Traumatic occlusion of the external iliac artery in a racing cyclist: a cause of ill defined leg pain

Alan J Taylor, William G Tennant, Mark E Batt, W Angus Wallace

Abstract

Stenosis of the external iliac artery in healthy athletes, although uncommon, has been reported in competition cyclists. A case of a racing cyclist whose chronic vague leg symptoms were incorrectly attributed to L4/5 nerve root irritation is reported. This highlights the importance of clinical vascular testing when assessing ill defined leg pain. The role of trauma as a causative factor in this condition has not been previously documented.

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Keywords: trauma; external iliac artery; endarterectomy; cycling

Stenosis of the external iliac artery (EIA) affecting competition cyclists was first reported by Walder *et al*¹ and Mosimann *et al*.² Since then less than 100 cases have appeared in the published literature. Increasing awareness of the condition on the continent has led to a recent proliferation of successful surgical procedures on professional cyclists (A Fotheringham, personal communication, 1996). Careful screening of athletes presenting with ill defined leg pain is required to identify the condition and to prevent delays in diagnosis or misdiagnosis.

Case report

A previously healthy 32 year old premier category racing cyclist presented complaining of a five year history of vague left leg pain, affecting the buttock, anterolateral thigh, calf and foot, together with a numb feeling in the middle three toes. Pain was worsened initially by cycling, swimming, walking, and more recently standing, sitting, and driving. The symptoms had been getting progressively worse since their onset. At the time of assessment the patient was unable to continue his work as an engineering fitter because of the pain experienced whilst standing, and had been unable to take part in competitive cycle racing for a number of months.

The patient was able to relate two specific incidents: (1) he was knocked from his cycle by a car in 1991 sustaining extensive bruising to his left buttock region and hindquarter; (2) a crash during competition later in the same year where he described "doing the splits" during the fall. The onset of the symptoms occurred soon after these two incidents.

Since that time, the patient had sought several specialist opinions, all of which had resulted in an unsubstantiated diagnosis of lumbar nerve root entrapment. Previous com-

puterised tomography and magnetic resonance imaging of the lumbosacral spine had demonstrated no significant abnormality.

Physical examination demonstrated full and unrestricted painless lumbar movement with no palpable tenderness proximally or distally. Muscle strength and lengths were normal, with no sign of muscle imbalance. Straight leg raise and slump test were normal with no sensorimotor deficit. Examination of the lower limb pulses revealed the left femoral pulse to be barely palpable, with absent distal pulses. A clinical diagnosis of vascular insufficiency with claudication pain was made.

Resting ankle brachial pressure index (ABPI) on the left side was 0.56 and 1.12 on the right (normal = 1.1). On treadmill testing, the patient developed claudication pain in the left calf at 40 metres, with a small reduction in post exercise ABPI. Lower limb arteriography demonstrated a short complete occlusion of the left EIA.

The patient underwent surgical vascular reconstruction. Operation was performed through a left retroperitoneal approach. The EIA was found to be of normal length, wall thickness, and diameter but was non pulsatile. There was a complete occlusion composed of both white and red thrombus densely adherent to the intima, typical of local trauma. There was no evidence of chronic atheromatous occlusive disease. Endarterectomy and vein patching were performed with immediate return of all distal pulses. The patient made a rapid recovery, was discharged five days after the operation and advised to refrain from training for three months.

Discussion

Arterial disease affecting racing cyclists has remained largely unreported in this country. Early in its course the patient usually complains of intermittent claudication of one limb, at a time of maximal strain—that is, climbing, sprinting, or time trialling, usually during competition or intensive training. Systolic ankle pressure may be normal at rest but markedly reduced after maximum exertion. Arteriography may show moderate stenosis (<40%) and sinuous lengthening.^{3,4} Pathological findings have included stenotic intimal thickening due to connective tissue.⁴

Certain predisposing anatomical conditions such as an arterial branch to psoas, the cramped position on the bicycle, high training mileage, and abnormal haemodynamics during exertion may lead to this condition.^{3,4} Mosimann *et al*² suggest that high cardiac output, arterial hypertension at maximal strain,

Department of
Orthopaedics, Queens
Medical Centre,
Nottingham NG7 2UH,
United Kingdom
A J Taylor
W G Tennant
M E Batt
W A Wallace

Correspondence to:
Mr Taylor.

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and local turbulence in the EIA, combined with strong thigh flexion and an abnormally sinuous section of artery, may be the cause of a slow progressive stenotic intimal thickening. The high incidence of falls or crashes at competition level cycling has not previously been considered as a predisposing factor.

This case was atypical in that complete rather than partial occlusion had occurred, so that the patient had begun to feel symptoms during normal activity and not just maximal exercise.

It appears from the pathological findings that, despite the transient nature of the early symptoms, trauma may have been a factor in the development of the symptoms. As far as we are aware this is the first report of ischaemia in an athlete caused by trauma connected with cycling.

Despite its rarity, awareness of this condition is essential to prevent delays in diagnosis. Our

case illustrates the importance of comprehensive evaluation of athletes. Vascular pathology must be considered when assessing patients complaining of vague unilateral leg pains during or after competition. Cyclists in particular may well be predisposed to stenosis or occlusion of the EIA.

- 1 Walder J, Mosimann F, Van Melle G, Mosimann R. A propos de l'endofibrose iliaque chez deux coureurs cyclistes. *Helv Chir Acta* 1984;51:793-5.
- 2 Mosimann R, Walder J, Van Melle G. Stenotic intimal thickening of the external iliac artery. Illness of competition cyclists. *Vascular Surgery* 1985;19:258-63.
- 3 Chevalier JM, Enon B, Walder J, Barral X, Pillet J, Megret A, et al. L'endofibrose iliaque externe du cycliste du competition. Une pathologie arterielle meconnue. *Annales de Chirurgie Vasculaire* 1986;1:297-303.
- 4 Rousselet MC, Saint-Andre JP, L'Hoste P, Enon B, Megret A, Chevalier JM. Stenotic intimal thickening of the external iliac artery in competition cyclists. *Hum Pathol* 1990;21:524-9.

Dynamic obstruction of the external iliac artery in endurance athletes and its relationship to endothelial function: the case of a long distance runner

Isabel A Wright, Neil D Pugh, Jonathan Goodfellow, Andrew M Wood, Ian F Lane, Alan G Fraser

Department of
Bioengineering,
University Hospital of
Wales, Heath Park,
Cardiff CF4 4XW,
United Kingdom
I A Wright
N D Pugh

Department of
Cardiology, University
Hospital of Wales,
Heath Park, Cardiff
CF4 4XW, United
Kingdom
J Goodfellow
A G Fraser

Department of
Radiology, University
Hospital of Wales,
Heath Park, Cardiff
CF4 4XW, United
Kingdom
A M Wood

Department of
Surgery, University
Hospital of Wales,
Heath Park, Cardiff
CF4 4XW, United
Kingdom
I F Lane

Correspondence to:
Miss I A Wright,
Department of Medical
Physics and Bioengineering,
University Hospital of Wales,
Heath Park, Cardiff CF4
4XW, United Kingdom.

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Abstract

There have been recent reports of exercise induced claudication in endurance trained athletes attributed to narrowing of the external iliac artery. Most patients have been competitive cyclists, and intimal hyperplasia has been cited as the cause. The case is reported here of a long distance runner who presented with similar symptoms.

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Keywords: external iliac artery; long distance runner; diabetes mellitus

Since the early 1980s there have been several reports documenting stenoses of the external iliac artery (EIA) in competitive cyclists.¹⁻³ They describe how intimal hyperplasia/endofibrosis produces a narrowing of the EIA, leading to lower limb claudication at maximal effort. It is thought that intimal hyperplasia may be produced in response to repetitive arterial injury. Endarterectomy and vein patch angioplasty have been used to treat some cases.²

Here we describe a long distance runner who presented with similar symptoms, apparently caused by arterial spasm.

Case report

A 39 year old female marathon runner presented with an 18 month history of acute pain in her right thigh and calf after running approximately 200 yards. She was a normotensive insulin dependent diabetic (of 18 months) and an ex-smoker (20 per day) from 12 years previously. Clinical examination was normal and there was no family history of cardiovascular disease.

Magnetic resonance imaging showed no evidence of lumbar canal stenosis or popliteal entrapment.

Resting Doppler studies showed a normal ankle/brachial systolic pressure index (ABPI) of 1.00 on the right and 1.06 on the left, with triphasic ankle waveforms bilaterally. There were raised velocities in the right common femoral artery (CFA) with a diffuse narrowing of the EIA as compared with the left on Duplex scan; however, this was not haemodynamically significant. Otherwise the lower limb vasculature appeared entirely normal.