INTRODUCTION

Peripheral vascular disease (PVD) is a nearly pandemic condition that has the potential to cause limb loss or even loss of life. PVD manifests as insufficient tissue perfusion initiated by existing atherosclerosis. It most commonly affects the arteries that supply blood to lower limbs importantly SFA. There is a wide range of modifiable and non-modifiable risk factors for PVD including advanced age, male gender, positive family history, smoking, diabetes, hypertension, hyperlipidaemia and obesity [1].

In patients developing limb-threatening critical ischaemia that is indicated by night pain, rest pain, ischaemic ulcers or gangrene, urgent intervention is required in order to avoid limb loss. In such patients, limb loss occurs at a rate of 80-90% if successful revascularization is not achieved [2].

The majority of patients diagnosed with PVD (70%) remain stable or improve in response to lifestyle modifications such as smoking cessation, physical activity and dietary changes, as well as pharmacological interventions including antiplatelet therapies, lipid lowering and glucose controlling agents, and antihypertensives [3].

However, for patients who do not improve with conservative management, revascularization procedures should be used to restore peripheral circulation. These include stenting

(implantation of a wire mesh scaffold) into the artery to keep it patent. Stents can be balloon-expandable, which require an angioplasty balloon to expand and set the stent within the artery, or self-expandable [4].

On the other hand A recent innovation is to employ drug-eluting stents (DES) and balloons (DEBs) which are coated with anti- proliferative drugs such as paclitaxel and sirolimus, that inhibit the proliferation of neointimal growth of vascular smooth muscle cells [5].

AIM OF THE WORK

The purpose of the study is to compare the outcome of using drug eluting balloons versus stenting in patients with SFA lesions for the following parameters:

1) Walking (claudication) distance before and 6 months after revascularization.

2) Ankle brachial index (ABI) before, immediately after and 6 months after intervention.

Chapter One

ANATOMY OF THE FEMORAL ARTERY

The femoral tree constitutes the major blood supply to the lower limb. In the thigh, the common femoral artery passes through the femoral triangle which is a wedge-shaped depression formed by muscles in the upper thigh. The medial and lateral boundaries of this triangle are formed by the medial margin of adductor longus and the medial margin of sartorius, respectively. Fascia lata constitutes the roof of the femoral triangle whilst the floor is formed by pectineus and adductor longus medially as well as by iliacus and psoas major laterally. Within this triangle, the common femoral artery is enclosed in the femoral sheath with the femoral vein. The common femoral artery and its branches supply most of the thigh as well as the leg and foot [6].

Anatomical Course

Midway between the anterior superior iliac spine and the symphysis pubis, the external iliac artery passes under the inguinal ligament into the femoral triangle and becomes the common femoral artery. The artery then travels down the anteromedial aspect of the thigh as the superficial femoral artery (SFA) before it passes through the adductor (subsartorial) canal the popliteal artery. When the SFA passes through the adductor hiatus it becomes [7].

Branches

The femoral artery gives off five branches in the femoral triangle and one in the adductor canal, to give six in total. These branches are referred to as the

- Superficial epigastric artery
- Superficial circumflex iliac artery
- Superficial external pudendal artery
- Deep external pudendal artery
- Profunda femoris
- Descending medial genicular artery

The superficial epigastric artery arises from the femoral artery 1 cm distal to the inguinal ligament. It travels through the cribiform fascia and ascends towards the umbilicus within the abdominal superficial fascia. It supplies the skin, superficial fascia and superficial inguinal lymph nodes [8].

The superficial circumflex iliac artery, the smallest branch of the femoral artery, arises near the superficial epigastric artery. Lateral to the saphenous opening, the artery passes through the fascia lata before coursing towards the anterior superior iliac spine. Like the superficial epigastric artery, it supplies the skin, superficial fascia and superficial inguinal lymph nodes.

The superficial external pudendal artery arises near the superficial epigastric and superficial circumflex iliac arteries. It travels through the cribiform fascia before crossing the spermatic cord deep to the long saphenous vein. It supplies the lower abdominal skin as well as the penile, scrotal or labial skin.

The deep external pudendal artery crosses the pectineus and adductor longus muscles before traversing the fascia lata. It supplies the skin of the perineum as well as the skin of the scrotum or labium majus.

The profunda femoris, or deep artery of the thigh, is the largest branch of the femoral artery, which arises 3.5 cm distal to the inguinal ligament. The profunda femoris is initially found lateral to the femoral artery before it passes deep to it towards the medial aspect of the femur. It travels between the pectineus and adductor longus muscles before passing between the adductor longus and adductor brevis muscles. It then descends between the adductor longus and adductor magnus muscles before it pierces the adductor magnus to anastamose with the muscular branches of the popliteal artery. The profunda femoris is the main blood supply to the muscles that extend, flex and adduct the thigh.

The descending medial genicular artery, arises from distal SFA just proximal to the adductor canal. It descends within the vastus medialis muscle to the medial aspect of the knee. Here, it anastomoses with the medial superior genicular artery. Branches of

this artery supply the vastus medialis and adductor magnus muscles as well as the proximomedial skin of the thigh [9, 10, 11].

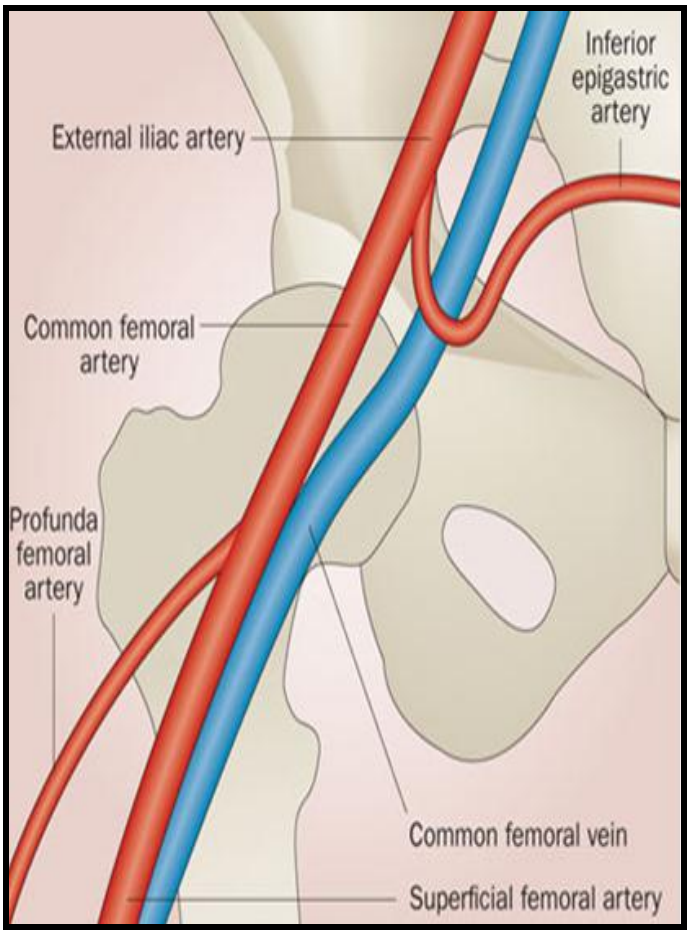

Figure (1): Femoral artery origin and course [8]

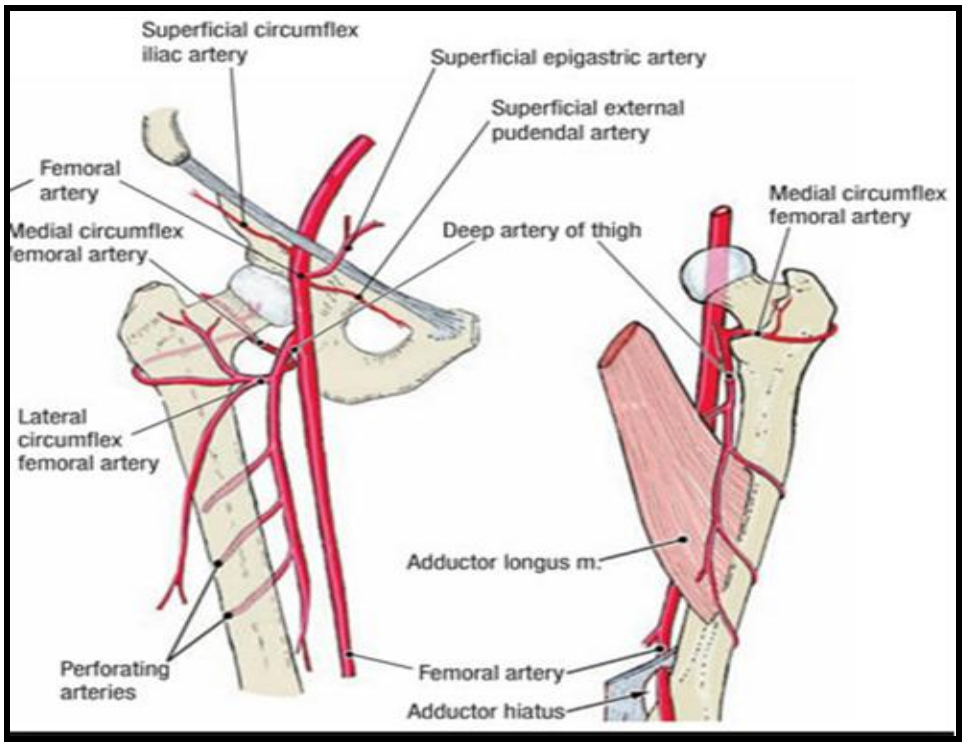

Figure (2): Femoral artery branches [10]

Chapter Two

PATHOPHYSIOLOGY OF PAD AND CLASSIFICATIONS

Atherosclerotic PAD involves several highly interrelated processes that include lipid disturbances, platelet activation, thrombosis, endothelial dysfunction, inflammation, oxidative stress, vascular smooth cell activation, altered matrix metabolism, remodeling, and genetic factors [12].

There are 7 stages of development of an atherosclerotic plaque; first of all when LDL particles move into the subendothelium and get oxidized by macrophages (stages 1 and 2). Then, release of growth factors and cytokines that attract additional monocytes (stages 3 and 4). Finally foam cell accumulation and smooth muscles proliferation resulting in growth of the plaque (stages 5, 6, and 7) [13].

Risk Factors for PAD development

The risk factor most correlated with onset and progression of peripheral arterial disease (PAD) is cigarette smoking. Smokers have a 1.7 to 5.6 folds increase in development of disease compared with nonsmokers [14].

Diabetes mellitus confers a similar increase in risk, augmenting rates of intermittent claudication in men with glycosuria 3.5-folds compared to men who are not diabetic [15].

Effects of dyslipidemia and hypertension are less impressive than diabetes and smoking. Relative risk for PAD is ≈1.1 for each 10-mg/dL increase in total cholesterol with similar increases for development of claudications [14].

Classifications

Rutherford Classification

The symptomatic classification was adapted by Rutherford in 1986 [16] and was revised in 1997 [17]. Rutherford classified PAD into acute and chronic limb ischemia, emphasizing that each presentation requires a different treatment algorithm. Rutherford also associated patient clinical symptoms with objective findings, including Doppler, ankle brachial indices (ABI), and pulse volume recordings. Acute versus chronic presentation implies timing of symptom onset; however, Rutherford did not include stringent temporal criteria in the definitions. This classification has been used widely in clinical settings to direct patient management as well as for research purposes [18].

Rutherford's chronic limb ischemia classification (table 1) most resembles Fontaine's classification, with the addition of objective noninvasive data [17]. The evaluation for any patient with chronic limb pain should include evaluation of the symptoms described in Rutherford's classification. The character of the patients' pain should be evaluated. Claudication onset should be determined, and can be reliably verified by

walking/treadmill tests in the noninvasive vascular diagnostic laboratory. Treadmill exercise testing with and without pre-exercise and post exercise ABIs helps differentiate claudication from pseudoclaudication in patients with exertional leg symptoms. Treadmill exercise testing may be useful to diagnose PAD with a normal resting ABI but a reduced post-exercise ABI. Treadmill exercise testing may objectively document the magnitude of symptom limitation in patients with claudication [18, 19].

Patients who cannot perform treadmill testing can undergo similar stress testing using plantar flexion or thigh blood pressure cuff compression to cause reactive hyperemia.

Table (1): Rutherford classification for chronic limb ischemia [16, 17].

Grade	Category	Clinical description	Objective criteria
0	0	Asymptomatic-no hemodynamically significant occlusive disease	Normal treadmill or reactive hyperemia test
	1	Mild claudication	Completes treadmill exercise, AP(Ankle Pressure) after exercise>50 mm Hg but at least 20 mm Hg lower than resting value
I	2	Moderate claudication	Between categories 1 and 3
	3	Severe claudication	Cannot complete standard treadmill exercise, AP after exercise<50 mm Hg
II	4	Ischemic rest pain	Resting AP<40 mm Hg flat or barely pulsatile ankle or metatarsal PVR (pulse volume recordings), TP(toe pressure)<30 mm Hg
III	5	Minor tissue loss-non healing ulcer, focal gangrene with diffuse pedal ischemia	Resting AP<60 mm Hg flat or barely pulsatile ankle or metatarsal PVR, TP<40 mm Hg
	6	Major tissue loss-extending above Transmetatarsal (TM) level, functional foot no longer salvageable	Same as category 5

Trans-Atlantic Inter-Society Consensus Document II (TASC II)

Fourteen societies representing disciplines in medicine, vascular surgery, interventional radiology, and cardiology from Europe and North America came together in 2000 to form a consensus in the classification and treatment of patients with PAD. The focus was to provide recommendations in the epidemiology of PAD, clinical evaluation, diagnosis, treatment, and follow-up of patients with intermittent claudication, ALI, and chronic limb ischemia. The resulting document was referred to as the Trans-Atlantic Inter-Society Consensus Document (TASC) [15]. In 2007, the consensus was updated and involved additional representatives from Australia, South Africa, and Japan and was referred to as TASC II[16]. TASC II is comprehensive in reviewing the literature relating to PAD up to 2007.

While TASC II addresses all aspects of PAD, the anatomic classification detailed in TASC II has received the significant focus of the review. Specific categories are assigned treatment algorithms (surgical vs. endovascular) based on lesion classification. TASC II divides anatomic distribution of lesions into aorto-iliac and femoro-popliteal [20]. Lesion patterns are grouped into A–D lesions. Based on this group recommendation, TASC A lesions are those that should have excellent results from endovascular management alone. TASC B lesions are those that should have good results from endovascular management, and endoluminal interventions

should be the first treatment approach. TASC C lesions are those for which surgical management provides superior long-term results and endovascular techniques should be reserved for patients who are surgically high risk. TASC D lesions should be treated by open surgery. While TASC II provides a framework to compare therapeutic techniques, advancement of endovascular techniques have led to many trials suggesting that endovascular management of TASC II C and D lesions is a potential alternative treatment to open strategies [21-30].

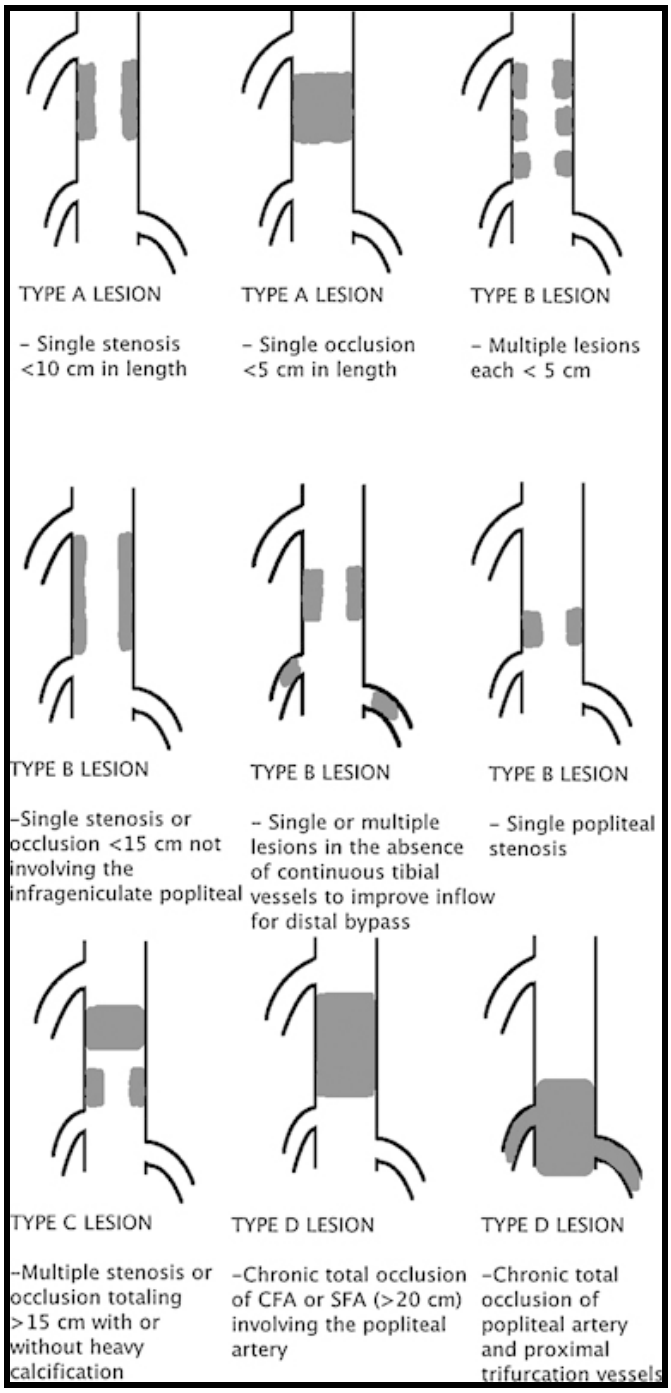

Figure (3): TASC II classification [20]