing of home care or other service providers by abuser Unpaid utility bills leading to loss of service
Neglect	Deep decubitus ulcers in multiple sites or foul-smelling, and necrotic ulcer may indicate neglect Refusal to eat may indicate improper feeding technics such as forceful assistance may lead to choking, aspiration and pneumonia. Need help with eating? Adverse side effects due to improper dosing of an indicated drug Overdosing patients to keep them quite and manageable Recent decline in personal care, dirty clothes, multiple insect bites are the signs of poor hygiene.

Table 3. Clinical markers indicating abuse or neglect [66, 67].

potentially life-threatening side effects such as gastrointestinal hemorrhage. And, it must be given with proton-pump inhibitor (PPI) cover. In carefully selected and monitored patients, opioids usually provide fast and effective pain relief. The weak opioids including co-codamol, codeine and dihydrocodeine may elicit adverse effects such as cognitional decline and constipation. Although tramadol's GI effects lesser than other weak opioids, potential to precipitate delirium and reduced seizure threshold may limit the usage [61]. Strong opioids include morphine, oxycodone and fentanyl may also be used to treat moderate and severe pain, especially if the pain causes functional impairment. Dose titration based on patient's response is required, in order to avoid side effects such as sedation, nausea or vomiting.

4.4. Elder abuse/maltreatment

Elder abuse is a global public health and human rights problem which is associated with morbidity and premature mortality. According to the latest data, the prevalence of elder abuse can vary widely. In USA, 10% of older adults have experienced some form of elder abuse [62].

Unfortunately, these statistics may represent an inaccurate underestimation because elder abuse often is not recognized and tends to be underreported. Elder abuse can be classified into five main categories and manifestations is shown in **Table 3**, but several types of abuse may occur simultaneously.

The risk factors can be stated as: shared living situation, social isolation, dementia, female gender, relationship of victim to perpetrator (spouse), personality characteristics of victim (hostility), race (black) [63].

Also it is crucial to screen for elder abuse in geriatric trauma patients, especially who have cognitive impairment or who are unwilling to report it due to fear. Health professionals are well positioned to identify elder abuse, detect vulnerabilities and evaluate interventions. If abuse or neglect is suspected or confirmed, management strategies should be applied.

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Although trauma victims constitute around one-tenth to one-eighth of the total patient volume in hospital emergency departments, the burden of trauma on humankind is beyond these statistics. The twenty-first century is witnessing a growing threat on human beings imposed by many sources, namely natural disasters, terrorism and other conflicts, warfare, and transportation accidents; all of which ignite the rise of major trauma incidents worldwide. Physicians, therefore, get involved in trauma management more and more frequently in time. They need to evaluate, diagnose, treat, and stabilize victims and help them take part in active and productive life as soon as possible.

Technological advances have provided many techniques to augment trauma care and resuscitation, fracture healing, wound care, casts and splints, sutures, and transfusions. However, the successful management of trauma warrants a collaboration of emergency medicine, surgical disciplines, intensive care medicine, and almost all the resources of a hospital. This work is an example of a multidisciplinary approach that is a must to maximize synergistic efforts to deliver contemporary care for trauma victims of all ages throughout the world.

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