Chapter 17
Distal Embolization in the Treatment of Peripheral Arterial Disease

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Historical Background

In early interventional radiology literature, writers reported a rate of distal embolization rates between 3.8 and 37% related to angioplasty of arteries in the iliac and femoropopliteal system [1–3]. In 2005, we submitted the first publications on the use of distal embolic protection with atherectomy devices used in the femoropopliteal artery and recorded a high incidence (10/10) of embolic plaque ranging in size from 0.5 to 10 mm [4, 5]. We did not expect the ensuing controversy about the creation and the treatment options for distal emboli during peripheral arterial interventions of the lower extremities [5, 6]. For nearly 16 years, this debate has continued with several studies and reviews despite the highly charged economic forces involved. There are multiple variables in addressing the problem, the most important being how relevant are the distal emboli.

The Problem: Distal Embolization from Endovascular Procedures

All arterial interventions be it crossing an atherosclerotic lesion with a wire, ballooning, stenting, or debulking all can result in distal embolic material [7]. (Refer to Fig. 17.1a–c). This distal embolization includes material ejected off the atherosclerotic plaque as well as thromboemboli. Distal embolization occurs in all arterial beds including the coronary, carotid, and intracerebral, renal, aortic, and pelvic and lower extremities. The clinical significance and the ability to diagnose the

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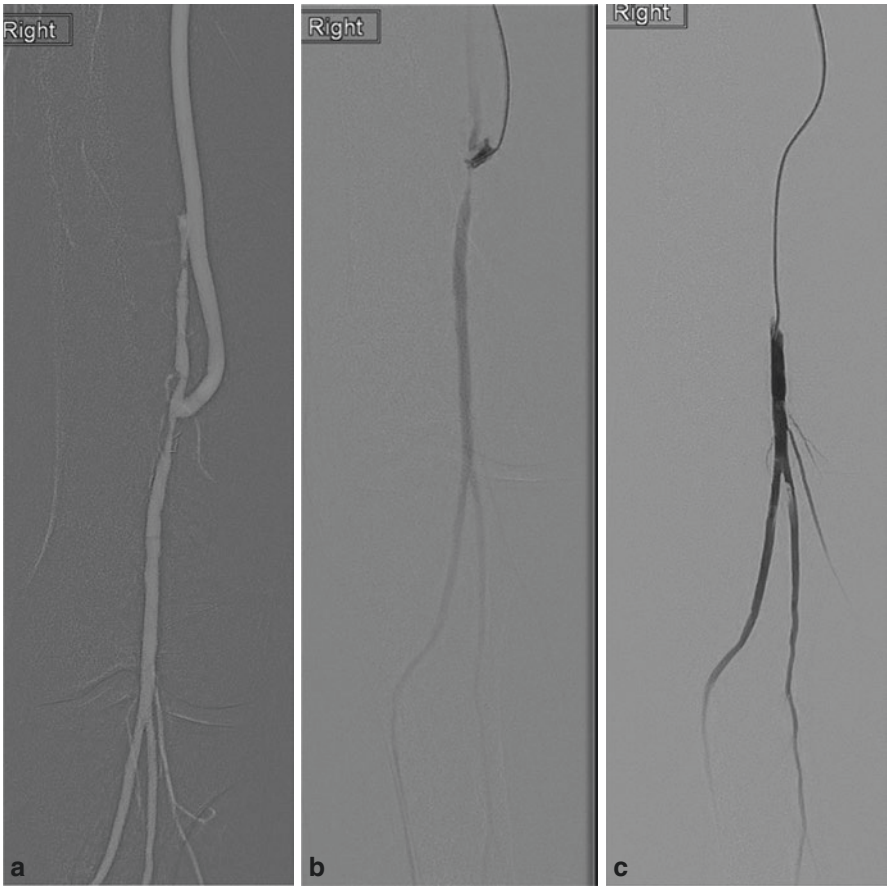

Fig. 17.1 (**a**) Initial carbon dioxide angiogram revealing moderate disease at the distal end of the fem-pop bypass graft. (**b**) Contrast injection with a 4-Fr Glide catheter near the distal anastomosis revealing greater stenosis along native popliteal artery and patent three vessels distally (not shown). (**c**) With the Glidecath across the stenosis, contrast injection reveals the new filling defect in the tibial peroneal artery (arrow)

occurrence of the distal emboli depend upon the region treated and the imaging available. For example, with carotid artery angioplasty and stent placement, the diagnosis of distal emboli depended much upon the use of diffusion-weighted MR images documenting the multiple ischemic insults. Neurological correlation would then be provided to determine the resultant clinical significance.

So how is distal embolization determined in PAD cases? This ranges from the following:

- Increased Doppler signals that occur distal to the treated area during a case.
- Whether there is material retained within the embolic protection device (EPD) basket if used. The material has been delineated to visible and macroscopic

material or as microscopic; the determining diameter of microscopic versus macroscopic material varies to under 2 mm in some studies and under diameter <100 μm in other studies [8, 9].
- Angiographic findings of distal embolization pre- and post-intervention which usually pertains to those large vessels of the trifurcation; there has been little written about the loss of the small arteries in the foot. Likewise, most studies did not employ an independent core lab. The quality of conventional digital subtraction angiography can vary resulting in different resolution and detection of small emboli [10].
- Whether further intervention is needed. Some studies have used "distal emboli" to denote the need for TLR or other interventions.

What are the Rates of Distal Embolization?

As the table below reveals, there is a wide range of reported distal emboli that occurs with PAD inventions depending upon a multitude of factors including:

1. Target location: Aortoiliac which has had larger vessel diameter with more chronic disease, femoropopliteal which has the largest series of data with extensive use of distal protection (DP), and below the knee (BTK) with generally less plaque volume and vessels too small for DP.
2. Acuteness of symptoms: Acute ischemia traditionally had higher fresh clot and clot burden with larger and more disastrous distal embolization downstream. Most studies deal with chronic ischemic disease with older plaque and established thromboemboli.
3. Whether EDP is used and how is the debris counted: Microemboli or macroemboli in the basket.
4. Who measures the severity of the distal emboli? (Table 17.1).

Incidence of Distal Emboli

The incidence of distal embolization during lower extremity arterial intervention is easily determined by visible or macroscopic debris particles, usually greater than 2 mm, retained in the EDP basket. In Krishnam's study of 508 patients, he had 62% of cases with macroscopic debris [14]. Yet in Shammas' study of 557 patients, there were only 2.4% [13]. This variation in incidence from two respective centers with similar procedures and patient types may reflect the random nature of the embolic debris created. Microscopic debris found in the filter baskets was highly prevalent in some studies such as Spiliopoulos and Lewis' studies showing 100% and 87%, respectively [9, 10]. Müller et al. evaluated the safety and effectiveness of EPD in reducing distal embolization during percutaneous lower extremity interventions

Table 17.1 Review of distal embolization incidence in major PAD studies

Author	Targ sites	Years	Pt no.	Lesion no.	Device	EDP filter?	% Distal emboli	% Clinical significance
Cassius [11]	Iliac/ SFA-POP	2017		10,875	All	Angio	1.7%	66%req'd treatment
Shirankde [12]	SFA-POP	2011	1029 18 18 55 570 740 736	2137	Jetstream CSI Laser PTA PTA/Stent SilverHawk	Angio	1.6% 22% 22% 3.6% 0.9% 0.7% 0.1%	
Shammas [13]	SFA-POP	2009	557	1183	All types	Yes	2.4%	2.4%
Krishnan [14]	SFA-POP	2017	508		All		62% macro	15% filter overflow
Balzer [15]	Iliac	2010	195	285	PTA/Stent	Yes	4.2%	
Definitive CA study [16]	SFA-POP	2014	133	168	SilverHawk	Yes	97.5%	3 cases
Milnerowicz [17]	Iliac/ SFA	2019	74		Rotarex	Angio.	8.1%	
PROTECT registry [18]	SFA-POP	2008	40	43 13	PTA/Stent SilverHawk	Yes Yes	28% 91%	>2 mm
DEEP Emboli [8]	SFA-POP	2009	20	44	Laser spectra	Yes	66%	Macrodebris in 12 cases
Spiliopoulos [9]	SFA-POP	2014	40		PTA/Stent	Yes	100%micro	No angio-occlusions
Lewis [10]	SFA-POP	2010	35	64	PTA/Stent	Yes	87% micro	3 DP baskets completely full
Hadidi [19]	SFA-POP	2012	30	36	All	Balloon	89%	Angiographic 5.5%

including angioplasty/stenting and directional atherectomy [20]. All patients undergoing directional atherectomy with the SilverHawk device demonstrated significant macroembolization, and 37.9% of patients undergoing angioplasty and/or stenting demonstrated significant macroembolization [20].

The incidence of distal embolization detected angiographically or clinically was reported between 1.6 and 8.1%; however, contemporary studies have demonstrated a much higher incidence of 3.8–67% [1, 10, 18, 21–24]. As for other modalities, Lam et al. used a transcranial Doppler to detect signals related to DE during superficial femoral artery (SFA) interventions and found 100% occurrence [25]. So why would the angiographic detection be so much lower for distal emboli detection compared to filter retention studies? It may be due to equipment used, limited views of the foot and distal circulation, and economic forces: Why look for trouble?

As the above chart shows, the incidence of distal emboli is difficult to determine from the various registries and single-center studies. In PTA/stent procedures involving the SFA-POP, distal emboli occurred from 0.7 to 100%. For atherectomy usage, the range for distal embolization was from 0.1 to 97.5%. Obviously, the rate of distal embolization is higher with atherectomy and other invasive means of treating atherosclerotic disease compared to angioplasty and stenting. In the PROTECT registry, basket captured distal embolization occurred in 28% of the PTA/stent cases versus 91% of the atherectomy (FoxHollow SilverHawk) [18]. In Shirankde's study, there was a 22% angiographic rate of embolization with the CSI and Jetstream versus 0.9% for PTA/stent [12].

A special risk is treating acute arterial occlusions: high rate of distal embolization often resulting in complete occlusions downstream. The rate of distal embolization during thrombolytic therapy in limb-threatening ischemia has been reported as 3.8–37% [22, 24].

The Clinical Relevance of Distal Emboli

All arterial interventions can cause distal embolic complications. For the PAD, positive embolic debris findings are reported either with distal protection devices with full or partially full baskets, with angiographic findings of distal vessels occluded, or by ultrasound. But for PAD cases, it is difficult to determine how significant the loss of a peroneal, anterior, or a posterior tibial artery following an embolic shower. These lost vessels raise questions: Does distal emboli result in discoloration? Does it result in the poor healing of a wound? Does it clear in time? Does the patient have worsening claudication, ABI, or other parameters? These are all questions with vague answers, which are easily overlooked, and which are why it is hard to justify and to subsequently provide adequate reimbursement for the use of distal protection and why after 16 years we ask the same question: Do we really need distal protection for PAD cases?

Muller et al. reported performing 30 lower extremity revascularizations for the femoropopliteal artery and used an embolic protection device (DP) in all of the cases. All of the filters were found to have debris under microscope examination, and 90% were deemed "clinically significant" because they were visible by the naked eye [20]. In our limited series in 2005, we were surprised by the large size of the fragments of plaque for the diameters of the tibial vessels are often under 2–3 mm [5]. As Shammas et al. said, large debris >2 mm do occur at a high frequency, ranging from 20 to >90% depending on lesions and devices used [18]. Thus, it would not take much to totally occlude the infrapopliteal vessels.

The pathological features of the captured distal emboli have been interesting to study. We found that when angioplasty and stent placement were performed in high-risk patients, the debris tended to be small fragments or microembolization. Treatment with atherectomy devices led to larger or more visible fragments. According to published data, embolic material during peripheral endovascular

procedures consists of plaque and vessel lumen components, such as fibrin, calcified deposits, cholesterol clefts, and inflammatory and endothelial cells [26, 27]. In Karnabatidis et al.'s study, they found in their 50 cases the total area of occlusion was 2.76 ± 6.5 mm^2 with 12% of the particles greater than 3 mm [27]. Collected particles included platelets, fibrin conglomerates, trapped RBC, inflammatory cells, and extracellular matrix which compose atherosclerotic plaque and thrombosed elements [27]. Hence, with such material occluding infrapopliteal arteries, it will be hard to succeed with standard thrombolytic therapy with tPA that only works on a thrombus 2–3 weeks or less.

The peripheral microcirculation in ischemic patients has been compromised, and any further downstream embolization may result in worsening or persistent ischemia [12]. This is supported by the findings of Karnabatidis in which histologic analysis demonstrated a greater amount of collected particles in the filter baskets positively correlating with increased lesion length and reference vessel diameter, acute thrombosis, and total vessel occlusion [4, 10]. All of these factors could lead to disastrous consequences for our typical CLI patient.

A main issue in the endovascular community has been the lack of consensus for the definition of "clinically significant" distal emboli and when, how, and in whom this should be treated. Operators at present might differ in their approach in defining and treating distal emboli [13]. However, it appears that only a small fraction (2.4% in Shammas et al.'s study) of these debris requires further treatment [13]. Limb-threatening distal embolization occurred in approximately 2% of patients during routine intervention in other major studies [28, 29] Given the previous discussion on the unreported incidence of distal emboli and the size and pathological features of these emboli, it is hard to understand why there are not more limb-threatening events. The consequence of distal embolization resulting in occlusion of the vascular bed in patients with poor arterial inflow, poor collateralization, or poor runoff may be devastating.

Possible reasons include the durability of the distal circulation of "healthy" PAD patients such as claudicants, patients with three vessel runoff, and nondiabetics. Namely, if the patient has limited reserve in the distal circulation, then he or she can take less embolic insult. Also, despite increased debris from our procedures, many patients seem to clear these obstructions over a week or 2 weeks. Possibly, the patients develop collaterals, the spasm reduces, or the inflammation improves allowing the flow to return.

This complication may require the use of additional intervention including thrombectomy or thrombolysis, resulting in longer procedure time, greater volumes of contrast administered, and increased radiation exposure [10, 18]. The acute outcomes are less symptom relief, worsening of symptoms, and increased emergent surgical bypass. Long-term outcomes are also adversely affected with decreased symptom relief at 2 years and increased above-the-knee, below-the-knee, and below-the-ankle amputations [13, 25, 30].

Freeman et al. defines high-risk patients as those with limited distal runoff, vulnerable or unstable plaque, history of thromboembolic disease, or aneurysmal disease [31]. Features separating patients with stable claudication from those with

ongoing ischemia include the length of the stenosis and the length of the lesion, presence of distal runoff, and chronicity of disease [30]. Embolism has also been reported in patients with concentric stenoses [26, 27]. However, Shammas et al. state that predicting which vessels will embolize based on lesion characteristics is not always possible [13].

Shirkande et al. showed some lesion types that are more prone to embolization [12]. TASC C and D lesions had higher rates of embolization than TASC A and B lesions (2.2% vs 0.9%, $p = 0.018$) [12]. In a study by Lam et al., there was no difference in sonographically detected embolic signals between TAIC classifications [25]. Shirkande et al. showed total occlusions (2.4%) or ISR (3.2%) had a higher rate of DE than native stenotic lesions (0.9%; $p<0.01$) [12]. The higher rate of distal emboli in ISR could reflect the nature of the material within the stent, which may be softer and more friable than standard atherosclerotic plaque [25]. Shammas provided a good review of patients with a high risk of distal emboli are those with a prior history of amputation, TASC D lesions, and the presence of angiographic thrombus. In his study, thrombotic lesions had 5.9 times greater odds of embolization than nonthrombotic ones [13]. Also, patients presenting with acute thrombotic occlusions (within 24 h of symptom onset) showed a trend toward more distal emboli than those presenting subacutely/chronically [13].

Indications for the Use of Distal Embolic Filters

Common sense indicates that EPD use may be of value in a lesion with vulnerable or unstable plaque, acute thrombosis, chronic total occlusions, or aneurysmal disease [10]. It is safe to generalize that all CTO, ISR, and thrombotic lesions treated with atherectomy merit the use of embolic protection devices because of high risk of embolization. Current recommendations support the use of these filters in cases of significant calcification, although operator discretion plays a pivotal role in their deployment. The decision for the use of EPD in calcific lesions and atherosclerotic lesions is dependent on lesion length. Lesions >140 mm when atherosclerotic and >40 mm when calcific warrant the use of an embolic protection device to prevent complications.

Although embolization is a potential complication of any atherectomy device, it remains unclear whether certain devices or techniques predispose patients to distal embolization. Further, certain devices may be preferred in a given lesion morphology, and additional research may be necessary to address this question. Third, the incremental cost of EPD is significant (often in the range of $1000), and additional cost-benefit analyses could help clarify the optimal clinical scenarios for both atherectomy and embolic protection device uses. Although the proposed algorithm is therefore a useful guide, it is not clear that this algorithm would apply to every clinical scenario in which atherectomy is being used [32].

Krishnan et al. prepared a good algorithm for the use of EPD with atherectomy devices depending upon lesion characteristics [30]. Once the diagnostic angiogram

is performed, the operator assesses the following morphology and anatomic characteristics: thrombotic, calcific, restenotic, CTO, lesion length, and runoff [30].

EPD filters are recommended for the following:

- Calcium with length greater than 4 cm.
- In-stent restenosis, thrombus, and complete total occlusions.
- Atherosclerotic lesions in lesions longer than 14 cm and with distal runoffs of less than 2 vessels.

Current Status of Distal Protection

Currently, there is no consensus for the use of EPD during atherectomy, and the only Food and Drug Administration-approved device with an indication in the femoropopliteal segment is the SpiderFX for use in conjunction with directional atherectomy in heavily calcified lesions [16].

There are currently two available EPD filter protections in treating PAD. The SpiderFX Embolic Protection Device (Medtronic, Minneapolis, Minnesota) is indicated for use as a guidewire and embolic protection system to contain and remove embolic material in conjunction with the TurboHawk (Medtronic, Minneapolis, MN), either during standalone procedures or together with PTA and/or stenting, in the treatment of severely calcified lesions in arteries of the lower extremities. The SpiderFX device (Fig. 17.2) has a braided nitinol filter that conforms to the vessel wall and maintains apposition. The pore size is 167–209 μm. The capture wire rotates and moves longitudinally independent of the filter and is available in diameter sizes 3–7 mm.

Advantages for the SpiderFX device include:

– The ease of use with the ability to cross a lesion with a 0.014–0.018″ wire of choice followed by the delivery catheter or the ability to deliver it through a 4-Fr compatible catheter.
– The EPD filter allows flow through the vessel while deployed reducing the chance of fibrin buildup to occlude flow.
– Recovery catheter (opposite end of the delivery catheter) is very easy to use and captures efficiently.

Disadvantages for the SpiderFX device and for many EPD include:

Fig. 17.2 SpiderFX embolic protection device

– The pore size is rather large and lets smaller debris (200 μm) flow through.
– The proximal busing holding the strut for the basket is susceptible to become stuck with coaxial balloon catheters, stents, and other devices.
– The filter basket with the nitinol basket design can easily become ensnared with self-expandable stents.
– When the filter becomes full of debris, it is very hard to get a catheter down to basket to aspirate the debris out, and if a solid plaque is captured, it may not be withdrawn into the overlying sheath.
– Then the length of the filter and diameter is limited, so it will not capture the large iliac vessels well.
– It is useful primarily with the FoxHollow (Medtronic line) atherectomy line.
– Once the system is retracted, a second wire must be passed to the treated lesion.

A second EPD filter is the Mednova later Abbott Emboshield Nav [6] (Plymouth, MN) (Fig. 17.3) which is indicated for use as a guidewire and embolic protection system to contain and remove embolic material (thrombus/debris) while performing angioplasty and stenting procedures while performing atherectomy, during stand-alone procedures or together with PTA and/or stenting, in lower extremity arteries. The diameter of the artery at the site of the filtration element placement should be between 2.5 and 7.0 mm. It has a centered wire design to prevent bias against the vessel wall; circumferential nitinol frame maintains optimal wall apposition.

Advantages for the Abbott Emboshield Nav [6] system include:

– The unique wire technology allows the wire to rotate and advance freely, independent of the filter. This allows the device to be used with most of the atherectomy devices.
– The filter is designed to stay in place during device delivery.
– Continued wire access, after the filter is fully retracted, allows for easy delivery of additional therapy.

Disadvantages of the Abbott Emboshield Nav [6] system include:

– The filter can be pulled back but cannot be easily pushed forward without recapturing it.

Fig. 17.3 Abbott Emboshield Nav [6]

– The two struts with plastic covering can deflect plaque to sides and can fail to capture them.
– The filter has 140 μm pore size which is very efficient in capturing small debris but can lead to a full basket.

Technique of Distal Protection

The standard technique for use of distal protection is simple. After an angiogram is performed involving the pelvis and/or lower extremity from either retrograde or antegrade approach, the site of EPD filter deployment is selected. Selection is based upon a region well enough distal to the targeted treatment zone. Other considerations include trying to preserve as many main vessels and collateral vessels as possible (Fig. 17.4a–g). Deployment is best if the vessel does not have extensive plaque and is fairly straight.

If the lesion is not terribly tight and we cross with our 4-Fr catheter or complete target occlusion (CTO) catheter which utilizes a 0.035″ wire, then we will simply remove the 0.035″ wire and advance our filter and deploy it. If we use a 0.014″ wire and are worried about the plaque/thrombus, we will use the special rapid exchange catheter to allow deployment of the filter. Once the filter is in place, we will move swiftly to perform the needed intervention. For atherectomy, we will be more vigilant for a full basket. After the procedure is done, we advance the filter recovery catheter quite slowly in the smaller vessel, capture the ringlike structure, and then remove the catheter and filter very carefully especially with a sent in the path of the filter and recovery system.

There are several inherent limitations with the use of EPD through selected lesions including chronic total occlusions which may pose a challenge. The EPD devices are routinely mounted on medium support wires, which are not intended to function as primary crossing wires.

EPD typically requires a rather long length of vessel for safe implantation, which may pose a challenge since many patients with CLI have extensive and diffuse atherosclerotic disease. Incomplete apposition of the device to the vessel wall may allow side escape of engendered debris. Furthermore, arterial spasm, arterial injury (including dissection), and de novo thrombus may occur as a result of the EPD device itself [27].

Revascularization procedures often result in the production of large amounts of macroscopic debris. The size of the filter basket may be inadequate for collection of debris. Extreme care must be taken since filter wire retrieval may also result in dislodgment of debris due to squeezing of the basket. Larger clots may also remain outside the struts of the EPD filter and are too large to be removed by filter closing and standard re-sheathing techniques. EPD provides no protection to the collateral circulation. Therefore, the collateral vessels are vulnerable to the sequelae of distal embolization should it occur.

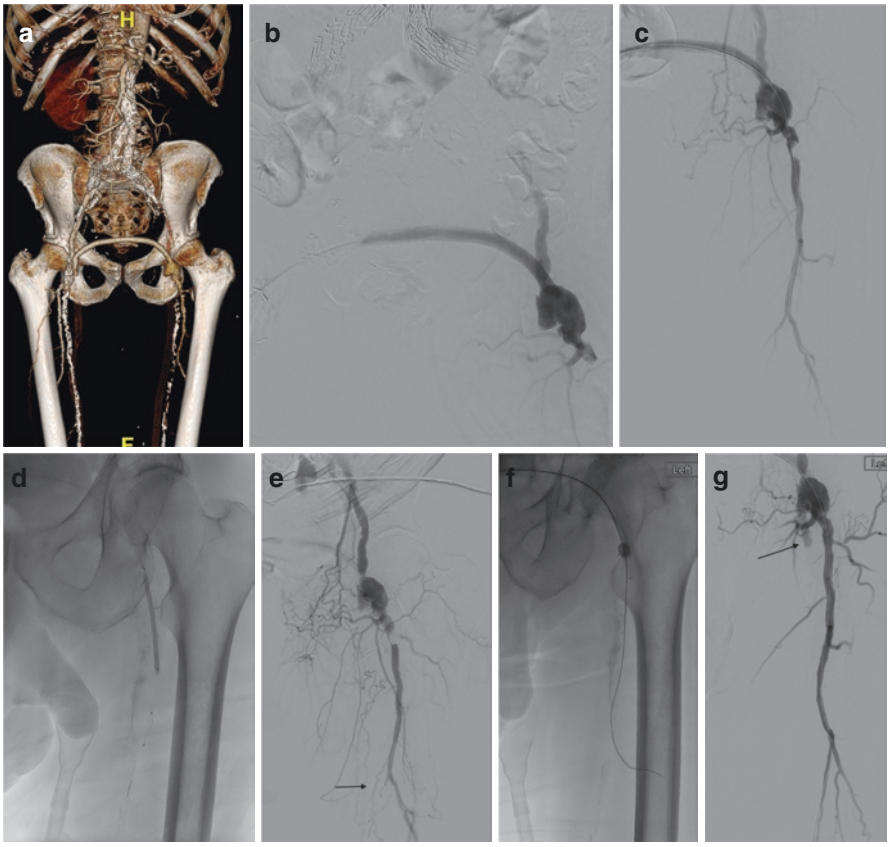

Fig. 17.4 (**a**) Complicated left leg rest pain in a patient with occluded native left iliac, SFA, and fem-pop bypass graft with a patent fem-fem bypass graft. CTA had been performed several months earlier. (**b**) Initial angiogram from the right end of bypass graft showing the occluded left main PFA. (**c**) Lesion was crossed and TPA administered overnight. High-grade stenosis seen at PFA origin. (**d**) The lesion is crossed with a 4-Fr Glidecath over the 0.035″ Wholey wire. Once across, a 6-mm-diameter SpiderFX is deployed in a distal portion. Penumbra aspiration and 4-mm PTA are then performed. (**e**) After the above intervention, increased collaterals are now seen, but PFA plaque or probable clot persists. The distal filter can be seen with wire extending into the medial side branch now with debris in the basket (arrow). Filter was removed with the recovery catheter. (**f**) With the native SFA open at the stump, decision was made to use a Fogarty balloon over the wire to pull the recalcitrant plaque/clot back into the CFA and have it float into the occluded SFA. (**g**) Final angiogram after additional angioplasty showing the improved flow to the PFA and its branches. The plaque/clot of the PFA can be seen in the SFA stump (arrow)

Conclusion and Future of Distal Protection

The role of distal protection in PAD cases has remained an enigma for most interventionalists. EPD is extremely useful in treating large caliber vessels such as the iliac, CFA, SFA, PFA, and popliteal arteries especially when atherectomy are being

used. EPD are useful in treating high-risk lesions such as thrombotic segments, long segment occlusions, in-stent restenosis, and heavy calcified lesions all complicated by limited distal runoff.

If not useful, why are EPD not used more frequently in treating PAD? There appears to be three main factors. Firstly, as the various studies, corporate-sponsored registries, and few randomized trials have shown, it is difficult to obtain standard complication rates. The fact that distal embolization occurs cannot be denied, but the rate of significant events related to the distal emboli is hard to determine; there are many variables that involve assessing and treating PAD patients; it is hard to determine what is successful and what is not in treating the PAD population.

Secondly, EPD are not perfect and have faults. You lose wire access when you need to get distal to the filter, filter baskets can get full and hard to clean out, and, importantly, filters cannot prevent distal emboli from getting past or through the filter. There is much improvement that can be done with EPD technology. Possibly, as EPD are used more frequently in the cerebral, coronary, and other circulations, there will be improvements in its use and design in the PAD.

Finally, after 20 years since their invention, EPD for PAD are still not reimbursed in the US market. We looked into creating a randomized trial for one of the EPD in 2006, but it was not possible to establish reliable endpoints. Strangely, the reimbursement of PTA and atherectomy in outpatient facilities is $11,000 versus $3500 for PTA alone (CPT 37,225–37,224). So with EPD costing approximately $1600–$2000 per device, there is much hesitation for interventionists especially at outpatient facilities to employ EPD use, especially in cases without atherectomy use.

In summary, distal embolization can occur with any intervention in treating PAD. When it does occur, it results in longer case time, contrast, and fluoroscopy at the minimum. Worse cases will result in worsened claudication, rest pain, amputation, and even death. The best approach to avoid distal embolization is to prevent its occurrence: though EPD filters are not perfect, they do provide protection from harmful embolic debris.

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