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NARRATIVE REVIEW

Impact of Popliteal Artery Entrapment Syndrome on Exercise Tolerance and Athletic Performance: A Narrative Review

a narrative review

HIGHLIGHTS

- Popliteal artery entrapment syndrome (PAES) is a rare but clinically important vascular cause of exercise-induced lower-limb ischemia in young, physically active athletes.

- Structural and functional variants both produce intermittent claudication, calf cramping, paresthesia, and impaired endurance — symptoms easily confused with musculoskeletal disorders.
- Static, resting imaging is frequently non-diagnostic; dynamic duplex ultrasound, provocative CTA/MRA, and post-exercise ABI are essential for confirming the diagnosis.
- Surgical decompression — release of the medial gastrocnemius or vascular reconstruction when needed — is the cornerstone of management and supports high return-to-sport rates.
- Frequent coexistence of PAES with chronic exertional compartment syndrome (CECS) mandates a combined vascular and musculoskeletal evaluation to optimise outcomes in athletes.

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ABSTRACT

BACKGROUND: Popliteal artery entrapment syndrome (PAES) is a rare vascular disorder observed mainly in young, physically active individuals. It involves compression of the popliteal artery, leading to exercise-induced ischemia and reduced athletic performance. The condition is frequently underdiagnosed due to symptom overlap with musculoskeletal disorders.

AIM: This narrative review aims to summarise current evidence on the impact of PAES on exercise tolerance and athletic performance, covering pathophysiology, clinical presentation, diagnostic approaches, and management strategies relevant to athletic populations.

MATERIALS AND METHODS: A non-systematic search of PubMed, Scopus, and Web of Science was performed for studies published up to 2026. The analysis focused on PAES in athletic populations, covering pathophysiology, clinical presentation, diagnosis, and management.

RESULTS: PAES may be structural or functional, both resulting in impaired blood flow during exercise. Common symptoms include intermittent claudication, muscle fatigue, and paresthesia, which negatively impact exercise tolerance. Diagnosis requires dynamic imaging techniques, as resting examinations are often inconclusive. Surgical treatment provides significant improvement in most symptomatic athletes.

CONCLUSIONS: PAES is an important but often overlooked cause of exercise intolerance in athletes. Early diagnosis and appropriate management are essential to restore performance and prevent long-term complications.

KEYWORDS popliteal artery entrapment syndrome; athletes; exercise tolerance; athletic performance; intermittent claudication; chronic exertional compartment syndrome; dynamic imaging; surgical management.

PLAIN LANGUAGE SUMMARY

Popliteal artery entrapment syndrome (PAES) is an uncommon problem that can affect young, active people — especially runners, cyclists, and other athletes. In PAES the main artery behind the knee gets squeezed by surrounding muscles when the leg is in certain positions, so the calf does not receive enough blood during exercise. The typical signs are calf pain, cramping, tingling or numbness, and reduced stamina that appear with effort and quickly disappear at rest. Because these symptoms look very similar to ordinary muscle injuries or compartment problems, the diagnosis is often delayed. A regular scan taken while the leg is relaxed may look completely normal — so doctors must use dynamic tests such as ultrasound during plantarflexion, exercise ankle–brachial index, or CT/MRI imaging with the foot held in provocative positions. Once recognised, PAES is usually treated with surgery to release the artery, and in selected cases to repair it. Most athletes recover well, especially when the syndrome is diagnosed early and rehabilitation is sport-specific. Better awareness of PAES among sports physicians, vascular specialists, and athletes themselves is key to preventing long-term vascular damage and to keeping athletes performing at their best.

GRAPHICAL ABSTRACT

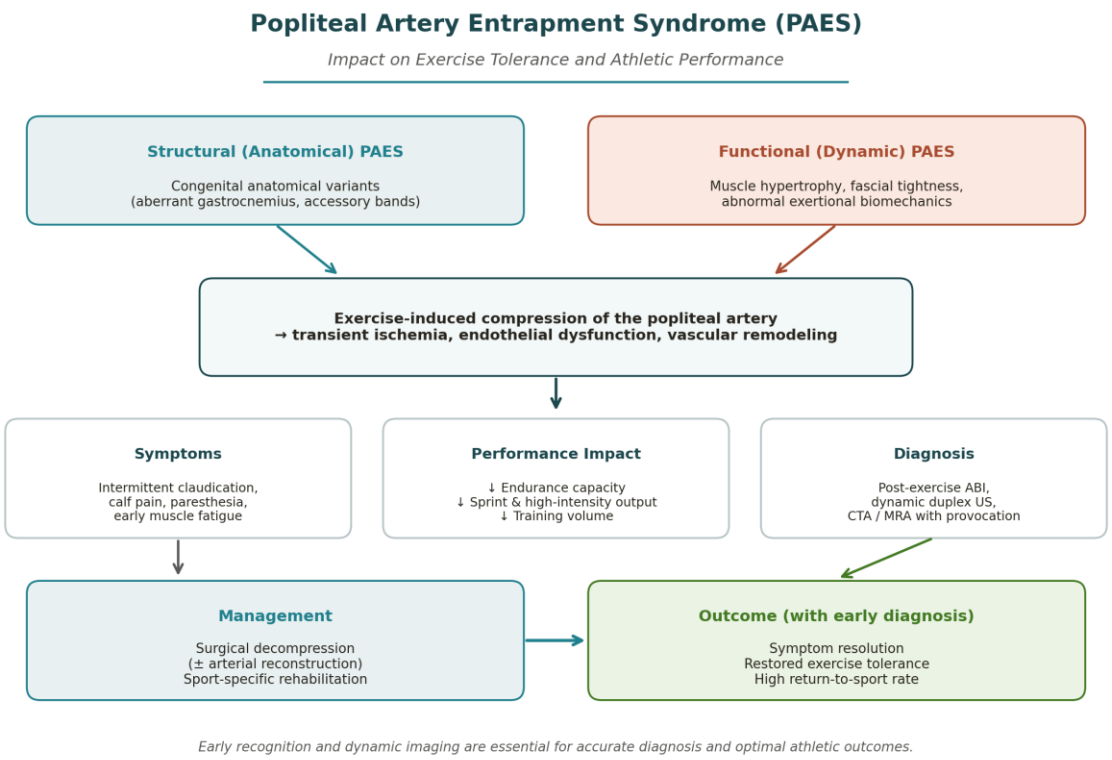

Figure 1. Graphical abstract summarising the etiology, mechanism, clinical impact, diagnosis, and management of popliteal artery entrapment syndrome (PAES) in athletes.

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1. INTRODUCTION

Popliteal artery entrapment syndrome (PAES) is a rare but clinically significant vascular disorder, characterised by extrinsic compression of the popliteal artery, leading to exercise-induced lower limb ischemia [1,2]. Initially described in young, physically active individuals, the condition is now more frequently identified among athletes, where repetitive knee flexion and plantarflexion may exacerbate arterial compression and precipitate symptomatic claudication [3,4]. Functional PAES is often missed because its symptoms mimic those of musculoskeletal disorders, contributing to delayed diagnosis [5,6].

The significance of PAES in athletes extends beyond intermittent claudication. Activity-related ischemia may cause transient paresthesia, fatigue, and reduced training or competition capacity [7]. Functional forms of PAES are particularly challenging, as standard imaging at rest may fail to identify dynamic arterial compression, delaying diagnosis and treatment [6,8]. Undiagnosed or mismanaged PAES may lead to vascular remodelling and persistent performance deficits. In rare cases, thromboembolic complications can occur [9].

Given these considerations, a focused review of PAES in athletic populations is warranted to clarify its impact on exercise tolerance and performance. This narrative review aims to:

- Summarise the epidemiology and significance of PAES in athletes [1,3].
- Elucidate the pathophysiological mechanisms underlying exertional symptoms [2,5].
- Provide an integrated framework for diagnostic evaluation and management strategies that optimise both vascular and functional outcomes [6,9].

2. METHODS

This study was conducted as a narrative review aimed at synthesising current evidence on popliteal artery entrapment syndrome (PAES) and its impact on exercise tolerance and athletic performance. A non-systematic literature search was performed using major electronic databases, including PubMed, Scopus, and Web of Science, to identify relevant studies published up to 2026. The search strategy combined keywords and Medical Subject Headings (MeSH) such as "popliteal artery entrapment syndrome," "PAES," "athletes," "exercise tolerance," "intermittent claudication," and "chronic exertional compartment syndrome."

Eligible sources included original research articles, clinical studies, narrative and systematic reviews, and case series focusing on PAES in physically active or athletic populations. Particular emphasis was placed on studies addressing pathophysiology, clinical presentation, diagnostic strategies, and the effects of PAES on functional capacity and sports performance.

Studies were included if they involved patients diagnosed with PAES, encompassing both anatomical and functional forms, and if they focused on athletic or physically active populations. In addition, only articles that addressed key aspects relevant to this review — namely pathophysiology, clinical features, diagnostic approaches, or the impact of PAES on exercise tolerance and performance — were considered. All included studies were required to be available in English and accessible in full-text form.

Studies were excluded if they were not directly related to PAES or did not concern lower-limb vascular compression syndromes. Articles focusing exclusively on non-athletic or predominantly atherosclerotic populations, without relevance to exercise-induced symptoms, were also excluded.

3. PATHOPHYSIOLOGY OF PAES

PAES is classified into structural and functional types, each with distinct pathophysiological features. Structural PAES arises from congenital anatomical variants, including aberrant medial or lateral heads of the gastrocnemius, accessory muscle slips, or anomalous popliteal artery origin, which may compress the vessel during knee flexion or plantarflexion [1,2]. In contrast, functional PAES occurs without anatomical anomalies and is often linked to muscle hypertrophy, fascial tightness, or abnormal biomechanics during exertion, particularly in athletes performing repetitive high-intensity activities [5,6,10]. It is particularly prevalent in athletes performing repetitive sprinting or jumping, where dynamic muscle compression causes transient ischemia [10]. Functional forms are often underrecognised in athletes, as static imaging may appear normal [8].

3.1. Mechanisms of arterial compression and ischemia

Repetitive lower limb activity transiently compresses the popliteal artery, resulting in ischemia, paresthesia, and exercise intolerance [2,3]. Prolonged compression may cause endothelial injury and intimal hyperplasia. It may also lead to post-stenotic dilation, increasing the risk of thromboembolic or aneurysmal complications [2,11]. These hemodynamic changes further promote oxidative stress, vascular remodelling, and impaired microcirculation [11]. Mechanical stress may also trigger local inflammation and oxidative stress, contributing to progressive vascular remodelling [10,12].

3.2. Vascular remodelling and endothelial changes

Repeated blood flow restriction leads to morphological alterations in the popliteal artery, including medial fibrosis, intimal thickening, and collateral vessel formation [2,11]. Endothelial dysfunction diminishes vasodilatory reserve, exacerbating exercise limitations [7]. Although collateralisation may partially compensate for intermittent obstruction, it does not fully restore perfusion during high-intensity activity [10,11].

3.3. Coexistence with other exercise-induced syndromes

This syndrome frequently occurs alongside chronic exertional compartment syndrome (CECS), complicating diagnosis and management due to overlapping symptoms of pain, cramping, and reduced performance [6,13]. Unrecognised concurrent CECS can lead to persistent functional limitations even after surgical decompression for PAES, emphasising the need for comprehensive evaluation in athletes presenting with exertional leg pain [13,14].

In summary, the pathophysiology of PAES is multifactorial, integrating anatomical variation, intermittent vascular obstruction, ischemia-induced vascular remodelling, and neuromuscular interactions. A thorough understanding of these mechanisms is essential for accurate diagnosis, differentiation from musculoskeletal syndromes, and optimisation of interventions aimed at restoring exercise tolerance and athletic performance [7,10].

PAES type	Mechanism	Main symptoms	Diagnostic tools	Key references
Structural	Congenital anomalies of the medial head of gastrocnemius or popliteal artery, accessory bands, or abnormal course of vessels	Constant or exercise-induced claudication, calf pain, numbness	CTA, MRA, DSA	Stager & Clement 1999 [1]; Levien 2003 [2]; Darling 1974 [3]; Sinha 2012 [5]
Functional	Compression due to hypertrophied or abnormally contracting muscles without anatomical vessel anomaly	Symptoms only during exertion, intermittent, variable intensity	Dynamic duplex US, CTA/MRA in provocative position, exercise ABI	Turnipseed 2009 [6]; Hislop 2014 [7]; Morgan 2023 [8]; Ghaffarian 2023 [9]; Pandya 2019 [10]
Mixed / Combined	Both congenital anatomical variant and functional muscle hypertrophy contributing to intermittent compression	Exercise-induced leg pain, sometimes persistent; possible concurrent CECS	Combination of imaging modalities + functional tests	Lawley 2022 [13]; Bellomo 2024 [14]; Miller 2021 [15]

Table 1. Summary of PAES types, their pathophysiological mechanisms, main clinical symptoms, diagnostic tools, and key references.

4. CLINICAL PRESENTATION

The syndrome manifests with a heterogeneous and often activity-dependent clinical picture, primarily affecting young, otherwise healthy individuals, particularly athletes engaged in repetitive lower limb exertion [1,3]. The underlying pathophysiology reflects dynamic arterial compression, which may progress from reversible functional impairment to fixed vascular pathology if untreated [15]. Prompt identification is crucial to avoid chronic ischemic damage and long-term vascular sequelae [12,16].

4.1. Symptom spectrum

The hallmark manifestation of PAES is intermittent claudication, typically localised to the calf and reproducibly triggered by exercise, with rapid resolution at rest [3,5]. Unlike atherosclerotic disease, these symptoms occur in younger populations without conventional cardiovascular risk factors [15,17]. Patients commonly describe exertional cramping, posterior compartment tightness, or a sensation of acute distension during activity [5,16].

Muscle fatigue is another common feature, reflecting compromised perfusion during sustained activity, which may significantly impair athletic performance and endurance capacity [17]. Additionally, paresthesia, including numbness or tingling in the foot or calf, may occur due to ischemia-induced neural involvement or concomitant compression of adjacent neurovascular structures [6,15]. In advanced or chronic cases, symptoms can progress to rest pain or critical limb ischemia, although this remains relatively uncommon in athletic populations [18,19].

4.2. Anatomical vs functional PAES

A critical distinction exists between anatomical (structural) and functional (dynamic) forms of PAES, which has direct implications for clinical presentation and diagnosis [5,7].

Anatomical PAES results from congenital aberrations in the relationship between the popliteal artery and surrounding musculotendinous structures, most commonly involving the medial head of the gastrocnemius muscle. This leads to fixed or reproducible arterial compression, which can progress to intimal damage, stenosis, or thrombosis over time [5,20]. Consequently, symptoms may become more persistent and less strictly exercise-dependent as the disease advances [20].

Dynamic arterial obstruction in functional PAES arises without anatomical defects and is often secondary to overdeveloped or overused surrounding musculature [6,7]. Vascular obstruction occurs solely during particular limb movements or muscle contractions. Patients often present with subtle, intermittent symptoms, frequently leading to delayed or missed diagnosis [7,10]. Functional PAES may also coexist with chronic exertional compartment syndrome, complicating diagnosis [13,14].

4.3. Activity- and position-dependent manifestations

Symptoms in PAES are highly activity- and position-dependent. Clinical manifestations are typically provoked by exercise involving repetitive plantarflexion, such as running or cycling [17]. Provocative limb positions, including forced plantarflexion or dorsiflexion, can exacerbate arterial compression [8,9].

Resting examination may appear unremarkable, whereas provocative manoeuvres can uncover attenuated distal pulses [21,22]. This dynamic variability underscores the importance of functional imaging to reproduce symptomatic conditions and confirm the diagnosis [9,23].

4.4. Differential diagnosis

Diagnosing PAES is challenging due to symptom overlap with other exercise-induced leg pathologies, particularly in athletes [24].

Chronic exertional compartment syndrome (CECS) is an important differential, presenting with exercise-induced pain, tightness, and occasionally paresthesia [14,25]. CECS results from increased intracompartmental pressure rather than vascular compromise, and symptoms usually resolve gradually after activity cessation [25]. Notably, PAES and CECS may coexist, necessitating careful evaluation [13,14].

Other musculoskeletal disorders — including muscle strains, tendinopathies, or stress injuries — must also be considered. These conditions generally present with more localised pain, are less reproducible with vascular manoeuvres, and lack characteristic pulse changes seen in PAES [15,24].

Given these overlaps, a high index of suspicion and a structured diagnostic approach are essential to differentiate PAES from these entities and to avoid misdiagnosis or inappropriate management [12,26].

5. DIAGNOSTIC APPROACHES

The diagnostic work-up of PAES requires a structured and multimodal approach, particularly in young, physically active individuals presenting with exertional lower limb symptoms [3,4]. A detailed clinical history is essential and should include the onset, reproducibility, and intensity of symptoms such as calf pain, tightness, paresthesia, and premature fatigue, typically resolving rapidly with rest [1,4]. Physical examination should incorporate vascular

assessment at rest and during provocative manoeuvres. Active plantarflexion and dorsiflexion may reproduce symptoms and lead to attenuation or disappearance of distal pulses, reflecting dynamic arterial compression [1,27].

Hemodynamic testing represents the first-line diagnostic step. Resting ankle–brachial index (ABI) is frequently within normal limits; however, post-exercise or provoked ABI measurements often demonstrate a significant decrease, indicating flow limitation [5,6]. Segmental pressure measurements and pulse volume recordings may further localise the level of arterial compromise and reveal characteristic waveform changes associated with dynamic stenosis [8,22].

Duplex ultrasonography is a cornerstone modality due to its ability to provide both anatomical and functional information. The examination should be performed under resting and dynamic conditions, including resisted plantarflexion and dorsiflexion. Findings may include increased peak systolic velocity, turbulence, or complete cessation of flow during provocation [21,28]. Duplex imaging also allows assessment of the spatial relationship between the artery and surrounding musculotendinous structures. This is particularly important for distinguishing anatomical from functional variants [29]. The implementation of dynamic protocols significantly enhances diagnostic sensitivity, especially in functional PAES where resting studies may be non-diagnostic [8,21].

Cross-sectional imaging techniques provide complementary structural detail. Computed tomography angiography (CTA) enables high-resolution visualisation of arterial morphology, including focal stenosis, post-stenotic dilatation, mural thickening, or thrombotic changes, as well as identification of congenital anatomical anomalies [22,24]. Magnetic resonance angiography (MRA) offers superior soft tissue contrast, facilitating detailed evaluation of adjacent muscles, fascia, and neurovascular relationships [22]. Importantly, both CTA and MRA should be performed with provocative positioning to demonstrate positional arterial compression or occlusion, which may not be apparent in neutral positioning [9,22].

Dynamic imaging modalities are now considered the diagnostic gold standard for functional PAES, enabling real-time evaluation of transient arterial compromise and enhancing identification of exercise-induced occlusion [9]. In selected or inconclusive cases, digital subtraction angiography (DSA) may be employed, particularly when surgical intervention is being considered. This modality can demonstrate characteristic positional narrowing, delayed distal filling, and development of collateral circulation [30,31].

Given the high rate of symptom overlap, differential diagnosis should systematically include chronic exertional compartment syndrome (CECS). Intracompartmental pressure measurements before and after exercise are recommended in patients with persistent or atypical symptoms, allowing identification of coexisting pathologies that may influence treatment outcomes [13,14]. Increasing evidence supports a combined vascular and musculoskeletal diagnostic strategy in athletes to reduce misdiagnosis and ensure comprehensive evaluation [6,7].

6. IMPACT ON EXERCISE TOLERANCE AND ATHLETIC PERFORMANCE

PAES can substantially impair exercise tolerance and athletic performance, primarily through mechanisms related to exercise-induced ischemia and muscle fatigue. During physical activity, dynamic compression of the popliteal artery reduces blood flow to the distal limb, particularly affecting the gastrocnemius and soleus muscles, which leads to transient ischemia and early onset of pain, cramping, and fatigue [1,32]. In functional PAES, arterial compression may occur without structural anomalies, yet still elicits significant ischemic symptoms during exertion [6,7]. The severity of ischemia varies depending on limb position, contraction intensity, and vascular anatomy. This leads to inconsistent symptom presentation and may complicate early diagnosis [5,24].

Those affected frequently exhibit reduced capacity in endurance, sprinting, and high-intensity activities. Endurance activities, such as long-distance running and cycling, are particularly affected due to sustained demand for lower-limb perfusion, often resulting in exercise-induced claudication and reduced training capacity [4,27]. Sprinting, plyometric movements, and repeated high-intensity efforts may be compromised because rapid muscle contraction exacerbates arterial compression and reduces oxygen delivery, causing premature fatigue and decreased anaerobic performance. High-intensity intermittent activity can further worsen symptoms, limiting an athlete's ability to maintain competitive performance over consecutive sessions or matches [8,29]. Notably, dynamic imaging and functional testing often reveal perfusion deficits that correlate with symptom severity, highlighting the direct impact of vascular compromise on exercise capacity [9,23].

Evidence supporting these limitations comes from a range of sources, including case reports, cohort studies, and systematic athlete assessments. Early descriptions by Darling et al. [3] and Rudo et al. [27] documented intermittent claudication in young athletes, with pain resolving at rest. Subsequent investigations using Doppler ultrasonography, CT angiography, and MR angiography have confirmed transient ischemia during exertion, even in patients without structural arterial anomalies [21]. Longitudinal studies of surgically treated athletes demonstrate substantial improvements in both endurance and sprint performance after decompression, confirming the causal link between ischemia and impaired exercise capacity [31,33].

Associated syndromes may amplify exercise limitations. Co-occurring CECS frequently coexists with functional PAES, producing overlapping leg pain and further restricting high-intensity activity [13,14]. Musculoskeletal overuse injuries, such as tendinopathies or biomechanical anomalies of the lower limb, may also compound exercise intolerance by increasing muscular fatigue and altering gait patterns [4,25]. Recognition of these coexisting pathologies is critical for accurate diagnosis, comprehensive management, and optimising athletic performance outcomes [10,12].

7. MANAGEMENT AND THERAPEUTIC APPROACHES

Management of PAES requires an individualised and multidisciplinary approach. It should consider symptom severity, underlying etiology (anatomical vs functional), and the athlete's demands. Initial management in suspected functional PAES may include conservative strategies such as activity modification, targeted physiotherapy, and biomechanical optimisation aimed at reducing excessive muscular compression during dynamic movement; however, evidence suggests that non-operative treatment is often insufficient in athletes with persistent symptoms or significant performance limitation [6,7]. In such cases, definitive treatment typically involves surgical intervention, particularly when dynamic imaging confirms hemodynamically significant arterial compression or when structural abnormalities are present [5,26].

Surgical decompression remains the cornerstone of therapy and involves the release of the popliteal artery from compressive musculotendinous structures, most commonly the medial head of the gastrocnemius, with or without arterial reconstruction depending on the extent of vascular damage [5,34]. In patients with advanced disease, additional procedures such as endarterectomy, patch angioplasty, or bypass grafting may be required to restore adequate blood flow and prevent long-term complications [2,5]. Outcomes following surgery are generally favourable, particularly in young athletic populations, with significant improvements in symptom resolution, exercise tolerance, and return-to-sport rates reported across multiple studies [31,33,35]. Early intervention is associated with better vascular and functional outcomes, whereas delayed treatment may result in irreversible arterial changes and suboptimal recovery [36].

In functional PAES, surgical strategies may also include myotomy or debulking of hypertrophied musculature to eliminate dynamic compression, even in the absence of clear anatomical anomalies [6,33]. Postoperative rehabilitation plays a critical role in optimising recovery, focusing on gradual return to activity, neuromuscular control, and correction of movement patterns that may have contributed to symptom development [26]. Importantly, coexisting conditions such as CECS must be identified and managed concurrently, as failure to address overlapping pathologies can lead to persistent symptoms despite technically successful vascular intervention [13,14].

Emerging approaches emphasise the importance of early diagnosis, dynamic imaging-guided treatment planning, and sport-specific rehabilitation protocols to maximise outcomes in athletes [12]. Long-term follow-up is recommended to monitor for recurrence, residual functional limitations, or late vascular complications, particularly in high-performance individuals exposed to repetitive lower limb stress [31,35]. Overall, effective management of PAES requires not only restoration of arterial patency but also a comprehensive strategy aimed at returning athletes to pre-symptom performance levels while minimising the risk of recurrence.

Improvement in athletic performance following treatment of PAES represents one of the most clinically significant outcomes, particularly in physically active populations. Successful surgical decompression restores adequate blood flow to the calf musculature during exercise, which directly enhances aerobic capacity and delays the onset of fatigue. In many patients, this leads to complete resolution of intermittent claudication and a marked increase in exercise tolerance, allowing a return to training at intensities comparable to pre-symptom levels [31,33,35]. Importantly, these improvements are observed in both endurance-based activities, such as long-distance running, and high-intensity or intermittent efforts, where oxygen delivery had previously been compromised.

Observational studies indicate that the majority of athletes are able to return to sport — often at a competitive level — within several months following surgical intervention. This recovery is not only attributable to the removal of mechanical vascular compression but also to the reversal of secondary metabolic disturbances within the affected musculature, including impaired oxygen utilisation and accumulation of fatigue-related metabolites [4,33]. Restoration of adequate perfusion supports normalisation of mitochondrial function and improves tolerance to high-intensity exercise [11,12].

An additional factor contributing to improved performance is the correction of compensatory movement patterns that develop in response to chronic ischemia and pain. With appropriate postoperative rehabilitation, athletes demonstrate improvements in neuromuscular control, movement efficiency, and muscle strength, further enhancing functional capacity [4,26]. In some cases, performance may even exceed pre-diagnosis levels, particularly when previously unrecognised PAES had chronically limited training potential [31].

However, the degree of recovery is closely related to the duration of symptoms prior to treatment. Early intervention is associated with near-complete restoration of function, whereas delayed diagnosis may result in persistent limitations due to irreversible vascular or muscular adaptations [5,36]. Furthermore, coexisting conditions such as CECS may negatively influence outcomes if not simultaneously addressed [13,14].

Overall, treatment of PAES — especially surgical management — results in substantial and multidimensional improvements in exercise capacity, encompassing both physiological parameters (e.g., perfusion and endurance) and functional performance (e.g., strength, coordination, and fatigue resistance). Better outcomes are associated with early diagnosis and sport-specific rehabilitation.

8. DISCUSSION

Category	Key aspects	Implications for athletes
Epidemiology	Rare vascular disorder in young, physically active individuals [1,12]	Frequently underdiagnosed; should be considered in exercise-induced leg pain
Etiology and types	Anatomical: congenital anomalies [2]; Functional: dynamic compression without structural changes [6,7]	Functional PAES more common in athletes; often missed
Pathophysiology	Repetitive arterial compression leading to ischemia and vascular remodelling [5,21,26]	Progressive impairment of muscle perfusion and oxygen delivery
Clinical presentation	Intermittent claudication, calf pain, tightness, fatigue, paresthesia [3,17]	Reduced exercise tolerance and training capacity
Activity dependence	Symptoms during running, sprinting, cycling; worsened by plantarflexion [4,14]	Reduced endurance and high-intensity performance
Differential diagnosis	Chronic exertional compartment syndrome (CECS) and musculoskeletal disorders [13,25]	High risk of misdiagnosis; coexistence may worsen outcomes
Diagnostic approach	Post-exercise ABI, duplex ultrasound, CTA/MRA with provocation [8,9]	Dynamic testing essential, especially in functional PAES
Impact on performance	Decreased endurance, early fatigue, impaired anaerobic capacity [4,27]	Reduced training volume and competitive performance
Management	Conservative (limited), surgical decompression (standard) [16]	Surgery enables return to sport and symptom resolution
Outcomes and prognosis	High return-to-sport rates; better outcomes with early treatment [31,33,35]	Delayed diagnosis may lead to persistent functional deficits

Table 2. Key aspects of popliteal artery entrapment syndrome (PAES) in athletes, including epidemiology, pathophysiology, clinical presentation, diagnostic approach, and management.

PAES remains a diagnostically challenging and often underrecognised condition in athletic populations, largely due to its dynamic nature and symptom overlap with more prevalent musculoskeletal disorders. This review highlights that the true clinical burden of PAES is likely underestimated, particularly in its functional form, where standard resting investigations may fail to demonstrate arterial compromise [5,6,8]. The findings synthesised in this narrative analysis emphasise that PAES should be systematically considered in the differential diagnosis of exercise-induced leg pain in young, otherwise healthy athletes, especially when symptoms are reproducible and resolve rapidly with rest [3,4].

Exercise intolerance in PAES arises from multiple mechanisms, encompassing transient ischemia, vascular remodelling, and adaptive neuromuscular changes. Repeated arterial compression contributes to endothelial dysfunction, impaired vasodilatory capacity, and reduced oxygen delivery during exertion, which collectively diminish exercise tolerance and athletic performance [2,5,29]. Importantly, even in functional PAES, where no fixed anatomical abnormality is present, dynamic compression can produce clinically significant hemodynamic consequences, reinforcing the need for dynamic and provocative diagnostic protocols [6,8,9].

Another critical aspect is the frequent coexistence of PAES with chronic exertional compartment syndrome (CECS), which complicates both diagnosis and management. Overlapping symptom profiles may lead to misdiagnosis or incomplete treatment, particularly when only one condition is addressed [13,14]. The literature consistently supports a comprehensive diagnostic approach that integrates vascular and musculoskeletal assessment, as failure to recognise concurrent pathologies may result in persistent symptoms and suboptimal functional outcomes [6,7].

From a therapeutic perspective, the evidence indicates that surgical decompression is highly effective in appropriately selected patients, particularly athletes with significant functional limitation. Restoration of arterial patency leads not only to symptom resolution but also to meaningful improvements in exercise capacity and return-to-sport rates [31,33,35]. However, the timing of intervention appears to be a critical determinant of outcome. Delayed diagnosis may allow for the development of irreversible vascular changes, thereby limiting the potential for full recovery [5,36]. These observations underscore the importance of early recognition and timely referral to specialised care.

Despite growing awareness of PAES, several gaps remain in the current evidence base. Most available data derive from case series and observational studies, with limited high-quality prospective research focusing specifically on athletic populations. Additionally, there is variability in diagnostic protocols, surgical techniques, and rehabilitation strategies, which complicates direct comparison of outcomes across studies. Future research should aim to standardise diagnostic criteria, evaluate long-term functional outcomes, and define optimal return-to-sport protocols tailored to different athletic disciplines.

9. LIMITATIONS

This narrative review has several limitations. First, the available literature on PAES in athletic populations is limited and largely based on case reports and small observational studies, which reduces the strength and generalisability of conclusions.

Additionally, heterogeneity in diagnostic criteria, imaging methods, and outcome measures makes direct comparison between studies difficult. Many reports also focus primarily on vascular outcomes, with limited use of objective, sport-specific performance metrics such as VO_2max or functional testing.

Finally, the frequent coexistence of PAES with conditions such as CECS may confound both diagnosis and treatment outcomes. Further prospective studies using standardised protocols and performance-based outcomes are needed.

10. CONCLUSIONS

PAES is an important yet frequently overlooked cause of exercise-induced lower limb symptoms in athletes, with a substantial impact on exercise tolerance and performance. Its pathophysiology is complex and multifactorial, involving dynamic arterial compression, ischemia, and secondary vascular and muscular adaptations. Accurate diagnosis requires a high index of suspicion and the use of dynamic imaging techniques capable of reproducing exercise-induced vascular compromise.

Effective management — most commonly surgical decompression — can lead to significant improvement in symptoms, restoration of exercise capacity, and successful return to sport, particularly when implemented early in the disease course. However, optimal outcomes depend on a comprehensive approach that includes recognition of coexisting conditions, individualised treatment planning, and structured rehabilitation.

In summary, early diagnosis and targeted intervention are essential to prevent long-term vascular complications and to maximise functional recovery in athletes with PAES. Further research is needed to refine diagnostic strategies and optimise management pathways, with particular emphasis on long-term performance outcomes and sport-specific rehabilitation.

DISCLOSURE

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CRediT Author Contributions (taxonomy)

Mapped to the CRediT (Contributor Roles Taxonomy, NISO Z39.104-2022). Author initials: AJ = Janaszek Agnieszka; KF = Frączek Karolina; AM = Majchrzyk Aleksandra; JŁ = Łyżwa Julia; AS = Strzępek Aleksandra; KK = Kałuża Kinga; AC = Chmurska Agnieszka; MP = Pater Michał; NK = Kałwa Natalia; MS = Smerdzyński Mateusz.

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