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The Role of Physical Activity in Lower Extremity Peripheral Artery Disease

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Abstract

Peripheral artery disease (PAD) is a common disorder characterised by a reduced blood circulation caused by stenosis or occlusion of peripheral arteries of the limbs, predominantly affecting the lower extremities. This condition is typically related to atherosclerosis, but it might also be caused by vasculitis, fibromuscular dysplasia, or adventitial cystic disease. Although atherosclerotic occlusive disease of the lower extremities has high mortality and morbidity rates, it remains underdiagnosed and undertreated condition. The risk factors of PAD are similar to those for coronary and cerebrovascular disease. The most common symptom of PAD is intermittent claudication, but recent studies show that asymptomatic PAD is several times more common in the population than intermittent claudication. The medical approach to peripheral artery disease is multifaceted and includes smoking cessation, lipid-lowering therapy, antiplatelet therapy, antithrombotic therapy, peripheral vasodilators, blood pressure management, glycemic control and various methods of revascularization. An important, although frequently neglected aspect is an exercise therapy. Walking exercise is first-line therapy for PAD-related walking impairment. Supervised walking exercise and structured home-based walking exercise each not only improve walking ability, but also have other benefits, which we would like to discuss in this review.

Purpose

This study reviews currently available methods of treatment lower extremities peripheral artery disease to propose the most effective therapeutic approach. Particular attention is given to the role of physical activity in the management of PAD.

Materials and Methods

A review of the current literature was conducted, focusing on exercise therapy categorised into two main types- supervised walking exercise and structured home-based walking exercise. Other treatment modalities were evaluated. Epidemiology, pathogenesis, clinical presentation, diagnostic approach and risk factors were discussed. The literature available in the PubMed and Google Scholar databases was searched using predefined keywords.

Results

Various methods of treatment may be performed. Individual approach and integrated care are the essential factors to provide satisfying management of the disease. Walking exercise is first-line therapy for PAD-related walking impairment. Supervised exercise therapy has particular importance in ensuring acceptable quality of patient’s life. Physical activity plays crucial role not only in treatment of PAD, but also is an important modifiable risk factor. Further studies are warranted to improve effectiveness of treatment.

Keywords

Peripheral Arterial Disease, Atherosclerosis, Ischemia, Intermittent Claudication, Endothelial Dysfunction, Exercise Therapy

1. Introduction

Peripheral artery disease (PAD) is a common disorder characterized by arterial narrowing and a consequent reduction in blood flow. In this review, we focus primarily on atherosclerotic occlusive disease of the lower extremities. Although PAD may result from a variety of etiologies, it is most commonly associated with atherosclerosis. Atherosclerosis is a chronic inflammatory disease characterized by the formation of lesions within the intima, known as atheromas or fibrofatty plaques. The development of these lesions is influenced by multiple factors, including genetic, metabolic, and environmental determinants, many of which are associated with elevated levels of low-density lipoprotein cholesterol (LDL) [1]. The formation of atheromas is initiated by endothelial activation in the presence of increased LDL concentrations. Subsequently, leukocytes are recruited to the arterial intima, where they interact with lipoproteins and their derivatives [2]. This process leads to the accumulation of lipids, fibrous components, and calcium deposits [3], followed by activation of inflammatory pathways. Inflammatory mediators play a key role in regulating both the progression and healing of fibrofatty plaques. These pathological changes within the arterial wall may ultimately result in significant luminal narrowing or complete vascular occlusion. PAD predominantly affects the arteries of the lower extremities. The most commonly involved vascular segments include the aortoiliac, femoropopliteal, and infrapopliteal arteries [4,5]. Less frequently, PAD involves the upper extremities. In some cases, other vascular territories, such as the abdominal aorta, renal arteries, and celiac arteries, may also be affected. Despite its clinical significance, PAD remains underdiagnosed and, consequently, undertreated. One of the primary reasons is the high proportion of asymptomatic cases [6]. Nevertheless, the global burden of PAD continues to rise. It is estimated that more than 200 million individuals worldwide are affected by PAD, with over half of cases remaining asymptomatic. The prevalence of PAD is higher in high-income countries than in low- and middle-income countries (7.4% vs. 5.1%), although the absolute number of affected individuals is greater in low- and middle-income countries due to larger population sizes [7].

2. Clinical presentation

Peripheral artery disease (PAD) may manifest across a broad spectrum of clinical presentations, ranging from asymptomatic disease to severe chronic limb-threatening ischemia [8]. Clinical symptoms largely depend on the location of the affected artery. Other important determinants include vessel size, metabolic demand, and the extent of collateral circulation [9]. The most characteristic symptom of PAD is intermittent claudication, defined as exertional, cramping calf pain that is absent at rest and resolves within approximately 10 minutes of rest [10]. Nevertheless, individuals with PAD frequently present with atypical lower extremity symptoms, which may be misinterpreted as manifestations of knee osteoarthritis or spinal stenosis [11]. Approximately 70–90% of individuals with an ankle–brachial index (ABI) of less than 0.90 are either asymptomatic or present with exertional leg symptoms that differ from classic claudication. A substantial proportion of patients do not report leg symptoms because they have compensated by reducing their level of physical activity or slowing their walking pace to avoid symptom onset. Consequently, approximately 75% of patients with PAD demonstrate significantly greater annual declines in six-minute walk performance compared with individuals without PAD [10]. Atypical lower extremity symptoms may include pain or discomfort that begins at rest but worsens with exertion, pain that does not limit walking, or exertional discomfort that is not relieved within 10 minutes of rest [12]. The course of the disease is progressive. Over time, patients with PAD may develop non-healing wounds, gangrene, as well as elevation pallor or dependent rubor. Abnormal peripheral pulses further increase the likelihood of PAD [13]. Classification systems, such as the Fontaine stages and

Rutherford categories, are useful for characterizing symptom severity and may assist in guiding treatment decisions [14].

Stage	Symptoms
I	Asymptomatic, incomplete blood vessel obstruction
IIA	Claudication at a distance > 200 m
IIB	Claudication at a distance < 200 m
III	Rest pain, mostly in the feet
IV	Necrosis and/or gangrene of the limb

Table 1. The Fontaine classification [15].

Grade	Category	Symptoms
0	0	Asymptomatic
I	1	Mild claudication
I	2	Moderate claudication
I	3	Severe claudication
II	4	Ischemic rest pain
III	5	Minor tissue loss
III	6	Major tissue loss

Table 2. The Rutherford classification [16].

Other symptoms, such as exertional pain in the hip, buttock, or lower back, may indicate involvement of arterial segments proximal to the femoral arteries. It is worth noting that intermittent claudication may be accompanied by impotence and absent femoral pulses. This constellation of symptoms is characteristic of Leriche syndrome [17]. Leriche syndrome, also known as aortoiliac occlusive disease, results from atherosclerotic involvement of the distal abdominal aorta, iliac arteries, and, in some cases, femoropopliteal vessels. The classic symptom triad warrants prompt clinical attention due to the high risk of progression to limb-threatening ischemia. Chronic limb-threatening ischemia (CLTI) represents the most severe form of PAD and is defined by the presence of PAD in conjunction with ischemic rest pain, gangrene, or non-healing ulceration persisting for more than two weeks. The ankle–brachial index is typically <0.4 [18]. CLTI affects approximately 11% of patients with PAD [10] and is associated with a markedly increased risk of all-cause mortality, cardiovascular mortality, and major adverse cardiovascular events over long-term follow-up. Additionally, it is linked to reduced life expectancy and a high risk of limb loss [19]. Two important terms related to lower extremity PAD are major adverse limb events (MALE) and major adverse cardiovascular events (MACE). MALE is defined as major amputation of the affected limb and/or

reintervention on the revascularized segment. In contrast, MACE encompasses cerebrovascular events (stroke), myocardial infarction, or death [20].

3. Diagnostic approach

Diagnosing lower extremity peripheral artery disease (PAD) remains challenging, primarily due to the asymptomatic course observed in the majority of patients. Accordingly, clinical guidelines have been established to facilitate accurate diagnosis. The resting ankle–brachial index (ABI) is recommended as the first-line diagnostic test, in accordance with the 2016 AHA/ACC Guideline on the Management of Patients with Lower Extremity PAD [13]. The ABI is a noninvasive measure obtained using a sphygmomanometer cuff and/or Doppler ultrasound. These techniques are used to determine systolic blood pressure (SBP) in the bilateral brachial arteries, as well as in the dorsalis pedis and posterior tibial arteries, with the patient in the supine position. The highest SBP measured in either arm is used as the reference value. An important consideration is the presence of inter-arm differences in SBP. A difference of ≥15 mm Hg suggests possible subclavian artery stenosis. The ABI for each leg is calculated by dividing the higher pressure of either the dorsalis pedis or posterior tibial artery by the higher brachial SBP [21]. An ABI value of 1.00–1.40 is considered normal, 0.91–0.99 borderline, and ≤0.90 in either limb is diagnostic of PAD [13]. In cases where the ABI is >0.90 but clinical suspicion of PAD persists, post-exercise ABI or other noninvasive tests, including imaging modalities, should be considered. A noncompressible ABI (ABI >1.40) is typically associated with arterial calcification, which may occur in conditions such as diabetes mellitus, hypertension, or chronic kidney disease. Arterial stiffness also increases with age [22]. In such cases, a toe–brachial index (TBI) is recommended. The TBI is defined as the ratio of toe systolic blood pressure to brachial systolic blood pressure, with values ≤0.70 considered diagnostic of PAD. The sensitivity and specificity of resting ABI for the diagnosis of PAD have been reported to exceed 80%. Additionally, a post-exercise decrease in ankle pressure of >30 mm Hg or a reduction in ABI of >20% is considered diagnostic. Importantly, ABI also serves as a marker of systemic atherosclerosis and is predictive of cardiovascular events and functional impairment, even in asymptomatic individuals. Although additional imaging modalities are not required for the diagnosis of PAD, they play an important role in planning revascularization strategies. Invasive angiography remains the gold standard; however, it is typically reserved for patients with inconclusive noninvasive testing or for procedural planning. Alternative imaging techniques include duplex ultrasonography, computed tomography angiography (CTA), and magnetic resonance angiography (MRA). Each modality has specific advantages and limitations. Duplex ultrasonography is noninvasive and relatively inexpensive; however, it is operator-dependent, provides limited assessment of proximal arterial segments, and may be hindered by vascular calcification. Both CTA and MRA require contrast agents and may be contraindicated in patients with impaired renal function. MRA may also be limited by the presence of incompatible medical devices, a tendency to overestimate stenosis, and venous contamination. In contrast, CTA is affected by arterial calcification and demonstrates reduced accuracy in evaluating distal tibial and pedal vessels [23].

4. Risk factors

For a long time, peripheral artery disease (PAD) has been underappreciated compared with other manifestations of atherosclerosis, such as coronary artery disease and cerebrovascular disease. PAD is a major contributor to cardiovascular morbidity and mortality [24]. Importantly, many risk factors are shared among these disorders. Classical risk factors can be categorised as modifiable or non-modifiable. Typical modifiable risk factors include diabetes mellitus, elevated low-density lipoprotein (LDL) cholesterol, hypertension, and obesity; these

are the primary targets of both clinical management and preventive guidelines. The association between smoking and PAD is stronger than that reported for other cardiovascular diseases. Additionally, exposure to secondhand smoke has been identified as an independent risk factor for PAD. Non-modifiable risk factors, including advanced age, male sex, genetic predisposition, and a positive family history, also play a significant role [25].

Modifable risk factors	Non-modifable risk factors
Cigarette smoking	Older age
Diabetes mellitus	Male sex
Dyslipidemia	Genetic predisposition
Hypertension	Positive family history
Obesity	
Inadequate diet	
Lack of physical activity	
Long-term stress	
Sleep deprivation	

Table 3. Conventional risk factors of PAD [26].

An often underestimated aspect of PAD management is nutrition. Evidence suggests that dietary factors may reduce the risk of developing PAD, slow disease progression, and decrease the likelihood of complications. Dietary interventions should be personalized and implemented according to individual needs. Further research in this area is warranted to optimize PAD management [27]. Another critical component is physical activity. Recent studies indicate a steep reduction in PAD risk when moving from a state of physical inactivity to approximately 5000 MET-minutes per week, corresponding to roughly 13–14 hours of walking per week, which is associated with an approximate 30% reduction in risk [26]. Aerobic physical activity exerts multiple protective effects against PAD and other cardiovascular diseases. These include reduction of sympathetic nervous system activity and promotion of vagal modulation, which economize cardiac work and enhance nitric oxide-mediated endothelial function. Additionally, physical activity has anti-inflammatory effects that may inhibit the progression of atherosclerotic processes, although these mechanisms remain incompletely understood. The effects of resistance training are more complex. Resistance exercise may induce skeletal muscle hypertrophy, reduce oxidative stress and inflammation, and decrease both blood pressure and arterial stiffness. Epidemiological data suggest that the risk of PAD, particularly among male smokers, is inversely related to physical activity in early middle age, highlighting a potential protective role of exercise [28]. While lifestyle interventions remain challenging to implement, they are essential for effective management of atherosclerotic PAD [29,30].

5. Rare causes of lower extremities PAD

Atherosclerosis is the most common cause of peripheral artery disease (PAD). However, nonatherosclerotic peripheral artery disease (NAPAD) also warrants clinical attention. NAPAD

may present with symptoms similar to atherosclerotic PAD, including intermittent claudication, rest pain, and tissue loss. Nonatherosclerotic causes should be suspected in younger patients, in those without conventional atherosclerotic risk factors, or in cases with unusual lesion distributions. Below, we outline selected causes of NAPAD.

5.1 Vasculitis

Vasculitis comprises a heterogeneous group of disorders that can affect vessels of all sizes: large, medium, and small. Each type of vasculitis has characteristic clinical and pathological features; however, diagnosis is challenging and typically requires a multidisciplinary approach. Buerger's disease is an inflammatory disorder of small- and medium-sized arteries and veins, affecting all three vessel layers in a segmental pattern. It is strongly associated with tobacco use and predominantly affects young males [31]. Takayasu arteritis involves large and medium vessels, primarily the aorta, its major branches, and the pulmonary arteries. Arm claudication is the most common manifestation in young women, although lower limb symptoms may also occur. Giant cell arteritis shares pathologic similarities with Takayasu arteritis, including giant cell infiltration and granuloma formation, but typically affects the temporal artery and occurs in individuals over 50 years of age. Common symptoms include headache, jaw claudication, and visual impairment. Behçet's disease is a multisystem vasculitis of unclear etiology, causing perivascular inflammation and thrombus formation. It can result in aneurysmal dilation, stenosis, or occlusion in arteries and veins of any size [32].

5.2 Fibromuscular Dysplasia

Fibromuscular dysplasia (FMD) is a non-inflammatory, nonatherosclerotic vascular disorder that can affect any artery, although it predominantly involves the renal and carotid arteries. FMD is typically diagnosed in women aged 20–60 years. Pathologically, FMD is classified as intimal, medial, or perimedial. The characteristic angiographic appearance is described as a "string-of-beads." FMD commonly presents as renovascular hypertension but may also cause cerebrovascular events in young adults. Less commonly, FMD affects lower extremity arteries, including the iliac, femoral, and popliteal arteries [33].

5.3 Adventitial Cystic Disease

Cystic adventitial disease (CAD) is characterised by the accumulation of mucinous material within the adventitial layer of arteries and veins. The popliteal artery is most frequently affected, although the femoral artery may also be involved. Cysts may cause vessel narrowing and subsequent ischemic symptoms. CAD predominantly affects middle-aged men. Angiographically, affected vessels exhibit a smooth, eccentric, and extrinsically narrowed appearance [34].

5.4 Endofibrosis

Endofibrosis is a rare condition primarily affecting young athletes, including cyclists, runners, triathletes, and skaters. It is caused by repetitive trauma to the external iliac artery, often due to repeated hip hyperflexion and hypertrophy of the psoas muscle, which compresses the artery. Pathological findings include intimal thickening and arterial narrowing due to collagen deposition, fibrous tissue, and smooth muscle proliferation. Endofibrosis is progressive and may cause exercise-induced pain, weakness, or numbness [35].

5.5 Popliteal Artery Entrapment Syndrome

Popliteal artery entrapment syndrome (PAES) is defined as compression of the popliteal artery by aberrant myotendinous structures within the popliteal fossa. PAES primarily affects young athletic males but can occur at any age. Six types of entrapment are described, broadly categorized as anatomic/congenital or functional. Clinical manifestations mimic PAD, including intermittent claudication and calf or foot pain during exertion [36].

5.6 Vasospasm

Lower extremity vasospasm is uncommon compared with cerebral or coronary vasospasm. Nonetheless, cases have been reported, often induced by agents such as ergotamine, cocaine, marijuana, amphetamines, or other drugs. Drug-induced vasospasm is typically bilateral, symmetric, and manifests as abrupt narrowing of arterial segments in the lower limbs [37].

6. Conservative treatment

Antiplatelet, antithrombotic, and lipid-lowering therapies constitute core components of medical management for patients with peripheral artery disease (PAD). The consequences of PAD can be debilitating, significantly reducing quality of life. Pharmacological treatment should aim to alleviate symptoms, such as intermittent claudication, and prevent the development of skin lesions. However, the primary goal of medical therapy is to reduce the risk of disease progression, particularly to limb-threatening ischemia. Another important objective is the reduction of major adverse limb events (MALE) and major adverse cardiovascular events (MACE). Conservative management should also include strict smoking cessation, adoption of a healthy diet, and reduction of sedentary behavior. Pharmacotherapy is recommended to support smoking cessation in patients with PAD. Blood pressure in hypertensive PAD patients should be controlled to <130/80 mmHg. There is no pharmacological agent specifically indicated solely for PAD. Glycemic control is important to reduce the risk of microvascular complications and cardiovascular events. Sodium-glucose cotransporter-2 (SGLT2) inhibitors and glucagon-like peptide-1 (GLP-1) receptor agonists have demonstrated beneficial cardiovascular effects, including in patients with PAD [38].

6.1 Lipid-lowering therapy

Dyslipidemia is a major risk factor for the development, progression, and prognosis of PAD, making it a key therapeutic target. Current guidelines recommend statin therapy for all patients with PAD, with the goal of achieving a ≥50% reduction in low-density lipoprotein cholesterol (LDL-C). Statin therapy has been shown to improve outcomes, including reductions in MACE and MALE. Despite strong evidence of safety and efficacy from large cohort studies, statins remain underprescribed in PAD populations, partly due to concerns regarding muscle-related adverse effects. If LDL-C remains ≥70 mg/dL despite maximally tolerated statin therapy, addition of a PCSK9 inhibitor is recommended. In cases where LDL-C remains ≥70 mg/dL despite combined statin and PCSK9 inhibition, ezetimibe may be added as a further step [39].

6.2 Antiplatelet and antithrombotic therapy

Platelets play a central role in atherosclerotic disease. Platelet adhesion initiates fibrofatty plaque formation, while platelet hyperaggregability and increased adhesion contribute significantly to PAD pathogenesis [40]. Accordingly, antiplatelet therapy is a cornerstone of

PAD management. Randomized clinical trials have demonstrated that clopidogrel monotherapy is more effective than aspirin in reducing cardiovascular events. More recent studies indicate that dual therapy with low-dose rivaroxaban (2.5 mg twice daily) combined with aspirin significantly reduces cardiovascular events, all-cause mortality, and MALE in PAD patients [38]. Consequently, aspirin in combination with rivaroxaban is now preferred in patients without contraindications. Therapy must be administered cautiously due to an increased risk of bleeding. Full-intensity oral anticoagulation should only be used when clinically indicated, such as in patients with atrial fibrillation.

6.3 Peripheral vasodilators

Intermittent claudication is a major contributor to functional impairment and reduced quality of life in PAD. Patients with claudication have a three- to six-fold increase in cardiovascular mortality. Cilostazol, a selective phosphodiesterase III inhibitor, is approved for improving walking distance in PAD patients. However, the benefits of cilostazol are modest, and evidence for its impact on cardiovascular events is limited. Common adverse effects include headache, palpitations, and diarrhea. Cilostazol is contraindicated in patients with heart failure [41]. In comparison, exercise therapy has been shown to produce significantly superior outcomes in terms of walking performance and overall functional status.

7. Exercise therapy

Supervised exercise training (SET) is recommended as first-line therapy for patients with chronic, symptomatic peripheral artery disease (PAD). The primary aims of this intervention are to improve functional status, walking performance, and quality of life. The underlying mechanisms are complex and multifactorial. Exercise training promotes the formation of collateral blood vessels and increases blood flow through angiogenesis; however, this is not the primary mechanism. Improvements in walking ability are also mediated by enhanced vascular endothelial function, increased muscle strength and endurance, and reduction of systemic inflammation. The precise mechanisms by which SET improves walking performance require further investigation [42]. Two structured types of exercise therapy are recognized for PAD: supervised exercise therapy and structured community-based exercise programs, including home-based training (HBT) [13]. Supervised exercise therapy (SET) involves intermittent walking on a treadmill, interspersed with rest periods when pain becomes moderate or severe. Sessions are conducted in a hospital or outpatient facility for 30–45 minutes of active exercise per 60-minute session, at least three times per week for a minimum of 12 weeks. The goal of SET is to progress to 30–45 minutes of continuous walking per session. Structured community-based exercise programs, including HBT, are self-directed but guided by qualified healthcare professionals and also focus on walking. These programs are performed in the patient's home environment and require long-term adherence. Patients must be educated on proper technique and follow recommendations strictly. Compared with SET, HBT is generally less effective. Self-monitoring using tools such as wrist-worn activity trackers or smartphone accelerometer applications has been reported to improve adherence [43]. Patients with chronic symptomatic PAD may also benefit from alternative non-walking SET modalities, such as recumbent stepping, although further research is required in this area. In conclusion, exercise therapy remains a cornerstone of PAD management. It not only improves walking ability, quality of life, and functional status, but also mitigates the psychological consequences of limited mobility. Symptomatic PAD significantly restricts physical activity, increasing the risk of depressive symptoms and chronic stress. Therefore, promoting increased physical activity represents a critical lifestyle intervention for these patients [44].

8. Surgical treatment

Surgical treatment is a rapidly evolving field. Multiple techniques, including endovascular and open surgical approaches, are available for the management of lower extremity peripheral artery disease (PAD). An individualized approach requires consideration of several factors, including symptom severity, risk of limb threat, patient comorbidities, and the anatomic distribution of the disease [45]. The urgency of revascularization varies depending on clinical presentation, ranging from patients with mild symptoms to those with acute limb ischemia. Technological innovations, such as advanced guidewires, catheters, drug-eluting devices, specialized balloons, and biomimetic stents, are continuously being developed. Each modality has specific indications, advantages, and limitations. Rapid advances in revascularization techniques allow for more precise tailoring of interventions to the individual patient [46]. However, surgical treatment is associated with potential challenges and adverse outcomes, including restenosis, vascular calcification, microvascular disease, and silent embolization.

9. Conclusion

Atherosclerotic occlusive disease of the lower extremities is a chronic and progressive condition. Although the risk factors for peripheral artery disease (PAD) are well established, preventing disease progression and optimizing management remain challenging. Implementation of effective treatment strategies for PAD faces multiple obstacles, including difficulties with smoking cessation, limited efficacy of peripheral vasodilators, and technical challenges associated with surgical interventions. Furthermore, lifestyle modifications are demanding for a substantial proportion of patients. Walking exercise remains a cornerstone of PAD management. This intervention is accessible, cost-effective, and can be performed in the patient's personal environment. However, patient adherence is crucial for achieving satisfactory outcomes. Without compliance, disease progression may continue, necessitating more invasive interventions, including surgical procedures or, in severe cases, amputation. Patients can significantly improve their prognosis by adopting healthy lifestyle modifications, such as regular physical activity, smoking cessation, a balanced diet, and weight reduction.

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