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Review Article

External Iliac Artery Endofibrosis in Cyclists

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Abstract

An underrecognized clinical condition that may afflict high level cyclists is external iliac artery endofibrosis (EIAE). EIAE is an intermittent claudication vascular condition that results from intimal narrowing most often of the external iliac artery (EIA). Symptoms are reported as thigh pain and loss of power that occur during high intensity efforts. EIAE is theorized to be a result of the mechanical and hemodynamic stress within the EIA heightened by psoas muscle hypertrophy in conjunction with the repetitive and extreme hip flexion coupled with high cardiac output. A combination of clinical tests (e.g. ankle-brachial index) in concert with imaging and vascular studies (e.g. duplex ultrasound) is necessary to arrive at an accurate diagnosis. The mean time from symptom onset to diagnosis is 3 years. Conservative interventions, which consist of bike hardware adjustments and/or posture modifications while riding, are generally not acceptable for a competitive cyclist. Surgical interventions take the form of percutaneous/endoscopic (e.g. balloon angioplasty, stent insertion) or open procedures (e.g. arterial release, endarterectomy, artery reconstruction) to restore arterial flow. Long-term outcomes following percutaneous procedures have followed a finite number of patients to date and are not recommended as a primary intervention for EIAE. Outcomes following open surgical procedures are strong with most riders being able to return to preinjury levels of competition. Greater awareness of EIAE among the scientific and medical community who work with cyclists is needed to improve the efficiency and overall management of EIAE.

Keywords

bicycling, claudication, cyclist, endofibrosis, endurance exercise, external iliac artery

1 Introduction

Hip pain originating from a musculoskeletal source is infrequently encountered in recreational (Weiss, 1985; Dannenberg, 1996) and professional cyclists (Barrios, 2015). In fact, the hip was the least frequently reported region when a group of amateur cyclists (n = 300) were queried about their injury profile following a 94.7-km race (van der Walt, 2014). There is a paucity of information related to overuse injuries in professional cyclists. Over a 12-month period, Clarsen et al. (Clarsen, 2010) surveyed 116 professional riders from seven different teams. 101 riders completed the survey (87% response rate) revealing the lumbar spine and knee regions were the most commonly affected while the hip/groin region was cited by only one rider as necessitating medical attention. Arterial flow limitations rather than musculoskeletal conditions are more of a concern as it relates to the hip in a competitive cycling population. Health care providers need to have a good understanding of external iliac arteriopathy as it can be a debilitating condition for elite cyclists. External iliac arteriopathy can be defined as intermittent claudication that impairs a cyclist's ability to generate maximal power (Schep, 2002a; Wijesinghe, 2001). The most common cause of external iliac arteriopathy is endofibrosis (Chevalier, 1986). The purpose of this clinical commentary is to familiarize the reader with external iliac artery endofibrosis (EIAE) including the anatomy/pathoanatomy, incidence/symptoms, aetiology, diagnosis, interventions and outcomes of this condition in cyclists.

2 Anatomy/Pathoanatomical Features

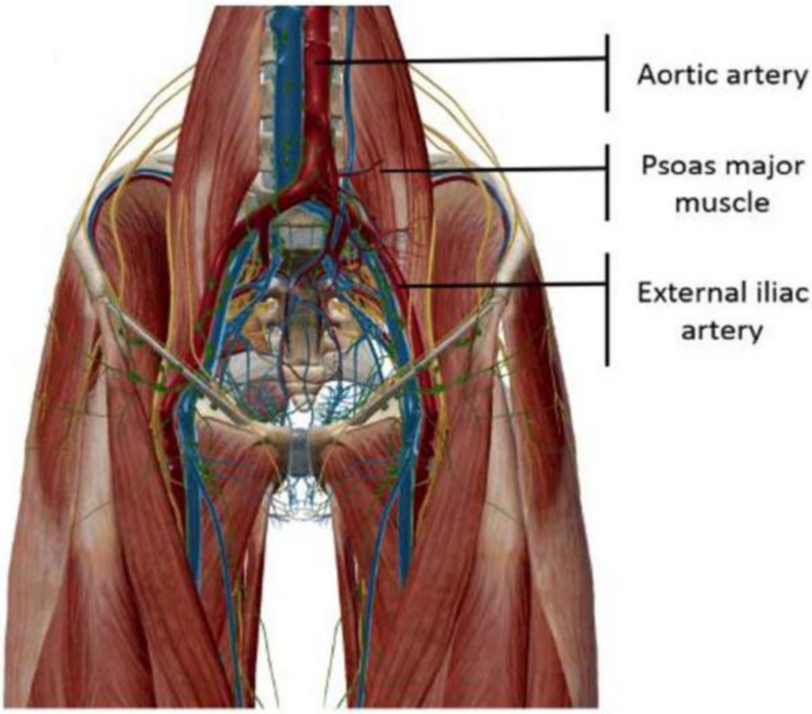

Figure 1. External iliac artery and its relationship to the psoas major muscle. *Image from the app 'Visible Body'.*

The external iliac artery (EIA) (Figure 1) is a significant blood vessel that plays a crucial role in lower limb circulation. It originates from the common iliac artery (CIA) and gives rise to various branches (e.g. inferior epigastric, deep circumflex) as it descends inferiorly and laterally towards the inguinal ligament (Nayak, 2012). After passing deep to the inguinal ligament, the external iliac artery becomes the femoral artery. Anatomically, the EIA is located in the retroperitoneal space, anterior to the psoas major muscle and posterior to the peritoneum. The artery maintains a consistent average length of 10 cm and has been reported to be approximately 1.0 cm in diameter (Nayak, 2019).

Some work has investigated the morphological variability of the EIA citing anatomical variants in 42% (20/48) of cadaveric specimens (Nayak, 2019). Of these, variations were appreciated in the shape and branching pattern of the artery. Specifically, in nine hemi-pelvises (5 right, 4 left) Nayak et al noted the EIA was either looped, twisted or 'S' shaped.

3 Incidence/Symptoms

External iliac artery endofibrosis is a pathological condition that is often overlooked and primarily affects highly trained cyclists (Abraham, 1999; Feugier, 2004; Venstermans, 2009; Wilson, 2010). Cyclists account for 89% of reported cases of EIAE (Feugier, 2004). A 0.66 per 100 incidence rate in professional cyclists over a five-year duration has been appreciated (Fernandez-Garcia, 2002) though the true incidence is unknown as the condition is underdiagnosed. Clarsen and co-authors reported 1.8% of professional cyclists had received surgical treatment to address the condition. Long distance runners and speed skaters (Peach, 2012) also demonstrate susceptibility, albeit at a reduced frequency. (Scavee, 1985; Maree, 2007; Schep, 2002b). The condition was initially documented in the English literature in 1985 by Mosimann et al., (Mosimann, 1985) who presented case reports of external iliac artery stenosis in two elite cyclists. While endofibrosis has been observed in the common iliac (14.4%) (Fisher, 2022; Rouviere, 2014), common (Fisher, 2022) or deep (Rouviere, 2014) femoral and profunda femoris arteries (Lindo, 2018), the condition is most commonly associated with the external iliac artery accounting for up to 96% of cases (Fisher, 2022; Rouviere, 2014).

As indicated by its name, EIAE causes constriction of the vessel which leads to reduced blood flow beyond the affected area. Symptoms are frequently reported as thigh pain and swelling which are perceived during high intensity efforts (Rouviere, 2014; Alimi, 2004). Additionally, patients complain of decreased power that may be accompanied by pain and/or burning in the affected lower extremity often located within the anterior thigh (Wilson, 2010). Additional symptoms may involve muscle cramps and numbness of the affected lower extremity (Kral, 2002).

The symptoms usually manifest unilaterally with a greater frequency observed on the left side (63%) (Fisher, 2022). Others, however, have appreciated (Rouviere, 2014) the condition may present bilaterally in a minority (15/180 = 8.3%) (Rouviere, 2014) of cases. The reason behind the higher prevalence of left-sided pathologies remains unclear. Rajasekaran and Finnoff (Rajasekaran, 2015) suggested that it might be linked to a higher occurrence of left-sided lumbar scoliosis, associated with left psoas hypertrophy. However, it is important to note that this suggestion is speculative, as it was based on a study involving general participants who were not cyclists with a mean age of 67.8 years (Kim, 2013).

4 Aetiology

The root cause of EIAE remains uncertain but is likely influenced by several factors. Anatomical risk factors include psoas muscle hypertrophy (Falor, 2013; Peach, 2012) and or increased arterial length (Fisher, 2022, Schep, 2002a) with or without kinking of the vessel (Rouviere, 2014; Bender, 2004; Scavee, 2003; Rousselet, 1990).

As the EIA rests anterior to the psoas muscle, hypertrophy of the psoas is thought to induce further lengthening or stretching of the EIA during hip flexion predisposing it to endothelial damage (Bender, 2004; Scavee, 2003). Chevalier et al reported the girth of the thigh and calf were 2 – 3 cm greater on the affected side of those diagnosed with EIAE. Whether the increased girth was due to muscle hypertrophy, swelling or other reasons was not reported (Chevalier, 1986). Scavee et al published a case study of a 51-year-old marathon runner with EIAE who demonstrated increased cross-sectional area (CSA) of the psoas on the affected side as measured by computed tomography (CT) without focal signs of inflammation (Scavee, 2003). Similarly, in a retrospective study of 33 patients with EIAE, Fisher et al (Fisher, 2022) reported psoas CSA was greater at all levels on the symptomatic side but only reached statistical significance at the L5 level. At L5, psoas CSA was 54 mm^2 greater ($P = 0.039$) on the affected (1450 mm^2) versus the unaffected (1396 mm^2) side as quantified by CT scan. To date, no studies have prospectively examined psoas muscle hypertrophy and its relationship with EIAE.

Other investigators have demonstrated increased arterial length (Alimi, 2004; Schep, 2002a;) with or without kinking (Rouviere, 2014; Peach, 2012) in patients suffering from EIAE. Of 14 cyclists diagnosed with EIAE who underwent surgical intervention, Alimi and colleagues appreciated increased arterial length in 50% (7/14) of the cases (Alimi, 2004). Similarly, Schep and colleagues found the external iliac artery length ratio (vessel length/straight line distance) of symptomatic cyclists (1.44 ± 0.23) was greater when compared to an asymptomatic control group (1.32 ± 0.13) ($P < 0.05$) (Schep, 2002a). Other work has found arterial kinking may also play a role (Flors, 2011) and if present is most likely to occur at the level of the common femoral artery as it traverses deep to the inguinal ligament (Fisher, 2022).

In addition to anatomic risk factors, contemporary theory posits that the pathogenesis of EIAE stems from mechanical and hemodynamic stress within the EIA due to repetitive and extreme hip flexion in conjunction with high cardiac output (Bender, 2004). Curvature of the EIA which is accentuated when the hip is in a flexed (e.g. cycling) posture (Lim, 2009) (Figure 2) may amplify the hemodynamic stress on the vessel wall of the EIA and/or increase the likelihood the vessel will become kinked (Rouviere, 2014; Bender, 2004).

The focal site of fibrosis is characterized as intimal thickening at a location along its course where the radius of curvature of the vessel is greatest. The pathological tissue contains an excess of smooth muscle cells, dispersed fibroblasts with minimal calcium or lipid build-up, and a small number of inflammatory cells (Vink, 2008; Maree, 2007; Kral, 2002). These findings along with where the pathology is situated within the vessel (Figure 3) suggest morphological differences between EIAE and atherosclerosis.

Vessel stenosis is almost always less than 50% regardless of whether the pathology is localized to the CIA or EIA (Rouviere, 2014). Despite identified anatomical risk factors, what is clear is the repetitive nature of hip flexion at or near maximal exertion seems to be an essential ingredient for the condition to manifest. Rouviere et al (Rouviere, 2014) reported male cyclists diagnosed with EIA had covered an average of 130000 ± 81800 kilometres at the onset of symptoms.

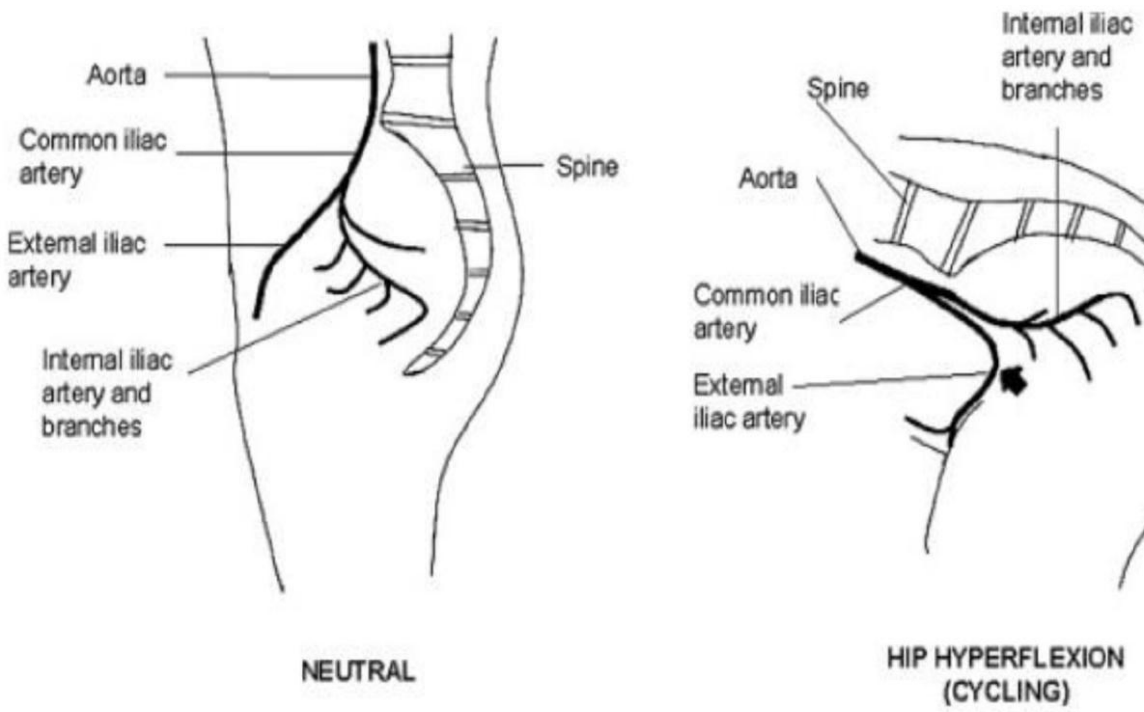

Figure 2. Orientation of the external iliac artery with the hip/spine in a neutral and flexed position. Image from: Lim CS, Gohel MS, Shepherd AC, Davies AH. (2009). Iliac artery compression in cyclists: mechanisms, diagnosis and treatment. *Eur J Vasc Endovasc Surg*., 38:180-186.

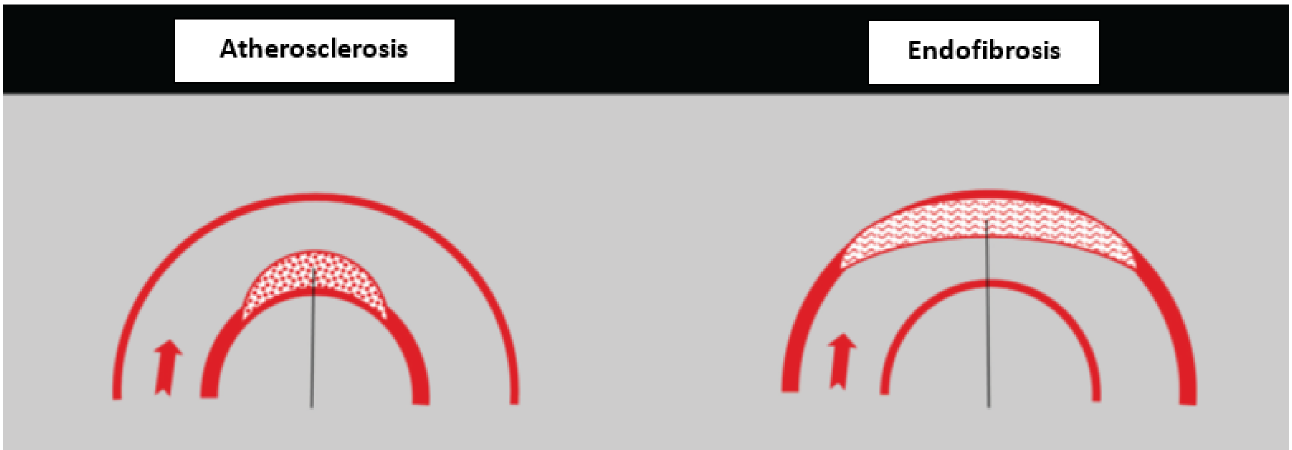

Figure 3. Lumen narrows differences between atherosclerosis and endofibrosis. Image modified from: Gahwiler R, Hirschmuller A, Grumann T, Issak A, Thalhammer C. (2021). Exercise induced leg pain due to endofibrosis of external iliac artery. *Vasa*., 50(2):92-100. Reproduced with permission.

5 Diagnosis

External iliac artery endofibrosis is diagnosed 8-10 times more frequently in males (Mazurova, 2023; Lindo, 2019) though this disparity has been attributed to the sex bias in competitive cycling (Lindo, 2019; Rouviere, 2014; Kral, 2002). The mean age of patients undergoing surgery was cited as 27 (Feguier, 2004) but documented cases range from 18 to 61 (Feguier, 2004; Scavee, 2003; Kral, 2002). The mean duration from the initiation of symptoms to determine a diagnosis is significant and has been reported to range between 12 – 41 months (Brunelle, 2016) with a mean of 36 months (Peake, 2018; Schep, 2002b). Diagnosing intermittent claudication poses a challenge since its signs and symptoms do not manifest at rest but become evident only during near-maximal exercise, making it a scientific conundrum (Veraldi, 2015). Compounding the issue, given the rarity of EIAE in the general population, the attending physician may not be familiar with the condition.

A stepwise approach starting with non-invasive assessment (e.g. clinical exam, ankle-brachial index [ABI]) is recommended (Hinchliffe, 2016). The clinical assessment should include the patient's age, activity level and determine the nature of the symptoms (e.g. provoked by high intensity exercise, relieved with rest) (Mazurova, 2023). If the healthcare practitioner suspects a vascular issue, measurement of the ankle-brachial index (ABI) before and after exercise is recommended (Mazurova, 2023; Hinchliffe, 2016; White, 2007). The ABI is the ratio of the systolic blood pressure at the ankle divided by the systolic blood pressure at the arm. A cycling protocol that gradually increases resistance at regular intervals (e.g. 50 W every 3-4 minutes) (Lindo, 2019; Fernandez-Garcia, 2002) should be carried out until the patient experiences symptoms (Hinchliffe, 2016). The pressure measures should be quantified in the supine position with the hip in an extended position before and after exercise (Hinchliffe, 2016). The first post-exercise measure should occur within 1 minute of exercise cessation and continue every 1-2 minutes for 10 minutes or until a pre-exercise index is attained (Lindo, 2019). Normal values for the ABI are 1.0 – 1.2 whereas 0.66 has been suggested as a criterion for diagnosis (90% sensitivity and 87% specificity) of moderate arterial lesions (Abraham, 2001). Expert consensus states an absolute drop in ankle systolic pressure between 21 and 40 mmHg is considered a positive result (Hinchliffe, 2016). The ABI has been shown to be a valid measure that can differentiate between healthy lower extremities and those with EIAE in elite and professional cyclists (Fernandez-Garcia, 2002).

Should the ABI be positive, duplex ultrasound is the next non-invasive assessment that is likely to be employed to aid in the diagnosis of EIAE. Duplex ultrasound combines both a traditional ultrasound image as well as details related to blood flow associated with doppler. Duplex ultrasound does not use ionizing radiation, is relatively inexpensive and available for use. An experienced ultrasound technician can identify arterial pathology and flow limitations in patients with EIAE (D'Abate, 2017; Schep, 2002b). Additional imaging modalities (e.g. CT angiography, Digital Subtraction angiography, MRA) may also be considered to aid with the diagnosis, pre-operative planning (Peach, 2012) and post-operative follow up (Flors, 2011). In standard clinical practice, a comprehensive patient history and the outcomes of various specific diagnostic assessments are typically considered before arriving at a conclusive diagnosis (Schep, 2002c). Once a diagnosis of EIAE has been confirmed, suitable interventions can be undertaken.

Key diagnostic lessons include the necessity of maintaining a high level of suspicion in young, otherwise healthy endurance athletes with unilateral exertional symptoms; the value of a structured, stepwise diagnostic approach beginning with exercise-provoked ankle-brachial index testing and the complementary role of duplex ultrasound and advanced vascular imaging to confirm flow limitation and guide management. Awareness that resting examinations are often normal and symptoms only manifest at near-maximal workloads is critical to avoiding prolonged diagnostic delays.

6 Interventions / Outcomes

Interventions can be classified as non-invasive (conservative) or invasive (percutaneous/endoscopic or open). Conservative interventions are commonly suggested for recreational athletes as a first step before contemplating invasive procedures. Non-invasive interventions include activity modification or ceasing the offending activity. Cessation is thought to arrest the progression of the disease, while ongoing participation results in additional advancement (Hinchliffe, 2016).

Activity modification may take different forms. On the bike, the goal is to decrease the extreme amount of hip flexion while riding (Peach, 2012). This may be accomplished by postural adjustments of the rider (e.g. maintenance of a more erect spinal posture) and/or bicycle hardware adjustments (e.g. altering the position of the handlebars and/or seat) (Ford, 2003; Schep, 2002a). Additionally, minimizing psoas muscle contraction and hypertrophy is desired. To achieve this, cyclists using a clipless pedal setup are discouraged from pulling up during the upstroke (Ford, 2003; Schep, 2002a). These alterations while possibly palatable for the recreational cyclist are likely not realistic adaptations for an elite or professional cyclist. Likewise, discontinuation is typically not a practical remedy for a high-calibre cyclist who may be at or near the pinnacle of their career. In these cases, invasive procedures may be pursued.

Invasive procedures can be classified as either percutaneous/endoscopic or open. Percutaneous/endoscopic options include balloon angioplasty, stent insertion, or a combination of these procedures (Maree, 2007; Giannoukas, 2006; Ford, 2003; Wijesinghe, 2001). Two case reports utilizing balloon angioplasty for elite cyclists are evident in the literature (Giannoukas, 2006; Wijesinghe, 2001). Both cases, however, were met with transient success as symptoms returned within weeks following the procedure. Given this, not surprisingly, the use of angioplasty was not recommended as a primary intervention for cyclists with EIAE by a panel of experts (Hinchliffe, 2016).

There is a scarcity of information regarding the utilization of a stent for cyclists with EIAE (Sharifi, 2024; Maree, 2007). Recent work on the long-term outcomes (8.4 ± 3.3 years) in a small group (10 patients; 13 limbs) of athletes (3 cyclists, 5 runners, 2 triathletes) with EIAE treated with endovascular stenting (Sharifi, 2024) identified seven patients remained symptom free while recurrence was appreciated in three patients. The time to recurrence in the three patients occurred at an average of 9.3 ± 2.1 months following the procedure. It is unclear whether these three were cyclists, runners, triathletes or a combination of therein. The long-term results of this study are encouraging however it remains a small sample size with only 3 cyclists enrolled. Numerous researchers have raised apprehensions about utilizing a stent in this area due to potential migration or arterial dissection (Getzin, 2010; Bender, 2004). These concerns are heightened given the average number of revolutions per hour generated by a competitive cyclist while riding often for hours at a time.

Open surgical management has become the preferred method of intervention for elite cyclists as well as for those where EIAE has made a significant negative impact on the quality of life (Martinez, 2022; Hinchliffe, 2016). In 2012, Peach et al (Peach, 2012) devised an algorithm with 4 different types of surgical procedures (e.g. arterial release, vessel shortening, endofibrosectomy, interposition grafting) largely guided by vessel length and degree of stenosis. A similar type of treatment algorithm was recently proposed by Mazurova and colleagues in 2022 (Mazurova, 2022).

While more research is needed to assess the outcomes of open procedures, the data that is available is favourable (van Hooff M, 2023; Peake, 2018). Both short (<1.5 years) and long-term (>5 years) symptom reduction and overall satisfaction rate in a group of 68 cyclists diagnosed with EIAE was 91.2% or greater following endarterectomy with autologous patching (van Hooff M, 2023). Additionally, the workload at which point symptoms began and peak workload were enhanced as were improvements appreciated with the ABI and peak systolic velocity. Complications were few with a 30-day complication rate of 5.1% (van

Hooff M, 2023). Wu and colleagues reported 82% of patients (n=18) (all high-performance endurance athletes) were able to return to their prior level of exercise following surgical intervention tailored to the specific needs of the case (Wu, 2016). Similar results were noted by Politano and colleagues who identified that patients (n=20) at mid-term follow up (32 months) were all still cycling following external iliac artery reconstruction (Politano, 2012).

A consensus amongst panel experts (Hinchliffe, 2016) was not reached regarding the duration a patient should refrain from cycling following a surgical procedure. Fifty-four percent of the panel agreed that a 6–8-week period was most appropriate though 34% suggested return to cycling should be assessed on an individual basis. From a tissue healing perspective, it is reasonable that after a less complex procedure (e.g. release of the artery), to permit a more aggressive approach to rehabilitation compared to a more complex procedure (e.g. arterial reconstruction).

The evidence supports that conservative management strategies such as activity or bike-fit modifications may be reasonable for recreational athletes but are often unacceptable or ineffective for elite cyclists. Percutaneous interventions (e.g. angioplasty or stenting) have demonstrated limited durability and carry biomechanical risks in this population, making them suboptimal as first-line treatments. In contrast, open surgical procedures tailored to the underlying anatomic pathology (e.g. arterial release, endofibrosectomy, vessel shortening, or reconstruction) consistently demonstrate high rates of symptom resolution, favourable hemodynamic improvements, and a strong likelihood of return to pre-injury levels of competition with relatively low complication rates. Collectively, these findings emphasize that timely recognition, appropriate vascular referral, and selection of definitive surgical management when indicated are central to optimizing long-term outcomes for cyclists with EIAE.

7 Conclusions

External iliac arteriopathy is a rare condition which most frequently occurs in elite cyclists. To effectively diagnose this condition, clinicians must be aware of the way the disease presents. Athletes suspected to have external iliac arteriopathy should be screened with the ABI and referred to a vascular physician for further consultation. Conservative interventions as previously noted will largely consist of modifying the athlete's position on the bike (e.g., stem, seat adjustments). Should conservative intervention fail, a variety of invasive options are available.

Conflicts of Interest: The author declares no conflict of interest.

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