



EVALUATION OF DIFFERENT ENDOVASCULAR TECHNIQUES OF RECANALIZATION OF LONG SUPERFICIAL FEMORAL ARTERY OCCLUSION

Thesis
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To My Family

Abstract

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 aim of this work was to evaluate the different techniques of crossing the superficial femoral artery lesions (TASC B and TASC C) in patients with critical limb ischaemia or incapacitating claudication regarding the feasibility, durability and complications. The morphological characteristics of the lesions that may affect the technique were studied. Also the risk factors affecting the patency were studied.

Key Words :

PAD – SFA – Endovascular .

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List of abbreviations

ABI : Ankle Brachial Index

ACD : Absolute Claudicating Distance

AP : Ankle Pressure

CFA : Common Femoral Artery

CT : Computed Tomography

CT : Cutting Ballon

CTO : Chronic Total Occlusion

ICD : Initial Claudicating Distance

ISR : In-Stent Restenosis

IVUS : IntraVascular UltraSound

LDL : Low Density Lipoprotein

MRA : Magnetic Resonance Angiography

PAD : Peripheral Arterial Disease

PIER : Percutaneous Intentional Extraluminal Recanalization

PTA : Percutaneous Transluminal Angioplasty

PVR : Pulse Volume Recording

SFA : Superficial Femoral Artery

SIA : SubIntimal Angioplasty

TASC : Trans Atlantic Inter- Society Consensus

TM : TreadMill

TP : Toe Pressure

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 , 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 has been proposed as a safe, effective and less expensive alternative to lower extremity arterial bypass surgery for treating critical limb ischemia and claudication. The less invasive nature of the endovascular surgery and the reports of excellent patency in selected cases have resulted in increasing the use of this modality as a primary treatment of lower extremity arterial stenoses and occlusions (*Richard et al., 2002*).

Angioplasty of long stenotic lesions or chronic total occlusions in the superficial femoral (SFA) and popliteal arteries may be suboptimal. Implantation of stent in this region has a high restenosis rate. Changes in

arterial flexion with daily activity can result in fatigue and fracture of metallic stents in the femoropopliteal arterial system (***Rabbi JF et al., 2004***).

Subintimal angioplasty, sometimes described as percutaneous intentional extraluminal recanalization (PIER) was first introduced in 1987 to treat femoropopliteal occlusive disease in intermittent claudication. Since that time, this technique has been extended to the treatment of stenotic and occlusive lesions in the iliac and popliteal arteries, trifurcation, and crural vessels where it has found an important role in the management of critical limb ischemia (***Bolia A et al., 1989***).

Recent studies have shown that SIA can produce good results not only in chronic critical limb ischemia but also in intermittent claudication. SIA can now help to extend the scope of endovascular therapy to include a large number of previously untreatable femoropopliteal and tibial occlusions and helps by increasing the long-term patency in these vessels. SIA can be attempted on those patients deemed poor candidates for surgery due to the anesthetic risks or because they have insufficient vein to allow bypass grafting, and also in patients with flush occlusions of the SFA (***Nasim A et al., 1995***).

In addition SIA can be used to re-open occluded native vessels following graft occlusion. The technique is simple, inexpensive, has low complication rates, good primary success rates, and good long-term outcomes (***Walker PR et al., 2001***).

AIM OF THE WORK

The aim of this work is to evaluate the different techniques of crossing the superficial femoral artery lesions (TASC B and TASC C) in patients with critical limb ischaemia or incapacitating claudication regarding the feasibility, durability and complications. The morphological characteristics of the lesions that may affect the technique will be studied. Also the risk factors affecting the patency will be studied.

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 adventitia 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 fibers 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 adventitia 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 adventitia (*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 (a relaxing factor) and endothelin (a vasoconstrictor) which affect the tone of smooth muscle in vessel walls. (*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*).

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. (*Crossman, 2005*).

Collagen fibrils are found in all three vessel layers. Type III collagen (reticulin) occupies much of the interstitial space between the muscle cells of the media, and is also found in the intima. Collagen is abundant in the