Angiography and Endovascular Therapy for Peripheral Artery Disease

Authored by Yoshiaki Yokoi, Keisuke Fukuda, Masahiko Fujihara, Akihiro Higashimori and Osami Kawarada

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Preface

This book grew out of my earlier textbook entitled *Angiography for Peripheral Vascular Disease* with a chapter on "Angioplasty, Various Techniques, and Challenges in the Treatment of Congenital and Acquired Vascular Stenosis." Although this previous work was well received, the subject matter was broad with only limited pages allowed. Thinking of most of the physicians who deal daily with peripheral artery disease, I felt the need to focus on lower limb angiography since there are not many textbooks on angiography as a preoperative imaging modality for peripheral artery disease. For example, compared to neurovascular intervention, there is no standardized technique for peripheral artery disease. This textbook is, thus, a comprehensive publication on angiography of the aortoiliac, femoropopliteal, and below-the-knee arteries in order to provide detailed assessments for endovascular treatment. Peripheral artery disease does not refer to atherosclerotic stenosis and/or occlusion, and clinicians should have basic knowledge not only in atherosclerotic causes but also in nonatherosclerotic causes of lower limb ischemia. Endovascular therapy by catheter techniques is now widely employed and is becoming the first-line approach in revascularization for symptomatic peripheral artery disease. As a vital tool in determining the final diagnosis and ensuring the best outcomes in endovascular therapy, angiography can be considered indispensable. Thus, I believe every interventionist should work to maximize his or her abilities and performance in order to obtain the best angiographic images possible.

Each chapter in this textbook is consistent in direction since all of the angiograms used were obtained in the same catheter lab with my colleagues. It does mean this book would not have been possible without the dedication of my friends and colleagues Dr. Masahiko Fujihara, Dr. Akihiro Higashimori, Dr. Keisuke Fukuda, and Dr. Osami Kawarada.

I do hope that this textbook *Angiography and Endovascular Therapy for Peripheral Artery Disease* will serve as a practical source of information for all vascular interventionists.

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Basics of Angiography for Peripheral Artery Disease

Yoshiaki Yokoi

Additional information is available at the end of the chapter

Abstract

Angiography has been historically used to image the peripheral artery system and still remains the gold standard for diagnostic and endovascular treatment. There is no standardized method for lower limb artery angiography. In this chapter, the basic standard technique for angiography of peripheral artery is described from aortoiliac, femoropopliteal and below the knee arteries. To obtain a good image, adequate contrast dose and image size must be determined with the appropriate catheter. For puncture, echo-guided approach is becoming popular; each lab needs to have echo machine to minimize the vascular complication. In cases of renal dysfunction, CO_2 angiography is suited. However, care must be taken to deliver gas into arterial system and to know the merit and demerit of CO_2 angiography.

Keywords: peripheral artery, peripheral artery disease, contrast angiography, echo-guided puncture, CO_2 angiography

1. Angiography-suite for endovascular therapy of peripheral artery disease (PAD)

1.1. Detector size

High resolution, accurate imaging is the key to success in endovascular therapies. In recent years, most machines provide fairly good images. An important point is the detector size of the angiography machine. Some physicians still use a coronary lab for peripheral artery intervention, however, when considering the vessel length and area, at least a 30 cm detector is needed. In **Figure 1**, two types of detectors are shown.

In peripheral artery angiography, the 30 cm system on the left (INNOVA 3100, 30 cm, GE healthcare, Uppsala, Sweden) (**Figure 1A**) is basically used while the 20 cm coronary system (INNOVA IGS620, 20 cm, GE healthcare, Uppsala, Sweden) (**Figure 1B**) is too small for peripheral artery angiography. For example, the superficial femoral artery (SFA) is the longest vessel and difficult to visualize in its entirety. In **Figures 1** and **2**, two SFA short lesions are shown.
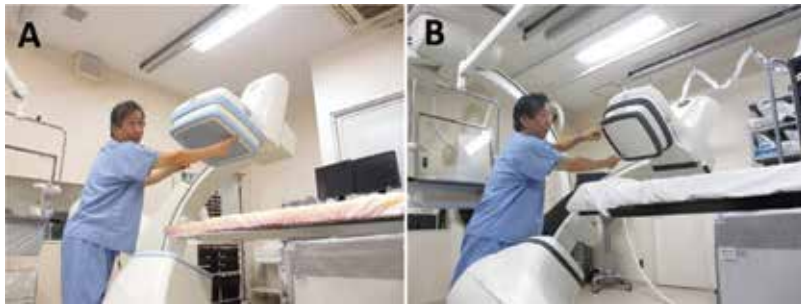
Figure 1. Detector size: (A) 30 cm × 30 cm, (B) 20 cm × 20 cm. In peripheral artery angiography, at least a 30 cm image size is needed. The 20 cm image size is too small for peripheral artery angiography.
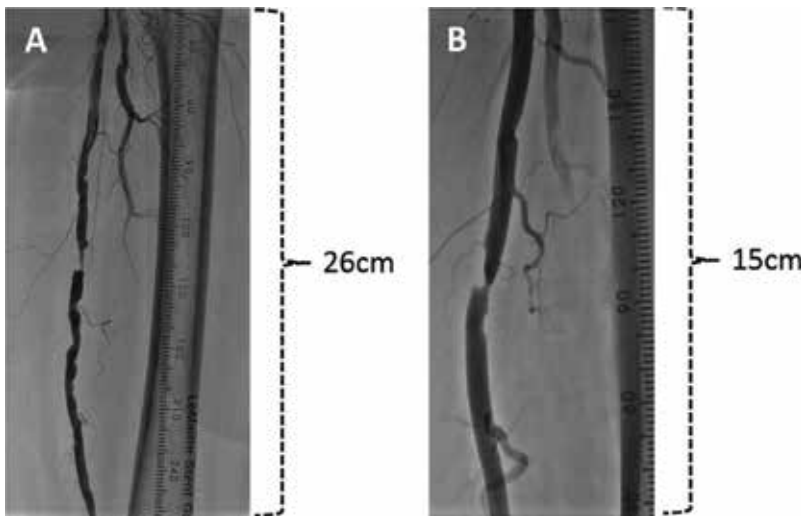
Figure 2. Image field of 20 cm and 30 cm detector in SFA. (A) In the 30 cm detector, about 26 cm of SFA is visualized. (B) In the 20 cm detector, only 13 cm of SFA is seen.

In the 30 cm panel, about 26 cm of the SFA can be visualized and intermediate stenosis around the culprit lesion (**Figure 2A**) can be discerned. On the other hand, the coronary detector could visualize only 13 cm of the SFA in 20 cm mode (**Figure 2B**). In a coronary lab, to visualize the SFA or below the knee (BK) arteries, the table is panned but a good static image of the lesion is difficult to obtain.

1.2. Extra monitor

In an angiographic suite, operators usually stand on the right side of the table. Most labs use one monitor and all medical staffs rely on this one screen. In right limb angiography via the left femoral artery approach, the operator who is standing on the right side has difficulty manipulating the catheter. In this situation, one operator needs to stand on the left side of the table to manipulate the catheter and hold the sheath. For this purpose, an extra-monitor should be installed (**Figure 3**).

Figure 3. Extra-monitor. In right superficial artery (SFA) intervention, the main operator stands on the left side of the table watching the extra-monitor while the assisting operator watches the central monitor. Without moving the central image monitor, the main operator can manipulate the catheter from the left side.

A typical right superficial artery (SFA) intervention is shown in **Figure 3**. The main operator is standing on the left side of the table and watching the extra-monitor while the assisting operator keeps an eye on the central monitor. Without moving the central image monitor, the main operator is able to perform the procedure. This extra-monitor is useful in the left brachial approach as well. It is a convenient way to intervene in the right femoropopliteal artery or cross-over approach for right below the knee arteries. In the left below the knee artery procedure via the cross-over approach, the C-arm is rotated to the left side. The cranial side operator may not see the central image. In this situation, the extra monitor can be placed on the left cranial side.

1.3. Injector

For most of the small vessels in selective angiography, hand injection of the contrast dye is adequate. However, for optimal opacification of high-flow blood vessels like the aorta, the use of a power injector is mandatory. A constant and high volume of dye should be injected through an electronically calibrated power injector. There are two types of injectors: one is a conventional power injector and the other is an assisted device that introduces small or large amounts of dye by an injector attached to the catheter table. The contrast volume is adjusted manually so that even a small dose of dye can be injected. However, the space on the left side of the table is occupied by this assisted device. Thus, a conventional power injector mounted to the ceiling is preferable since it affords more space around the catheter table. Furthermore, the distance allows a significant reduction in radiation exposure during dye injection. With the assisted device, radiation exposure is difficult to prevent since the operator has to be beside the table during dye injection (**Figure 4**).

Figure 4. Power injector mounted to the ceiling. The ceiling-mounted injector allows more space around the catheter table.

1.4. Contrast dose

Contrast-related factors include the vascular access site, injection time duration, injection rate, contrast volume and dye concentration. The key factor is the injection rate. An increased rate of injection can induce a greater extent of vascular opacification but the safety and total volume of the contrast dose must be carefully monitored. The contrast volumes for opacification of the major arteries are shown in **Table 1**. These are the injection volumes mainly used in our catheter laboratory although the actual contrast volume depends on the patient's condition, the catheter size, amount of contrast and speed of injection. Therefore, the contrast dose should be individualized for each case.

Location	Catheter	Injection rate (ml/s)	Total volume (ml)
Aortoiliac	5Fr Pig tail	14–16	15–25
CFA-SFA-Pop A	4–5Fr MP	5–7	16–20
Run: CFA-BTK	4–5Fr MP	4	9–12
SFA	4–5Fr MP	4–5	8–10
Run: BTK	4Fr MP	3–4	10–12
BTK	4Fr MP	3–4	5–7
Below the ankle	4Fr MP	3–4	5–7

Table 1. Contrast injection rate and injection volume.

There is no universally agreed upon threshold in the degree of renal dysfunction beyond which intravascular iodinated contrast medium should not be administered. We use Visipaque 320 [2]. Contrast-induced nephropathy (CIN) is an infrequent adverse reaction to iodinated contrast agents [3]. In endovascular procedures, particular complex procedures are associated with CIN and larger doses of contrast are considered a risk factor. Thus, as a precaution against CIN, the use of contrast media at the lowest dosage possible is advised. To minimize the contrast dose, we dilute Visipaque 320 by adding 30 cc of saline solution in a 100 cc bottle. The key factor is the injection rate which indicates the amount of dye per second. In our experience, 1/3rd diluted contrast does not decrease image quality.

1.5. Radiation safety

Angiography machines which use fluoroscopy for endovascular work are equipped with pulsed fluoroscopy instead of continuous fluoroscopy and this, to a large extent, helps to reduce the radiation dose (three radiation pulse mode). During this procedure, both the patient and physician are exposed to a certain degree of radiation so that its dose needs to be minimized. Constant measurement of radiation doses in patients and personnel is vital. Above all, the shielding in the room is particularly important. We use a suspended ceiling shield as well as a floor installed shield (**Figure 5**). During digital subtraction angiography (DSA) imaging, other comedical staffs are outside the angiosuite. The main operator besides the patient is protected by a ceiling-mounted radiation shielding glass. After the procedure, radiation exposure levels must be routinely recorded and archived.

Figure 5. Radiation shield. Operator uses the ceiling-mounted radiation shield and the assistant is behind the shield during contrast injection.

2. Imaging techniques

2.1. Sheath

2.1.1. 4Fr sheath

The 4Fr sheath is mainly used for the antegrade femoral approach. For initial access, a 4Fr sheath is placed from the common femoral artery (CFA) to the SFA. The reason is that an antegrade puncture is technically more demanding and if we fail to make the puncture, the sheath can be withdrawn or repositioned. While keeping the 4Fr sheath in the profunda femoris artery (PFA), we can even place an additional 4Fr sheath into the CFA. The long 4Fr sheath is for below the knee work. However, it is easily kinked and there may be an increased risk of hematoma formation. In interventions below the knee arteries, most occlusion balloons accept the 4Fr sheath with the use of a 0.014 or 0.018 in. guidewires. And to minimize sheath size in the ipsilateral CFA approach, a 4Fr long sheath is ideal for patients with critical limb ischemia (CLI) (**Figure 6A**).
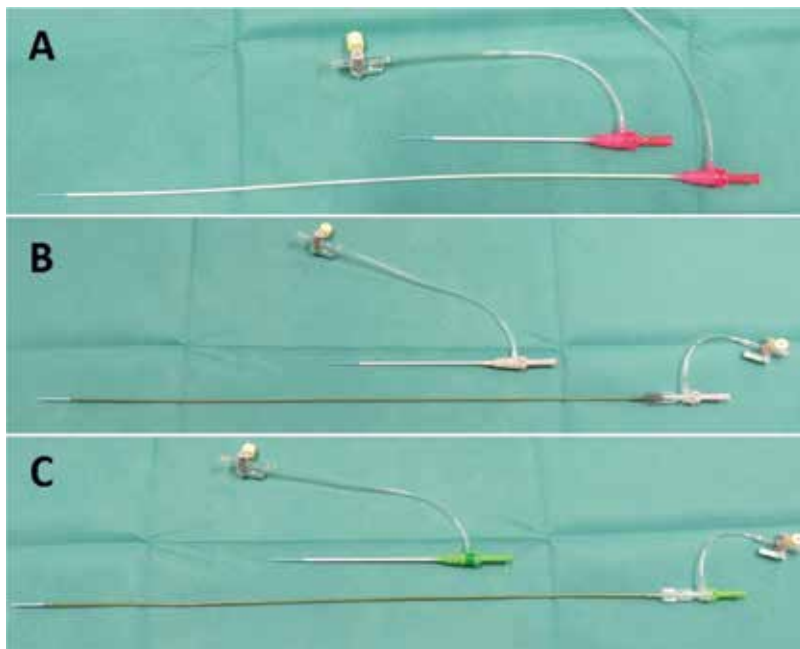

Figure 6. Sheath. (A) 4Fr sheath, (B) 5Fr sheath, (C) 6Fr sheath.

2.1.2. 5Fr sheath

In ad hoc interventions, we have standardized the 5Fr sheath for the initial retrograde CFA approach. When stent implantation is planned, we start with a 6Fr sheath. Either

a 4Fr or 5Fr pigtail catheter can be used for aortography. With a 4Fr pigtail catheter, the amount of dye is limited to around 10–13 cc/s. To opacity the terminal aorta to both the iliac and common femoral arteries, the rate of injection should be 15–20 cc/s and this flow rate can be achieved with at least a 5Fr pigtail catheter. Introducer sheaths are used for all angiography and endovascular procedures. The 5Fr 45 cm cross-over sheath is used for either the retrograde or antegrade approach. In a contralateral SFA intervention, a 5Fr 45 cm crossover sheath is used. However, when stenting is performed, the sheath should be replaced with a 6Fr crossover sheath. In the antegrade approach for BK interventions, a 5Fr 45 cm crossover sheath gives more back-up support to intervene on the tibial arteries (**Figure 6B**).

2.1.3. 6Fr sheath

When an iliac artery stent is already planned, a 6Fr short sheath should be placed in a retrograde manner. In a cross-over approach, a 6Fr 45 cm cross-over sheath is employed. The advantage of the 6Fr system is that the closure device can be applied after the procedure. In some medical centers, the antegrade 6Fr short sheath is placed for SFA stenting. However, we do not routinely use the 6Fr sheath for antegrade work (**Figure 6C**) (**Table 2**).

Advantage	Disadvantage
Better wire control with short wire	Complication related to antegrade common femoral artery (CFA) puncture
Short distance to the lesion	Might miss proximal superficial femoral artery (SFA) lesion
Precise stent placement	Need caution of proximal end of stent
Access to below the knee arteries	Compression of ischemic side after procedure

Table 2. Advantage and disadvantage of ipsilateral antegrade approach.

2.2. Wires

2.2.1. 0.035 in. wire

There are three types of tips for the 0.035 wire. We do not use a regular J-tip Radifocus wire (Terumo, Tokyo, Japan) (**Figure 7A**). The initial wire is always a 1.5 mm J-type Radifocus wire (Terumo, Tokyo, Japan) (**Figure 7B**). The tip of this wire has a 1.5 mm round shape and is quite safe when the wire migrates into the small branches or other vessels. Once the guidewire crosses the lesion, we change to a regular 0.035 in. spring wire (**Figure 7C**). The Radifocus wire is slippery and is difficult to keep in place while regular spring wires tend to stay in place. Thus, for stability, the wire should be changed to a spring wire once the lesion is crossed. These three types of 0.035 in. wires should always be at hand.
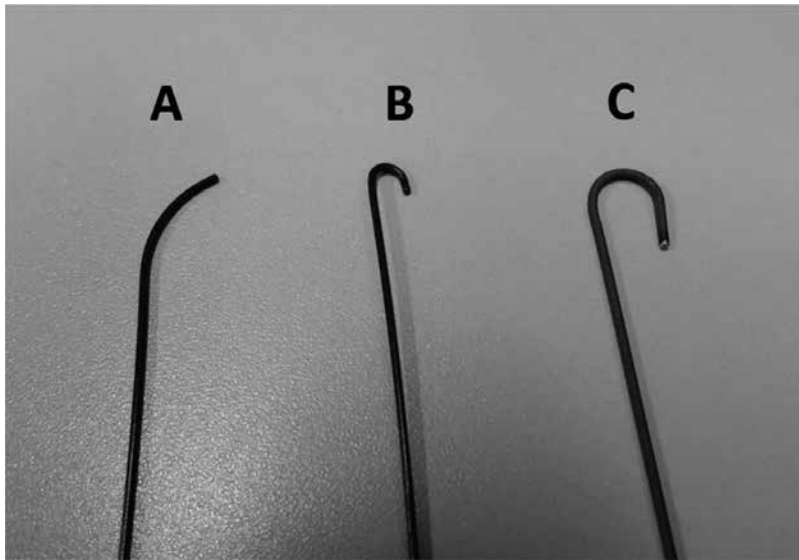
Figure 7. 0.035 in. guidewires. There are three types of tips for the 0.018 in. wire. We do not use regular a J-tip Radifocus wire (A). The initial wire is always a 1.5 mm J-type Radifocus wire (B). Once used to cross the lesion, it is exchanged to a 0.018 in. spring wire (C).

2.2.2. 0.018, 0.014 in. wires

Basically, we do not use the 0.018 in. wire as a regular wire. Chronic total occlusion (CTO), a 0.018 in. Treasure 12-g (Asahi Intec, Nagoya, Japan) wire is initially selected. It has a 12-g tip load and is best suited as a peripheral CTO wire. The V 18 (Boston Scientific, Cambridge, MA, USA) wire has a strong main shaft with a soft tip and can be used for cross-over ballooning or stent implantation. There are many 0.014 in. wires and their purposes vary. For below the knee artery work, the 0.014 in. wire is the basic wire used.

2.3. Digital subtraction angiography (DSA) vs. digital angiography (DA)

Digital subtraction angiography (DSA) has long been the gold standard for evaluation of atherosclerotic lesions in patients with PAD. Image quality has been further improved by replacing traditional image intensifiers with flat panel detectors so that regular digital angiography (DA) is now replacing DSA. When considering the high radiation doses, not all cases need imaging by DSA. Above all, critical limb ischemia is difficult to manage and some patients have difficulty staying still during injection of the contrast dye. Thus, adequate optimization with either DSA or DA should be employed to obtain accurate imaging of the diseased segments.

2.3.1. Aortoiliac artery

A typical DSA image of the iliac artery is shown in **Figure 8**. In the 30 cm image, we can see from the terminal aorta to both common femoral arteries (**Figure 8A**). In the 20 cm image, a clearer view can be seen (**Figure 8B**).

In our routine, we first take a 30 cm image by DSA (**Figure 9A**). Next, we take a 20 cm image by DA for the purpose of intervention (**Figure 9B**).
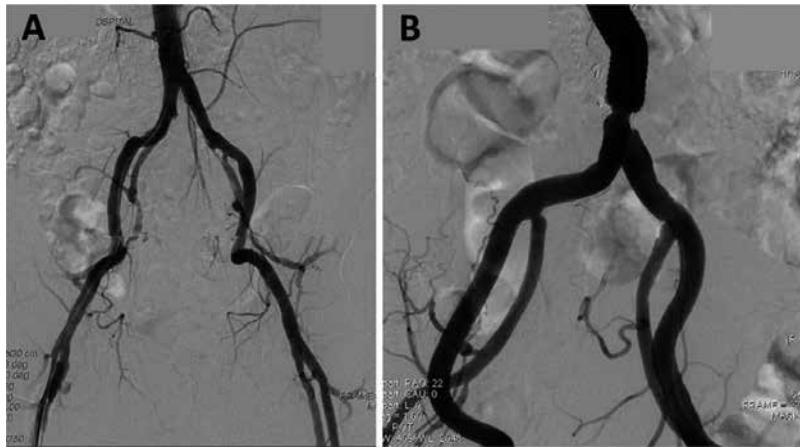

Figure 8. Iliac artery angiography, 30 cm vs. 20 cm image. (A) 30 cm image, we could see from the terminal aorta to both common femoral arteries. (B) 20 cm image, a clearer view is obtained.

Figure 9. DSA vs. DA image of the iliac artery. (A) 30 cm image by DSA for diagnostic purposes. (B) 20 cm image by DA for interventions. DSA, digital subtraction angiography; DA, digital angiography.

DA is more practical for stent implantation since it provides the background image. In the aortoiliac artery segment, the image is hampered by bowel and gas movements. Aortoiliac artery angiography is basically taken by DSA, however, due to bowel and gas movements, the image is blurred (**Figure 10A**). In such a circumstance, we change to the DA image (**Figure 10B**). In **Figure 10**, a left common iliac aneurysm with distal stenosis can be seen; the DSA image is blurred while the DA image clearly reveals stenosis.

Figure 10. DSA vs. DA image of the iliac artery. (A) DSA image is blurred by bowel gas. (B) DA image shows clear image of left common iliac aneurysm with distal stenosis. DSA, digital subtraction angiography; DA, digital angiography.

2.3.2. Femoropopliteal artery

The initial angiographic image is the ipsilateral angled view. Either DSA or DA can provide a reasonable image (**Figure 11**), although the DSA image (**Figure 11A**) is shown to be better than the DA image (**Figure 11B**). In the DA image, the background is shown and can be used as reference (**Figure 11B**).
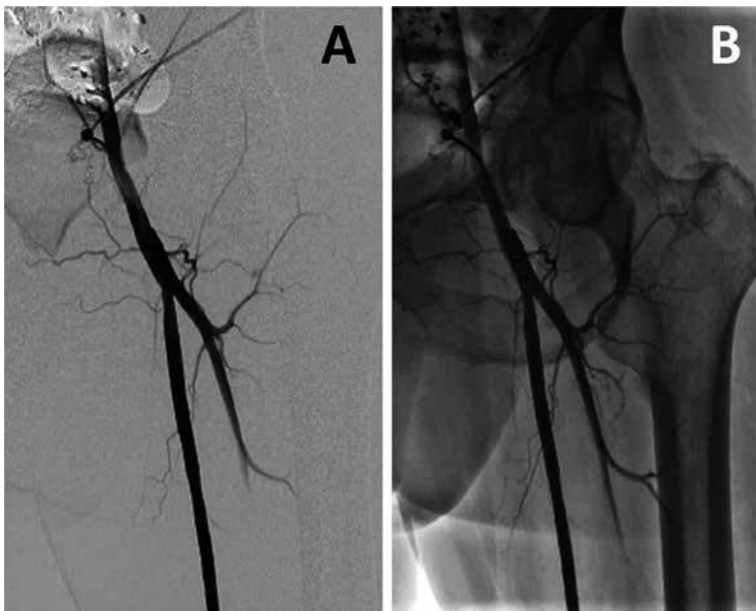

Figure 11. Proximal femoral artery. (A) DSA image for ipsilateral angled view of the left proximal femoral artery. (B) DA image shows the background and identifies bifurcation point.

A calcified lesion is often seen in the common femoral artery. In such cases, DSA provides a clearer view than the DA image (**Figure 12**). DSA clearly shows the calcified lesion (**Figure 12A**) while, in contrast, the lesion could not be determined in the DA image due to low contrast (**Figure 12B**).

For the SFA, we use either the DSA or DA image. For a calcified lesion, DSA is preferable (**Figure 13A**), but in most cases, DA provides a reasonably good image (**Figure 13B**).
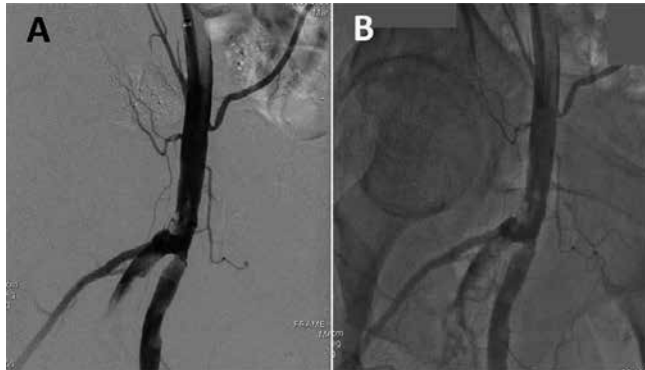
Figure 12. Calcified common femoral artery. (A) DSA clearly shows calcified lesion. (B) DA image could not determine lesion due to low contrast.

Figure 13. SFA angiography, DSA vs. DA. (A) DSA shows clearer image and branches are well seen. (B) DA gives reasonably good image.

In a SFA lesion, measurement of the lesion length is important to decide the interventional strategy and we prefer a DA image for the pre-interventional angiogram. The popliteal artery is located deep in the posterior fossa of the knee joint. Surrounded by a bony structure, the popliteal artery is very difficult to visualize by DA. Basically, a DSA image is taken for the popliteal artery (**Figure 14A**). In **Figure 14A**, tight stenosis of the mid-popliteal artery is well visualized with rich collateral circulation. In the DA view, stenosis is well observed but most of the collateral vessels are not visualized (**Figure 14B**).
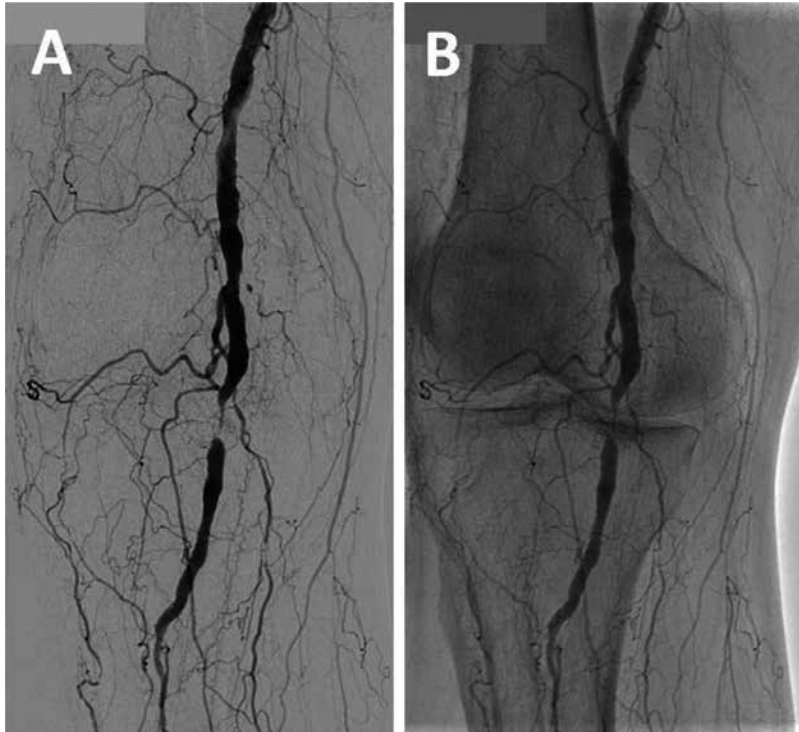

Figure 14. Popliteal artery angiography, DSA vs. DA. (A) Popliteal artery surrounded by bone and basically taken with DSA. (B) DA view shows well visualized stenosis but most collateral vessels unclear.

2.3.3. Below the knee arteries

Diseases of below the knee arteries are closely associated with critical limb ischemia (CLI) and detailed anatomical information is required to plan intervention. Compared to other limb arteries, angiography remains the imaging method of choice in most cases of CLI. How to take a good image is the cornerstone of successful endovascular therapy. DSA is a must for imaging of below the knee arteries. In **Figures 2–10**, comparisons of the DSA and DA images of the left proximal tibial arteries are shown. In the DSA image, posterior tibial artery occlusion is well observed (**Figure 15A**). On the other hand, the DA image failed to show the tibioperoneal trunk and occlusion of the posterior tibial artery (**Figure 15B**).

Figure 15. Proximal below the knee angiography, DSA (A) vs. DA (B).

A similar case of the left proximal below the knee artery is shown in **Figure 16**. In the DSA image, three tibial arteries are shown with multiple stenosis (**Figure 16A**). In the DA image, precise diagnosis cannot be made (**Figure 16B**).

Assessment of the distal tibial arteries is vital in evaluating below the ankle disease. Continuation from the anterior tibial artery to the dorsal artery and the posterior tibial artery to the planted artery must be clarified. However, due to the bony structure, the DA image could not show these distal tibial and below the ankle arteries (**Figure 17**). In **Figure 17A**, the planter artery is not clearly visualized in the DSA image. In the DA image, most of the vessels remain un-visualized (**Figure 17B**).

2.4. Basic angiography for PAD

2.4.1. Angiography from the terminal aorta to below the knee artery

In an angiographic approach for PAD diagnosis, we need to assess three segments of the lower limb artery, that is, the aortoiliac, femoropopliteal and below the knee arteries. In **Figure 18**, the basic angiography is shown. First, angiography of the aortoiliac artery was taken (**Figure 18A**). The second angiography is an ipsilateral view of the proximal femoral artery (**Figure 18B**). In the right leg, a 30° right anterior oblique (RAO) view was chosen to separate the proximal SFA and PFA. Third, angiography from the CFA to the distal below the

ankle artery was taken by running the table (**Figure 18C**). After observing these three angiograms, we could assess in which segment stenosis or occlusion was located.

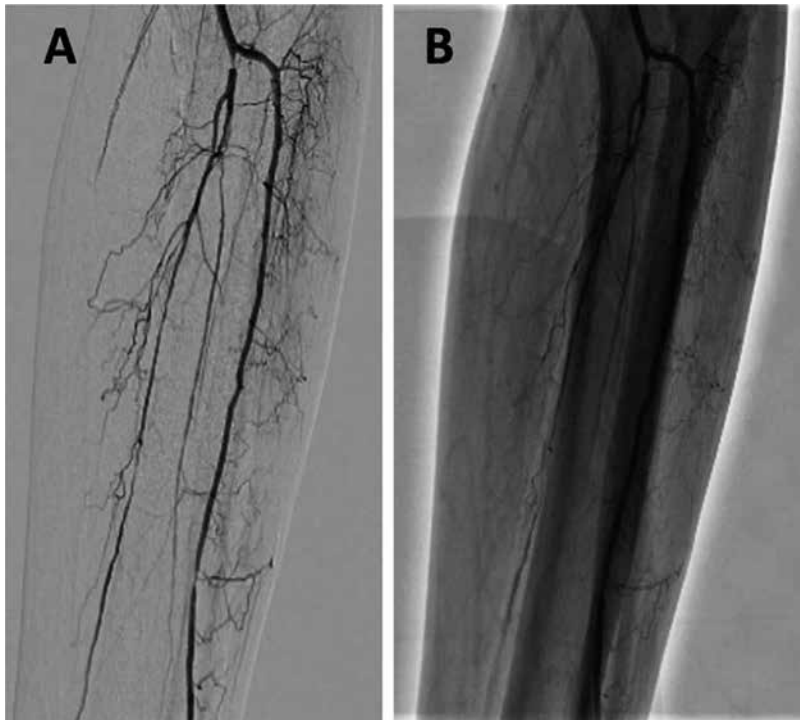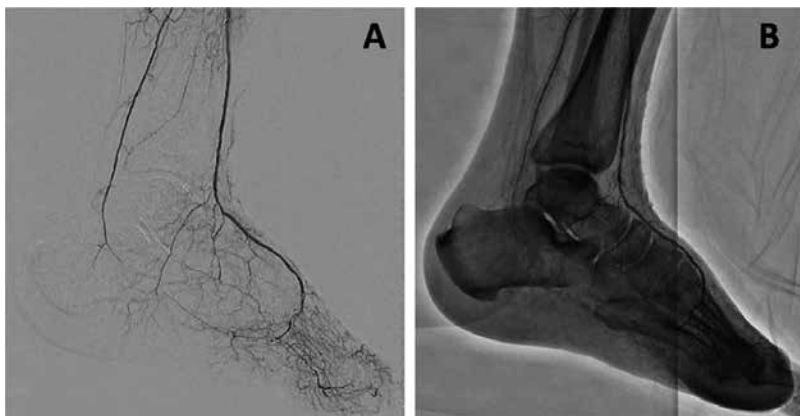**Figure 16.** Mid-below the knee angiography, DSA (A) vs. DA (B).

Figure 17. Below the ankle angiography, DSA (A) vs. DA (B).

A typical claudication with SFA disease is shown in **Figure 19**. Aortoiliac artery angiography showed no significant stenosis (**Figure 19A**). In the proximal femoral artery, there was no stenosis in the SFA and PFA (**Figure 19B**). Left limb angiography showed focal stenosis in the

mid-SFA while the left anterior tibial artery was not visualized (**Figure 19C**). By using DA, left SFA angiography was taken and revealed focal tight stenosis in the mid-SFA (**Figure 19D**). This DA image was used as reference in interventional work (**Figure 19D**).
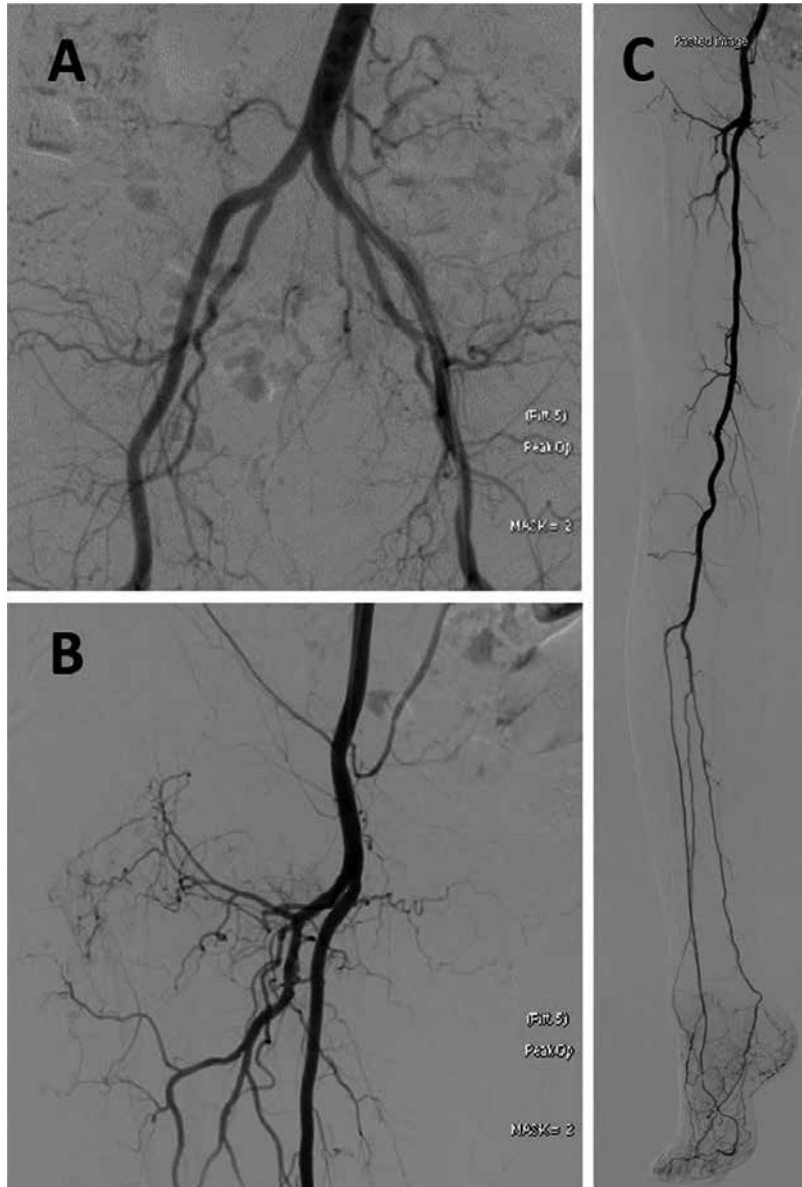
Figure 18. Basic lower limb artery angiography. (A) Aortoiliac artery angiography. (B) Proximal femoral artery by a 30° right anterior oblique view. (C) From the right common femoral artery to the distal below the ankle artery.

Figure 20 shows isolated below the knee artery disease. From the iliac to femoropopliteal artery level, no atherosclerotic changes could be observed (**Figure 20A** and 20B). A lesion is located in the right below the knee arteries. Below the knee arteries showed a stenotic lesion

of the anterior tibial artery, and the posterior tibial artery and peroneal artery are occluded (**Figure 20C**). This type of lesion, that is, "isolated below the knee artery disease" is often found in patients with critical limb ischemia.

Figure 19. A typical claudication with SFA disease (A) Aortoiliac artery angiography showed no significant stenosis. (B) In 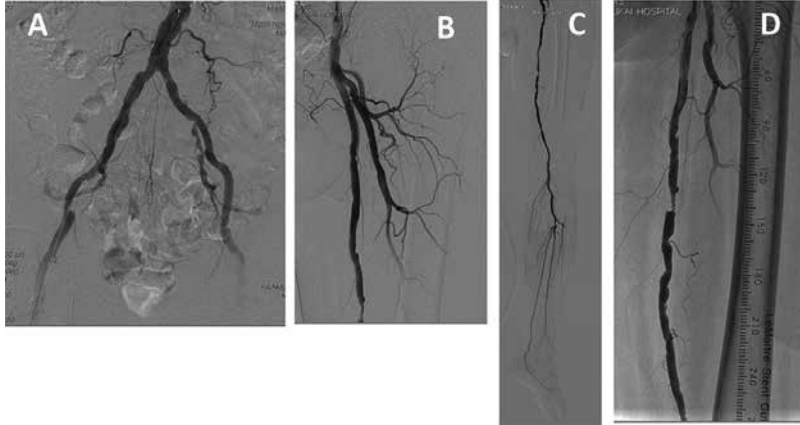the proximal femoral artery, there was nostenosis in the SFA and PFA. C: Left limb angiography showed focal stenosis in themid-SFA. (D) LeftSFA angiography revealed focal tight stenosis in the mid-SFA.

2.4.2. Magnification of images

The image size has two purposes: one is to see the whole vessel, for example, in the aortoiliac artery, visualization from the terminal aorta to both the right and left CFA (**Figure 21A**). The other is to better intervene on the target lesion utilizing appropriate magnification of the image size (**Figure 21B**). For wiring to this lesion, a 20 cm magnified image was taken and successful wiring was carried out using a 0.014 in. wire (**Figure 21B**).

In **Figure 22**, right SFA stent restenosis was visualized with a 30 cm image (**Figure 22A**). Moreover, using the 20 cm magnified mode, stent restenosis was well observed (**Figure 22B**).

In below the knee arteries, the whole image shows which vessels are diseased (**Figure 23A**). However, this running image does not give detailed information on the three tibial arteries. In the 30 cm image, the three proximal tibial arteries are well observed, and the peroneal and posterior tibial arteries are diffusely diseased (**Figure 23B**). The further magnified 20 cm image revealed that there is tight stenosis at the ostium of the right anterior tibial artery (**Figure 23C**).

2.4.3. Pre- and postinterventional image

Basically, all interventional work requires two images to be taken, that is, pre- and postintervention. These two images reveal the angiographic changes pre and post procedure. In **Figure 24A**, the femorofemoral bypass was occluded and a long total occlusion of the right iliac artery is seen. After successful recanalization and stenting, angiography of the exact same iliac artery was taken (**Figure 24B**).
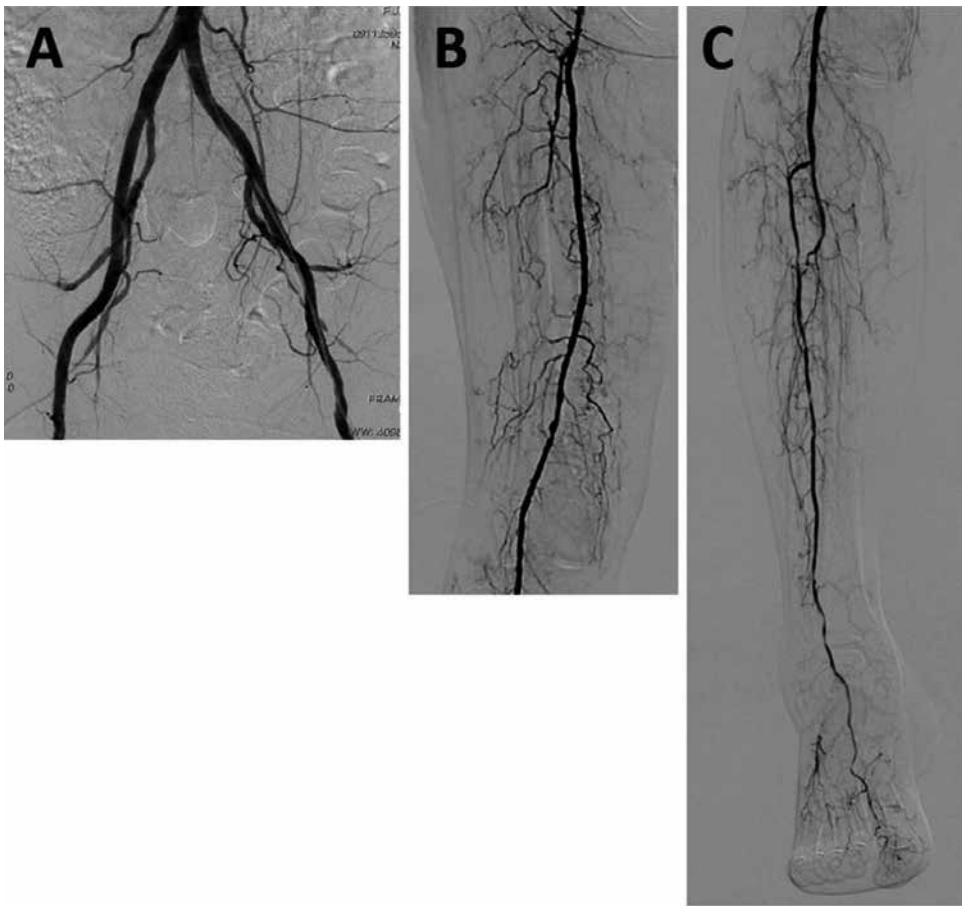
Figure 20. Basic lower limb artery angiography. Isolated below the knee artery disease. (A) Aortoiliac artery showed no disease. (B) Right femoropopliteal artery showed no disease. (C) Right below the knee arteries showed a stenotic lesion of the anterior tibial artery, and the posterior tibial artery and peroneal artery are occluded.
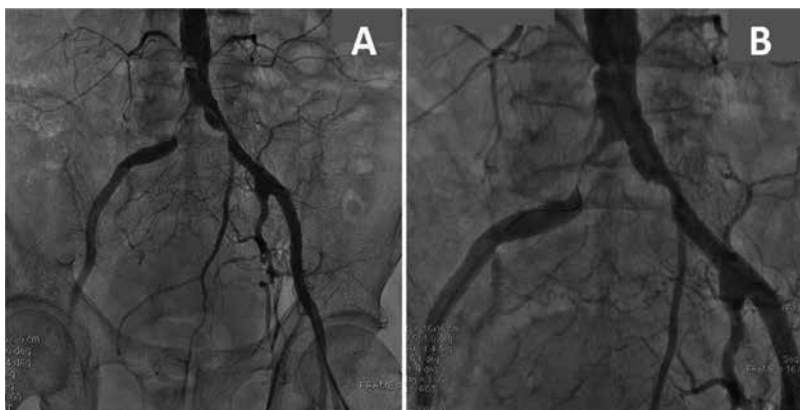
Figure 21. Iliac artery angiography, 30 cm (A) vs. 20 cm image (B).
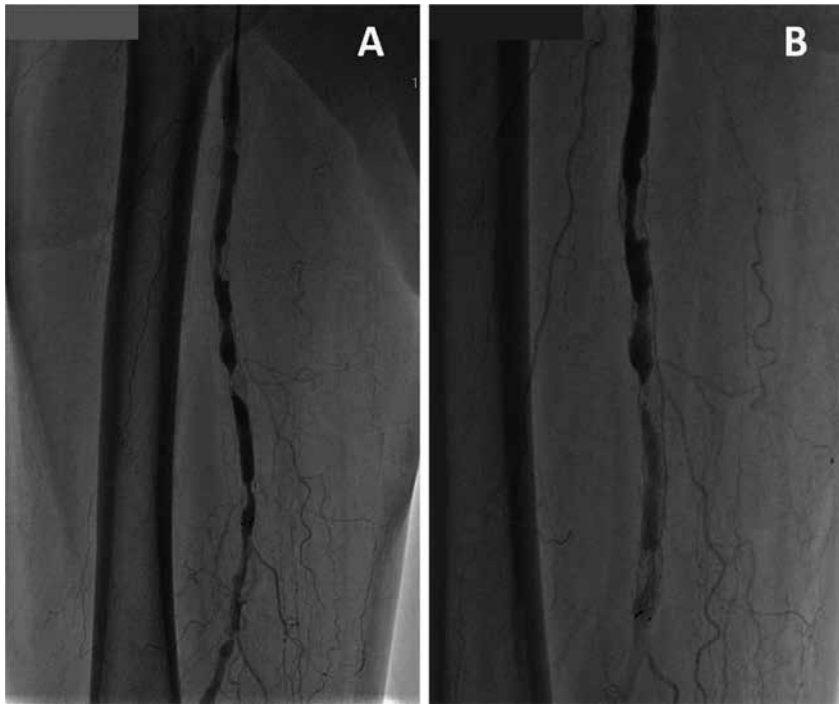
Figure 22. SFA angiography, 30 cm (A) vs. 20 cm image (B).
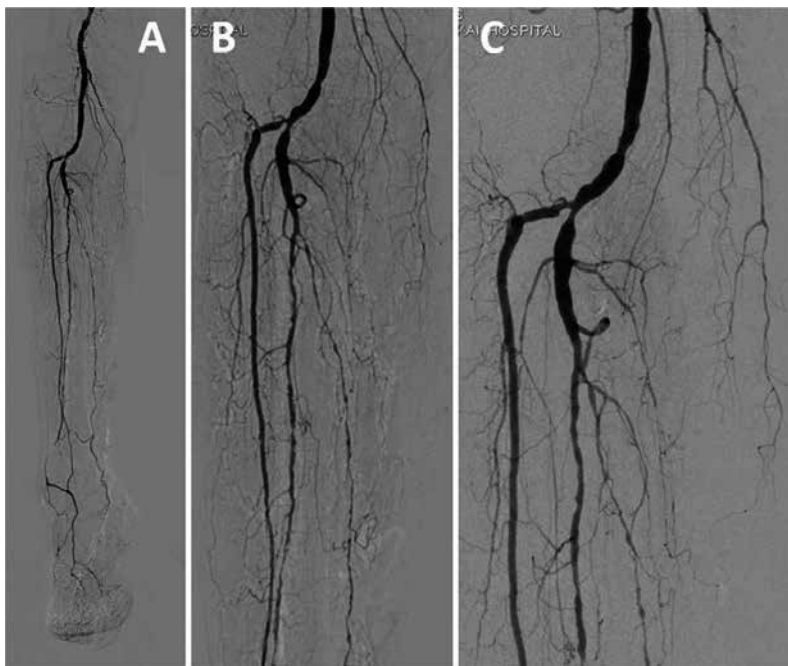
Figure 23. Below the knee angiography, 30 cm (A), 20 cm (B) and 16 cm images (C).
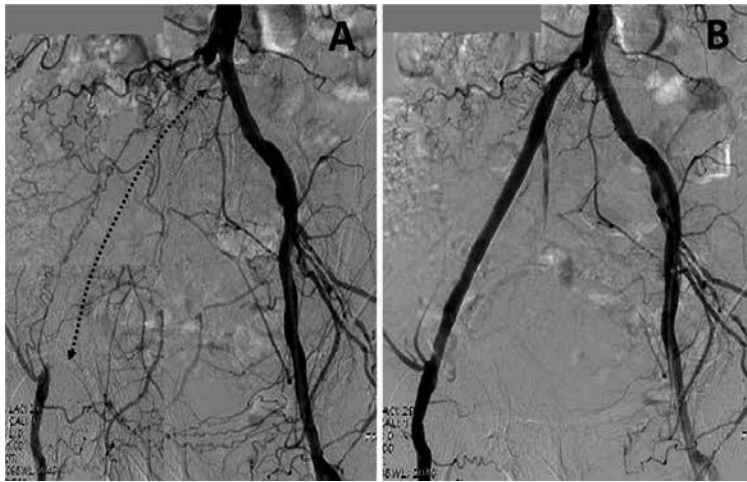
Figure 24. A case of right iliac artery occlusion. Pre (A) and post iliac artery angiography (B).

In **Figures 2–20**, typical left SFA occlusion was seen (**Figure 25A**). After balloon angioplasty, dissection and incomplete dilatation were observed (**Figure 25B**). Post stent angiography showed excellent dilatation of the left SFA lesion (**Figure 25C**). During the procedure, the table was frequently moved and oftentimes, post angiographic images were not taken in the different positions, giving a false impression of the postinterventional image.
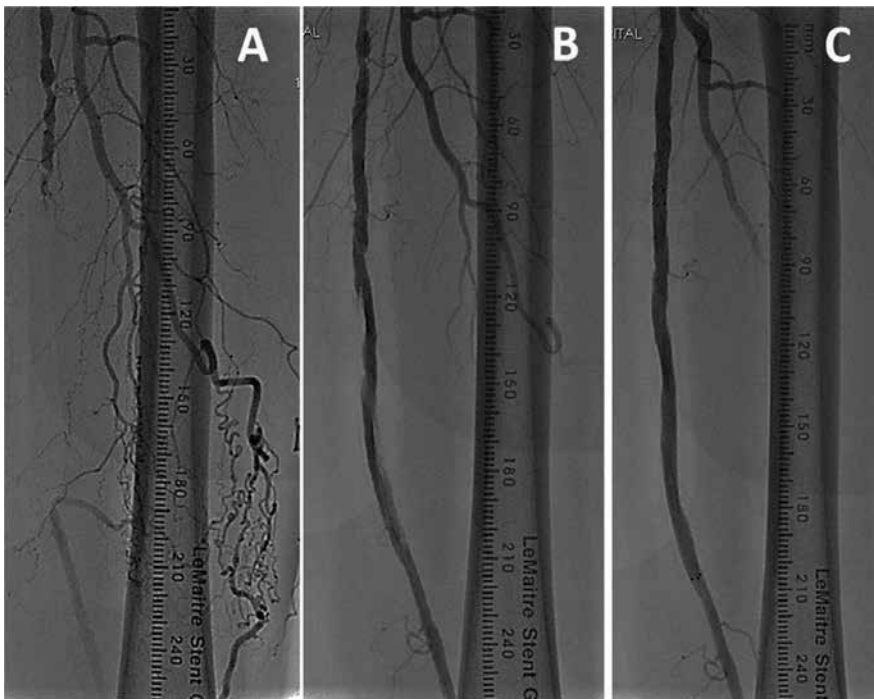
Figure 25. A case of SFA occlusion. Pre (A) and post SFA angiography (B).

3. Echo-guided puncture femoral artery puncture

The common femoral artery (CFA) remains the most widely accepted site for endovascular artery access. Vascular access site-related complications are a major cause of periprocedural morbidity among patients undergoing percutaneous endovascular intervention. In particular, patients with PAD may be more likely to have atherosclerosis affecting the CFA. Ultrasound guidance is an emerging trend for all percutaneous procedures and its use for femoral artery puncture has decreased vascular complications and improved first-pass success rates [4–6].

3.1. Retrograde common femoral artery puncture

The CFA is the main access site for angiography and interventional procedures. Among the various puncture sites, the retrograde CFA puncture is the most commonly employed and the basis of arterial punctures. We have described a safe and echo-guided technique for avoiding femoral access site complications.

3.1.1. Puncture point

The inferior border and upper border of the femoral head should be realized by fluoroscopy (**Figure 26A**). After checking the maximum arterial pulse (**Figure 26B**), Xylocaine is given 1 cm below the middle of the femoral head (**Figure 26C**).

Figure 26. Puncture point of common femoral artery. (A) Realizing the inferior border and upper border of the femoral head by fluoroscopy. (B) Marking middle of femoral head. (C) Xylocaine to be given 1–2 cm below.

3.1.2. Preparation

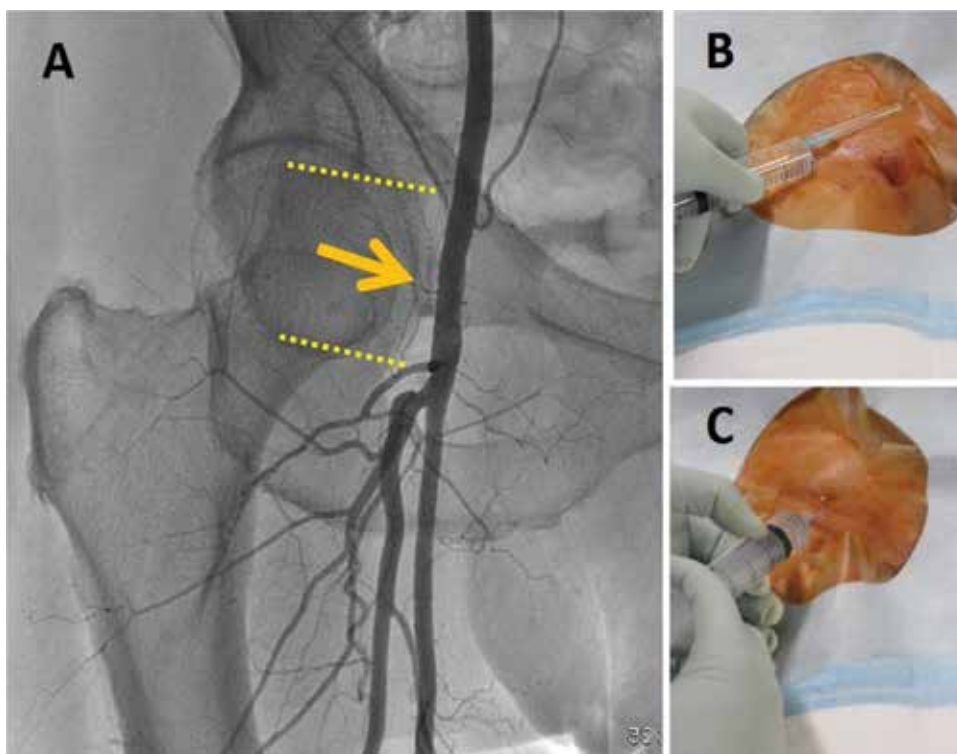A sheath and two types of wires were prepared. Once a puncture is performed, the wire should be ready to be inserted and if there is resistance, change to a different kind of wire is advised (**Figure 27**).
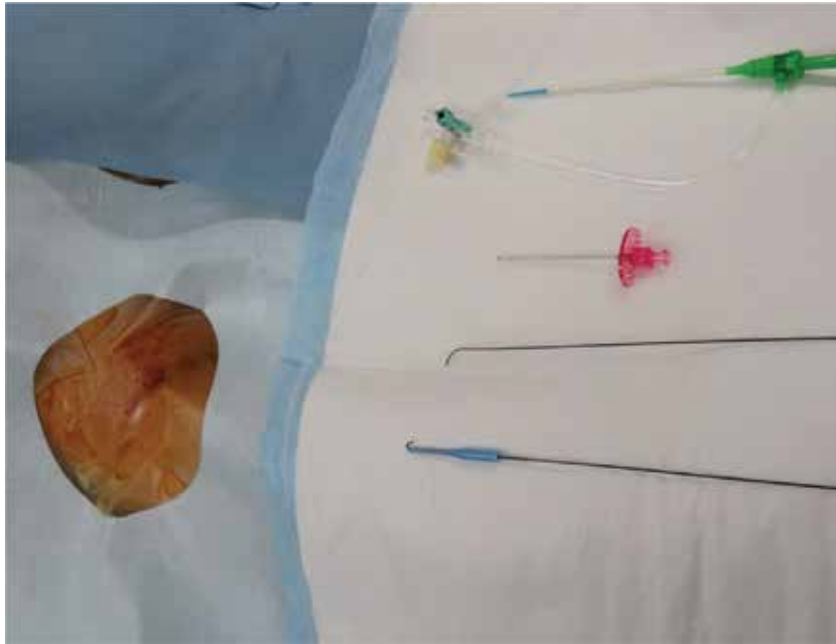
Figure 27. Preparation of sheath and two types of wire. Prepare sheath and two type wires close to puncture site. Once puncture completed, insert wire and if resistance encountered, change to different shape wire.

3.1.3. Echo scanning

For an echo-guided puncture (NEMIO MS, Toshiba, Tochigi, Japan), first, echo scanning was carried out from the upper CFA to proximal SFA (**Figure 28**). We could identify where the bifurcation is located. Either a long axis (**Figure 28A**) or short axis can be obtained (**Figure 28B** and **C**). A scan is basically made by a short-axis view. The ideal puncture site of the CFA can then be located (**Figure 28B**) and the bifurcation site can be identified (**Figure 28C**).

An echo image is best seen from the upper common femoral artery to the distal external iliac artery. When total reliance is on echo guidance, the puncture site locates higher than the middle femoral head. To avoid too low or high punctures, rechecking the puncture site by fluoroscopy is advised (**Figure 26A**).

3.1.4. Puncture

Arterial access was obtained with an 18-G needle (COOK Medical, Bloomington, Indiana) using the modified Seldinger technique. The needle was inserted at an angle of about 45° from the skin at a level just below the center of the femoral head. In viewing the short axis, the aim

should be for the top of the vessel. During flash backs of blood, a gentle wire insertion must be made. When resistance is felt, change from a straight wire to round shaped wire is advised (**Figure 29**). When the plaque in the CFA is found, a normal CFA puncture site should be located. In **Figure 29A**, the long-axis view showed the plaque in the CFA.
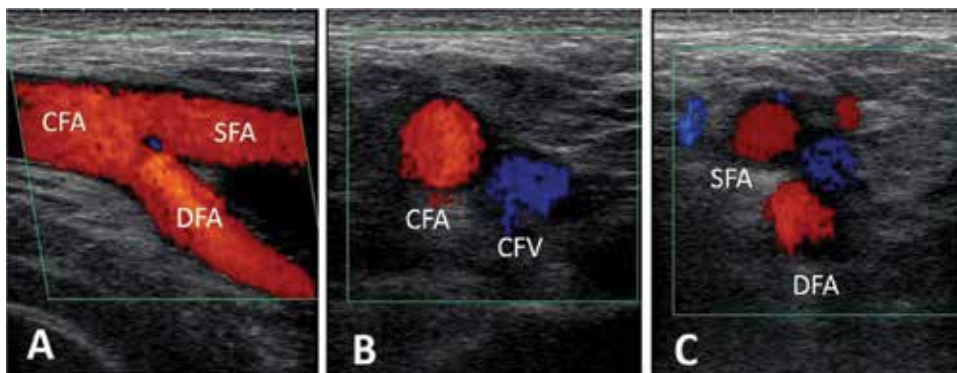
Figure 28. Color Doppler scanning from CFA to SFA and PFA. (A) Long-axis view of CFA and SFA. (B) Short-axis view of CFA. (C) Short-axis view of SFA and PFA. Using color Doppler, scan from upper CFA to SFA and PFA. Locate the ideal puncture site of CFA and identify the bifurcation point. CFA, common femoral artery; SFA, superficial femoral artery; PFA, profunda femoral artery.
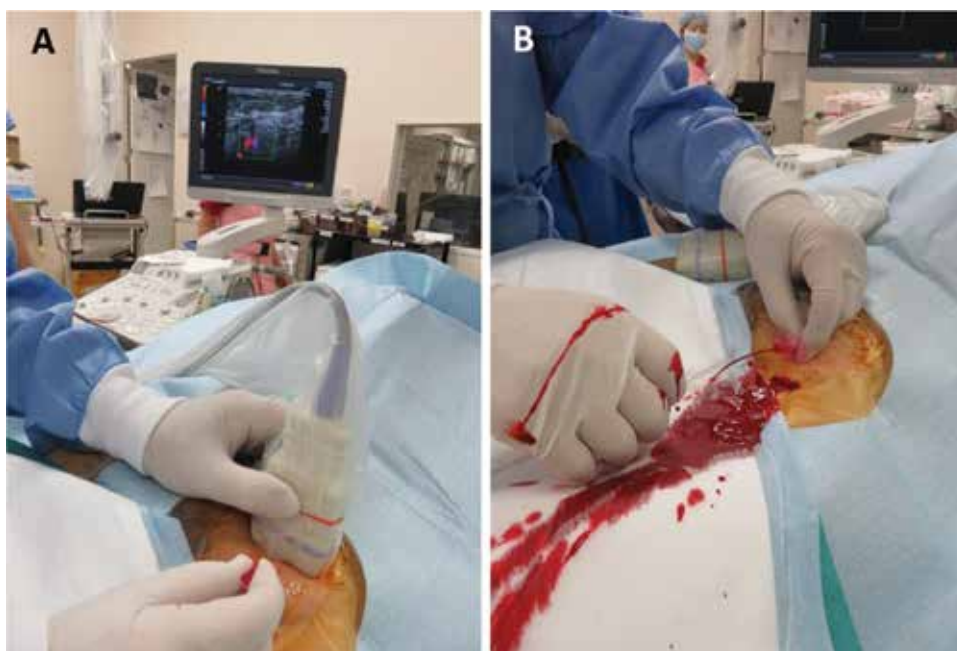
Figure 29. Puncture. (A) Echo guidance. (B) 18G needle puncture. In viewing short axis, aim for top of the vessel. During flash-back of blood, gentle wire insertion should be made. If resistance encountered, change straight wire to round shaped wire.

In this situation, a plaque free zone within the CFA should be located (**Figure 30B–D**).

In **Figure 31**, a puncture was made at the site of CFA disease and the wire went into false lumen, resulting in the total occlusion of the CFA.
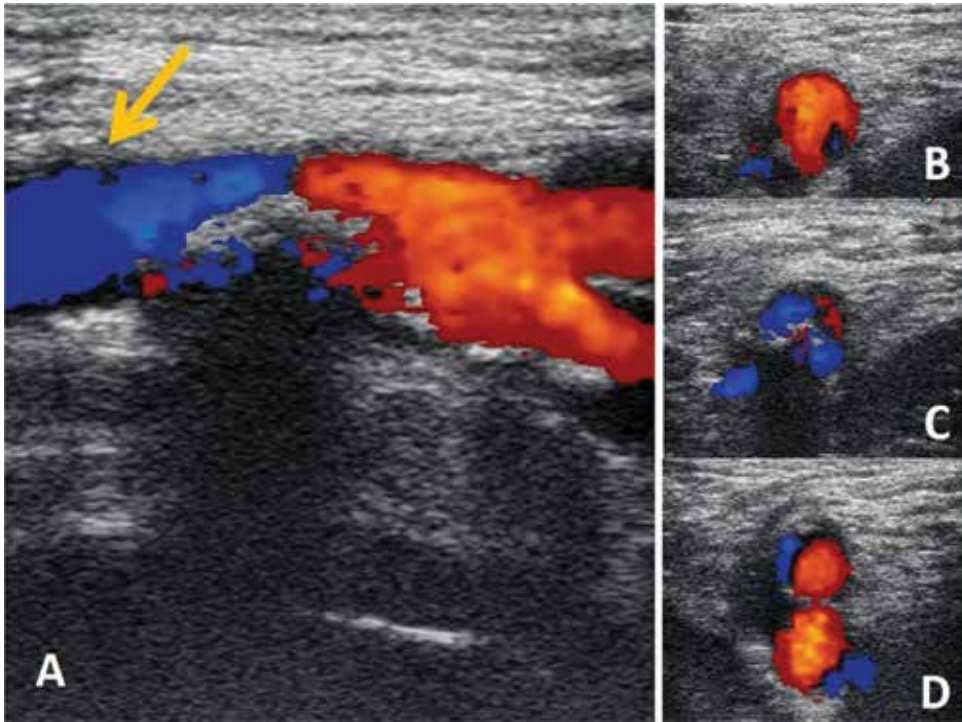
Figure 30. Presence of CFA plaque. (A) Long-axis view of CFA and SFA. Note CFA plaque. (B) Plaque free site of CFA in short axis. (C) Presence of plaque. Should not be punctured. (D) SFA and PFA level. When finding plaque in the CFA (A), should look for normal CFA puncture site. Must find plaque free zone within CFA. CFA, common femoral artery; SFA, superficial femoral artery; PFA, profunda femoral artery.
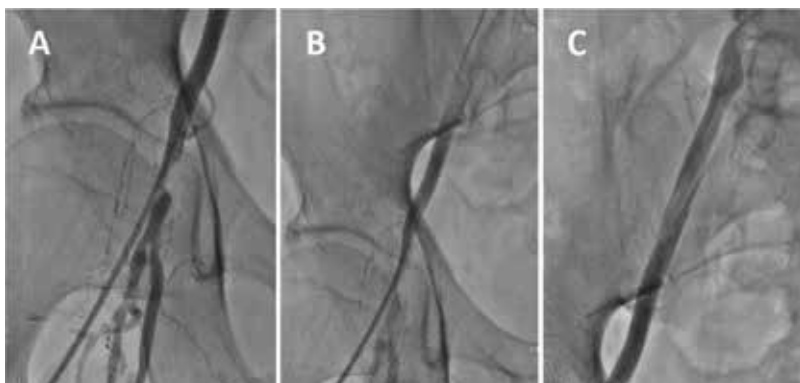
Figure 31. Puncture of common femoral artery plaque. (A) Puncture into CFA plaque. Creates false lumen. (B) TIMI 0 flow. (C) Dissection of iliac artery. Without knowledge of CFA disease, puncture was made. Wire went into false lumen and ended up in total occlusion of CFA.

3.2. Antegrade common femoral artery puncture

For treatment of femoropopliteal artery disease, the standard approach has been to access the contralateral common femoral artery (CFA). However, an ipsilateral, antegrade CFA approach has certain advantages. Compared to the contralateral approach, access to the lesion distance is short which in turn improves the responsiveness of the wire handling used to perform the intervention. In other clinical situations such as post aorto-bi-femoral surgical bypass, deployment of iliac kissing stents, post stent grafting and for aortoiliac occlusive disease, an antegrade approach is the method of choice to reach the lesion. The advantages and disadvantages are shown in **Table 2**.

The CFA is approximately 4–5 cm in length and arises from the external iliac artery (EIA) as it passes below the inguinal ligament. It then bifurcates into the PFA and SFA. An anatomical knowledge of the level of origin for the PFA is important in avoiding retroperitoneal bleeding, iatrogenic femoral arterial-venous fistula and/or formation of a pseudo aneurysm. The most lethal complication of femoral access remains retroperitoneal hemorrhaging due to a high puncture. Thus, the best first step toward reducing the incidence of retroperitoneal bleeding is to prevent high punctures.

3.2.1. Preparation of an antegrade puncture

As we perform in a retrograde puncture, two kinds of wires should be at hand. The initial sheath we place is always the 4Fr size sheath (**Figure 32**).

The main reason is, when obtaining access to the CFA fails, the sheath can be easily withdrawn or left in the PFA. Once placing the sheath in the SFA is successful, it can be changed to any sheath as desired. Pointing to the middle of the femoral head, local xylocaine should be given around the inguinal ligamentum (**Figure 33**).

Echo was applied in the same way. However, the proximal CFA to external iliac artery is well observed by echo and may result in a very high puncture site. Under fluoroscopic guidance with echo assistance, point to the middle of the CFA.

3.2.2. Puncture

A puncture should be made by aiming an imaginary line over the center of the femoral head. The maximum level of bifurcation should be at or below the inferior border of the femoral head (**Figure 34A**). In about 1/4th of cases, bifurcation locates in the CFA (**Figure 34B**). In **Figure 34B**, the bifurcation point is in the middle of the CFA and there is a short margin for the antegrade puncture site.

3.2.3. Two-wire technique

Even when the puncture site is above the bifurcation, the wire may go to the PFA. In this situation, we use a two-wire technique (**Figure 35**). If the wire goes to the PFA, the first step is to place a 4Fr sheath into the PFA. Two short 0.025 in. wires are inserted into the RFA (**Figure 35A**). Withdrawing the sheath, one 0.025 in. wire should be manipulated into the SFA

(**Figure 35B**). Once the SFA is accessed, leaving one wire in the PFA, the other wire should be advanced to the SFA (**Figure 35C**). After confirming the wire in the SFA, the other PFA wire is withdrawn and a 4Fr sheath should be placed into the SFA (**Figure 35D**). If the sheath comes out, it can be repositioned back into the PFA by a 0.025 in. wire.

Figure 32. Preparation of antegrade puncture. 18G needle, 4Fr sheath and two kinds of wire at hand.
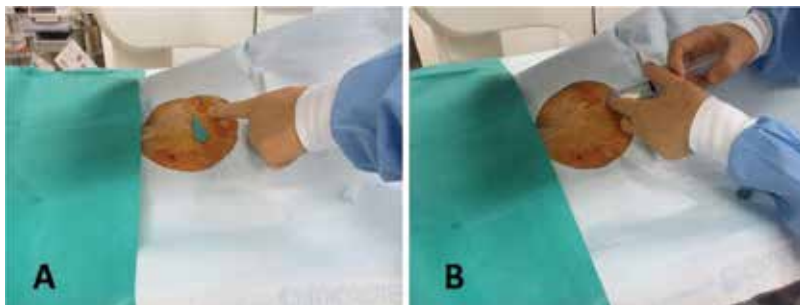

Figure 33. Antegrade puncture of common femoral artery. Puncture site. (A) Locate middle of femora; head. (B) Local xylocaine to be given at inguinal ligamentum.

Figure 34. Bifurcation point of CFA to SFA. (A) Level of the bifurcation is below the inferior border of the femoral head. (B) The bifurcation point is in the middle of CFA and only short margin for ideal puncture sire. CFA, common femoral artery.
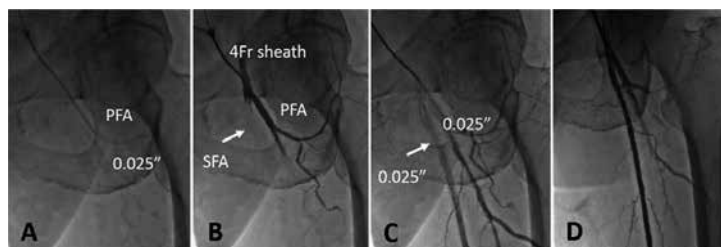
Figure 35. Two wire technique. (A) If wire goes to PFA, the first step is to place a 4Fr sheath into PFA. Two short 0.025 in. wires are inserted into PFA. (B) Withdrawing sheath, one 0.025 in. wire to find SFA leaving another wire in PFA (**Figure 10B**). (C) Once SFA obtained, wire advanced to SFA (**Figure 10C**). (D) After confirming wire in SFA, other PFA wire withdrawn and 4Fr sheath placed into SFA. SFA, superficial femoral artery; PFA, profunda femoral artery.

3.2.4. High bifurcation case

After surveying the CFA by echo, we may find high bifurcation of the SFA and PFA. In these cases, high puncture carries the risk of retroperitoneal bleeding. The puncture point should be in the range of the femoral head. In this situation, puncturing the SFA is one option. In **Figure 36**, there is high bifurcation and a CFA puncture is almost impossible. In this case, we decided to puncture the proximal SFA.

3.2.5. Sheath kinking

The angle of puncture should be more than 60° and almost vertical. After sheath insertion, care to avoid sheath kinking is advised. Once a hematoma is observed with sheath kinking, change to a larger size anti-kink sheath is necessary. In **Figure 37**, the initial 4Fr sheath was kinked (**Figure 37A**) and hematoma formation was detected. After the 4Fr sheath was replaced with a 6Fr sheath, the hematoma was stabilized (**Figure 37B**).

Figure 36. SFA puncture in high bifurcation case. After surveying CFA by echo, observed high bifurcation of SFA and PFA. In this situation, puncturing SFA is one option.

Figure 37. Sheath kinking during antegrade puncture. (A) Angle of puncture is more than 60° and almost vertical. After inserting sheath, observed sheath kinking. (B) 4Fr sheath replaced with 6Fr sheath and hematoma stabilized.

4. CO_2 angiography

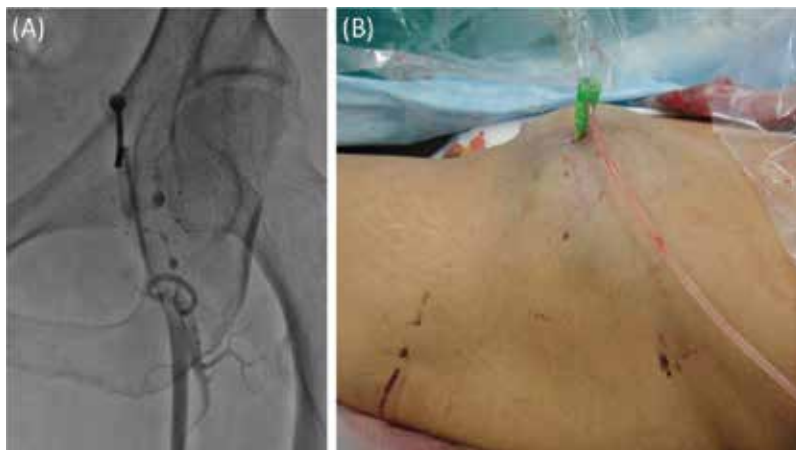The number of patients with chronic kidney disease (CKD) complicated with PAD is significantly increasing. In these patients, iodinated contrast may enhance the risk of contrast-induced nephropathy (CIN). CIN is an acute renal injury and may lead to irreversible loss of renal function. Carbon dioxide (CO_2) gas angiography is indicated for those with renal insufficiency and high-risk patients who are allergic to iodinated contrast material [7]. CO_2 is imaged using digital subtraction equipment with a CO_2 software program. Modern DSA equipment has a software program that allows integration of multiple images into a single composite image.

4.1. CO_2 delivery system

The system consists of a medical grade CO_2 gas cylinder with a regulator, a disposable sterile plastic tube with a bacteria-removal filter, and a 50-ml delivery syringe (**Figure 38**).
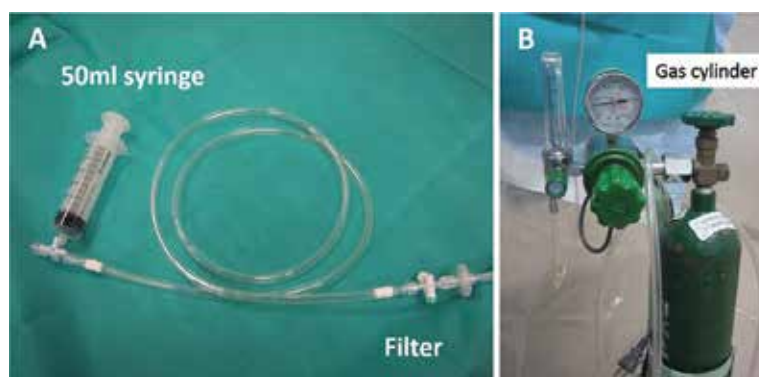
Figure 38. CO_2 delivery system. (A) A disposable, sterile plastic tube with a bacteria-removal filter and a 50-ml delivery syringe; (B) a medical grade CO_2 gas cylinder with a regulator.

Collection of CO_2 to the syringe and injection system should be separated to avoid erroneous gas injection to an artery (**Figure 39**).

The gas should be purged three to four times during collection to prevent room air contamination from the tube and delivery syringe in the circuit and then filled with gas at a stationary flow of 2 l/min. About 40 cc of aspirated gas was filled into the delivery syringe and 30–40 cm^3 of CO_2 gas was manually injected into the vessel leaving about 5 cm^3 in the injection syringe (**Figure 40**).

After gas injection, the remaining gas and blood were carefully aspirated into the syringe. Gas injections were spaced at least 30 s apart. Although we do not have experience in mechanical injection, manual injection is sufficient to inject 30–40 cc of CO_2. However, the safety of injecting large amounts of CO_2 is not guaranteed [8]. If a patient complains of abdominal pain, further CO_2 injection should be avoided. And if the angiogram shows a slow flow, further CO_2 delivery by syringe should also be stopped (**Table 3**).

Figure 39. Separate system between CO_2 suction and injection. Collection of CO_2 to syringe (left) and injection system (right) should be separated to avoid erroneous gas injection to artery.

Figure 40. Infusion and injection of CO_2 gas by 50 cc syringe. (A) Gas was purged 3–4 times during collection to exclude room air contamination from the tube and delivery syringe in circuit. Filled with gas at a stationary flow of 2 l/min. (B) 40 cc of aspirated gas filled into delivery syringe, 30–40 cm^3 of CO_2 gas manually injected into the vessel, leaving about 5 cm^3 in the injection syringe.

4.2. Iliac artery angiography by CO_2

The iliac artery is a large sized vessel and its inflow is the larger abdominal aorta. Moreover, there are two internal iliac arteries and two femoral arteries. CO_2 angiography requires displacement of all or most of the blood to achieve adequate images. Due to such anatomical reasons, the iliac artery is not well suited for CO_2 angiography. In **Figure 41A**, CO_2 was administered from a 5Fr pigtail catheter at the terminal aorta and, in the left external iliac artery, CO_2 was unfilled and there appears to be stenosis. With contrast angiography, no stenotic lesion is seen in the left external iliac artery (**Figure 41B**).

- Make a separate system with CO_2 cylinder
- Average dose of CO_2 is about 30 ml by using 50 ml syringe
- Be sure complete air excretion
- Manual injection not a mechanical injection
- If patient complains of abdominal pain, avoid further injection
- When a slow flow are observed, avoid an further CO_2 injection

Table 3. Setup of CO_2 delivery system and CO_2 injection.

Figure 41. Iliac artery angiography by CO_2 and DSA. (A) CO_2 administered from 5Fr pigtail catheter at the terminal aorta, in left external iliac artery, CO_2 unfilled and stenosis suspected. (B) In DSA, no stenotic lesion in left external iliac artery observed.

In **Figure 42A**, total occlusion of the left external iliac artery is observed. CO_2 injection from the terminal aorta shows chronic total occlusion (CTO) of the left external iliac artery. To confirm CTO, a crossover sheath was positioned at the left common iliac artery and CO_2 injection was repeated at a right anterior oblique (RAO) projection of 30° (**Figure 42B**). In this angiogram, CTO is clearly visualized and the left common femoral artery is well observed via the collateral flow from the deep circumflex artery. In CO_2 angiography of the iliac artery, the angiogram is hampered by bowel and gas movements.

In **Figure 43A**, the left iliac artery is not seen, but contrast angiography shows a clear picture of the entire iliac arteries (**Figure 43B**).

Generally speaking, when the iliac artery is not well visualized by CO_2 angiography, increasing the volume of CO_2 in the iliac abdominal aorta might be considered. However, there are important visceral vessels and the risk of various complications due to the injection of gas in these vessels must also be considered.
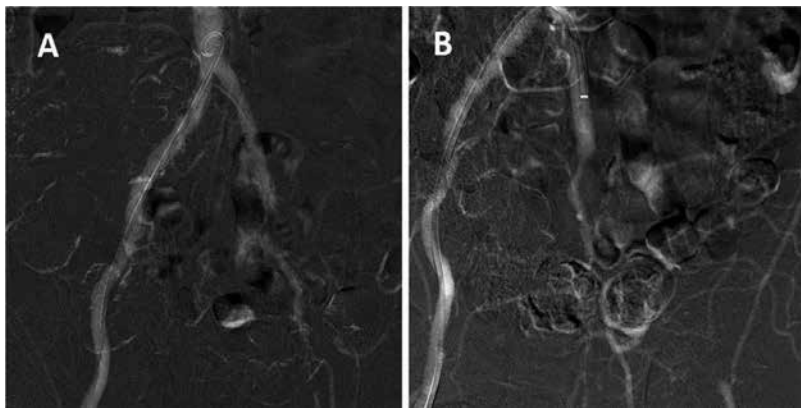
Figure 42. Left external iliac artery occlusion by CO_2 angiography. (A) CO_2 injection from terminal aorta in AP view. Total occlusion of left external iliac artery. (B) CO_2 injection from left common iliac artery by RAO 30.
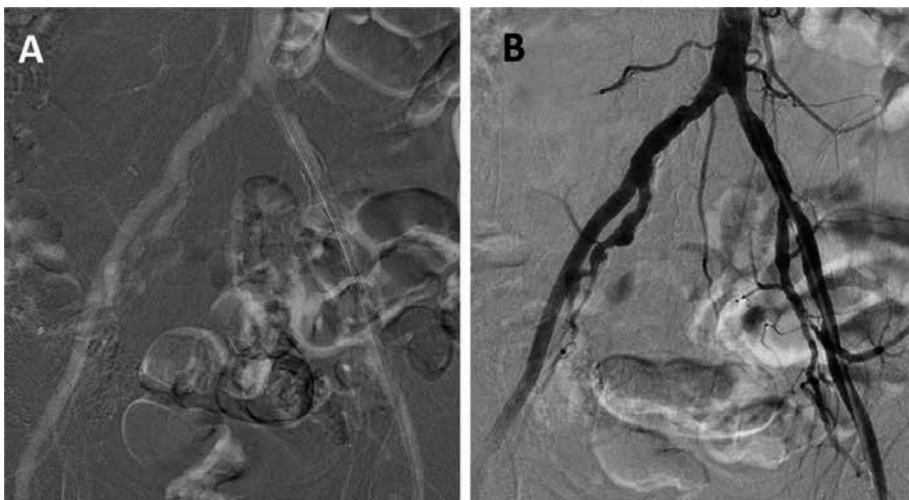
Figure 43. Bowel gas in iliac artery angiography. (A) In CO_2 angiography, left external iliac artery is hampered by bowel gas. (B) DSA shows a clear picture of whole iliac arteries.

4.3. Femoropopliteal artery angiography by CO_2

Visualization by CO_2 angiography is best suited for the femoropopliteal artery segment. The main reason is that the superficial femoral artery (SFA) is a straight vessel with small branches. The vessels sizes are about 4–7 mm and could easily be filled by CO_2 gas. In **Figure 44**, there are three kinds of SFA angiograms for the same patient. Digital angiography enabled visualization of the background (**Figure 44A**) while DSA could obtain the highest quality angiogram (**Figure 44B**). CO_2 angiography has poor visibility of small distal branches. However, it could visualize SFA fairly well and can be used as a substitute for contrast angiography (**Figure 44C**).

Similarly, the popliteal artery could be well observed even with the CO_2 angiogram (**Figure 45**).

Figure 44. SFA angiography by digital, DSA and CO_2. (A) Digital angiography could visualize background to be used as reference. (B) DSA obtained most accurate image. (C) CO_2 angiography cannot replace digital angiography, but can be used as a substitute for contrast angiography.

Figure 45. Popliteal artery angiography by digital, DSA and CO_2. (A) Digital angiography could visualize background to be used as reference. (B) DSA obtained most accurate image. (C) CO_2 angiography obtained similar image to DSA.

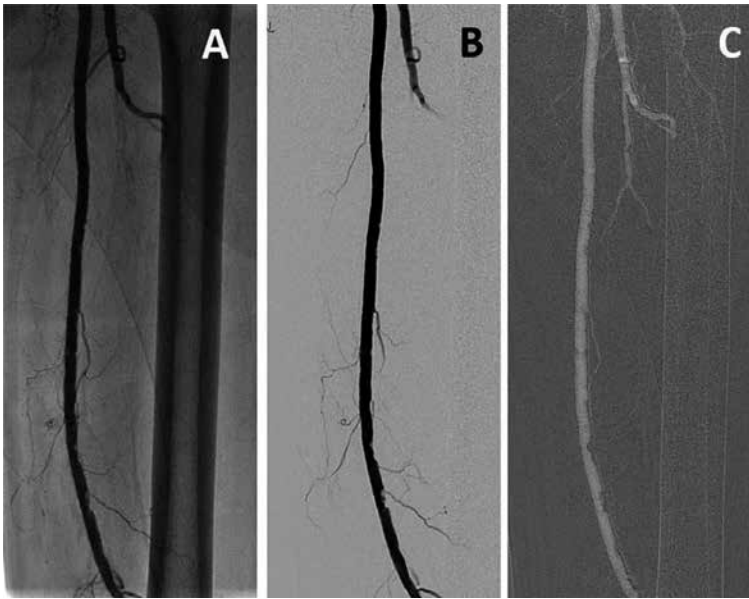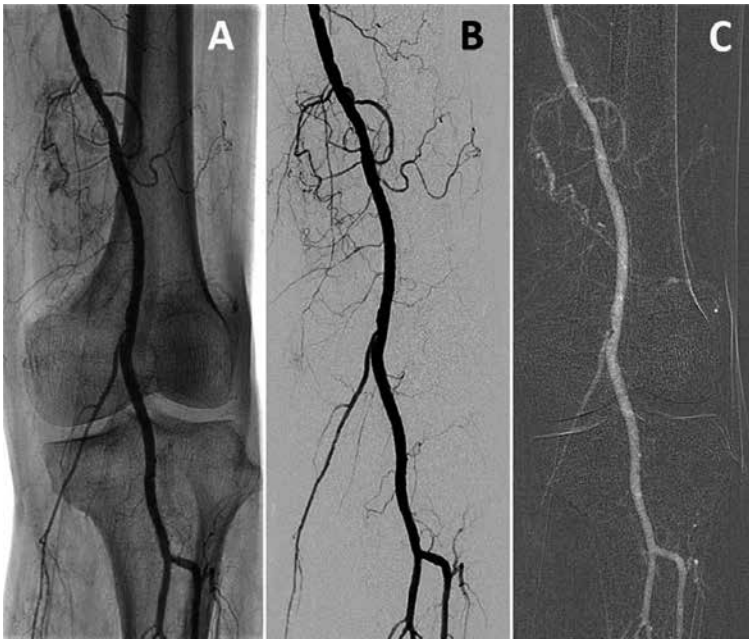In the DA angiogram, distal SFA is not well visualized compared to the DSA image (**Figure 45A**). In fact, a perfect image was obtained by DSA (**Figure 45B**). The CO_2 angiogram shows a fairly clear picture of the distal SFA and popliteal artery (**Figure 45C**) while the right femoropopliteal artery was visualized by CO_2 (**Figure 46**). In proximal SFA, separation between the SFA and deep femoral artery (DFA) is well observed (**Figure 46A**). In the mid-SFA, no stenosis is seen (**Figure 46B**). In the distal SFA and popliteal artery, moderate stenosis is detected (**Figure 46C**).
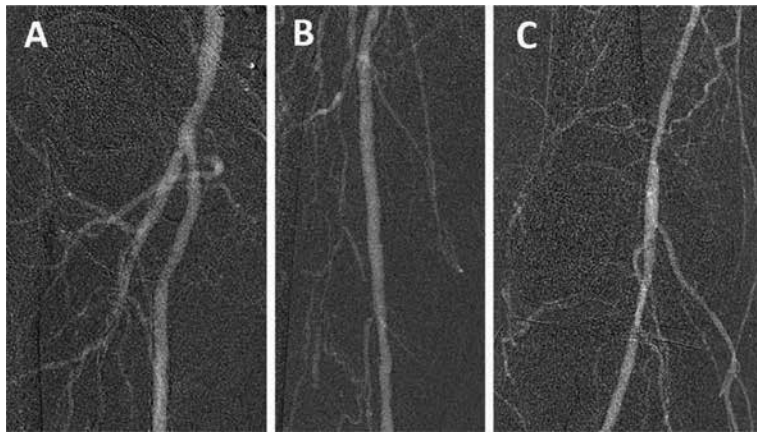
Figure 46. CO_2 angiography for right femoropopliteal artery. (A) In proximal SFA, separation between SFA and deep femoral artery (DFA) is well shown by RAO view. (B) In mid-SFA, there is no stenosis. (C) In distal SFA and the popliteal artery, there is moderate stenosis.

Comparisons between CO_2 angiography and digital angiography for the diseased SFA are shown in **Figures 47** and **48**. Stenosis is seen in the distal SFA in **Figure 47**. Both CO_2 (**Figure 47A**) and DA images (**Figure 47B**) could identify distal SFA stenosis.

The totally occluded left SFA was well visualized by CO_2 angiography (**Figure 48A**). Although DSA shows a clearer image with a rich collateral network (**Figure 48B**), the CO_2 image can also be used for interventional work.

The CTO of the left SFA was intervened using CO_2 angiography (**Figure 49**) in a patient with stage 4 CKD. The CO_2 angiogram showed typical CTO of the SFA (**Figure 49A**). After successful wiring, balloon angioplasty was performed (**Figure 49B**). Contrast was only used in the final angiogram (**Figure 49C**).

4.4. Below the knee angiography by CO_2

CO_2 angiography cannot be applied in below the knee (BK) work. The arterial vessel size below the knee is between 1.5 and 3 mm in diameter and the accuracy of CO_2 angiography is insufficient. Above all, in BK cases, most of the patients have critical limb ischemia and cannot tolerate large amounts of gas injection. In **Figure 50**, proximal right below the knee angiogra-

phy was performed by CO_2 (**Figure 50A**) and DSA (**Figure 50B**). In the CO_2 angiogram, stenosis of the peroneal trunk could be seen; however, the right anterior tibial artery and posterior tibial artery are not well visualized when compared to DSA.
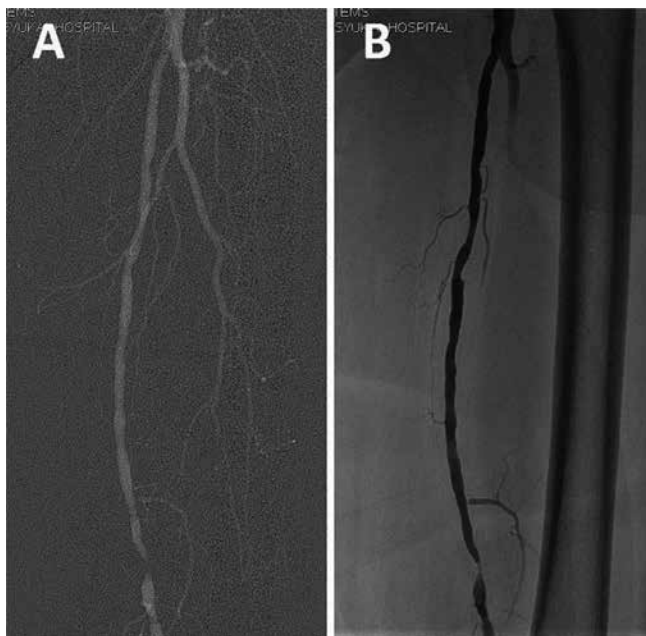
Figure 47. Left SFA stenosis by CO_2 and digital angiography. (A) CO_2 angiography shows moderate stenosis in mid SFA and tight stenosis in distal SFA. (B) Digital angiography confirmed these two lesions. Image quality is similar between CO_2 and digital angiography.

4.5. Problems of CO_2 angiography

CO2 angiography can be performed with minimal or no contrast media and can be used on CKD patients. However, CO2 angiography carries several potential risks (8). Gas delivery into the vessel is basically contraindicated. Moreover, erroneous injection of excessive volumes may result in catastrophic clinical consequences. There are many reports about transient lower limb pain and transient abdominal pain. Fujihara et al. have conducted a multi-center prospective CO_2 study and have reported that two patients (2%) developed CO_2-related non-occlusive mesenteric ischemia which resulted in death. These non-occlusive mesenteric ischemia cases were caused by the trapping of CO_2 gas in the celiac, superior and/or inferior mesenteric arteries [8]. The quality of CO_2 angiography is still not clear enough in the iliac artery and should not be employed in below the knee arteries. It should be used for the femoropopliteal artery although, even in the femoropopliteal artery, precise lesion evaluation may be difficult in some cases. Other complementary modalities such as surface echo, IVUs and/or pressure measuring should also be employed to confirm lesion severity (**Table 4**).

In treating claudication, safety is the first priority so that CO_2 use may be limited for most patients except those who have anaphylaxis to iodinated contrast media.
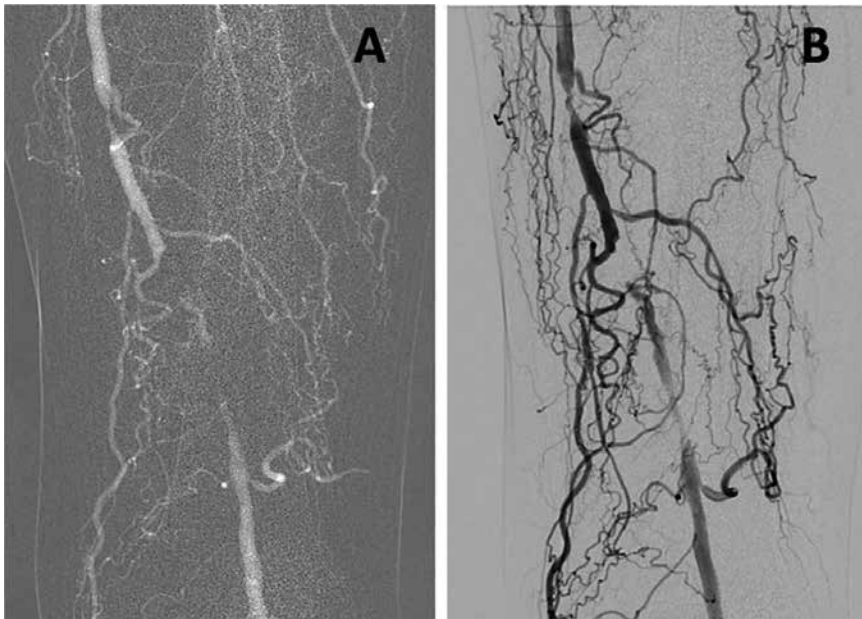
Figure 48. Left SFA occlusion by CO_2 and DSA. (A) CO_2 angiography shows totally occluded left SFA. (B) DSA shows clearer image with more collateral visualization.
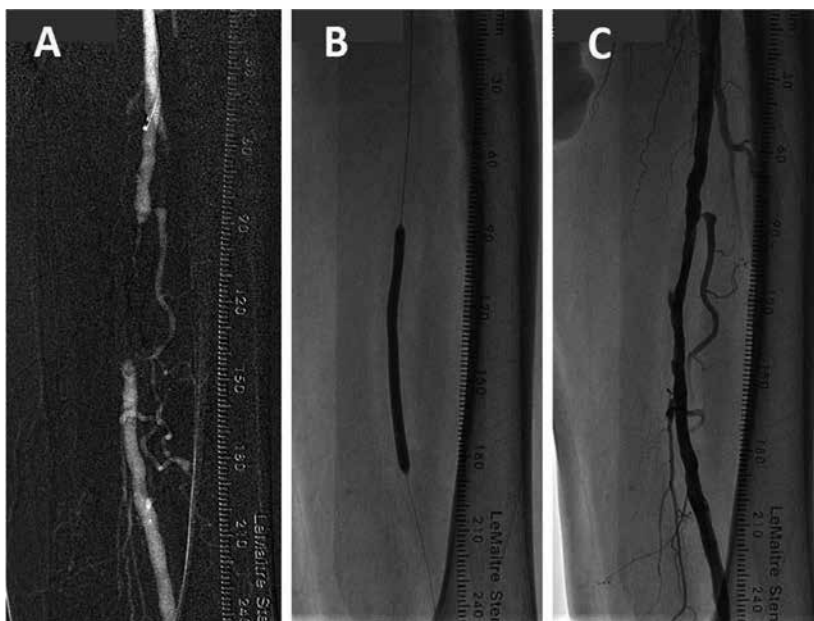
Figure 49. Left SFA CTO intervention by CO_2 angiography. (A) CO_2 angiography showed typical SFA CTO. (B) After wiring, balloon angioplasty was performed. (C) Contrast used only in final angiogram.
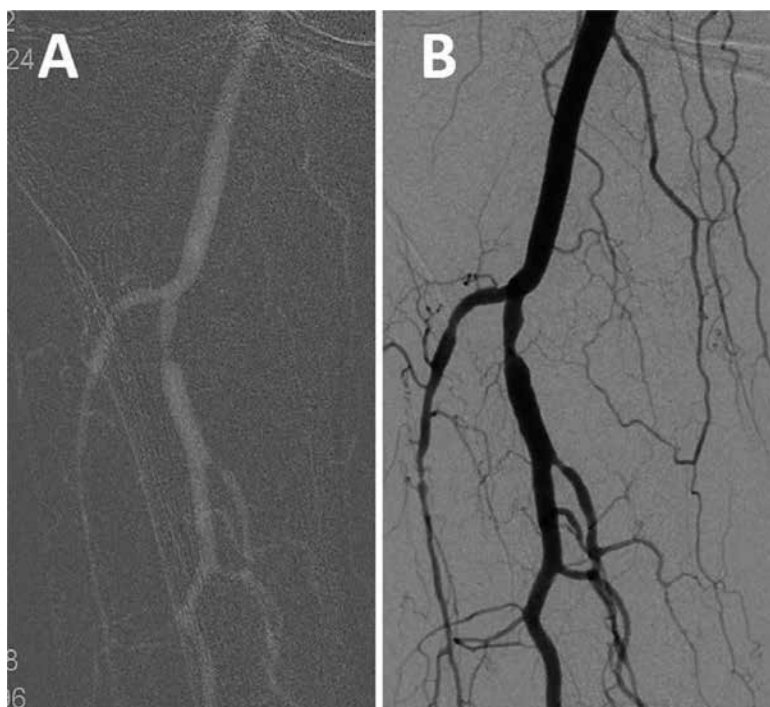
Figure 50. Below the knee angiography by CO_2 and DSA. (A) Proximal below the arteries by CO_2 angiography. Stenosis of peroneal trunk could be seen, otherwise, unable to identify right anterotibial and posterior tibial artery. (B) DSA shows detail of proximal below the knee arteries with small branches.

- Cause abdominal pain and leg pain
- Poor quality angiogram in iliac artery by bowel gas and movement
- Not applicable to below knee artery
- Rapid transition to venous circulation
- Risks of cerebral infarction
- Risk of nonobstructive mesenteric ischemia

Table 4. Problems of CO_2 angiography.

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Angiography and Endovascular Therapy for Aortoiliac Artery Disease

Keisuke Fukuda

Abstract

The treatment of aortoiliac occlusive disease has shifted to endovascular therapy. Even in complex lesion, precise angiography enables successful treatment in endovascular work. However, complications-related endovascular intervention of these lesions is catastrophic. Accurate angiographic assessment is mandatory to complete the procedure. This article reviews angiographic approach for aortoiliac artery disease in diagnosis, endovascular intervention, and complications.

Keywords: aortoiliac occlusive disease, TASC II classification, access

1. Introduction

Aortoiliac occlusive disease results from the common cardiovascular condition atherosclerosis, accounting for approximately one-third of all symptomatic peripheral artery disease (PAD) [1]. Most cases of aortoiliac artery disease presents with lower limb claudication and can potentially progress to critical limb ischemia. In revascularization for aortoiliac disease, endovascular therapy is increasingly being employed instead of surgical bypass. Even in complex aortoiliac artery diseases such as bifurcation disease, long segment occlusion, and aortic occlusion, endovascular therapy is being employed with the application of stents [2, 3]. The most commonly quoted classification of iliac lesions has been set forth by the Trans-Atlantic Inter-Society Consensus (TASC II) group with recommended treatment options [4]. These classifications of their morphology have finally been defined by assessment in accurate detail with angiography. Therefore, precise angiographic assessment is vital to success in aortoiliac artery interventions. This article reviews an angiographic approach to aortoiliac artery disease presenting various detailed images and analyses pre- and postrevascularization.

2. Angiographic technique for the aortoiliac artery

2.1. Vascular anatomy of the aortoiliac artery

A solid understanding of the vascular anatomy of the aortoiliac artery in angiography is indispensable to diagnostic or interventional work. The vasculature within range from the infra-renal artery to the common femoral artery is shown in **Figure 1**.
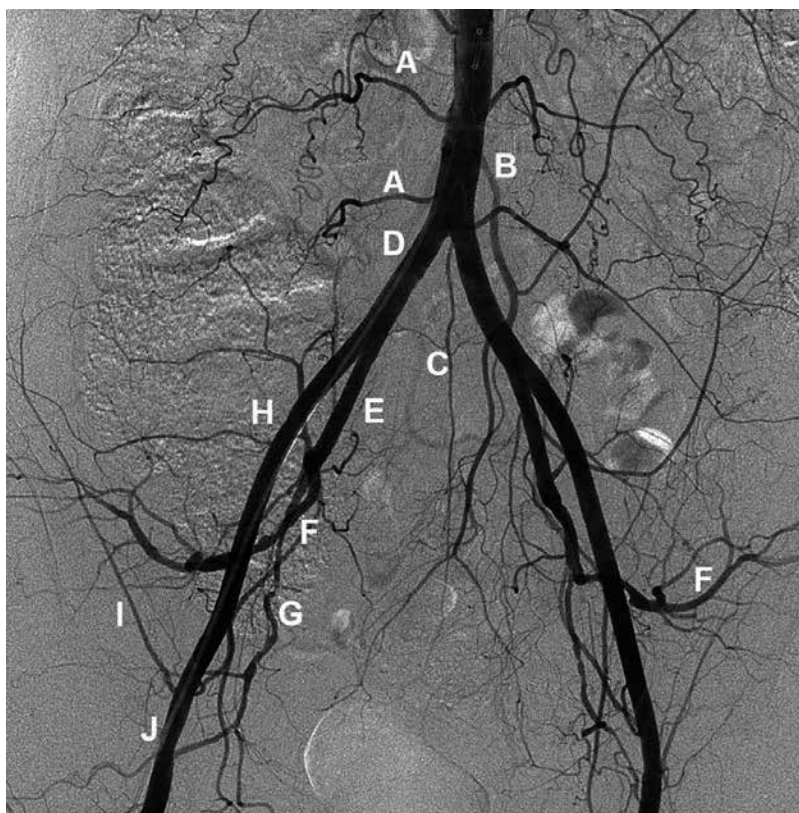
Figure 1. Anatomy in aortoiliac artery angiography. Aortoiliac angiography image includes abdominal aorta, lumber branch (A), inferior mesenteric artery (B), median sacral artery (C), common iliac artery (D), internal iliac artery (E), superior (F) and inferior (G) gluteal arteries, external iliac artery (H), deep circumflex iliac artery, (I) and common femoral artery (J).

Commonly, the aortoiliac angiographic image includes the abdominal aorta, lumber branch, inferior mesenteric artery, median sacral artery, common iliac artery (CIA), internal iliac artery (IIA), superior and inferior gluteal arteries, external iliac artery (EIA), deep circumflex iliac artery, and common femoral artery (CFA). Safe and successful procedures are accomplished by meticulous manipulation of a wire and/or catheter to a target vessel. Particularly, in the case of complex aortoiliac disease with total occlusion or calcification, a wire or catheter is more likely to stray unintentionally into unexpected vessels. Therefore, a thorough understanding of the vasculature including a normal vasculature, anomalies, and complex collateral networks is required before any intervening procedure.

2.2. Image size and optimal view

To visualize aortoiliac disease, an image field of more than 30 cm is required. Information on the vessel size and lesion length is needed as well. The wide 30 cm image could not confirm the precise morphology of the lesion, while a 20 cm image was better suited (**Figure 2**).
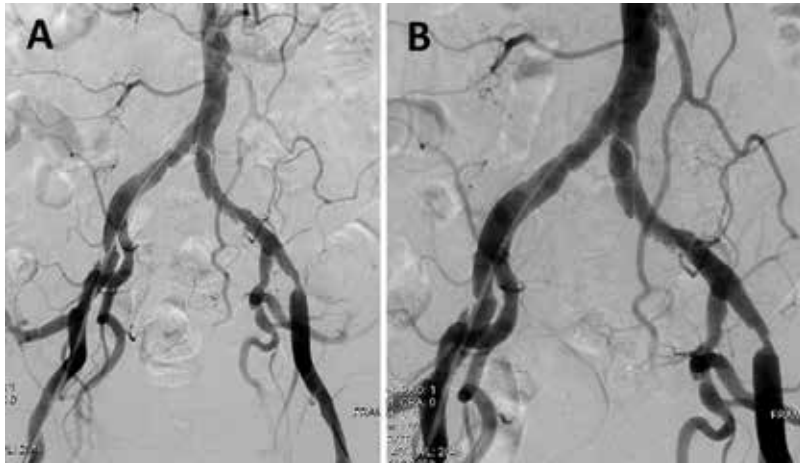
Figure 2. Aortoiliac artery angiography by 30 and 20 cm image size. (A) By 30 cm DSA image, whole aortoiliac disease is visualized. (B) By 20 cm DSA image, the precise morphology of left common iliac artery ulcerated lesion and tight stenosis of left external iliac artery are well visualized.

Unlike femoropopliteal or below the knee arteries, the aortoiliac arteries are located in the intraperitoneal space and form three-dimensionally from the terminal aorta to the common femoral artery (**Figure 3**).
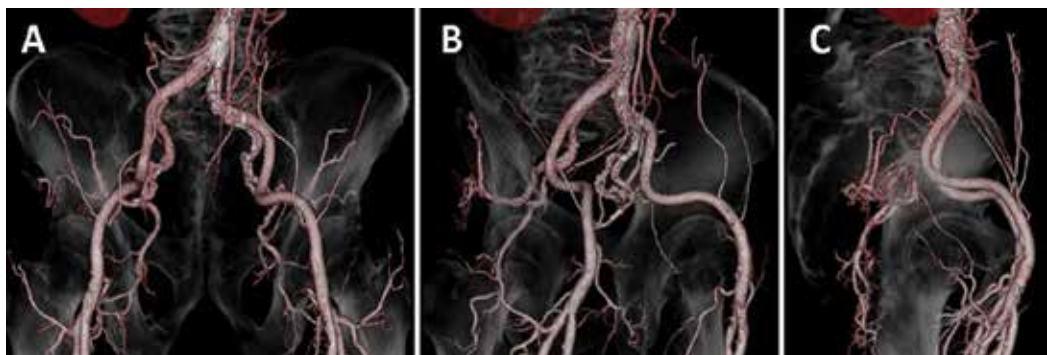
Figure 3. Aortoiliac artery by 3D CT angiography. (A) AP view, (B) RAO 45° view, (C) lateral view. RAO view and lateral view show that aortoiliac arterial system located in intraperitoneal space with three-dimensional CT angiography.

Multidirectional projection in angiography is also useful to achieve successful endovascular revascularization. The optimal view for interventional work is commonly obtained by magnification and various oblique projections. Not only anterior-posterior views but also a contralateral view is important (**Figure 4**).
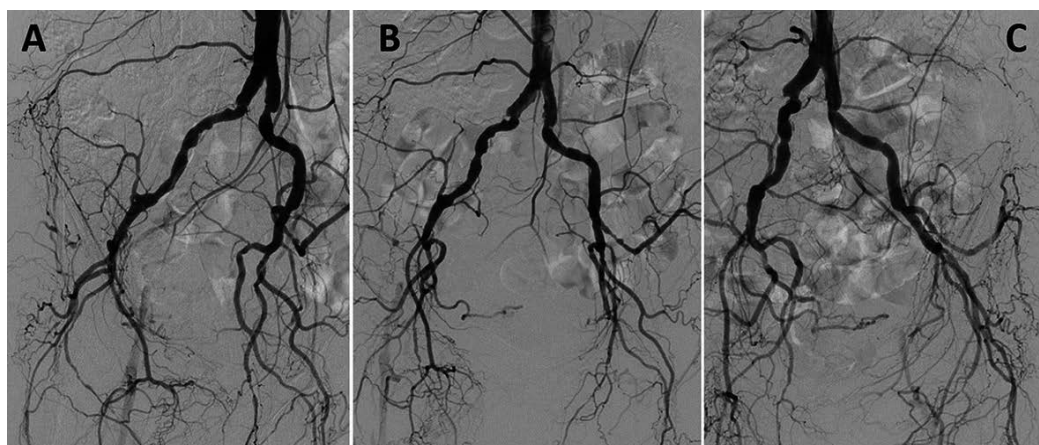
Figure 4. Basic three aortographic views. (A) DSA in LAO view. (B) DSA in AP view. (C) DSA in RAO view.

If a lesion could not be identified or is not well visualized, angiography should be performed using a contralateral oblique (20°–30°) projection. Particularly in EIA lesions, this angled view is imperative in obtaining accurate information on a lesion (**Figure 5**).
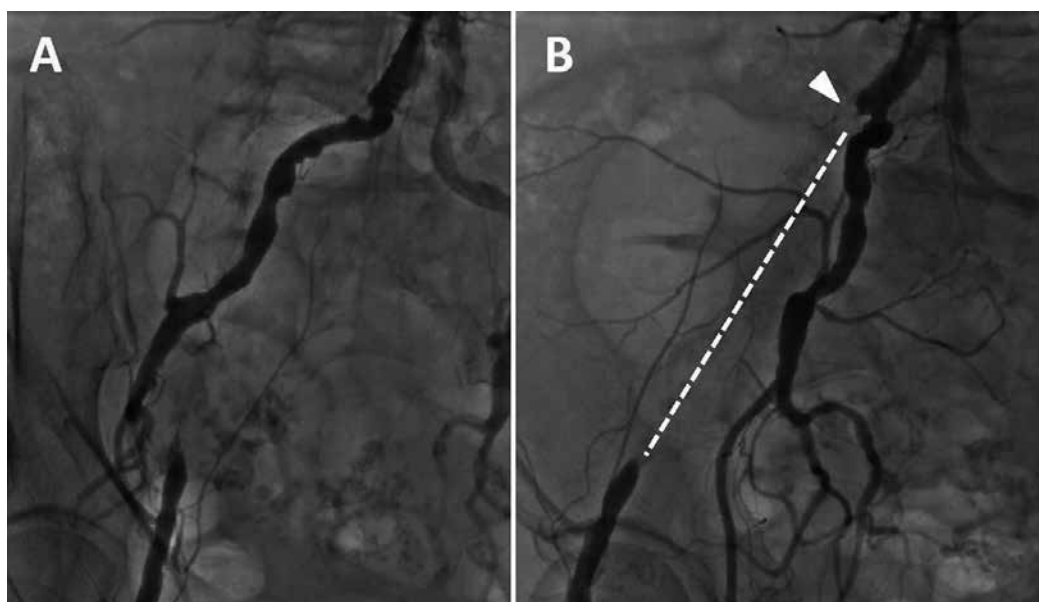
Figure 5. Right EIA occlusion in LAO and RAO views. (A): in LAO view, stump is hidden and occluded lesion overlaps with internal iliac artery. (B): In RAO view, stump (arrow head) can be clearly visualized and occluded lesion is well-isolated (white dot line).

Digital subtracted angiography (DSA) images are considered to be the standard modality for iliac artery disease, but visualization of the pelvic vessels is often interfered by bowel movement and gas in a 30 cm image. Breath holding during contrast injection is requested in conscious

patients. With recent advanced digital angiography (DA), similar images to DSA can be obtained. Moreover, to take excellent images for diagnostic angiography, the 5-Fr pigtail catheter is preferred over a 4-Fr system. Decisions for intervention are thus made after diagnostic angiography. We exchange to a 6-Fr system if intervention is indicated.

2.3. Access

2.3.1. Antegrade upper limb access

Upper limb access including the brachial or radial artery has several advantages compared to conventional femoral access in terms of anatomical reasons, fewer bleeding complications, and/or patient preference [5]. In common iliac artery disease, the contralateral femoral artery approach may encounter difficulty when crossing the contralateral common iliac artery lesion. Left brachial or radial artery access can be employed for aortography [6]. This approach is useful in cases with suspected or known total occlusion in aortoiliac artery lesions [7]. Generally speaking, the left brachial or radial artery approach is anatomically suited to reach the terminal aorta through the aortic arch due to the shorter length and more linear route from the access site to the terminal aorta, so that catheter manipulation of the catheter and wire is much easier as compared to the right upper limb approach (**Figure 6**).
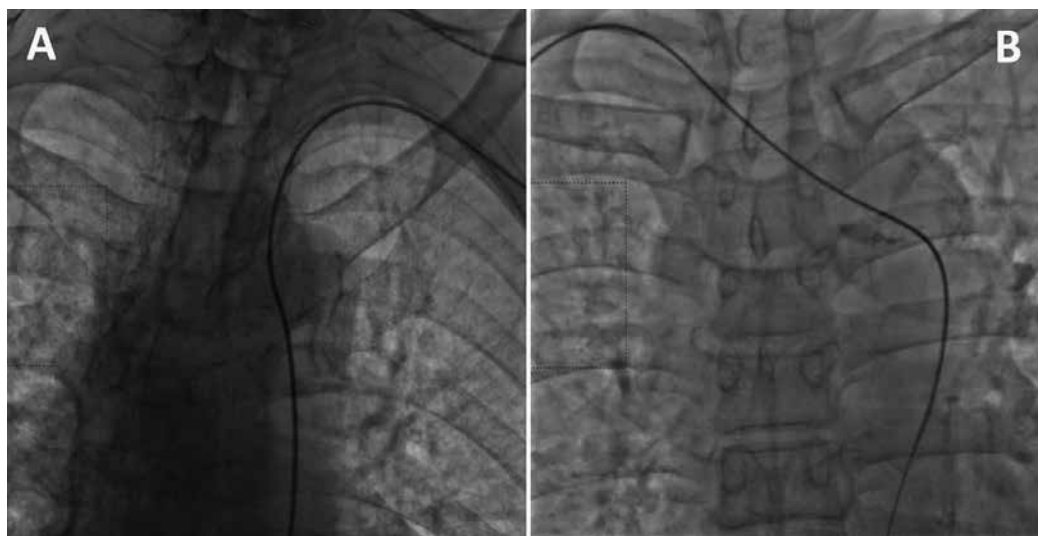
Figure 6. Difference between left and right upper limb approach. (A) From left arm approach, the shape of the wire is in linear form with shorter length. (B) From right arm approach, the wire and catheter have to pass through the aortic arch.

In complex aortic bifurcation or proximal CIA occlusions, the antegrade approach is necessary and effective in conjunction with the femoral approach for successful intervention. Additionally, this approach is vital in cases when the crossover technique cannot be employed due to previous kissing stenting at the aortic bifurcation or when CFA access is hindered due to severe stenosis or anastomosis for a bypass graft. A lesion may be easily

crossed with a wire through a 6-Fr-long sheath from the left brachial artery, and ballooning or stenting can then be performed by this approach. In recent endovascular devices for PAD, there are systems with long shafts of between 130 and 150 cm in length and these devices enable an upper limb approach. However, these are limited to balloons and stents. Large diameter size devices such as those for atherectomy are not advised for a brachial or radial artery approach.

2.3.2. Retrograde ipsilateral CFA access

The retrograde ipsilateral CFA approach is most frequently used as a standard technique. For a precise CFA puncture, we routinely use echo with fluoroscopy guidance. After confirming no CFA disease, an ipsilateral approach is selected for iliac artery intervention. If there is no CFA disease, we advance a 4-Fr or 5-Fr pigtail catheter to the terminal aorta. By power injection of the contrast dye at the terminal aorta, we inject 13 cc/s for a total of 26 cc by a 4-Fr pigtail and 16–18 cc/s for a total of 25 cc by a 5-Fr pigtail catheter. Manual contrast injection is not recommended at the aortoiliac artery. In TASC ll A or B lesions, retrograde sheath injection can be performed. In bilateral iliac lesions, if a contralateral CIA ostial lesion is not involved, treatment can proceed by a retrograde and crossover antegrade technique at one femoral artery access site (**Figure 7**).
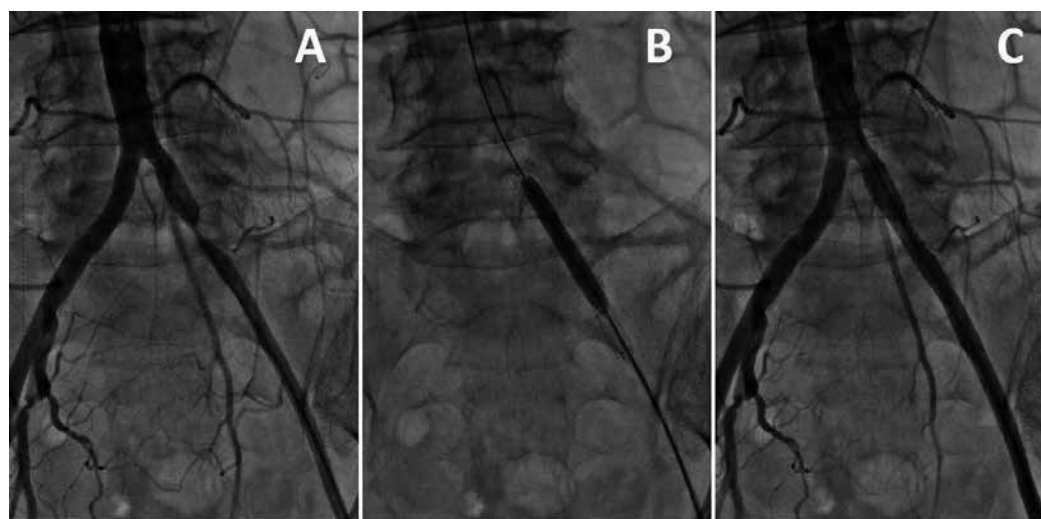
Figure 7. Angiography and treatment from ipsilateral CFA sheath. (A) Angiography from retrograde sheath injection shows severe stenosis of left EIA. (B) Ballooning and stenting performed from retrograde sheath. (C) Completion of angiography from retrograde sheath shows good results.

In intervention for total occlusion of the common iliac artery with or without distal EIA disease, a bidirectional wiring technique is the key to successful recanalization. Therefore, ipsilateral retrograde intervention in conjunction with an antegrade approach from the contralateral femoral side or from the brachial or radial artery is essential for successful wiring.

2.3.3. Retrograde contralateral CFA access

Retrograde contralateral CFA access is an indispensable technique for interventions of peripheral artery disease at each level of the lower extremity, including the iliac, femoropopliteal, and below the knee arteries. For interventions from the iliac artery to below the knee, the crossover technique is requisite. Mastering the crossover technique is required of every interventionist since, in patients with CFA, proximal SFA, and/or distal EIA disease, this approach is mainly selected. Operators who are experienced and skillful in this technique can freely select any access site, leading to safer procedures and more successful outcomes. Total occluded iliac artery interventions commonly require a bidirectional approach from both CFA access sites. To cross the guidewire to the contralateral iliac artery, we generally use a JR diagnostic (4 or 5 Fr) catheter placed to the bifurcation of the terminal aorta. If there is an acute angle at the bifurcation of the iliac artery, the 4-Fr IMA catheter can be used. We do not use a pigtail catheter for crossover purposes since the movement of the wire is unpredictable and may damage the vessel wall. Exchange to a stiff guidewire was performed to advance the crossover sheath (5 or 6 Fr). In the case of heavily tortuous or calcified CIA, it is very difficult to advance the sheath to the contralateral side. In such a case, the balloon anchor technique is helpful to crossover the sheath (**Figure 8**) [8].
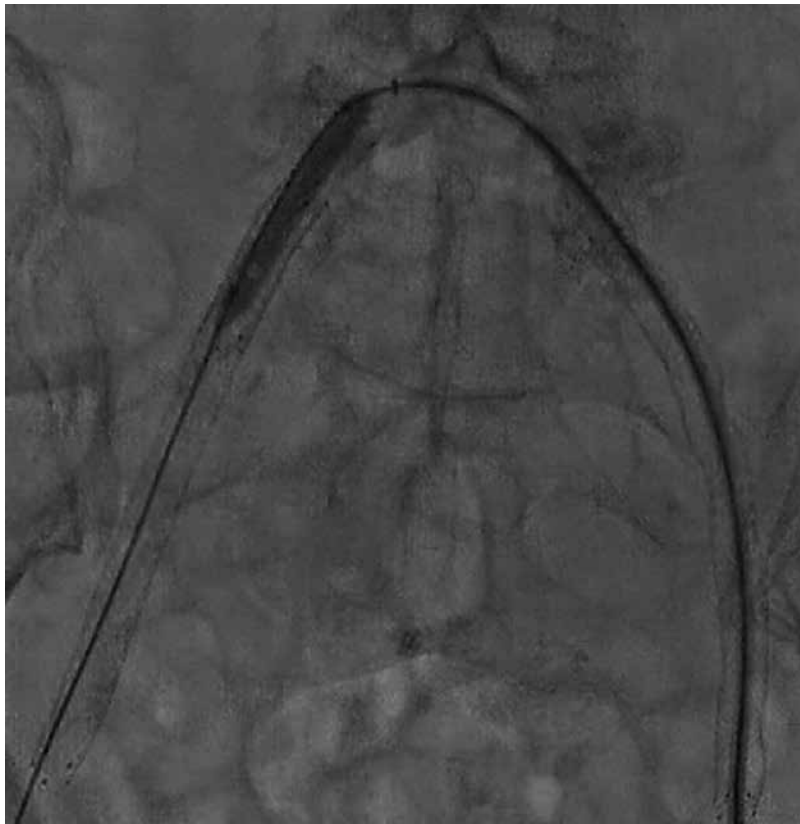
Figure 8. Using balloon anchor technique, the crossover sheath was advanced to contralateral iliac artery.

However, there are some cases when crossover at the bifurcation is not possible (**Figure 9**), for example, with a sharp-angled isthmus, poststent graft, or postkissing stent. Therefore, detailed reinterventional planning is essential.

Figure 9. Crossover technique is not indicated in the case of sharp-angled isthmus, poststent graft, or postkissing stent.

3. Aortic stenosis and aortic occlusion

Many studies have demonstrated that endovascular therapy for extensive aortoiliac occlusive disease shows better long-term patency and clinical outcomes [9–11]. The main strategy of endovascular therapy for this field has increasingly shifted to primary stenting [12–14]. Balloon expandable stents, self-expanding stents, or covered stents are basic treatment options. In iliac artery stenting, a self-expandable stent is preferred to a balloon expandable system. The self-expandable stent is compatible with a 6-Fr sheath, and sizes up to 10–12 mm are available. Selection of the stent type may be left to the operator based on lesion location and characteristics. In the treatment of aortic occlusion, catastrophic complications may occur. Operators need to keep in mind the risks of rupture, dissection or emboli to the renal, mesenteric artery or distally to the pelvic arteries, or lower limb arteries. There are

reports that aortic occlusion can be treated with an endovascular technique using the three way access sites including the upper limb or both CFAs [15–18].

Infra-renal aortic stenosis of less than 3 cm and a focal lesion is classified as TASC ll B which is considered a good indication for endovascular intervention as first line therapy. Isolated infra-renal aortic stenosis is shown in **Figure 10A**. To clearly visualize and identify aortic stenosis, a lateral view is useful (**Figure 10B**) and measurement of the pressure gradient across the lesion is a factor in making the final decision.
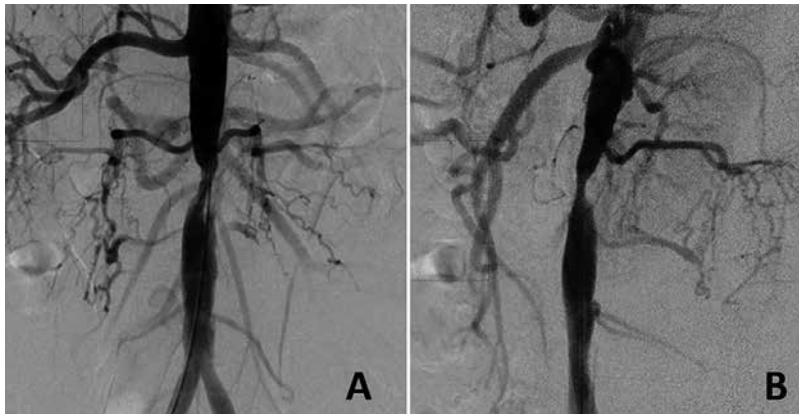
Figure 10. Isolated infra-renal aortic stenosis. (A) DSA image in AP view shows aortic stenosis of less than 3 cm. (B) In lateral view, stenosis is more visible.

Aortic stenosis is mostly treated with a self-expandable stent and postdilatation because of the large vessel size (**Figure 11**).

Aggressive postdilatation is not recommended in order to avoid the above mentioned complications. Even if moderate residual stenosis remains, reducing the pressure gradient across the lesion (<10 mmHg) is acceptable. In the case of aortoiliac occlusion involving bilateral CIAs (**Figure 12A**), upper limb and bilateral CFA access is required. Wiring is performed from the upper limb through a long sheath with simultaneous retrograde angiography from the CFA to guide the direction of the wiring (**Figure 12B**). Kissing stent insertion and kissing ballooning resulted in excellent recanalization on this complex occlusion at the terminal aorta (**Figure 12C** and **D**). Stenting above the aortic bifurcation combined with the kissing stent technique can be considered an effective alternative option to open surgical reconstruction for extensive aortoiliac occlusive disease.

Complex distal aortic occlusive disease often involves either the CIA or EIA (**Figure 13A**). A retrograde 0.035 wire carefully passed through the bifurcation to the abdominal aorta, and initial stent was placed at the aorta (**Figure 13B**). After the first stenting, the next wire was carefully crossed inside the aortic stent from the other side of the CFA access. After the two wires were crossed inside the aortic stent, a kissing stent technique was performed (**Figure 13C**), and the final angiogram was taken (**Figure 13D**). In this poststenting DSA, care must be taken to check for any perforations or residual thrombi.
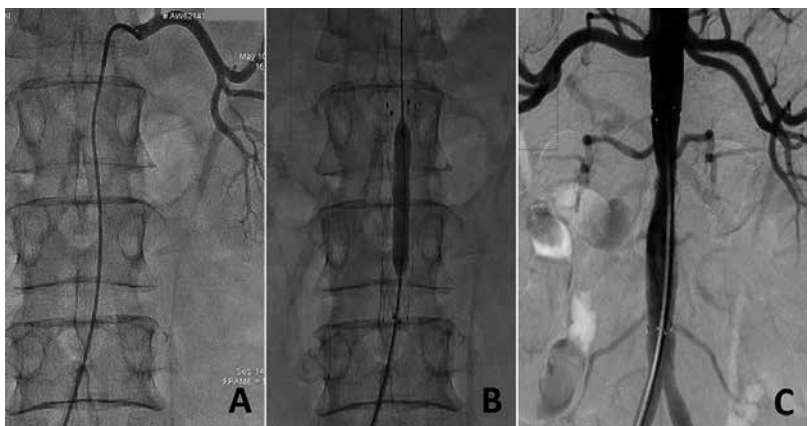
Figure 11. EVT for isolated infra-renal aortic stenosis. (A) Ostium of renal artery is confirmed by selective renal artery angiography. (B) After stenting, postdilatation is performed by lower diameter balloon at relatively lower pressure. (C) Final angiography shows acceptable dilatation of the lesion and no complications.
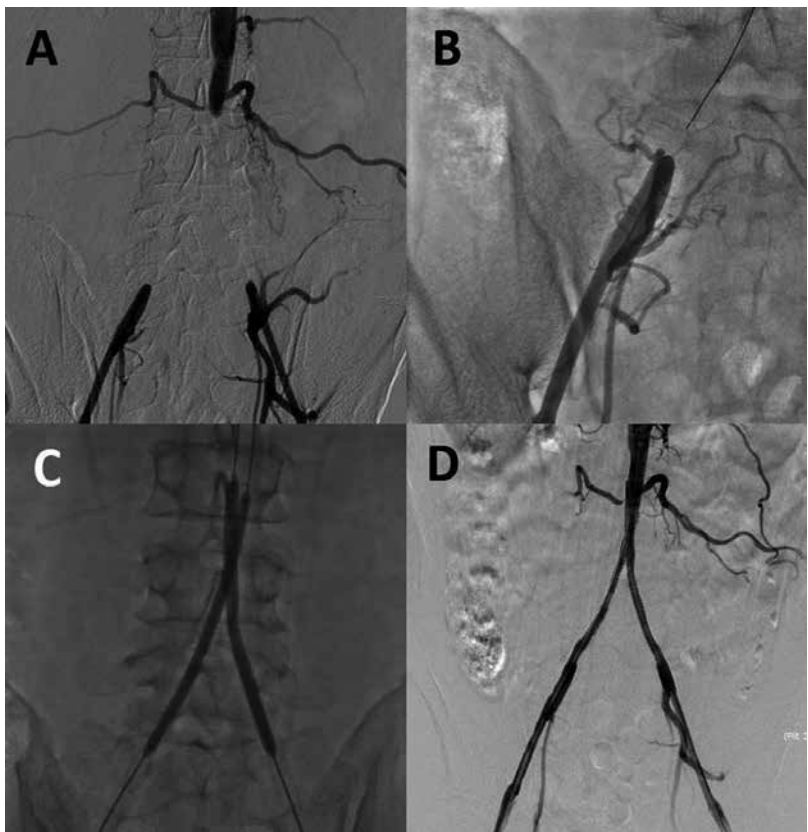
Figure 12. Aortic occlusion involving bilateral CIA. (A) DSA injected from three ways access can clearly visualize total occlusion of aortic bifurcation. (B) Simultaneous retrograde angiography from CFA in order to direct wiring from upper limb. (C) Kissing stent technique and kissing postdilatation are performed from bilateral CFA. (D) Completion of DSA angiography shows excellent recanalization.
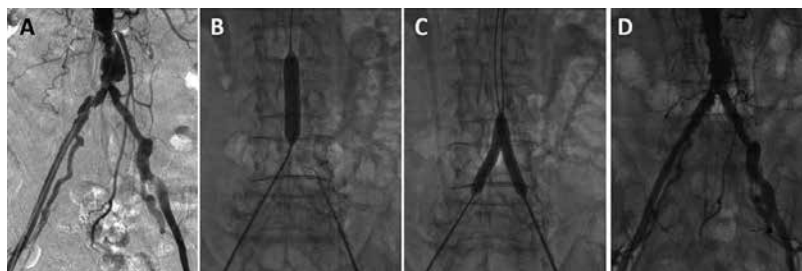
Figure 13. Complex distal aortic occlusive disease involving either CIA or EIA. (A) The 30 cm DSA image shows aortic stenosis extending to both CIAs and the left EIA. (B) Final angiogram is taken in DA, which shows complete recanalization. (C) Initial stent is placed at aorta, and postdilatation at lower pressure was performed. (D) After two wires are carefully crossed inside the initial stent from both CFA sheaths, a kissing stent is implanted.

4. Iliac artery disease: TASC ll A-C

Endovascular treatment has become a major part of revascularization of iliac artery occlusive disease [19–21]. Among the various interventional devices, stent is main stay of treatment strategy. From TASCII A to TASCII C, the stent implantation is now the standard technique and shows reasonable good long-term outcome with low reinterventions rate. In TASCII D, the basic strategy is considered to be surgical approach. However, recent development of endovascular technique enabled endovascular first approach, and in experienced hands, fairly high success rate can be achieved.

The iliac stent choice can be largely categorized into choosing either a balloon expandable or self-expanding stent based on lesion characteristics. It depends on introducer size, access site, vessel tortuosity, and lesion location. However, we are using self-expandable stent in every iliac artery lesion. Newer-generation nitinol self-expanding stents exhibit minimal foreshortening and have a more predictable length with 12 mm in size by 6-Fr sheath.

In image size for iliac artery intervention, 20 cm size with DA image is preferred to 30 cm image because position adjustment and determination of stent size or length. In recanalization of aortoiliac occlusive lesion, measurement of pressure gradient across the lesion should be checked pre- and postprocedure. This hemodynamic assessment can be helpful to make the final decision to terminate the procedure even after angiographic moderate stenosis remained [22]. This section shows that typical TASCII A-C iliac artery lesion underwent endovascular therapy.

4.1. TASC II A lesion

Case 1: In **Figure 14A**, a typical lesion of the left CIA is presented. The DSA image (30 cm) of aortography with a 5-Fr pigtail catheter is taken from a 6-Fr sheath placed at the ipsilateral left CFA. Isolated severe stenosis with ulceration of the left CIA can be observed. For intervention of this lesion, a DA image by a contralateral right anterior oblique (RAO) view with a 20 cm image size is employed as the standard method (**Figure 14B**). Compared to a 30 cm image, more precise information on lesion characteristics can be obtained. After diagnostic

angiography, the lesion was crossed by a 0.035-inch wire through a diagnostic pigtail catheter. After visual reference of the bony landmarks on the DA image, a self-expanding stent was implanted followed by postdilatation by balloon (**Figure 14C**). The final angiogram is shown in **Figure 14D**. To avoid vessel rupture and dissection, aggressive postdilatation is not recommended. We usually use a smaller balloon of up to 7 or 8 mm in diameter and inflation with lower pressure. When considering a balloon size of more than 8 mm for postdilatation, careful vessel size assessment is required.

Figure 14. Left CIA stenosis classified as TASCII type A. (A) DSA image in 30 cm size of aortography in AP view shows unilateral left CIA stenosis. (B) DA in larger size (20 cm) and in contralateral oblique (RAO) view more clearly revealed lesion. (C) Stenting and ballooning are performed in reference to visual bony landmark. (D) Final angiogram shows complete recanalization.

Case 2: Aortography of a bilateral short EIA lesion is shown in **Figure 15A**. However, this 30 cm image size and AP projection failed to visualize the precise characteristics of each EIA lesion. The 20 cm size image with contralateral oblique angiography could identify the EIA lesions (**Figure 15B** and **C**). Endovascular intervention was performed by the right CFA approach. First, from the right CFA sheath, a 0.035-inch guidewire was used to cross the right EIA lesion followed by stenting and ballooning. After confirmation of a good expanded stent on the right EIA lesion, a crossover approach was introduced to treat the left EIA lesion. For the left EIA lesion, a RAO view is generally taken. This left lesion was also treated by stenting and ballooning (**Figure 15D** and **E**). These views could clearly visualize the lesion in high tortuosity in the EIA. Generally, a TASC II A lesion is treated from only one access site by employing a crossover approach. The advantage of this technique is that both the right and left lesions can be treated through a single-access site, minimizing access site complications.

4.2. TASC II B lesion

Case.3: EIA stenosis between 3 and 10 cm not extending into the CFA (TASC II B) could be treated by an endovascular technique (**Figure 16A**). In cases of EIA lesions, either an antegrade crossover approach or retrograde sheath approach is taken. Selecting the approach site depends on the distance from the lesion to the CFA and/or configuration of the terminal

aorta when unable to use a crossover technique. When both approaches are not feasible, radial or brachial access is another option. The CFA is a so-called no stenting zone. To avoid CFA stenting, angiography should include the femoral head. Intervention should be performed based on the final control angiogram (**Figure 16B** and **C**). When we see the reference angiogram, the table should not be moved until stent expansion. The antegrade approach allows the stent to be placed more accurately close to the CFA as compared to the retrograde approach.
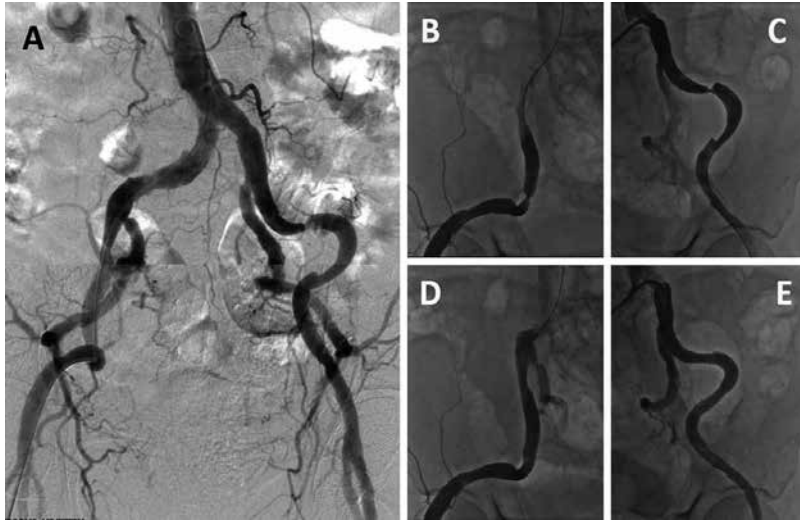
Figure 15. Bilateral short EIA lesion. (A) DSA in AP view could not reveal bilateral EIA stenosis due to severe iliac tortuosity. (B and C) Angiography in deep LAO (B) and RAO (C) can precisely reveal right EIA stenosis and left EIA stenosis, respectively. (D and E) Both lesions can be treated with stent placement using crossover technique from one access site.
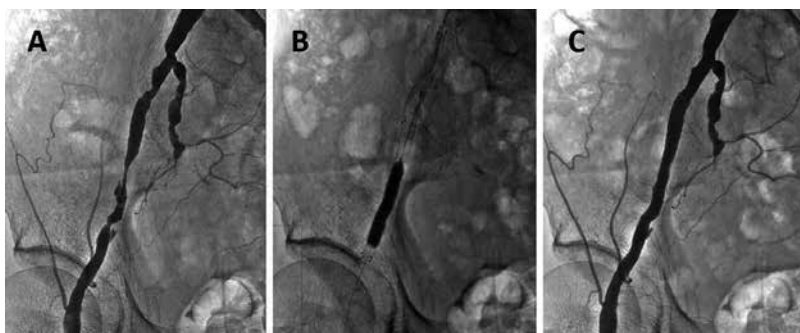
Figure 16. Multiple EIA lesions treated without changing the working view. In the crossover approach from the left CFA, angiography shows multiple stenotic lesions of the right EIA not extending to the right CFA (A). Keeping working view including the femoral head, the stent position is carefully determined so as not to implant in "no stenting zone" (B). Final result also confirmed with this view (C).

Case 4: Unilateral CIA occlusion is classified as TASC II B (**Figure 17A**). This aortography is performed from the left brachial artery and shows occlusion of the left CIA orifice. Seemingly, the total occluded part looks quite long; however, delayed-phase angiography revealed a patent external iliac artery, and only occlusion of the CIA was found (**Figure 17B**). Viewing up to the delayed phase, in particular for total occlusion, is very important to identify the actual occluded segment. The ipsilateral left CFA was punctured by echo guidance, and a 6-Fr sheath was positioned. Angiography by a 20 cm image was performed by simultaneous injection from the terminal aortography with hand injection from the left CFA sheath, more clearly revealing an image of the target lesion (**Figure 17C**). From the left CFA sheath, a 0.035-inch wire with a 5-Fr multipurpose catheter is carefully advanced with the knuckle wire technique (**Figure 17D**). After crossing the CTO lesion, a self-expandable stent was implanted, followed by postdilatation at low pressure with the same size balloon as predilatation. The final angiogram from the terminal aorta shows successful and complete recanalization of the total occlusion of the CIA (**Figure 17E**). Finally, we checked the pressure gradient between the left CFA and aorta. No pressure gradient was found across the stented segments.
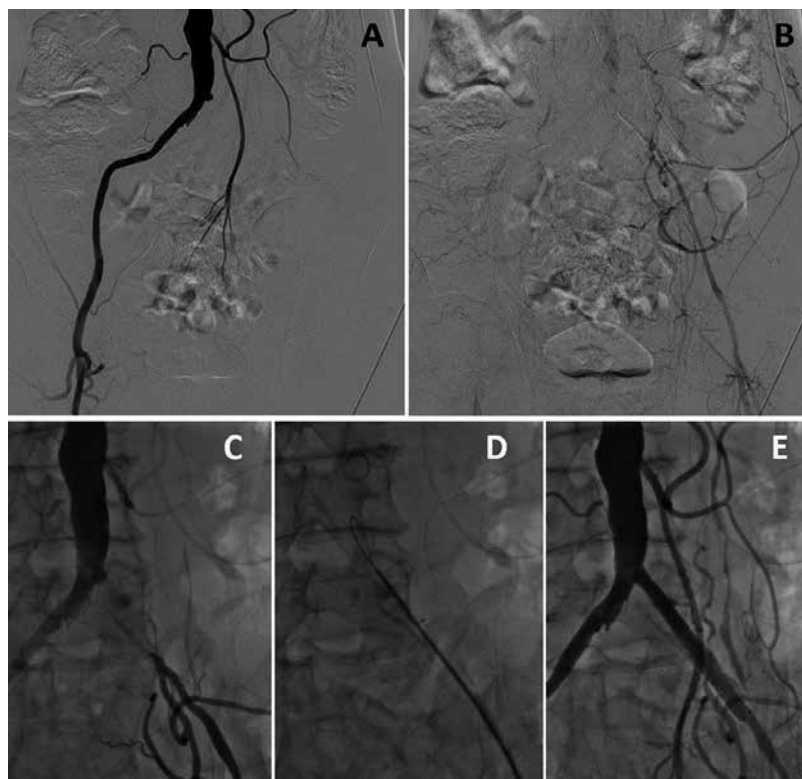
Figure 17. Left CIA occlusion. (A) Unilateral CIA occlusion is clearly found in angiogram from upper limb access. (B) Delayed phase of this angiogram reveals patent EIA supplied from collateral vessels. (C) DA (20 cm) performed by simultaneous injection from terminal aorta, while left CIA sheath reveals more precise lesion characteristics. (D) From left CFA sheath, 0.018-inch wire by knuckle wire technique supported with 5-Fr multipurpose catheter successfully used to pass the lesion. (E) Final angiography from terminal aorta shows successful recanalization.

4.3. TASC II C lesion

Case 5: Long total occlusion of the EIA with CFA involvement is classified as TASC II C which is relatively safe and can be effectively treated with endovascular therapy. For successful recanalization, angiographic work plays a crucial role. The first image to understand is the whole view of both iliac arteries with a 30 cm DSA image (**Figure 18**).
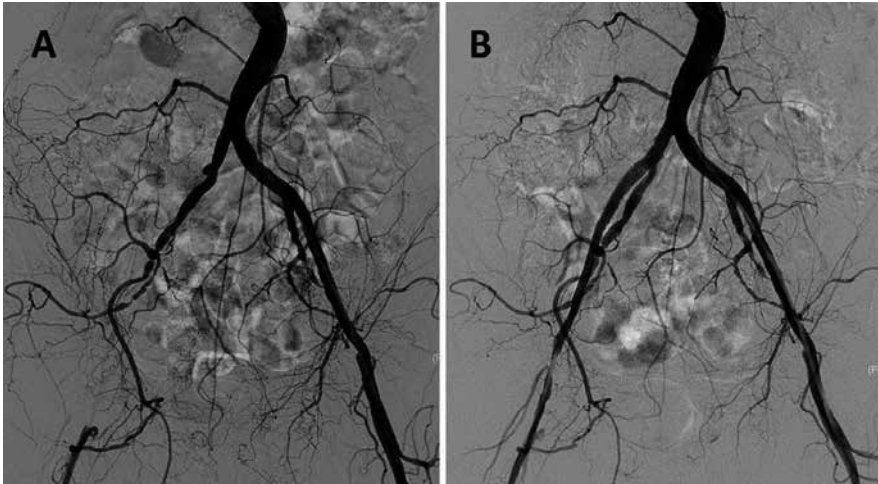
Figure 18. Long total occlusion of right EIA with involvement of CFA. (A) 30 cm DSA shows long EIA occlusion with involvement of CFA. (B) Complete recanalization is shown compared to initial diagnostic angiography.

In the delayed phase, the left CFA is visualized. After confirmation of an occluded EIA, the 20 cm contralateral DA image was positioned for working view. A 20 cm DA image in a contralateral left anterior oblique (LAO) view is actually used for interventions (**Figure 19**).

Figure 19. EVT for long total occlusion of right EIA with involvement of CFA. (A) 20 cm DA image in contralateral LAO view is actually used for interventions. (B) Final angiography is taken in working view.

For the bidirectional approach, an ipsilateral CFA puncture is performed under angiographic guidance where the 6-Fr sheath is placed (**Figure 20A**). From the crossover sheath, a 0.035-inch J-tip wire supported with a 5-Fr diagnostic catheter is advanced by antegrade access with the knuckle wire technique through the lesion (**Figure 20B**). Generally speaking, a bidirectional approach has a higher chance of wire crossing than a one way approach. When the antegrade wire fails to cross the lesion, retrograde wiring is the next step. After successfully crossing the occlusion, the wire position should constantly be monitored to see that it is in the distal true lumen. Predilatation should be performed by an undersized balloon, and a self-expandable stent should be placed. Careful attention is necessary to confirm that the distal stent does not extend to the CFA (**Figure 20C**). Adjunctive postdilatation by balloon at lower pressure was performed and the completion of angiography achieved.

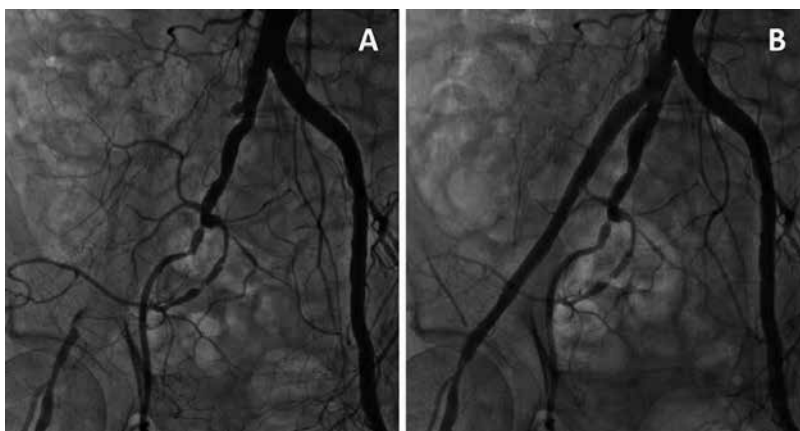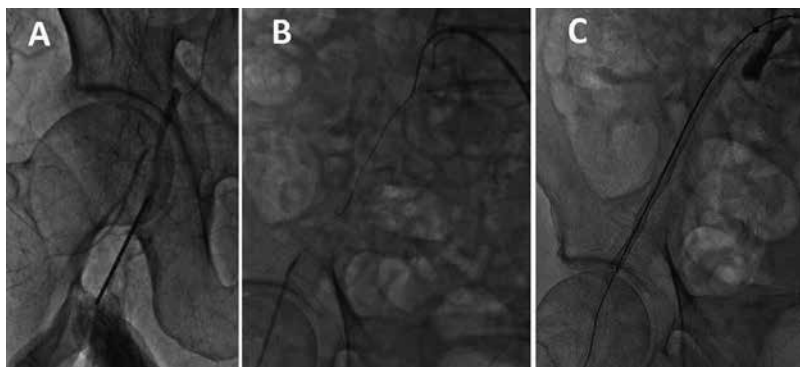**Figure 20.** Long total occlusion of right EIA with involvement of CFA. (A) Ipsilateral CFA puncture is performed under angiographic guidance for bidirectional approach. (B) 0.035-inch J-tip wire supported with 5-Fr diagnostic catheter advanced with knuckle wire technique from the crossover sheath. (C) Self-expanded stent is carefully implanted not to cover the CFA.

5. Approach to complex iliac artery disease

Advanced atherosclerotic disease of the iliac artery oftentimes involves an abdominal aortic aneurysm or extensive aortic bifurcation with or without unilateral or bilateral long total occlusions of the iliac artery. Most cases are classified as TASC II D, and in some cases, no TASC classification can be applied. Moreover, patients with these complex iliac artery diseases are commonly not candidates for surgical revascularization due to comorbidities. These patients benefit by an endovascular approach by employing various tools or angiographic techniques. In some medical centers, endovascular therapy for TASC II D is considered to be first line revascularization with safe and effective treatment by experienced interventionists [23–25]. When total occlusion is involved, aortography should be performed up to the delayed phase in which the distal patent flow could be visualized through the collateral flow (**Figure 21**). Additionally, angiography taken from multiple angles is important to navigate the guidewire correctly.

Figure 21. Aortography of aortic occlusion in the early phase (A), middle phase (B), and delayed phase (C), which can visualize distal arterial flow via collateral vessels.

Case 6: This patient has right EIA occlusion and left severe EIA stenosis. Both the right EIA occlusion and left EIA stenosis could be treated with a contralateral crossover technique from left CFA access. Both EIA lesions were successfully treated 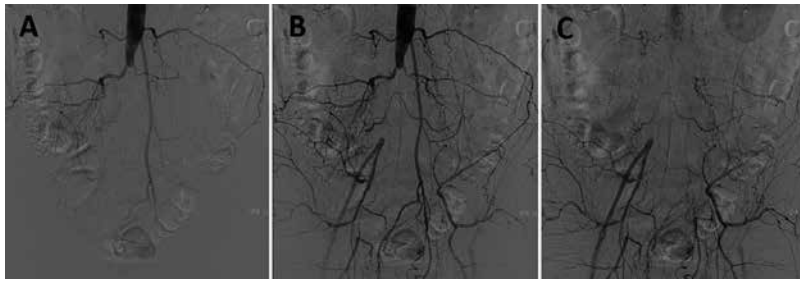by only the left anterior oblique (LAO) projection as the working image. First, aortography revealed right EIA total occlusion and left EIA stenosis (**Figure 22A**). In the delayed phase, the right CFA is slightly visible (**Figure 22B**). **Figure 22C** is the angiogram for simultaneous injection from the terminal aorta and right CFA sheath in the LAO projection. In this image, the occluded lesion can be visualized precisely. From the left CFA, a 6-Fr crossover sheath is advanced to the right CIA after a 0.035-inch J-tip Radifocus wire is used to cross the right internal iliac artery. The 0.035-inch J-tip wire with a 4-Fr support catheter could cross the occluded EIA to the right superficial femoral artery (SFA) (**Figure 22D**). Predilatation by a balloon was performed followed by the implantation of a self-expandable stent (**Figure 22E**). Postdilatation was performed by the same balloon and successful recanalization was confirmed by angiography from the crossover sheath. After opening the occluded right EIA, stenting and ballooning were performed for the left EIA stenosis (**Figure 22F**) and the final angiogram showed complete revascularization of both EIA lesions (**Figure 22G**).

Case 7: In **Figure 23A**, aortography shows an infra-renal aortic aneurysm, heavily calcified stenosis of the right CIA, and moderate stenosis of the left CIA. From the right CFA, a 0.035-inch J-tip wire was advanced, however, this wire could not cross the right CIA stenosis and was exchanged to a 0.014-inch wire. This 0.014-inch wire succeeded in crossing the lesion. The 4-Fr diagnostic catheter was gently advanced through the aneurysm, and the 0.014-inch wire was then exchanged to a 0.035-inch spring wire. After predilatation by a balloon at the right CIA, kissing stenting with a self-expandable stent was performed (**Figure 23B** and **C**). The final angiography in 20 cm DSA shows excellent results (**Figure 23D**) and pressures at both CFA sheaths showed equalized systemic arterial pressure.

Case 8: Extensive heavily calcified stenosis from the aortic bifurcation to both iliac arteries is treated by endovascular techniques (**Figure 24**). In a heavily calcified lesion, 3D CTA is not suited for diagnosis of the stenosis (**Figure 24D**).

Compared to angiography, these 3D CTA images are completely different. Patients with heavily calcified lesions are at high risk for open surgical treatment, especially those on

hemodialysis, and endovascular reconstruction is often required. In this case, an upper limb approach is not available due to the AV shunt and left CFA access is unfeasible because of a pulseless or indeterminable blood flow by ultrasound. Viewing the angiography from the right CFA sheath, left CFA access could be obtained. Wire crossing by a retrograde approach from the right CFA sheath was successful but failed from the left side due to heavily calcified stenosis of the left CIA (**Figure 25A**). Using a snare device to pull the wire into the left sheath, a microcatheter was advanced to the abdominal aorta (**Figure 25B**). Kissing stenting and mild postdilatation was performed, and an adjunctive overlap stent in both iliac arteries was placed (**Figure 25C** and **D**). Successful recanalization is confirmed in **Figure 25E**.
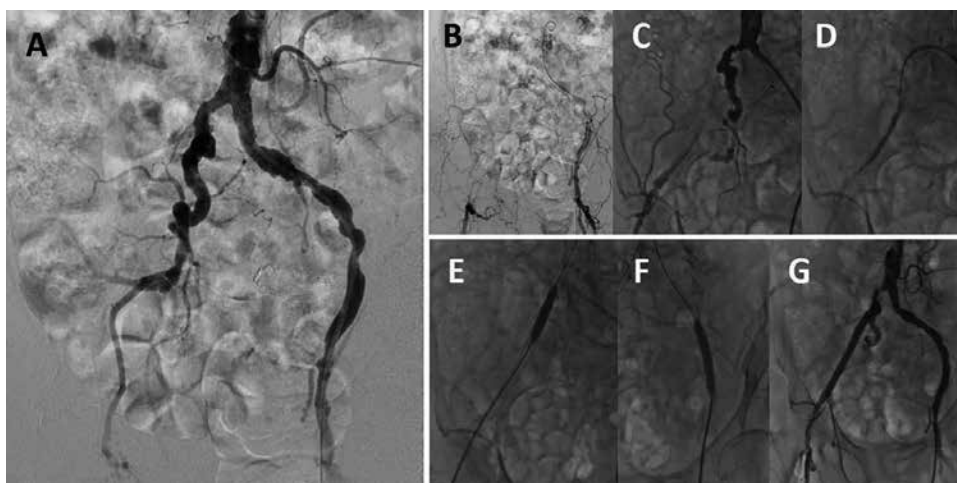
Figure 22. Right EIA occlusion with diffuse stenosis from right CIA to EIA. (A) Initial diagnostic DSA reveals right EIA total occlusion and multiple stenosis from left CIA to EIA. (B) In delayed phase, right CFA is slightly visible. (C) Simultaneous angiography from terminal aorta and right CFA sheath in deeper LAO view used for intervention, clearly revealing lesion morphology. (D) 0.018-inch wire with 4-Fr support catheter could cross the occluded EIA. (E) Stenting for distal EIA is performed from crossover sheath and, for proximal CIA, stenting is performed from retrograde ipsilateral CFA sheath. (F) After treatment for right iliac lesion, left side lesions are treated with stenting and ballooning. (G) Final angiogram shows complete revascularization of both CIA and EIA.
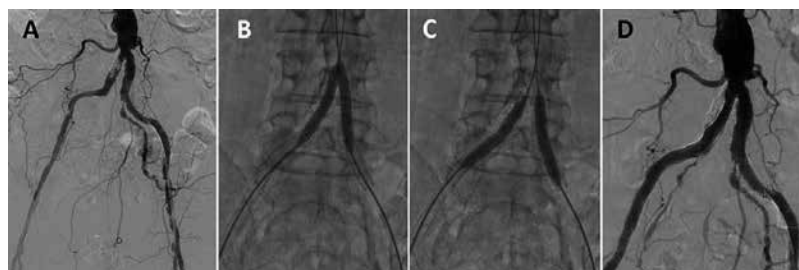
Figure 23. CIA stenosis with severe calcification and abdominal aortic aneurysm. (A) Aortography shows infra-renal aortic aneurysm, heavily calcified stenosis of right CIA, and moderate stenosis of left CIA. (B and C) Kissing stenting at aortic bifurcation (B) and postdilatation of both CIA (C) are carefully performed due to heavily calcified lesion. (D) Final angiography taken in DSA and 20 cm image shows good expansion and no complications such as rupture, dissection, or embolism.
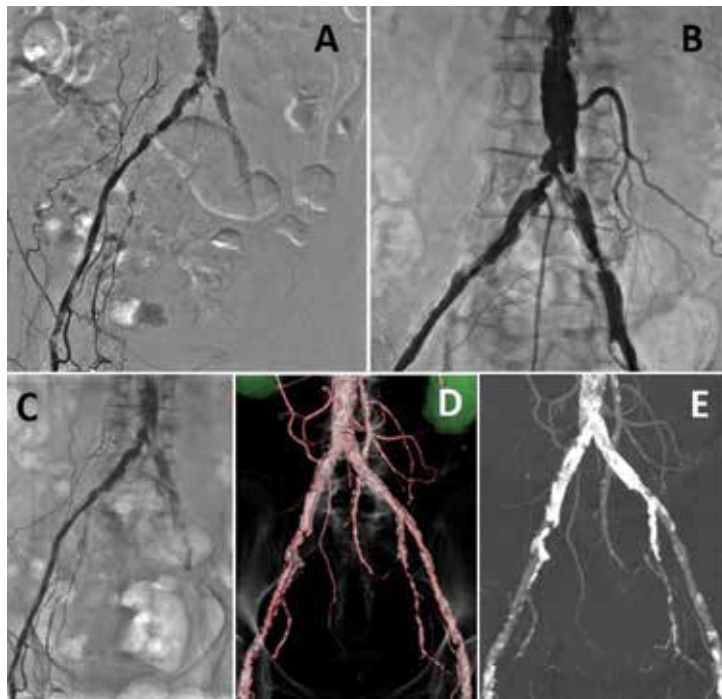
Figure 24. Heavily calcified aortic bifurcation disease involving orifices of bilateral CIA. (A) Extensive heavily calcified stenosis from aortic bifurcation to both CIAs is shown in 30 cm DSA. (B) More precise information of lesion can be obtained from 20 cm DA. (C–E) In case with severe calcification, CT angiogram is not suited as diagnostic or assessment tool since details of intravascular conditions due to calcification not visualized.
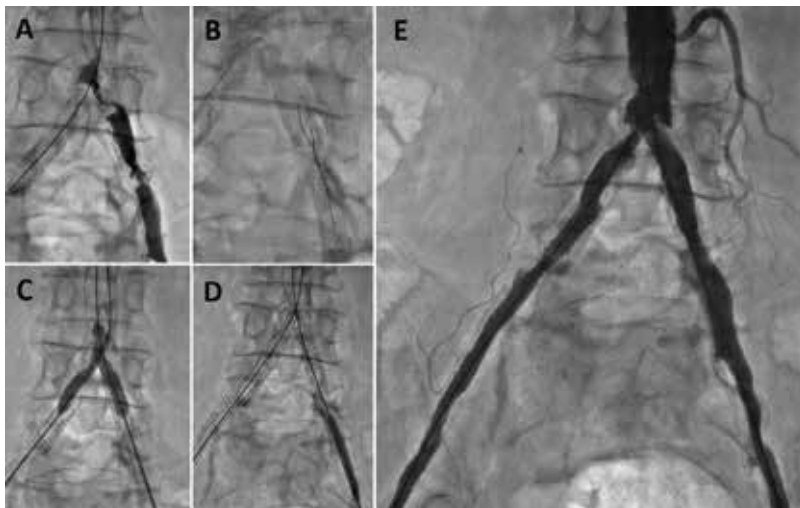
Figure 25. Heavily calcified aortic bifurcation disease involving orifices of bilateral CIA. (A) Wire crossing by retrograde from right CFA sheath is successful but due to heavily calcified stenosis of left CIA, unable to cross the wire. (B) Snare device pulls the wire from right iliac artery into the left CFA sheath. (C) Kissing stenting and mild postdilatation are performed at aortic bifurcation. (D) Adjunctive overlapping stents placed in both iliac arteries. (E) Successful recanalization is clearly visualized in final angiography.

6. Complications related to aortoiliac artery intervention

Complications in iliac artery intervention, especially bleeding due to perforation, are the most serious. Iliac artery perforation immediately causes hemorrhagic shock which may result in cardiac arrest. The iliac artery runs in the posterior abdominal cavity, and therefore, bleeding cannot be controlled with manual compression to establish hemostasis. Too much time is required to convert to open surgical repair so this is not a practical solution. Immediate endovascular repair should be attempted, and a covered stent should be positioned at the perforation site [26].

Wire perforation of the EIA is shown in **Figure 26A**. From another angle, the perforation site could not be detected (**Figure 26B**). Extravasation is clearly visualized in DSA. When suspecting perforation, DSA should be taken in several projections. The covered stent should be prepared at the same time as balloon inflation is being carried out to minimize bleeding (**Figure 26C**). After implantation of the covered stent, extravasation is not seen and complete shielding has succeeded (**Figure 26D**). To repair a perforation, aneurysm, or the other vessel injury, the covered stent is the most useful device and should be prepared along with the appropriate catheter.

Figure 26. Wire perforation and extravasation. (A) Wire perforation of EIA is clearly detected in DSA. (B) In another angle view, perforation site could not be detected. (C) Balloon inflation can minimize bleeding. (D) After implantation of covered stent, extravasation is not seen in DSA.

Dissection caused by intervention may cause acute occlusion or stop the flow to the distal vessel. In **Figure 27A**, the large dissection is caused by sheath placement due to a tortuous external iliac artery. This dissection was successfully wired to the true lumen and treated by self-expandable stent placement (**Figure 27B**). The dissection could be commonly repaired by a self-expandable stent to support the dissected flap to the vessel wall. After crossing the true lumen, we preferred a 0.014-inch wire since it is more atraumatic compared to a 0.035-inch wire.

In dealing with an aortoiliac artery bifurcation lesion with either ballooning or stenting, the potential complication is plaque shift. Therefore, a kissing stent or balloon technique has been widely employed. **Figure 28A** and **B** is typical images of plaque shift at the aortoiliac bifurcation. A large plaque shifted to the contralateral iliac artery where acute occlusion may cause acute limb ischemia on the contralateral limb. Therefore, precise information on the aortoiliac bifurcation lesion is essential, especially on the inward position of the plaque and/or large plaque burden by various different angled views before interventions.

Figure 27. The large dissection caused by sheath. (A) Large dissection by sheath with flap is found in DA. (B) Complete repair is achieved with stent implantation.
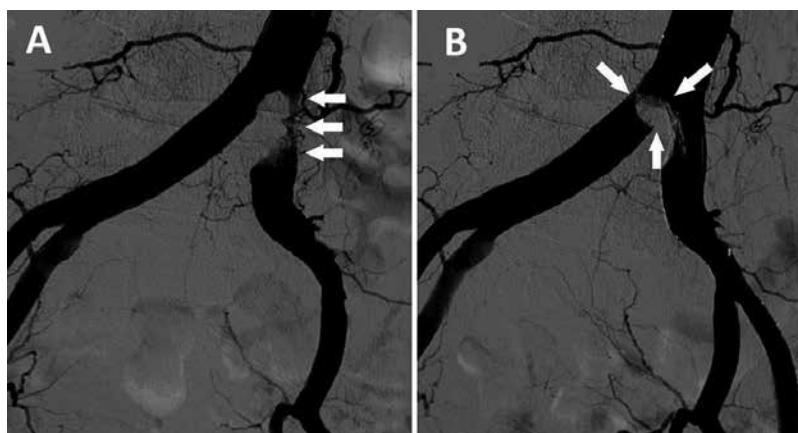
Figure 28. Unfavorable plaque shift. Right CIA stenosis with large burden of plaque (A; white arrow) is shifted to contralateral left CIA (B; white arrow) after ballooning (Special Courtesy by Dr. Yoshito Kadoya).

Recently, transcatheter aortic valve replacement (TAVR) has emerged as a promising procedure, and a transfemoral approach is the standard technique. However, vascular complications, in particular iliac artery injury, are the major clinical problem [27]. A large profile sheath in TAVR often injures the iliac artery. The DSA image clearly visualizes the injured EIA (**Figure 29A**). This complication can immediately cause catastrophic bleeding and shock. In this case, a 0.035-inch wire is carefully advanced from the ipsilateral CFA sheath for TAVR and successfully used to cross the ruptured site of the EIA followed by deployment of a covered stent (**Figure 29B**). Complete and successful repair could be achieved. The final angiogram by DSA showed no extravasation and established a TIMI 3 flow (**Figure 29C**).
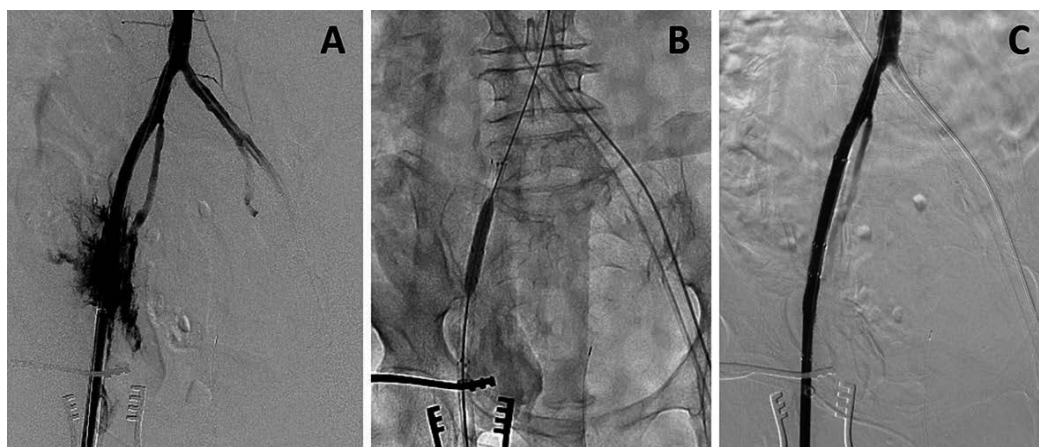
Figure 29. Ruptured EIA in TAVR. (A) DSA image can clearly visualize the ruptured EIA caused by large profile sheath for TAVR. (B) 0.035-inch wire was carefully advanced from ipsilateral sheath for TAVR and successfully crossed the ruptured site of EIA followed by deployment covered stent. (C) Successful endovascular repair is completely achieved. The final angiogram by DSA showed no extravasation and established TIMI 3 flow.

7. Conclusion

No randomized controlled trials (RCTs) have definitively established the magnitude and durability of the benefit of open surgical vs endovascular strategies. However, there has been an increase in the adoption of the endovascular first strategy for even the most complex anatomies up to TASC ll D in clinical practice. In aortoiliac artery disease, a 12-month primary patency for TASC D lesions treated with stents was considered to be fairly high. Therefore, the trend for the "endovascular first" approach will not change in addition to patient preference. And more complex TASC C-D aortoiliac lesions will be treated by stent. To succeed in treating complex lesions, careful angiographic assessment is of vital importance and unless a clear image of the lesion is not obtained, complex aortoiliac artery lesions cannot be revascularized, resulting in high complication rates.

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Angiography and Endovascular Therapy for Femoropopliteal Artery Disease

Masahiko Fujihara

Additional information is available at the end of the chapter

Abstract

Femoropopliteal artery disease accounts for a significant proportion of endovascular interventions (EVTs) for peripheral artery disease (PAD) in patients with disabling claudication or chronic limb ischemia. The femoropopliteal artery starts from the common femoral artery (CFA) to the superficial femoral artery (SFA) and ends at the popliteal artery. The SFA is the longest vessel, and it is hard to visualize the entire vessel in one image. However, it is the main target for endovascular works. Before EVT procedure, full evaluation by the angiography is needed. These include anatomical variation, lesion length, lesion characteristics, calcification, and stent restenosis pattern. Endovascular approach is based on these information. The benefit of revascularization is considered to correspond to the severity of ischemia. Their assessment led to optimal endovascular strategy for femoropopliteal occlusive disease. However, to keep the patency after procedure by current endovascular approach still remains unsolved.

Keywords: femoropopliteal artery, superficial femoral artery, popliteal artery, angiography, endovascular therapy

1. Introduction

Femoropopliteal artery disease accounts for a significant proportion of endovascular interventions (EVTs) for peripheral artery disease (PAD) in patients with disabling claudication or chronic limb ischemia. PAD is commonly classified into either the Fontaine stages or Rutherford classifications [1, 2]. The femoropopliteal artery is the longest vessel and crosses two joint structures, i.e., the hip and knee joints. This vessel courses through the muscular adductor canal in the thigh, which places the artery at increased mechanical stress; specifically on the distal superficial femoral artery (SFA) and proximal popliteal artery (PPA), which are the most common anatomic locations of lower extremity atherosclerosis.

There has been a marked increase in the use of endovascular interventions in the treatment of peripheral arterial disease, with femoropopliteal interventions accounting for more than 55% of cases [3]. Stents in the femoropopliteal system have historically been associated with increased rates of stent fracture, which is related to high rates of restenosis [4]. This vascular segment has limited long-term patency rates so that the clinical value of EVT requires more investigation. Even though new technology has been introduced to femoropopliteal artery disease, it is still challenging to treat and interventions are often limited by its unique anatomic, hemodynamic, and mechanical constraints. Contrast angiography provides detailed information on the arterial anatomy and is recommended as the "gold standard" method for evaluation of patients with lower extremity PAD, especially when revascularization is contemplated. In this chapter, a basic angiographic technique for femoropopliteal artery disease is presented along with various angiographic images of pre- and postangioplasty.

2. Basic angiography for the femoropopliteal artery

The femoropopliteal artery starts from the common femoral artery (CFA) to the superficial femoral artery (SFA) and ends at the popliteal artery. The first branch is the profunda femoral artery (PFA). The SFA is the longest vessel, and it is difficult to visualize the entire vessel in one image; however, it is the main target for endovascular works.

2.1. Bifurcation angiography

In the antero-posterior (AP) view, the profunda femoris (PFA) overrides the SFA (**Figure 1**).

In the proximal SFA, the initial angiography should be taken by an ipsilateral view (**Figure 2**).

In this angled view, the clear separation between the left SFA and left PFA was made by the left anterior oblique (LAO) view. In **Figures 1** and **2**, the same two images are shown. In the digital subtraction image (DSA), the background is not clearly visible so that the bifurcation point is difficult to identify. In contrast, digital angiography (DA) shows the background so that we could understand where the bifurcation point starts at the common femoral head. This angled view is of particular importance in visualizing a diseased proximal femoral artery or diseased deep femoral artery (DFA) (**Figure 3**).

2.2. Anatomy of the CFA in relation to the femoral head

In this angled view, we could locate the level of the bifurcation point at the common femoral head. In this angiography, whether the DFA shows high or low take-off needs to be evaluated. In **Figure 4A** and **B**, the DFA arises from the middle femoral head. In these cases, careful attention is mandatory before a puncture to the CFA by either a retrograde or antegrade approach.

According to a report by Ho-Young Ahn et al. [5] (**Figure 5**), the proportion of cases in which the location of femoral artery bifurcation was above the center of the femoral head was 4.59%, and the proportion of cases in which the location of femoral artery bifurcation was in zone 3 was 10.1%, zone 4 was 36.7%, and zone 5 in which bifurcation is below the femoral head was 48.6% (**Figure 6**).
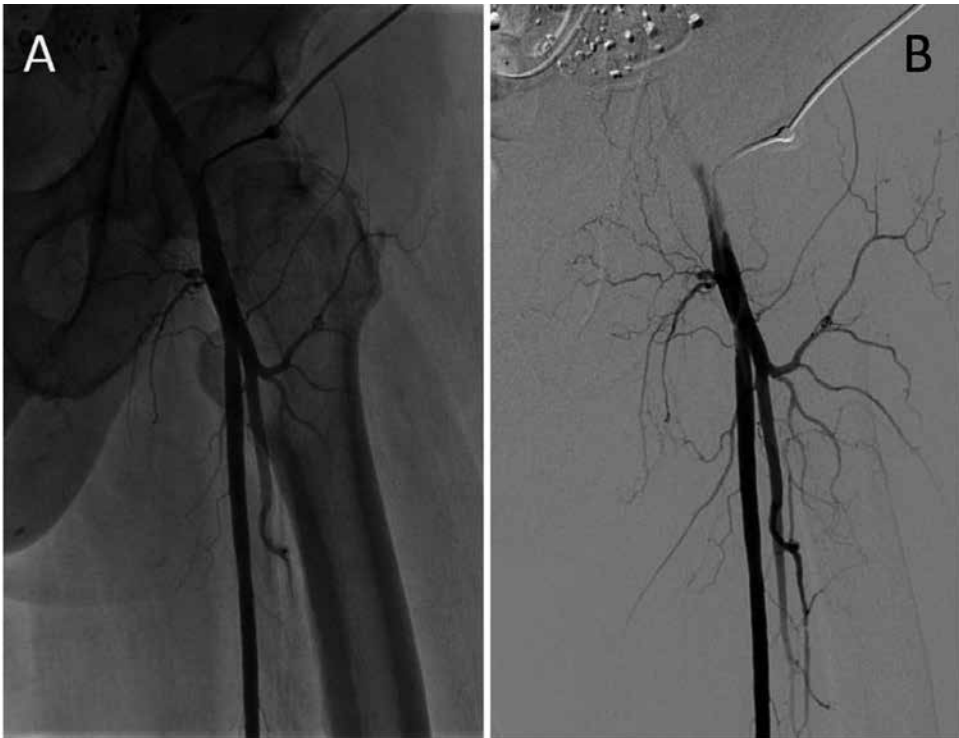
Figure 1. Anteroposterior view of proximal left femoral artery by (A) DA image; (B) DSA.
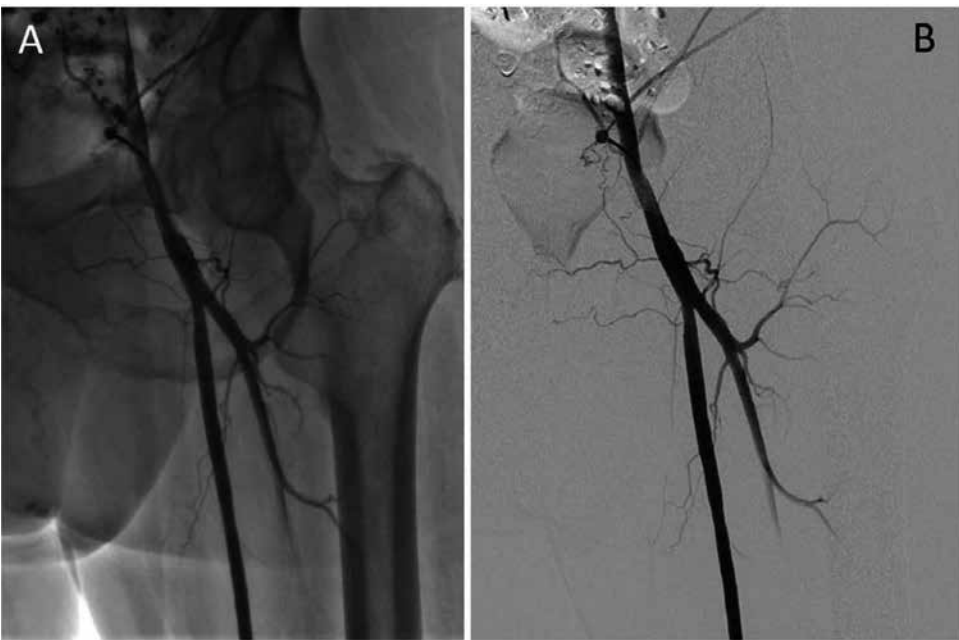
Figure 2. Left anterior oblique view of proximal left femoral artery by (A) DA image; (B) DSA.
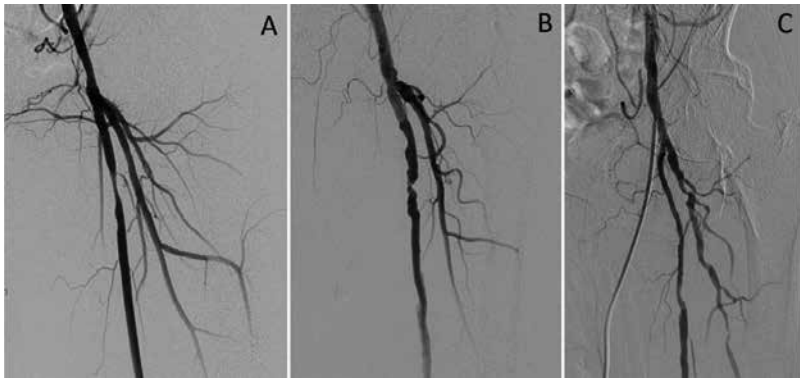
Figure 3. Left anterior oblique view for proximal left femoral diseased artery. (A) Proximal focal SFA stenosis is well visualized. (B) Complex SFA disease and ostial stenosis PFA are well visualized. (C) Both SFA and PFA are diffusely diseased.

Figure 4. High take-off of profunda artery. (A) Located in zone 3. (B) Located in zone 4.
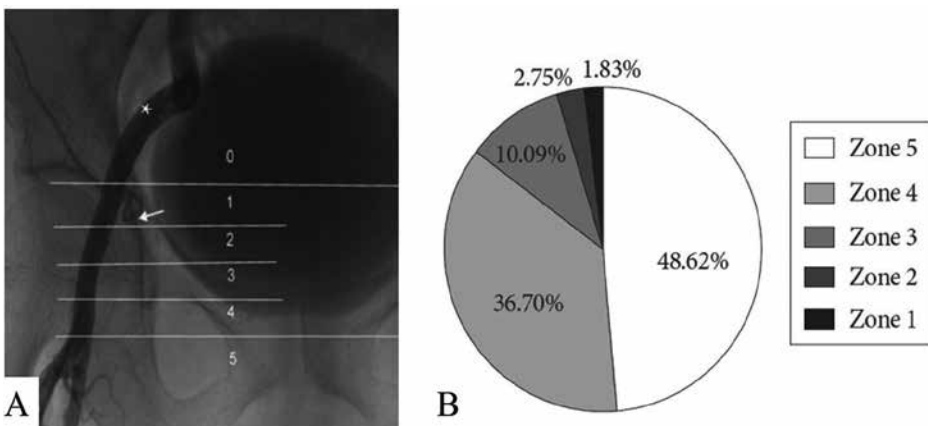
Figure 5. Angiographic anatomical study of the common femoral artery from Korea. (A) the definition of location for femoral artery bifurcation (B) the proportion of femoral artery bifurcation location, zone 5 in which bifurcation is below the femoral head was 48.6%.

Figure 6. Low take-off of profunda artery.

To reduce vascular complications, the bifurcation point needs to be assessed to determine the optimal puncture location. The second point is the direction of the PFA. In most cases, the PFA is directed outward, but in some cases, the PFA goes inward then turns outward. This is an anomaly of the deep femoral artery bifurcation (**Figure 7**). Accurate knowledge of such anatomical variations regarding the origin of the PFA and femoral circumflex arteries is essential for clinicians. Information on the precise anatomy of the PFA will provide the foundation for assessments to minimize puncture-related complications.

2.3. Middle to distal SFA

From the mid to distal SFA portion, the angiographic view is the antero-posterior (AP) view (**Figure 8**).
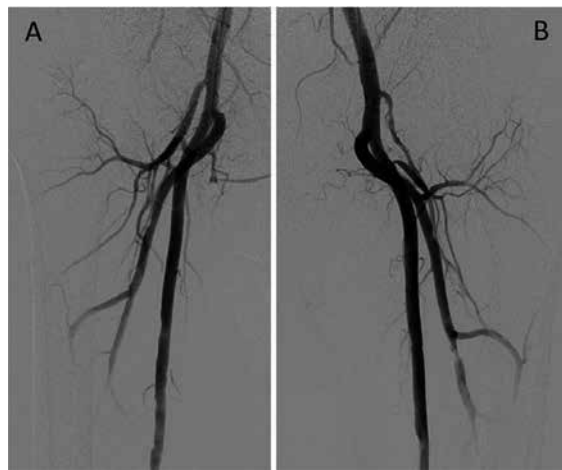
Figure 7. Variations of the profunda femoral artery. Both right (A) and left (B) deep femoral artery directed medially with separate origin of lateral circumflex artery.

Figure 8. Anteroposterior view for middle SFA with a DSA image.

Distal SFA and popliteal artery angiography may require an angled view (**Figure 9**) since it runs posterior to the knee joint (**Figure 10**). Bolus chasing (running table) is a commonly employed method to visualize a long segment of the femoropopliteal to below the knee artery. In some angiographic systems, this angiography can be performed by digital subtraction with a small amount of contrast.

Figure 9. (A) Left anterior oblique view for distal to popliteal disease. (B) Anteroposterior view.

Figure 10. Anteroposterior view of left popliteal artery. (A) Left popliteal artery clearly shown by DSA image. (B) Due to boney structure overriding the popliteal artery, DA image has limitations in visualizing arterial edge and other branches.

We usually inject 4 cc/second for a total 16 cc of contrast to enhance visualization from the common femoral to the distal tibial artery (**Figure 11**).

2.4. Popliteal artery

The popliteal artery is a continuation of the distal SFA and courses through the popliteal fossa. It is located in the knee and back of the leg. Due to the bony structure of the knee joint, the popliteal artery is well visualized by DSA. In **Figure 12**, the DA image does not reveal any branches.

The popliteal artery segments are defined as follows: P1 segment, from the intercondylar fossa to proximal edge of the patella; P2 segment, from the proximal part of the patella to the center of the knee joint space; and P3 segment (below knee popliteal artery), from the center of the knee joint space to the origin of the anterior tibial artery (**Figure 13**) [6]. In the DSA image, the right superior lateral genicular artery and inferior lateral genicular artery are well visualized.
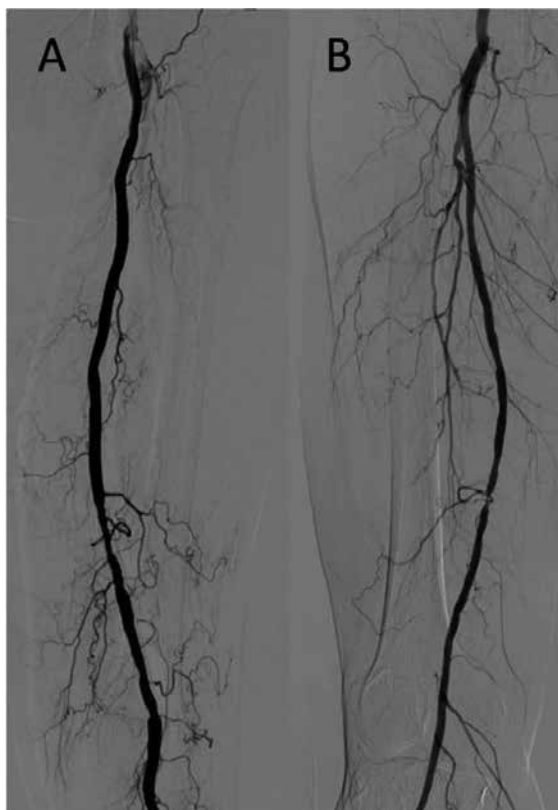

Figure 11. Bolus chase limb vessel angiography in DSA image. To see the whole femoropopliteal artery, bolus chasing is a very useful method. Angiogram can be obtained by digital subtraction with small amount of dye.
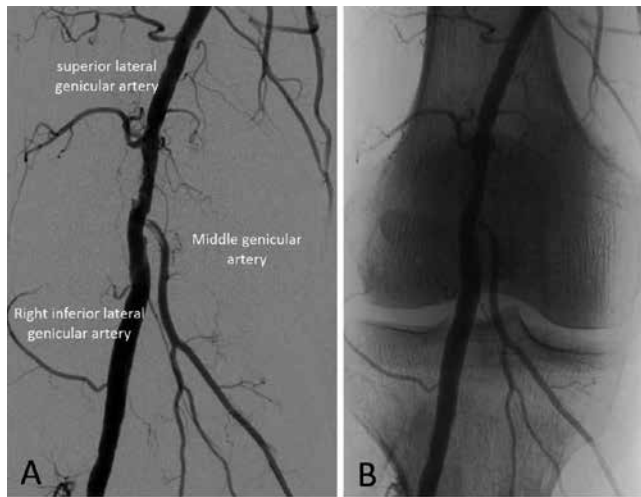
Figure 12. Anatomy of popliteal artery by (A) DSA image and (B) DA image.

Figure 13. Definition of popliteal artery segments.

3. Common femoral artery disease

Most CFA diseases are localized short lesions and have coral-like calcifications (**Figure 14**).

The ideal angiographic image is provided by an ipsilateral view. This image shows the circumflex iliac artery and deep femoral artery bifurcation (**Figure 15**).

The CFA is the so-called nonstenting zone and stent implantation should be avoided due to issues regarding long-term durability and stent fracture. There are some cases in which acceptable results with long-term patency could be obtained by balloon procedure alone (**Figure 16**).

However, in most cases, ballooning is insufficient in retaining patency. Standard revascularization in CFA disease should be treated by endarterectomy (**Figure 17**).

Endarterectomy is a surgical cut-down and removal of the plaque. This so-called femoral endarterectomy with or without patch angioplasty has long been the favored approach for the treatment of patients with symptomatic common femoral artery disease. On the other hand, the endovascular approach is regarded as a less effective treatment strategy for CFA stenosis/occlusion. The plaque in the CFA is often bulky, eccentric, and heavily calcified and may not respond well to balloon dilation. Thus, stent implantation is contraindicated in the CFA.
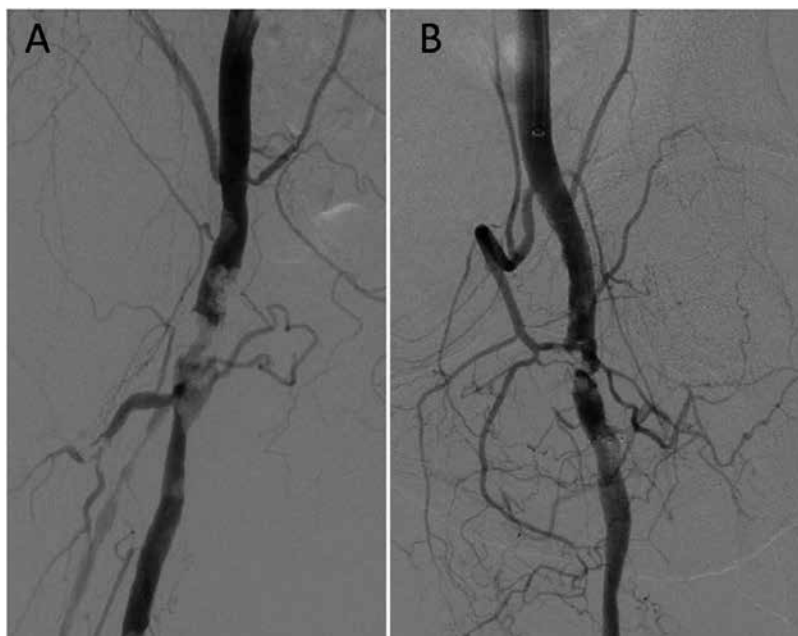
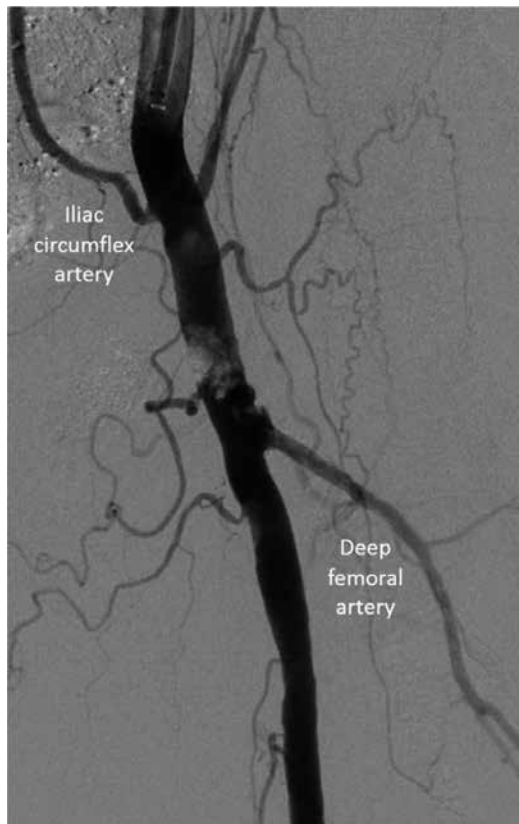**Figure 14.** CFA disease with typical coral-like calcification disease on (A) right and (B) left.

Figure 15. Identification of circumflex iliac artery and deep femoral artery bifurcation by left anterior oblique view.
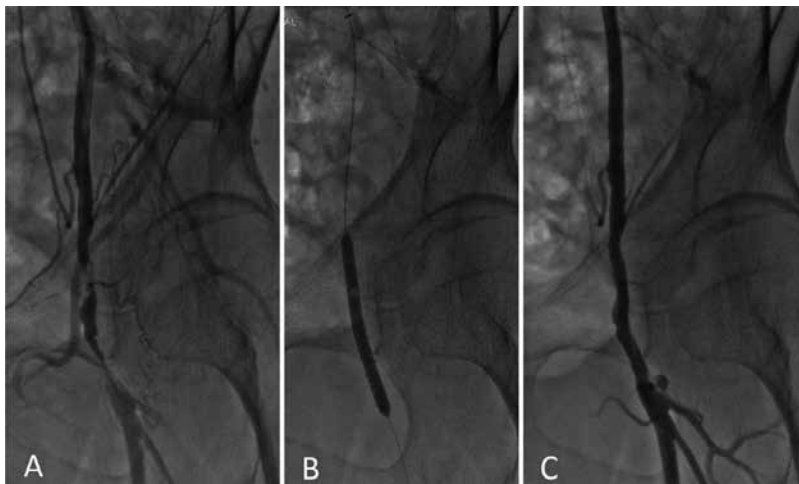

Figure 16. Balloon angioplasty for CFA lesions. (A) Preprocedure shows tight stenosis of left CFA; (B) balloon angioplasty by 5 × 40 mm balloon; (C) postprocedure shows successful angioplasty with minor dissection.
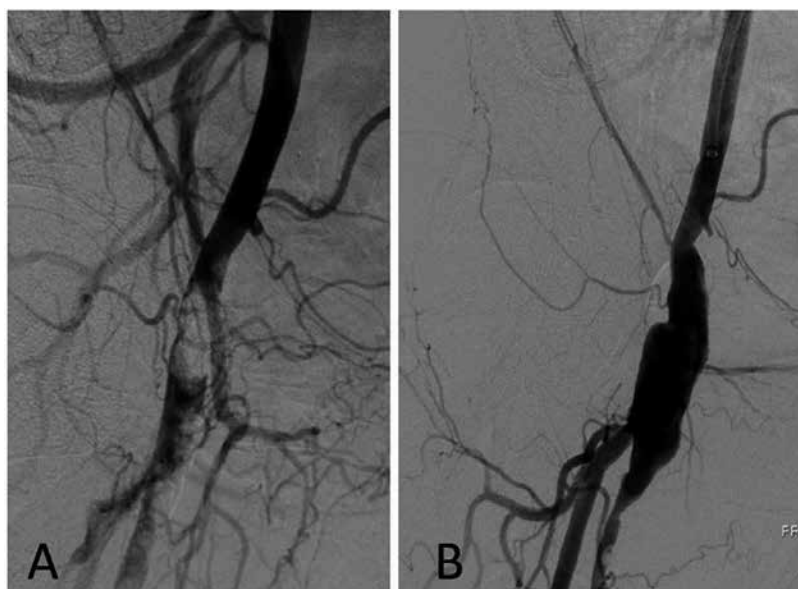
Figure 17. Endarterectomy with patch angioplasty. (A) Presurgical repair. (B) Postsurgical repair.

4. Evaluation of SFA disease

The etiology of SFA disease varies depending on such factors as fibromuscular dysplasia, repetitive occupational trauma, external compression, and inflammatory disease. However, most SFA lesions are a manifestation of arteriosclerosis and its clinical symptoms are induced by stenosis and occluded lesions. Evaluation of SFA disease includes lesion length, morphology, location, calcification, and pattern of restenosis in cases of reintervention.

4.1. Lesion length measurements

Lesion length and morphology can be classified according to the TASC II guidelines [7]. It provides the standard indications of either interventional treatment or bypass surgery. **Figure 18A** shows a single focal lesion of less than 10 cm in length and did not involve SFA origins. This lesion is classified into type A. **Figure 18B** is a single chronic total occlusion (CTO) lesion of less than 15 cm and considered to be type B. **Figure 18C** shows multiple stenosis of more than 15 cm lesion length and a typical example of type C. **Figure 18D** shows the long CTO and the lesion length is more than 20 cm. This is a typical type D lesion and stenting for this complex type of lesion involves a high risk of restenosis.

For lesion length measurements, a tape measure is attached to the front of the thigh (**Figure 19**).

Figure 18. Focal lesion was measured at 1 cm (A). Short CTO was measured at 10 cm (B). Short CTO and stenosis measured at over 15 cm (C). SFA long CTO was measured at 20 cm (D).

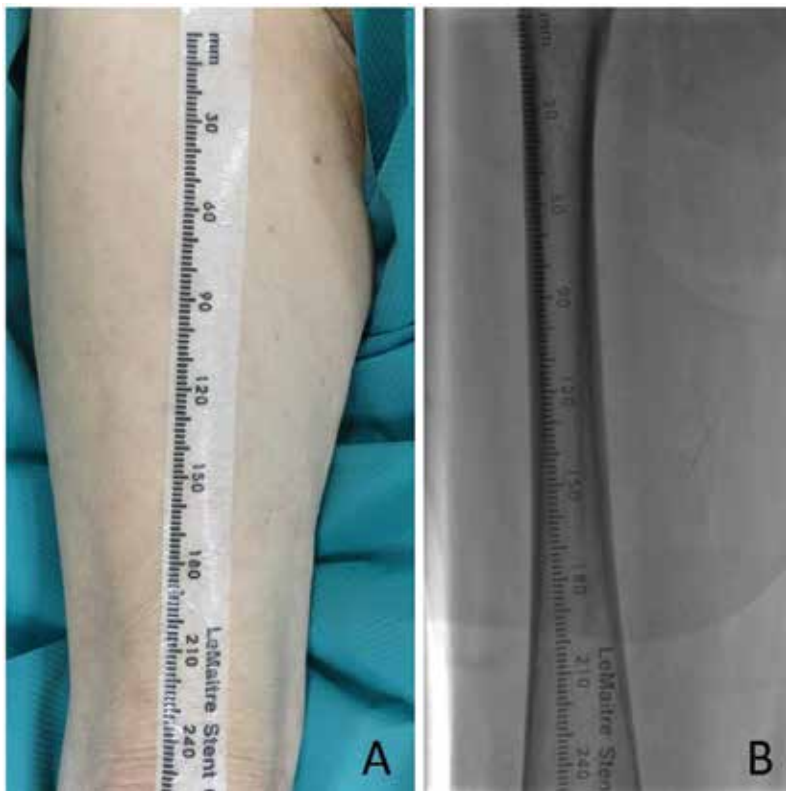**Figure 19.** For lesion length measurement, a tape measure is attached to the front of the thigh.

4.2. Lesion location

- Proximal segment: the feasibility of intervention requires assessment not only by TASC ll classifications alone [7]. In a SFA CTO lesion, the initial entry point needs to be clarified. Does the SFA occlusion begin with a proximal stump (**Figure 20A** and **B**) or not (**Figure 20C** and **D**)? This is a key factor for successful wiring in a CTO lesion.

- Distal segment: a distal portion of the SFA lesion needs to be visualized (**Figure 21**).

If antegrade wiring should fail, we may change to a retrograde approach. If we plan to puncture at the distal SFA, the exact location must be clearly identified. To puncture at the distal SFA, the lesion must be collateralized above the popliteal artery (**Figure 22**). If popliteal artery disease is involved, it is contraindicated to puncture at the distal SFA or popliteal artery for the retrograde approach.
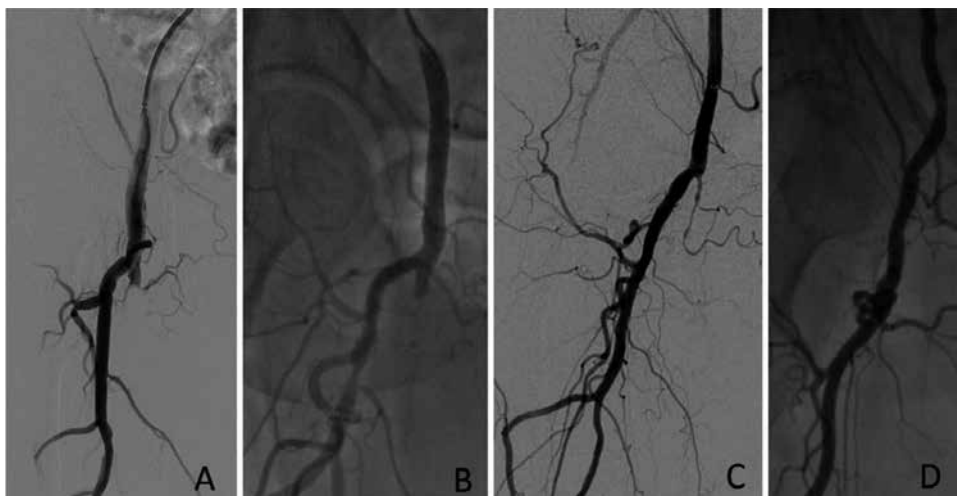
Figure 20. In right anterior oblique view, the stump of SFA ostium occlusion is well visualized by (A) DSA image; (B) DA the stump of SFA ostium occlusion is not well visualized; (C) DSA image; (D) DA.

4.3. Evaluation of calcification

Most endovascular devices are unable to cope with calcified lesions and there is no ideal solution for the treatment of severely calcified lesions. Calcification on the SFA is associated with increased cardiovascular morbidity and mortality [8]. There are two types of calcification, i.e., intimal calcification and media calcification. The first type is associated with stenosis and/or obstruction of the vessel, while the second type is associated with arterial stiffness, increased pulse pressure, and increased cardiac overload. However, current modalities for assessing calcified lesions are limited. From a practical viewpoint, we use only plain X-P and intravascular ultrasound (IVUS) to distinguish between intimal and media calcification in nonoverlapping areas of the vessels. There are two proposed definitions of a calcified lesion. One is the proposed peripheral arterial calcium scoring system (PACSS) diagnosis by

Figure 21. Distal portion of SFA lesion (A) uninvolved popliteal artery and (B) involved proximal popliteal artery.

Figure 22. Puncture of distal SFA visualized from collateral flow from PFA.

fluoroscopy [9]. The cut-off value of a severely calcified lesion is defined as more than 5 cm in length. Moreover, a determination of either unilateral or bilateral calcification along with a separation of intimal or medial calcification is required. The second is the calcium burden a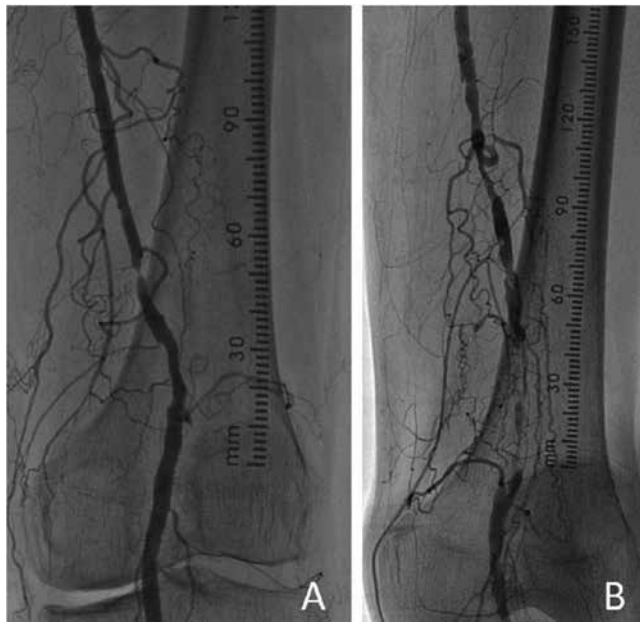ssessment and less than 3 cm or more than 3 cm has been proposed to determine lesion calcification. Along with the length, assessment of the axial view of the vessel by CT is also recommended. The circumferential distribution of calcification by plain CT is analyzed [10]. Both criteria are based on calcified lesion length and measurement of the calcified angle in the axial section. **Figure 23** shows severe calcification in a lesion evaluated by fluoroscopy. As for the calcified arc, IVUS is more practical in actual clinical situations. However, the meaning and usefulness of this modality are as yet not well known.
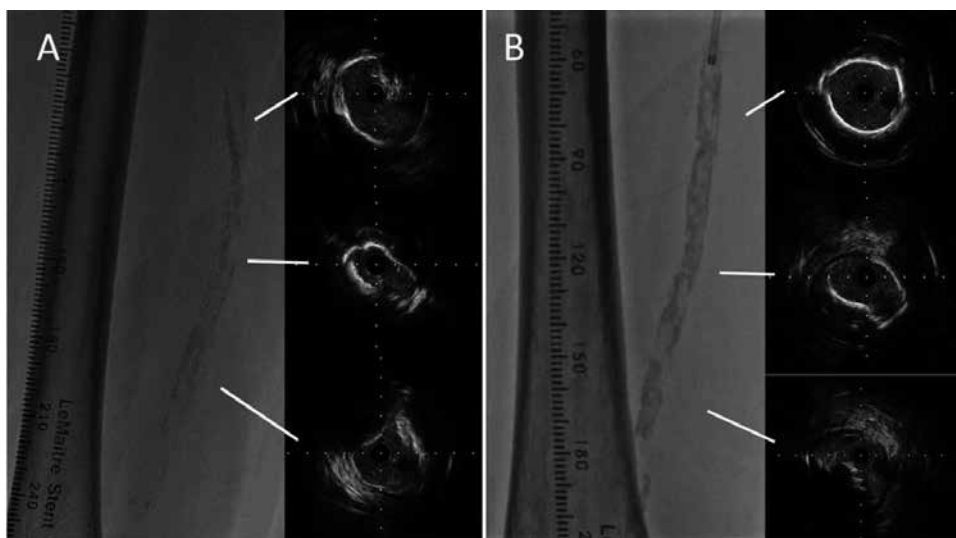
Figure 23. (A) Severe calcification of SFA in mid-portion (>15 cm) evaluated by fluoroscopy and IVUS. (B) Super severe calcification of SFA mid-portion (>10 cm) evaluated by fluoroscopy and IVUS.

4.4. Stent fracture

Over the past decade, stent technologies, which may offer improved clinical outcomes over balloon alone procedure, have been developed. This fact was proved by recent randomized trials demonstrating its superiority over simple percutaneous angioplasty. However, nitinol self-expanding stents are subject to both axial and bending deformation when implanted into the SFA, and stent fracture in SFA is a growing concern. In most cases, stent fracture is not related to restenosis (**Figure 24**).

The stent fracture is classified according to types 1–5 [11]. In a severe form of fracture, aneurysm formation was observed (**Figure 25**). The recent development of nitinol stents, which are more flexible in structure, is expected to lead to a lesser degree of stent fracture, but data for its long-term use are lacking and careful observation and assessment are still required.

Figure 24. Stent showed complete traverse liner separation without displacement. (A and B) Type 3 stent dissection; (C) no restenosis observed at fracture site.

Figure 25. Complete stent fracture at distal right SFA to proximal popliteal artery. (A) The stent showed no restenosis; however, (B) complete break at SFA distal portion with aneurysm formation.

4.5. Stent restenosis

As far as primary patency is concerned, the superiority of stents over balloon angioplasty has been evidenced by various studies. However, although nitinol stents can be implanted for primary use, stent restenosis is still a serious issue 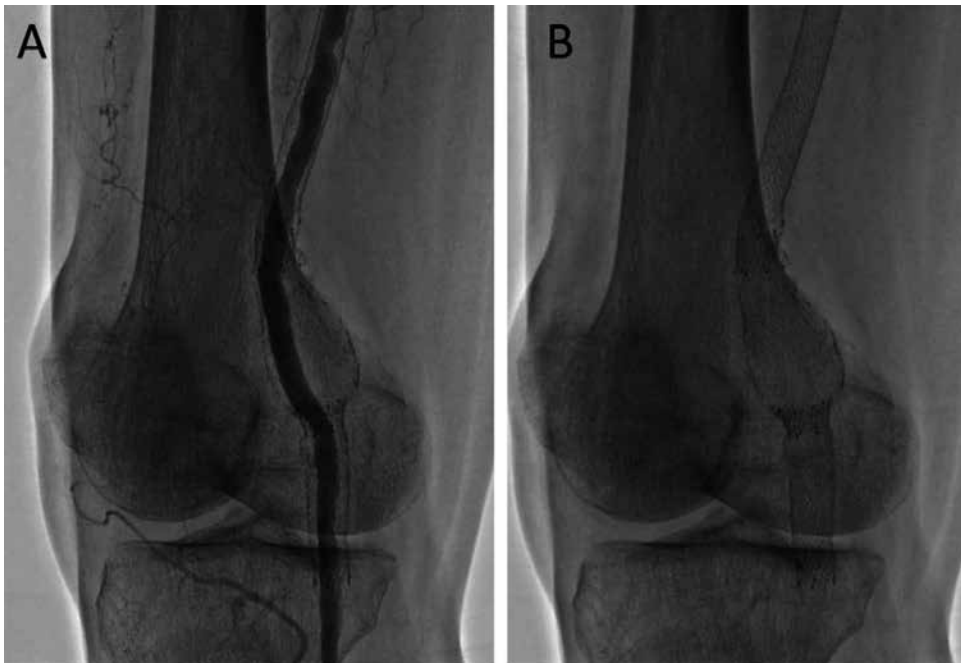in primary stenting. The SFA long CTO lesion originates from the SFA ostium, and successful recanalization was obtained by stenting. However, angiography at 6 months revealed not only SFA stent restenosis with but also new stenosis in the profound femoral artery initiated by the SFA stent (**Figure 26**).

There are three types of restenotic patterns: Class I includes focal lesions (<50 mm in length, **Figure 27A**); Class II includes diffuse lesions (>50 mm in length, **Figure 27B**); and Class III includes totally occluded in-stent restenosis (ISR) (**Figure 27C**). Restenotic patterns after FP stenting are significant predictors of recurrent ISR and occlusion [12].

Moreover, neointimal hyperplasia is the decisive factor for the restenosis of the SFA stent. However, careful observation shows another cause associated with restenosis. In **Figure 28**, no in-stent restenosis is observed but edge restenosis, showing that the stent edge initiated hyperplasia.

Figure 29 shows the case of short occlusion in the SFA (A). A successful recanalization was performed by stenting (B), but restenosis was found at the site of the original occluded segment, and an eccentric calcified lesion is seen (C). In this restenosis, the calcified lesion is the main cause of restenosis. As shown in these cases, SFA stent restenosis is multifactorial and multifaceted, accounting for the high rate of restenosis in SFA stents and no single solution at present.

Figure 26. (A) Nitinol stent restenosis. After 6 months stent implantation angiography showed SFA stent restenosis with new stenosis in profunda femoral artery. (B) This restenosis is clearly seen in nonsubtracted angiogram.
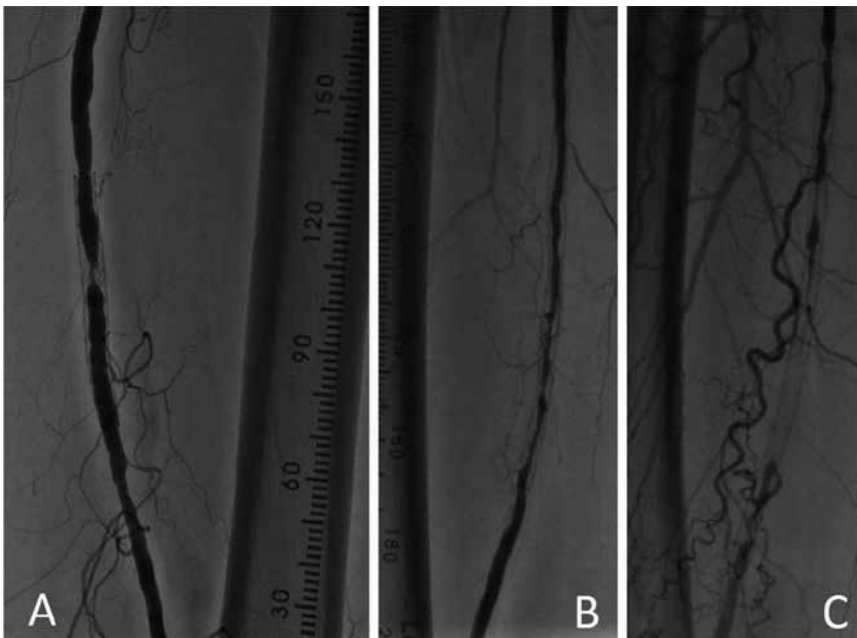
Figure 27. ISR lesions were classified by visual estimate on angiography: Tosaka class I, the focal ISR group (<50 mm in length), included lesions positioned at the stent body, stent edge, or a combination of these sites (A). Tosaka class II, the diffuse ISR group (>50 mm in length), includes not only stent body lesions but also stent edge lesions (B). Tosaka class III is the totally occluded ISR group (C).
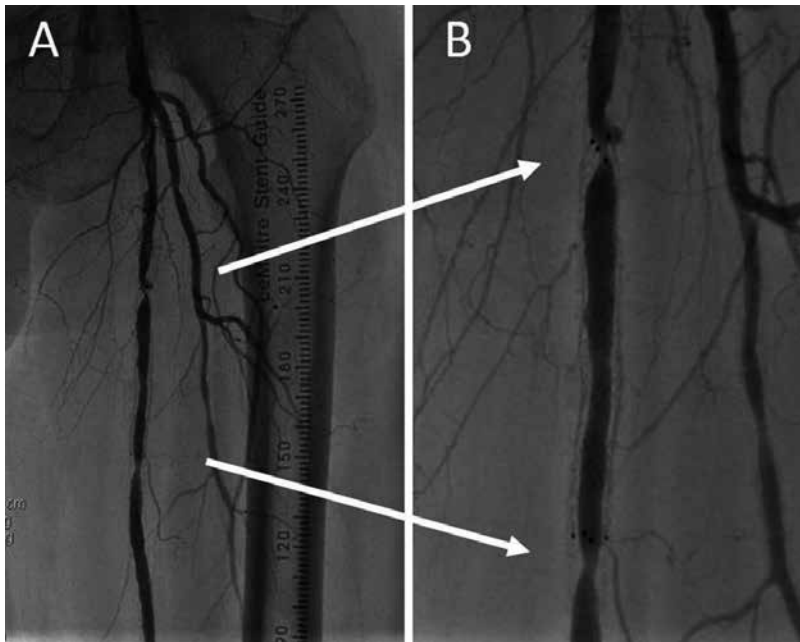
Figure 28. The case of stent edge initiated hyperplasia without in-stent restenosis.
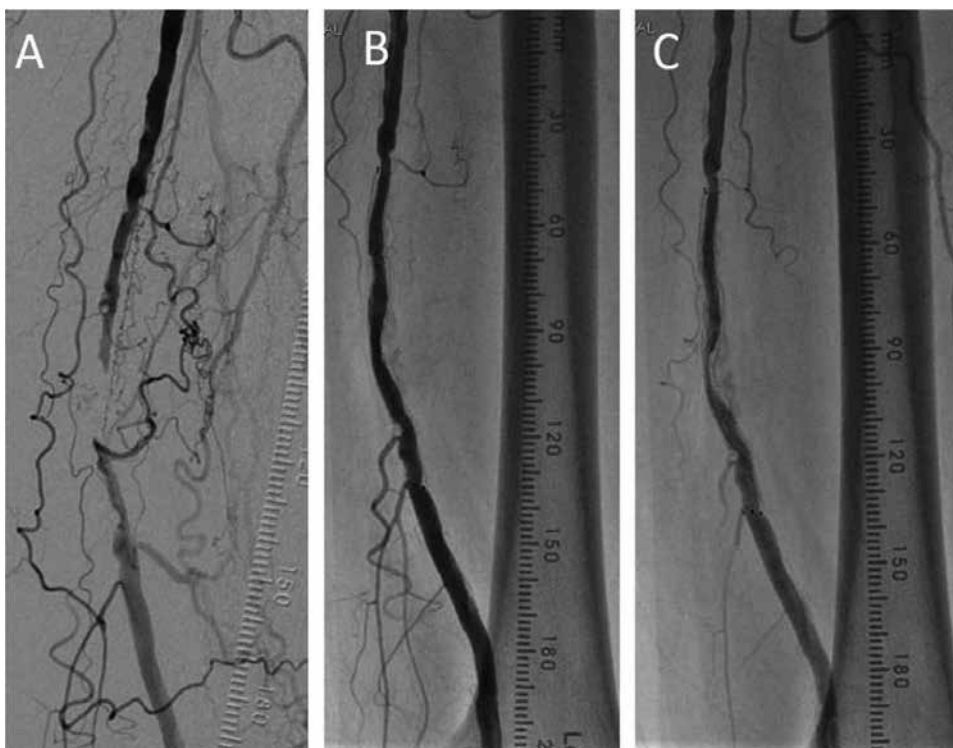
Figure 29. The case of short calcified occlusion in the SFA (A). A successful recanalization was performed by stenting (B). Restenosis was found at the site of the original occluded segment, and an eccentric calcified lesion (C).

5. Endovascular treatment for SFA disease

Endovascular therapy is indicated in the treatment of disabling claudication despite optimal medical therapy or critical limb ischemia. The benefit of revascularization is thought to correspond to the severity of ischemia or the presence of other risk factors for limb loss such as wound and infection severity. Otherwise, retaining the patency rate is yet an unsolved issue. However, there are several options in SFA recanalization (**Figure 30**).

5.1. Balloon angioplasty and nitinol stent implantation

In femoropopliteal artery disease, conventional balloon angioplasty is considered to be the standard approach for solitary lesions. However, balloon angioplasty alone does not have sustained benefits. In the 1990s, many stent trials were started. Stainless steel stents did not show acceptable long-term durability [13], but nitinol stents have shown good results in simple SFA lesions compared to conventional balloon angioplasty [14]. Based on these trials, the world trend has shifted from balloon alone to the era of nitinol stents. Thus, until recently, nitinol stent implantation was the main treatment option for SFA revascularization. However, as mentioned earlier, it became known that nitinol stents showed fracturing mid to long term and a low patency rate in complex lesions. In 2011, the Zilver PTX nitinol stent (COOK Medical,

Figure 30. Different techniques to intervene in (A) long SFA disease including (B) balloon angioplasty, (C) spot stenting with short nitinol stent, (D) full cover nitinol stent implantation, (E) stent grafting, and (F) bypass surgery.

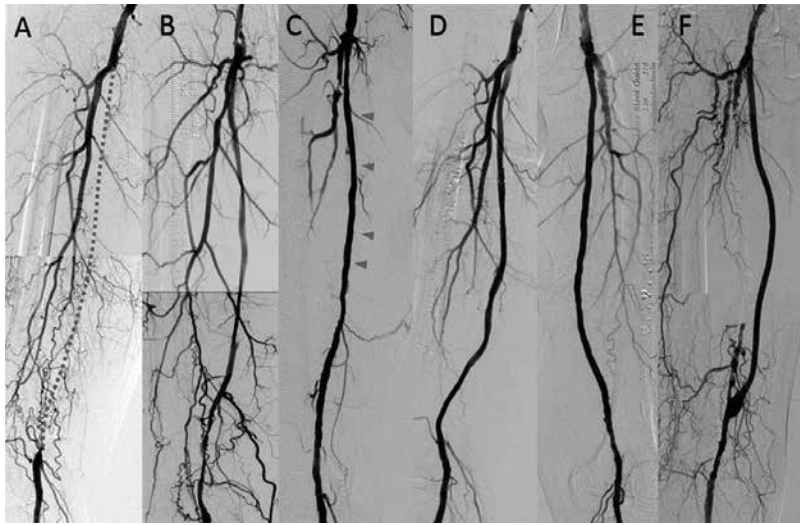Bloomington, Indiana) became the first polymer-free paclitaxel-coated nitinol stent (drug-eluting stent; DES) approved for the treatment of SFA disease. A pivotal randomized controlled trial (RCT) conducted in 2011 reported that the use of a drug-eluting stent (DES) resulted in a higher patency rate of 83.1% at 12 months over percutaneous transluminal balloon angioplasty (PTA) [15]. In short lesions, the initial approach is still balloon angioplasty (**Figure 31**).

The angiogram of postballoon angioplasty shows acceptable results. However, in reality, most of the lesions required stent implantation. **Figure 32** shows a typical 5 cm stenotic lesion and balloon angioplasty was performed.

Balloon dilatation resulted in dissection and bail-out stenting was performed. Nitinol stent implantation could seal the dissection with no residual stenosis. Although such pivotal trials proved the superiority of stents over the balloon, the results could not be extrapolated to real-world cases. First, these studies excluded patients with backgrounds of complex risk factors such as kidney dysfunction. Second, long lesions classified as TASC (II) C/D lesions were also excluded. The real-world population study did not prove the expected patency using DES [16]. From our experience, we are aware that long-segment stent implantation has many disadvantages included stent fracture and thrombosis. Only the covered stent, VIABAHN (Gore, Flagstaff, AZ, USA) showed acceptable results in long lesions (**Figure 33**) [17]. However, the application of covered stents in the SFA is still not well known so a cautious approach should be taken in VIABAHN implantation.

5.2. Drug-coated balloon and atherectomy devices

Since a drug-coated balloon (DCB) was first introduced, there have been many arguments about primary stenting. DCB trials proved to be safe and effective in SFA lesions with better outcomes
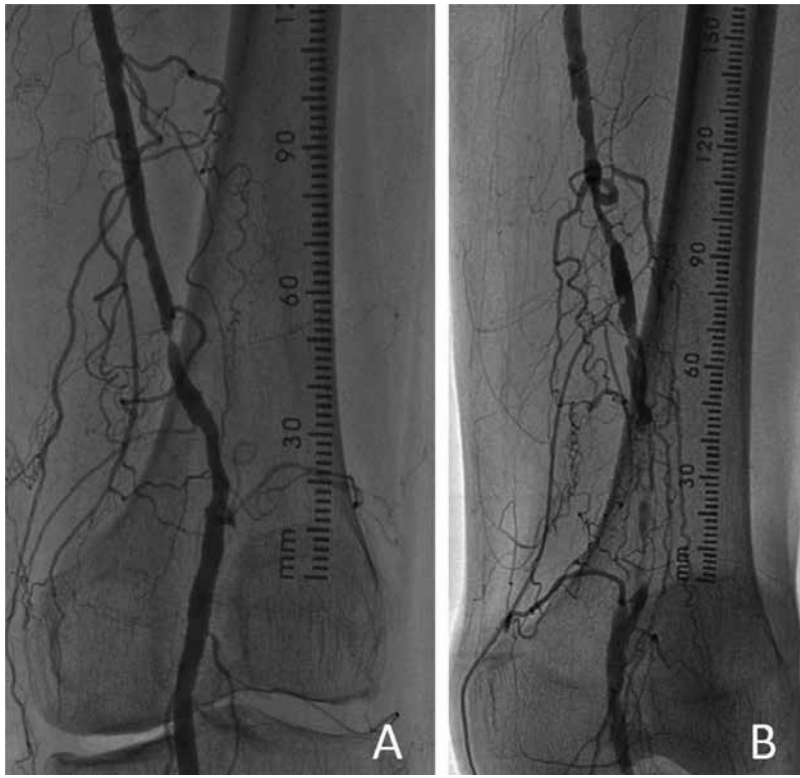
Figure 31. In a short lesion, initial approach is (A) balloon angioplasty; (B) short focal lesion in distal SFA; and (C) balloon angioplasty performed with 5 × 40 mm balloon. Postballoon angioplasty showed residual stenosis of less than 50% at ballooning site.
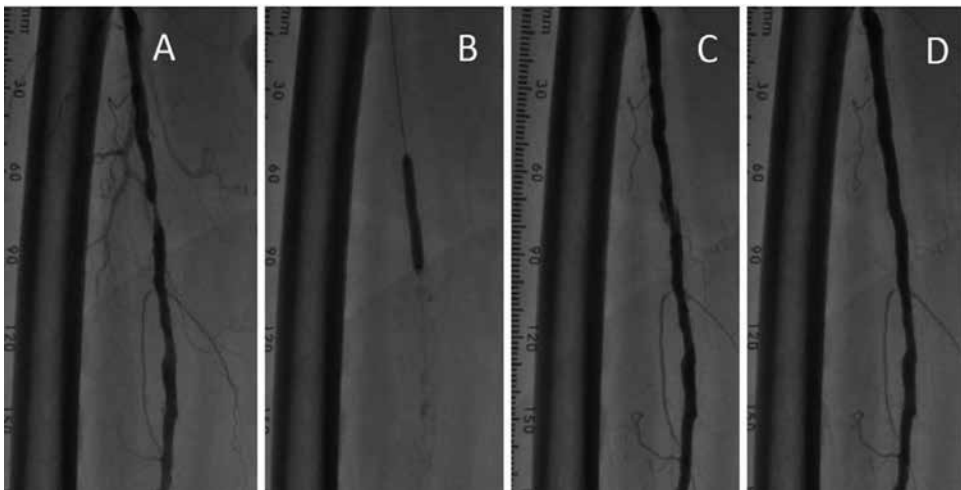
Figure 32. Bail out stenting post balloon angioplasty procedure of (A) 3 cm stenotic lesion; (B) balloon angioplasty performed; (C) dissection; (D) nitinol stenting for bail out purpose successful in sealing dissection.
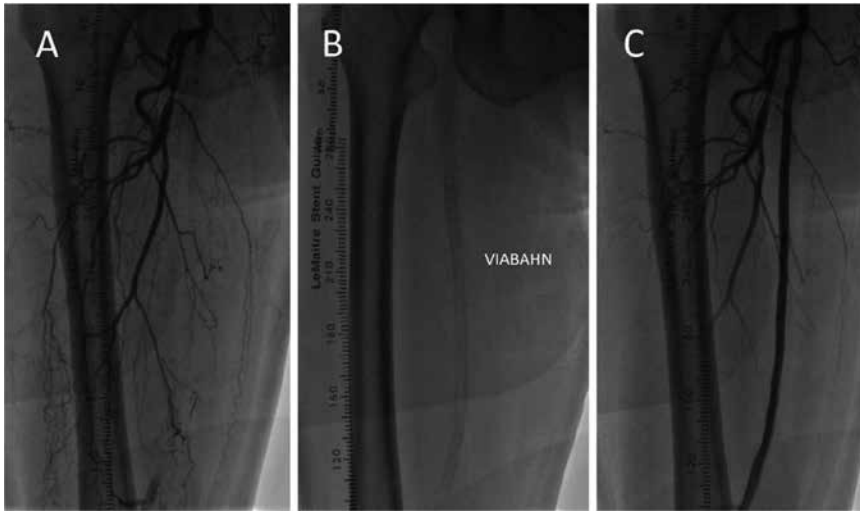
Figure 33. (A) Covered stent implantation for long right SFA CTO lesion; (B) CTO length is over 20 cm lesion; (C) VIABAHN (Gore, Flagstaff, AZ, USA) 6 × 250 mm stenting after balloon angioplasty.

over conventional balloon angioplasty in simple lesions (IN.PACT DCB, Medtronic Inc., Santa Rosa, CA, USA and Lutonix DCB, Bard Peripheral Vascular; Tempe, AZ, USA) (**Figure 34**) [18–20].

Furthermore, various atherectomy devices are being developed. At present, there are two types of atherectomy devices: one is a rotational cutting device (JETSTREAM, Boston Scientific, Cambridge, MA, USA [21, 22] (**Figure 35**) and Diamondback, Cardiovascular Systems,

Figure 34. DCB angioplasty procedure. (A) Short focal lesion in mid-SFA; (B) after conventional balloon angioplasty and drug coated balloon angioplasty with LUTONIXX DCB (Bard Peripheral Vascular; Tempe, AZ) 5 × 100 mm balloon; (C) postballoon angioplasty showed residual stenosis of less than 50% at ballooning site.
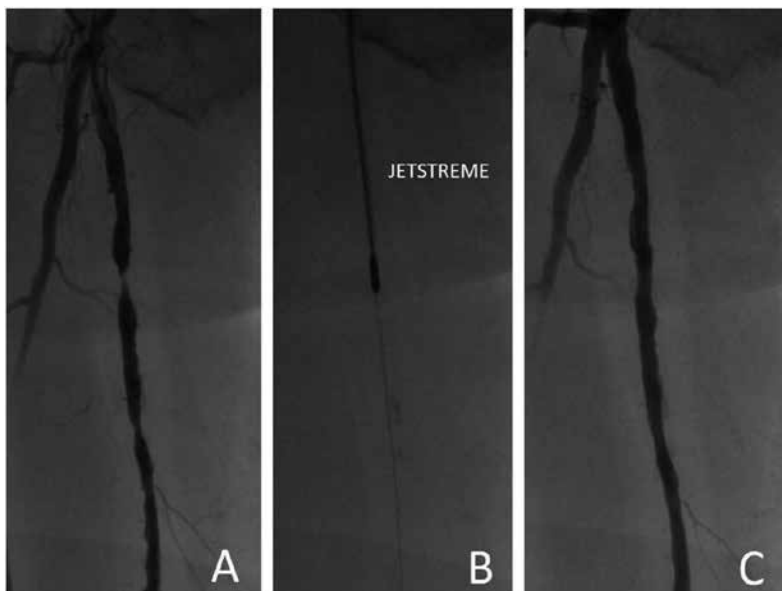
Figure 35. Atherectomy device. (A) Short focal lesion in mid-SFA; (B) atherectomy with JETstreme (Boston Scientific, Cambridge, MA, USA) atherectomy device; (C) postprocedure.

Inc., St. Paul, MN, USA 360 [23]), and the other is a directional cutting device (TurboHawk (**Figure 36**), Silverhawk, Medtronic Inc., Santa Rosa, CA, USA) [24, 25]. However, although solid data on the efficacy of atherectomy devices are still lacking, the worldwide trend toward the "nothing left behind" approach favors these treatment options.
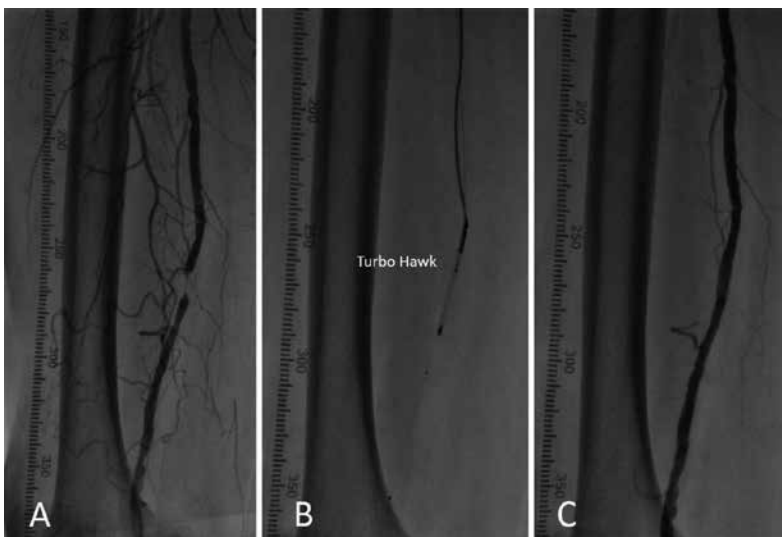
Figure 36. Atherectomy device. (A) Short focal lesion in mid-SFA; (B) atherectomy with TurboHawk (Medtronic Inc, Santa Rosa, CA) atherectomy device; (C) postprocedure.

6. Approach to complex femoropopliteal artery disease

Recanalization of complex SFA lesions is still challenging. In particular, the approach to a chronic total occlusion (CTO) lesion is not yet standardized and technically more demanding.

6.1. Approach site

In general, a contralateral CFA approach is the standard procedure mainly because the puncture site is not on the ischemic limb side, and the proximal SFA and DFA can be safely approached. However, to intervene in a complex lesion, deft manipulation of the wiring is vital, and for that reason, some interventionists prefer the antegrade approach as the initial step. Furthermore, a recent advance in SFA intervention is the use of the bidirectional approach. The distal puncture sites presently being employed are the distal SFA, popliteal artery, and dorsal pedis artery. The bidirectional approach has a higher chance of recanalization compared to a unidirectional approach. However, the safety of employing these new puncture sites is not well elucidated so that a cautious approach is necessary.

6.2. Wire

The antegrade approach via ipsilateral or contralateral CFA access is the standard approach. First, we try to cross the lesion with 0.035 inch guidewires. Subintimal tracking with a 0.035 inch J-loop wire and support catheter is the traditional method of CTO recanalization (**Figure 37**).

When crossing the CTO by the antegrade approach fails, a bidirectional approach with a distal puncture should be attempted. Suitable points for the distal approach include the popliteal, distal SFA, and tibial puncture sites (**Figure 38**).
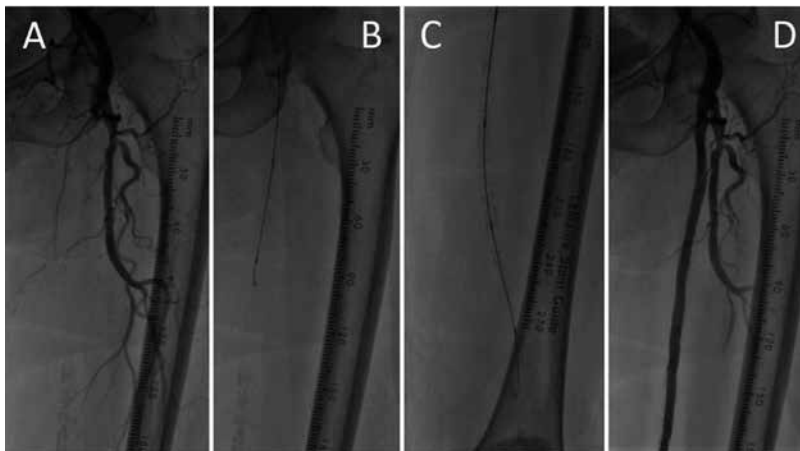
Figure 37. Antegrade approach with 0.018 inch guidewire. (A) SFA long total occlusion from proximal SFA to distal portion; (B) 0.018 inch J-type guidewire with support catheter into CTO lumen; (C) guidewire passage to distal true lumen; (D) final angiography after nitinol stent implantation.

Figure 38. Retrograde access with distal puncture to recanalize SFA total occlusion lesion. (A) Popliteal artery puncture; (B) distal SFA puncture; (C) distal anterior tibial artery puncture; and (D) distal posterior tibial artery.

6.3. CTO crossing devices

CTO crossing devices are being approved, but the safety and efficacy of these devices have not yet been proven. Two types of CTO crossing devices are used broadly: first is a reentry device and the Outback (Cordis Endovascular, Bridgewater, NJ, USA) [26] (**Figure 39**), Pioneer (Medtronic Inc., Santa Rosa, CA, USA) [27], and OffRoad (Boston Scientific, Cambridge, MA, USA) [28] devices are categorized in this group while the other includes devices for true lumen crossing such as the Crosser (Bard Peripheral Vascular; Tempe, AZ, USA) [29] (**Figure 40**),
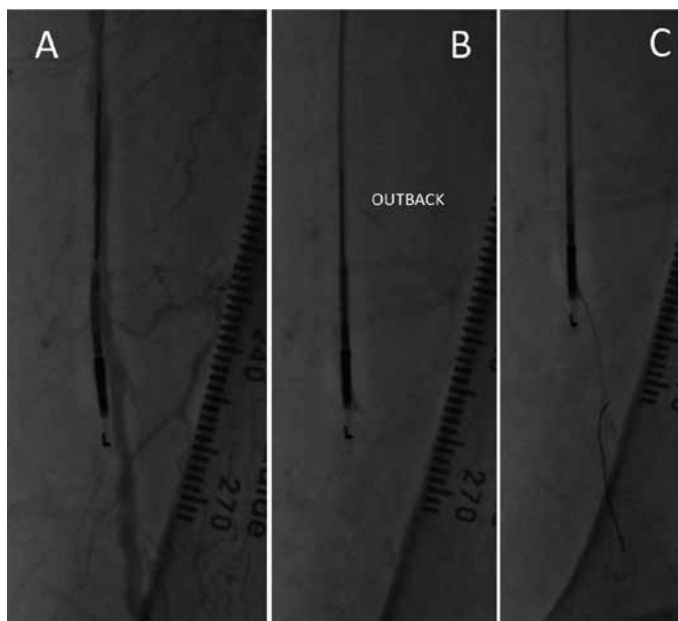
Figure 39. Outback reentry device (Cordis Endovascular, Bridgewater, NJ, USA). (A) Outback reentry device was placed in subintimal space of SFA mid portion. (B) Deploying the cannula from subintimal space to true lumen. (C) Wire crossing.
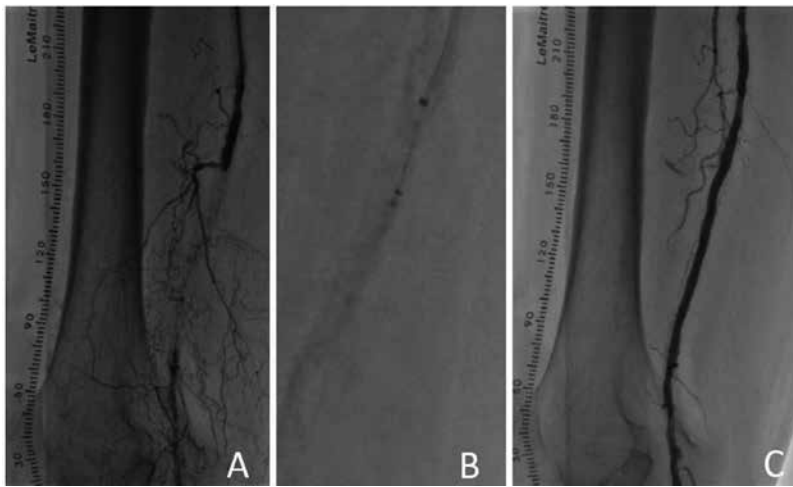
Figure 40. CTO crossing device. (A) Heavy calcified occlusive disease at distal SFA portion; (B) Crosser CTO crossing device (Bard Peripheral Vascular; Tempe, AZ) advanced without guidewire; (C) after crossing device, nitinol stent implantation.

TruePath (Boston Scientific, Cambridge, MA, USA) [30], Frontrunner (Cordis Endovascular, Bridgewater, NJ, USA), and Wildcat (Avinger, Redwood City, CA, USA).

7. Complications in femoropopliteal artery intervention

The rate of complications varies depending on how they are defined. In addition, the operator's experience contributes to the complication rate. Serious complications in femoropopliteal artery intervention are less frequent in experienced hands. Most important is to foresee unexpected events in order to prevent an adverse outcome. The factors most related to complications are complex lesions, long procedure times, the use of many catheters, and excessive long wire manipulation. The other factors are patient related such as obesity, evidence of critical limb, hemodialysis dependence, etc. Understanding such potential risks is essential for those engaging in interventional procedures.

7.1. Vessel perforation

Perforations occur in femoropopliteal artery interventions from various causes. The common causes of femoropopliteal artery perforations have been divided into four types. First is puncture-related perforation. **Figure 41A** shows a puncture-related vessel perforation by the antegrade approach at the CFA. In a retrograde approach, the popliteal artery was perforated by a puncture (**Figure 41B**).

Figure 42 shows the CFA perforation caused by the crossover sheath.

The second are guidewire perforations. Guidewire perforation is usually of little consequence since they are typically small and rarely result in significant or continuous bleeding. They

Figure 41. Puncture site vessel perforation. (A) Antegrade approach via CFA puncture; (B) retrograde approach via popliteal artery.

Figure 42. Procedure-related vessel perforation: vessel perforation occurred by cross-over sheath from contra lateral CFA puncture access.

usually occur when the wire is advanced into an occlusion site (**Figure 43A**) or migrate into small side branches (**Figure 43B**). In SFA intervention, after successful wire passage, the tip of the guidewire is not visible due to the long SFA vessel and the distal tip of the wire may advance deeply to perforate the below the knee arteries (**Figure 43C**). The third type is atherectomy or CTO crossing device-induced perforation. These perforations occur due to the direct cutting or mechanical penetration of the adventitial layer. CTO crossing devices have a perforation rate of 1–6% [29, 30].

Figure 44 shows the vessel perforation caused by the Crosser device. Atherectomy devices have a perforation rate of between 0.5 and 2.2% [20–24]. Although atherectomy and CTO-crossing device-related perforation rates are relatively low, interventionists should have a definite strategy to cope with these complications. The last is balloon angioplasty-related perforation. The mechanism of balloon dilatation is to disrupt the intima and media of the vessel wall to obtain an increased luminal area. When the area up to the adventitia is penetrated, vessel perforation occurs. Heavy calcification, high-pressure ballooning, and the use of oversized balloons are the potential risks of balloon perforation.

Figure 45 shows the vessel perforation after balloon angioplasty with or without stent implantation.

7.2. Acute thrombosis and distal emboli

Acute arterial thrombosis is another common problem seen in femoropopliteal interventions. This complication may occur after EVT as a result of the erosion or rupture of the atherosclerotic plaque, and distal emboli. There are not many reports on acute or subacute stent occlusion by drug-eluting stent or balloon, but this paclitaxel-coated device has the potential risk of thrombotic occlusion. When intervening for in-stent-occlusion, there is a high risk of distal emboli during the procedure (**Figure 46**).
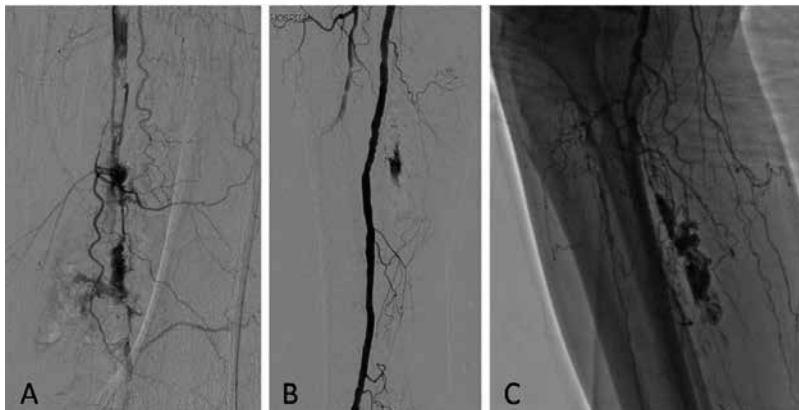
Figure 43. Vessel perforation: (A) caused by hard guidewire advanced into total occlusion portion; (B) aberration to small SFA side branches; (C) too deeply advanced guidewire to below the knee arteries.
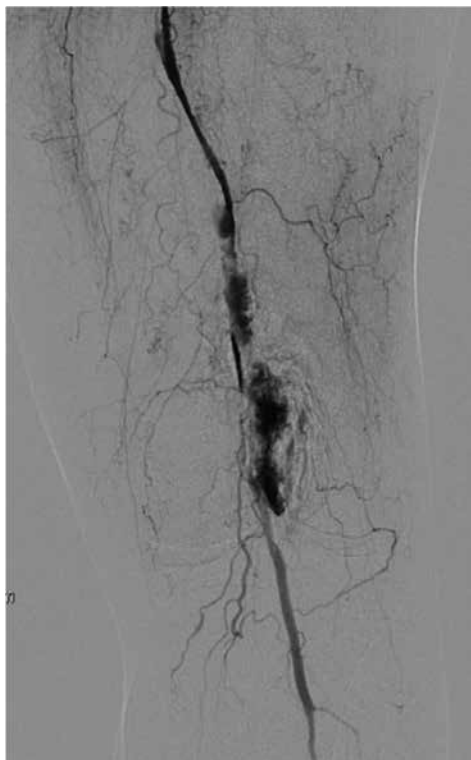
Figure 44. CTO crossing device related vessel perforation: vessel perforation by Crosser device at SFA mid-portion occlusive disease.
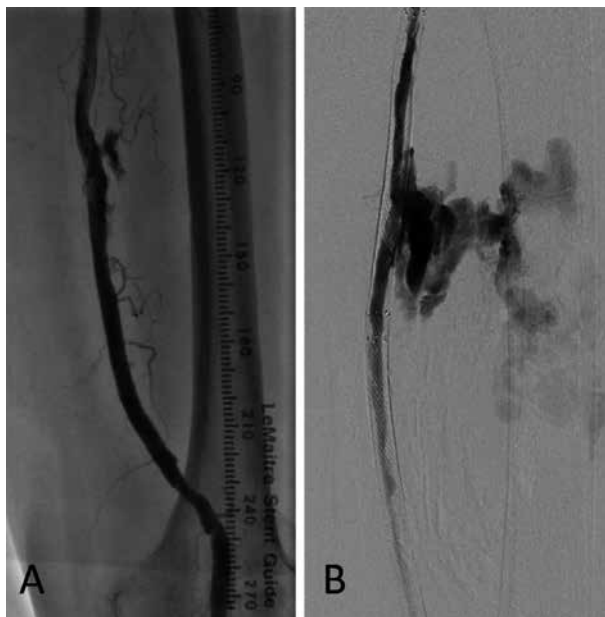
Figure 45. Balloon dilatation related vessel perforation (A) and (B).
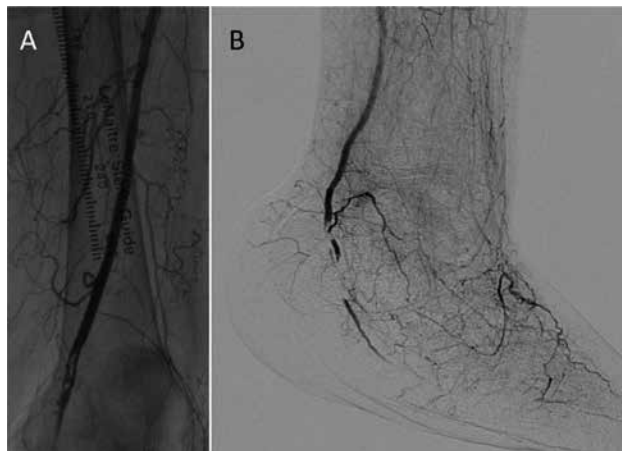
Figure 46. Distal emboli after femoropopliteal intervention. (A) Total occlusion of popliteal artery caused by distal emboli after SFA stent implantation; (B) distal emboli into PTA distal portion.

7.3. Arterial-venous fistula

Arterial-venous communications, which are often seen after subintimal angioplasty, are the least concerning. Irregular AV communications usually represent a series of microperforations between the artery and adjacent vein (**Figure 47**).

Most of these cases may be shielded by balloon or stent implantation (**Figure 48**). In case when balloon shielding is unsuccessful, the covered stent may be the next option.
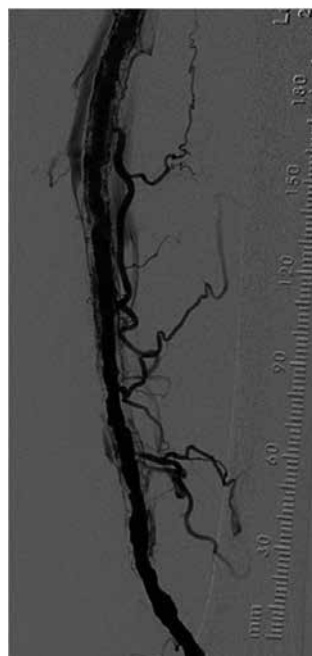
Figure 47. AV fistula: occurred after balloon angioplasty.
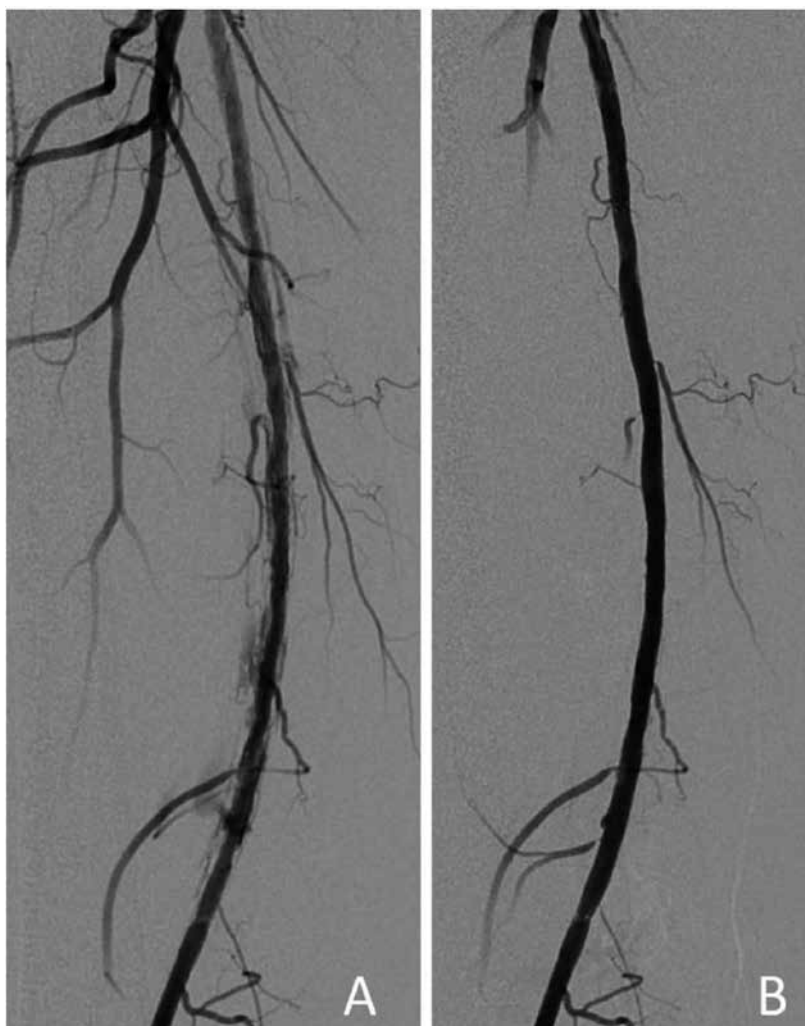
Figure 48. Stent implantation can repair the AV fistula in most cases. (A) Balloon angioplasty performed and resulted in AV fistula; (B) nitinol stenting for bail out purpose succeeded in sealing AV fistula.

8. Popliteal artery disease

The popliteal artery is a deeply placed continuation of the femoral artery after it passes through the adductor hiatus or opening in the distal portion of the adductor magnus muscle. It courses through the popliteal fossa and ends at the lower border of the popliteus muscle, where it branches into the anterior and tibio-peroneal trunk. In the popliteal artery, there are various types of arterial disease such as atherosclerosis stenosis or occlusion, popliteal aneurysms, popliteal artery entrapment syndrome, cystic adventitial disease, and Buerger's disease. The ideal angiographic image is provided by a contralateral view (**Figure 49**).

Figure 49. Popliteal artery disease; clear image by contra-lateral angled view.

Surgical therapy for popliteal artery occlusion involves a bypass of the occlusion, which can be achieved with grafts, including the great saphenous vein or PTFE grafts (**Figure 50**).

Endovascular therapy is a less invasive intervention in the treatment of popliteal artery occlusive disease. It is indicated for short and noncalcified lesions. The popliteal artery is a nonstenting zone and stent implantation should be avoided (**Figure 51**).

Some studies have suggested that the use of drug-coated balloons is safe and effective for proximal popliteal disease, especially for preventing restenosis [16–18]. Recently, the SUPERA (Abott Vascular, Santa Clara, California, USA) stent, which is a high kink and fracture-resistant stent, has been introduced and evaluated for primary treatment of the proximal popliteal artery for its flexibility [31].

Figure 50. Popliteal artery aneurysm case (A) preprocedure; (B) after surgical therapy with SVG bypass.
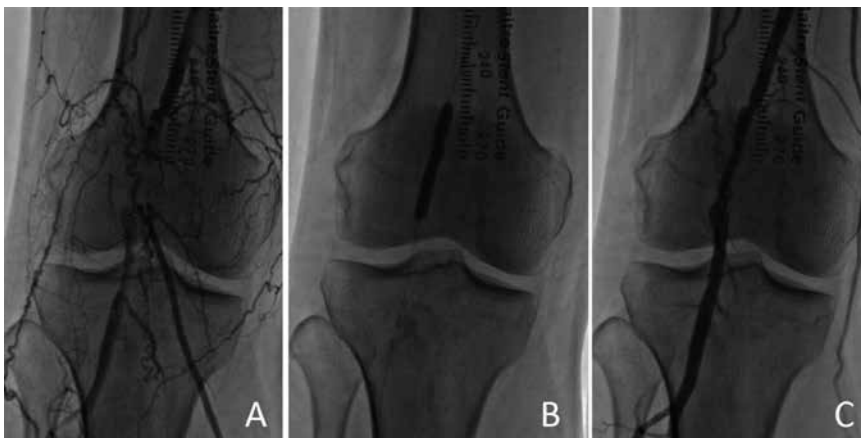
Figure 51. In short popliteal lesion. (A) Short focal lesion in mid-popliteal artery; (B) balloon angioplasty performed with 5 × 40 mm balloon; (C) postballoon angioplasty showed residual stenosis of less than 50% at ballooning site.

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Angiography and Endovascular Therapy for Below-the-Knee Artery Disease

Akihiro Higashimori

Additional information is available at the end of the chapter

Abstract

Critical limb ischemia (CLI) is growing in global prevalence and is associated with high rates of limb loss and mortality. "Endovascular-first" approach is considered to be the current standard care for symptomatic infrainguinal atherosclerotic disease. Given the facts that many CLI patients have severe comorbidities and endovascular-first approach is a common practice and may reduce the magnitude of the surgical trauma and systemic complications. In this chapter, updated angiographic approach for below-the-knee disease is described with endovascular technique.

Keywords: critical limb ischemia, below-the-knee disease, angiosome, indigo carmine

1. Introduction

Critical limb ischemia (CLI) is growing in global prevalence and is associated with high rates of limb loss and mortality. The "endovascular-first" approach is considered to be the current standard treatment for symptomatic infrainguinal atherosclerotic disease [1]. Given the fact that many CLI patients have severe comorbidities, the endovascular-first approach is most common since it reduces the magnitude of surgical trauma and systemic complications. In this chapter, an updated angiographic approach for below-the-knee artery disease is described with endovascular techniques.

2. Basic angiographic technique for below-the-knee artery

2.1. Vascular anatomy of the below the knee

Normally, from the direct continuation of the popliteal artery and past the branch of the anterior tibial artery (ATA) is the tibioperoneal trunk. This vascular segment splits into the posterior tibial artery (PTA) and the peroneal artery (**Figure 1**).
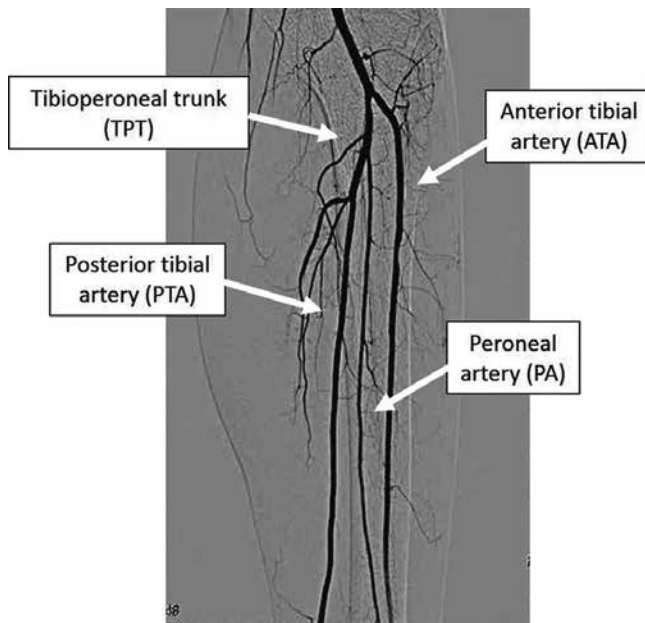
Figure 1. Anteroposterior view of the tibial trifurcation shows anterior tibial artery, tibioperoneal trunk, peroneal artery, and posterior tibial artery.

The distal peroneal artery splits into the anterior malleolar branch and the calcaneal branch (**Figure 2**).
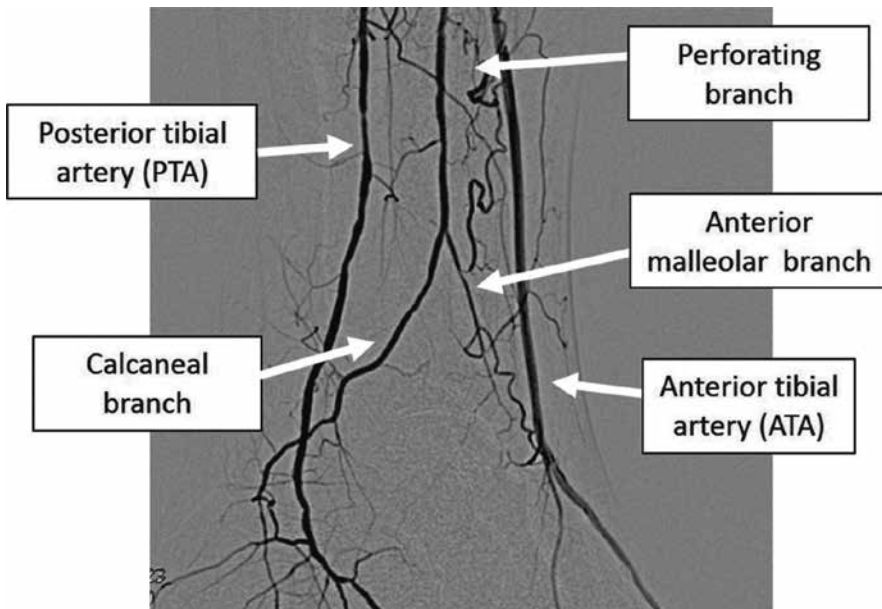
Figure 2. Lateral view at the distal leg shows the distal anterior and tibial arteries and peroneal artery. Distal peroneal artery splits into the anterior malleolar branch and calcaneal branch.

The dorsal pedis artery is a continuation of the ATA in the foot. The PTA bifurcates into the lateral plantar artery and medial plantar artery (**Figure 3**).
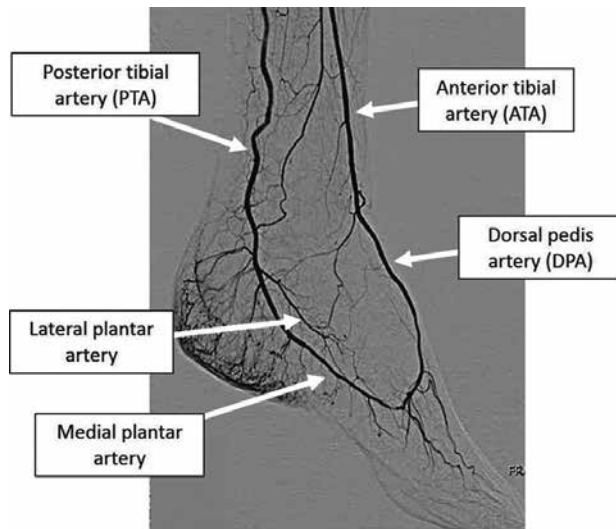
Figure 3. Lateral view of the ankle shows distal anterior tibial artery and posterior tibial artery. Dorsal pedis artery is a continuation of the anterior tibial artery in the foot. Posterior tibial artery bifurcates into the lateral plantar artery and medial plantar artery.

By definition, the popliteal artery ends at the origin of the first tibial artery, which typically is the ATA. In about 4% of cases, we find a so-called high origin of the ATA at the level of the knee joint or even some centimeters more proximal (**Figure 4**).
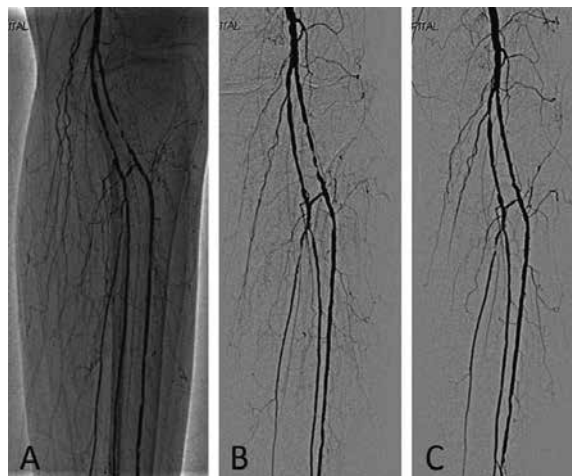
Figure 4. (A and B) High division of popliteal artery branching and anterior tibial artery at the knee joint. The anterior tibial artery was diseased. (C) Balloon angioplasty was performed in the anterior tibial artery.

Similarly, in just as small proportion of the population, a high origin of the PTA has been described. As a variation, a trifurcation of the popliteal artery into all three lower leg arteries at the same point has been observed in 0.4% of patients. The PTA may be missing completely in 1–5% of the normal adult population (**Figure 5**) [2].

Figure 5. The posterior tibial artery is missing completely. Peroneal artery connects through the tibial arteries.

Branching variations of below-the-knee arteries are common. A practical triad classification of anatomical variations in the branching pattern has been reported [3], that is, Type 1, Type 1-B, Type 2-A, Type 3-A, and Type 3-B (**Figure 6**) [4].
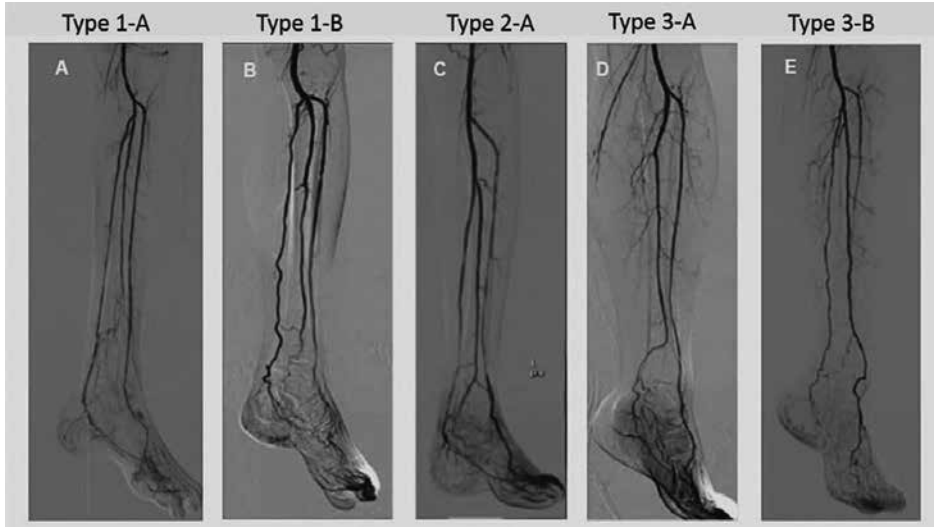
Figure 6. Anatomical variation of tibial arteries. (A) Type 1-A: normal level of arterial branching and most common pattern. The first branch is anterior tibial artery and peroneal trunk separates into peroneal and posterior tibial arteries. (B) Type 1-B: normal level of arterial branching. The three tibial arteries show trifurcation. (C) Type 2-A: high division of popliteal artery branching and the anterior tibial artery arises at the knee joint. (D) Type 3-A: hypoplastic posterior tibial artery and peroneal artery provide a distal supply to the plantar side of the foot. (E) Type 3-B: hypoplastic anterotibial artery and peroneal artery provide distal supply to the dorsal side of the foot.

There are many more anatomical variations and we have to keep in mind that these variant arteries are rather common (**Figures 7** and **8**).
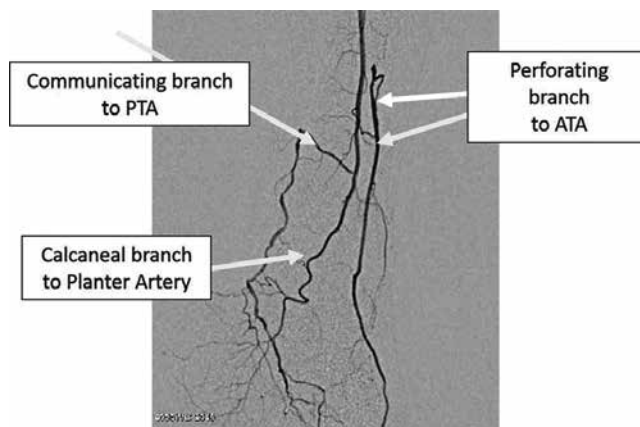
Figure 7. When ATA and PTA are occluded, the peroneal artery may function as the sole blood supply of the foot. Left ATA and PTA are occluded. Anterior terminal branch (perforating branch) of peroneal artery gives collateral to ATA. Similarly, posterior terminal branch (communicating branch) gives collateral to PTA.
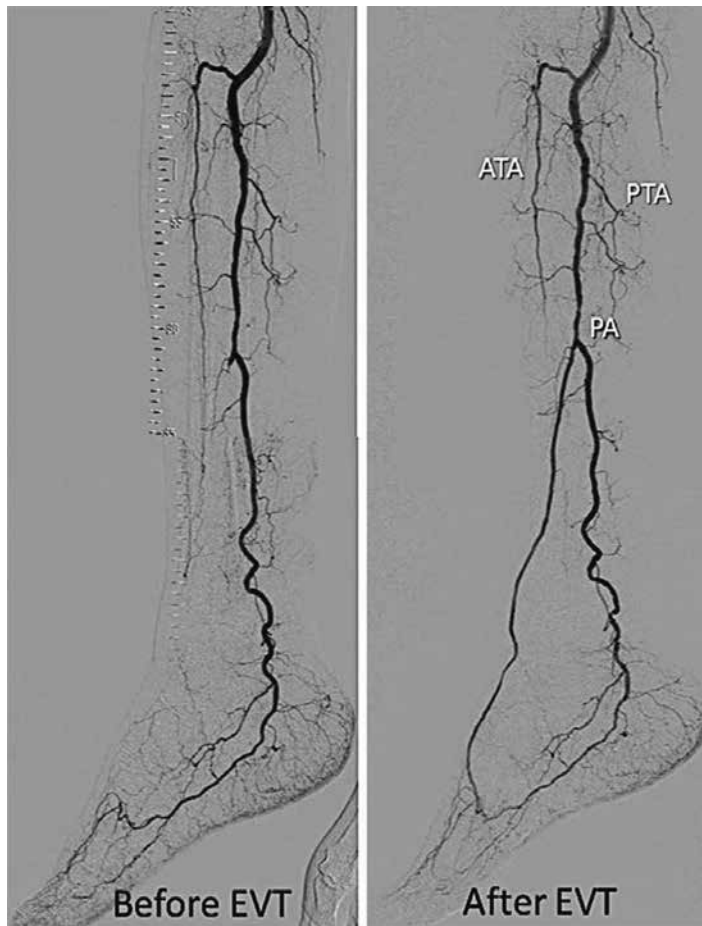
Figure 8. Based on pre-interventional below-the-knee angiography, it was assumed that anterior tibial artery is occluded. Post-interventional below-the-knee angiography showed that anterior tibial artery is hypoplastic. Peroneal artery continues to the dorsal pedis artery.

2.2. Angiography for below-the-knee arteries

Before intervention in below-the-knee vessels, the target vessel should be clearly identified. After confirming that no inflow disease is present, below-the-knee angiography is performed since the presence of inflow disease results in a less clear image [4]. **Figure 9** shows the basic image of below-the-knee angiography.

Only the AP view is insufficient to clearly visualize the below-the-knee anatomy and diseased segment. Four views are mandatory to see all below-the-knee arteries. There are two right anterior oblique (RAO) views and two left anterior oblique (LAO) views. In the upper RAO view, the proximal left ATA is well visualized and, in the lower RAO view, the three distal tibial arteries are well observed. In the upper LAO view, bifurcation of the peroneal artery and PTA is well visualized. In the lower LAO view, the ATA and peroneal artery overlap and these two vessels cannot be separated (**Figure 10**).
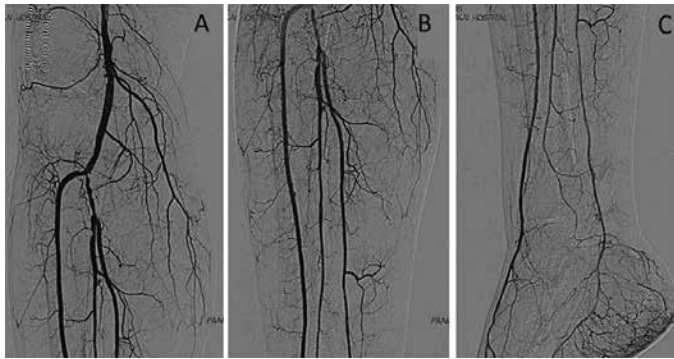
Figure 9. Basic below-the-knee angiography. Only AP view is insufficient to visualize the below-the-knee anatomy and diseased segment.
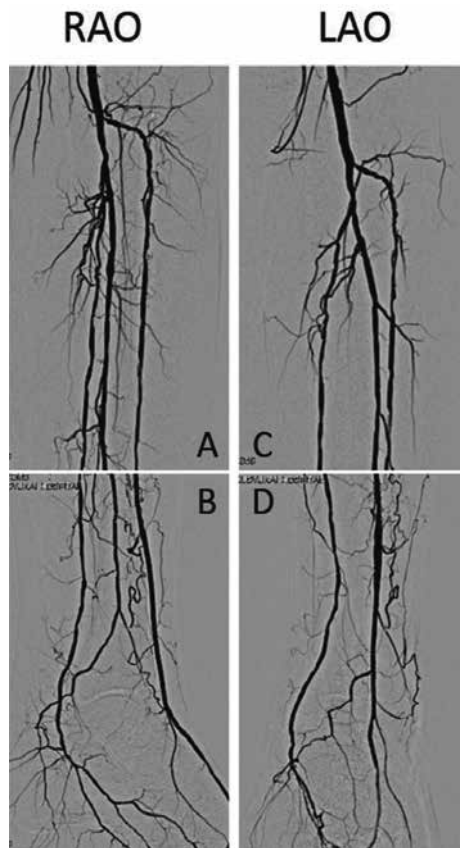
Figure 10. Basic below-the-knee angiography. (A) Upper right anterior oblique view. Proximal anterior tibial artery is well visualized, but peroneal artery and posterior tibial artery are overlapped. (B) Lower right anterior oblique view. In this view, distal three tibial arteries are separated. (C) Upper left anterior oblique view. Proximal peroneal artery and posterior tibial artery are well separated. (D) Lower left anterior oblique view. Distal posterior tibial artery is well visualized, but distal anterior tibial artery and peroneal artery are overlapped.

Figure 11 shows the basic angiographic steps. A femoropopliteal artery angiogram should be taken to rule out inflow disease (**Figure 11A**). After confirming that no inflow disease is present, below-the- knee angiography is performed through a catheter located at the distal popliteal artery (**Figure 11B**). Four views are taken before angioplasty (**Figure 11C**) and four more views after angioplasty (**Figure 11D**).
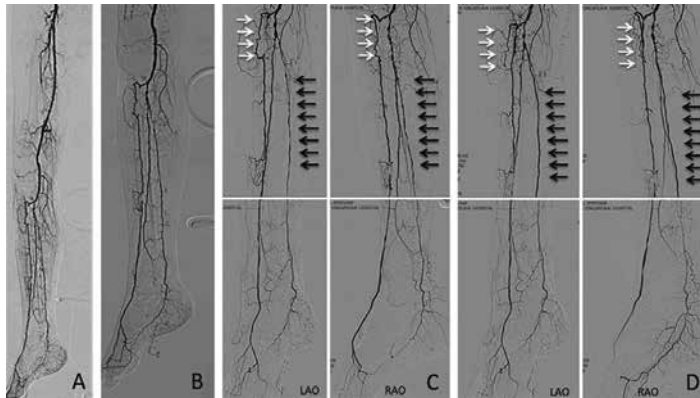
Figure 11. (A) Pre-interventional angiography for critical limb ischemia. Ipsilateral right femoropopliteal artery angiogram which can rule out inflow disease. (B) Below-the-knee angiography showed the entire three tibial arteries. (C) Pre-interventional below-the-knee angiography. This angiography showed the diseased proximal anterior tibial artery (white arrow) and mid-portion of the posterior tibial artery (black arrow). (D) Post-intervention below-the-knee angiography showed proximal anterior tibial artery and mid-posterior tibial artery successfully recanalized. Pre- and post-angiography were taken in the same angle and view.

If the patients have impaired renal function, four views are not mandatory. Moreover, pre- and post-interventional angiography should be taken in the same image size and angle (**Figures 12** and **13**).
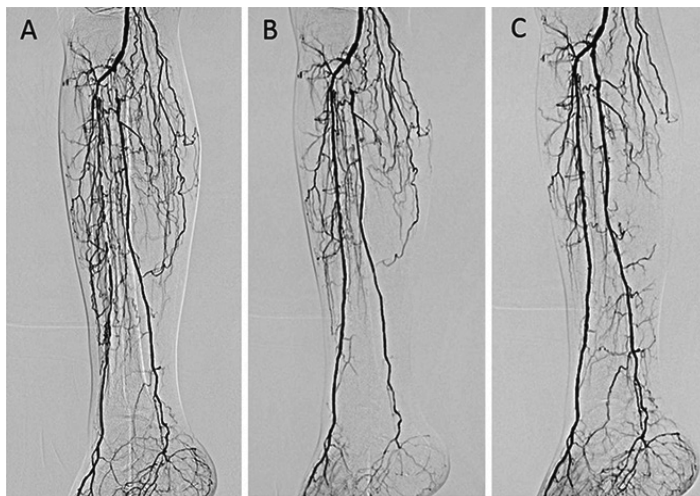
Figure 12. (A) Below-the-knee angiogram of pre-intervention. Right anterior tibial artery, peroneal artery, and posterior tibial artery were occluded. (B) Angiography of postanterior tibial artery intervention. (C) Angiography of postanterior and posterior tibial artery intervention. Peroneal artery was still occluded but sufficient blood flow seems to be obtained.
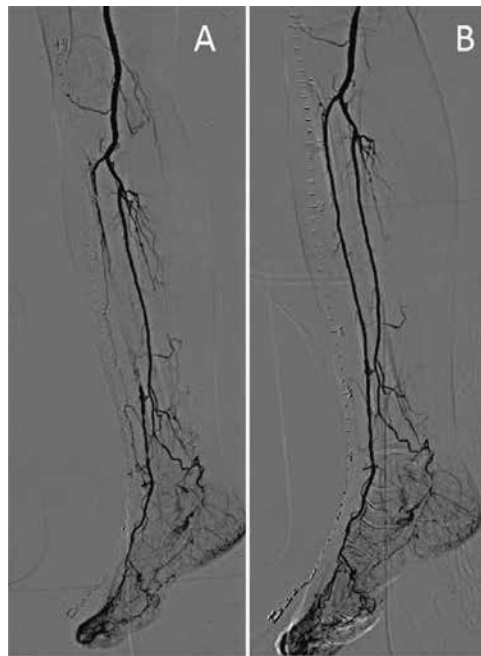
Figure 13:. Pre- (A) and post- (B) interventional angiography should be taken in the same image size and angle.

3. Basic angiographic technique for below-the-ankle arteries

3.1. Vascular anatomy of below-the-ankle arteries

The vascular anatomy of the foot is composed of the anterior and posterior circulation connected through the pedal arch. The anterior circulation consists of the dorsal pedis artery, lateral tarsal artery, and arcuate artery (**Figure 14**).
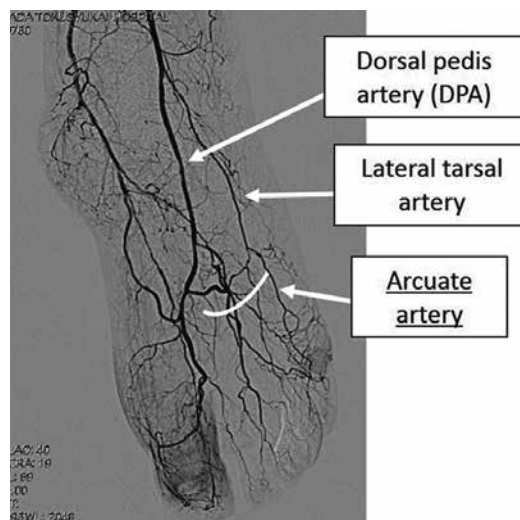
Figure 14. Anterior circulation consists of dorsal pedis artery, lateral tarsal artery, and arcuate artery.

The dorsalis pedis artery is a continuation of the anterior tibial artery distal to the ankle. The branches of the dorsalis pedis include the lateral and medial tarsal, first dorsal metatarsal, deep plantar, and arcuate arteries. The arcuate artery arises near the medial cuneiform, passing laterally over the metatarsal bases deep to the digital extensor tendons and gives rise to the second through fourth dorsal metatarsal arteries before anastomosing with the lateral tarsal artery. The arcuate artery is classically described as being the primary source of blood for the second, third, and fourth dorsal metatarsal arteries [5]; however, a combination of the arcuate, lateral tarsal, and proximal perforating arteries may be involved depending on an individual's vascular anatomy. The posterior circulation consists of the medial plantar artery and lateral plantar artery. These two arteries have an artery-to-artery connection which constitutes the deep plantar arterial arch. The first to fourth plantar metatarsal arteries are the branches of the deep plantar arterial arch (**Figures 15–17**).
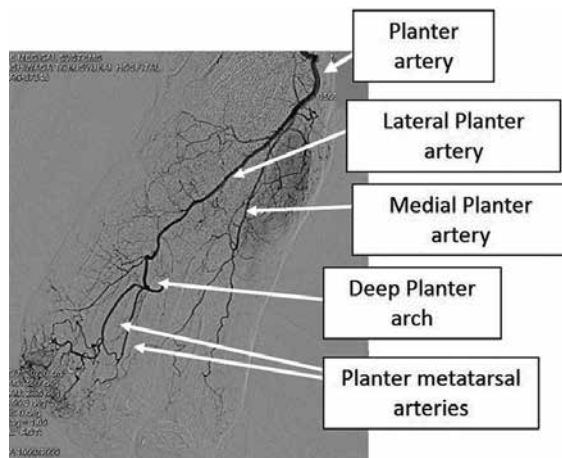
Figure 15. Posterior circulation consists of medial plantar artery and lateral plantar artery.
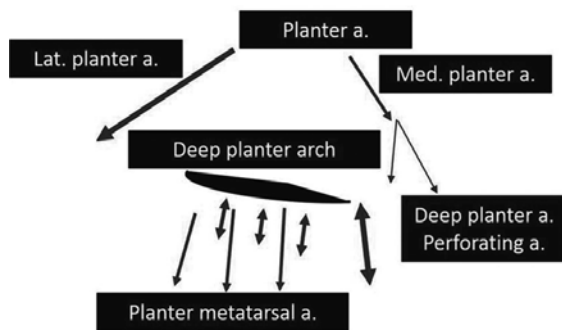
Figure 16. The digital branches originate from the anterior and posterior circulation. There are dorsal branches for each toe.

Figure 17. The digital branches originate from the anterior and posterior circulation. There are plantar branches for each toe.

Normally, the area between the anterior and posterior circulation forms the pedal arch. It primarily constitutes the deep perforating branches of the dorsal artery of the foot and the medial plantar artery. This arcade is the main supply for all distal forefoot circulation (**Figure 18**).
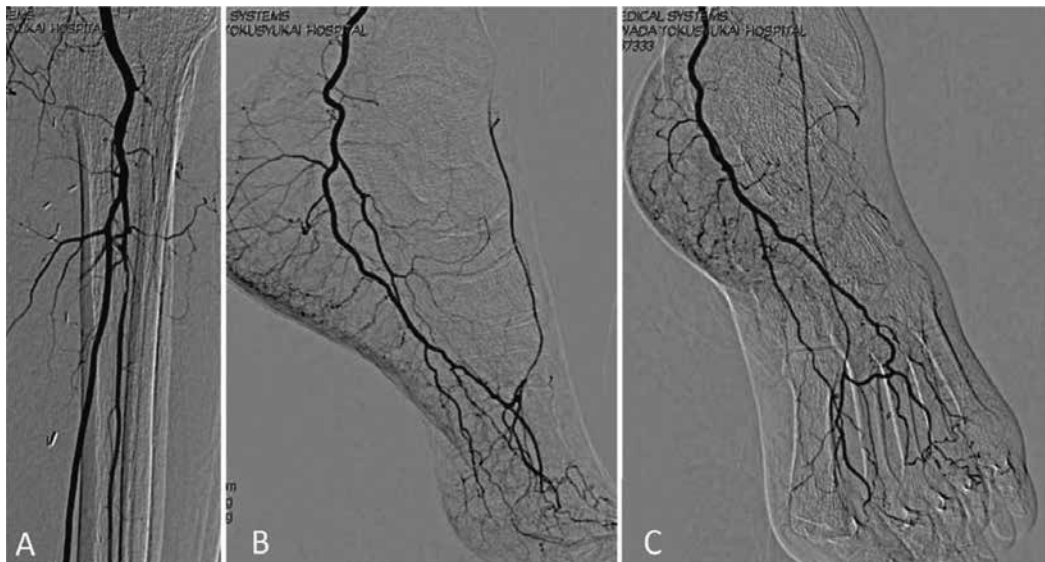
Figure 18. (A) Right anterotibial artery occluded at the proximal portion. (B) Lateral view of the right foot. Right dorsal pedis is retrograde visualized. (C) Dorsal pedis is visualized through the collateral via the deep plantar artery.

Moreover, we should keep in mind that anatomically variant arteries are quite common (**Figure 19**).
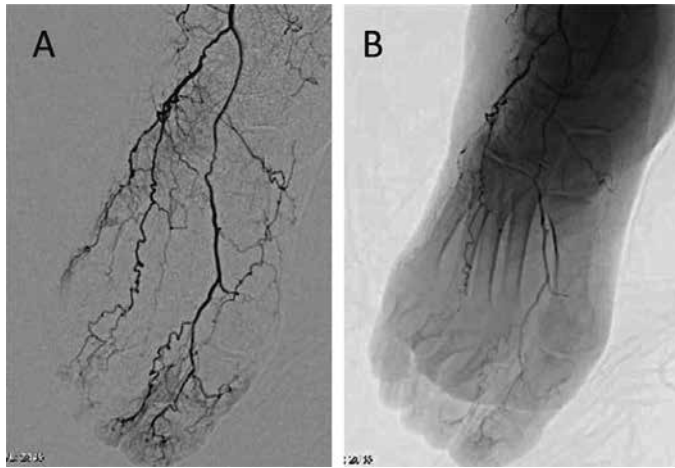
Figure 19. Angiography shows the absence of the arcuate artery and the anomalous origin of the dorsal metatarsal arteries (A) DSA image and (B) DA image.

The pedal arch at the completion of angiography has been classified as follows: Type 1, both dorsal pedis and plantar arteries are patent; Type 2A, only the dorsalis pedis artery is patent; Type 2B, only the plantar artery is patent; and Type 3, both the dorsalis pedis artery and plantar arteries are occlusive (**Figure 20**) [6].
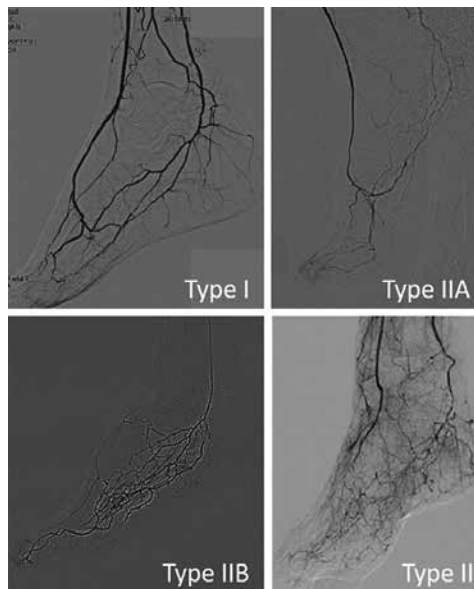
Figure 20. The pedal arch at the completion of angiography was classified into Types I, IIA, IIB, and III.

3.2. Angiography for below-the-ankle arteries

With severe ischemic limb, foot artery angiograms are hard to obtain due to patient leg movements. To minimize this problem, an injection of small amounts of contrast with inflow revascularization is essential. A typical below-the-ankle artery is shown in **Figure 21** [4].
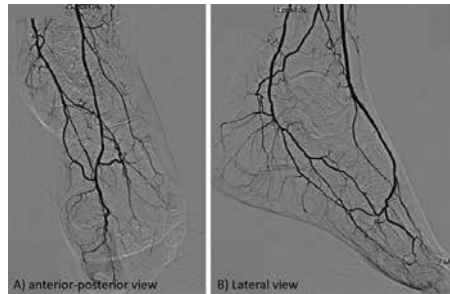
Figure 21. In the anterior view, plantaris medial and lateralis are well separated (A). In the lateral view, dorsal pedis artery is well seen but the lateral tarsal arteries are not well separated (B).

Basically, two views are sufficient: one is the anteroposterior view and the other a lateral view. In the lateral view, the dorsal pedis artery is well visualized but the lateral tarsal arteries are not well separated. In the anterior view, the medial and lateral plantar arteries are well separated. In the anterior view, the dorsalis pedis is well visualized and suited for retrograde dorsal pedis puncture (**Figures 22** and **23**).

Figure 22. White arrow shows the dorsal pedis artery. Black arrow shows the puncture needle.

Figure 23. In anterior view, dorsal pedis artery is well visualized. Black arrow shows severe stenosis (A). This lesion was treated with 1.5 × 20-mm balloon (B). Post-angiography (C).

In the lateral view, the distal posterior tibial artery and proximal dorsalis pedis artery are well visualized and suitable for retrograde posterior tibial artery puncture (**Figures 24** and **25**).
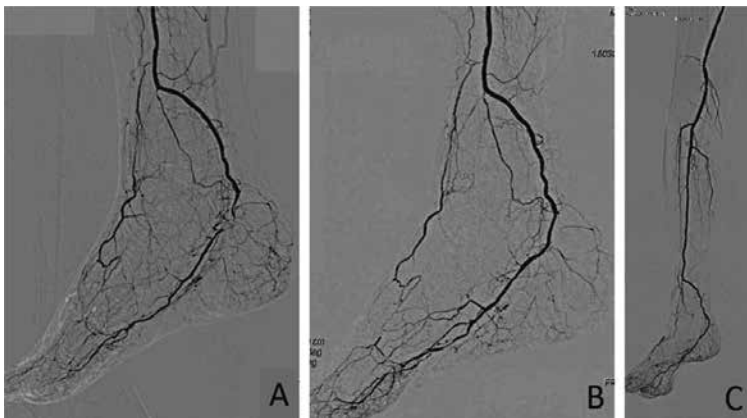
Figure 24. Balloon angioplasty to occluded plantar artery. Based on lateral angiogram, plantar artery was totally occluded (A). Using 2 × 40-mm balloon (B), the occluded vessel was recanalized (C).
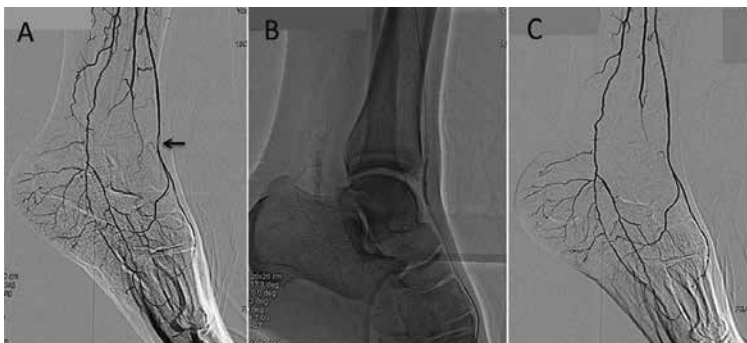
Figure 25. Based on lateral angiogram, proximal dorsal pedis artery disease is well visualized (black arrow) (A). This lesion was treated with 1.5 × 20-mm balloon (B). The stenosis lesion was recanalized (C).

4. Technical aspects of below-the-knee artery intervention

4.1. Endovascular therapy for below-the-knee artery disease

Endovascular therapy for the tibial vessels and foot arteries should be the first-line treatment in patients with CLI due to its good technical and clinical outcomes. Endovascular treatment is possible in most cases with a known low complication rate. The primary indications for tibial and foot artery intervention are limb salvage to avoid amputations. Patients with chronic leg ischemia face major future challenges and, in fact, the long-term survival rate with CLI is significantly lower than that of a matched population so that limb salvage is of major importance to such patients [2].

4.2. Access site

Antegrade access in the common femoral artery is, in our experience, the best approach to perform tibial and foot artery revascularization, with excellent guide ability of the wire and good pushability of the catheter balloons since this access site is closer to the lesions. Moreover, ultrasound-guided punctures may reduce radiation doses, screen time, and complications.

4.3. Guidewires

The tip load, tip stiffness, hydrophilic coat of the tip and body, guidewire flexibility, ability to shape, shaping memory, shaft support, torque transmission, trackability, and pushability are all critical components for a below-the-knee intervention guidewire [7]. Selection should be based on the specificities of the lesion such as localization, stenosis or chronic total occlusion (CTO), and lesion length. Non-hydrophilic guidewires allow a better tactile feel and a more controlled torque response when compared with hydrophilic wires. They are less likely to cause dissection of a vessel but have a higher resistance within the lesion, which decreases the chance of crossing, particularly "ronic total occlusions." But some guidewires, rather than increased sharpness, may have greater tip stiffness due to weight addition which increases their penetration ability. Hydrophilic wires typically advance with minimal resistance, providing good maneuverability in tortuous and long vessels but at a cost of reduced tactile feel [8]. In the field of coronary artery disease, we only use 0.014 inch wires, but in the field of peripheral artery disease, we use 0.014 inch and 0.018 inch wires. Our first wire is the 0.014 inch Runthrough peripheral (Terumo, Tokyo, Japan) and the second wires are the 0.014 inch Cruise (Asahi Intec, Nagoya, Japan) or 0.014 inch Command (Abott Vascular, Santa Clara, CA, USA). For chronic total occlusion lesions, we use 0.018 inch wires. **Table 1** shows the wires we use in our daily practice. These five wires cover almost all of our below-the-knee and below-the-ankle procedures.

0.014" guidewires	Hydrophilic	Tip load	Remarks
Runthrough peripheral (Terumo)	N	1 g	1st wire
Cruise (Asahi intecc)	Y	1 g	2nd wire
0.018" guidewires	Hydrophilic	Tip load	Remarks
Treasure (Asahi intecc)	N	12 g	CTO lesion
Astato (Asahi intecc)	N	30 g	CTO lesion

Table 1. Guidewires for below-the-knee artery intervention.

4.4. Balloon catheter

There are many low-profile balloons of various sizes and lengths for below-the-knee intervention. The balloon platforms are mostly dependent on either a 0.014-inch or 0.018-inch system. The main objective in below-the-knee intervention is to cross a total occlusion. For this purpose, we prefer a 0.018-inch lumen size for the over-the-wire angioplasty balloon (OTW) with increased shaft strength. These balloons give adequate pushability and wire support, particularly in complex chronic total occlusions when compared to the rapid exchange monorail technique. The over-the-wire balloon catheter system with the 0.018-inch platform allows good control of the wire without resistance and a smooth exchange to the 0.018 inch or 0.014 inch wire. They also allow wire exchange without sacrificing the progress made through the lesion. Additionally, their lumen can be used to inject contrast as well as verify position and distal outflow (**Figures 26** and **27**).
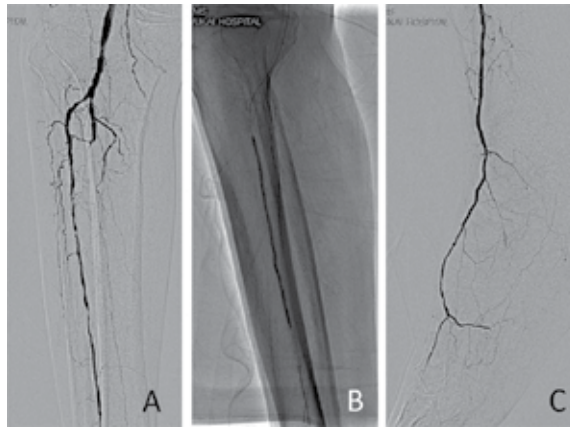
Figure 26. A 0.018-inch over-the-wire balloon catheter can be used to inject contrast, also verify position and distal outflow. (A) Pre angiography (B) Balloon delatation (C) Tip injection.

Figure 27. Injection of contrast through over-the-wire balloon. Contrast medium administered using a 1-ml Luer Lock syringe.

4.5. Transluminal recanalization for total occlusion

The first step in transluminal recanalization is to pass the proximal stenosis or negotiate the total occlusion by various wires until reaching the distal patent lumen. A drilling motion of the guidewire is used to properly penetrate and cross the lesion. As the initial wire, we prefer a 0.014-inch soft guidewire (**Table 1**) with an angulated tip since these very soft tips with coated wires have a higher chance of crossing the micro-channel without penetrating the subintimal space.

When we could not cross this soft 0.014 inch wire, it was exchanged with a 12-g 0.018-inch hard guidewire. The 0.018-inch wire has more controllability and penetration power compared to hard 0.014-inch wires. These wires are used with the OTW balloon catheter for wire support and exchange. In addition, the OTW balloon catheter can be used for tip injection to guide the wire and to understand what the problem is. However, in a 0.014-inch OTW balloon, there is a limitation for contrast injection (**Figure 28**).
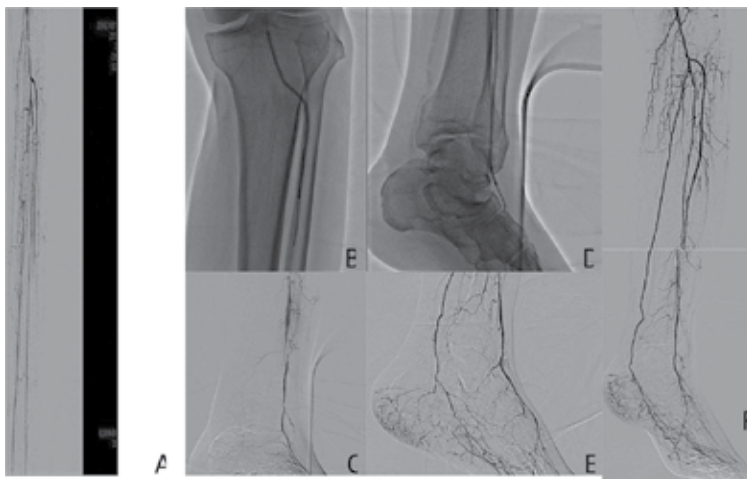
Figure 28. (A) Baseline angiography showed popliteal artery had severe stenosis and all below-the-knee vessels were occluded. (B) 0.014-inch soft guidewire was advanced into anterior tibial artery with 2.5 × 100-mm over-the-wire balloon catheter. The balloon was inflated at the distal popliteal artery and proximal anterior tibial artery. (C) Injection of contrast through over-the-wire balloon at proximal anterior tibial artery. Angiogram clarifies the position of the distal tip of the over-the-wire balloon and occluded segment. (D) 0.014-inch guidewire was changed to 0.018-inch hard guidewire which succeeded in crossing the occluded segment. A 2.0 × 100-mm balloon was inflated at the occluded segment. (E) Injection of contrast through over-the-wire balloon at the distal anterior tibial artery. Angiogram shows below-the-ankle vessels. (F) Final angiography shows complete revascularization was obtained.

4.6. Subintimal recanalization for total occlusion

A subintimal recanalization in below-the-knee arteries is one of the options after intraluminal attempts have failed and it was not possible to cross the lesion. Subintimal angioplasty was first described by Bolia [9], and since then there have been many publications to confirm its value and assess the clinical results of this technique to treat tibial vessels. We normally perform a subintimal recanalization in the crural arteries with a 0.018-inch J-tip guidewire (**Figure 29**) or 0.014-inch hydrophilic guidewire (Command, Abott Vascular). A balloon catheter is used to support the guidewire during subintimal progression.
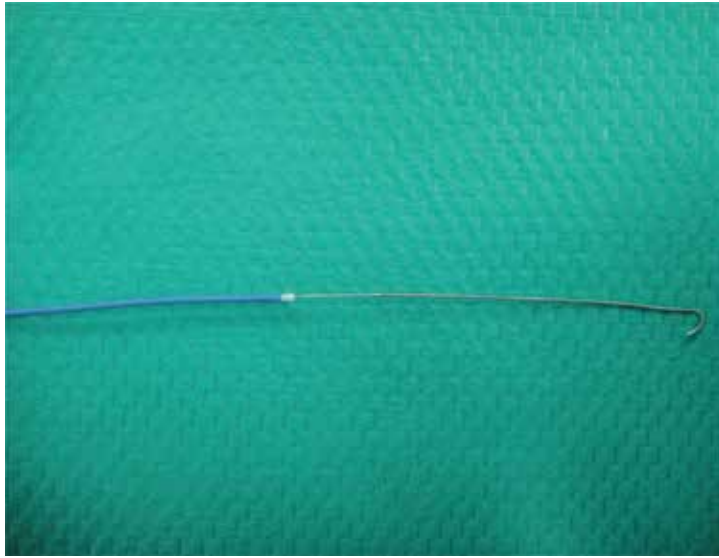
Figure 29. A 0.018-inch 1.5-mm J-tip wire (NEXUS NT 1.5J; Future Medical Design, Saitama, Japan).

4.7. Retrograde recanalization for total occlusion

When antegrade wiring cannot pass the lesion, such as the inability to reenter the patent distal true lumen after subintimal recanalization, a retrograde approach is the choice to cross the total occlusion. In these situations, the retrograde approach is an effective and safe technique for CTO interventions. Compared to other techniques such as the pedal plantar loop technique and/or collateral wiring, retrograde distal access is easier and less risky without damaging the collateral vessels. In our daily procedures, distal retrograde access is the best technical strategy for reentry or to resolve any problems.

4.8. Techniques for distal puncture

All tibial vessels, including the anterior tibial, posterior tibial, and peroneal arteries, can be accessed in retrograde fashion. Generally, the distal anterior tibial artery or posterior tibial artery is selected because these approaches are easier to establish hemostasis under external compression. The peroneal artery can be punctured but it is difficult to achieve hemostasis. The degree of vessel opacification can be enhanced using vasodilators through the femoral access site to maximize the caliber of the arterial target. Usually, nitroglycerin is administered from the distal tip of a catheter. Patient cooperation and proper use of sedation are of utmost importance for the success of this approach. Local anesthesia at the puncture site should be minimized to make the precise puncture into the 1–2-mm vessel. The position of the foot during the access procedure is also important. When puncturing the dorsal pedis or anterior tibial artery, the foot is placed in plantar flexion (**Figure 30**).

When accessing the posterior tibial artery, we place the foot in eversion and dorsal flexion (**Figure 31**).
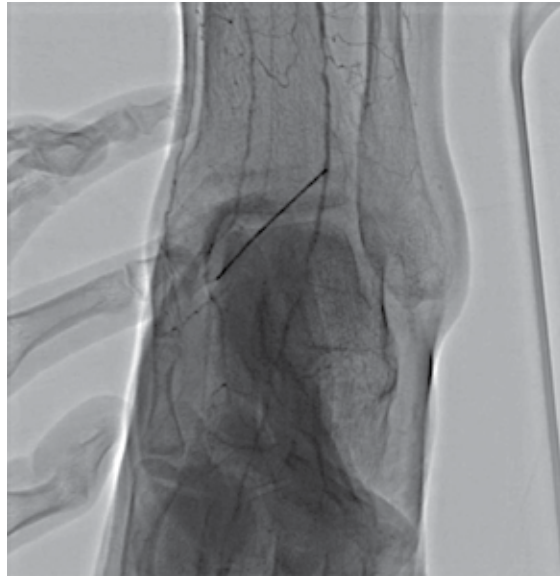
Figure 30. The dorsal pedis artery can be accessed percutaneously under fluoroscopic guidance with a micro-puncture. When accessing the dorsal pedis or anterior tibial artery, the foot is placed in plantar flexion.
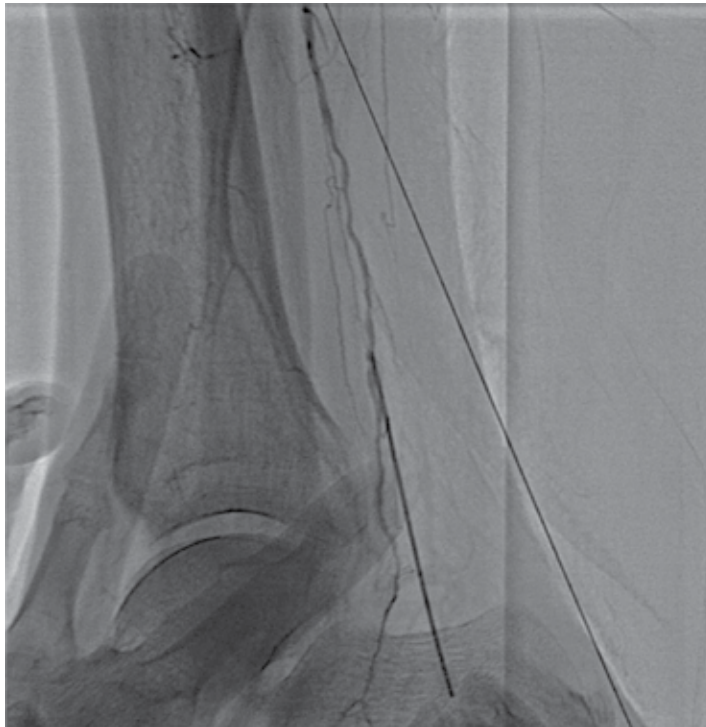
Figure 31. When accessing the posterior tibial artery, the foot is placed in eversion and dorsal flexion.

Selection of the puncture point is of vital importance in the cannulation of a 0.014-inch wire so that it is necessary to find a fairly normal vessel site. A micro-puncture needle (Cook Medical Inc., Bloomington, IN, USA) is best suited for this purpose. Under fluoroscopic guidance, a small amount of contrast is injected through a proximal vessel, most often the popliteal artery. After successful puncturing which is confirmed by back bleeding, the 0.014-inch hydrophilic wire (Cruise; Asahi Intec, Tokyo, Japan) is passed through the needle into the vessel under fluoroscopic guidance (**Figure 32**). The needle is then removed and inserted into a micro-catheter (Ichibanyari; Kaneka Medical Products, Osaka, Japan). This sheathless strategy can reduce the risk of disrupting the access vessel and is easier to achieve hemostasis (**Figure 32**).

Figure 32. The retrograde micro-catheter is withdrawn and hemostasis is secured through digital compression and/or low-pressure balloon dilatation with appropriately sized balloons.

4.9. Complications

A vessel perforation by wiring could happen while attempting to cross a tibial chronic total occlusion. In most cases, bleeding due to wire perforation is of no clinical significance. However, the perforated site must be sealed. If the wire has crossed the lesion, balloon infla-tion is applied with low pressure and, in most cases, hemostasis can be established. However, when bleeding cannot be stopped or the wire did not cross the lesion, external compression guided by angiography or temporary balloon occlusion at the proximal to perforated site

should be tried. In conjunction with proximal balloon occlusion, an external compression method should also be performed. The Tometa-Kun compression system (Zeon Medical, Tokyo, Japan) is generally used to achieve complete hemostasis at the radial artery. The Tometa-Kun compression system is easy to use and quite an effective method for external compression (**Figure 33**).

Figure 33. Perforation site is compressed with Tometa-Kun device which allows effective eternal compression.

5. Indigo carmine angiography

There are many issues regarding local perfusion problems of the foot. A non-healing ulce-ris often caused by macrovascular obstructions in combination with disease at the level of microcirculation in the foot. In addition to macrovascular ischemia, microcirculation,

particularly in a diabetic foot, is often damaged. Endovascular revascularization of the macrovessels is the first-line treatment; however, there are no validated tests to predict the clinical outcome after successful revascularization and often ulcer healing is the hard end point.

5.1. Indigo carmine

In the field of oral surgery, the injection of indigo carmine to tumor-feeding arteries has been used to determine the main feeding artery to the tumor. After confirming the color change of indigo carmine, the super-selective administration of chemotherapy infusions is performed [10]. The application of indigo carmine in patients with CLI has previously been reported, providing visual information regarding foot perfusion at the microcirculation level (**Figure 34**) [11].

Figure 34. The application of indigo carmine in patients with CLI has been reported, providing visual information regarding foot perfusion at the microcirculation level.

5.2. The angiosome concept

The angiosome concept was introduced by Taylor and Palmer who showed the foot and ankle area to be composed of six distinct angiosomes supplied by the anterior tibial artery, posterior

tibial artery, and peroneal artery [12, 13]. The ATA supplies the dorsal side of the foot and toes while the PTA supplies the plantar side of the foot, toes, interdigital web spaces, and inner side of the heel. The PA supplies the lateral ankle and lateral side of the heel (**Figure 35**) [8].
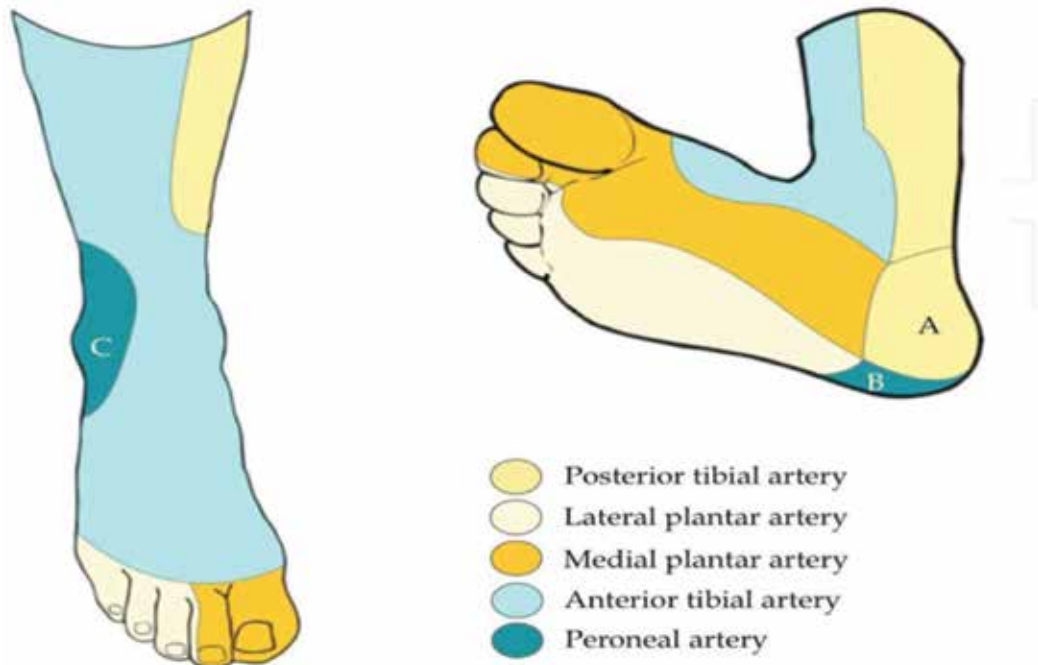
Figure 35. Six distinct angiosomes supplied by the anterior tibial artery (ATA), posterior tibial artery (PTA), and peroneal artery (PA).

However, there are extensive connections between below-the-knee arteries coursing through-out the foot and the inter-angiosome connections by choke vessels could be more important for revascularization in ischemic foot. Whether angiosome-guided EVT is essential for revas-cularization is not yet well elucidated since there is no objective method to evaluate foot per-fusion after successful revascularization.

5.3. Indigo carmine angiography

Indigo carmine angiography to provide visual information is not a difficult process. Generally, 5-ml indigo carmine is injected after endovascular therapy. If the indigo carmine is injected from the distal portion of the below-the-knee artery, indigo carmine angiography shows the territory of the vessel. If indigo carmine is injected from the distal popliteal artery, indigo car-mine angiography shows all of the below-the-knee feeding territories (**Figure 36**).

Indigo carmine angiography depicts microcirculation clearly by making the circulated area visible with blue dye. If the ulcerated area remains undyed after the EVT procedure, we can

speculate that sufficient blood flow was not established, informing us of the necessity for further revascularization with either distal bypass or EVT. Indigo carmine angiography is thus a completely new and effective method of evaluating distal microcirculation.

Figure 36. Indigo carmine angiography clearly depicts microcirculation by making the circulated area visible with blue dye.

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Nonatherosclerotic Peripheral Artery Disease

Osami Kawarada

Additional information is available at the end of the chapter

Abstract

Nonatherosclerotic peripheral artery disease (NAPAD) remains underappreciated compared to atherosclerotic peripheral artery disease. However, under- or misdiagnosis of NAPAD can potentially lead to serious adverse outcomes. There is a broad spectrum of disorders including vasculitis, thrombophilia, and other vascular anatomical or functional disorders in the context of NAPAD. This section briefly overviews vascular imaging, mainly invasive angiography, to optimize the management of NAPAD.

Keywords: awareness, nonatherosclerosis, peripheral artery disease

1. Introduction

Nonatherosclerotic peripheral artery disease (NAPAD) remains underappreciated compared to atherosclerotic peripheral artery disease (APAD) due to its low prevalence. Despite common symptoms such as claudication, rest pain, and tissue loss, most clinicians are unfamiliar with the diagnosis of NAPAD. NAPAD should be suspected clinically in younger patients, and in older patients with few atherosclerotic risk factors, few atherosclerotic features, or unusual lesion distributions. There is a broad spectrum of pathophysiologies in NAPAD, with the most common being arterial wall abnormalities, abnormal external and internal forces, spasm, vasculitis, and thrombophilia [1]. Under- or misdiagnosis of NAPAD can lead to serious adverse outcomes that, with awareness of its distinctive symptoms and signs, may be avoided or minimized [2–5]. Thus, this section briefly overviews vascular imaging, mainly invasive angiography, to optimize the management of NAPAD.

2. Workup for differential diagnosis

When there is a high clinical index of suspicion of NAPAD, the combination of blood examination (biochemical and serological tests) and vascular imaging is an integral part of the differential diagnosis process (**Figure 1**).
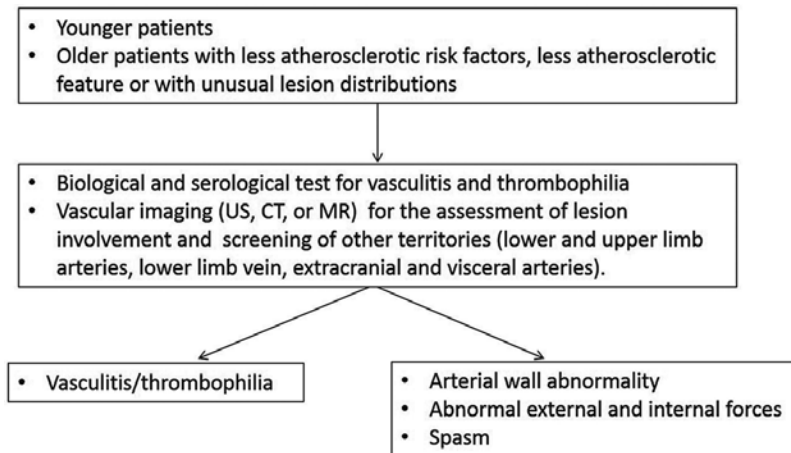
Figure 1. Approach to diagnosis of nonatherosclerotic peripheral artery disease.

In combination with vascular imaging, the assessment of macrocirculation by the ankle-brachial index is important. Furthermore, in regard to microcirculation assessment, either skin perfusion pressure (SPP) or transcutaneous oxygen pressure (TcPO$_2$) can be applied in patients with critically ischemic limbs. Limb ischemia in younger patients warrants a high clinical suspicion of NAPAD. Even in older patients, most cases of NAPAD may be underdiagnosed or misinterpreted as an atherosclerotic condition.

3. Arterial wall abnormalities

3.1. Fibromuscular dysplasia

Fibromuscular dysplasia (FMD) is a noninflammatory disease that produces arterial narrowing, aneurysms, dissection, and occlusion. Although the cause is unknown, 90% of cases occur in females [6], most frequently in the renal and carotid arteries, followed by the mesenteric artery. Although lower extremity arteries are less commonly affected, FMD is one of the most significant causes of NAPAD [4, 7]. Pathologically, FMD can mainly be classified into three types, that is, intimal, medial, and perimedial. Angiographic classification identifies the multifocal type with multiple stenoses and the so-called "string-of-beads" appearance, the tubular type, and the focal type. The "string-of-beads" sign that is frequently associated with the medial type is the most indicative of FMD, while the tubular and focal types may mimic atherosclerotic lesions [8] (**Figure 2**).
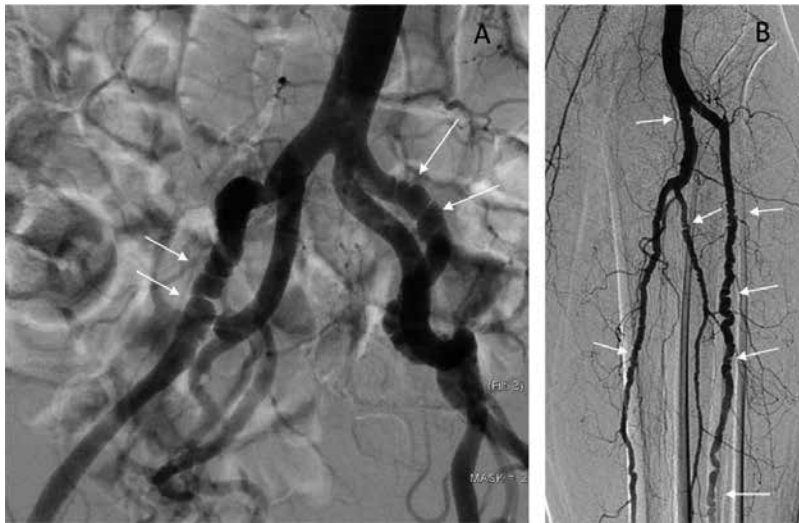
Figure 2. Fibromuscular dysplasia. (A) String-of-beads appearance in bilateral external iliac arteries (arrows). (B) Multiple string-of-beads appearances in the left crural artery (arrows).

There are isolated reports of FMD mimicking vasculitis such as polyarteritis nodosa, Takayasu's arteritis (TA), and other disorders such as Ehlers-Danlos's syndrome, Alport's syndrome, and pheochromocytoma [9].

3.2. Adventitial cystic disease

Adventitial cystic disease is characterized by a collection of mucin in the adventitial layer, typically in the popliteal artery. In rare cases, the external iliac artery or femoral artery can be affected. This disorder is typically observed in middle-aged persons, with a male-to-female ratio of 5–15:1 [4, 10]. Duplex ultrasound is considered a reasonable first-line method to diagnose adventitial cystic disease. Although stenotic lesions may develop into occlusion, the angiographic findings show a smooth, eccentric, and extrinsically narrowed appearance (**Figure 3**).

3.3. Midaortic syndrome

Coarctation of the aorta is mostly located just distal to ligament arteriosum. Midaortic syndrome (MAS) is a rare condition characterized by coarctation of the abdominal aorta or distal descending thoracic aorta and thought to arise from an embryonic development disorder [2]. It is essential to differentiate MAS such as involvement of the abdominal aorta from other causes in large-vessel vasculitis. In addition to idiopathic MAS, the association of MAS with neurofibromatosis, FMD, mucopolysaccharidosis, Alagille syndrome, and William's syndrome could be a genetic etiology. Others include tuberous sclerosis, retroperitoneal fibrosis, moyamoya disease, congenital rubella syndrome, epidermal nevus syndrome, and autosomal dominant supravalvar aortic stenosis syndrome [11, 12]. The most common anatomic type of MAS is suprarenal (60%), followed by intrarenal (25%) and infrarenal (15%) (**Figure 4**).
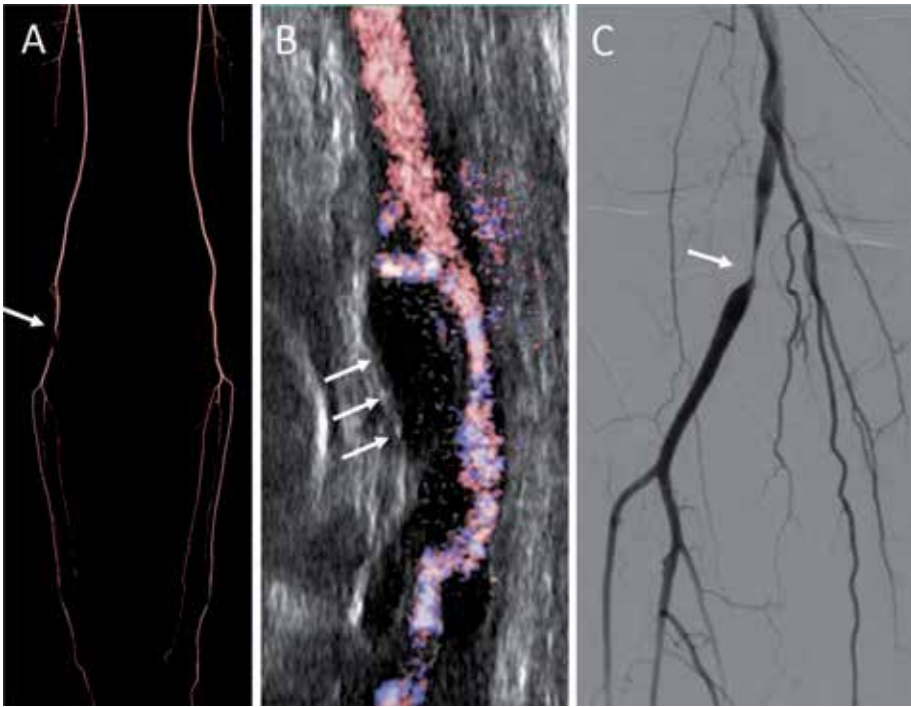
Figure 3. Adventitial cystic disease. (A) A 70-year-old male presenting moderate claudication. Enhanced CT shows focal stenosis in the midsegment of the right popliteal artery (arrow). (B) Ultrasonography revealed a low-echoic cystic lesion along the vessel wall causing significant stenosis in the popliteal artery (arrows). (C) Angiography could confirm focal stenosis in the right popliteal artery (arrow).
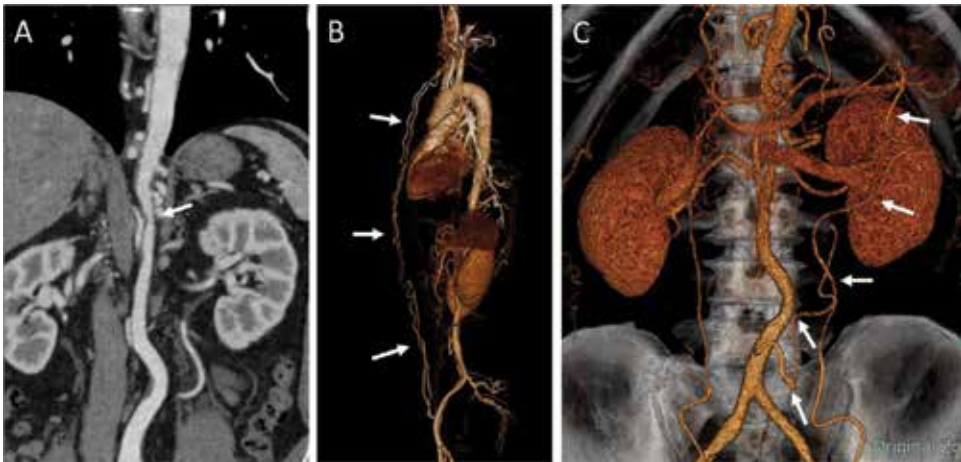
Figure 4. Idiopathic midaortic syndrome. A 51-year-old male presenting renovascular hypertension. Although the ABI was 0.70/0.67 (right/left), intermittent claudication was absent. (A) Enhanced CT showing suprarenal abdominal aortic coarctation below the origin of the superior mesenteric artery (arrow). (B) Lateral view demonstrates Winslow's pathway which is a collateral vessel developing from the subclavian arteries, internal thoracic (mammary) arteries, superior epigastric arteries, inferior epigastric arteries into the external iliac arteries (arrows). (C) Anteroposterior view AP view reveals the Arc of Riolan which is a mesenteric meandering artery between the superior and inferior mesenteric arteries (arrows).

It is usually discovered during workups for hypertension in children. Renal vessels, mesenteric vessels, or both may also be affected to varying degrees. According to previous reports, if the syndrome is left untreated, the majority of patients will die from complications of severe hypertension and ischemia by the age of 40 because of myocardial infarction, heart failure, intracranial hemorrhage, or aortic rupture [13, 14]. Recent study suggests that good long-term outcomes of MAS can be obtained by medical management [15]. Intermittent claudication might be an uncommon clinical presentation compared to manifestation of hypertension.

4. Abnormal external and internal forces

4.1. Endofibrosis

Endofibrosis typically involves the narrowing of the external iliac artery in young athletes such as cyclists, runners, triathletes, and skaters [16]. The disorder is characterized by intimal thickening and subsequent narrowing of the artery by collagen fibers, fibrous tissue, and smooth muscle proliferation [17]. The pathogenesis is presumed to involve repetitive vessel stretching during extreme hip flexion, external compression by psoas muscle hypertrophy, repeated vessel kinking during exercise, and shear stress during high cardiac output. This disorder is progressive and may lead to occlusion, frequently occurring (85%) unilaterally on the left. In addition to the external iliac artery (85%), the common femoral artery (5%) and superficial femoral artery (<5%) can be affected [4]. Since no specific angiographic findings are observed, a high clinical suspicion of this disorder is required for diagnosis and proper treatment.

4.2. Popliteal artery entrapment

Popliteal artery entrapment can be caused by compression of the popliteal artery in the popliteal fossa by adjacent or surrounding musculotendinous structures and ligaments. This disorder can occur bilaterally (30–67%) and is predominant in young males, although cases of elderly patients up to the age of 70 have been reported [4, 18]. The condition may become evident when the popliteal artery is abnormally positioned, or in cases of fibrous bands or abnormal muscle insertions or slips. There are six types of entrapment based on the anatomical compression of the popliteal artery [1]. Computed tomography (CT) angiography or magnetic resonance (MR) angiography may be useful techniques for identifying the structures causing external compression of the artery (**Figure 5**). Angiography may also reveal medial or occasionally lateral displacement of the popliteal artery if it is still patent. However, the position of the popliteal artery may be normal if the compression is due to the plantaris or popliteus muscles. In addition, pre-stenotic or post-stenotic dilatation can be associated with this disorder (**Figure 5**). Although popliteal artery narrowing induced by extension of the knee and dorsiflexion of the foot may support the diagnosis of this condition, there is some concern regarding the potential for false-positive results since popliteal artery compression can occur with active plantar flexion even in healthy individuals [19].
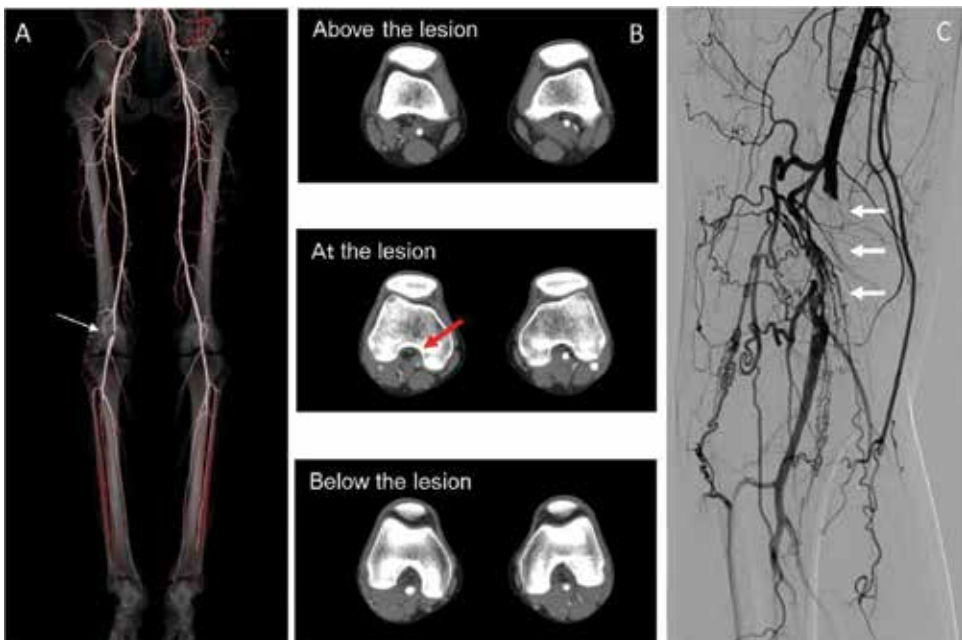
Figure 5. Popliteal artery entrapment syndrome. A 30-year-old male presenting moderate claudication. (A) Enhanced CT revealed tight stenosis and post-stenotic dilatation in the right popliteal artery (arrow). (B) Horizontal view of the CT showing compressed popliteal artery by the surrounding muscle (arrow). (C): Subsequent angiography revealed the progression to occlusion (arrows).

4.3. Adductor canal outlet syndrome

Adductor canal outlet syndrome involves the compression of the distal superficial femoral artery by the adductor canal. It is most commonly reported in runners and skiers, who present with exercise-induced intermittent claudication symptoms and paresthesias. Symptoms are typically chronic but can progress to occlusion and cause acute limb ischemia due to thrombus. This condition may be rare but it is possible relationship to acute intimal injury and thrombosis should be considered in order to save limbs that may otherwise be lost [20–22].

4.4. Other conditions

Other conditions including neoplasma, pseudoxanthoma elasticum, and Baker's cyst can cause lower limb ischemia [4].

5. Vasospasm

Vasospasm can occur even in the lower extremity arteries. There are a variety of causes, including idiopathic or certain vasospastic agents (e.g., ergotamine, cocaine, marijuana, and amphetamine) [23, 24]. The characteristic findings of drug-induced vasospasm are bilateral, symmetric, and abrupt narrowing of any segment of a lower limb artery. Vasospasm can be resolved by discontinuing the offending drug or administering vasodilators (**Figure 6**).
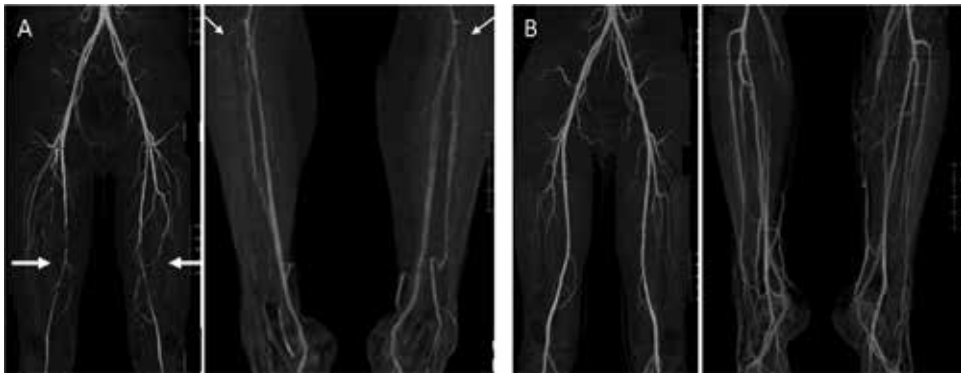
Figure 6. Idiopathic vasospasm (Ref. 24). A 28-year-old male presenting claudication with subsequent acute limb ischemia. (A) Diagnostic enhanced CT angiogram showing tight narrowings in the bilateral femoropopliteal segments (large arrows) and disruptions in the bilateral anterior tibial arteries (small arrows). Also, the proximal segment in the superficial femoral artery seems to be spastic. (B) After initiation of medical treatment during hospitalization, complete recovery of the disruptive lesions is observed though the crural arteries are superimposed on the veins.

6. Vasculitis

Vasculitis may confuse clinicians since it comprises a heterogeneous group of disorders characterized by inflammation and necrosis of blood vessels. However, the key to diagnosis when considering the possible presence of some type of vasculitis is to employ a multidisciplinary approach that involves rheumatologists as well as vascular specialists. Based on the size of the arteries involved and the underlying cause, vasculitis can be categorized as large vessel, medium vessel, or small vessel. The effects of vascular damage including arterial narrowing, thrombosis, or aneurysm formation become prominent over the course of these conditions. Invasive angiography is the gold standard for detecting such lesions and can be used to measure the trans-lesional pressure gradient. However, there are some concerns regarding invasive angiography for vasculitis. First, sheath or catheter insertion may cause vascular injury in the presence of active inflammation. Second, the potential exists for hypersensitivity reactions to the contrast dye as well as contrast nephropathy and volume overload. Moreover, invasive angiography does not provide any information on changes in vasculitis activity.

Takayasu's arteritis and giant cell arteritis (GCA) are typical large- and medium-vessel vasculitis that affect the aorta and its main branches, including the subclavian, carotid, vertebral, renal, mesenteric, and iliac arteries. The affected aortoiliac arteries may cause lower limb ischemia. Behcet's disease and Buerger's disease are representative conditions affecting various-sized arteries and venous systems. Medium-vessel vasculitis mainly comprise polyarteritis nodosa, anti-neutrophil cytoplasmic antibodies (ANCA)-related vasculitis (granulomatosis with polyangiitis (GPA), microscopic polyangiitis, and eosinophilic granulomatosis with polyangiitis), and Kawasaki disease. Assessment of the patient's clinical background and systemic examination are indispensable for the diagnosis of this vasculitis. They can also potentially emerge in an atypical vascular bed for each disorder, mimicking other types of vasculitis [25].

6.1. Takayasu's arteritis

This inflammatory vasculitis of large and medium elastic arteries, also called nonspecific aortitis, is characterized pathologically by giant cell infiltration and granuloma formation. Destruction of the entire vascular wall and progressive adventitial fibrosis can cause stenosis or dilatation that can be complicated with superimposed calcification at the chronic stage. This disorder is typically but not exclusively observed in young women of Asian or Latin descent. It primarily affects the aorta, its major branches, and the pulmonary arteries, including but not limited to the brachiocephalic, carotid (common carotid), vertebral, subclavian (proximal subclavian), renal, iliac, femoral, and coronary arteries. Clinically, it usually first presents in the second or third decade, but can occur at older ages. Many patients initially complain of fever, arthralgias, and malaise. Although the most common symptom of TA is arm claudication, observed in greater than 60% of cases, aortoiliac artery involvement can result in lower limb ischemic symptoms, and even the femoral artery may be involved [26, 27].

There are no serological tests to identify TA. The diagnosis of TA is based on clinical findings in the presence of compatible vascular imaging abnormalities (**Table 1**) [1].

1990 criteria for the classification of Takayasu arteritis
1. Age at disease onset <40 years
2. Claudication of extremities
3. Decreased brachial artery pulse
4. Difference of >10 mmHg in systolic blood pressure between arms
5. Bruit over subclavian arteries or abdominal aorta
6. Arteriogram abnormality (arteriographic narrowing or occlusion of the entire aorta, its primary branches, or large arteries in the proximal upper or lower extremities, not due to arteriosclerosis, fibromuscular dysplasia, or similar causes; changes usually focal or segmental)
Takayasu arteritis is defined clinically if at least three of these six criteria are present. The presence of any three or more criteria yields a sensitivity of 90.5% and a specificity of 97.8%.

Table 1. American College of Rheumatology diagnostic criteria for Takayasu arteritis.

Angiography can reveal the extent of luminal narrowing, with or without dilatation/aneurysm, in order to differentiate TA from other diseases. While CT angiography or MR angiography can provide the whole image alternative to angiography, measurement of the pressure gradients is one of the major advantages of invasive angiography (**Figure 7**). It can also provide opportunities for surgical or endovascular intervention. However, in terms of evaluating vessel wall thickening and edema, duplex US, CT, and MR are more informative than angiography.

6.2. Giant cell arteritis

Although giant cell arteritis is pathologically similar to TA, this type of vasculitis commonly affects the temporal artery. The disorder is observed in men and women of around 50 and older, and is particularly prevalent in patients aged 70 and older. The arteries potentially affected include the aorta and its branches, with a predilection for the distal subclavian, axillary, and proximal brachial arteries, as well as the branches of the carotid arteries, in particular the ophthalmic artery. Therefore, headaches, jaw claudication, and visual impairment can occur in addition to arm claudication. Also, a normal erythrocyte sedimentation rate (ESR) is more useful in excluding giant cell arteritis than an elevated ESR is in diagnosing this disease [1] (**Table 2**).

Figure 7. Takayasu arteritis. A 20-year-old female presenting mild claudication and renovascular hypertension. Invasive angiography revealed significant stenosis in the descending thoracic aorta (arrow). The pullback pressure gradient was 20 mmHg.

1990 criteria for the classification of giant cell arteritis

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1. Age at disease onset <50 years
2. New headache
3. Temporal artery abnormality
4. Elevated erythrocyte sedimentation rate
5. Abnormal artery biopsy (biopsy specimen with artery showing vasculitis characterized by a predominance of mononuclear cell infiltration or granulomatous inflammation, usually with multinucleated giant cells)

Giant cell arteritis is defined clinically if at least three of these five criteria are present. The presence of any three or more criteria yields a sensitivity of 93.5% and a specificity of 91.2%.

Table 2. American College of Rheumatology diagnostic criteria for giant cell arteritis.

Lower limb arteries can also be affected by this disorder [28, 29]. According to positron emission tomography scan studies, the iliac artery was involved in 37% of cases and the femoral artery in 37% (subclavian artery 70%, axillary artery 40%) [30]. Other studies have reported that superficial femoral artery was involved in 33%, common femoral artery in 14%, internal iliac artery 11%, deep femoral artery in 6%, and popliteal artery in 6% of cases [4, 31–34]. US studies have also detected the involvement of distal lower limb arteries such as the femoropopliteal, tibial, and peroneal arteries [35]. It is sometimes challenging to differentiate lesions from atherosclerosis in older patients.

Findings of arteritis from a temporal artery biopsy can be supportive but not essential for a diagnosis. An accurate diagnosis of GCA requires a comprehensive approach that includes assessment of clinical manifestations, physical examination, laboratory studies, vascular imaging, and arterial biopsies. Positive temporal artery biopsies can occasionally be seen in other types of arteritis and the ESR may be normal in up to 10% of GCA patients so that cautious interpretation is required. Differential diagnosis of GCA should include brain disease, infectious disease, and malignant disease. It should be noted that other vasculitis such as polyarteritis nodosa or ANCA-related vasculitis may rarely present with temporal artery involvement. Polymyalgia rheumatic may also be included in this group of vasculitis as it is often regarded as one clinical entity with GCA.

Although angiography can confirm the extent of affected vessels, less invasive tools such as duplex US, CT angiography, and MR angiography should also be considered. In particular, CT is informative for the extent of calcification. The advantage of angiography is that it allows measurements of pressure gradients to identify hemodynamically significant lesions.

6.3. Behcet's disease

Vasculitis is observed in less than one-third of Behcet's disease cases. The etiology remains unclear and may involve both genetic and environmental factors. It can potentially be characterized by concomitant oral and genital ulcerations, skin lesions, uveitis, central nervous system, and gastrointestinal involvement. Approximately 80% of Behcet's disease patients have the human leukocyte antigen (HLA)-B51 allele. However, since no symptoms or laboratory findings are pathognomonic for Behcet's disease, diagnosis depends on the patient meeting a set of established clinical criteria. The major histopathological features of this disorder are predominantly perivascular inflammatory infiltrates and a tendency to thrombus formation in both veins and arteries of every size. In particular, venous disease is characteristic, including superficial phlebitis, varices, and thrombosis of the deep veins, vena cava, and cerebral sinuses. Large vessels frequently show luminal narrowing, aneurysm, or rupture. Medium and small vessels may also be affected [36, 37] (**Figure 8**).

6.4. Buerger's disease

Buerger's disease, also known as thromboangiitis obliterans (TAO), was first reported by Winiwarter in 1879, and later described in detail by Buergers in 1908 [38, 39]. Although the etiology remains unclear, this disorder is a segmental inflammatory disease typically affecting small- to medium-sized arteries of the upper and lower extremities, with occasional extension

to the veins and nerves of the extremities [40–42]. Atypically, multiple large vessels can be affected [43]. This condition is more common in men than in women and is almost exclusively observed in patients who use tobacco so that it is widely recognized that tobacco is associated with the onset, progression, and recurrence of the disease. Symptoms can include claudication, rest pain, and ischemic tissue loss such as ulceration and gangrene. Unlike other vasculitis, inflammatory markers such as the ESR and C-reactive protein are typically normal. Angiography is often required to evaluate lesion extent and runoff conditions since there is the potential for over- or underestimation of the lesion with MR and CT imaging. Angiographic findings include segmental arterial occlusions of small- and medium-sized vessels while large arteries are typically spared (**Figure 9**) [44].
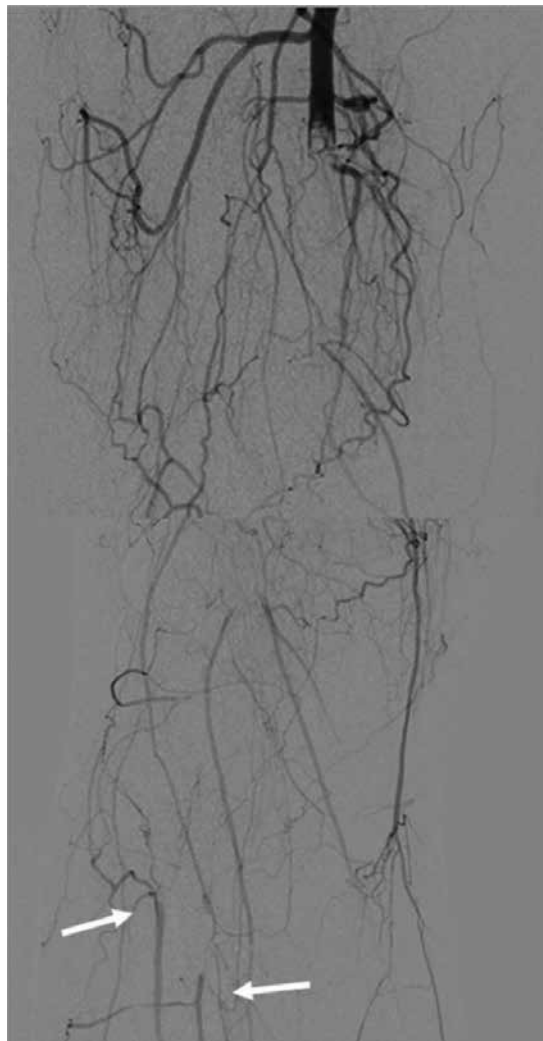

Figure 8. Behcet's disease. A 64-year-old male with a history of deep vein thrombosis and cerebral vein thrombosis presenting acute onset of rest pain and claudication in the right leg. Invasive angiography revealed right femorocrural occlusion. The proximal crural artery was reconstituted through the collateral vessels (arrows).
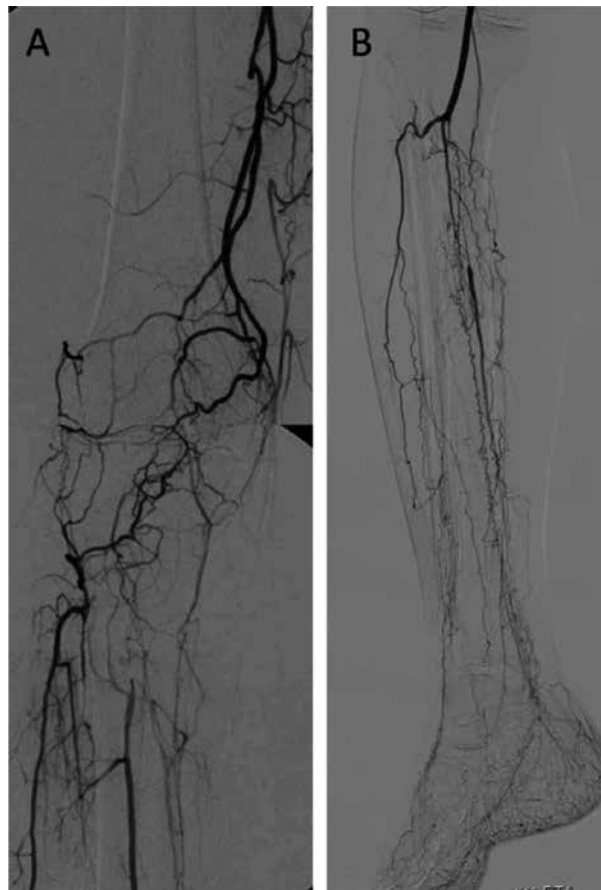
Figure 9. Buerger's disease. (A) Femorocrural occlusion beyond the right knee joint in a 38-year-old female presenting foot gangrene. (Ref. 44). (B) Crural artery occlusion in a 37-year-old male presenting right toe gangrene. Long total occlusions in the tibial arteries extending to the pedal arch.

The term "corkscrew" has recently been used to describe the appearance of collateral vessels in Buerger's disease patients. However, the original article attributes the corkscrew appearance to the recanalization of the affected native artery [45, 46]. Moreover, the corkscrew appearance is not pathognomonic for Buerger's disease as it may be seen in patients with other disorders including connective tissue disease. Thus, several different criteria have been proposed for the diagnosis of Buerger's disease (**Tables 3** and **4**) [40, 41].

- History of smoking
- Onset before age 50
- Infrapopliteal arterial occlusions
- Either arm involvement or phlebitis migrans
- Absence of atherosclerotic risk factors other than smoking

Table 3. Criteria of Buerger's disease by Shionoya [40].

- Age under 45
- Current or recent history of tobacco use
- Presence of distal extremity ischemia as indicated by claudication, pain at rest, ischemic ulcers, or gangrene, and documented by noninvasive vascular testing
- Exclusion of autoimmune diseases, hypercoagulable states, and diabetes mellitus
- Exclusion of a proximal source of emboli by echocardiography or arteriography
- Consistent arteriographic findings in the clinically involved and noninvolved limbs

Table 4. Criteria of Buerger's disease by Olin [41].

6.5. Other vasculitides

Other rare diseases, including Cogan's syndrome and relapsing polychondritis, can cause vasculitis of large- or medium-sized vessels. Small-vessel vasculitides include cryoglobulin-emic vasculitis, leukocytoclastic vasculitides such as Henoch-Schonlein purpura and isolated cutaneous leukocytoclastic vasculitis, and vasculitis secondary to systemic autoimmune disease, including rheumatoid arthritis, systemic lupus erythematosus (SLE), and scleroderma [30, 47]. It is worth noting that there is the possibility of atypical lesion distribution for any type of vasculitides (**Figures 10** and **11**).
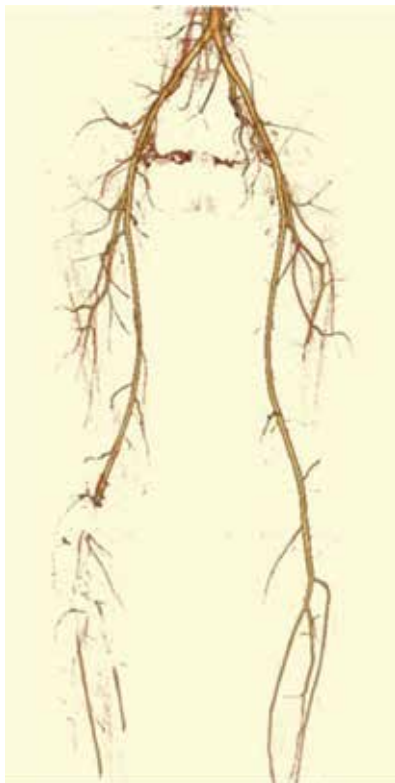

Figure 10. Systemic lupus erythematosus A 16-year-old female presenting claudication with subsequent acute limb ischemia as an initial clinical manifestation. Enhanced CT revealed severe femorocrural occlusion. Although the mid-tibial arteries were reconstituted, the distal tibial and pedal arteries were occluded.

Figure 11. Scleroderma. An 80-year-old female presenting toe gangrene with a history of scleroderma. Invasive angiography showing multiple stenosis in the right crural artery.

For example, there have been case reports of large- or medium-vessel vasculitis in patients with rheumatoid arthritis, SLE and ANCA-related vasculitis [30, 47–62], and uncommon diseases including hypereosinophilic syndrome, Kimura disease, and angiolymphoid hyperplasia with eosinophilia can also mimic Buerger's disease [63–65]. Moreover, antiphospholipid syndrome (APS) can occur concomitantly with vasculitis secondary to systemic autoimmune disease (frequently SLE), and can develop into catastrophic APS [66–73] (**Figure 10**). Certain kinds of vasculitis can be complicated by thrombosis and potentially develop into thrombotic storm which has a devastating clinical course [74–77]. Radiation arteritis can occur years after high-dose radiotherapy for pelvic malignant disease. In such cases, stenotic or occlusive disease can be seen within the radiation field. Thus, with typical and atypical cases in mind, a careful diagnostic workup is vital for vasculitis.

7. Arterial thrombophilias

There are inherited and acquired disorders in which thrombosis develops in the arterial system. Inherited disorders include hyperhomocysteinemia/hyperhomocystinuria, antithrombin deficiency, protein S deficiency, and protein C deficiency, as well as gene polymorphisms such as Factor V Leiden. Acquired disorders are more common and can be caused by APS, malignancies, hormone therapy, and such myeloproliferative disorders as polycythemia vera, thrombocythemia, heparin-induced thrombocytopenia, and thrombotic thrombocytopenic purpura (**Figure 12**) [78].
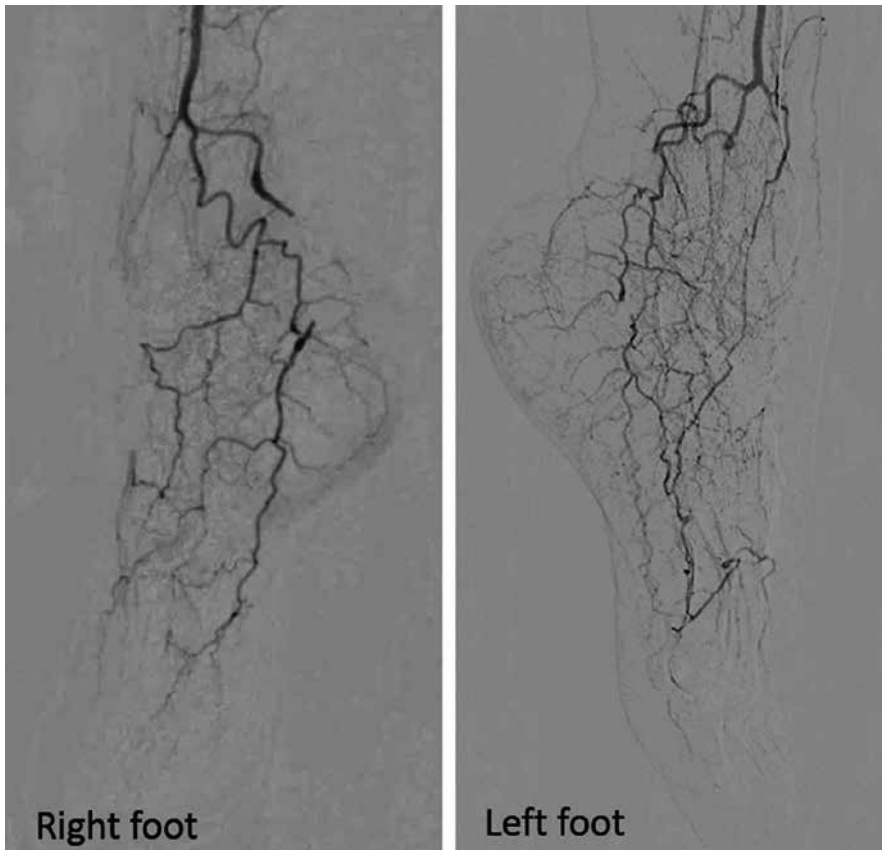
Figure 12. Primary antiphospholipid syndrome [78]. A 75-year-old female presenting symmetric peripheral gangrene in four limbs. Angiography shows the occlusions in the pedal arteries as well as the palmar arteries due to antiphospholipid syndrome.

Thrombosis can be seen in vasculitis [74, 79]. In particular, inflammation-induced thrombosis is considered to be a feature of certain kinds of vasculitis (systemic autoimmune diseases such as SLE, rheumatoid arthritis, and Sjogren's syndrome, and other vasculitis). Thus, primary or secondary APS concomitant with autoimmune diseases such as SLE can cause thrombosis and potentially develop into catastrophic APS [80–82].

8. Congenital variants

Congenital malformations of the iliofemoral arterial system are rare, but accurate diagnosis is essential to avoid unnecessary revascularization treatment. Congenital absence or hypoplasty of the common iliac artery, external iliac artery, and SFA has been reported [83, 84]. Congenital variants of the external iliac artery have been classified into three groups [85]: group 1, anomalies in the origin or course of the artery; group 2, hypoplasia or atresia compensated for by persistent sciatic artery (PSA); and group 3, isolated hypoplasia or atresia. Although group 1 may not be associated with lower limb ischemia and is most often discovered at autopsy, group 2, the so-called persistent sciatic artery, and group 3 are most likely to present with lower limb ischemia.

Above all, PSA is a popular variant. Failure of regression of the sciatic artery during fetal development is often associated with superficial femoral artery hypoplasia, and the PSA then provides the dominant arterial inflow to the lower limb. Therefore, there is continuation of the internal iliac artery into the thigh through the greater sciatic notch. This variant can cause not only acute lower limb ischemia due to thromboembolism but also chronic lower limb ischemia, with a high incidence of aneurysm formation and arteriosclerosis of the sciatic artery (**Figure 13**) [4, 86].
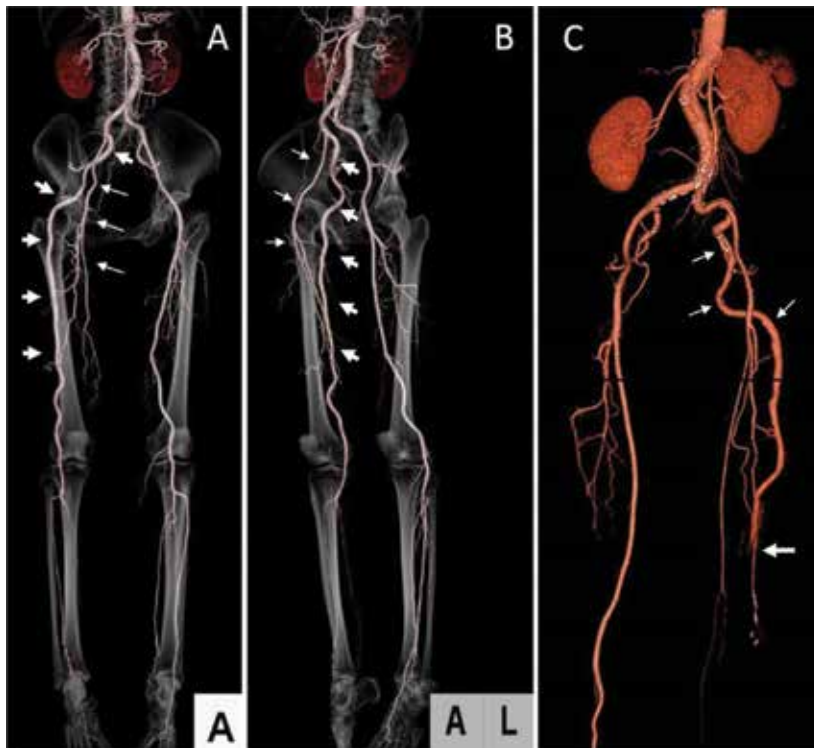
Figure 13. Persistent sciatic artery. (A, B) A 64-year-old male. Enhanced CT incidentally found persistent sciatic artery (large arrows) in the right. The right external iliac artery connects with the hypoplastic superficial femoral artery (small arrows). (C) A 60-year-old female presenting acute limb ischemia. The left external iliac artery connects with the hypoplastic superficial femoral artery whereas the persistent sciatic artery through the left internal iliac artery is the dominant blood supply (small arrows). The distal part of the sciatic artery is occluded due to thromboembolism (large arrow).

9. Embolism

Embolism can potentially cause manifestations of chronic lower limb ischemia, such as clau-dication and critical limb ischemia, but not acute limb ischemia in particular in the elderly population. Embolisms may have a number of sources including cardiac, aortic, and right-to-left shunts (paradoxical embolism from the venous circulation).

10. Vascular injury

Orthopedic surgery or trauma may cause lower limb ischemia because of dissection or throm-botic occlusion [87]. Additionally, pediatric cardiac catheterization using the transfemoral approach could be a cause of iliofemoral occlusion or stenosis due to thrombosis formation or intimal hyperplasia (**Figure 14**). This disorder may be asymptomatic until adulthood, but long-term uncorrected circulatory impairment can potentially cause limb growth retardation even in the absence of symptomatic evidence of ischemia [88–93].
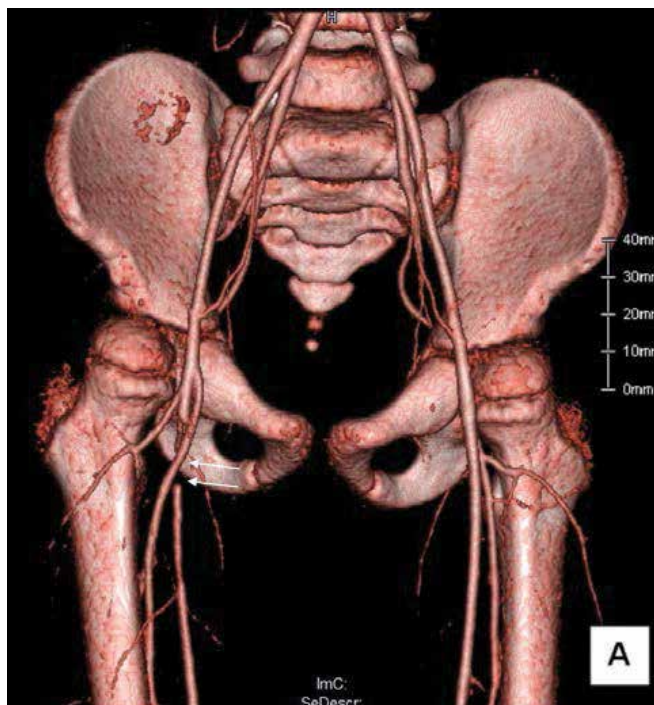

Figure 14. Vascular injury following catheterization. A 4-year-old boy experienced a pale foot on the right following catheterization. Enhanced CT revealed a short occlusion due to puncture site thrombosis in the proximal segment of the right superficial femoral artery (arrows).

11. Conclusions

This section is intended to focus on vascular imaging, mainly invasive angiography, for NAPAD. From a clinical standpoint, an increase in opportunities to experience the symptoms and signs of APAD heightens the importance of the differential diagnosis of NAPAD in daily practice. NAPAD cannot benefit from a one-size-fits-all approach compared to APAD. Thus, differentiation between NAPAD and APAD may be a challenging task but we clinicians need to increase our knowledge of the diversity of NAPAD so that such awareness can be translated into improved patient care.

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This textbook on Angiography and Endovascular Therapy for Peripheral Artery
Disease provides a comprehensive angiographic approach to assess and determine
optimal treatment strategies for peripheral artery disease (PAD). Each chapter focuses
on angiography as it relates to the outcomes of endovascular work. It is an overview of
the results obtained from a large number of actual cases with over 100 angiographic
images of aortoiliac, femoropopliteal, below-the-knee, and below-the-ankle artery
disease. Diagnostic and angiographic images of nonatherosclerotic PAD are also
included. Anyone who is engaged in or preparing to engage in endovascular work for
PAD should find this book useful as a reference and as an instruction material.

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