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REVIEW ARTICKLE

Posterior ankle impingement syndrome : A clinical review with reference to horizontal jump athletes

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Study carried out within United Kingdom Athletics

Posterior ankle impingement syndrome is the term attributed to the clinical disorder characterized by posterior ankle pain that occurs in forced plantar flexion. In this review article, we discuss the anatomy, aetiology, clinical and radiological features of posterior ankle impingement syndrome and consider the conservative and surgical management and the guidelines used for post operative rehabilitation. Finally we provide illustrative cases and review the biomechanical features of this condition in horizontal jump athletes.

Keywords : PAIS ; impingement ; ankle ; long jump.

INTRODUCTION

Posterior ankle impingement syndrome (PAIS) is the term attributed to the clinical disorder characterized by posterior ankle pain that occurs in forced plantar flexion (*11*). This clinical syndrome is well recognised in ballet dancers (*6*), gymnasts, footballers (*3,14*) and cricketers but has not been previously described in horizontal jumpers. There can be a number of anatomical and causative factors involved in PAIS, and it is often difficult for the clinician to identify the exact source of the symptoms. As a result, accurate diagnosis can be difficult and lead to long delays for the athlete from initial presentation to final diagnosis. It has long been known that an accessory ossicle known as the os trigonum, and other anatomical variants in the posterior ankle region can give rise to symptoms and that excision may be necessary for the relief of symptoms (*10*). This paper reviews the current understanding of PAIS along with radiological and management considerations, with specific reference to six cases of PAIS in elite horizontal jump athletes.

ANATOMY OF THE POSTERIOR PROCESS OF THE TALUS

The anatomy of the posterior process of the talus and, in particular, the origin of the os trigonum has been subject to some debate over the past 150 years. When the lateral tubercle of the posterior process of

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the talus projects excessively it is known as Stieda's process, after Ludwig Stieda, a German anatomist who published the paper 'Ueber secundäre Fusswurzelknochen' (about secondary tarsal bones) (*16*) in 1869. He (correctly) believed there to be a secondary ossification centre of the talus posterior to the lateral tubercle.

In 1882, Shepherd (*15*) reported 3 cases of what he considered to be fractures of the posterior process of the talus. He believed the ossicle he observed was due to a fracture of the lateral tubercle of the posterior process of the talus (Stieda's process). In contrast to Stieda, Shepherd felt the talus developed from a single ossification centre. Shepherd's fracture is still used by some authors to describe an avulsion fracture of the lateral tubercle following ankle inversion resulting from traction on the posterior talofibular ligament (*1*).

In 1933, Burman and Lapidus observed a separate ossicle in 64 of 1000 radiographs of feet and an elongated lateral tubercle (Stieda's process) in 429 of the 1000 radiographs (*4*). By 1955, McDougall (*10*) had produced radiographic evidence to support Stieda's notion that the posterior process of the talus develops from a secondary centre of ossification. He also suggested that in an active individual, repeated impingement of the tubercle against the tibia will lead first to a groove at the point of contact and eventually to separation of the tubercle from the body of the talus. It is currently accepted that the os trigonum is an accessory bone located directly posterior to the lateral tubercle that arises from a secondary ossification centre of the talus, between the ages of 8 and 11 years. In most children, it fuses to the lateral tubercle within one year of its appearance but in up to14% may persist as a separate ossicle (*4*), attached to the talus by a cartilaginous synchrondosis (*7*).

AETIOLOGY OF PAIS

The literature is conflicting as to what causative factors and pathology constitute PAIS. By contrast with some authors who include acute injuries as a cause for PAIS, we believe PAIS is a chronic injury with posterior ankle pain that is reproduced in plantar flexion of the ankle. Numerous terms have previously been used to describe this syndrome, including soft tissue impingement, bony impingement, posterior block of the ankle, posterior triangle pain, talar compression syndrome, os trigonum syndrome, os trigonum impingement, posterior tibiotalar impingement syndrome and nutcracker type syndrome (*14*). In addition, posterior ankle pain may have several alternative causes (table I) which need to be excluded prior to diagnosing PAIS.

Table I.
Differential Diagnoses for Posterior Ankle Pain (*2,13*)

Achilles tendon pathology
Retrocalcaneal bursitis
Sever's disease in adolescents
Accessory soleus muscle
Referred pain from lumbar spine
Peroneal tendon subluxation
Ankle osteoarthritis
Tarsal tunnel syndrome
Haglund's deformity
Tarsal coalition
Inflammatory arthropathies
Calcaneal stress fracture
Tibialis posterior tendinopathy
Flexor Hallucis Longus (FHL) tendinopathy
Medial calcaneal nerve entrapment

Posterior ankle impingement syndrome results from the repeated abutment of the posterior process of the talus or Os Trigonum between the tibia and calcaneus during plantar flexion of the ankle. This results in localised soft tissue inflammation and progressive bony damage to either the posterior process or the Os Trigonum. Additionally, in the presence of an Os Trigonum there may be irritation of the synchondrosis. The development of secondary inflammatory tissue may then perpetuate the cycle of impingement and inflammation. In addition to these anatomical variations, generalised hypermobility or previous disruption to the anterior talo-fibular ligament and subsequent anterior talocrural laxity may predispose some athletes to PAIS.

CLINICAL FEATURES OF PAIS

Typically, patients describe pain localized to the posterior aspect of the ankle, which is aggravated by forced plantar flexion activities e.g. dancing en pointe position, kicking, downhill running, walking in high heel shoes (*14*) and in our experience the take-off phase of horizontal jumping.

On examination, athletes may exhibit tenderness on palpation in the region of the posterior ankle joint and most posterior aspect of the subtalar joint. Posterior impingement testing, whereby pain is reproduced on passive plantar flexion of the ankle is invariably positive. This is best assessed with the patient in a relaxed prone position (fig 1a). Involvement of the FHL tendon may be suspected if resisted isometric plantar flexion of the first MTPJ results in posterior ankle pain while in this position (fig 1b). Further localization of symptomatology may be obtained by injecting a small volume of local anaesthetic (preferably under image guidance) around the posterior talus, with a subsequent negative passive plantar flexion test supporting the diagnosis (*2*).

RADIOLOGICAL FEATURES OF PAIS

Usually, plain radiography is the initial imaging option for posterior ankle impingement. Lateral radiographs obtained with the foot in plantar flexion may show the presence of a Stieda process or os trigonum (fig 2). It may be difficult to differentiate between an os trigonum and a fractured lateral tubercle on lateral radiographs. Usually, an os trigonum is round or oval with well-defined corticated margins, whereas a fractured lateral tubercle has irregular serrated margins. In difficult cases, CT with its high spatial resolution may be helpful in evaluating the osseous structures around the ankle. Technetium 99m diphosphonate bone scans shows increased blood flow and active bone repair.

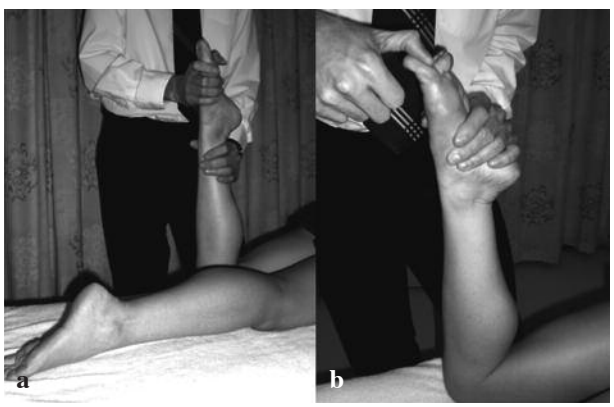

***Fig. 1.* —** Posterior ankle Impingement test - (a) Passive plantar flexion of the left ankle (with overpressure) with patient in relaxed prone position (b) Resisted isometric plantar flexion of first MTPJ to test for FHL involvement.

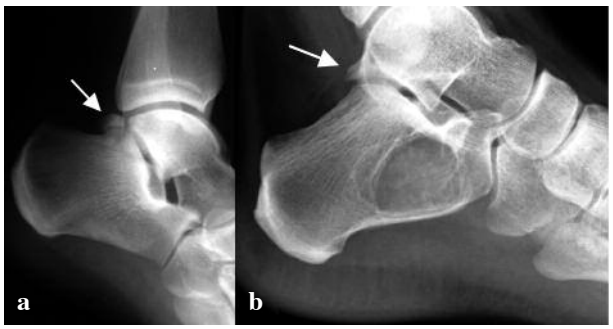

***Fig. 2.* —** Lateral radiographs in posterior Impingement - (a) 'Os trigonum' located at the posterior aspect of the tibiotalar joint (b) 'Stieda process' projecting from the talus.

MR imaging has convincingly replaced all the other modalities for the accurate delineation of the soft tissue and osseous structures involved in posterior ankle impingement (*3,13*). It can demonstrate occult bony abnormalities along the posterior aspect of ankle joint and is well suited for the assessment of soft tissue structures including the posterior synovial recess of subtalar and tibiotalar joints and the FHL tendon sheath. The soft tissues compressed in posterior ankle impingement include the posterior talofibular ligament, transverse tibiofibular ligament, posterior tibiofibular ligament and the tibiotalar capsule (*14*).

MR imaging depicts the low-signal intensity of the os trigonum and/or lateral talar tubercle on T1-weighted images and high signal intensity of these structures on fat-suppressed T2-weighted images, consistent with bone marrow oedema (fig 3).

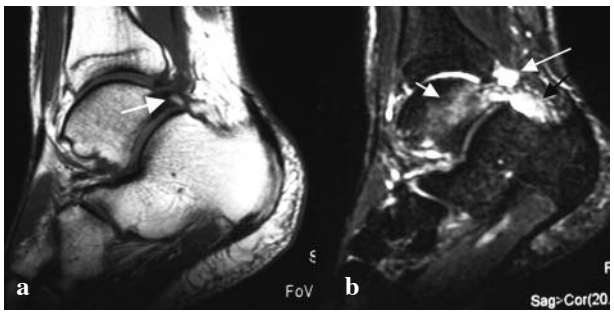

***Fig. 3.* —** MRI Posterior Ankle Impingement – (a) Sagittal T1-weighted image showing the typical position of Os trigonum, an anatomical variant that may predispose to the posterior ankle impingement. (b) Sagittal STIR image depicting the typical findings of PAIS including oedematous os trigonum (long white arrow), marrow oedema in the talus (short white arrow) and increased signal within thickened soft tissues indicating posterior synovitis at the posterior aspect of the subtalar joint.

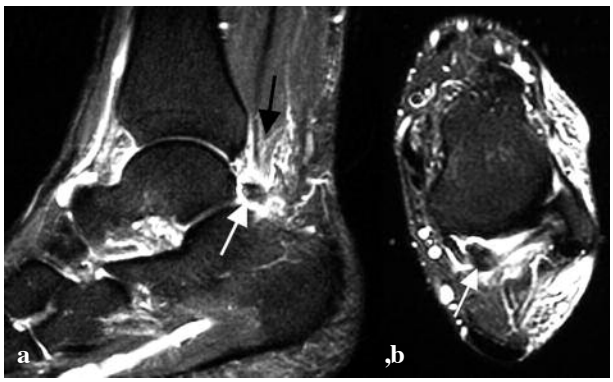

***Fig. 4.* —** MRI showing flexor hallucis longus abnormality in PAIS - (a) Sagittal STIR and (b) Axial T2-weighted images showing os trigonum with surrounding inflammation and fluid in the posterior synovial recess indicating synovitis. This inflammation extends along the flexor hallucis longus (FHL) tendon (black arrow).

These structural abnormalities are best appreciated on the sagittal MR images. In addition, MRI may demonstrate findings including increased signal intensity and distension of the posterior capsule indicating presence of fluid or synovitis. An increased amount of synovial fluid may also be seen extending within the FHL tendon sheath (fig 4). MR imaging also helps to evaluate the intermalleolar ligament which has been described as a cause of posterior ankle impingement in professional soccer players (*5*) and ballet dancers. MRI typically demonstrates a thickened, hypo-intense structure in the posterior talofibular space.

MANAGEMENT OF PAIS

1. Conservative

PAIS can be managed conservatively with relative rest, bracing or taping to limit plantar flexion, manual mobilisation of the subtalar, talocrural and midfoot joints, anti-inflammatory medication and electrotherapeutic modalities. Technique assessment is important to determine any causative factors. If the condition persists a corticosteroid injection around the area of maximal tenderness may reduce pain and inflammation. Ultrasound is useful in this condition for targeted injection of local anaesthetic and steroid into the synovial thickening or the os trigonum synchondrosis. Although temporary, the effect is particularly helpful in professional athletes to delay surgery (*12*). However, the presence of a long posterior process or an Os Trigonum may preclude a positive response to conservative management and surgical excision is required.

2. Surgical

Surgery for posterior impingement of the ankle can be undertaken by open (*8*) or endoscopic techniques (*17*). Open techniques utilise either an antero-medial or antero-lateral approach. Whilst acknowledging potential advantages from an endoscopic procedure in terms of size of incision, the open antero-medial approach allows direct visualisation, mobilisation and protection of the posterior tibial nerve and vessels, full exposure of FHL and the capacity to release its fibro-osseous tunnel as far distally and medially as the sustentaculum tali. The posterior osseous structures are easily identified and excised as required. Potential damage to the sural nerve from an anterolateral approach is avoided. The procedures are performed under general anaesthesia with tourniquet control and the procedure can be undertaken through a 4 cm incision. All procedures are per-

formed under fluoroscopic control to ensure full osseous decompression and full ankle plantarflexion at the end of the procedure. A mini-vac drain is used post-operatively and patients remain in hospital overnight. Immediate active ankle mobilisation is encouraged although full weight-bearing is avoided for 2 weeks until the wound is healed. From 2-6 weeks progressive weight-bearing and ankle mobilisation is undertaken supplemented by hydrotherapy and aqua-jogging. From 6 weeks, according to progress and comfort, a return to land-based impact activities is allowed. Guidelines for post operative rehabilitation are outlined in table II.

Table II. - Guidelines for Post Operative Rehabilitation

Phase	**Goal of phase**	**Time post surgery**	**Physiotherapy treatment**	**Exercise program**	**Functional / sport related activity**
Phase 1	PWB – FWB Eliminate swelling Scar resolution	0-2 weeks	Cryotherapy Electrotherapy Compression Manual therapy Gait re-education	Gentle active exercises Low grade proprioception prior to walking drills	None
Phase 2	No swelling Aim to have DF normalised prior to starting walking drills Good balance and control Unrestricted walking	2-6 weeks	Cryotherapy Electrotherapy Compression Manual therapy – start neural mobilisations Mobilisation TC / STJ/ Mid-foot /1st MTP Gait re-education Exercise progression	Increased active and passive movements – PF/ DF/INV/EVERS Single leg calf raises/ Intrinsics +/- ther band Core/Gluteal activation, strength work Balance and proprioceptive drills single leg	Non-impact fitnessStationary bike Pool work
Phase 3	Close to full ROM Full strength and power Return to jogging	6-10 weeks	As above	Progression of walking drills over 2-3 weeks Sand walking Low level sports specific strengthening Low grade running drills	Stationary bike Walking Straight line jogging
Phase 4	Full ROM Full strength and power Return to restricted sports specific drills	10-14 weeks	Manual therapy Supervised exercise with progression/modification of activities as appropriate	Start running Easy technical running on grass event specific Low grade lateral and multidirectional stepping Low grade plyometrics – bouncing NOT bounding progress over 2 weeks Start jumping drills	Progressing to sport specific running and agility (progressively sequenced) Plyometrics Jumping
Phase 5	Return to full training/competition	14 weeks +	As above	Run / jump drills revisiting technique	Unrestricted training / competition

DF = Dorsi-flexion, INVERS = inversion, EVERS = eversion, FWB = full weight bearing, MTP = metatarsophalangeal joint, PF = plantar flexion, PWB = partial weight bearing, ROM = Range of Motion, TC = talocrural joint, STJ = subtalar joint.

CASE SERIES

All five athletes were international standard horizontal jump athletes and presented between July 2003 and August 2007. One athlete re-presented with identical symptoms on the contra-lateral side 26 months after the first ankle. Each athlete opted for surgery after a brief trial of conservative management with all 6 procedures undertaken by one surgeon (WJR) using the anteromedial open incision described above. There were no significant complications and all returned to full training. A summary of the series is presented in table III.

Table III. – Case Series

Age at Surgery (years)	18	25	27	22	16	19
Sex	M	M	M	M	F	M
Event(s)	LJ	LJ	LJ	TJ	LJ Heptathlon	LJ Decathlon
Duration of symptoms prior to surgery (months)	3	2	1.5	4	8	3
Side affected	Right	Left	Right	Left	Right	Left
Take Off Leg	Right	Left	Left	Left	Right	Left
Pathology/imaging results	Large oedematous os trigonum fused to talus	Long posterior process of talus (Stieda's Process)	Moderate posterior process with FHL tendinopathy	Large os trigonum	Small os trigonum	Oedematous posterior process of talus
Time to return to competitive action-months	4	4	4	3	4	4
Residual symptoms/signs at final follow-up	Lost 3-5 degrees plantar flexion	Nil	Nil	Nil	Nil	Nil

LJ = long jump, TJ = triple jump.

BIOMECHANICAL FEATURES OF THE HORIZONTAL JUMP AND PAIS

The primary aim of the run up in horizontal jumps is to attain the fastest controllable take-off velocity at the board. During the take-off phase, the athlete is required to sacrifice horizontal velocity in order to generate the necessary vertical velocity, to allow sufficient flight time and create the horizontal distance. Numerous postural adjustments affect an athlete's ability to gain vertical and minimise the horizontal velocity loss at take off. At initial contact with board, the take off leg is placed in front of the body, with a typical whole foot or rear foot strike, the dorsiflexed ankle undergoes a rapid compressive phase in which rapid plantar flexion occurs over 40-70ms. During this compressive phase the whole body absorbs the forces (up to 10-15 times body weight) generated by the extended lead leg and attempts to transfer horizontal velocity into vertical velocity in order to gain the flight time and horizontal distance. The further the lead leg is in front of the body's centre of mass (fig 5), the greater the braking impulse exerted on the leg and thus the body. Although techniques vary amongst elite horizontal jumpers on the board, it is thought that a lead leg action that is seen to be moving in the opposite direction to the motion of travel of the body (backwards towards the ground) will reduce these braking forces. However, if the lead leg is left to passively contact the ground in front of the body, without such a movement backwards, the resultant is a huge blocking action which inevitably causes

***Fig. 5.* —** Take off phase of horizontal jump illustrating plantar flexion of the ankle joint during the compressive phase with the position of the centre of mass highlighted.

increased forces transmitted through the ankle, lower leg and the body. We believe that it is this repetitive rapid and high force plantar flexion in the take-off phase which predisposes to PAIS. The combination of increasing the angle of leg plant, no deliberate action backwards of the lead leg and the distance the lead leg strikes the ground in front of the body will all contribute to increased loading and risk for the athlete.

DISCUSSION

Posterior ankle impingement syndrome has been well recognised in certain sports such as gymnastics, football and in dance arts such as ballet. Repetitive plantar flexion in combination with anatomical variants such as a long posterior process or Os Trigonum may predispose to its development. We believe that horizontal jumping has a high risk for PAIS due to the high speed plantar flexion and high forces involved during the take-off stride. A posterior centre of gravity at take-off may increase the risk of impingement and this should be assessed early in the process of evaluation. Early diagnosis is aided by a high index of suspicion, positive impingement signs and early imaging with lateral radiographs and MRI. This combination of imaging provides an optimal awareness of the bony and soft tissue factors involved in the condition. While conservative management can be successful, those athletes with a long posterior process or Os Trigonum should be considered for surgery at an early stage. In those athletes with anatomical predispositions, conservative management may be successful in managing the pain during the season, however definitive management is surgical. All the athletes in this case series had surgery and returned to competitive action within 4 months with minimal residual symptoms or signs.

An understanding of both the anatomy of the posterior ankle and the biomechanics of the horizontal jump take-off are vital if a practitioner is to understand the aetiological factors involved in the development of PAIS in this group of athletes. We believe that this is a common condition which can be adequately treated when properly identified.

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