Exercise induced leg pain due to endofibrosis of external iliac artery

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Summary: Due to monotonous movement patterns, muscular hypertrophy, and increased cardiac output peripheral vasculature of athletes are subject to extreme stresses during athletic performance. Individuals suffering from exercise induced non-traumatic lower leg pain may display underlying vascular pathology such as external iliac artery endofibrosis. Therefore, it is essential in the course of to discriminate vascular from non-vascular findings and prime the correct diagnostic path within the course of clinical examination. In this regard, interdisciplinary thinking and profound knowledge in exercise-associated pathologies of the musculoskeletal, nervous and vascular system is indispensable. Consequently, provocation testing displays an indispensable diagnostic tool and has to be continued until symptoms occur, or complete exhaustion is attained. Finally, selective assessment of conservative and surgical treatment options, as well as its ethical evaluation, are of major importance in order to protect, preserve and promote the health and physical integrity of our patients who are keen to perform.

Keywords: endofibrosis, iliac artery, endurance athlete, sports medicine

Introduction

The exact identification of underlying pathology in competitive athletes suffering from exercise induced non-traumatic lower leg pain often represents an intellectual and technical challenge. In this regard, interdisciplinary thinking and profound knowledge in exercise-associated pathologies of the musculoskeletal, nervous and vascular system is indispensable. There is often a significant delay of diagnosis and treatment in terms of vascular pathologies, which might be devastating when it comes sports medical care of competitive athletes. Possible differential diagnosis of non-traumatic exercise-induced leg pain might contain pathologies of; muscles and tendons (tenosynovitis, metabolic or neoplastic myopathies, structural or ultrastructural muscle damage), bones (stress fracture, medial tibial periostalgia/shin splints, metabolic or neoplastic bone disease), nerves (nerve entrapment, neurogenic claudication), and vessels (chronic exertional compartment syndrome, atherosclerosis, vascular entrapment, aneurysms). The distinction between underlying musculoskeletal, nervous or vascular pathology in patients with non-traumatic leg pain is essential in order to prime the correct diagnostic and therapeutic pathway. Hence, the aim of this narrative review is to equip the reader with some basic knowledge about the special entity of external iliac artery endofibrosis

as a potential cause of vascular leg pain in athletes. As a result, the reader might develop a certain sensitiveness refer to the identification of this rare entity and may also get a comprehensive overview in terms of epidemiology, etiology, diagnosis and management of this pathology.

General evaluation of suspected vascular pathology in athletes

Detailed questioning about the development and quality of symptoms in order to identify vascular pain represents an indispensable necessity. Similar to neurologic lesions also vascular compromise may lead to pain, paresthesia or paralysis [1]. Consequently, basic vascular examination includes inspection of the affected limb and search for accompanied findings such as pallor, pulselessness and prolonged capillary refill [2, 3]. Furthermore, a sports medicine physician may also suspect vascular etiology if athletes complain about “increased fatigability” of a specific extremity. Decisive questions within a sport medical assessment regarding a possible vascular pathology are summarized in Table I and might be recalled by the notion “the fatiguing 7 P’s of vascular sports medicine”.

Table 1. *The fatiguing 7 P's in vascular sports medicine:* Questions and examination findings for identification of vascular pathology within sports medical consultation (modified from Brearley S. et al., BMJ 2013, [3])

Fatigue	Fatigibility	Does the patient complain about increased fatigibility of one leg while performing his/her sport?
P1	Pain	Is there an exercise inducible pain in the thigh/buttock/calf which may be aggravated/paralyzing near maximal effort?
P2	Pallor	Does one leg show paler skin compared to the other side?
P3	Perishing	Does one leg sense colder compare to the other side?
P4	Pulselessness	Can you palpate and count pedal pulses? Are they diminished in one leg or another?
P5	Paraesthesia	Does the patient suffer from reduced sensation or numbness in the affected leg?
P6	Paralysis	Is the patient able to move legs and feet?
P7	Prolonged capillary refill	Is there prolonged capillary refill (more than 2–3 seconds) after squeezing the toes?

Peripheral vessels of athletes are exposed to significant stress factors due to monotonous movement patterns, anatomic predisposition, muscular hypertrophy and increased cardiac output. The identification of an underlying vascular pathology is very difficult, because of its unremarkable examination findings at rest. It becomes even more challenging with respect to their low prevalence. With reference to the vascular system of the lower extremities, one can define several anatomic locations that predetermine for vascular damage due to excessive hemodynamic and biomechanical stress during training. As Akuthota et al. state “any athlete exposed to repetitive movements and rigorous training is susceptible to these disorders; however, there are sport-specific factors that predispose athletes to certain types of vascular injuries” [1]. As a matter of fact, endofibrosis of the external iliac artery represents a rare but important vascular pathology in endurance athletes.

Epidemiology of external iliac artery endofibrosis

In 1985 a couple of Swiss surgeons of the University Hospital in Lausanne published a case report in which they described an iliac endofibrosis in two cycling racers [4]. Subsequently, Chevalier et al. added a case series of seven competitive bicycle racers who had been surgically treated by endarterectomy and shortening of the artery due to suffering from intermittent claudication of one lower limb at “near maximal” exercise [5]. Histopathological findings in the cross-section of resected external iliac arteries confirmed endofibrosis with intimal hyperplasia [5]. Obviously, endofibrosis of the iliac artery causing intermittent claudication in young endurance athletes was gaining increased awareness within the last three decades. Although, reliable insight in the prevalence is insufficient. Therefore, 10–20% of elite road cyclists are estimated to suffer from hemodynamic relevant blood-flow restrictions in their legs due to either intravascular endofibrosis or functional kinking (which may result in endofibrosis) of iliac arteries [6]. According to Schep et al. about 90% of the intravascular lesions are identified in external iliac arteries [7]. Nevertheless, the pathology is not solely limited to cyclists. Approximately 75% of affected athletes

are cyclists, followed by speed skaters, triathletes, runners, and even soccer or rugby players [8–11]. Within a small retrospective study of 13 highly trained athletes Alimi et al. noted that mean age at onset of symptoms and mean age at diagnosis was significantly lower in professional athletes than in the amateur group (25.8 vs. 27.1 years and 43.6 and 44.6 years respectively, $p = 0.0001$) [12]. While scrutinizing characteristics of affected cyclists an average of 120,000 cycled kilometers (range 30,000–500,000) prior to onset of symptoms could be identified [12]. Nevertheless, with respect to formation of the lesion intensity of training seems to be as significant as overall distance cycled. Therefore, athletes cycling more than 14,500–20,000 km a year appear to be at higher risk of developing endofibrosis [12, 13].

Etiology and pathophysiology

The etiology of this entity is multifactorial. In the course of the initial publications, describing intraoperative findings of the disease Chevalier et al. suggested three factors that might contribute to these kinds of vascular lesions. First, in the arteriography arterial segments exhibited lengthening of external iliac arteries resulting in a so-called “kinking” which could be accentuated by hip flexion [5]. Second, a small nutrient collateral artery arising from the origin of the external iliac artery marked the upper limit of the lesion, and was suspected to induce a tethering of the vessel against the highly developed psoas muscle [5]. Third, the tethering/kinking in combination with a high cardiac output during training seemed to cause an increased stress to the vessel wall, which resulted in histologically validated intimal hyperplasia located on the side of greater curvature of arterial bend [5]. In 2003 Scavée et al suggested an additional explanation for the development of endofibrosis and their relationship with psoas hypertrophy in highly trained athletes. Based on findings in a 51-year old marathon runner, they stated that the iliac artery is also fixed by iliac bifurcation proximally and adheres to the surrounding inguinal ligament distally. As a result, its movement is limited. Thus, an enlarged psoas muscle may induce excessive stretch and medial elongation of the vessel [14].

Potential pathogenetic mechanism such as high blood flow volumes and increased blood pressure have already been suggested within the first publication of Walder et al. [4]. Furthermore, repeated arterial kinking and compression due to muscle hypertrophy may also cause elevated shear stress in the vessel wall. As a result, vascular smooth muscle cells respond directly (and indirectly) to these biomechanical variations by inducing proliferation, migration, and differentiation of contractor and secretory components in the vessel wall that finally lead to intimal hyperplasia [15, 16]. With reference to the “shear stress model”, it is essential to differentiate between the location of endofibrosis compared to the usual sites of atherosclerotic plaque formation. According to Cunningham and Gotlieb’s model of atherogenesis, atherosclerotic plaque formation takes place at a focal region of decreased hemodynamic shear stress around the small curvature of the vessel [16]. In contrast, Chevalier et al. identified sites of intimal hyperplasia and endofibrosis at the level of the vessel curve with greater radius [5] (Figure 1).

Vink et al. conducted a comparison of histopathological findings in endofibrosis of high-performance cyclists (age 29 ± 8 years) and atherosclerotic segments of the external iliac artery from elderly individuals (age 82 ± 10 years). Their observations suggest that involvement of inflammation in the development of endofibrosis is absent or limited. Correspondingly, lymphocytes were present in 21% of endofibrotic cases, but in 80% of atherosclerotic lesions [17]. Furthermore, macrophages could be identified in 16% of endofibrotic, and in 70% of atherosclerotic sites, respectively [17].

In a case series of 23 symptomatic cyclists, Rousselet et al. generally characterized cells in the affected zone of endofibrosis as subendothelial loose connective tissue labeled with anti-actin and anti-myosin antibodies. Furthermore, electron microscopy revealed features of synthetic smooth muscle cells [18]. Suitably, Chevalier et al. described their histological findings as follows: “The intima was richly cellular with myofibroblasts suggesting *medialization* of the intima” [5]. Posthuma et al. suggested that myofibroblasts and the accompanying collagen and elastin synthesis might be critical elements in the pathogenesis of endofibrosis. Furthermore, increased presence of protease-activated receptors (PARs), coagulation factor X or tissue factor (TF) suggests that protease-mediated cell signaling may also contribute to mechanisms in development of endofibrosis [19].

Endofibrosis appears to affect the external iliac artery in over 90% of patients with an involved vessel segment between 2–6 cm in length [7, 13]. Interestingly, there seems to be a predominance of left sided pathologies accounting for at least 68% of symptomatic limbs in men [9]. This fact is well described in several publications, whereas its pathophysiological correlate remains unclear [6, 13]. An association regarding the individual preference to a dominant or nondominant leg is conceivable [20]. However, 15% of patients reveal bilateral symptoms [21].

Unfortunately, within a professional and high-performance sports framework the question about possible association of endofibrosis and abuse of illicit performance-enhancing drugs is apparent. Although, there is a lack of randomized-controlled evidence-based information concerning vascular effects of doping, many theories

Figure 1. Distinction of location of vessel wall thickening due to either endofibrosis or atherosclerosis. Figures modified from (left side) Cunningham & Gotlieb et al., Laboratory Investigation, 2005 [16] and from (right side) Chevalier et al., Annals of Vascular Surgery, 1986 [5]).

remain hypothetical. Nevertheless, the authors of this review consider the topic of doping to be important in this context. As a matter of fact, anabolic-androgenic steroids (AAS) have multiple effects on the cardiovascular system [22]. Therefore, elevated erythropoiesis due to supraphysiologic testosterone application may lead to augmented blood viscosity [23]. In combination with steroid-induced psoas muscle hypertrophy shear stress to the iliac artery vessel wall might be accentuated. Appropriately, Khaira et al. documented a case of external iliac endofibrosis in a bodybuilder who ingested androgenic anabolic steroids [24]. Furthermore, assuming exercise induced micro-trauma to the vasculature, also systemic effects of the erythropoiesis inducing peptide-hormone erythropoietin (EPO) are debatable. Particularly, external iliac artery endofibrosis predominantly affects high-performance cyclists. Therefore, Reddy et al. reported induction of excessive neointima formation in injured carotid arteries of rats after intraperitoneal administration of EPO (5,000 IE/kg) [25]. Also in mice, EPO seems to accelerate smooth muscle cell-rich neointima formation [26]. Finally, the combination of EPO and AAS may promote the development of iliac artery endofibrosis.

Diagnostic procedures

Intermittent claudication due to iliac endofibrosis is a relatively rare condition within a specific cohort of athletes. Therefore, exclusion of potential orthopedic or neurological differential diagnoses such as physiologic exhaustion, (pseudo-) radicular causes or hip impingement is essential. Exceptionally, non-atherosclerotic, non-inflammatory vessel diseases like fibromuscular dysplasia may also take place in iliac arteries [27]. Nevertheless, prompt diagnosis of iliac endofibrosis represents a considerable challenge in sports medical practice, what in turn may trigger significant delay of treatment. Since symptoms occur at a maximum level of exertion, routine diagnostics might not be able to detect the disease adequately. Therefore, athletes suffering from non-traumatic leg pain while performing at their level of maximum performance may undergo systematic sports-specific and provocative vascular tests. In order to reliably recognize vascular causes of leg complaints in endurance athletes Schep et al. analyzed the diagnostic value of several test variables within a study population of 80 patients suffering from leg symptoms (pain, powerlessness, cramping) performing endurance sports for a minimum of 5 hours a week for at least 3 years [8]. An adapted summary of the diagnostic values and test variables analyzed by Schep et al. is pictured in Table II.

The authors illustrate that multivariate examination of patients is crucial in order to classify findings correctly into “vascular” and “non-vascular”. Furthermore, the authors state that 79% of cases could be identified when using all twelve significant test variables obtained from patient history and physical examination in a discriminant analysis [8]. 86% of cases were classified correctly by adding variables such as ankle brachial index and systolic ankle

pressure to the discriminate analysis test [8]. And, 91% of cases were classified correctly by using all 19 univariate significant test variables from the patient history, physical examination, exercise test and echo-Doppler examination [8].

For clinical practice, the rating of variables by Schep and colleagues is enormously important to keep in mind while examining an athlete, because neither orthopedic examination, nor neurological tests were able to discriminate vascular discomfort from non-vascular causes. Nevertheless, no single test variable revealed high sensitivity combined with a high specificity. However, these data show that a stepwise approach in the investigation of symptomatic athletes is crucial. In conclusion, focused questioning about the patient’s athletic history in combination with a deliberate examination of the hip and lower back, as well as auscultation of femoral bruits (with hip extended and flexed) identifies almost 80% of patients suffering from vascular causes of leg pain.

Currently, there are no guidelines concerning to optimal diagnosis and management of iliac endofibrosis. Consequently, the INSITE collaborators (INternational Study group for Identification and Treatment of Endofibrosis) published an agreement about the presenting symptoms and the investigations required to confirm (or exclude) the diagnosis of iliac endofibrosis based on a Delphi consensus methodology [28]. Thus, there was consensus (means $\geq 70\%$ of respondents were in agreement) that investigations such as clinical assessment, exercise testing and duplex ultrasound were required to confirm the diagnosis of iliac endofibrosis. However, experts did not agree on the need of CT-angiography, magnetic resonance angiography or angiography as required diagnostic tools [28].

In addition, the authors of this review recommend to further consider the following information about the use of different diagnostic tools:

Clinical assessment

INSITE-collaborators consented that leg weakness, thigh pain, and resolution of symptoms within 5 minutes of cessation of exercise represent hallmarks of iliac endofibrosis [28].

Exercise testing

INSITE-collaborators agreed that measuring pre- and post-exercise ankle pressure (ankle-brachial pressure index, ABPI) was the most appropriate way to confirm (or exclude) iliac endofibrosis. Furthermore, it seems to be essential to measure pressure within 1 minute of exercise cessation, and pressure drop of between 21 mmHg and 40 mmHg (in individuals with unilateral pain) or ABPI drop >0.3 was thought to indicate a positive test [28].

Duplex ultrasound

Velocity measurements ought to be performed in supine position and repeated with the hips and knees flexed 90 degrees in order to be able to detect kinking, which

Table II. Adapted summary of diagnostic value and test variables from Schep G et al. [8]

Test variable*	Vascular (n = 58)	Non-vascular (n = 34)	Sensitivity	Specificity
Patient history				
1. Disappearance of complaints <5 minutes of rest	97% (56)	71% (24)	0.97	0.29
2. Progression of complaints	79% (46)	47% (16)	0.79	0.53
3. Calf complaints	34% (20)	0% (0)	0.34	1.00
4. Adductor complaints	34% (20)	12% (4)	0.34	0.88
5. Hamstring complaints	64% (37)	44% (15)	0.64	0.56
6. Complaints in >3 muscle areas out of 6**	48% (28)	6% (2)	0.48	0.94
7. Relative (parent, uncle) under 60 years old with atherosclerotic disease	17% (10)	44% (15)	0.17	0.56
8. No backache	43% (25)	24% (8)	0.34	0.76
Physical examination				
9. Normal sacro-iliac joint	90% (52)	74% (25)	0.90	0.26
10. Normal back mobility	91% (53)	71% (24)	0.91	0.29
11. Femoral bruit with extended hip	36% (21)	6% (2)	0.36	0.94
12. Femoral bruit with flexed hip	76% (44)	35% (12)	0.76	0.65
Cycling test				
13. Ankle pressure <107 mmHg	53% (31)	15% (5)	0.53	0.85
14. Ankle Brachial Index <0.54	43% (23)	0% (0)	0.43	1.00
Doppler-Sonography examination (psv = peak systolic velocities in m/sec)				
15. PSV extended >1.48 m/sec	28% (15)	3% (1)	0.28	0.97
16. PSV flexed >1.70 m/sec	49% (26)	7% (3)	0.49	0.93
17. PSV exercise >3.18 m/sec	75% (31)	6% (2)	0.57	0.94
18. Kinking present	39% (21)	0% (0)	0.39	1.00
19. Lesion present	61% (33)	7% (2)	0.61	0.93

*All variables obtained above tested significantly different ($p < 0.05$) between vascular and non-vascular legs.

**Definition of muscle areas: anterior (Quadriceps), posterior (Hamstrings), medial (Adductors) and/or lateral part of upper leg, in the buttock, and/or in the calf.

represents a major cause of flow limitation [29]. Furthermore, symptomatic legs demonstrated significantly higher peak systolic velocities compared to their references [30]. Finally, color ultrasound is not able to detect the presence of psoas muscle arteries [12, 30].

CT-angiography (CTA)

In his review, Peach et al. concluded that CT-angiography is likely to become increasingly popular as CT resolution and 3D reconstruction improve. But it has not yet been shown to provide any diagnostic advantage over MRA combined with Duplex ultrasonography [13].

Magnetic-resonance angiography (MRA)

Schep and Bender stated that MRA appeared to be more sensitive than DSA with regard to visualization of lesions in the external iliac artery. In contrast, it seems to be less sensitive than duplex ultrasound [6].

Digital subtraction angiography (DSA)

According to Alimi et al. DSA has a high specificity (100%) and moderate sensitivity (57.1%) in order to identify psoas muscle arteries and long iliac external arteries [12].

In summary, timely detection of vascular syndromes in the lower extremities remains a major challenge. Therefore, Schep et al. established and validated a decision algorithm in order to classify symptomatic legs into vascular or non-vascular causes. Following this algorithm 87% of the

athletes could be categorized [29]. The authors of this review presumed to expand this algorithm with reference to an interdisciplinary approach, which is essential in a sports medicine setting and may also enhance diagnostic sensitivity (Figure 2).

Therapy and management

The decision about the optimal treatment modality in affected patients is quite delicate from various perspectives. First, athletes usually do not have symptoms while performing tasks of daily life, respectively not related to maximal exertion. Second, affected patients undergoing surgical vascular repair are usually less than 40 years old and probably influenced by a competitive environment consisting of several stakeholders. Third, there is scarce scientific ground about specific medical therapies or long-term outcomes in patients with iliac artery endofibrosis.

Initially, the exploitation of every conservative treatment option must be at the forefront of sports medicine care. Therefore, the duration and intensity of training need to be reduced. Maximal hip-flexion has to be avoided by raising the cycle handlebars or change saddle position. Finally, stimulation of psoas hypertrophy needs to be minimized by abandonment of click-pedals, which enable athletes to pull the pedals upwards. Feugier et al. also recommend the

Figure 2. Adapted diagnostic algorithm for detection of vascular pathologies in the athlete (Algorithm modified and adapted from Schep G et al. [29] and Peach G. et al. [13]).

ingestion of folate supplements [31]. Unfortunately, restrictions like this might be accepted within a cohort of leisure athletes, but they are not a long-term option for promising competitive young athletes. Consequently, competitive athletes who want to pursue their athletic career goals may favour surgical vascular repair. Corresponding to this, INSITE-collaborators agreed that medical therapies are

ineffective in the management of endofibrosis, and that surgery should be offered to anyone with severely impacted quality of life [28]. Furthermore, possible complications of endofibrosis such as thrombosis [32] might be avoided by early surgical intervention. In addition, Bender et al. consider that the disease process will progress in 80% of affected patients which might act as another argument

Table III. Proposed surgical strategy in the management of iliac endofibrosis by Peach et al. [13]

Surgical technique	Individual requirements
Arterial release	If stenosis is <15% AND arterial length ratio is NOT >1.25
Vessel shortening ± endofibrosectomy*	If arterial length ratio is >1.25
Endofibrosectomy + patch angioplasty	If presence of intravascular lesion is proven, but NO excessive vessel length co-exists
Interposition grafting	Alternative technique if endofibrosectomy with patch angioplasty is not feasible

*Endofibrosectomy = endarterectomy.

for surgery [33]. Finally, there was consent about the fact that endovascular interventions have no implication in patients with endofibrosis [28].

In fact, there is scarce evidence-based information concerning long-term outcomes following surgical intervention of endofibrotic iliac arteries. In accordance with the first publications by Walder and Chevalier in 1985, the authors suggested endarterectomy with venous patch angioplasty [4, 5]. In 2004, Feugier and Chevalier published long-term data of 334 athletes with a mean follow-up of 53 months after surgery. 237 patients (68%) underwent endofibrosectomy and arterial shortening, 110 patients (31%) received arterial bypass surgery with saphenous vein graft, and three patients (1%) were treated by percutaneous transluminal angioplasty. According to their investigation 332 athletes (99%) returned to sport with a mean delay of 2.84 months [31].

Considering the fact that surgery needs to be individually tailored according to the characteristics of underlying lesions, Peach et al. proposed a strategy for surgical management of endofibrosis [13]. In the course of this systematic review the authors focus on four surgical approaches (see Table III).

Additionally, Politano et al. concluded that inguinal ligament release alone does not appear to reduce the need for subsequent arterial intervention [34]. Furthermore, postoperative patient dissatisfaction was significantly higher for patients who received interposition graft [34].

With regard to postoperative management, INSITE-collaborators agreed on the administration of aspirin after surgery, whereas duration of treatment remains unclear. Annual clinical outcome assessment with non-invasive imaging may help to identify potential periinterventional complications such as pseudo-aneurysms greater than 3 cm subsequent to vein patch angioplasty [28]. Generally, overall complication rate seems to be as low as 3% [31]. In accordance to this, Bender et al. documented no detection of aneurysm formation in mid-term results (mean 15.6 months) within 33 legs of endurance athletes treated by endarterectomy with vein patch [35]. A retrospective case series of 63 patients (2011–2017) by Peake et al. reported one benign venous path dilatation (2 cm in diameter) out of 27 limbs treated with endarterectomy and vein patch angioplasty [9]. Finally, Politano et al. considered intimal hyperplasia as the most frequent complication necessitating reoperation [34].

In view of future perspectives, Howard et al. propagates the option of minimally invasive surgical repair of iliac artery fibrosis with endoscopic great saphenous vein

harvest and subsequent endarterectomy and vein patch of external iliac artery. Accordingly, a combined laparoscopic and endoscopic approach provides the following advantages: the opportunity to directly inspect the entire iliac system, potential faster recovery or return to sport by avoiding oblique abdominal incision, and reduced associated morbidity by vein harvesting through a single 2 cm above knee incision [36]. In the end, laparoscopic approach is only feasible in cases where plaque load is limited and does not extend into the common femoral artery [36].

Finally, within a sports medicine framework the vast majority of patients are represented by healthy young individuals. Hence, consideration of ethical reasoning in order to evaluate certain therapeutic (e.g. surgical) approaches gain in importance. Athletes affected by external iliac endofibrosis are young, manipulable, and susceptible to treatment options that focus primarily on short-term success in order to maximize their chance to participate within the next competition. In fact, the condition of external iliac endofibrosis usually does not restrict an individual's daily life, and is only symptomatic in the state of maximal exhaustion. Consequently, ethical assessment of treatment decisions may undergo an intensified evaluation in this context as feasibility of medical interventions in otherwise healthy individuals is almost unlimited, and therefore highly delicate. Nevertheless, becoming a professional athlete is not a human right. As physicians, we are obliged to protect, preserve and promote the health and physical integrity of our athletes. With reference to artificial performance enhancement Swedish philosopher Torbjörn Tännsjö stated the medical challenge as follows: "In (elite) sport we can test out the results of such enhancements and see, not where the limits are of the (given) human nature, but how far we can push them. And we can thank the athletes for taking the inconvenience to test them out (before us)" [37]. In this concern, several institutions including the Swiss Society of Sports Medicine developed an ethics charta which may serve as an infrastructure and bioethical guidance in cases like endofibrosis of external iliac artery [38].

Conclusions

Due to monotonous movement patterns, muscular hypertrophy, and increased cardiac output peripheral vasculature of athletes are subject to extreme stresses during

athletic performance. In this respect, a major challenge of vascular pathologies in the lower extremities of athletes is their timely detection. Exercise-induced leg pain represents an intersection of disciplines such as vascular medicine, orthopedic surgery and neurology. In the course of the clinical examination, it is essential to discriminate vascular from non-vascular findings and prime the correct diagnostic path. External iliac artery endofibrosis is a pathological entity of the vasculature that is rare, but important in sports medical practice. Admittedly, athletes represent a special cohort with very individual performance capacities and pain thresholds. Consequently, provocation testing displays an indispensable diagnostic tool and has to be continued until symptoms occur, or complete exhaustion is attained. Finally, selective assessment of conservative and surgical treatment options, as well as its ethical evaluation, are of major importance in order to protect, preserve and promote the health and physical integrity of our patients who are keen to perform.

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