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Approach to Peripheral Arterial Disease (PAD)

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Abstract

Peripheral arterial disease is a complex disease affecting the arterial system of the lower extremities. It has a multifactorial etiology presenting with a wide spectrum of symptoms. Clinical examination, laboratory evaluation and imaging are essential for accurate assessment of the severity of the disease. Treatment is multidisciplinary comprising medical therapy as well as surgical intervention. The article provides a systematic approach to assessment and treatment of peripheral arterial disease.

Keywords: Peripheral, Arterial, Disease, Risk Factors, Diagnosis, Treatment

Introduction

Peripheral arterial disease is a common problem faced by the general surgeon. Increased comorbidities such as diabetes, hypertension and smoking are associated with rising incidence of peripheral arterial disease (PAD).[1] A systematic approach to clinical assessment, investigation and treatment strategies is essential to reduce the morbidity in the form of limb loss and mortality associated with vascular accidents such as stroke and myocardial infarction (MI).[2] This article provides the comprehensive approach to patient presenting with peripheral arterial disease.

Clinical assessment

Patient suffering from peripheral arterial disease can be categorized into 4 groups, as per the guidelines outlined by American Heart Association and American Association of Cardiology.[3] The guideline is specific for PAD involving the lower extremity. The specific categories are as follows

1. Patients with age 65 years or older
2. Patients with an age group ranging from 50 and 64 accompanied with risk factors for atherosclerosis

namely smoking,diabetes, hypertension, hyperlipidemia or with a family history of PAD

3. Patients younger than 50 years of age with diabetes mellitus predisposed to having 1 or more additional risk factor for atherosclerosis
4. Patientswith atherosclerosis involving other systems such as coronary, carotid, renal, subclavian and mesenteric vessels.

History taking

A detailed history is essential. History of PAD symptoms which include claudication, rest pain, non-joint related lower extremity symptom's, impairment of walking and non-healing wounds.[4]

Intermittent claudication is the commonest symptom of PAD. Cramp like pain in the ischemic muscle on exercising is diagnostic. The severity of claudication can be assessed by correlating the pain and exercise potential despite the pain.

Two classification systems are commonly used to grade the severity of claudication which include

- Rutherford classification based on performance of 5-minute treadmill test at 2 mile per hour on 12-degree incline. (Table 1) [4,5]
- Fontaine classification which assigns based on symptoms. (Table 2) [4,5]

Rest pain is the end result of severe vascular compromise. Non healing wounds in the lower extremity are suggestive of severe vascular compromise. Previous history of healed ulceration and amputation which include amputation of toes or major amputation. Erectile dysfunction also needs to be taken into consideration while evaluating peripheral vascular disease. Many a times symptoms of PAD maybe a local manifestation of a systemic problem. Therefore, other vascular beds need to be evaluated. These include carotid for TIA's and strokes, cardiac system for angina and any previous history of cardiac events including surgical interventions, renal system associated with uncontrolled hypertension and mesenteric system presenting with post prandial abdominal angina and mesenteric vascular thrombosis.[6] A detailed evaluation of co-morbidities and their treatments along with level of response to treatment is essential for developing an effective plan for further management as this could impact a successful outcome.

Physical Examination

Includes general and local examination.

General Examination includes assessment of level of cerebration followed by physical examination.

Assessment of all peripheral pulses which include carotid, subclavian, abdominal aortic, femoral, popliteal, ankle pulses is pivotal in identifying the site of weakened vascularity. (Table 3)

Scars of previous vascular interventions are also important in order to quantify the severity of compromise. This includes scar of carotid endarterectomy, scar of CABG on the chest, abdominal aneurysm surgery, iliofemoral and femoral-popliteal bypass surgery.

Local examination includes elaborate assessment of peripheral pulses, temperature differentials along the lower extremities in order to localize the potential site of narrowing or block. In case of ulcerated lesions, assessment of the ulcer as to whether it's in healing phase is important. As one can decide adjuvant therapy to accelerate the healing process. In case of diabetic patients, assessment of both the lower extremities is essential to identify the foot at risk which necessitates proper advice.

Variation in presentation of PAD

Asymptomatic PAD may occur in patients with atherosclerotic disease in the absence of symptoms. The purpose of identifying patients with asymptomatic PAD is important as such patients are at very high risk of vascular accidents.[7] Such patients may benefit by cardiovascular risk modification. Symptoms may include limitation of ability to exercise by virtue of comorbidities example, CCF and COPD. Altered pain perception due to peripheral neuropathy in diabetics may mask the symptoms of PAD. Atypical leg pain which is of two types may be seen. [7.8] First type is pain on exertion and rest. This is distinct from rest pain in chronic ischemia. Leg pain which is exertional which doesn't stop the patient from continuation to walk. Patient suffering from leg pain tend to have functional impairment and rapid decline. Incidence of leg pain accounts for about 40-50% in PAD.[7]

Acute limb ischemia (ALI) is sudden decrease in lower limb perfusion which threatens limb viability in patients who present within 2 weeks of the insightingevent. [8,9] Symptoms are pain, pallor, paresthesia, pulselessness, paralysis, poikilothermia

ALI a surgical emergency requiring immediate treatment. [10] ALI is caused by thrombosis of a pre-existing stenotic lesion and embolism. It may also result from arterial dissection and thrombosis of the aneurysm or occlusion of the stent or bypass graft.

Chronic ischemia (Critical limb ischemia) is a condition characterized by presence of ischemic rest pain, nonhealing ulcers and gangrene over a period of 14 days. [11] Rest pain typically occurs in fore foot and is relieved on positioning it in dependent position.

Diagnostic Tests for PAD

Ankle Brachial Index (ABI)

This is a diagnostic test to establish the diagnosis of PAD.[11]First the systolic BP in both arms and from both ankle pulses is obtained after the patient is at rest in supine position for 10mins. ABI is calculated by dividing the highest systolic blood pressure in the foot by highest systolic BP in the arm. An abnormal ABI is less than or equal to 0.9 whereas borderline ranges from 0.91 to 0.99. A Normal ABI ranges from 1.0-1.4.Non compressible atherosclerotic artery may exhibit an ABI of 1.4.

Exercise treadmill ABI

Patient with exertional non joint related symptoms who have normal or borderline ABI (0.9-1.4) should undergo exercise treadmill test. Abnormal treadmill ABI is defined as decrease in ABI by 20% or greater after exercise and is diagnostic for PAD. A normal exercise ABI is defined as no change in ABI or increased ABI after exercise.[11]

Absolute Toe pressure (TBI)

TBI is similar to ABI. [12,13]It is the ratio of systolic BP of the great toe to higher of 2 arms brachial artery pressure. TBI less than 0.5 is classified as abnormal. An abnormal TBI is specific for PAD. In patients with suspected PAD wherein ABI is greater than 1.4 due to thickened artery, TBI should be measured to confirm the diagnosis of PAD. This type of response is seen in diabetes and end stage renal disease. TBI is also helpful in assessing the chance of wound healing and absolute toe pressure above 30mm Hg favors better wound healing. However, in diabetic patients an absolute toe pressure of above 40-55mm Hg is necessary for adequate healing.[14]

Evaluation of pulse volume recording (PVR), transcutaneous O2 measurement (TCPO2) and skin perfusion pressure (SPP)

PVR wave forms are obtained non-invasively.The nature of the wave form reveals the severity of the disease at each level of the leg. A normal PVR is characterized by a steep systolic upstroke with the sharp peak followed by a downstroke with prominent dichotic notch. Mild to moderate loss of systolic peak amplitude, dicrotic notch and outward projection of the down stroke is seen in PAD. In severe PAD the amplitude of systolic peak is severely diminished. [15]

TCPO2 and SPP provide information on the status of tissue perfusion.[16] Normal TCPO2 at the level of the foot usually exceeds 50mm Hg. A TCPO2 of greater than 50mm Hg suggest that the wound may heal whereas a TCPO2 of less than 20mmHg indicates severe ischemia and low probability of wound healing.Sucha patient is an ideal candidate for attempted revascularization.

SPP less than 30mm Hg has good specificity and sensitivity in diagnosing PAD.

More than 30mm Hg is associated with high probability of wound healing.

Imaging for anatomical assessment of patients with PAD is essential for selecting patient for vascular intervention only. Symptomatic patient may be considered for revascularization. Duplex USG, CT-angiography, MR-angiography are helpful in determining the anatomical location and severity of the disease.[17] Digital subtraction angiography (DSA) is specifically useful in patients with CLI. Arterial anatomy is best delineated by DSA with respect to

- Site of the block
- Length of the block
- Quantity of collateral circulation
- Distal runoff

These are the four findings which need to be critically assessed. Patients of PAD with more proximal block, apparent collateral circulation and good distal runoff are ideal candidates for vascular intervention with promising outcomes. [18]

Treatment

No single modality of treatment can is effective or prevent complication arising from PAD. Every patient needs to be critically studied and multi-disciplinary approach has to be planned. Each patient will exhibit a better response to a particular modality. Therefore, planning a comprehensive algorithm for each patient is the mainstay of treating the patient suffering from PAD.[18]

Medical

Medical therapy plays a pivotal role. The main aim of medical therapy is the risk modification and improvement in the functional status through this modification. Risk Modification includes cessation of smoking, control of blood pressure, control of cholesterol and diabetes. Anti-platelet and cilostazol are also needed. Smoking cessation is the most important modality for treating PAD. Immediate, complete and permanent cessation of smoking prevents progression of disease especially in cases of TAO. [19,20]

Anti-hypertensive therapy is prescribed to all patients of PAD suffering from hypertension. Angiotensin converting enzyme inhibitor and Angiotensin receptor blocker significantly reduce cardiovascular events in patients with PAD. In addition to the above, diuretics, B-blockers and calcium channel blockers are also suitable in certain cases of hypertension. [20]

Statin therapy is to be always prescribed in patients having PAD. There is significant reduction in cardiovascular events including mortality in patients with PAD who are on stains.[20]

Meticulous multispecialty treatment is essential for good glycemic control. It helps in lowering the rate of amputation and at the same time improves revascularization in patient suffering from CLI. [21]

Long term antiplatelet therapy with aspirin alone(75-350mg) or clopidogrel(75mg) is recommended in symptomatic PAD. Clopidogrel shows better results in preventing CVS events.[22,23]

Cilostazol and pentoxyphylline is a phospho-diesterase inhibitor and is recommended for symptomatic treatment of patients with claudication.[24] Patients may experience significant improvement in vascular symptoms with these medications. Structured exercise therapy which includes regular physical activity within physiological limits reduces cardiovascular events, improves lipid profile, reduces weight and blood pressure.[25]

Surgical

Proper wound care for active ulceration or gangrene of the lower extremity necessitates debridement and evaluation of arterial patency. Sharp debridement with removal of all necrotic tissue and use of de-sloughing agents helps in improvement of the wound. Non adherent dressings are preferred. Dressing may also be used to prevent excessive pressure which may exacerbate ischemia.

Vascular intervention includes angioplasty or stenting depending upon the findings on DSA. With proximal blocks bypass grafting may be helpful. [25, 26]

Hyperbaric oxygen therapy (HBOT) is salvage for patients with PAD wherein all modalities have been utilized with poor results.

HBOT controls infection and induces neo-angiogenesis thereby improving the healing potential of the compromised limb. The number of sessions required may be variable and needs to be decided based on periodic clinical evaluation with respect to enhanced wound healing

Conclusion

PAD is a very complex disease which concomitantly affects various vascular beds.A critical and elaborate vascular assessment is essential which include clinical evaluation and laboratory testing including vascular imaging. Every patient needs to be individualized based on the findings. A multi-modality approach is always associated with improved outcomes thereby reducing limb loss and improve wound healing.

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Legend Tables

Table 1: Rutherford's Classification

Grade	Clinical Inference
0	Asymptomatic
1	Mild claudication
2	Moderate Claudication
3	Severe Claudication
4	Ischemic rest pain
5	Minor tissue loss like non healing ulcers of limb, localized gangrene
6	Major tissue loss typically extending above trans-metatarsal

Table 2: Fontaine's Classification

Grade	Clinical Inference
1	Asymptomatic
IIa IIb	Mild claudication Moderate – Severe Claudication
III	Ischemic Rest Pain
IV	Ulceration or Gangrene

Table 3: Assessment of Peripheral Pulses

Peripheral Arterial Pulses	Anatomical Landmark
Superficial Temporal	Just Anterior to the Tragus
Carotid	Palpate the carotid artery by placing your fingers near the upper neck between the stern mastoid and trachea roughly at the level of cricoid cartilage (Medial to sternocleidomastoid)
Brachial	palpated medial side of antecubital fossa, just medial to tendinous insertion of the biceps
Radial	Palpated Lateral to the Tendon of Flexor Carpi Radial is Muscle and medial to the styloid process of radius
Femoral	Inferior to the inguinal ligament and midway between ASIS and pubic symphysis
Popliteal	Deep in the popliteal fossa medial to the midline, Knee should be semi-flexed while examination
Posterior Tibial Pulses	Posteroinferior to the medial malleolus in the groove between the malleolus and the heel
Dorsalis Pedis	Lateral to the tendon of Extensor Hallucis Longus