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Abstract Background: The inferior calcaneal spur has long been associated with inferior heel pain. Traditionally, many physicians have believed the spur to be associated with the plantar aponeurosis, though more recently others report the spur to lie within the intrinsic musculature of the plantar rear foot. While previous anatomical studies have generally involved cadaveric specimens, there is a relative paucity of research which has specifically analysed the spur of surgical samples from symptomatic heels. Objectives: This study was carried out to investigate the nature, affiliation and histopathology of the heel spur and the tissue in which the spur resides. Methods Heel spur samples were taken from five healthy participants undergoing open heel surgery for recalcitrant inferior heel pain involving the presence of a heel spur. Results: The inferior calcaneal spur was found to lie consistently within the origin of the intrinsic musculature of the first layer of the plantar aspect of the foot. The plantar aponeurosis proper was observed to course inferior to the heel spur and had attachment to the spur along the spur's inferior margin. Microscopically, the entheses of the intrinsic musculature of the first layer and the plantar aponeurosis were amalgamated to form a single broad origin of the inferior medial calcaneal tubercle. The spurs lacked a cortical shell and endochondral ossification was a prominent feature. Overt fracture of the heel spur itself was a common finding. Conclusions: Observations largely suggest a weight-bearing compressive disturbance, with secondary traction of the respective enthesis–bone unit as the underlying cause of spur growth. In this series, spurs were noted to manifest by means of endochondral ossification.

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The Inferior Calcaneal Spur

A Review of the Literature

The inferior calcaneal or heel spur defines the osseous outgrowth that projects anteriorly from, and across the entire surface of, the medial tubercle of the calcaneus (DuVries 1957; Hauser 1950; Hiss 1949; Steindler & Smith 1938). It manifests in a horizontal manner and tapers anteriorly along the same course as the plantar fascia (McCarthy & Gorecki 1979). The heel spur, with the implementation of radiography, was first noted by Plettner in 1900 (Steindler & Smith 1938) and for decades its presence was often the exclusive focus for medical and allied health practitioners when confronted with patients suffering inferior heel pain, often resulting its dramatic surgical removal (Wachter & Sonnenschein 1915). The heel spur has long been discussed and investigated in association with insertional plantar fasciitis. It is often considered a concomitant feature of this common foot pathology (McCarthy & Gorecki 1979), wherein both are described as a product of traction at the origin of the plantar fascia itself and referred synonymously by the term "heel spur syndrome". This review of the literature will focus primarily on the spur itself, however, as the two entities are inherently investigated together, where appropriate, insertional plantar fasciitis will be covered.

ETIOLOGY

The inferior calcaneal spur has been affiliated with a multitude of pathologies. Even hereditary factors have been implicated in the genesis of the spur (Gould 1942). In the early 20th century, heel spurs were believed to be a manifestation of gonorrheal infection, syphilis etiology and arteriosclerosis (Baer 1906; Miltner & Chang 1934; Swett & Stoll 1916). However, beginning with Von Lackum and Palomoque (1930) and Blokhin and Vinogradova (1937), it is generally considered these pathologies play no role in the etiology of the inferior calcaneal spur.

The association of plantar spurs with more widespread systemic conditions, such as Marie-Strümpell disease, psoriatic arthritis, rheumatoid arthritis, spondylitis, lupus, gout and diffuse idiopathic hyperostosis (DISH) syndrome is well documented (Chang & Miltner 1934; Davis & Blair 1950; Furey 1975; Lapidus & Guidotti 1965; Rubin & Witten 1963). While inferior calcaneal spurs are

often a clinical finding of DISH and systemic arthropathies, spurs related to these pathologies are not the focus of this study and will not be discussed further.

Increased weight, associated with plantar fasciitis and spur formation, has been reported by a number of authors (DuVries 1957; Fuller 2000; Furey 1975, Lapidus & Guidotti 1965; Lester & Buchanan 1984; O'Brien & Martin 1985; Rubin & Witten 1963; Shikoff, Figura & Postar 1986; Tountas & Fournasier 1996). Hill and Cutting (1989) in a study of heel pain and body weight involving 77 cases found 84.4% had weights above the 50th percentile of standardized tables. Of their subjects, 41 patients were reviewed radiographically and 70.7% demonstrated heel spur formation – of these, 82.2% had weights greater than the 50th percentile. Snook and Chrisman (1972) discovered one half of their study of 27 patients with heel pain to be overweight and 13 of the 27 demonstrated plantar spurs, although they did not relate what proportion of those overweight subjects' had a spur. Excessive weight was a common finding (67%) in a series of operative treatment for subcalcaneal pain on 21 patients, 16 of whom had heel spurs present (Tountas & Fornasier 1996).

According to Rubin and Witten (1963), the most important factor influencing the occurrence of spurs, is age. In their study of the 125 patients with heel spur, 98.4% were over 40. They concluded that some unknown factor related to the aging process is an essential pre-requisite. A number of other authors support this trend, noting a higher proportion of patients with heel pain and spur formation are over 40 years of age, (DuVries 1957; Furey 1975; Hill & Cutting 1989; Lapidus & Guidotti 1965; Shikoff et al. 1986).

Another area that has received attention relating to heel pain and spur formation is occupations involving prolonged weight bearing. Of the patients with heel spur (125) in Rubin and Witten's (1963) study, 66% stood most of the time at work. In Lapidus and Guidotti's (1965) study, the predominating occupations of those patients suffering inferior heel pain and calcaneal spurs were those requiring continual standing and walking. Tountas and Fornasier (1996) concluded, albeit vaguely: "all (21 patients) presented with pain under the heel triggered by weight bearing", (Tountas & Fornasier 1996), p. 171).

Of the postulations and literature regarding inferior heel spur formation and heel pain, there is general agreement that the fundamental cause is repetitive stress and tension of the soft tissue structures that insert directly into medial calcaneal tubercle (DuVries 1965; Fishco, Goecker & Schwartz 2000; Fuller 2000; Gormley & Kuwada 1992; Hauser 1939; Hiss 1949; Karr 1994; Kwong, Kay, Voner & White 1988; Leach & Gorzyca 1991; LeMelle, Kisilewicz & Janis 1990; Lester & Buchanan 1984; Lewin 1959; Mann 1978; McCarthy & Gorecki 1979; Schepisis, Forman & Green 1990; Snider, Clancy & McBeath 1983; Snook & Chrisman 1972; Rosenfeld 1985).

Fuller (2000), Hiss (1949) and Hauser (1939, 1950) suppose that it is frequently seen in both pes plano valgus and cavus deformity, wherein both pathologies place excessive strain on the plantar aponeurosis at its origin at the medial calcaneal tubercle. Indeed, Shama, Kominsky and Lemont (1983) reviewed 1000 weight-bearing radiographs and demonstrated a statistically significant relationship between heel spur presence and postural foot pronation – of 132 subjects with heel spur, 62% demonstrated a pronated foot posture; and of those with painful spurs, 81% were deemed to pronate, (as measured by anterior break in cyma line and the long axis of the talus falling below the long axis of the 1st metatarsal on lateral weight bearing radiographs).

Following the logic that traction influences the formation of inferior calcaneal spurs, it seems reasonable that each obesity, prolonged weight bearing and certain foot postures/ pathologies that place a greater stress on the plantar fascia and intrinsic musculature and, by virtue of their attachments to the medial calcaneal tubercle, likely result in spur formation. Furthermore, considering the daily repetitive weight bearing that generally occurs over the course of many years – as the foot is repeatedly loaded and the plantar structures placed on rudimentary tension – the supposition that spurs are more prevalent with age, appears sound.

Of academic and clinical importance, is the observation by some authors – and the general imperative of the literature – is the belief that a heel spur noted on radiographic evaluation is an incidental finding and not the source of the pain *per se* (DuVries 1965; Lapidus & Guidotti 1965; Mann 1986; Snook & Chrisman 1972). Only 10% of 125 non-arthritic heels with calcaneal spurs were found to be symptomatic in Rubin and Witten's (1963) large series of subjects. Tanz (1963) reviewed 100 radiographs of patients with asymptomatic heels and found a 16% incidence of heel spur, although the author did not note whether there was a previous history of heel pain in this cohort. In another series of 29 patients with heel pain, Tanz (1963) reports that approximately only 50% had spurs. Shama, Kominsky and Lemont (1983) reviewed 1000 random patient radiographs and found the incidence of heel spur to be 13.2% and of these, 61% were not painful.

What these studies fail to report is **(1)** the actual size of the spur *related* to symptomology, and **(2)** whether there is a previous history of heel pain symptomology and the presence of the spur in a 'presently' asymptomatic subject. Regarding the actual size of the heel spur, although Baxter and Thigpen (1984) previously developed a classification for heel spur length (small: 2-5 mm and large: ≥ 6 mm); a more recent study of heel fat pad thickness and elasticity on heel pain, further segregated spur length into small (1-2mm), medium (3-5mm) and large (≥ 6 mm), (Özdemir, Söyüncü, Özgörge & Dabak 2004) – (see Fig 1). While not statistically significant, the author's believed that larger heel spurs were associated with a decrease in heel pad elasticity and *increase* in length of heel pain symptoms.

Figure 1. Measurement of subcalcaneal spur length. **A-B** indicates the lowest part of the medial calcaneal tuberosity; **C-D**, the lowest point of the subcalcaneal spur; and **п-Ø**, the length of the subcalcaneal spur. From: Özdemir, H., Söyüncü, Y., Özgörge, M. & Dabak, K. (2004) Effects of Changes in Heel Fat Pad Thickness and Elasticity on Heel Pain. *Journal of the American Podiatric Medical Association*. Vol. 94, No. 1, p. 48.

ANATOMY

Before considering the pathophysiology of the subcalcaneal spur, it is prudent to review the pertinent anatomy of the inferior heel to help differentiate and appreciate the structures involved in – and predisposed to – its genesis and the implication of its common presence with clinical syndromes, such as insertional plantar fasciitis.

The calcaneus is the largest bone of the foot and occupies the ‘heel’ region (Sarrafiian 1993). It articulates distally with the cuboid, superiorly with the talus and, by virtue of the spring ligament, the navicular superomedially (McCarthy & Gorecki 1979). Two convex tuberosities occupy the base posteriorly: the large medial tubercle and the smaller lateral, a triangular space or groove separates the two tubercles, (see Fig 2). The medial tubercle is the main weight-bearing segment of the calcaneus and has an average width of 2cm. In an anatomical series of 50 calcanei, Sarrafiian (1993) noted a ‘heel spur’ or shelf like anterior bony projection originating from the medial tubercle in 36% of cases, though he did not extrapolate on this finding.

The plantar aponeurosis, a structure largely implicated in the formation of a heel spur, is a strong band of collagen fibers that forms an investing layer in the sole of the foot (Mitchell, Meyer & Kreuger 1991). It is subcutaneous and extends from the heel region to the ball of the foot. There are three components to the plantar aponeurosis: the medial, central and lateral portions (see Fig. 3). The central component is the major constituent of the aponeurosis and, for the purposes of this review, the thinner medial and lateral components will not be covered.

Figure 2. Inferior Surface of the Calcaneus.
From: Snook, G.A. & Chrisman, O.D. (1972) The Management of Subcalcaneal Pain. *Clinical orthopaedics and Related Research*. Vol. 82, p. 164.

Figure 3. The Plantar Aponeurosis. From: Mitchell, I.R., Meyer, C. & Kreuger, W.A.

(1991) Deep Fascia of the Foot: Anatomical and Clinical Considerations. *Journal of the American Podiatric Medical Association*. Vol. 81, No. 7, p. 374.

The central component originates from the plantar aspect of the posteromedial calcaneal tuberosity. According to Sarrafian (1993), the origin is approximately 1.5-2cm wide and conforms to the convexity of the tuberosity. Mercado (1987) further articulates that the insertion of the plantar aponeurosis is just below the tuberosities, and a cadaver study of 200 specimens reports the plantar aponeurosis to be between 2-4mm thick immediately proximal to its insertion (Barrett, Day, Pignetti & Egli 1995). Distally, the plantar aponeurosis becomes thinner and broader and, at the mid-metatarsal region, divides into five bands which split superficial and deep, whereupon they blend and insert subcutaneously and deep, respectively, at the level of the metatarso-phalangeal and proximal digital architecture (Bojsen-Møller & Flagstad 1976; Mitchell et al. 1991).

The plantar aponeurosis stabilizes the medial longitudinal arch (Basmajian & Stecko 1963). Hicks (1955) likened the plantar aponeurosis to a windlass mechanism, whereby as the toes are extended, the plantar aponeurosis is pulled anteriorly around the metatarsal heads. This causes elevation of the arch by virtue of a shortening of the distance between the metatarsal heads and calcaneus (Hicks 1955), (see Fig 4). This takes place in late stance, as the digits are dorsiflexed at heel off (Fuller 2000).

Figure 4. The Windlass Mechanism causes elevation of the longitudinal arch of the foot.

From: Hicks, J.H. (1955) The Mechanics of the Foot: II The Plantar Aponeurosis and the Arch. *Journal of Anatomy*, Vol. 88, p. 29.

The flexor digitorum brevis muscle (FDB) originates from the posteromedial calcaneal tuberosity, the posterior third of the deep surface of the plantar aponeurosis and the lateral and medial intermuscular septum (Sarraffian 1993), (see Fig 5). Although Grodinsky (1937) describes a fascial cleft between the plantar aponeurosis and FDB, Sarraffian (1993) notes that the muscular attachment of FDB to the deep surface of the posterior third of the aponeurosis is dense, “allowing separation only by sharp dissection” (Sarraffian 1993, p. 247); this is supported by Forman and Green’s (1990) study. The calcaneal origin of FDB is sandwiched between the abductor hallucis medially, abductor digiti quinti anterolaterally and the aponeurosis infero-posteriorly (see Figs 5, 6). It divides into four muscular fascicles that give off tendon slips that insert into the base of the proximal phalanx of the lesser four digits (Sarraffian 1993). FDB stabilizes the lesser rays and the midtarsal joint about the oblique axis against ground reaction forces.

Figure 5. Representation of the origin of the Flexor Digitorum Brevis **(1)** and tendon slips **(1')**; the Abductor Hallucis Muscle **(2)**; Abductor Digiti Quinti **(3)**. From: Sarraffian, S.K. (1993) *Anatomy of the Foot and Ankle: Descriptive, Topographic, Functional*. 2nd Ed. J.B. Lippincott Company, p. 249.

The abductor hallucis muscle (AbH) extends from the inferior and medial aspect of the medial tubercle, the deep surface of the plantar aponeurosis, the posterior end of the medial intermuscular septum and the flexor retinaculum (Sarraffian 1993), (see Figs 5, 6). It inserts into the tibial sesamoid and medial aspect of the base of the proximal phalanx of the hallux. This muscle helps stabilize the first metatarso-phalangeal joint, abducts and plantarflexes the hallux.

A

B

Figure 6. A – Plantar Surface of Right Calcaneus. B – Medial Surface of Right Calcaneus.

From: McCarthy, D.J. & Gorecki, G.E. (1979) The Anatomical Basis of Inferior Calcaneal Lesions:

A Cryomicrotomy Study. *Journal of the American Podiatry Association*, Vol. 69, No. 9, p. 534-535.

The Abductor Digiti Quinti (AbQ) is the muscle of the lateral border of the foot and originates from the plantar aspect of the posterolateral tuberosity, the adjacent posteromedial tubercle of the calcaneus, slightly anterior to the FDB and the deep surface of the lateral component of the plantar aponeurosis, (Sarraffian 1993), (see Fig 6). It inserts into the plantar plate of the 5th metatarso-phalangeal joint and the lateral aspect of the base of the proximal phalanx of the 5th digit. This muscle causes plantarflexion and abduction of the 5th toe.

The quadratus plantae muscle (QP) is a trapezoidal, flat muscle formed by two muscular heads: the medial and lateral (Sarraffian 1993). It is situated between the FDB and the long plantar ligament (see Fig 6). The lateral head originates from the posterolateral calcaneal tuberosity and inferolateral aspect of the calcaneocuboid joint, while the medial head arises from the medial surface of the calcaneus (Sarraffian 1993). This muscle is relatively short and inserts into the lateral border of the common flexor digitorum tendon before it sends tendinous slips to the lesser four digits (McCarthy & Gorecki 1979). The QP muscle serves to “straighten” the oblique pull of the long flexor tendons.

According to Mann and Inman (1964), the intrinsic muscles act as a functional unit, exert a considerable flexion force on the fore part of the foot and play a principle active role in the stabilization of the foot during propulsion (see Fig 7). Both Mann and Inman (1964) and Basmajian (1963) agree that muscle activity is not evident or necessary to support the arches of a fully loaded foot at rest.

Figure 7. The intrinsic muscles of the foot stabilize the arch and aid in the elevation of the arch along with the plantar aponeurosis. From: Mann, R. & Inman, V.T. (1964) Phasic Activity of Intrinsic Muscles of the Foot. The Journal of Bone and Joint Surgery, Vol. 46A, No. 3, p. 477.

HEEL SPUR GEOGRAPHY

For the volumous literature dedicated to inferior heel pain, there have been relatively few studies dedicated to *defining the exact tissue* into which the spur itself propagates (Barret et al. 1995; Forman & Green 1990; McCarthy & Gorecki 1979).

DuVries (1957), Lapidus and Guidotti (1965) and Lewin (1959) report the spur to insert into the plantar fascia origin, while Hauser (1939, 1950), Hiss (1949) and Jahss (1992) suggest the heel spur arises in the plantar aponeurosis and intrinsic muscular attachments to the calcaneus. The term “intrinsic musculature” is rather ambiguous; the aforementioned authors do not specify *which* intrinsic muscle/s play a role – FDB, AbH, ABD, QP – or the relative importance of one structure over another in the propagation of the spur. Barret et al. (1995) in their cadaver analysis of 200 specimens, reported that of 42 demonstrable heel spurs, 52.4% were identified as originating within

the plantar fascia and 47.6% were superior to the plantar fascia, (assumedly within the FDB muscle origin).

Baxter, Pfeffer and Thigpen (1989) and Baxter and Pfeffer (1992) emphatically relate the heel spur to be dorsal to the fascial structure, within the FDB muscle. Tanz (1963) inserted a metallic marker into the calcaneal origin of the plantar aponeurosis and another in the origin of FDB in one cadaver. Subsequent radiographs demonstrated the portion of the calcaneus from which the muscle arose was the area in which the heel spur was noted, with the plantar fascia situated more proximal. McCarthy and Gorecki (1979) in a cryomicrotomy study of two 'variations' of heel specimens demonstrating spurs, implicated the position of a large heel spur in one specimen to be within the musculature of the FDB, (see Fig 8). In another, much smaller spur; the spur is more closely associated with the plantar fascia (see Fig. 9). They concluded that: "It is clear...that m. flexor digitorum brevis is often associated with plantar heel spurs," (McCarthy & Gorecki 1979, p. 533).

Forman and Green (1990) studied the anatomic relationship of the calcaneal spur to the plantar aponeurosis and associated intrinsic musculature in an unspecified number of freshly amputated limbs demonstrating heel spurs. They discovered the FDB to maintain the apex of the heel spur, as well as small involvement from the abductor hallucis muscle.

Figure 8. Microtomy section of one specimen with heel spur (*arrow*) evidently originating in the flexor digitorum musculature; (*p*) panniculus adipose; (*a*) plantar aponeurosis; (*f*) flexor digitorum brevis; (*q*) quadratus plantae; (*v*) lateral plantar vessels.

From: McCarthy, D.J. & Gorecki, G.E. (1979) The Anatomical Basis of Inferior Calcaneal Lesions: A Cryomicrotomy Study. *Journal of the American Podiatry Association*, Vol. 69, No. 9, p. 530.

Figure 9. Microtomy section of one specimen with heel spur (*arrow*) evidently originating in the flexor digitorum musculature; (*p*) panniculus adipose; (*a*) plantar aponeurosis; (*f*) flexor digitorum brevis; (*q*) quadratus plantae; (*v*) lateral plantar vessels.

From: McCarthy, D.J. & Gorecki, G.E. (1979) The Anatomical Basis of Inferior Calcaneal Lesions: A Cryomicrotomy Study. *Journal of the American Podiatry Association*, Vol. 69, No. 9, p. 531.

Although McCarthy and Gorecki's (1979) study was comprehensive to detail, there were only two anatomical specimens used to make detailed generalizations; and Forman and Green (1990) neglected to delineate how many specimens were involved in their study. Apart from Barrett et al. (1995) whose anatomic involvement of intrinsic muscle and plantar fascia seemed to be equivocal regarding heel spurs, there is a generally scarcity of large sample investigations – although a large number of suppositions exist.

Amis, Jennings, Graham and Graham (1988) demonstrated the presence of a “saddle sign” – a small lytic indentation – present on the tuberosity of the calcaneus posterior to the heel spur, if a spur was present, in patients with subcalcaneal heel pain (see Fig. 10). Amis et al. (1988) believes this point to represent a fatiguing of the tuberosity where the FDB attaches and that the heel spur itself is a ‘healing’ of this apparent ‘saddle injury’. The senior author suggests that the leading edge of the spur has nothing attached to it and that it is simply healing callous being layered above FDB.

Figure 10. The saddle sign (*arrow*) reported by Amis et al. (1988).

HEEL SPUR PATHOPHYSIOLOGY

At the pathological level, Fuller (2000) and Lewin (1959) supposes that traction of the plantar aponeurosis and intrinsic musculature on the medial calcaneal tubercle pulls off a portion of the periosteum, resulting in an osteogenic proliferation underneath, which forms new bone. Forman and Green (1990) hypothesize a similar pathology, wherein traction leads to periostitis and microtrauma, followed by calcification and bone spur formation.

Barrett et al. (1995), Hiss (1949) and McCarthy and Gorecki (1979) believe that fibrous tissue undergoes differentiation into bone. The authors’ implicate irritation to the periosteum in the origin of both plantar aponeurosis and intrinsic musculature. McCarthy and Gorecki (1979) describe an inflammatory response with osteogenic cell activation and fibrous connective tissue and cartilage matrix formation. An exostosis is ultimately formed through the process of calcification, resorption and recalcification.

DuVries (1965) and Mann (1978) relate the early stages of spur formation to a fibrositis of low chronicity and, should excessive tension in the aponeurosis at the medial calcaneal tubercle continue, bone deposition and osteophytic changes occur in the “sulcus”, anterior to the tuberosity. It is assumed this “sulcus” that Mann (1978) and DuVries (1965) suggest is the “natural tissue plane” that Barrett et al. (1995) report to exist between the 1st layer of intrinsic muscles and the plantar aponeurosis. They theorize that this area is the ideal location for the accumulation of serosanguaneous fluid to accumulate and subsequently ossify.

McCarthy and Gorecki (1979) examined a plantar calcaneal spur with its insertion into plantar fascia from one cadaver at an electron microscopic level. They reported both the *calcified*

region and the *aponeurosis* were characterized by highly irregular oriented fibrillar network of collagen, parallel to the weight-bearing surface. Lacunae were present, where presumably osteogenic cells and their associated territories were situated. The highly irregular collagenous network would seem to reflect previous microtrauma, likely traction – especially given their propensity to manifest along lines of tension – followed by fibroplasia and subsequent calcification, as was noted previously (McCarthy & Gorecki 1979).

Schepisis, Leach and Gorzyca (1991) and Snider, Clancy and McBeath (1983) analysed histologic samples of plantar aponeurosis resected from the origin of the aponeurosis in patients undergoing surgery for recalcitrant insertional plantar fasciitis. Similar to McCarthy and Gorecki's (1979) findings, both authors identified collagen degeneration, angiofibroplasia, chondroid metaplasia with lacunae containing cells in a number of samples examined. They postulated inflammation at the attachment of the plantar aponeurosis as a result of traction was underlying cause of the condition. There was no mention of the presence of heel spur presence.

More comprehensive studies have been reported recently (Lemont, Ammirati & Usen 2003; Tountas & Fornasier 1996). Tountas and Fornasier (1996) reported on 21 specimens submitted from surgery for subcalcaneal pain. Their samples contained the proximal insertion of the plantar aponeurosis and, if present, a heel spur. Results demonstrated varying degrees of fascial degeneration and reactive changes, from mucoid degeneration of dense collagenous tissue of fascia to tears and rupture distal to insertion of fascia. There was often an ingrowth of 'oedematous' connective tissue (angiofibroblastic tissue) noted, although the authors' stipulated that active inflammation was not a histological feature (see Fig 11A-C). The authors' postulated that, although not seen histologically, inflammation, not at the proximal attachment of the plantar aponeurosis, but of the subcalcaneal periosteum is the underlying cause of subcalcaneal pain.

The histology of the calcaneal spurs evaluated in the same study (Tountas & Fornasier 1996) consisted of mature lamellar bone with appositional periosteal woven bone deposition on its surface. There were also small foci of enchondral ossification at its apex and this was noted to be the only metabolically active area (Tountas and Fornasier 1996), (see Fig. 11C). This would agree with McCarthy and Gorecki's (1979) conclusion that a spur is a result of chondroid metaplasia of the reactive fibrous tissue, followed by calcification.

Lemont et al. (2003) reported on the histologic samples of 50 cases of "heel spur surgery", however they did not report on the specific implication of **(1)** whether heel spur resections were carried out, or **(2)** whether just the proximal plantar fascia was resected, or **(3)** if both were performed. In this study, the common pathologic findings were fiber fragmentation and myxoid degeneration, (see Fig 12). There was no mention of angiofibroplasia, although, a proportion demonstrated vascularization of the attached bone, with multiple dilated vessels present under low power magnification (see Fig 13). Lemont et al. (2003) suggests this is possibly the marrow oedema observed in magnetic resonance (MR) imaging sections of chronic plantar fasciitis patients. Based on their results, the authors conclude there is substantial evidence for degeneration, but little evidence of inflammation in subcalcaneal pain involving insertional plantar fasciitis.

Figure 11. (A) Light Photomicrographs showing mucoid degeneration and; **(B)** fibrinoid degeneration; **(C)** Longitudinal section of a heel spur; the arrow points to the only area metabolically active. From: Tountas, A.A. & Fournasier, V.L. (1996) Operative Treatment of Subcalcaneal Pain. *Clinical Orthopaedics and Related Research*. No. 332, p. 175.

Figure 12. (A) Zones of dark-staining areas representing mucopolysaccharide (*curved arrows*), with degeneration of collagen (*straight arrow*); **(B)** Close-up view of degenerated collagen (*fascia*). From: Lemont, H., Ammirati, K.M. & Usen, N. (2003) Plantar Fasciitis: A Degenerative Process (Fasciosis) Without Inflammation. *Journal of the American Podiatry Association*, Vol. 93, No. 3, p. 236.

Figure 13. (A) Low power view of calcaneal marrow demonstrating vascular engorgement; **(B)** High-power view of calcaneal marrow demonstrating dilated vasculature. From: Lemont, H., Ammirati, K.M. & Usen, N. (2003) Plantar Fasciitis: A Degenerative Process (Fasciosis) Without Inflammation. *Journal of the American Podiatry Association*, Vol. 93, No. 3, p. 236.

It is interesting to note that although the intrinsic musculature is implicated in heel spur formation and geography, there seems to be no evidence of muscle tissue origin in the heel spur samples histologically.

Insertional plantar fasciitis and heel spur have also been the focus of studies utilizing ultrasound and MR imaging modalities. Both MR and ultrasound studies report a pathologic thickening of the proximal portion of the plantar aponeurosis compared to controls (Berkowitz, Kier & Rudicel 1991; Cardinal, Chhem, Beauregard, Aubin & Pelletier 1996; Grasel, Schweitzer, Kovalovich, Karasick, Wapner, Hecht & Wander 1999; Narvez, Narvez, Ortega, Aguilera, Sanchez & Andia 2000; Theodorou, Theodorou, Farocki, Kakitsubata, Lektrakul, Resnick 2001; Theodorou, Theodorou, Kakitsubata, Lektrakul, Gold, Roger & Resnick 2000; Timmins 2000; Wall, Harkness & Crawford 1993).

The most prevalent finding in MR investigations into inferior heel pain involving insertional plantar fasciitis is perifascial oedema, and to a lesser extent intrafascial oedema (Grasel et al. 1999; Theodorou et al. 2001; Theodorou et al. 2000; Timmins 2000,). Grasel et al. (1999) believes this perifascial oedema to manifest as a result of microtears at the plantar aponeurotic attachment into the calcaneus. Interestingly, when reviewing the available published MR pictures in the aforementioned respective articles dealing with acute and chronic plantar fasciitis, there is often *significant* oedema within the proximal origin of the intrinsic muscles, specifically the region of the FDB and QP musculature, where they insert into the calcaneus (Grasel et al. 1999; Theodorou et al. 2001; Theodorou et al. 2000; Timmins 2000), (see Figs 14-16).

Figure 14. Sagittal fat-suppressed T2-weighted MR image of patient with acute plantar fasciitis; reveals thickened plantar aponeurosis (*solid straight arrows*) with small amount of abnormal intrasubstance signal intensity (*open arrow*), and widespread abnormal high signal intensity (oedema) in perifascial soft tissues (*curved arrows*). From: Theodorou, D.J., Theodorou, S.J., Kakitsubata, Y., Lektrakul, N., Gold, G.E., Roger, B. & Resnick, D. (2000) Plantar Fasciitis and Fascial Rupture: MR Imaging Findings in 26 Patients Supplemented with Anatomic Data in Cadavers. *Radiographics*, Vol. 20, p. 194.

Figure 15. (A) T2-weighted fast spin-echo MR image with fat saturation of patient with acute plantar fasciitis; shows thickening of plantar aponeurosis (*straight arrows*), extensive high signal intensity in perifascial soft tissues (*curved arrows*), and abnormal intermediate intrafascial signal intensity. From: Theodorou, D.J., Theodorou, S.J., Farocki, S., Kakitsubata, Y. & Resnick, D. (2001) Pictorial Review: Disorders of the Plantar Aponeurosis: A Spectrum of MR Imaging Findings. *AJR: American Journal of Roentgenology*, Vol. 176, January, p. 98.

Figure 15. (B) Unenhanced T1-weighted spin-echo image of chronic plantar fasciitis displays abnormal high signal intensity in superficial and

deep tissues to plantar aponeurosis (*solid and open arrows*). From: Theodorou, D.J., Theodorou, S.J., Farocki, S., Kakitsubata, Y. & Resnick, D. (2001) Pictorial Review: Disorders of the Plantar Aponeurosis: A Spectrum of MR Imaging Findings. *AJR: American Journal of Roentgenology*, Vol. 176, January, p. 99.

Figure 16. Sagittal fast short inversion time recovery (STIR)

of 'plantar fasciitis' reveals oedema (*arrows*) above and below

plantar aponeurosis at insertion to calcaneus. From: Grasel,

R.P., Schweitzer, M.E., Kovalovich, A.M., Karasick, D., Wapner, K.,

Hecht, P. & Wander, D. (1999) MR Imaging of Plantar Fasciitis:

Edema, Tears and Occult Marrow Abnormalities Correlated with

Outcome. *AJR: American Journal of Roentgenology*. Vol. 173, p. 700.

Also of interest is that while these articles relate the mere presence of 'perifascial oedema', they do not necessarily describe which perifascial tissue is involved, that which appears to be the fat pad and the proximal FDB, ABD and QP. Furthermore, they do not relate the magnitude or severity of the signal intensity. One might agree with Grasel et al. (1999), wherein the oedema has infiltrated into the perifascial tissues as a result of trauma to the plantar aponeurosis – fascial trauma and collagen degeneration is certainly well documented to be evident (Lemont et al 2003; Schepsis et al. 1991; Snider et al. 1983; Tountas & Fornasier 1996) – however, Lemont et al (2003) and Tountas and Fornasier's (1996) studies relate no evidence of inflammation in the fascia per se. Though perhaps the oedema has resulted within the perifascial tissues themselves and this tissue should be the focus of further investigation, especially given that evidence exists for the manifestation of the heel spur in this 'perifascial region', i.e. the FDB. Theodorou et al. (2001) notes specifically the location of the calcaneal spur as originating in the FDB and AbD musculature (see Fig 17).

In Grasel et al's (1999) study, calcaneal bone marrow abnormalities were the second most common finding in the study of insertional fasciitis. The authors related a poorly circumscribed area of marrow oedema "close" to the attachment of the plantar fascia, suggesting an injury mechanism based on tensile or avulsion injury. However, when one inspects the MR pictures, the marrow oedema is adjacent not to the aponeurotic insertion, but the intrinsic musculature, (see Fig 18). Theodoreau et al. (2001) also reported on marrow oedema associated with insertional plantar fasciitis, one figure presented in the study demonstrates signal intensity at both the plantar fascial insertion and intrinsic musculature insertion (see Fig 19). In Theodorou et al's (2000) study of 26 patients with insertional plantar fasciitis and rupture, bone marrow oedema was the least common finding.

A

B

Figure 17. (A) Unenhanced T1-weighted spin-echo MR image of asymptomatic patient shows calcaneal enthesophyte (*curved arrow*); plantar aponeurosis (*straight arrow*); adjacent soft tissue is unremarkable. **(B)** Gradient echo MR image of patient with chronic heel pain; calcaneal enthesophyte noted (*curved arrow*); abnormal high signal intensity in soft tissues superficial to plantar aponeurosis consistent with oedema (*open arrow*). From: Theodorou, D.J., Theodorou, S.J., Farocki, S., Kakitsubata, Y. & Resnick, D. (2001) Pictorial Review: Disorders of the Plantar Aponeurosis: A Spectrum of MR Imaging Findings. *AJR: American Journal of Roentgenology*, Vol. 176, January, p. 100.

Figure 18. Sagittal fast STIR MR image of patient with chronic plantar fasciitis demonstrating marrow signal abnormality (*arrows*). From: Grasel, R.P., Schweitzer, M.E., Kovalovich, A.M., Karasick, D., Wapner, K., Hecht, P. & Wander, D. (1999) MR Imaging of Plantar Fasciitis: Edema, Tears and Occult Marrow Abnormalities Correlated with Outcome. *AJR: American Journal of Roentgenology*. Vol. 173, p. 700.

Figure 19. Fast spin-echo T2-weighted MR image of patient with acute plantar fasciitis; signal intensity changes of oedema in perifascial soft tissues (*straight arrows*). Note foci of abnormal bone marrow high signal intensity at plantar fascial and intrinsic muscular origin (*curved arrows*). From: Theodorou, D.J., Theodorou, S.J., Farocki, S., Kakitsubata, Y. & Resnick, D. (2001) Pictorial

CONCLUSION

The inferior calcaneal spur has been the subject of much investigation and is widely associated with the commonly encountered clinical syndrome of insertional plantar fasciitis. Both of these entities, which have traditionally been studied concomitantly, are believed to be the manifestation of stress and traction at the site where both the plantar aponeurosis and the intrinsic musculature originate from the medial calcaneal tubercle. The direction of the spur (and its collagenous structure) – longitudinal and in line with the pull of the aforementioned structures – would seem to affirm this hypothesis. Overweight, prolonged weight-bearing, age and foot type have been demonstrated to influence the prevalence of heel spur formation and heel pain.

Singularly, the plantar aponeurosis, the flexor digitorum brevis or both the plantar aponeurosis and flexor digitorum brevis are implicated as the tissues responsible for heel spur formation. The literature seems equivocal on this point, providing evidence for the presence of spurs in each the plantar fascia and the flexor digitorum brevis, although there has been no mention of muscular tissue evident in histological examinations of heel spurs. It seems logical, given the firm attachment of the plantar aponeurosis and flexor brevis anatomically to each other; and their similar function in foot stability and arch maintenance in late stance, where both could act simultaneously as a single unit pulling at the calcaneal attachment.

Regardless, this traction by either or both of these structures results in micro trauma, and:

- (1)** collagen degeneration and fibrous proliferation of the plantar aponeurosis at its insertion site;
- (2)** inflammation and oedema of the origin flexor digitorum brevis, plantar fascial origin and fat pad;
- (3)** irritation at the osseous interface of these structures, resulting in oedema of calcaneal bone marrow and ultimately both appositional bone growth and enchondral ossification, resulting in a spur. It is interesting to note that traditionally the intrinsic musculature is closely 'freed' from osseous/ spur interface before spur resection during heel spur surgery and may be the reason behind a lack of muscle 'presence' in histological samples.

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