Chapter 3

Angiography and Endovascular Therapy for Femoropopliteal Artery Disease

Masahiko Fujihara

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/67181

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**).

http://dx.doi.org/10.5772/67181

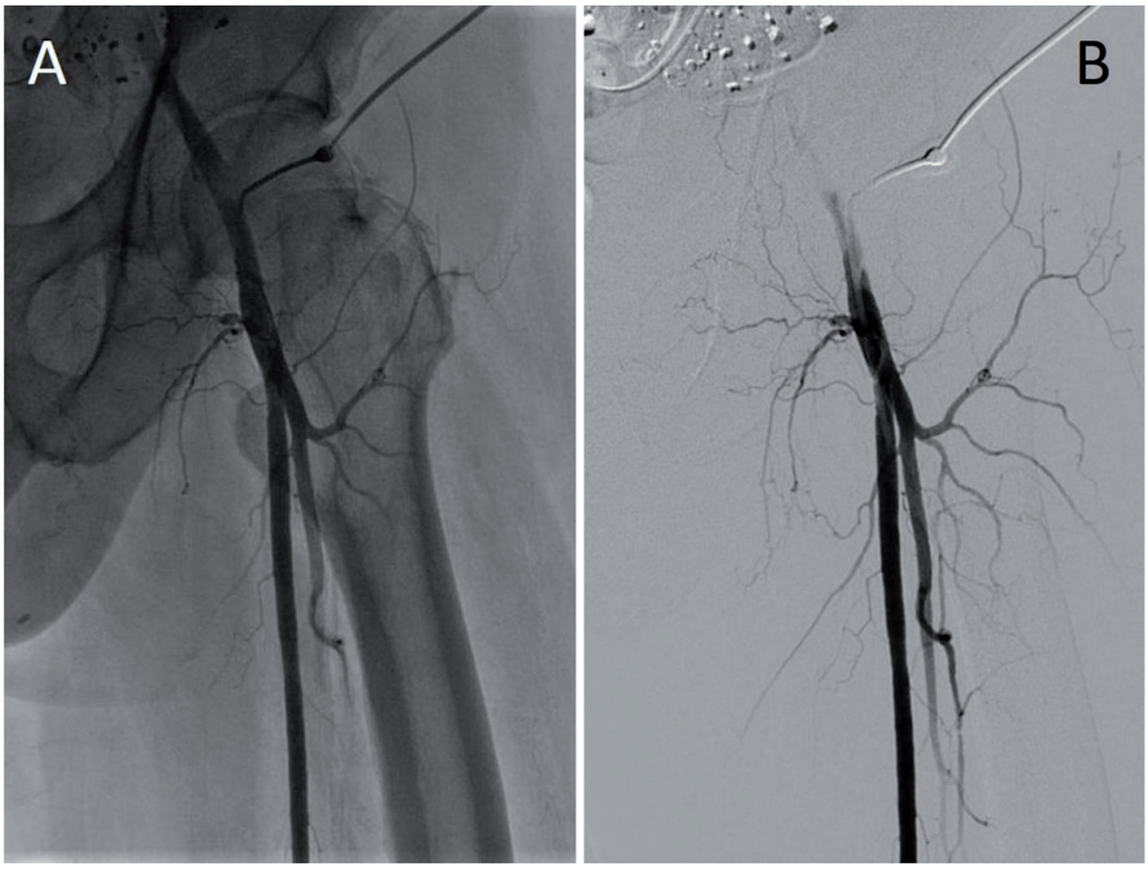

Figure 1. Anteroposterior view of proximal left femoral artery by (A) DA image; (B) DSA.

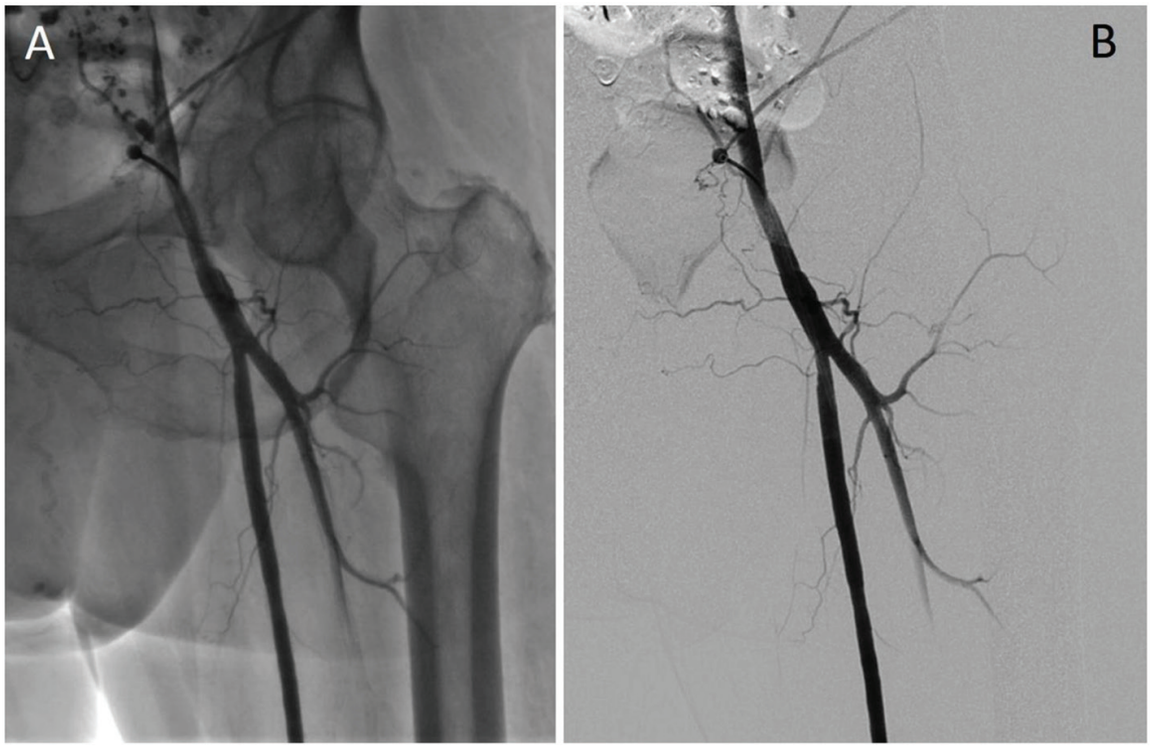

Figure 2. Left anterior oblique view of proximal left femoral artery by (A) DA image; (B) DSA.

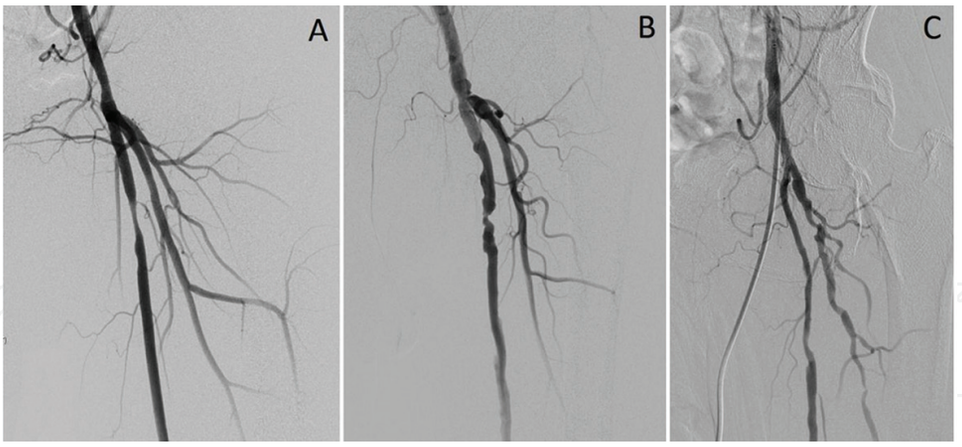

Figure 3. Left anterior oblique view for proximal left femoral diseased artery. (A) Proximal focal SFA stenosis is well visualized. (B) Complex SFA disease and ostial stenosis PFA are well visualized. (C) Both SFA and PFA are diffusely diseased.

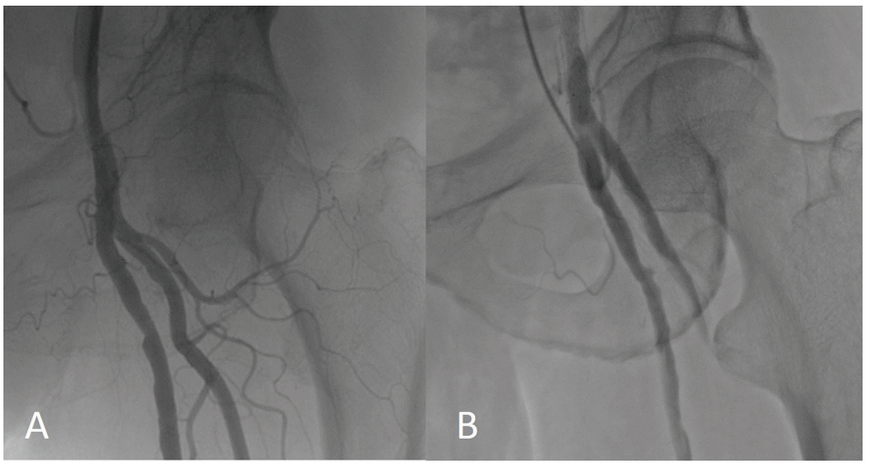

Figure 4. High take-off of profunda artery. (A) Located in zone 3. (B) Located in zone 4.

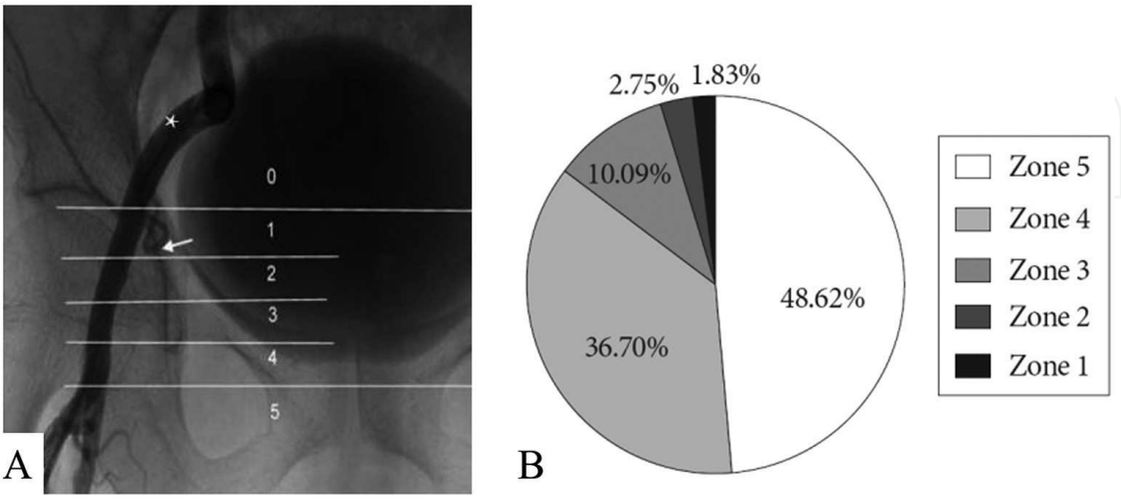

Figure 5. Angiographic anatomical study of the common femoral artery from Korea. (A) the definition of location for femoral artery bifurcation (B) the proportion of femoral artery bifurcation location, zone 5 in which bifurcation is below the femoral head was 48.6%.

http://dx.doi.org/10.5772/67181

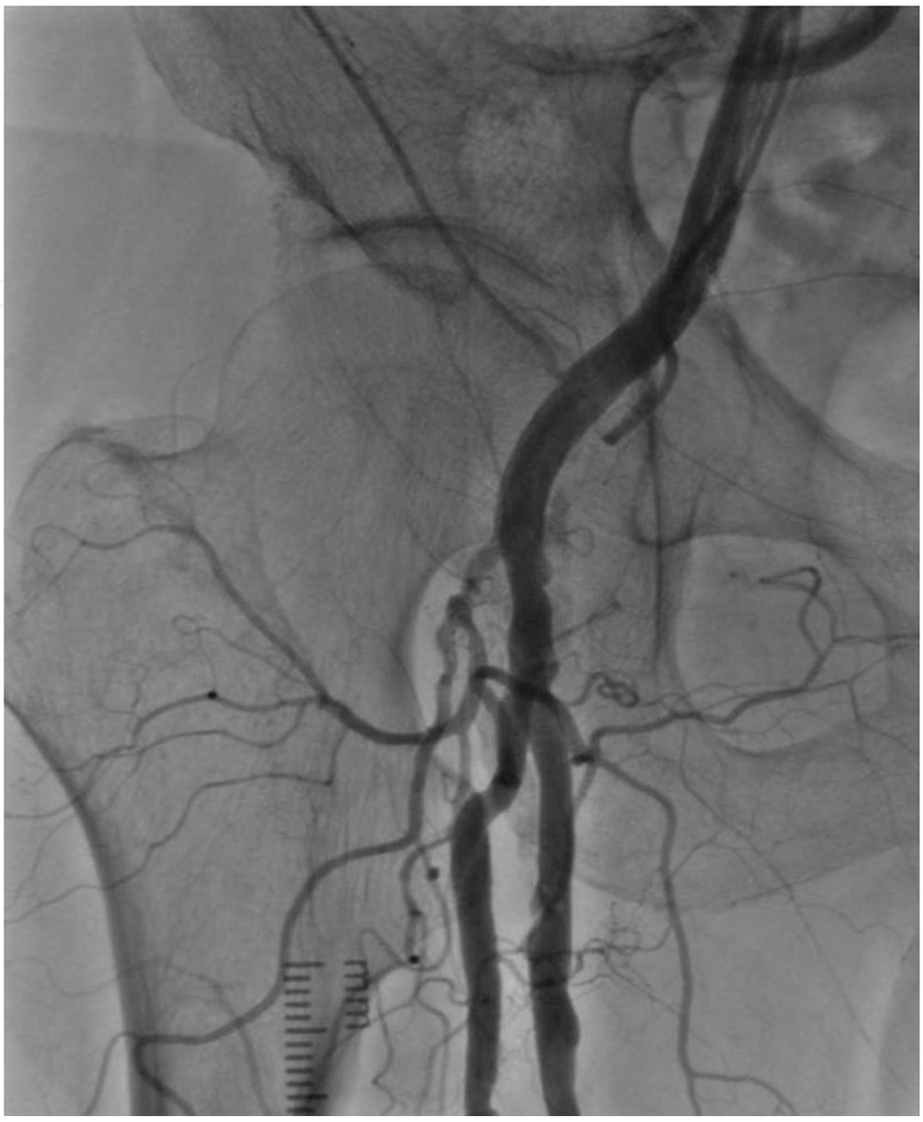

Figure 6. Low take-off of profunda artery.

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**).

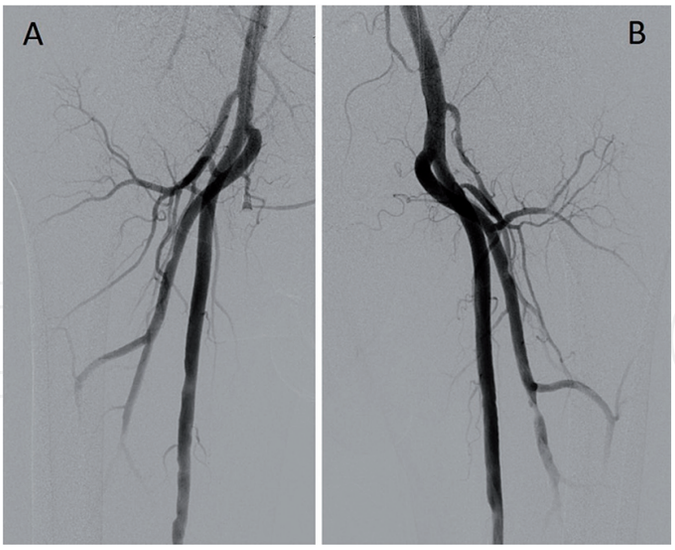

Figure 7. Variations of the profunda femoral artery. Both right (A) and left (B) deep femoral artery directed medially with separate origin of lateral circumflex artery.

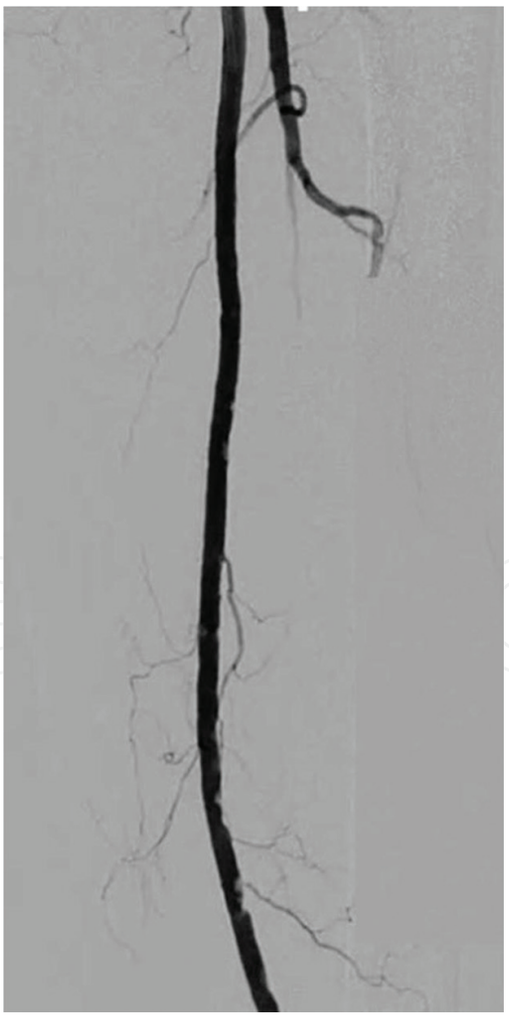

Figure 8. Anteroposterior view for middle SFA with a DSA image.

http://dx.doi.org/10.5772/67181

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.

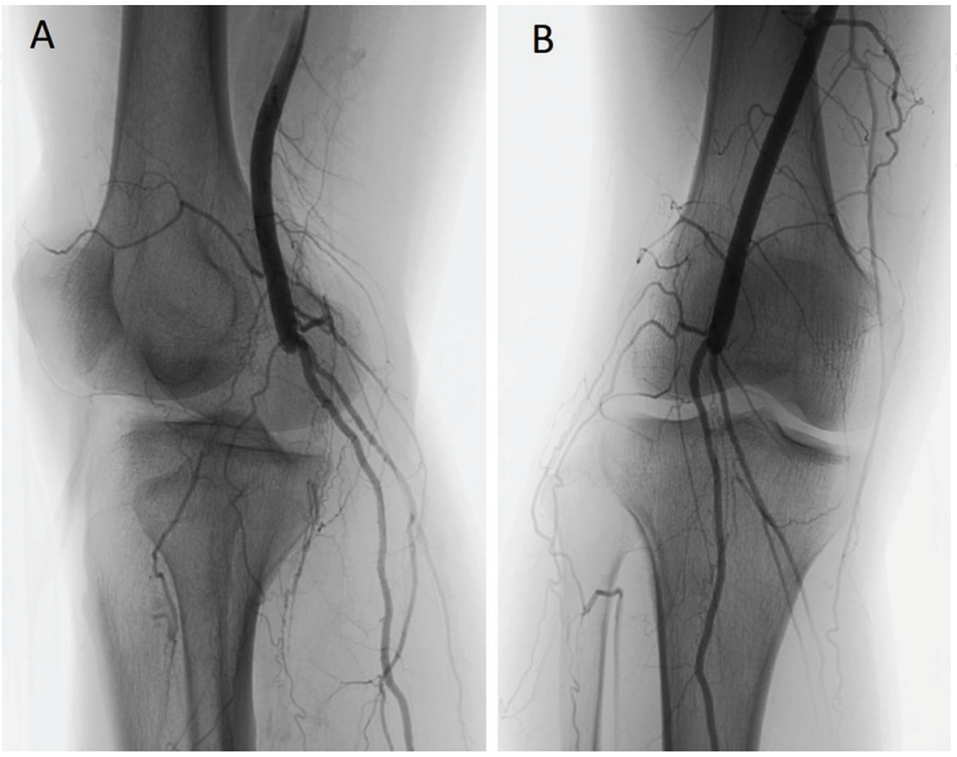

Figure 9. (A) Left anterior oblique view for distal to popliteal disease. (B) Anteroposterior view.

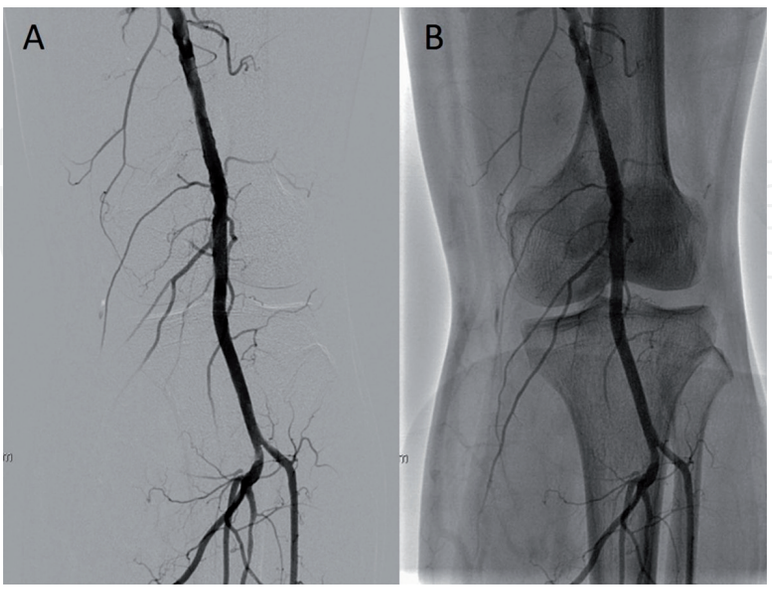

Figure 10. Anteroposterior view of left popliteal artery. (A) Left popliteal artery clearly shown by DSA image. (B) Due to boney structure overriding the popliteal artery, DA image has limitations in visualizing arterial edge and other branches.

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.

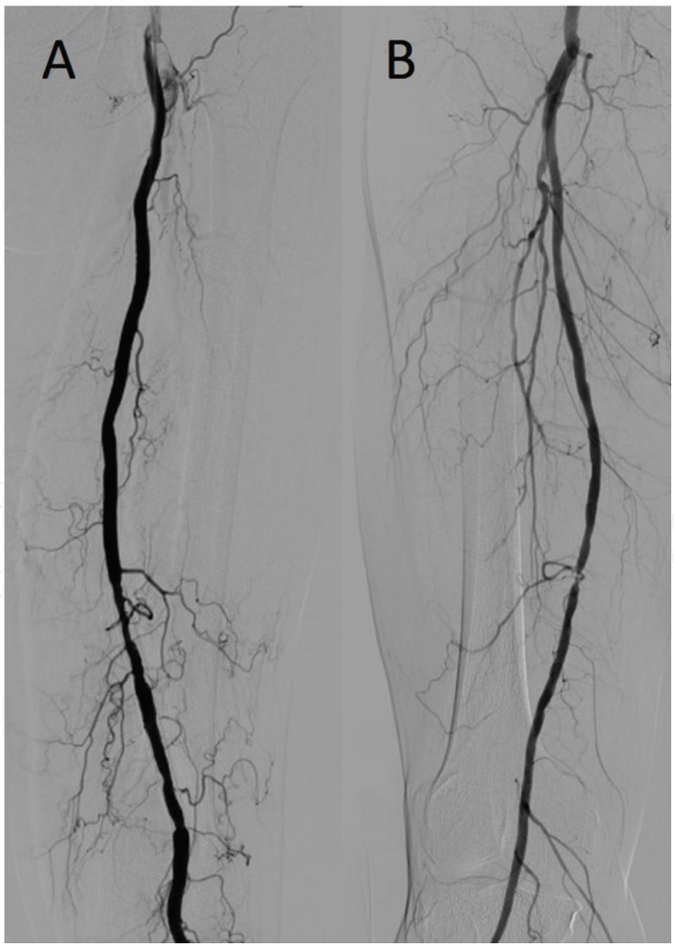

Figure 11. Bolus chase limb vessel angiography in DSA image. To see the whole femoropopliteal artery, bolus chasing is a very useful method. Angiogram can be obtained by digital subtraction with small amount of dye.

http://dx.doi.org/10.5772/67181

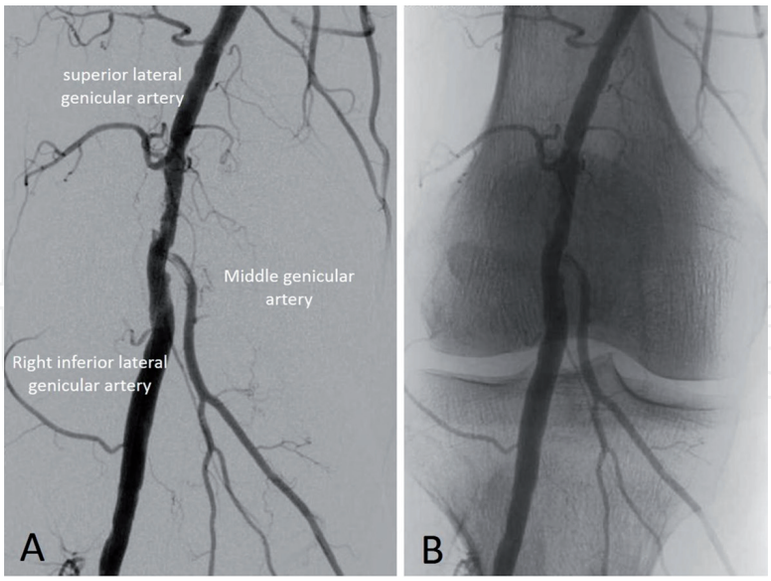

Figure 12. Anatomy of popliteal artery by (A) DSA image and (B) DA image.

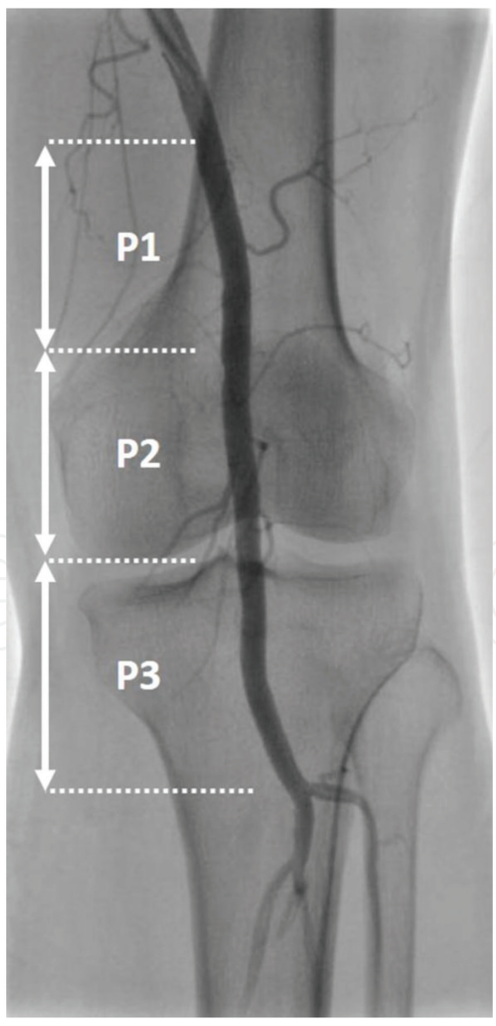

Figure 13. Definition of popliteal artery segments.

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.

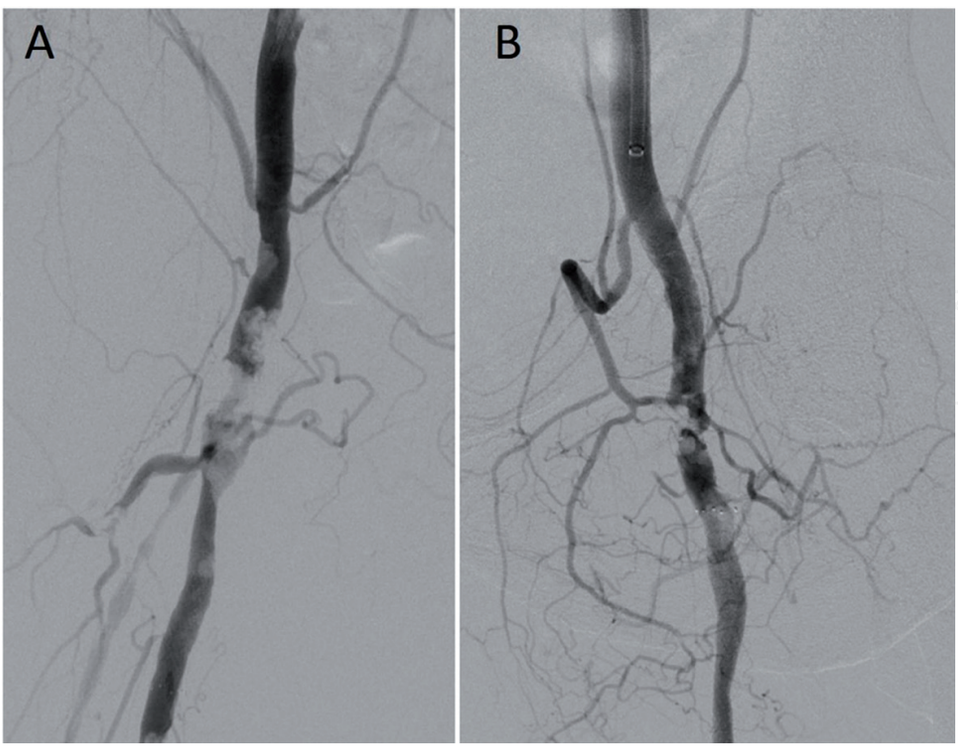

Figure 14. CFA disease with typical coral-like calcification disease on (A) right and (B) left.

http://dx.doi.org/10.5772/67181

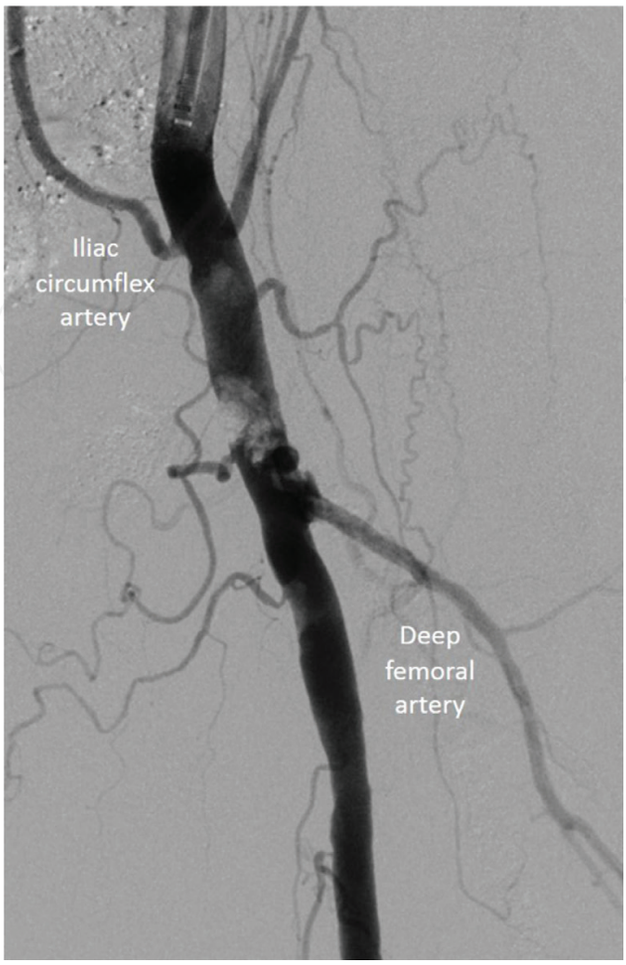

Figure 15. Identification of circumflex iliac artery and deep femoral artery bifurcation by left anterior oblique view.

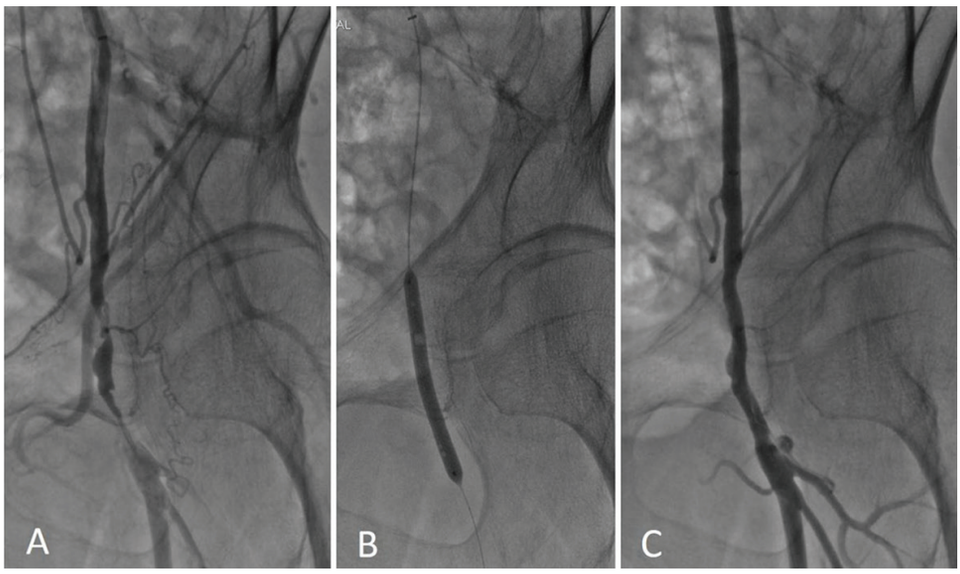

Figure 16. Balloon angioplasty for CFA lesions. (A) Preprocedure shows tight stenosis of left CFA; (B) balloon angioplasty by 5 × 40 mm balloon; (C) postprocedure shows successful angioplasty with minor dissection.

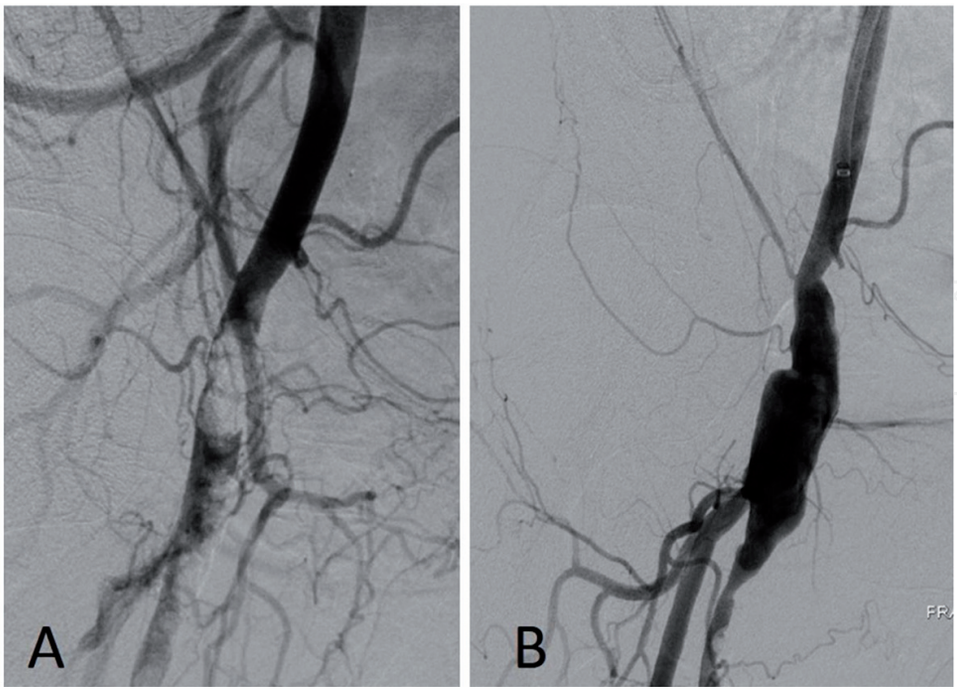

Figure 17. Endarterectomy with patch angioplasty. (A) Presurgical repair. (B) Postsurgical repair.

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**).

http://dx.doi.org/10.5772/67181

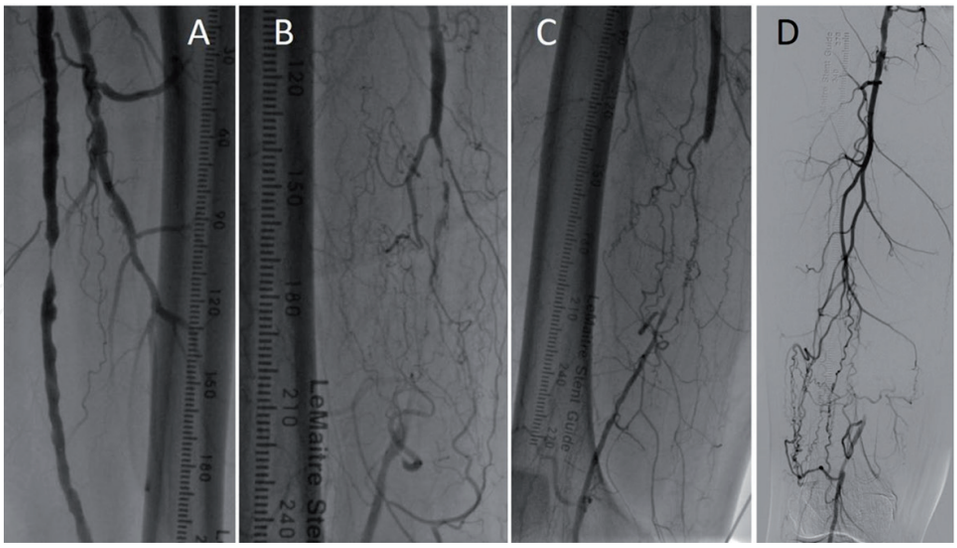

Figure 18. Focal lesion was measured at 1 cm (A). Short CTO was measured at 10 cm (B). Short CTO and stenosis measured at over 15 cm (C). SFA long CTO was measured at 20 cm (D).

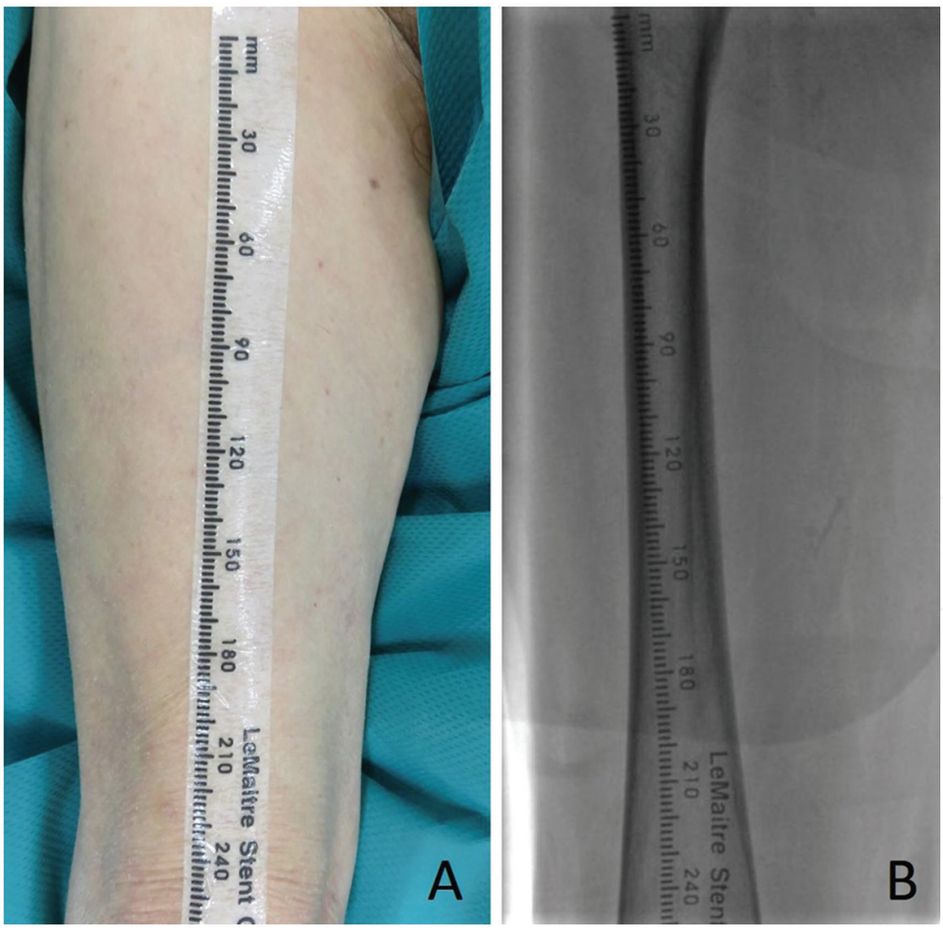

Figure 19. For lesion length measurement, a tape measure is attached to the front of the thigh.

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.

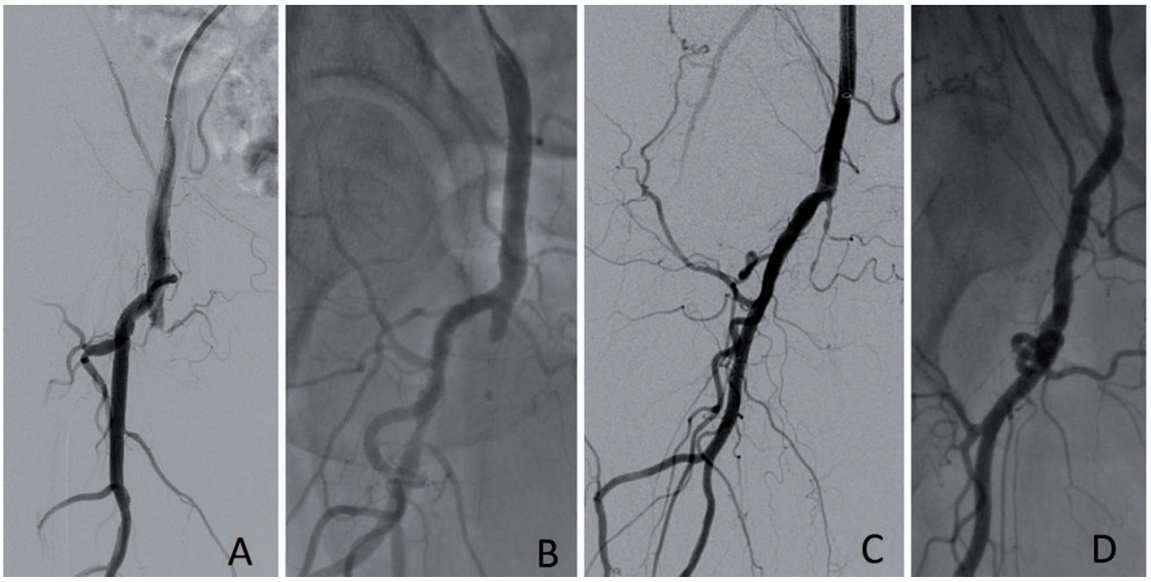

Figure 20. In right anterior oblique view, the stump of SFA ostium occlusion is well visualized by (A) DSA image; (B) DA the stump of SFA ostium occlusion is not well visualized; (C) DSA image; (D) DA.

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

http://dx.doi.org/10.5772/67181

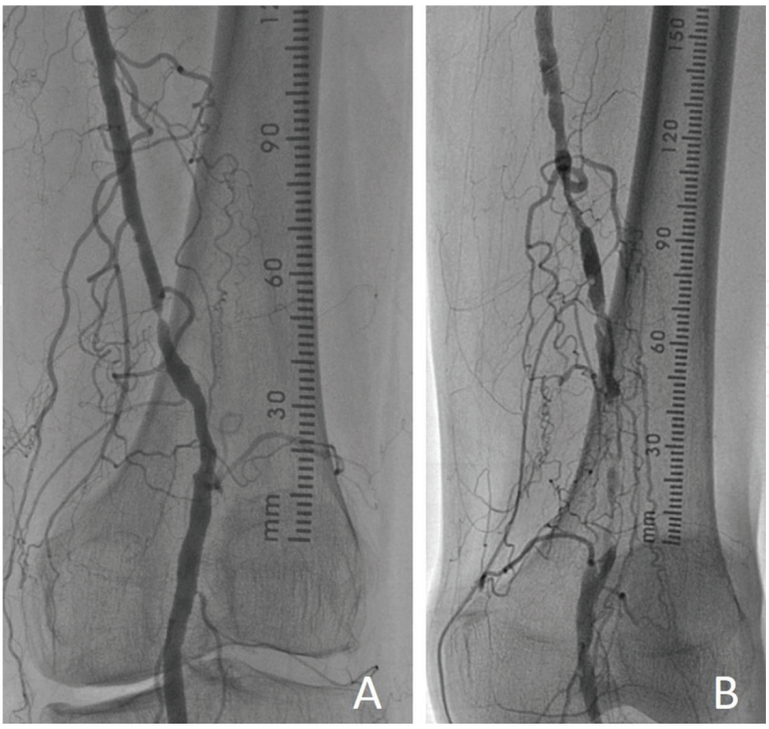

Figure 21. Distal portion of SFA lesion (A) uninvolved popliteal artery and (B) involved proximal popliteal artery.

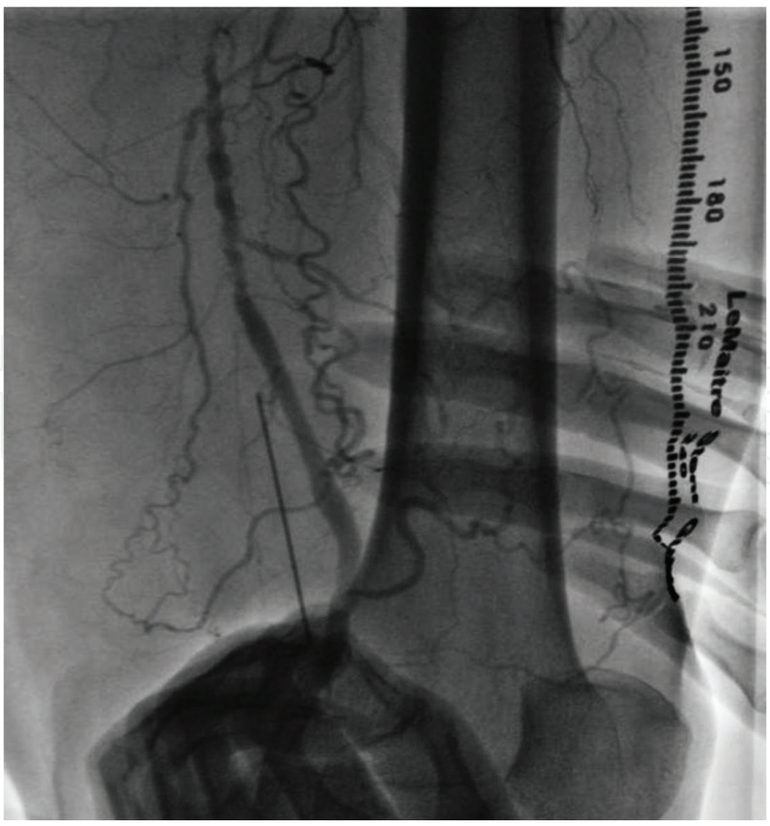

Figure 22. Puncture of distal SFA visualized from collateral flow from PFA.

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 assessment 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.

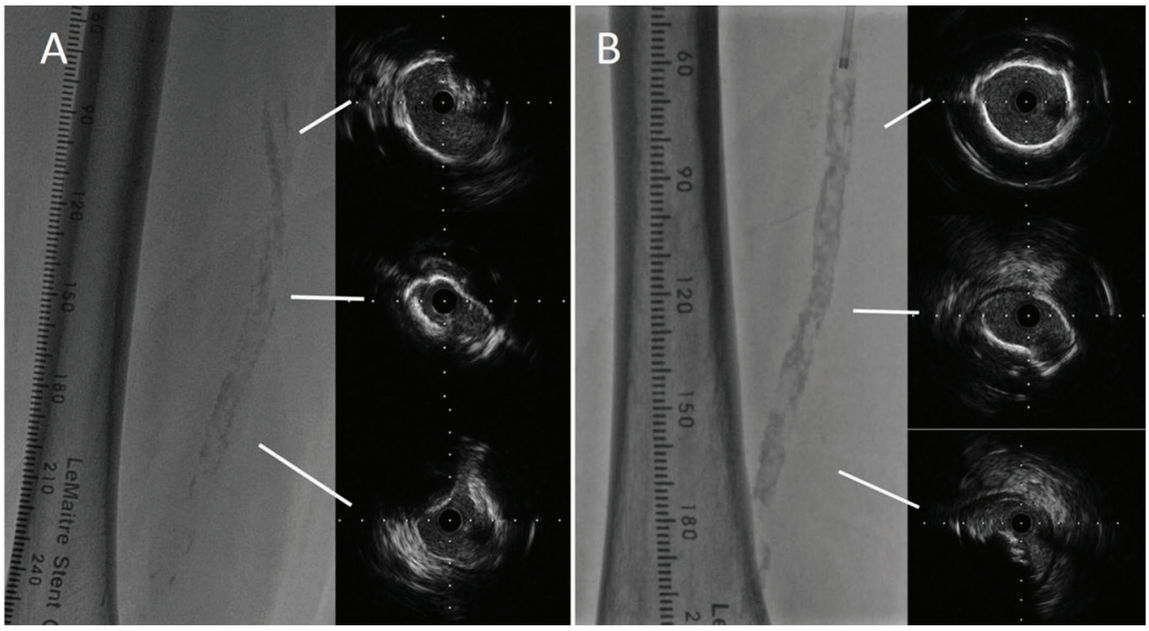

Figure 23. (A) Severe calcification of SFA in mid-portion (>15 cm) evaluated by fluoroscopy and IVUS. (B) Super severe calcification of SFA mid-portion (>10 cm) evaluated by fluoroscopy and IVUS.

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.

http://dx.doi.org/10.5772/67181

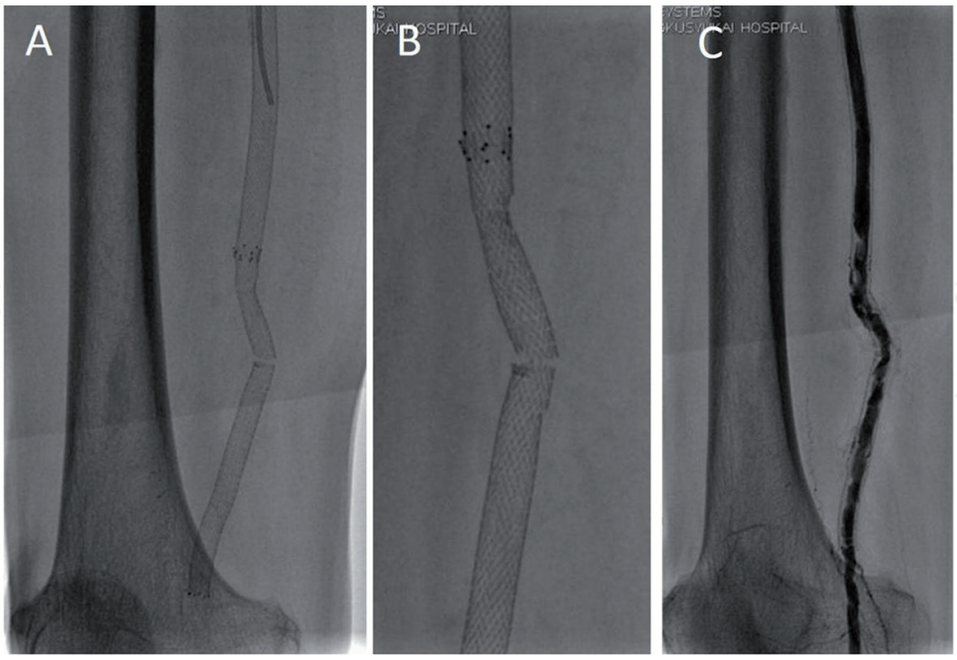

Figure 24. Stent showed complete traverse liner separation without displacement. (A and B) Type 3 stent dissection; (C) no restenosis observed at fracture site.

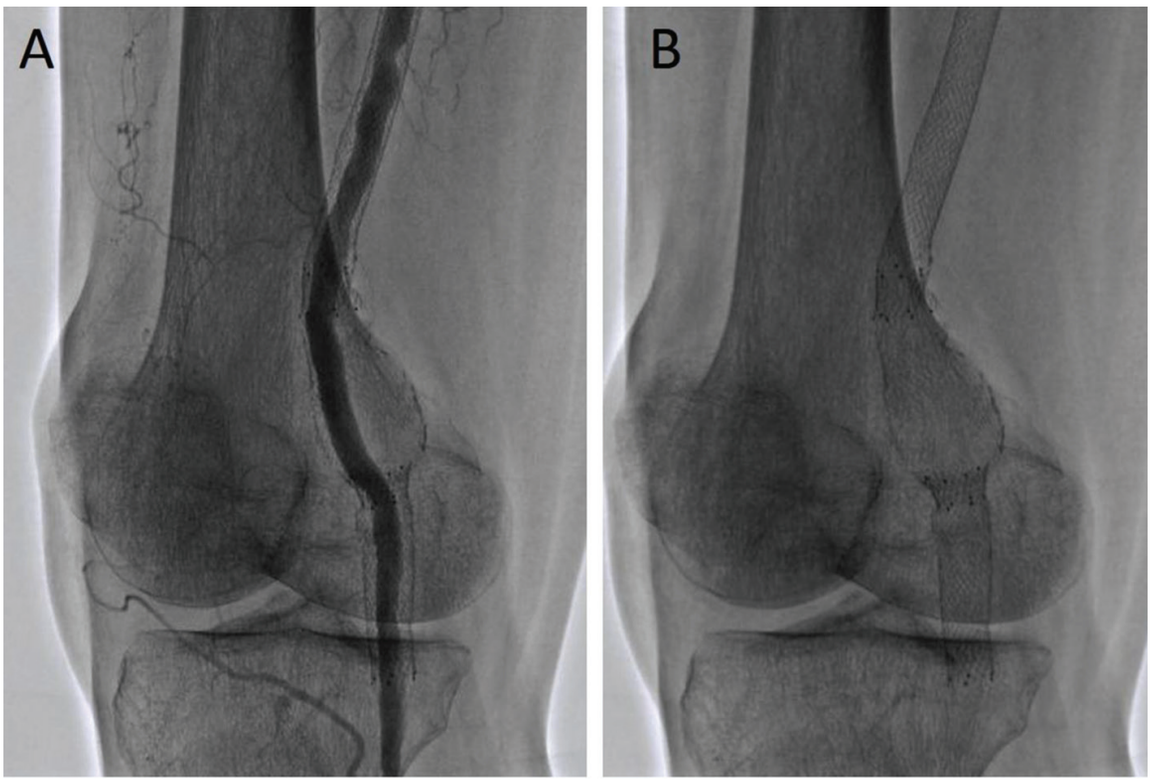

Figure 25. Complete stent fracture at distal right SFA to proximal popliteal artery. (A) The stent showed no restenosis; however, (B) complete break at SFA distal portion with aneurysm formation.

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.

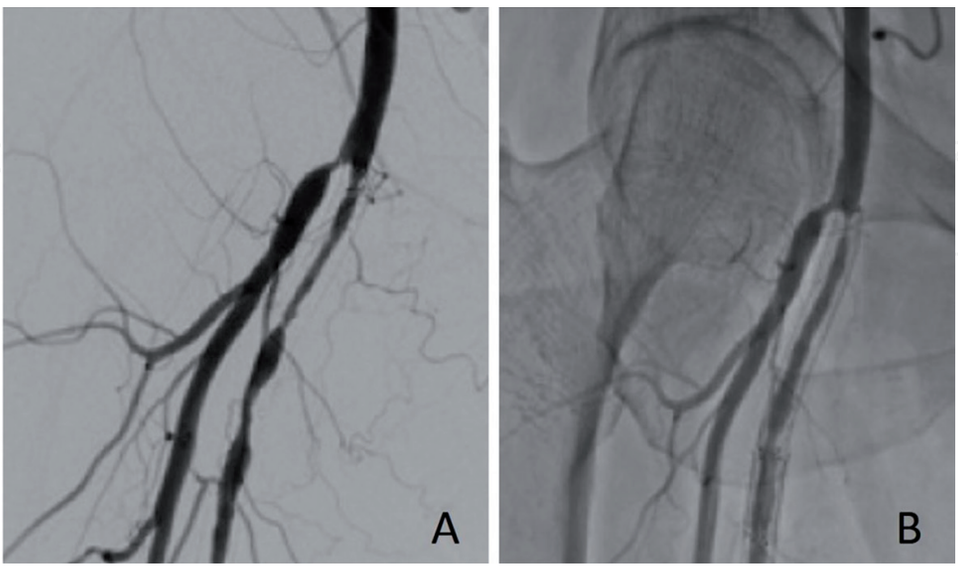

Figure 26. (A) Nitinol stent restenosis. After 6 months stent implantation angiography showed SFA stent restenosis with new stenosis in profunda femoral artery. (B) This restenosis is clearly seen in nonsubtracted angiogram.

http://dx.doi.org/10.5772/67181

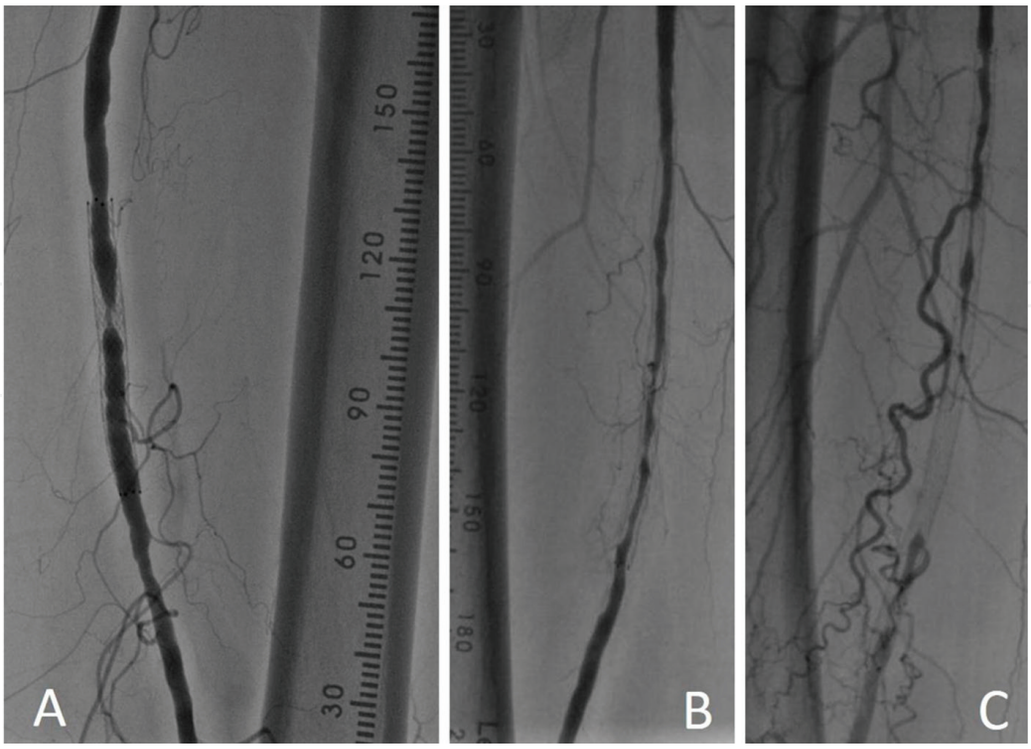

Figure 27. ISR lesions were classified by visual estimate on angiography: Tosaka class I, the focal ISR group (<50 mm in length), included lesions positioned at the stent body, stent edge, or a combination of these sites (A). Tosaka class II, the diffuse ISR group (>50 mm in length), includes not only stent body lesions but also stent edge lesions (B). Tosaka class III is the totally occluded ISR group (C).

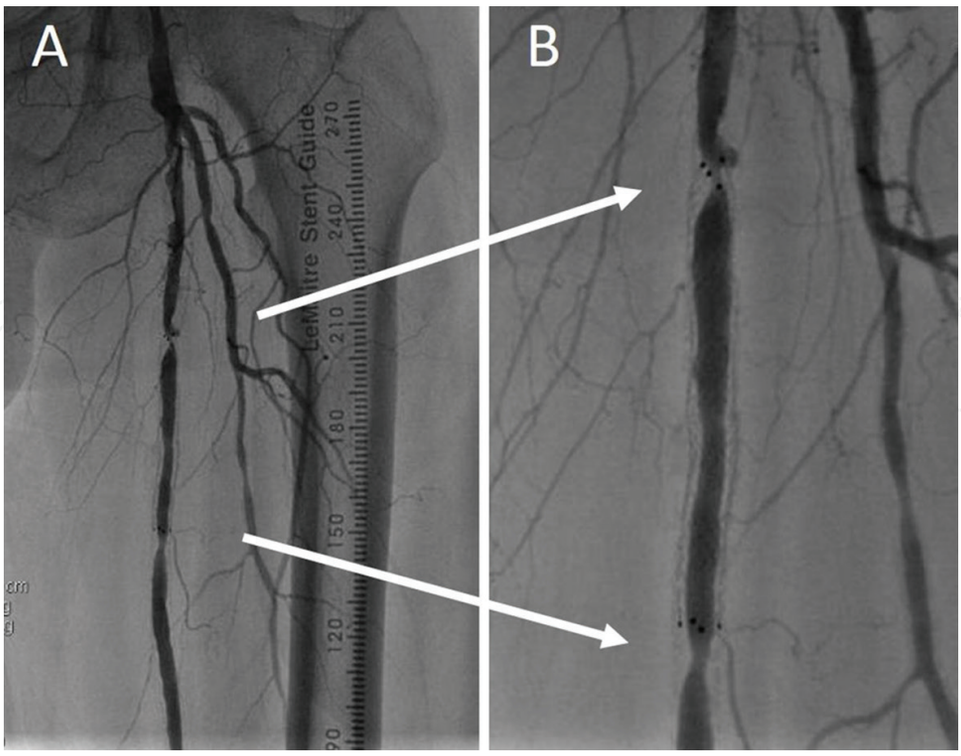

Figure 28. The case of stent edge initiated hyperplasia without in-stent restenosis.

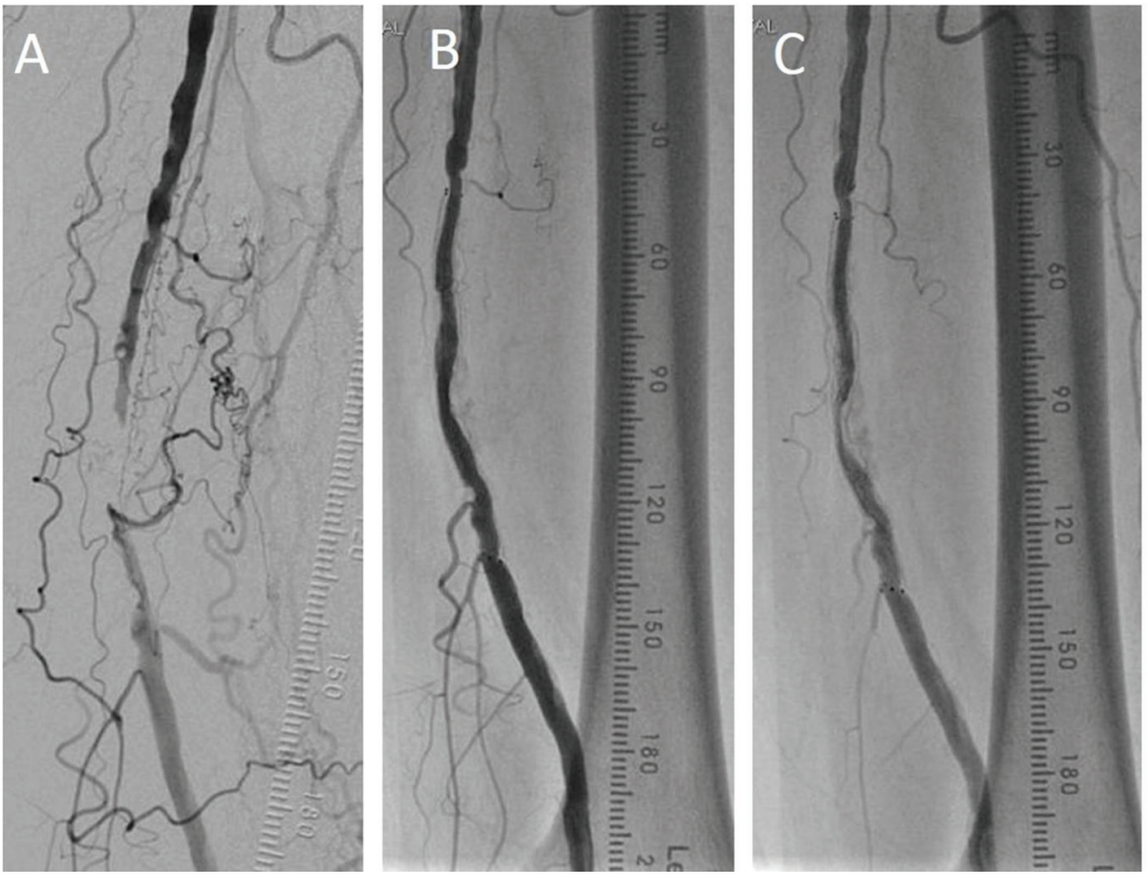

Figure 29. The case of short calcified occlusion in the SFA (A). A successful recanalization was performed by stenting (B). Restenosis was found at the site of the original occluded segment, and an eccentric calcified lesion (C).

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,

http://dx.doi.org/10.5772/67181

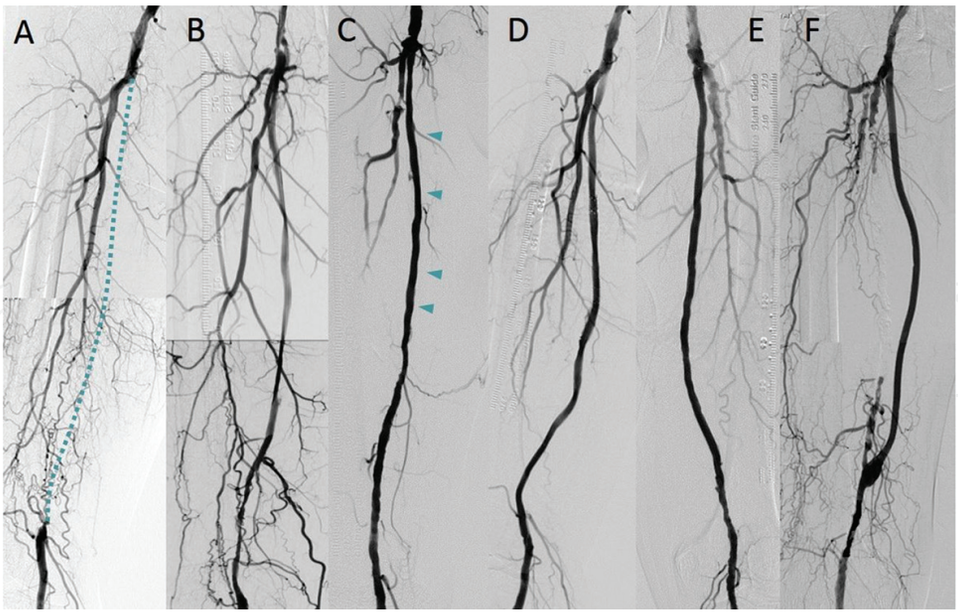

Figure 30. Different techniques to intervene in (A) long SFA disease including (B) balloon angioplasty, (C) spot stenting with short nitinol stent, (D) full cover nitinol stent implantation, (E) stent grafting, and (F) bypass surgery.

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

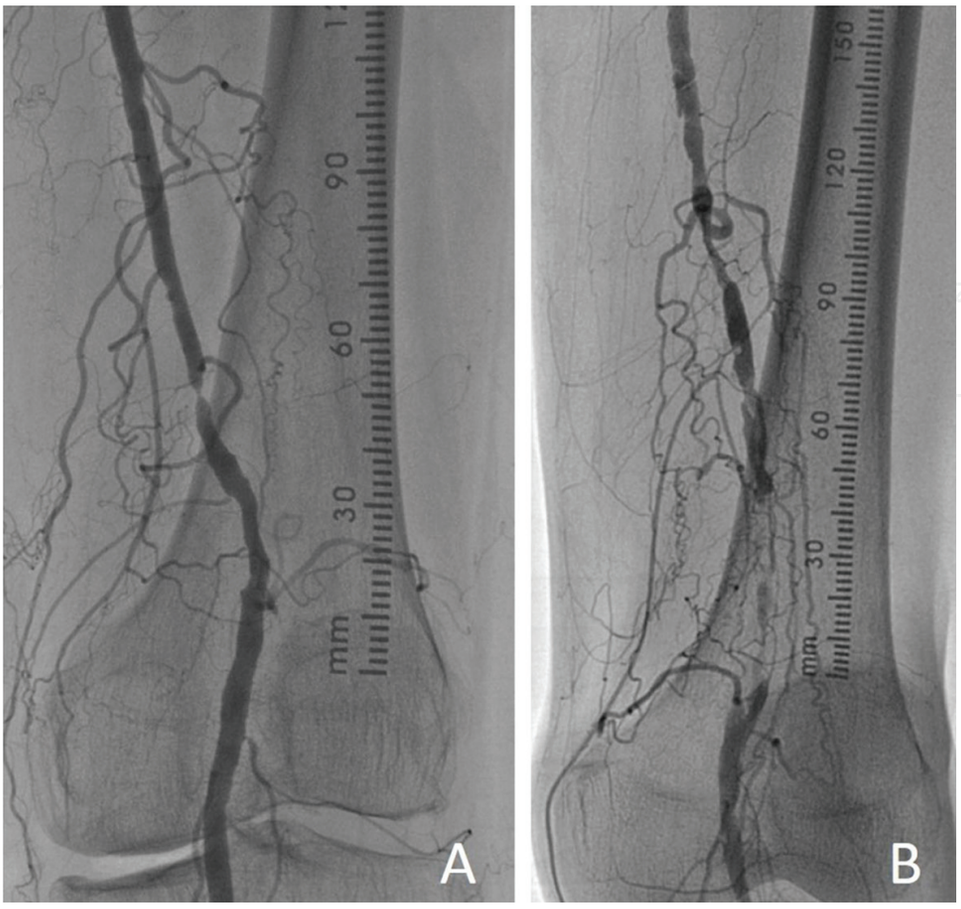

Figure 31. In a short lesion, initial approach is (A) balloon angioplasty; (B) short focal lesion in distal SFA; and (C) balloon angioplasty performed with 5 × 40 mm balloon. Postballoon angioplasty showed residual stenosis of less than 50% at ballooning site.

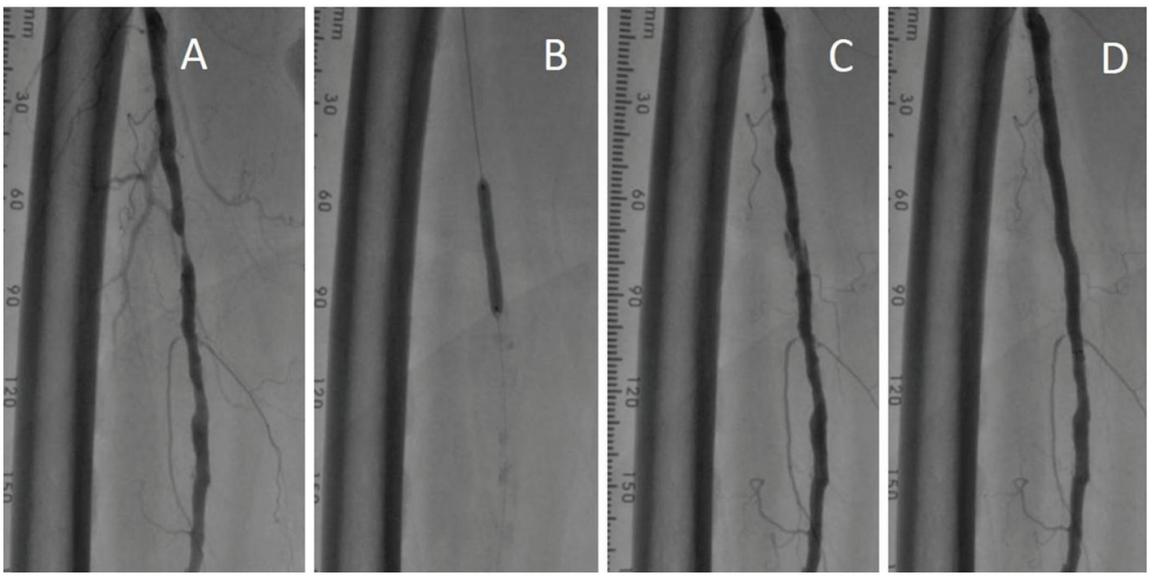

Figure 32. Bail out stenting post balloon angioplasty procedure of (A) 3 cm stenotic lesion; (B) balloon angioplasty performed; (C) dissection; (D) nitinol stenting for bail out purpose successful in sealing dissection.

http://dx.doi.org/10.5772/67181

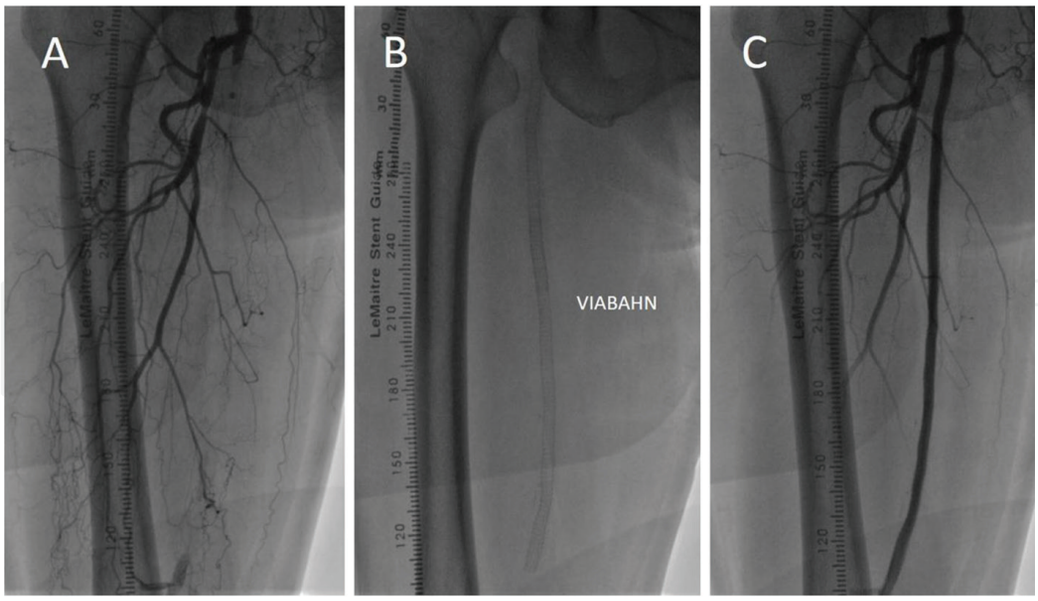

Figure 33. (A) Covered stent implantation for long right SFA CTO lesion; (B) CTO length is over 20 cm lesion; (C) VIABAHN (Gore, Flagstaff, AZ, USA) 6 × 250 mm stenting after balloon angioplasty.

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,

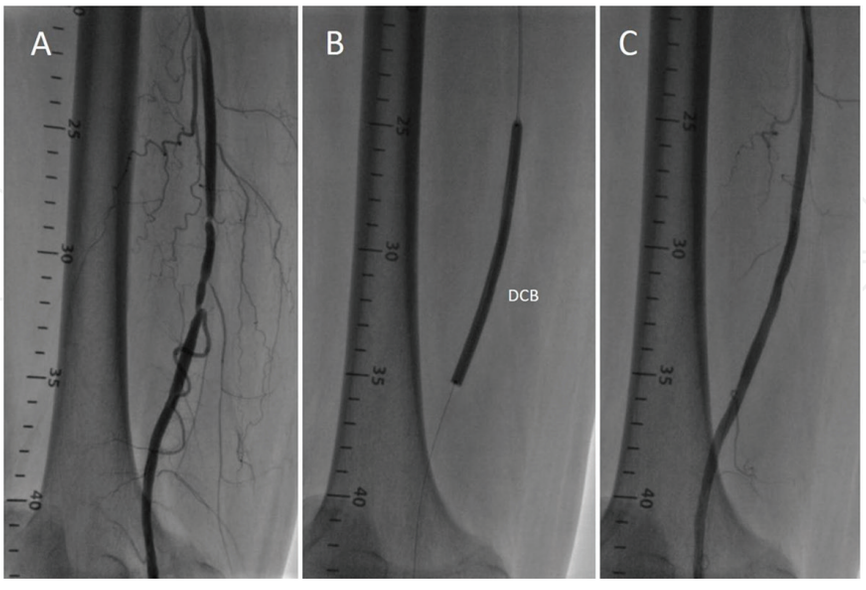

Figure 34. DCB angioplasty procedure. (A) Short focal lesion in mid-SFA; (B) after conventional balloon angioplasty and drug coated balloon angioplasty with LUTONIXX DCB (Bard Peripheral Vascular; Tempe, AZ) 5 × 100 mm balloon; (C) postballoon angioplasty showed residual stenosis of less than 50% at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

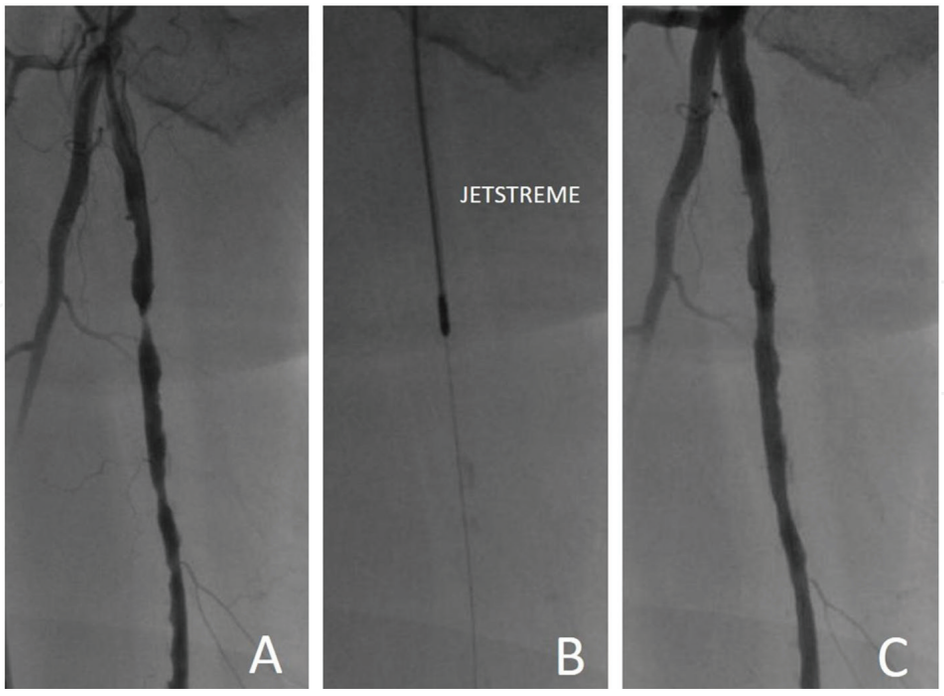

Figure 35. Atherectomy device. (A) Short focal lesion in mid-SFA; (B) atherectomy with JETstreme (Boston Scientific, Cambridge, MA, USA) atherectomy device; (C) postprocedure.

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.

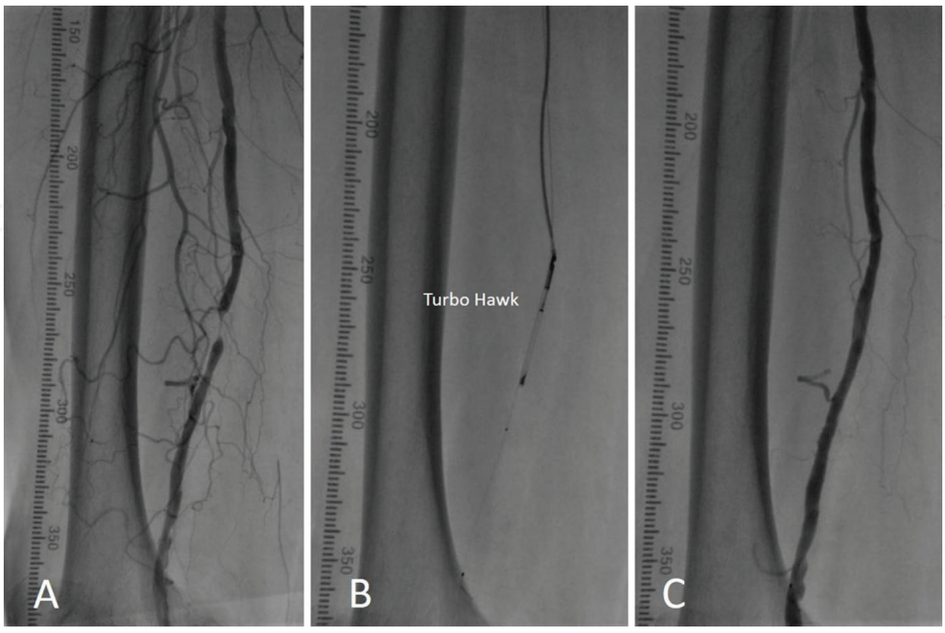

Figure 36. Atherectomy device. (A) Short focal lesion in mid-SFA; (B) atherectomy with TurboHawk (Medtronic Inc, Santa Rosa, CA) atherectomy device; (C) postprocedure.

http://dx.doi.org/10.5772/67181

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**).

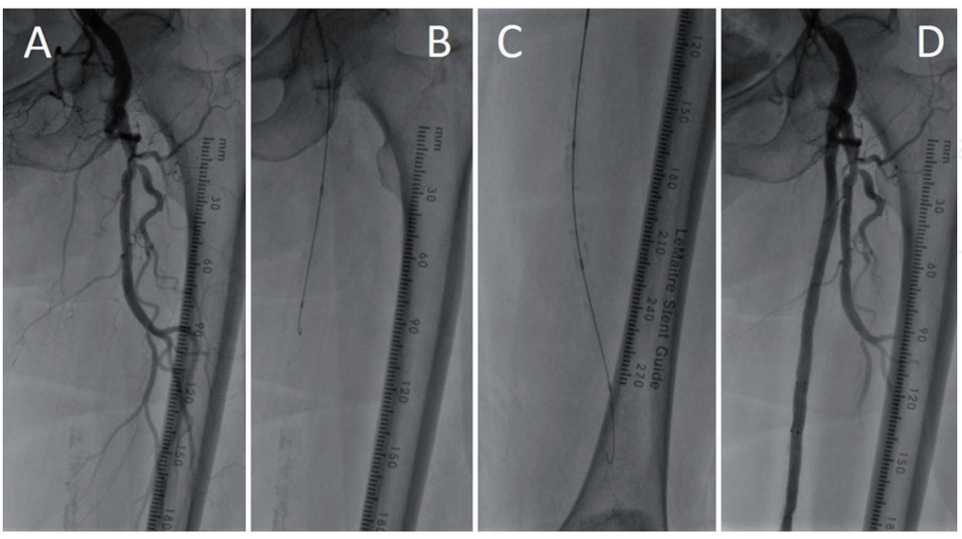

Figure 37. Antegrade approach with 0.018 inch guidewire. (A) SFA long total occlusion from proximal SFA to distal portion; (B) 0.018 inch J-type guidewire with support catheter into CTO lumen; (C) guidewire passage to distal true lumen; (D) final angiography after nitinol stent implantation.

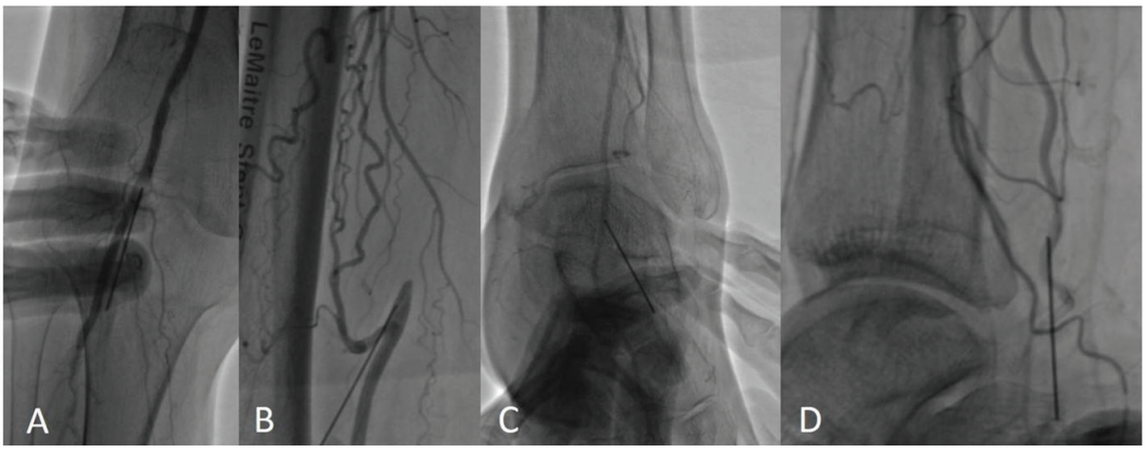

Figure 38. Retrograde access with distal puncture to recanalize SFA total occlusion lesion. (A) Popliteal artery puncture; (B) distal SFA puncture; (C) distal anterior tibial artery puncture; and (D) distal posterior tibial artery.

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**),

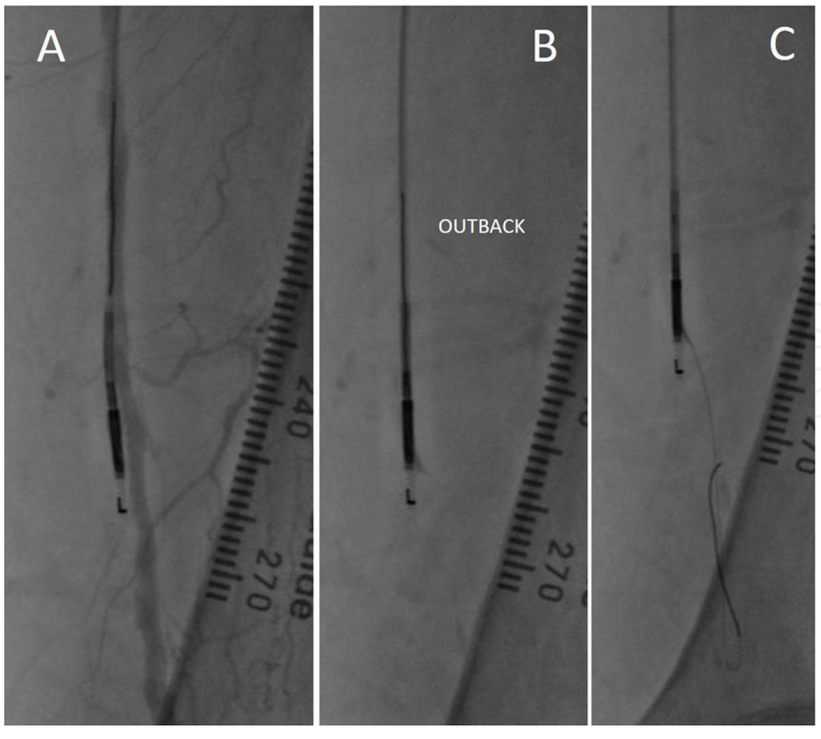

Figure 39. Outback reentry device (Cordis Endovascular, Bridgewater, NJ, USA). (A) Outback reentry device was placed in subintimal space of SFA mid portion. (B) Deploying the cannula from subintimal space to true lumen. (C) Wire crossing.

http://dx.doi.org/10.5772/67181

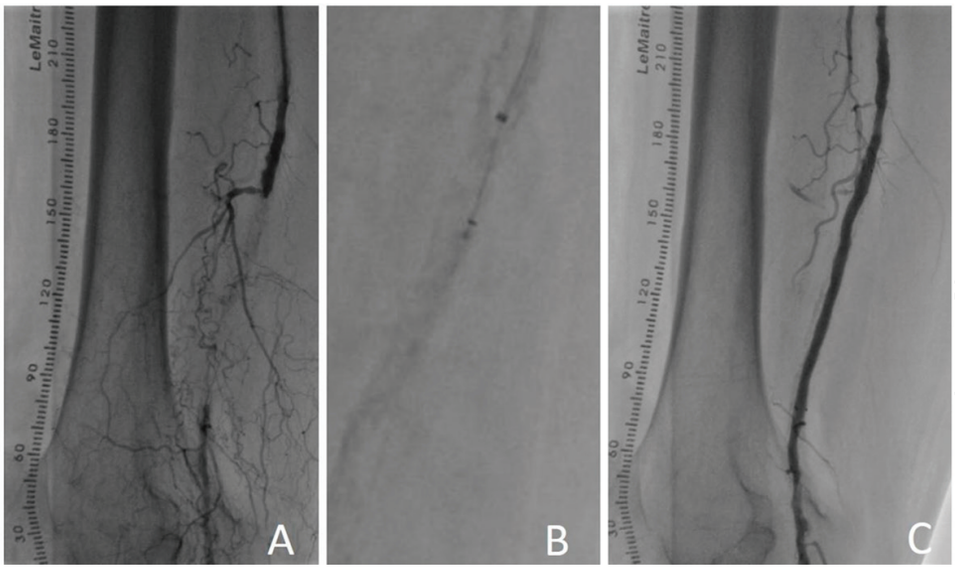

Figure 40. CTO crossing device. (A) Heavy calcified occlusive disease at distal SFA portion; (B) Crosser CTO crossing device (Bard Peripheral Vascular; Tempe, AZ) advanced without guidewire; (C) after crossing device, nitinol stent implantation.

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

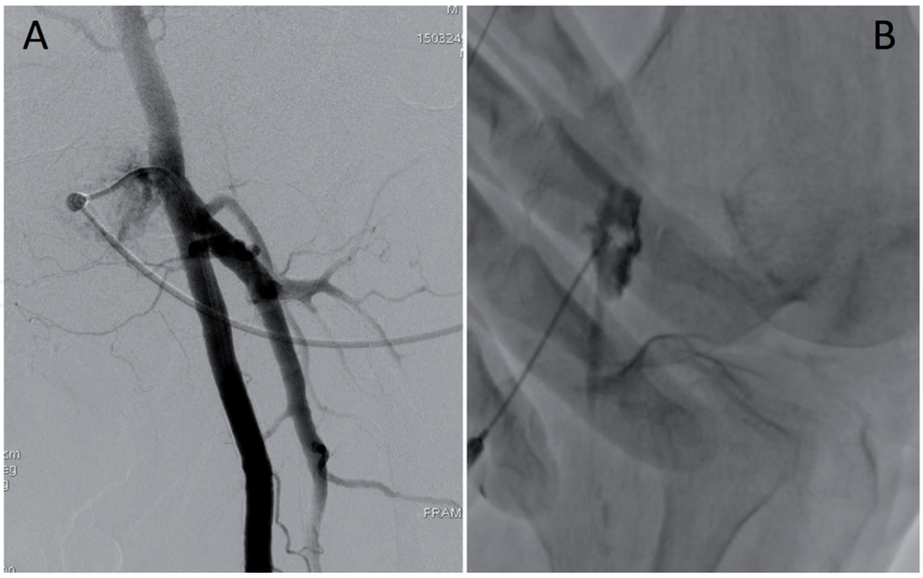

Figure 41. Puncture site vessel perforation. (A) Antegrade approach via CFA puncture; (B) retrograde approach via popliteal artery.

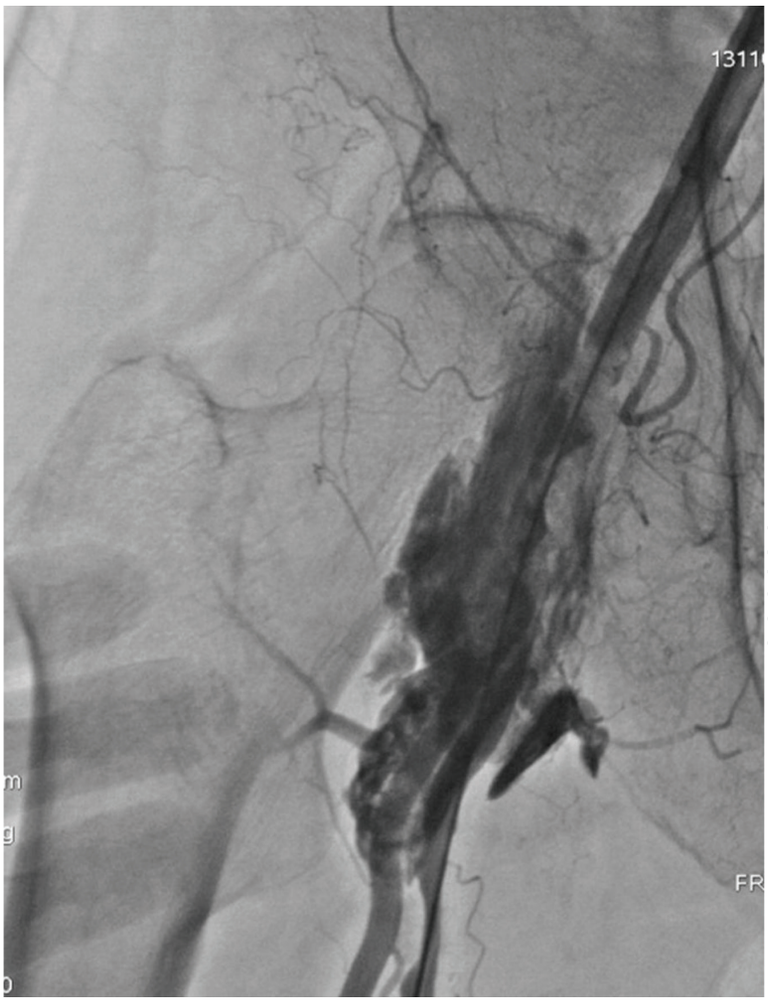

Figure 42. Procedure-related vessel perforation: vessel perforation occurred by cross-over sheath from contra lateral CFA puncture access.

http://dx.doi.org/10.5772/67181

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**).

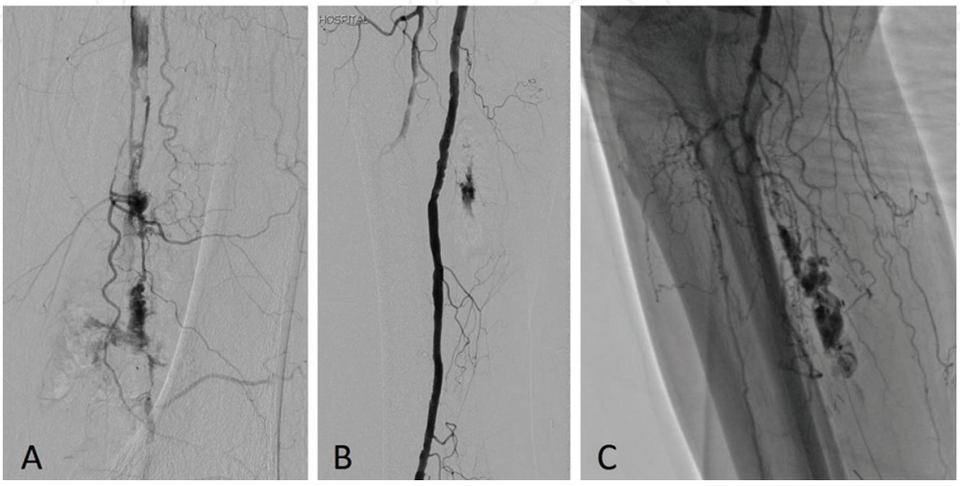

Figure 43. Vessel perforation: (A) caused by hard guidewire advanced into total occlusion portion; (B) aberration to small SFA side branches; (C) too deeply advanced guidewire to below the knee arteries.

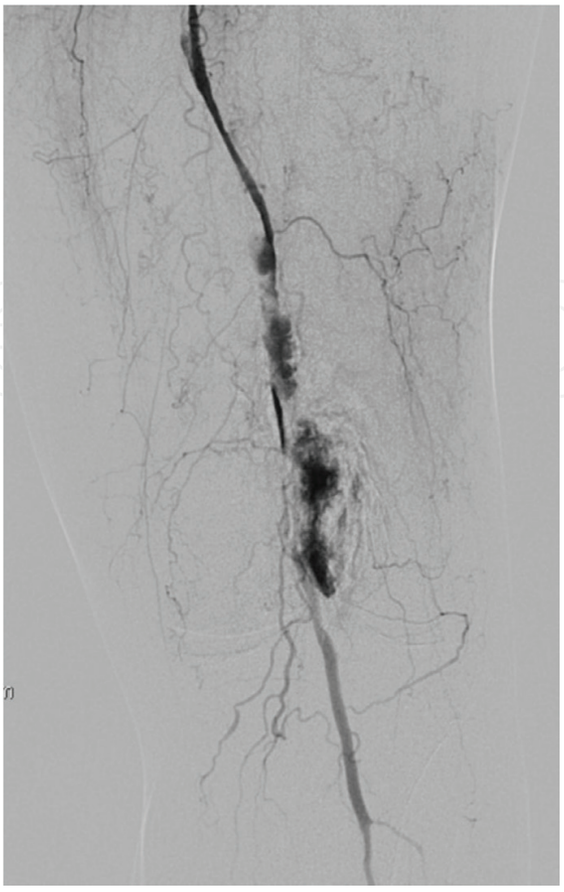

Figure 44. CTO crossing device related vessel perforation: vessel perforation by Crosser device at SFA mid-portion occlusive disease.

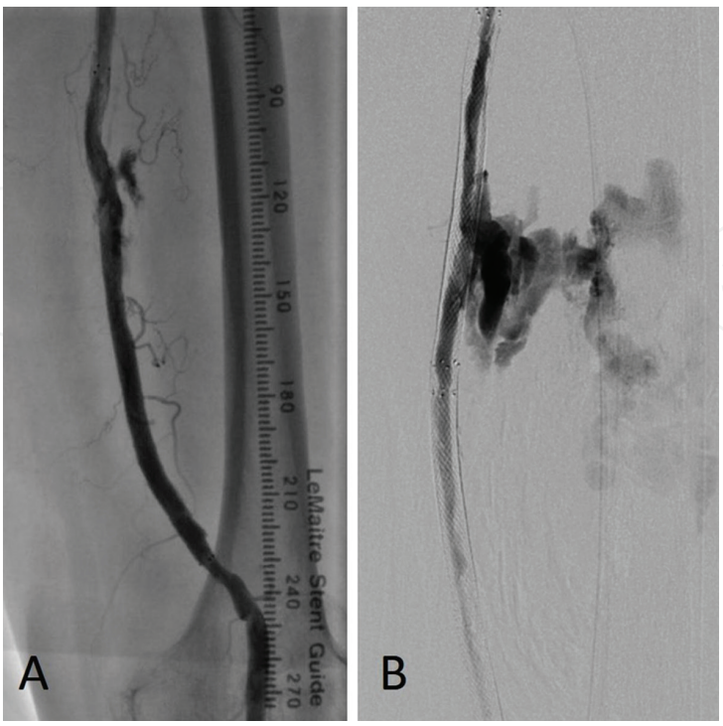

Figure 45. Balloon dilatation related vessel perforation (A) and (B).

http://dx.doi.org/10.5772/67181

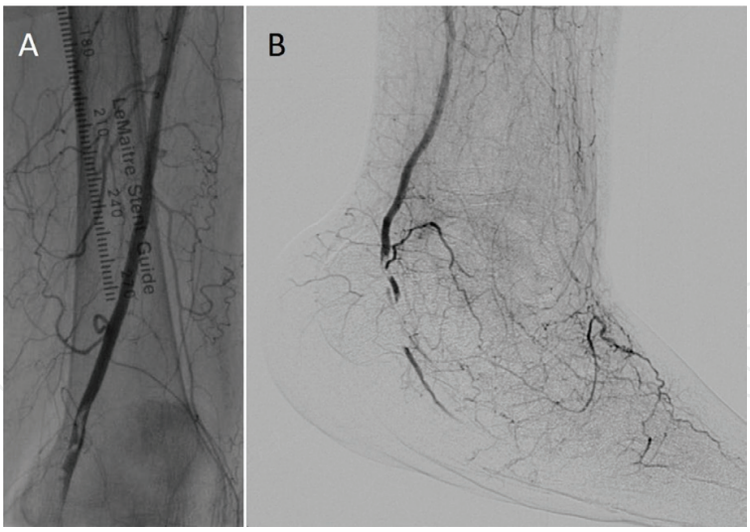

Figure 46. Distal emboli after femoropopliteal intervention. (A) Total occlusion of popliteal artery caused by distal emboli after SFA stent implantation; (B) distal emboli into PTA distal portion.

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.

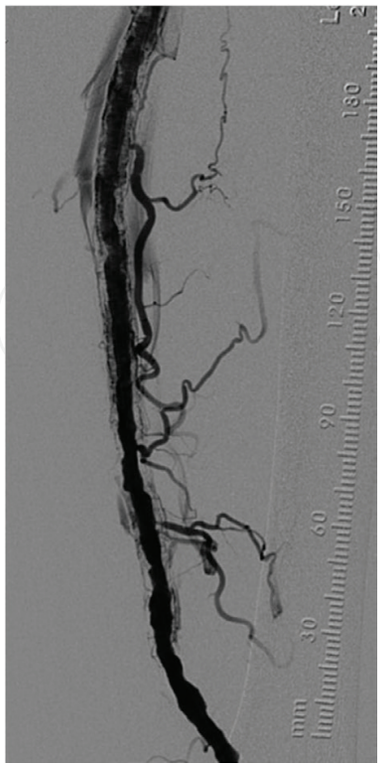

Figure 47. AV fistula: occurred after balloon angioplasty.

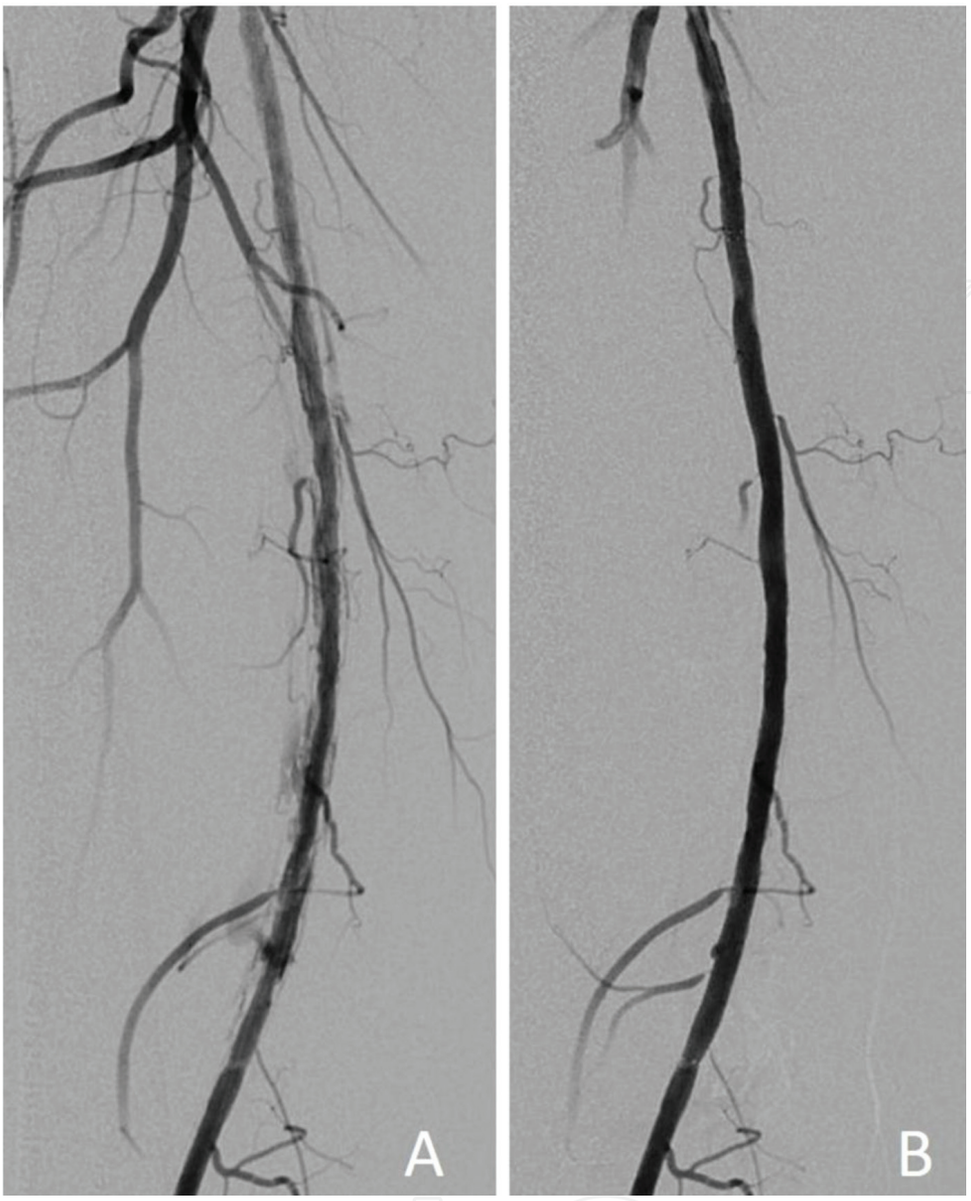

Figure 48. Stent implantation can repair the AV fistula in most cases. (A) Balloon angioplasty performed and resulted in AV fistula; (B) nitinol stenting for bail out purpose succeeded in sealing AV fistula.

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**).

http://dx.doi.org/10.5772/67181

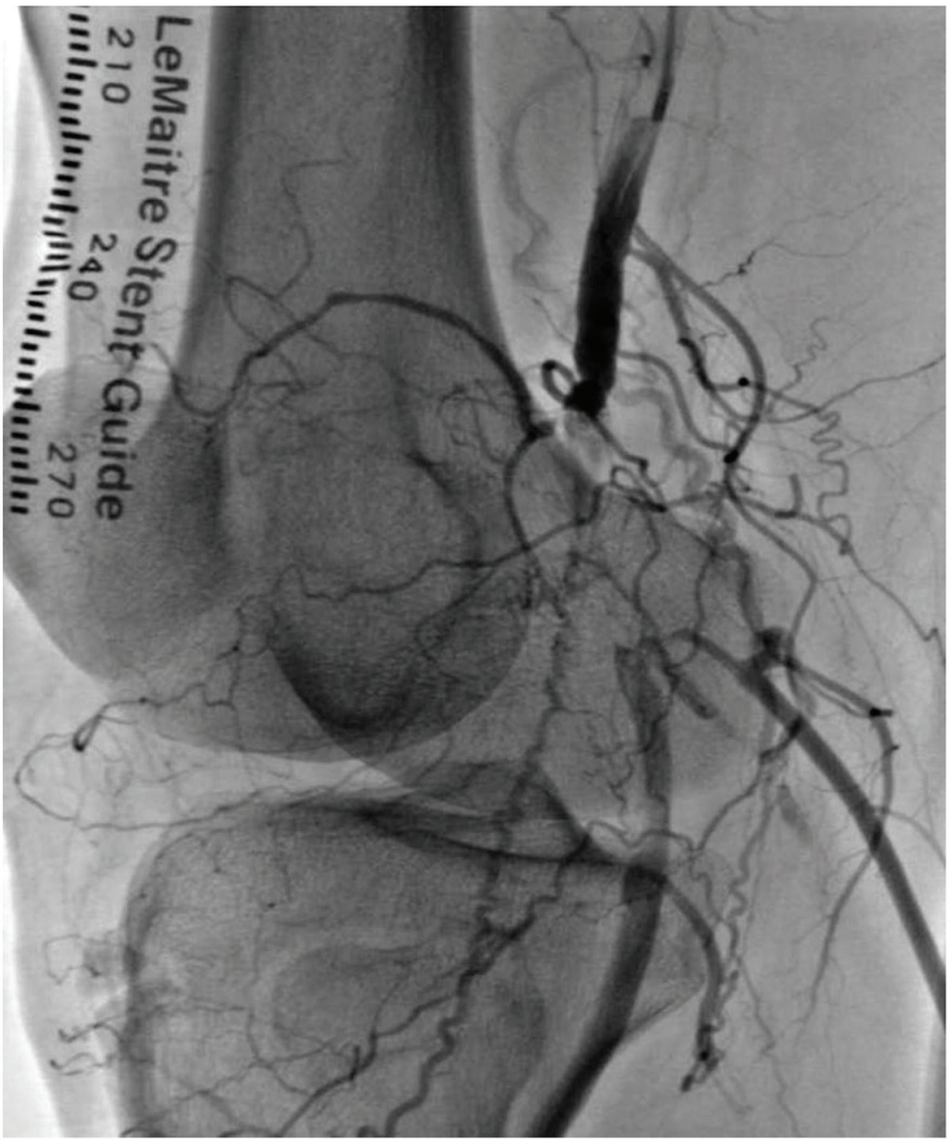

Figure 49. Popliteal artery disease; clear image by contra-lateral angled view.

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 non-stenting 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].

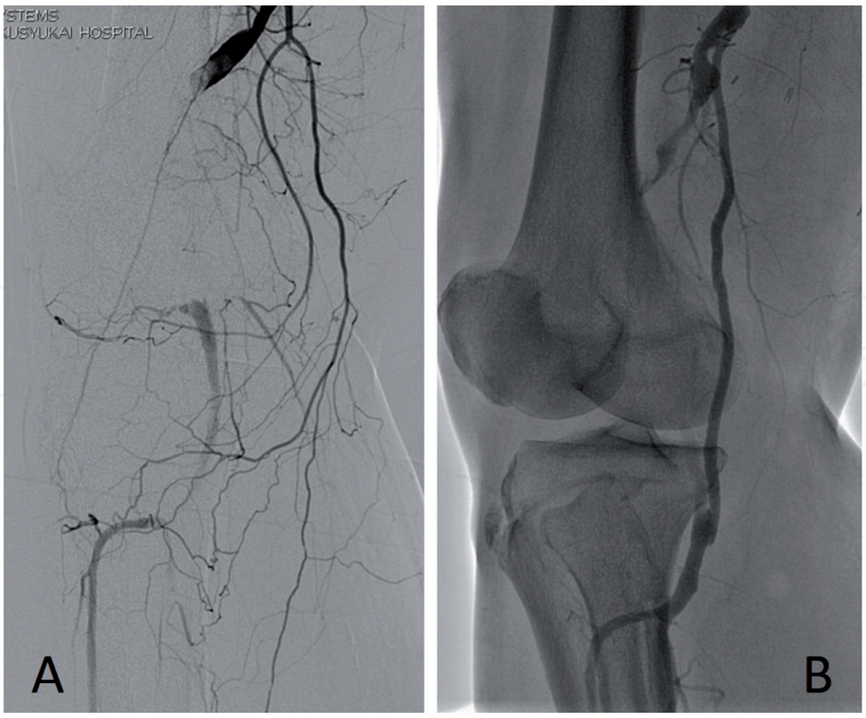

Figure 50. Popliteal artery aneurysm case (A) preprocedure; (B) after surgical therapy with SVG bypass.

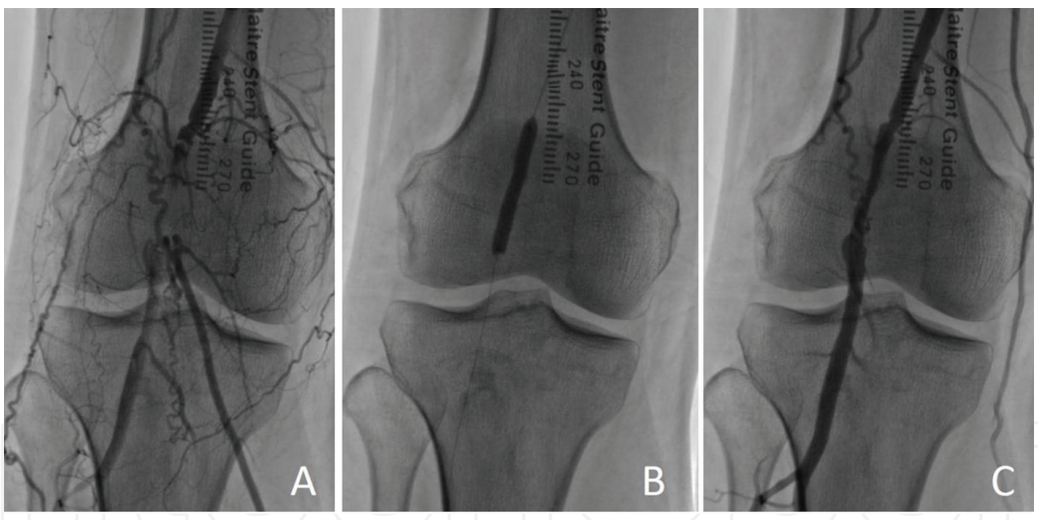

Figure 51. In short popliteal lesion. (A) Short focal lesion in mid-popliteal artery; (B) balloon angioplasty performed with 5 × 40 mm balloon; (C) postballoon angioplasty showed residual stenosis of less than 50% at ballooning site.

Author details

Masahiko Fujihara

Address all correspondence to: masahiko-fujihara@themis.ocn.ne.jp

Kishiwada Tokushukai Hospital, Osaka, Japan

http://dx.doi.org/10.5772/67181

References

[1] Fontaine R, Kim M, Kieny R; Kim, K. Surgical treatment of peripheral circulation disorders. Helvetica Chirurgica Acta 1954; 21 (5/6):499–533.

[2] Rutherford RB, Becker GJ. Standards for evaluating and reporting the results of surgical and percutaneous therapy for the peripheral arterial disease. J Vasc Interv Radiol. 1991; 2:169–74.

[3] Goodney PP, Beck AW, Nagle J, Welch HG, Zwolak RM. National trends in lower extremity bypass surgery, endovascular interventions, and major amputations. J Vasc Surg. 2009; 50(1):54–60.

[4] Scheinert D, Scheinert S, Sax J, Piorkowski C, Bräunlich S, Ulrich M, Biamino G, Schmidt A. Prevalence and clinical impact of stent fractures after femoropopliteal stenting. J Am Coll Cardiol. 2005; 45(2):312–5.

[5] Ho-Young Ahn, et al. Assessment of the Optimal Site of Femoral Artery Puncture and Angiographic Anatomical Study of the Common Femoral Artery. J Korean Neurosurg Soc 2014; 56(2):91–7.

[6] Scheinert D, Werner M, Scheinert S, Paetzold A, Banning-Eichenseer U, Piorkowski M, Ulrich M, Bausback Y, Bräunlich S, Schmidt A. Treatment of complex atherosclerotic popliteal artery disease with a new self-expanding interwoven nitinol stent: 12-month results of the Leipzig SUPERA popliteal artery stent registry. JACC Cardiovasc Interv. 2013; 6(1):65–71.

[7] Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FG, Bell K, Caporusso J, Durand-Zaleski I, Komori K, Lammer J, Liapis C, Novo S, Razavi M, Robbs J, Schaper N, Shigematsu H, Sapoval M, White C, White J, Clement D, Creager M, Jaff M, Mohler E 3rd, Rutherford RB, Sheehan P, Sillesen H, Rosenfield K, TASC II Working Group. Inter-society consensus for the management of peripheral arterial disease (TASC II). Eur J Vasc Endovasc Surg 2007; 33(suppl 1):S1–75.

[8] London GM, Guerin AP, Marchais SJ, Metivier F, Pannier B, Adda H. Arterial media calcification in end-stage renal disease: impact on all-cause and cardiovascular mortality. Nephrol Dial Transplant 2003; 18:1731–40.

[9] Rocha-Singh KJ, Zeller T, Jaff MR. Peripheral arterial calcification: prevalence, mechanism, detection, and clinical implications. Catheter Cardiovasc Interv. 2014;83(6):E212–20.

[10] Fanelli F, Cannavale A, Gazzetti M, Lucatelli P, Wlderk A, Cirelli C, d'Adamo A, Salvatori FM. Calcium burden assessment and impact on drug-eluting balloons in peripheral arterial disease. Cardiovasc Intervent Radiol. 2014; 37(4):898–907.

[11] Jaff M, Dake M, Pompa J, Ansel G, Yoder T. Standardized evaluation and reporting of stent fractures in clinical trials of noncoronary devices. Catheter Cardiovasc Interv. 2007; 70(3):460–2.

[12] Tosaka A, Soga Y, Iida O, Ishihara T, Hirano K, Suzuki K, Yokoi H, Nanto S, Nobuyoshi M. Classification and clinical impact of restenosis after femoropopliteal stenting. J Am Coll Cardiol. 2012; 59(1):16–23.

[13] Zdanowski Z, Albrechtsson U, Lundin A, Jonung T, Ribbe E, Thörne J, Norgren L. Percutaneous transluminal angioplasty with or without stenting for femoropopliteal occlusions? A randomized controlled study. Int Angiol. 1999;18(4):251–5.

[14] Laird JR, Katzen BT, Scheinert D, Lammer J, Carpenter J, Buchbinder M, Dave R, Ansel G, Lansky A, Cristea E, Collins TJ, Goldstein J, Jaff MR, RESILIENT Investigators. Nitinol stent implantation versus balloon angioplasty for lesions in the superficial femoral artery and proximal popliteal artery: twelve-month results from the RESILIENT randomized trial. Circ Cardiovasc Interv. 2010; 3(3):267–76.

[15] Dake MD, Ansel GM, Jaff MR, Ohki T, Saxon RR, Smouse HB, Zeller T, Roubin GS, Burket MW, Khatib Y, Snyder SA, Ragheb AO, White JK, Machan LS, Zilver PTX Investigators. Paclitaxel-eluting stents show superiority to balloon angioplasty and bare metal stents in femoropopliteal disease: twelve month Zilver PTX randomized study results. Circ Cardiovasc Interv 2011; 4:495–504.

[16] Iida O, Takahara M, Soga Y, Nakano M, Yamauchi Y, Zen K, Kawasaki D, Nanto S, Yokoi H, Uematsu M, ZEPHYR Investigators. 1-Year Results of the ZEPHYR Registry (Zilver PTX for the Femoral Artery and Proximal Popliteal Artery): predictors of restenosis. JACC Cardiovasc Interv. 2015; 8(8):1105–12.

[17] Lammer J, Zeller T, Hausegger KA, Schaefer PJ, Gschwendtner M, Mueller-Huelsbeck S, Rand T, Funovics M, Wolf F, Rastan A, Gschwandtner M, Puchner S, Ristl R, Schoder M. Heparin-bonded covered stents versus bare-metal stents for complex femoropopliteal artery lesions: the randomized VIASTAR trial (Viabahn endoprosthesis with PROPATEN bioactive surface [VIA] versus bare nitinol stent in the treatment of long lesions in superficial femoral artery occlusive disease). J Am Coll Cardiol. 2013; 62(15):1320–7.

[18] Tepe G, Laird J, Schneider P, Brodmann M, Krishnan P, Micari A, Metzger C, Scheinert D, Zeller T, Cohen DJ, Snead DB, Alexander B, Landini M, Jaff MR, IN.PACT SFA Trial Investigators. Drug-coated balloon versus standard percutaneous transluminal angioplasty for the treatment of superficial femoral and popliteal peripheral artery disease: 12-month results from the IN.PACT SFA randomized trial. Circulation. 2015; 131(5):495–502.

[19] Rosenfield K, Jaff MR, White CJ, Rocha-Singh K, Mena-Hurtado C, Metzger DC, Brodmann M, Pilger E, Zeller T, Krishnan P, Gammon R, Müller-Hülsbeck S, Nehler MR, Benenati JF, Scheinert D, LEVANT 2 Investigators. Trial of a paclitaxel-coated balloon for femoropopliteal artery disease. N Engl J Med. 2015; 373(2):145–53.

[20] Zeller T, Beschorner U, Pilger E, Bosiers M, Deloose K, Peeters P, Scheinert D, Schulte KL, Rastan A, Brodmann M. Paclitaxel-coated balloon in infrapopliteal arteries: 12-month results from the BIOLUX P-II randomized trial (BIOTRONIK'S-First in Man study of the Passeo-18 LUX drug releasing PTA Balloon Catheter vs. the uncoated Passeo-18 PTA balloon catheter in subjects requiring revascularization of infrapopliteal arteries). JACC Cardiovasc Interv. 2015; 8(12):1614–22.

[21] Zeller T, Krankenberg H, Steinkamp H, Rastan A, Sixt S, Schmidt A, Sievert H, Minar E, Bosiers M, Peeters P, Balzer JO, Gray W, Tübler T, Wissgott C, Schwarzwälder U, Scheinert D. One-year outcome of percutaneous rotational atherectomy with aspiration in infrainguinal peripheral arterial occlusive disease: the multicenter pathway PVD trial. J Endovasc Ther. 2009;16(6):653–62.

[22] Maehara A, Mintz GS, Shimshak TM, Ricotta JJ 2nd, Ramaiah V, Foster MT 3rd, Davis TP, Gray WA. Intravascular ultrasound evaluation of JETSTREAM atherectomy removal of superficial calcium in peripheral arteries. EuroIntervention. 2015; 11(1):96–103.

[23] Shammas NW, Lam R, Mustapha J, Ellichman J, Aggarwala G, Rivera E, Niazi K, Balar N. Comparison of orbital atherectomy plus balloon angioplasty vs. balloon angioplasty alone in patients with critical limb ischemia: results of the CALCIUM 360 randomized pilot trial. J Endovasc Ther. 2012; 19(4):480–8.

[24] Ramaiah V, Gammon R, Kiesz S, Cardenas J, Runyon JP, Fail P, Walker C, Allie DE, Chamberlin J, Solis M, Garcia L, Kandzari D; TALON Registry. Midterm outcomes from the TALON Registry: treating peripherals with SilverHawk: outcomes collection. J Endovasc Ther. 2006; 13:592–602.

[25] Roberts D, Niazi K, Miller W, Krishnan P, Gammon R, Schreiber T, Shammas NW, Clair D, DEFINITIVE Ca^{++} Investigators. Effective endovascular treatment of calcified femoropopliteal disease with directional atherectomy and distal embolic protection: final results of the DEFINITIVE Ca^{++} trial. Catheter Cardiovasc Interv. 2014; 84(2):236–44.

[26] Kitrou P, Parthipun A, Diamantopoulos A, Paraskevopoulos I, Karunanithy N, Katsanos K. Targeted true lumen re-entry with the outback catheter: accuracy, success, and complications in 100 peripheral chronic total occlusions and systematic review of the literature. J Endovasc Ther. 2015; 22(4):538–45.

[27] Scheinert D, Bräunlich S, Scheinert S, Ulrich M, Biamino G, Schmidt A. Initial clinical experience with an IVUS-guided transmembrane puncture device to facilitate recanalization of total femoral artery occlusions. EuroIntervention. 2005; 1(1):115–9.

[28] Schmidt A, Keirse K, Blessing E, Langhoff R, Diaz-Cartelle J, European Study Group. Offroad re-entry catheter system for subintimal recanalization of chronic total occlusions in femoropopliteal arteries: primary safety and effectiveness results of the re-route trial. J Cardiovasc Surg (Torino). 2014; 55(4):551–8.

[29] Laird J, Joye J, Sachdev N, Huang P, Caputo R, Mohiuddin I, Runyon J, Das T. Recanalization of infrainguinal chronic total occlusions with the crosser system: results of the PATRIOT trial. J Invasive Cardiol. 2014; 26(10):497–504.

[30] Bosiers M, Diaz-Cartelle J, Scheinert D, Peeters P, Dawkins KD. Revascularization of lower extremity chronic total occlusions with a novel intraluminal recanalization device: results of the ReOpen study. J Endovasc Ther. 2014; 21(1):61–70.

[31] Scheinert D, Grummt L, Piorkowski M, Sax J, Scheinert S, Ulrich M, Werner M, Bausback Y, Braunlich S, Schmidt A. A novel self-expanding interwoven nitinol stent for complex femoropopliteal lesions: 24-month results of the SUPERA SFA registry. J Endovasc Ther. 2011; 18(6):745–52.

IntechOpen