

**PREDICTORS OF SUCCESS AFTER
FEMOROPOPLITEAL ANGIOPLASTY IN
MANAGEMENT OF CHRONIC LOWER
LIMB ISCHEMIA**

Thesis

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Abstract

Key Words

(PTA- stents- Femoropopliteal- ABI- Distal runoff)

Percutaneous transluminal angioplasty (PTA) has a definite place in the management of peripheral arterial occlusive disease of the lower limb. It has widely accepted as a line of treatment for many patients with femoropopliteal disease. The low complication rate and relatively non invasive nature of PTA has made it an increasing popular intervention. The ABI , pressure gradient and distal runoff status are predictors of success after femoropopliteal angioplasty.

Contents

Acknowledgment	
List of tables	
List of figures.	
Introduction	
Aim of the work.	
Review of literature:	
Chapter (I):	
Arterial wall anatomy-----	5
Chapter (II):	
Pathology of atherosclerosis-----	12
Chapter (III):	
Diagnosis of peripheral arterial occlusive disease-----	24
Chapter (IV):	
Percutaneous vascular access-----	39
Chapter (V):	
Femoropopliteal angioplasty-----	53
Chapter (VI):	
Indications and contraindications of PTA-----	66
Chapter (VII):	
Clinical application of Intravascular Stents-----	76
Chapter (VIII): Complications of PTA-----	84
Patients and methods-----	95
Results-----	102
Discussion-----	128
Summary and conclusion-----	137
Recommendations-----	139
References-----	140
Arabic summary-----	

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List of tables

No.	Title of table	Page No.
1	Progression of atherosclerosis	20
2	Clinical categories of limb ischemia	28
3	Balloon diameter selection according to angioplasty site.	47
4	Preferred options for treating femoropopliteal lesions: <i>(TASC 2000)</i>	63
5	Preferred options for treating femoropopliteal lesions: <i>TASC II 2007</i>	64
6	Clinical improvement after successful percutaneous femoropopliteal angioplasty	65
7	Categories of endovascular intervention	68
8	American Heart Association guidelines for clinical improvement after percutaneous interventions	100
9	Patients demographics	103
10	Clinical indications	104
11	Lesion positions	105
12	Lesions morphology	107
13	Runoff score	109
14	procedural complications	110
15	Clinical improvement after successful percutaneous femoropopliteal angioplasty	111
16	Long term patency of femoropopliteal angioplasty	134

List of figures

No.	Title of figures	Page No.
1	Catheter Head shape determines function	41
2	Balloon angioplasty catheter	48
3	balloon angioplasty of SFA and popliteal artery	51
4	Effect of diabetes	58
5	Effect of runoff score	61
6	Number of male & female patients	102
7	Risk factors for atherosclerosis	103
8	Indications for intervention.	104
9	Lesion positions	105
10	Mean ABI in the pre, immediate and follow up period.	107
11	TASC classification	108
12a-b-c	Case No 1 Pre & post treatment	112
13a-b-c	Case 2 Pre & post treatment	113
14 a-b-c	Case 3 Pre & post treatment	114
15a-b-c	Case 6 Pre & post treatment	115
16a-b-c-d	Case 8 Pre treatment	116
16 e-f	Case 8 post treatment	117
17a-b-c	Case 9 Pre & post treatment	118
18 a-b-c	Case 12 Pre & post treatment	119
19a-b-c	Case 17 Pre & post treatment	120
20a-b-c-d	Case 19 Pre treatment	121
20 e-f	Case 19 post treatment	122
21 a-b-c	Case 21 Pre & post treatment	123
22a-b-c	Case 32 Pre & post treatment	124
23a-b-c	Case 25 Pre & post treatment	125
24a-b-c	Case 26 Pre & post treatment	126
25 a-b-c	Case 27 Pre & post treatment	127

INTRODUCTION

Catheter based endovascular surgical techniques are increasingly being used for the treatment of vascular pathology. Angioplasty, stenting, atherectomy and endovascular grafts have allowed conventional surgical interventions to be converted to be less invasive endovascular techniques. It is for this reason vascular surgeons need to become familiar with endovascular devices, instrumentations, basic principles, indications and limitations (*Daniel et al., 1999*).

Peripheral arterial disease (PAD) is a common manifestation of atherosclerosis. The prevalence of PAD continues to increase, with recent data suggesting that almost 30% of at-risk populations have PAD. The alternatives for treatment of PAD are rapidly expanding. These new options include pharmacotherapy (i.e. cilostazol, angiogenesis), improved surgical techniques, and endovascular therapy. In order to make appropriate decisions regarding therapy, an accurate diagnosis must be established. The diagnosis of PAD includes historical clues, physical diagnostic findings, physiologic tests and arterial duplex ultrasonography (*Michael R., 2002*).

Endovascular surgery offers many benefits as it decreases morbidity and mortality by decreasing the anaesthetic time, the magnitude of the operation and the potential operative injuries further more, the need for post operative intensive care unit and in-stay hospital diminish(*Ahn et al., 1992*).

Percutaneous transluminal angioplasty (PTA) has obtained a definite place in the management of peripheral arterial occlusive disease of the lower limb, it was accepted as a first line treatment for many patients with lower arterial insufficiency. The low complication rate and relatively non invasive nature of PTA made it an increasing popular intervention. Often, there is a dramatic improvement in the presenting symptoms after dilatation as pressure gradients are reduced, flow is improved and distal ischemia is relieved (*Paul Capek et al., 1991*).

PTA has been proposed as a safe effective less expensive alternative to lower extremity arterial bypass graft surgery for treating limb threatening ischemia and claudication. The less invasive nature of PTA and the reports of excellent patency in selected cases have resulted in increasing use of this modality as primary treatment of lower extremity arterial stenoses and occlusions (*Rechard et al., 1998*).

For many years, PTA was applied almost exclusively to the aorto-iliac system for dilating stenoses and reopening short occlusions in these circumstances, there is a little doubt that the long term patency is good especially when combined with stent placement (*White et al., 1995*).

Many interventional radiologists propose that the role of PTA should be extended to include the management of femoro-popliteal and even infra-popliteal arterial occlusions (*Buckenham et al., 1993*).

Chronic lower extremity occlusive disease is most often manifested by mild symptoms of claudication that can be managed conservatively. When conservative therapy fails, endovascular procedures may be effective, particularly if the disease extent is minimal. Surgery may be considered for selected patients with claudication who fail endovascular therapy or are not candidates for it. Patients with more severe symptoms of extremity occlusive disease typically have more extensive disease that is best treated with surgery or with combination of surgery and endovascular therapy. Occasionally, endovascular procedures are performed for patients with extensive disease who are poor candidates for surgery because of severe comorbidity. Further advances in endovascular technology may improve patency in these patients (*Schermerhorn et al., 2003*).

AIM OF THE WORK

The aim of this work is to evaluate the results of femoropopliteal angioplasty according to; the category of the lesion, the type and character of the lesion, the run off state, the associated medical comorbid factors, the use of stent or not, and correlate that with the improvement of Ankle-Brachial Index (ABI) and the clinical outcome.

ARTERIAL WALL ANATOMY

An artery consists of three histologically discrete concentric layers. The inner most, luminal part of the artery, the intima, contains a densely adherent monolayer of endothelial cells, bound together by tight junctions, which provide a barrier that strictly control the entrance of substances to the arterial wall (*Christopher et al., 2000*).

The endothelial cell layer is adherent to the internal or basal elastic lamina, a network of areolar and elastic tissue. This layer is more marked in the medium size and larger arteries. The media contains vascular smooth muscle cells arranged in a closely adherent monolayer or multiple layers depending on the size of the artery (*Christopher et al., 2000*).

The adventia is the outer coat of the vessel, and consists of connective tissue, nerves and vessel capillaries. It links the vessels to the surrounding tissues (*Gartner and Hiatt, 1997*)

Large elastic arteries :

The aorta and its largest branches (common carotid, subclavian and common iliac arteries) are large elastic arteries which conduct blood to the medium-sized distributing arteries. The intima is made of an endothelium, resting on basal lamina, and subendothelial connective tissue layer. The endothelial cells are flat, elongated and polygonal in outline, with their long axes parallel to the direction of blood flow. The subendothelial layer is well developed, contains elastic fibres and type I collagen fibrils,

fibroblast and smooth muscle- like myointimal cells. The latter accumulate lipid with age and in an extreme form, this feature contributes to atherosclerotic changes in the intima. Thickening of the intima progresses with age and is more marked in the distal than in the proximal segment of the aorta(*Gartner and Hiatt, 1997*).

A proximal internal elastic lamina, sometimes split, lies between intima and media. This lamina is smooth, and with the elastic lamellae of the media, is stretched under the effect of the systolic pressure, recoiling elastically in diastole. The media has a marked layered structure, in which fenestrated layers of elastin (elastic lamellae) alternate with interlamellar muscle cells, collagen and fine elastic fibers. The arrangement is very regular, such that elastic lamella and adjacent interlamellar zone is regarded as a ; lamellar unit; of the media. In the human aorta there are 52 lamellar units (*Gartner and Hiatt, 1997*).

The adventia is well developed. In addition to collagen and elastic fibers, it contains flattened fibroblast with extremely long thin processes, macrophages and mast cells, nerve bundles and lymphatic vessels. The vasa vasorum are usually confined to the adventia (*Gartner and Hiatt, 1997*).

FUNCTIONAL MICROSTRUCTURE OF VESSELS:

INTIMA :

The intimal lining of the blood vessels consist of an endothelium, and a variable amount of subendothelial connective tissue, depending on the vessel (*Crossman, 2005*).

Endothelium;

The endothelium is a monolayer of flattened polygonal cells which extends continuously over the luminal surface of the entire vascular tree. Its structure varies in different regions of the vascular bed (*Crossman, 2005*).

The endothelium is a key component of the vessel wall, and subserves several major physiological roles. Endothelial cells are in contact with the blood stream and thus influence blood flow. They regulate the diffusion of substances and migration of cells out of and into the circulating blood. Endothelial cells participate in the formation of blood clots (by secreting clot-promoting factors; Von Willebrand factor); in minimizing clot formation (by secreting prostacyclin, thrombomodulin); and in the process of clot dissolution or fibrinolysis (by secreting tissue plasminogen activator). They have selective phagocytic activity and are able to extract substance from blood. Endothelial cells secrete nitric oxide (NO, relaxing factor) and endothelin (a vasoconstrictor) which affect the tone of smooth muscle in vessel walls. They are sensitive to the dilatation of vessels imposed by the pulse, via stretch sensitive ion channels in the cell membrane. Endothelial cells synthesize components of the basal lamina. They proliferate to provide new cells during the growth in size of a blood vessel, to replace damaged endothelial cells to provide solid cords of cells which develop into new blood vessels (angiogenesis) (*Crossman, 2005*).

Subendothelial connective tissue ;

Subendothelial connective tissue, also termed the lamina propria, is a thin but variable layer. It contains a typical fibrocollagenous extracellular matrix, a few fibroblasts and occasional smooth muscle cells. Endothelial Von Willebrand factor concentrates in this layer and participates in the clotting process when the overlying endothelium is damaged (*Williams et al., 1995*).

Media;

The media consists chiefly of concentric layers of circumferentially or helically arranged smooth muscle cells with variable amounts of elastin and collagen (*Williams et al., 1995*).

Smooth muscle;

Smooth muscle forms most of the media of arteries and arterioles. Contraction of smooth muscle in arteries and arterioles reduces the caliber of the vessel lumen, which reduces blood flow through the vessel and raises the pressure on the proximal side. This role is particularly effective in small resistance vessels where the wall is thick, relative to the diameter of the vessel. Smooth muscle can also alter the rigidity of the wall, without causing constriction (isometric contraction), and this affect the distensibility of the wall and propagation of the pulse(*Crossman, 2005*).

The smooth muscle cells synthesize and secrete elastin, collagen and other extracellular components of the media which bear directly on the mechanical properties of the vessels (*Crossman, 2005*).

In large arteries, where the blood pressure is high, the muscle cells are shorter 60-200 μm and smaller in volume than in visceral muscle. The muscle cells of the arterial media can be regarded as multifunctional mesenchymal cells. After damage to the endothelium, muscle cells migrate into the intima and proliferate, forming bundles of longitudinally oriented cells which reform the layer (*Crossman, 2005*).

Collagen and elastin:

Components of the extracellular matrix are major constituents of vessel walls, and in large arteries and veins they make up more than half of the mass of the wall, mainly in the form of collagen and elastin. Other fibrous components such as fibronectin, amorphous proteoglycans and glycosaminoglycans, are present in the interstitial space(*Williams et al., 1995*).

Elastin found in all arteries and veins is especially abundant in elastic arteries and anastomose with each other to form net-like structure. The internal elastic lamina is seen in arteries between intima and media. This is a tube of elastic material which allows the vessel to recoil after distension. Fenestrations in the elastic lamina allow materials to diffuse between intima and media. An outer elastic lamina, similar in appearance to, but markedly less well developed and less compact than the internal elastic lamina, lies at the outer aspect of the media at its boundary with the adventitia. These laminae are less evident in elastic arteries, where elastic fibers occupy much of the media (*Crossman, 2005*).