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External Iliac Artery Endofibrosis in Cyclists and Other Endurance Athletes: An Underrecognized Cause of Exercise - Induced Limb Ischemia - A Narrative Review

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Abstract

Background: External iliac artery endofibrosis (EIAE) is a non-atherosclerotic cause of exercise-induced limb ischemia, reported mainly in cyclists and other endurance athletes. Resting pulses and ankle-brachial index are often normal, which can delay diagnosis.

Aim. To review evidence on pathophysiology, presentation, diagnosis, treatment, and return to sport.

Material and methods: PubMed/MEDLINE was searched through 4 August 2026. Reference lists and relevant full-text sources were also screened. Reviews, consensus papers, diagnostic studies, cohorts, and surgical series were prioritized.

Results: EIAE is linked to repeated hip flexion, high-flow shear stress, arterial fixation, and elongation or kinking. Athletes usually report reproducible unilateral loss of power, thigh or buttock discomfort, cramping, or paresthesia at high workloads. Assessment should reproduce symptoms and combine immediate post-exercise pressures with dynamic duplex ultrasonography; CTA or MRA defines anatomy. Activity and bike-fit changes may help mild cases. In selected athletes, endarterectomy with autologous patching or correction of kinking has the most consistent outcome data, whereas evidence for stenting remains limited.

Conclusions. EIAE should be considered in endurance athletes with repeatable unilateral leg failure despite normal resting findings. Sport-specific exercise testing is central to timely diagnosis and appropriate treatment.
Keywords: iliac artery endofibrosis; cycling; endurance athlete; exercise-induced ischemia; ankle-brachial index; duplex ultrasonography; return to sport

Introduction

Endurance athletes with exertional leg symptoms are usually assessed first for musculoskeletal overload, chronic exertional compartment syndrome, lumbar or peripheral nerve disease, or training-related fatigue. This is reasonable because vascular disorders are uncommon. Nevertheless, a flow-limiting lesion may become clinically important when the limb is required to sustain near-maximal blood flow. External iliac artery endofibrosis (EIAE) is the best-described non-atherosclerotic lesion in this setting. It is characterized by intimal thickening, usually in the external iliac artery, and may coexist with arterial elongation, dynamic kinking, vasospasm, or thrombosis [1-4].

Although EIAE was first described in competitive cyclists, it remains unfamiliar to many clinicians outside specialist sports-vascular centers [5-10]. Pulses and ankle-brachial index (ABI) may be normal at rest, while imaging performed with the hip extended can underestimate an obstruction that develops during hard exercise or sustained hip flexion. A typical athlete reports that one leg repeatedly loses power at a predictable workload even though breathing and overall fitness would allow continued effort. That mismatch between systemic capacity and unilateral limb performance is an important diagnostic clue [3,9-11].

Cycling creates the classic combination of prolonged hip flexion, repetitive force production, and exceptionally high lower-limb blood flow. Similar iliac flow limitations have also been reported in triathletes, runners, speed skaters, rowers, cross-country skiers, and other endurance athletes. The condition is not confined to men or professionals; women and recreational competitors are affected as well [1,2,12-14].

When the diagnosis is missed, athletes may spend months or years undergoing orthopedic imaging, physiotherapy, compartment-pressure testing, spinal assessment, or even surgery without relief. The opposite error is also possible: a minor anatomical irregularity on imaging

may be mistaken for the cause of symptoms. A clinically meaningful diagnosis requires concordance between the athlete's complaint, exercise-induced loss of perfusion, and a lesion in the corresponding arterial segment [1-4,15,16].

This narrative review examines the epidemiology, mechanisms, clinical presentation, investigation, treatment, and return-to-sport considerations associated with EIAE and related iliac flow limitations. Particular attention is given to practical diagnostic reasoning, differentiation from more common causes of exertional leg pain, and areas where the evidence remains incomplete.

Material and methods

PubMed/MEDLINE was searched for relevant literature, and PubMed Central was used to access available full texts. Reference lists and publisher pages were screened for additional reports. The final search was completed on 4 August 2026. The principal search combined disease and population terms: ("iliac artery endofibrosis" OR "external iliac artery endofibrosis" OR "flow limitation in the iliac arteries") AND (athlete* OR cyclist* OR runner* OR endurance). Additional searches included exercise testing, ABI, duplex ultrasonography, CTA, MRA, NIRS, endarterectomy, patch angioplasty, arterial release, shortening, stenting, and return to sport.

Priority was given to consensus statements, systematic and scoping reviews, diagnostic studies, cohort studies, surgical outcome series, and landmark pathological or mechanistic reports. Case reports were retained when they described an uncommon sport, a female athlete, an acute vascular complication, or a diagnostic pitfall not represented in larger studies. Eligible publications addressed non-atherosclerotic iliac flow limitation in athletes or highly active people and provided clinically relevant information on mechanism, diagnosis, treatment, or outcome. Literature devoted solely to conventional atherosclerotic peripheral artery disease was excluded, except where it informed differential diagnosis.

The evidence was not suitable for meta-analysis. Most studies were retrospective, many originated from a small number of specialist centers, and terminology, exercise protocols, diagnostic thresholds, operations, and outcome definitions varied substantially. A formal study-by-study risk-of-bias score was therefore not applied. Instead, the evidence was synthesized by clinically relevant themes: disease definition and mechanisms, presentation, diagnostic testing, differential diagnosis, treatment, and return to sport. Recent reviews were used to map the literature, while primary studies were consulted for specific diagnostic and outcome data [1-4].

Table 1. Evidence domains included in the narrative synthesis.

Evidence domain	Representative source types	Contribution to the review
Definition and mechanisms	Landmark case series, histopathology, anatomical studies	Defines endofibrosis, arterial elongation, kinking, fixation, shear stress, and vasospasm
Diagnosis	Consensus, exercise-physiology studies, duplex and angiographic studies	Supports sport-specific provocation, immediate pressure testing, dynamic imaging, and multimodal confirmation
Treatment	Systematic/scoping reviews and surgical cohorts	Describes conservative care, endarterectomy and patching, arterial release or shortening, bypass, and endovascular uncertainty
Athlete diversity and differential diagnosis	Case reports, female series, runner reports, exertional-leg-pain reviews	Prevents overrestriction of the diagnosis to elite male cyclists and clarifies competing diagnoses

Results

Terminology, anatomy, and disease definition

Endofibrosis is a non-atherosclerotic disorder of the arterial intima. The external iliac artery is affected most often, although lesions have also been reported in the common iliac, common femoral, profunda femoris, and superficial femoral arteries. Histological studies describe concentric or eccentric intimal thickening composed of loose connective tissue and myofibroblastic elements, with relative preservation of the media and without the lipid-rich core or calcification typical of atherosclerosis [5,6,17,18]. The term EIAE is most accurate when a structural intimal lesion has been demonstrated. The broader expression flow limitation

in the iliac arteries (FLIA) also includes obstruction caused by excessive arterial length, kinking, dynamic compression, vasospasm, or mixed disease [9,10].

The external iliac artery follows the medial border of the psoas muscle, passes beneath the inguinal ligament, and becomes the common femoral artery. In an aerodynamic cycling position, repeated hip flexion alters both the course and longitudinal tension of the vessel. Branches, fascial attachments, and the inguinal region may limit arterial mobility and concentrate stress over a short segment. Psoas hypertrophy and an enlarged psoas branch have also been proposed as local contributors, although neither is found consistently [11,19].

A fixed intraluminal stenosis should be distinguished from a predominantly functional kink. Endofibrosis may be visible at rest but become more restrictive as flow rises. Conversely, an elongated iliac artery can appear almost normal with the hip extended and form a sharp angle only in the racing position. Mixed lesions are common. Static imaging can therefore be misleading, and treatment should be directed at the dominant mechanism rather than at the label alone [4,9,20-22].

Epidemiology and athlete profile

The true prevalence of EIAE is unknown. There has been no population-based screening, referral is highly selective, and athletes with normal resting tests may never reach a vascular clinic. Estimates from specialist cycling cohorts cannot be generalized to the wider athletic population [1-3]. The available evidence supports a cautious conclusion: EIAE is uncommon, but it is probably underrecognized among athletes with substantial cumulative endurance exposure.

Early series largely involved male competitive cyclists in their twenties to forties after years of high-volume training. More recent reports include women, recreational cyclists, runners, triathletes, and speed skaters [12-14]. The apparent male predominance in older studies may reflect participation and referral patterns as much as biological susceptibility. Reports in women also show that disease may be bilateral and may initially be attributed to a nonvascular condition [13].

Symptoms are usually unilateral, and several series have reported a left-sided predominance. Individual anatomy, limb dominance, cycling position, and differences in branch fixation have all been suggested as explanations, but none has been confirmed. Bilateral disease remains possible and should be considered when both legs fail at high workloads or when contralateral abnormalities are seen on imaging [1,3,22].

A long history of high-intensity endurance training appears to increase risk, but no practical dose threshold has been established. Published studies do not identify a dependable number of annual kilometers, weekly training hours, power output, or career years above which EIAE becomes likely. A younger or recreational athlete should therefore not be excluded simply because the accumulated training load is lower than that of a professional cyclist.

Pathophysiology and sport-specific mechanisms

The proposed pathophysiology is multifactorial. Maximal exercise produces high cardiac output, blood velocity, and shear stress, while repeated hip flexion continually changes arterial geometry and longitudinal tension. If the vessel is partly tethered by branches or surrounding tissues, recurrent deformation may injure the endothelium and stimulate intimal proliferation. Resting flow can remain sufficient for years, but flow reserve gradually falls until symptoms appear at a particular intensity [5,9-11,17].Excessive arterial length adds an important dynamic component. Magnetic resonance and operative studies have demonstrated elongated iliac arteries in symptomatic endurance athletes, with some lesions caused mainly by kinking during hip flexion rather than by a fixed stenosis [20,23,24]. This distinction matters clinically: endarterectomy treats diseased intima, whereas release or shortening is intended to correct abnormal geometry.

Local anatomy may influence where and how the lesion develops. A hypertrophied psoas can alter the arterial course, while a prominent psoas branch or another side branch may tether the vessel during pedaling. Surgeons may divide or mobilize these structures when they appear relevant. Their inconsistent presence, however, makes it unlikely that a single anatomical feature explains every case [11,19].

Vasospasm can coexist with structural disease and may partly explain why results differ between tests. Shalhub et al. described athletes with endofibrosis and vasospastic features who improved after surgery [25]. Temperature, sympathetic activation, exercise intensity, and the interval between symptom onset and measurement may all affect the apparent severity of obstruction. Testing after complete recovery can therefore miss a dynamic component. EIAE is distinct from atherosclerosis, but it is not always benign. Progressive narrowing can lead to severe stenosis, thrombosis, or occlusion, and isolated reports describe dissection or acute limb ischemia. These complications are uncommon, yet they justify timely vascular assessment when an athlete develops a consistent unilateral loss of power or symptoms that persist longer than usual after exercise [1,3,16,17].

Clinical presentation and reasons for diagnostic delay

The most characteristic presentation is an intensity-dependent problem in one limb. Athletes may describe deep thigh or buttock discomfort, cramping, tightness, paresthesia, a swollen sensation, early fatigue, or an abrupt fall in power. Symptoms often emerge during climbing, time trials, high-resistance intervals, racing, or prolonged work near maximal aerobic capacity. Easier training may remain symptom free, and complaints usually settle within minutes after power is reduced or exercise stops [1-4,10]. Not every athlete describes pain, and many do not recognize their symptoms as claudication. Common phrases are that the leg feels empty, heavy, blocked, or unable to push. The other leg and the cardiorespiratory system may still feel capable of continuing. Power-meter asymmetry, observations from a coach, or a reproducible decline on one side may therefore be as informative as the pain history.

Resting examination is often unrevealing. Femoral and distal pulses may be palpable, the limb warm, and resting ABI normal. A bruit can be absent until the hip is flexed or the athlete has exercised. In a young person without conventional vascular risk factors, these normal findings can create false reassurance unless the assessment is extended to exercise [4,9,21].

The symptom pattern overlaps with chronic exertional compartment syndrome, lumbar radiculopathy, hip disease, muscle injury, bone stress injury, and peripheral nerve entrapment. Some athletes consequently undergo prolonged rehabilitation or fasciotomy before a vascular cause is considered. A useful history defines the exact location, workload threshold, recovery time, laterality, relation to posture, and reproducibility of the complaint [15,26].

Urgent vascular assessment is needed when the usual rapid recovery no longer occurs or when new resting signs appear. A pulse deficit, persistent pain, coolness, pallor, prolonged sensory change, rest pain, or sudden loss of function may indicate thrombosis, dissection, or severe fixed obstruction rather than uncomplicated exercise-induced flow limitation [1,3,16].

Physical examination and exercise provocation

Both limbs should be examined systematically. Femoral, popliteal, posterior tibial, and dorsalis pedis pulses are assessed with the hips neutral and, where practical, in a flexed position that resembles cycling. Auscultation over the iliac and femoral regions may reveal a position-dependent bruit. Blood pressure, limb temperature, neurological findings, hip movement, lumbar signs, and focal musculoskeletal tenderness should also be documented because competing diagnoses may coexist [4,15]. The diagnostic test must reproduce the athlete's usual

symptoms. In cyclists, an incremental cycle-ergometer protocol should continue to the workload that limits training or racing. A submaximal treadmill test may be falsely negative because it does not recreate the same power demand or hip position. Runners should generally be tested by running, and other athletes may need a sport-specific protocol. Reaching a target heart rate is less important than provoking the familiar unilateral complaint [2,4,10]. Ankle pressures should be measured immediately after exercise. Perfusion can recover within minutes, so even a short delay may conceal a clinically important fall. Although protocols differ, bilateral ankle pressures and ABI should be obtained as quickly as possible and, when useful, repeated during recovery. Recording the workload at symptom onset and comparing the symptomatic and contralateral limbs improves interpretation [4,21].

A normal resting ABI does not exclude EIAE. A post-exercise pressure drop, a clear side-to-side difference, or delayed recovery supports a hemodynamically important lesion, but proposed thresholds vary between studies and centers. Results should therefore be interpreted alongside symptom reproduction and imaging. Measurements obtained with the hip flexed may improve sensitivity when obstruction is posture dependent [2,4,21].

Diagnostic imaging and physiological testing

Duplex ultrasonography is usually the first imaging test because it is noninvasive, repeatable, and able to assess both structure and hemodynamics. A focused study should examine the distal aorta, common and external iliac arteries, and common femoral arteries bilaterally. Relevant findings include intimal thickening, focal narrowing, increased peak systolic velocity, post-stenotic disturbance, and kinking during hip flexion. Pre- and post-exercise measurements are particularly useful when the resting study is equivocal [4,7,21].

Duplex assessment is limited by operator dependence and the depth of the pelvic vessels. A mild lesion may be missed during a routine resting study, especially when the hip remains extended or scanning begins after pressures have recovered. The referral should therefore state that iliac endofibrosis is suspected and request dynamic, exercise-related assessment rather than a generic lower-limb arterial examination [4,21]. Computed tomography angiography (CTA) and contrast-enhanced magnetic resonance angiography (MRA) demonstrate the full course of the iliac arteries and can identify stenosis, elongation, kinking, occlusion, or another pelvic abnormality. CTA is fast and offers high spatial resolution; MRA avoids ionizing radiation, which is advantageous in young athletes. Either technique may underestimate functional obstruction if performed only in a neutral position. Hip-flexed acquisitions and curved planar

reconstructions can make abnormal geometry more apparent [20,22,27]. Catheter angiography is generally reserved for unresolved cases, procedural planning, or a contemplated endovascular intervention. It is invasive and can still appear normal if provocative positioning is omitted. Pressure gradients or intravascular ultrasound may add information in selected centers, particularly before stenting, but neither is a routine requirement for diagnosis [1,22,28]. Near-infrared spectroscopy (NIRS) measures changes in muscle oxygenation during exercise and may demonstrate the downstream effect of iliac flow limitation. Pilot and subsequent diagnostic studies found abnormal deoxygenation patterns in affected athletes, including some with inconclusive conventional investigations [29,30]. The method is attractive because it records physiology during exercise, although protocols, equipment, and thresholds still require broader validation. The diagnosis is most convincing when three elements agree: the typical symptom is reproduced at high intensity, impaired perfusion is documented during or immediately after exercise, and imaging shows a plausible lesion on the symptomatic side. When findings are discordant, the clinician should revisit exercise intensity, timing, hip position, technical quality, and alternative diagnoses rather than proceeding directly to surgery or dismissing the complaint [4,15].

Table 2. Diagnostic tools used in suspected external iliac artery endofibrosis or related iliac flow limitation.

Test	**Most useful finding**	**Strengths**	**Important limitations**
History and sport-specific provocation	Reproducible unilateral loss of power, thigh or buttock symptoms, or paresthesia at a predictable high workload	Defines symptom concordance and guides all further testing	Symptoms overlap with musculoskeletal and neurological disorders
Resting ABI and pulse examination	Baseline asymmetry or fixed obstruction	Widely available and useful for severe disease	Often normal in dynamic or moderate disease; cannot exclude EIAE

Test	Most useful finding	Strengths	Important limitations
Immediate post-exercise ABI/ankle pressures	Pressure fall, side-to-side difference, or delayed recovery after typical symptoms	Demonstrates physiological significance	Rapid recovery and variable thresholds make immediate timing essential
Dynamic duplex ultrasonography	Wall thickening, focal acceleration, turbulence, or posture-dependent kinking before/after exercise	Noninvasive; combines anatomy and hemodynamics	Operator dependent; deep pelvic segments may be difficult to visualize
CTA or MRA	Stenosis, arterial elongation, kinking, occlusion, or alternative pelvic pathology	Defines arterial course and supports operative planning	Neutral-position imaging may underestimate dynamic obstruction; CTA involves radiation
Catheter angiography	Provoked narrowing or lesion anatomy requiring procedural clarification	High temporal resolution and option for pressure assessment	Invasive; may be falsely reassuring without provocation
Near-infrared spectroscopy	Abnormal exercise-related muscle deoxygenation pattern	Measures downstream physiological consequence during exercise	Emerging technique with limited availability and evolving thresholds

Differential diagnosis

Differential diagnosis becomes more manageable when symptom location, triggering activity, recovery time, and objective findings are considered together. Chronic exertional compartment syndrome usually produces tightness, pain, or paresthesia within a defined lower-leg compartment after a repeatable duration of exercise. Intracompartmental pressure testing may be appropriate when suspicion remains high, but iliac testing deserves priority when symptoms involve the thigh or buttock, pulses change with exercise, or loss of power is the dominant complaint [26].

Popliteal artery entrapment syndrome is another vascular cause of exertional symptoms in young athletes, although pain is usually centered in the calf or foot. Plantar flexion may alter distal pulses or Doppler flow, and cross-sectional imaging is directed at the popliteal fossa rather than the iliac axis [31]. Because resting ABI may be normal in both conditions, careful localization and appropriately targeted provocative imaging are essential.

Lumbar radiculopathy and peripheral nerve entrapment may also cause pain, paresthesia, or weakness, but symptoms may be linked to spinal position and can persist longer after exercise. Hip impingement, labral pathology, tendinopathy, muscle injury, and bone stress injury may mimic or coexist with EIAE. A musculoskeletal abnormality should be accepted as causal only when it reproduces the athlete's usual high-intensity complaint.

Atherosclerotic peripheral artery disease is more relevant in older recreational athletes and in those with cardiovascular risk factors. Other possibilities include thromboembolism, arterial dissection, cystic adventitial disease, vasculitis, fibromuscular dysplasia, and congenital arterial anomalies. These diagnoses warrant particular attention when the clinical course is atypical or imaging does not fit the usual EIAE pattern [3,15,26].

Table 3. Differential diagnosis of reproducible exercise-induced leg symptoms in athletes.

Condition	**Typical symptom pattern**	**Resting examination**	**Most informative test**
External iliac artery endofibrosis / iliac flow limitation	Unilateral thigh, buttock, or diffuse leg fatigue; loss of power at high intensity; rapid improvement with reduced workload	Often normal pulses and ABI	Sport-specific maximal exercise with immediate pressures and dynamic iliac duplex; CTA/MRA if needed
Chronic exertional compartment syndrome	Tightness, pain, or paresthesia in a defined lower-leg compartment after a reproducible exercise duration	Usually normal	Clinical assessment plus appropriately performed intracompartmental pressure testing or selected post-exercise MRI
Popliteal artery entrapment syndrome	Exertional calf pain, cramping, or foot paresthesia; may be provoked by plantar flexion	May show dynamic pulse change	Provocative duplex and cross-sectional imaging of the popliteal region
Lumbar radiculopathy / peripheral nerve entrapment	Dermatomal pain or paresthesia, weakness, possible posture dependence; recovery may be slower	Neurological signs may be present	Focused neurological examination, electrodiagnostic testing, and selective spine/peripheral nerve imaging

Condition	Typical symptom pattern	Resting examination	Most informative test
Bone stress injury or muscle-tendon disorder	Focal pain related to impact or loading; may progress to pain at lower intensity or at rest	Local tenderness or provocative test	Targeted radiography, MRI, or musculoskeletal ultrasonography
Atherosclerotic or other arterial disease	Claudication at lower workloads; risk factors or persistent vascular signs more likely	Reduced pulses or ABI more common	Resting and exercise ABI, full arterial duplex, CTA/MRA, and etiological work-up

Conservative management

Conservative care is reasonable when symptoms are mild, physiological significance remains uncertain, performance goals are modest, or the athlete wishes to avoid surgery. The most reliable way to prevent ischemic symptoms is to reduce the intensity or volume that provokes them. This may be acceptable for a recreational cyclist but may not meet the goals of a competitor whose symptoms appear only near race pace [1,2,11]. Bike-fit changes are intended to reduce sustained hip flexion and arterial deformation. Options include a less aggressive trunk position, higher handlebars, a different saddle position, shorter cranks, or a shift from high-torque to higher-cadence pedaling. These adjustments require individual testing because they may not alter the relevant arterial geometry and can create other biomechanical problems. The supporting evidence remains observational [2,11]. Rehabilitation may improve hip mobility, trunk and pelvic control, secondary pain, and tolerance of a modified riding position, but it cannot remove established intimal fibrosis. Stretching, manual therapy, and strengthening are therefore supportive measures rather than curative treatment for a fixed arterial stenosis [1,2,11].

Antiplatelet, anticoagulant, or lipid-lowering medication has not been shown to reverse uncomplicated endofibrosis. Drug treatment should be linked to a clear indication, such as acute thrombosis, perioperative management, a vascular reconstruction, or coexisting cardiovascular

disease [1-4]. Acute thrombosis requires urgent vascular care and should not be managed as stable exertional symptoms.

The main weakness of conservative management is the lack of evidence for a durable return to competition. A 2024 scoping review found no standardized nonoperative protocol supported by adequate outcome data [2]. Athletes with documented ischemia, a falling workload threshold, or persistent restriction despite modification should be reviewed by an experienced multidisciplinary vascular team.

Surgical and endovascular treatment

Surgery should be considered only when the familiar symptoms have been reproduced, an exercise-related perfusion deficit has been demonstrated, and imaging identifies a lesion that explains the affected side. Planning must distinguish focal intimal disease from excessive arterial length, dynamic kinking, external tethering, or long-segment occlusion. A technically successful operation aimed at the wrong mechanism may not restore performance [3,4,15].

For focal EIAE, endarterectomy with an autologous vein patch is the most established reconstruction. The diseased intima is removed and the artery enlarged, commonly with great saphenous vein. Autologous tissue avoids a prosthetic implant in a young patient and in an arterial segment exposed to repeated movement. Tethering branches or constraining tissues may be released during the same procedure when they contribute to the lesion [3,15,32].

The most detailed long-term data come from a 2023 specialist-center cohort of 68 patients with 79 treated limbs. Cycling workload at symptom onset increased after surgery, 91.2% of patients reported early symptom reduction, and 94.5% continued to report improvement after a median follow-up of 11.1 years [32]. These results support endarterectomy with patching in carefully selected athletes, but they should not be extrapolated to vague symptoms or incidental minor lesions.

When excessive arterial length and kinking are the principal abnormalities, release or surgical shortening may be more appropriate than endarterectomy. Specialist-center cohorts describe improvement and durable satisfaction after these procedures in selected endurance athletes [23,24]. Differences between subjective recovery and objective hemodynamic change in some series reinforce the need to define the dominant lesion before surgery. Bypass is usually reserved for long occlusions, thrombosis, failed reconstruction, or anatomy that cannot be treated with a focal repair. Autologous vein is often preferred when suitable. Although bypass can restore perfusion, graft durability and decades of repetitive hip movement are major

considerations in a young athlete [3,33]. Acute limb ischemia may require thrombectomy, thrombolysis, endarterectomy, or bypass depending on anatomy and limb viability. Balloon angioplasty and stenting remain controversial. Angioplasty does not remove fibrotic intima or correct kinking, while a stent in a mobile iliac segment is exposed to repeated bending and compression. A 2024 series reported favorable long-term outcomes in a small group who declined surgery, but recurrent symptoms occurred in three of ten patients and there was no comparison group [28]. Endovascular treatment may be reasonable in exceptional circumstances, yet current evidence does not support routine first-line use in young high-performance athletes. Informed consent should cover wound and lymphatic complications, thrombosis, restenosis, nerve or venous injury, and the possibility that a patent reconstruction will not restore previous performance. The goal is to re-establish safe perfusion and relieve reproducible ischemic symptoms, not to guarantee a return to the former race level. Age, bilateral disease, training history, competitive goals, and willingness to modify position should all inform shared decision-making [15,23,24,32,34].

Table 4. Management options and the lesion mechanism they are intended to address.

Management option	**Best-supported clinical context**	**Potential advantages**	**Main concerns**
Activity and bike-fit modification	Mild symptoms, uncertain physiological significance, recreational goals, or a preference to avoid surgery	Noninvasive; may reduce hip flexion and symptom-provoking exposure	Evidence is limited; established fibrosis does not regress and high-intensity symptoms may persist
Endarterectomy with autologous vein patch	Focal, physiologically important endofibrotic stenosis with concordant symptoms	Removes diseased intima, enlarges lumen, and has encouraging long-term specialist-center outcomes	Open-surgical complications, restenosis, and incomplete performance recovery remain possible

Management option	Best-supported clinical context	Potential advantages	Main concerns
Arterial release	Dynamic kinking related to tethering without major stenosis or excessive length	Preserves the artery and addresses external fixation	Success depends on accurate mechanism classification; symptoms may recur
Arterial shortening / resection and reanastomosis	Excessively long, kinked iliac artery with exercise-induced flow limitation	Corrects geometry and has reported durable satisfaction	Open reconstruction in a young athlete; risk of recurrence or reintervention
Bypass reconstruction	Long occlusion, thrombosis, complex anatomy, or failure of a previous focal repair	Can restore flow when focal repair is unsuitable	Graft durability, conduit choice, and repetitive hip-motion exposure
Angioplasty or stenting	Exceptional selected cases after specialist multidisciplinary review	Less invasive and potentially rapid recovery	Does not correct all kinking or fibrosis; long-term comparative evidence and device durability remain uncertain

Return to sport and postoperative surveillance

Return-to-sport practice after iliac reconstruction is not standardized. Published pathways vary with the operation, incision, disease severity, and level of competition, and a recent scoping review found no validated universal timetable [2]. Athletes should be advised that progression depends on healing, symptoms, vascular findings, and workload tolerance rather than on a single calendar date. Early rehabilitation centers on wound healing, distal perfusion, and recognition of thrombotic or lymphatic complications. Walking generally precedes cycling, followed by low-resistance stationary riding once the reconstruction is judged stable. Hip

flexion, duration, and intensity are then increased gradually. New swelling, changing pulses, worsening incision pain during loading, or recurrent ischemic symptoms should prompt reassessment [2]. Progression is best guided by clinical milestones: a healed wound, symmetric resting vascular findings, absence of ischemic symptoms in daily life, recovered hip motion, tolerance of easy cycling, and graded workload without the previous unilateral loss of power. Before unrestricted competition, many specialist pathways repeat maximal sport-specific exercise with immediate pressure measurements and duplex assessment [2,23,24,32]. Arterial patency does not automatically mean that performance has recovered. A long diagnostic delay may lead to deconditioning, compensatory movement, and anxiety about recurrent symptoms. Rehabilitation should therefore integrate strength and conditioning, bike fit, nutrition, and psychological readiness. Coaches need clear workload limits and warning signs rather than a simple statement that the artery has been repaired [2]. Long-term follow-up is reasonable because restenosis, contralateral disease, progression, and recurrent kinking can occur. The literature does not define an optimal surveillance interval. Clinical review and duplex assessment are appropriate when symptoms return and may be scheduled periodically for high-level athletes after reconstruction. A sudden change in pulses or symptoms that persist after exercise requires urgent evaluation [2,23,24,32].

Prevention and early recognition

Routine screening of asymptomatic cyclists cannot currently be recommended because the natural history and a useful risk threshold are unknown. Prevention therefore depends mainly on early recognition. Athletes with a characteristic symptom pattern should have access to exercise-based vascular testing before disease progresses to severe stenosis or thrombosis [1,2]. Athletes and coaches should know that repeatable unilateral loss of power, deep thigh cramping, or numbness at high intensity is not always a training or bike-fit issue. Power-meter records, including workload at onset and side-to-side asymmetry, can make the history more precise and shorten the route to appropriate testing. Bike fitting may reduce provocation in some athletes, but no single position has been shown to prevent EIAE. A substantial increase in time spent in extreme hip flexion should be introduced gradually, and new unilateral symptoms after a position change deserve assessment. Any adjustment must also account for comfort, overall biomechanics, aerodynamics, and the athlete's wider injury profile [2,11]. After treatment, risk reduction relies on gradual reloading, avoidance of an abrupt return to maximal torque, prompt investigation of recurrent symptoms, and appropriate vascular follow-up. Continued

surveillance is supported by expert practice and observational evidence, although no evidence-based lifelong schedule has been established [1,2].

Discussion

EIAE is difficult to recognize because the vascular deficit may be absent at rest. Symptoms appear when high flow demand, repeated hip flexion, and an abnormal iliac segment interact. Resting pulse examination, ABI, and neutral-position imaging may therefore all be normal. A negative work-up is persuasive only if the athlete's usual symptoms were reproduced and measurements were obtained before recovery [1-4,21]. A practical diagnostic framework separates three questions. Is the symptom pattern compatible with iliac flow limitation? Is perfusion objectively impaired during or immediately after exercise? Does imaging identify a lesion that explains the physiological deficit on the symptomatic side? Symptoms alone are nonspecific, a structural irregularity may be incidental, and an abnormal exercise test without lesion localization may reflect technical error or disease elsewhere in the arterial tree. Surgical series show that carefully selected athletes can improve substantially, but the evidence has clear limitations. Most cohorts are retrospective, originate from a small number of highly experienced centers, and use different definitions of diagnosis, success, symptom relief, and return to sport. Operated athletes are also a motivated and selected group with specialist follow-up. High satisfaction rates demonstrate that durable benefit is possible; they do not justify reconstruction of every subtle imaging abnormality [1-3,23,24,28,32]. For focal intimal disease, endarterectomy with autologous patching has the strongest long-term outcome data. Release and shortening are supported when dynamic tethering or excessive arterial length predominates [23,24,32]. These studies also expose the limits of using EIAE as a single label: some athletes have mainly intimal fibrosis, others abnormal geometry, and many a mixture of both. Future reports should describe the preoperative phenotype, provocation protocol, lesion morphology, operation, objective hemodynamics, complications, and sport-specific outcomes using common definitions. The role of stenting is unresolved. A small 2024 series suggests that long-term symptom control may be possible in selected patients who decline surgery, but recurrence occurred and comparative data are lacking [28]. In view of the young age of many athletes, repetitive hip motion, and the possibility of later reconstruction, stenting should be presented as an individualized alternative rather than an established equivalent to open repair.

Important evidence gaps remain. Women and athletes outside cycling are underrepresented, bilateral disease is inconsistently reported, and the natural history of mild or asymptomatic

lesions is uncertain. Post-exercise ABI, duplex velocity criteria, hip-flexed testing, and NIRS protocols are not standardized. Return-to-sport guidance is based mainly on expert practice and observational cohorts rather than controlled trials [2,4,13,29,30]. Even within these limitations, the diagnostic pathway can be improved. Normal resting pulses should not end the assessment of an endurance athlete with repeatable unilateral leg failure at high intensity. Equally, surgery should not be recommended from imaging alone. The most defensible decisions are made when symptoms, exercise physiology, and arterial anatomy are concordant.

Practical implications for sport quality

Delayed recognition affects both athlete health and training availability. It can interrupt effective training for long periods, lead to repeated low-yield investigations, and expose athletes to inappropriate rehabilitation or surgery. A coherent pathway should link primary care, sports medicine, vascular laboratories, radiology, vascular surgery, physiotherapy, and coaching instead of leaving the athlete to navigate each specialty separately. A practical pathway includes eight steps: (1) recognize repeatable unilateral loss of power or thigh/buttock symptoms at high intensity; (2) document workload, posture, laterality, and recovery; (3) examine both limbs at rest; (4) reproduce the familiar symptoms with a maximal sport-specific test; (5) obtain immediate post-exercise pressures and dynamic duplex ultrasonography; (6) use CTA or MRA when anatomy is uncertain or intervention is being considered; (7) review concordant findings within a multidisciplinary team; and (8) use criteria-based rehabilitation with repeat physiological testing before unrestricted competition.

A useful test report states whether the athlete's familiar symptoms occurred, the workload achieved, the interval before pressure measurement, the hip position, and whether the lesion corresponds to the symptomatic side. A routine conclusion of "normal arterial circulation" after resting assessment can be misleading. Likewise, a test that did not reproduce the usual complaint may not answer the clinical question [4,15,21]. The topic also fits the broader discussion of quality and safety in endurance sport. Recent Quality in Sport reviews have addressed physical activity in peripheral artery disease and the balance between the health benefits and overuse risks of cycling [35,36]. EIAE illustrates that boundary clearly: endurance exercise is beneficial for most participants, yet a small subgroup develops a sport-related vascular disorder that requires timely recognition. High-quality care protects athletes without medicalizing normal training adaptation.

Conclusions

External iliac artery endofibrosis is an uncommon but clinically important cause of exercise-induced limb ischemia in cyclists and other endurance athletes. Repeated high-flow stress, hip flexion, arterial fixation, and elongation or kinking appear to contribute to intimal injury and functional obstruction. The typical presentation is a reproducible unilateral decline in leg power or function at high intensity despite normal or near-normal findings at rest. Diagnosis depends on agreement between symptoms, exercise physiology, and anatomy. The athlete's usual complaint should be reproduced with a maximal sport-specific test, followed immediately by bilateral pressure measurements and focused dynamic duplex ultrasonography. CTA or MRA helps define lesion morphology and plan treatment. NIRS is promising but remains an adjunct rather than a replacement for established assessment. Activity reduction and bike-fit modification may be appropriate for mild or uncertain disease, although evidence for sustained competitive return is limited. Endarterectomy with autologous patching has the strongest long-term support for focal intimal lesions, while release or shortening can address selected kinking phenotypes. Stenting may be considered in exceptional circumstances, but the current evidence is insufficient for routine use in young athletes. Earlier recognition and a standardized referral pathway are likely to provide the greatest immediate benefit. Multidisciplinary, criteria-based care can reduce unnecessary procedures, identify athletes at risk of thrombosis, and support a safer return to sport. Future studies should use common diagnostic and outcome definitions, include more women and non-cycling athletes, and compare conservative, open, and endovascular strategies prospectively.

Disclosure

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