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Chapter 1

Introductory Chapter: Superficial Femoral Arterial Disease

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Additional information is available at the end of the chapter

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1. Introduction

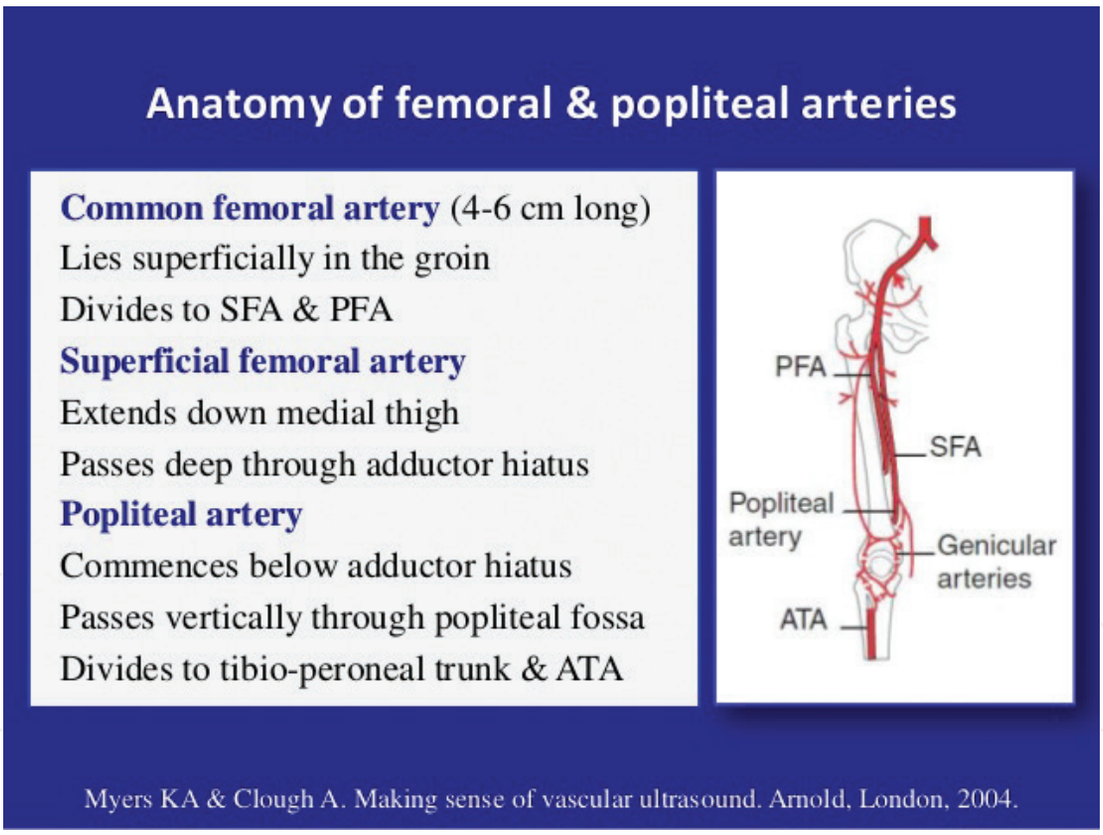
Superficial femoral artery, traditionally grouped together with the popliteal artery, as femoral popliteal segment, is the most common localization of peripheral artery disease (70%). Atherosclerotic disease of superficial femoral artery is deemed high risk to present with diffuse distribution, frequent calcification, large plaque burden, and high progression rate to total occlusion [1]. Additionally, femoral popliteal segment is exposed to significant flexion, bending, and compression forces. Hence, unique anatomical features, along with nonfavorable atherosclerotic plaques characterize in this region, contributes to a significant challenge, when treating this disease. Apart from the variation in the anatomy and the presentation of the pathology in superficial femoral artery area, there are many unique features in the presentation and diagnosis as well. These in concert with the downstream areas of supply at risk, can present with pain on exertion, pain at rest, or/and with nonhealing ulcers extending anywhere from thigh to feet area. It is crucial to understand that while these issues could arise from focal stenosis at the presentation level, consideration should always be given to an inflow lesion.

2. Anatomy

Superficial femoral artery arises, at the level of the femoral head, as the continuation of the common femoral artery, medially and anteriorly to profunda femoral artery. After the origin, superficial femoral artery enters the femoral triangle, coursing toward the abductor canal. Subsequently, it leaves the abductor canal through abductor hiatus to enter the popliteal fossa.

At this level, the artery name is changing to the popliteal artery, which usually bifurcates into the anterior tibial artery and tibioperoneal trunk. One of the unique features of superficial femoral artery is lack of the major branches. The only named branch is the descending genicular branch, contributing to collateral flow of the knee when there is a stenosis in the area. Additionally, when discussing the superficial femoral artery disease, profunda femoral branch deserves a special attention, as the potential source of significant collateral flow, which should be evaluated before decision-making about the intervention on superficial femoral artery [2].

Anatomy is especially important when evaluating the superficial femoral arterial stenosis in patients with concerning clinical presentation on abdominal aiortogram with run-off. In cardiac catheterization laboratory, we place a pigtail in the abdominal aorta above the level of iliac bifurcation. There is an option of performing stepped digital subtraction views with 100 ml contrast at the rate of 10–15 ml/sec. The alternative option is to cross over the iliac artery of the affected lower extremity and then use 50 ml contrast at the flow rate of 10 ml/sec for the imaging. Adequate images can be obtained if patient does not move the lower extremities.



3. Pathophysiology of superficial femoral arterial (SFA) disease

While the pathology can range from atherosclerosis to thrombosis, the majority is attributed to atherosclerosis in the lower extremities. The detailed pathophysiology has been described in the chapter of aortoiliac disease. As outlined above, atherosclerotic disease of superficial femoral

artery is characterized by diffuse distribution, significant plaque burden, frequent calcification, and the high risk of progression to total occlusion, which is likely dictated by distinguishable anatomical features, including significant length of the segment, measuring about 50 cm in an average adult patient. The management is dictated by the extent, location, and intensity of the stenosis and calcification. It can range from medical therapy alone to aggressive interventional approaches. The interventional approaches can range from percutaneous interventions to surgical interventions, again based upon the extent of pathology and patient clinical risk factors.

4. Clinical symptoms and diagnosis

Clinically, intermittent claudication is the classic symptom of peripheral arterial disease (PAD). Usually, patients with SFA disease endorse pain in the upper two-thirds of the calf. Nevertheless, approximately only 10% of patients report these symptoms. About half of them complain about atypical leg pain and about 40% remain asymptomatic. Acute limb ischemia is an uncommon presentation (1–2%) [3]. However, it can be limb threatening.

Usually, the diagnosis of PAD is made clinically based upon symptoms and signs. Physical examination typically reveals diminished peripheral pulses and skin changes over the poorly supplied area. Elevation pallor and dependent rubor are signs of advanced PAD. Physical examination may be helpful in localizing the segment of obstruction. Intact femoral pulses, diminishing peripherally and vascular bruit over the SFA are suggestive about this vessel involvement. Ankle brachial index (ABI) is a useful confirmatory test for PAD (ABI ratio < 0.9). Pursuing topography is justified in case of potential intervention. The approach to the patient with suspected PAD and information about testing modalities were outlined in detail in the chapter dedicated to aortoiliac disease.

5. Classification

Clinical symptoms are the fundaments of Fontaine classification. This is a widely used system, encompassing four stages of PAD, ranging from asymptomatic disease to apparent necrosis of the limb (**Table 1**). Similarly, Rutherford classification included patient's symptoms, but was enriched by objective data, not requiring invasive measures (**Table 2**) [4].

Anatomically, SFA has been grouped together with neighboring arterial vessels as the femoral popliteal segment. This facilitates the management even more significantly. Further anatomical details about the lesion, including the disease pattern and the number of changes, were incorporated into Trans-Atlantic Inter-Society Consensus (TASC II). This classification distinguishes type A, B, C, and D type. Accordingly, lesions vary from relatively short stenosis or occlusion to more diffuse changes. This dictates the management plan from medical therapy to revascularization. Importantly, TASC II additionally provides guidelines regarding the treatment strategy, based on intervention success rate. Detailed classification, in relation to femoral popliteal segment was presented in **Table 3** [5].

Stage I – No symptoms
Stage II – Mild claudication
Stage IIa – Intermittent claudication >200 m walking distance
Stage IIb – Intermittent claudication <200 m walking distance
Stage III – Pain at rest
Stage IV – Ulceration/gangrene due to ischemia

Table 1. PAD classification system by Fontaine.

Grade	Category	Presentation	Objective measures
0	0	No symptoms	Treadmill test and reactive hyperemia test within normal limits
	1	Mild intermittent claudication	Can complete treadmill test. Ankle pressure post exertion >50 mmHg, but ≥20 mmHg lower than at rest
I	2	Moderate intermittent claudication	Between mild and severe claudication
	3	Severe claudication	Unable to complete treadmill test. Ankle pressure post exertion <50 mmHg
II	4	Symptoms at rest	Ankle pressure <40 mmHg at rest. Toe pressure <30 mmHg
III	5	Minor tissue loss, focal gangrene, and ischemic ulceration	Ankle pressure <60 mmHg at rest. Toe pressure <40 mmHg
	6	Tissue loss spreading above metatarsal level, extremity cannot be preserved any longer	Same as above

Table 2. PAD classification system by Rutherford.

Type A lesion
Solitary stenosis <10 cm in length
Solitary occlusion <5 cm in length
Type B lesion
Solitary, severally calcified stenosis <5 cm in length
Many lesions, each <5 cm in length
Solitary lesion <15 cm in length
Type C lesion
Multiple lesions totaling >15 cm in length
Recurring lesions after treatment failure
Type D lesion
Chronic total occlusion of SFA > 20 cm in length.

Table 3. SFA lesions – TASC II classification system.

6. Treatment

All patients with PAD should be treated initially the same way with pharmacotherapy, including antiplatelet medications and high-intensity statin, risk factors control, and exercise program, irrespectively of the localization of the lesion. Medical therapy is paramount in the management and should be instituted. Revascularization is a modality reserved for selected cases, including no adequate symptoms control despite appropriate conservative treatment and acute limb ischemia [6]. Detailed information regarding conservative management based on the most recent trials was outlined in the chapter of aortoiliac disease.

7. Revascularization

Revascularization, as mentioned above, is a treatment option only for selected patients with SFA disease. Traditionally, femoral popliteal segment used to be treated with vein bypass surgery. Nevertheless, over the last decades, a rapid evolution of endovascular techniques revolutionized the treatment of PAD. Admittedly, endovascular treatment of SFA disease appears to be specifically complex due to unique biophysical forces over his body area along with often diffuse and calcified atherosclerotic lesions, leading to suboptimal endovascular treatment results and stent restenosis as the significant problem affecting long-term outcome. However, the tendency is pointing toward improving morbidity and mortality index after endovascular treatment [7]. Currently, according to TASC II Update (2015), endovascular approach is the preferred method of treating femoropopliteal lesion up to 10 cm in length [5]. Different modalities of invasive treatment have been presented in a separate chapter.

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References

[1] Klein AJ, Chen SJ, Messenger JC, et al. Quantitative assessment of the conformational change in the femoropopliteal artery with leg movement. Catheterization and Cardiovascular Interventions. 2009;**74**:787-798

[2] Casserly IP, Sachar R, Bajzer C, Yadav JS. Utility of IVUS-guided transaccess catheter in the treatment of long chronic total occlusion of the superficial femoral artery. Catheterization and Cardiovascular Interventions. 2004;**62**(2):237-243

[3] Hirsch AT, Criqui MH, Treat-Jacobson D, et al. Peripheral arterial disease detection, awareness, and treatment in primary care. JAMA. 2001;**286**:1317-1324

[4] Rutherford RB, Baker JD, Ernst C, Johnston KW, Porter JM, Ahn S, et al. Recommended standards for reports dealing with lower extremity ischemia: Revised version. Journal of Vascular Surgery. 1997;**26**(3):517-538

[5] Michael R, Jaff DO, Christopher J, White MD, William R, Hiatt MD, et al. An update on methods for revascularization and expansion of the TASC lesion classification to include below-the-knee arteries: A supplement to the inter-society consensus for the management of peripheral arterial disease (TASC II): The TASC steering committee. Journal of Endovascular Therapy. 2015;**22**:657-671

[6] Anderson JL, Halperin JL, Albert NM, et al. Management of patients with peripheral artery disease (compilation of 2005 and 2011 ACCF/AHA guideline recommendations): A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2013;**127**(13):1425-1443

[7] Diehm NA, Hoppe H, Do DD. Drug eluting balloons. Techniques in Vascular and Interventional Radiology. 2010;**13**:59-63