Heel Spur Formation and the Subcalcaneal Enthesis of the Plantar Fascia

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ABSTRACT. ***Objective.*** To describe the structure and significance of subcalcaneal heel spurs associated with the plantar fascia.

Methods. The enthesis of the plantar fascia was removed from 17 elderly cadavers by sagittal saw cuts either side of the medial tuberosity, radiographs were taken, and the tissue was processed for routine histology. Sagittal sections were stained with toluidine blue, Masson's trichrome, or alcian blue, and sections were matched with the corresponding radiographs.

Results. Spurs develop on the deep surface of the plantar fascia but their formation is heralded by degenerative changes that occur within it. According to differences between small and large spurs, we propose that there are 3 stages in their development: (1) an initial formation of cartilage cell clusters and fissures at the plantar fascia enthesis; (2) thickening of the subchondral bone plate at the enthesis as small spurs form; (3) development of vertically oriented trabeculae buttressing the proximal end of larger spurs. The spurs grow by a combination of intramembranous and chondroidal ossification.

Conclusion. Contrary to popular belief, subcalcaneal heel spurs cannot be traction spurs as they do not develop within the plantar fascia itself. They are thus fundamentally different from heel spurs in the Achilles tendon. We suggest instead that they develop as a consequence of degenerative changes that occur in the plantar fascia enthesis. (J Rheumatol 2002;29:1957–64)

Key Indexing Terms:
HEEL SPUR PLANTAR FASCIA ENTHESIS
ENTHESOPATHY HISTOPATHOLOGY

Subcalcaneal heel pain is very common and well known to rheumatologists and orthopedic surgeons alike. It is characterized by extreme tenderness along the plantar-medial aspect of the heel, and often presents as a repetitive strain injury in distance runners or in middle aged patients with a history of prolonged standing[1-7]. However, heel pain can also be linked to underlying systemic disorders such as rheumatoid arthritis (RA), ankylosing spondylitis, and Reiter's syndrome[8-12]. In these conditions, the symptoms are often bilateral. Although the cause of heel pain is unclear, it is frequently associated with bony spurs[1,2] — although these have also been identified in 15% of asymptomatic patients[13]. The most commonly accepted theory for spur formation is a biomechanical one, for they are widely thought to form as a result of excessive traction at the origin of the plantar fascia and flexor digitorum brevis[1,4,10,14-16]. The spurs are viewed as a reactive ossification to chronic inflammation[1,17].

We recently reported that traction spurs developing in the Achilles tendon of elderly rats are initiated by endochondral ossification following vascular invasion from the bone marrow, with vessel ingrowth proceeding along rows of enthesis fibrocartilage cells[18]. Here we describe the structure of bony spurs of different sizes in the plantar fascia of elderly dissecting room cadavers and suggest a hypothesis for their formation. As the plantar fascia is mechanically linked to the Achilles tendon by fibrous tissue passing below the calcaneus and by a series of regularly oriented bony trabeculae[19], we predicted that the spurs form by a similar mechanism. However, our findings show that this cannot be the case and that the spurs must be fundamentally different.

MATERIALS AND METHODS

The subcalcaneal enthesis of the plantar fascia, including the entire medial tuberosity of the calcaneus and adjacent connective tissues, was removed from 17 elderly cadavers (64–97 years of age, 10 male, 7 female). Medical histories were not available other than the cause of death, and the cadavers were selected from a total of 34 available specimens according to the quality of preservation and the absence of gross abnormalities in the foot. The cadavers were fixed with embalming fluid containing 4% formaldehyde and 25% alcohol. Entheses were removed from one limb only by making parallel saw cuts either side of the medial tuberosity of the calcaneus in the sagittal plane. Radiographs were taken of all specimens at this stage and the tissue subsequently processed for routine histology as described[19]. Briefly, the samples were further fixed in 10% neutral buffered formal saline, decalcified in 5% nitric acid, dehydrated in graded alcohols, cleared, and embedded in paraffin wax. Serial sagittal sections were cut at 8 μm and 6 sections collected systematically at 1 mm intervals throughout the blocks of tissue. Peripheral sections were discarded in order to remove the risk of viewing artefactual damage in the tissue that resulted from the saw cuts. Adjacent slides were stained with alcian blue, hematoxylin and eosin, Masson's trichrome, and toluidine blue.

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The maximum thickness of the plantar fascia was compared in specimens with and without radiologically detectable heel spurs. Estimates were made on slides with a micrometer eyepiece at ×100 magnification. Measurements were made at each sample point in the blocks, and the largest value was taken as the fascial thickness. Differences in the mean thickness were assessed by an unpaired Student t test and a value of $p < 0.05$ was regarded as significant.

RESULTS

Bony spurs. Bony spurs were common and a graded series of spurs of different sizes are illustrated in Figure 1 in an attempt to mimic a "developmental sequence." Possible stages in their development are summarized in Figure 2. In 8 specimens, the spurs were visible radiologically, and in a further 3 they could be detected microscopically. Larger spurs showed both radiological and histological evidence of cortical bone thickening (Figures 1d, e, i, j). The mean thickness of the plantar fascia was significantly greater ($p < 0.02$) in radiographically visible spurs than in specimens that either lacked spurs or had microscopical ones (5.1 ± 0.39 mm compared with 4.0 ± 0.44 mm, respectively). While the precise location of the spurs was not always evident from radiographs, the corresponding histological sections clearly showed that they were always located immediately deep to the plantar fascia on its dorsal side, and thus were not embedded within it. This applied not only to the 5 spurs illustrated in Figure 1, but also to the remaining spurs that are not illustrated. The spurs were covered by a pad of uncalcified fibrocartilage on their plantar side (Figures 3a and 4d), and their tips were separated from the fascia by a fatty or fibrous connective tissue, richly supplied with blood vessels and nerves (Figures 4d and e).

Bony spurs of all sizes showed abundant evidence of ossification and an irregular surface of the bone commonly suggested active bone turnover — osteoclasts lying in Howship's lacunae were present on their marrow cavity side (Figure 3e). Periosteal bone deposition by intramembranous ossification was seen on the outer, dorsal aspect of the spur (Figures 3c and d), but chondroidal ossification (i.e., the method by which chondroid bone is formed) occurred on the plantar side (Figures 3a and b). Chondroid bone was characterized by the presence of fibrocartilage cells embedded in bone-like matrix and was particularly common in larger bony spurs (Figure 3b). Typical signs of endochondral ossification (cartilage cell hypertrophy, cell death, and vascular invasion) were not detected in any of the specimens, and calcified fibrocartilage was only rarely present at the surface of the spur.

Histopathology of the plantar fascia enthesis. The subcalcaneal enthesis was highly fibrocartilaginous and regions of calcified and uncalcified fibrocartilage were readily identifiable at the insertion site (Figure 4a). These 2 zones were separated by one or more tidemarks that defined the outer limit of calcification (Figure 4a). In most specimens, there was a clear distinction between fiber arrangement in the uncalcified fibrocartilage of the dorsal ("deep") and plantar ("superficial") parts of the enthesis. In the latter, the fibers were sagitally arranged, but in the former, fascicles of transversely arranged fibers were interwoven with others that ran sagitally, thus creating a distinctive basketweave of fiber bundles (Figure 4b). In the dorsal part of the enthesis, the different bundles were separated by thin films of loose connective tissue ("endoligament" in Figure 4b) and the fibrocartilage was generally more highly cellular (Figure 4c). Muscle fibers from flexor digitorum brevis were attached to the dorsal side of the plantar fascia (Figure 4d) and numerous small blood vessels and nerves were present immediately deep to it, in a region of fatty or fibrous connective tissue close to the enthesis (Figures 4d and e). However, the enthesis fibrocartilage itself was generally avascular and aneural. The subchondral bone plate was consistently thin (Figures 1f to j) and the deeper trabeculae were regularly aligned along the direction of tensile force at the enthesis, and continuous with similar trabeculae at the Achilles tendon insertion (Figure 4f). In contrast, trabeculae were vertically arranged at the anterior margin of the calcaneus (Figure 4f).

Histopathological changes were common, especially in the dorsal region of enthesis fibrocartilage. They included cartilage cell clustering, longitudinal fissure formation, and local erosion of subchondral bone (Figures 5a to f). The cartilage cell clusters were located close to the bone and were surrounded by an extracellular matrix (ECM) that stained intensely with alcian blue (Figures 5a to c). The longitudinal fissures were splits in the fibrocartilage that contained a strongly-staining, amorphous ECM, suggesting mucoid degeneration (Figure 5d). The subchondral bone was locally absent at some parts of the enthesis, and either blood vessels in the marrow cavity were in direct contact with the uncalcified enthesis fibrocartilage or the defect was filled with a vascular loose connective tissue (Figure 5f).

DISCUSSION

The location of the subcalcaneal heel spurs just deep to the plantar fascia (rather than within it) challenges the widely held view that they are traction spurs developing in response to tension[1,4,10,14-16]. We suggest instead that they represent a response to mechanical stress at the enthesis and are comparable to the peripheral osteophytes of articular cartilage in patients with osteoarthritis[20,21]. Whether the spurs are the cause or an effect of the plantar fascial thickening reported here and by Berkowitz, *et al*[22] is unclear. The cell clusters and longitudinal fissures resemble those in osteoarthritic cartilage[21,23] and have been reported in the entheses of human tendons[24,25]. Intriguingly, calcaneal spurs are also more frequent in patients with osteoarthritis than those without — and the frequency rises with age[11,26]. In terms of repetitive loading experienced, the fibrocartilage of the

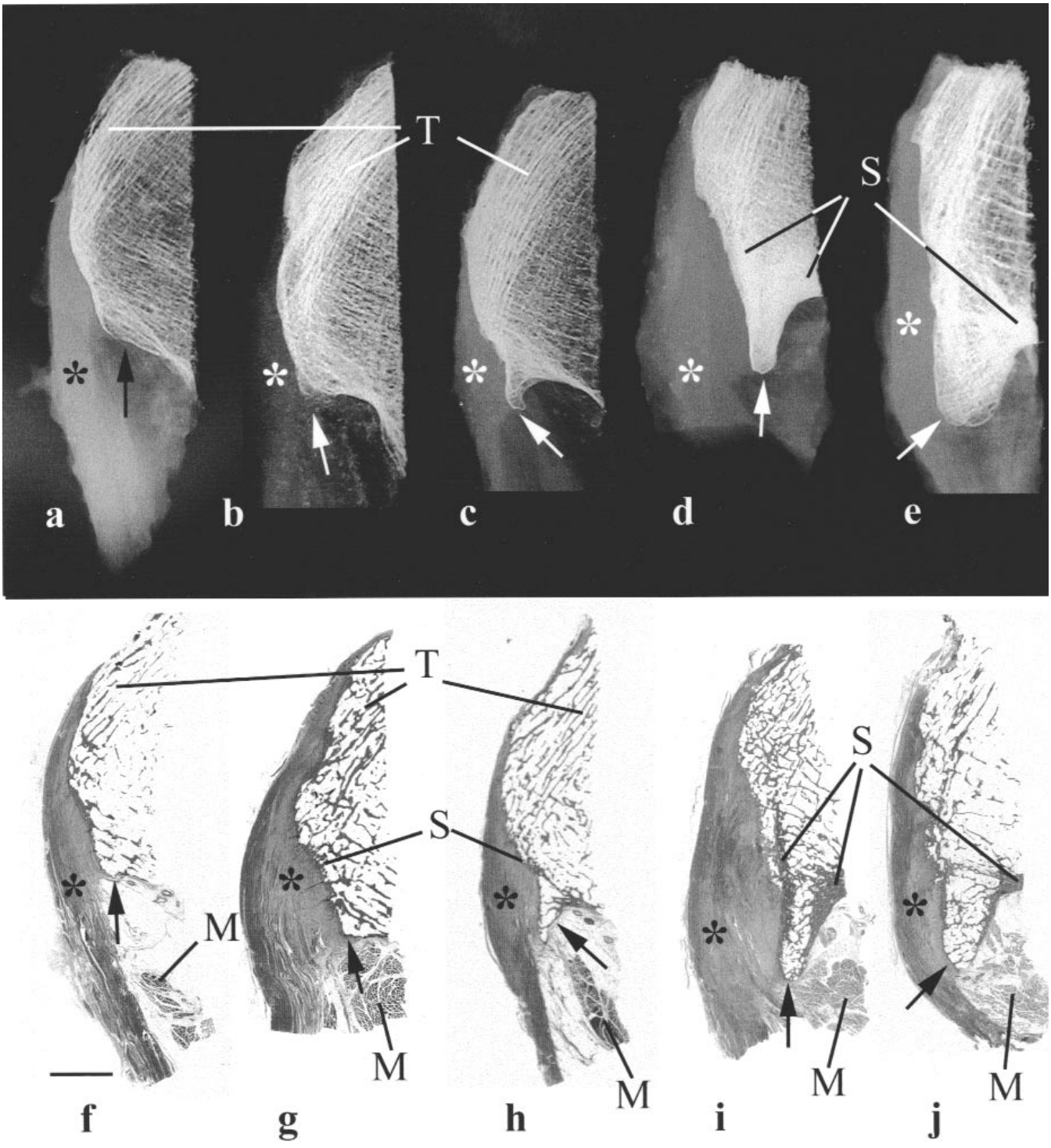

Figure 1. Radiographs of heel spurs (arrows), arranged in a "developmental sequence" (a–e) and shown together with a single histological section chosen by an unblinded observer from the middle of the section series (f–j), in order to enhance the interpretation of both. The radiographs, printed so that the plantar fascia (*) is visible in the soft tissue relief, clearly show that the spurs develop adjacent to the plantar fascia but not within it. In the specimens with smaller spurs (a–c, corresponding to f–h), the parallel trabeculae (T) linking the enthesis of the Achilles tendon to that of the plantar fascia are clearly visible. In all cases, there is little compact bone at the attachment site, although there is evidence of subchondral bone sclerosis (S) in the region of most spurs — especially the largest (spurs in d and e, corresponding to i and j). M: flexor digitorum brevis muscle fibers. Histological sections are stained with Masson's trichrome. Scale bar = 5 mm.

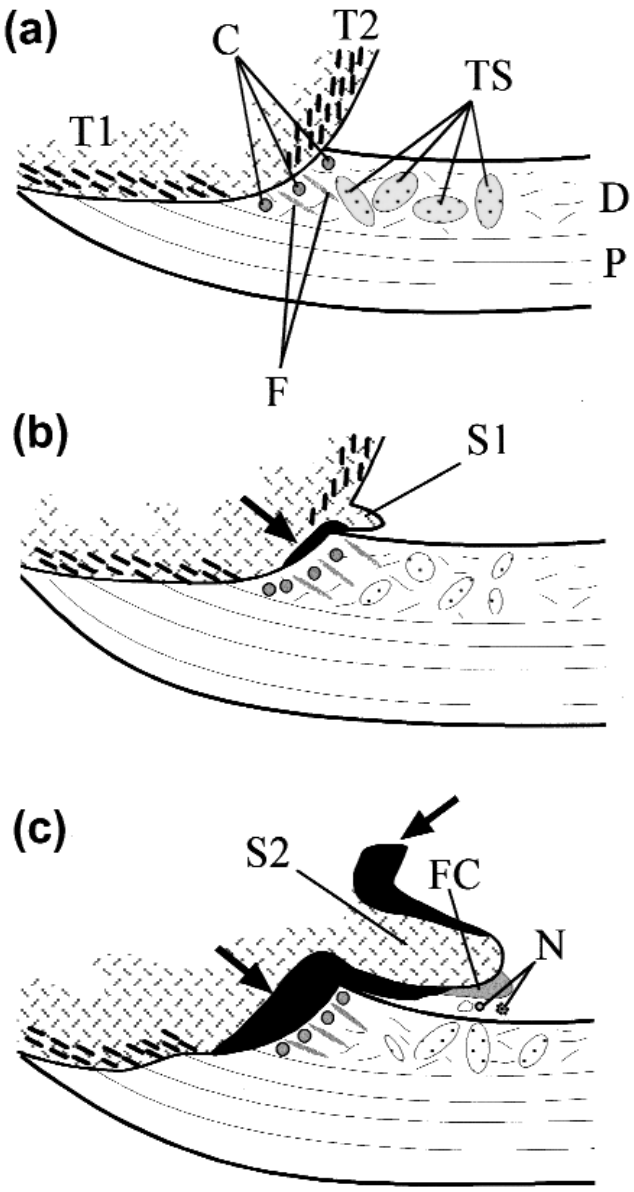

Figure 2. Three possible stages in the formation of a heel spur. Each drawing shows the T1 and T2 groups of trabeculae at the plantar fascia enthesis. The former are oriented along the direction of pull of the fascia and the latter along the line of force transmission that is associated with weight-bearing. Note the distinction between the dorsal (D) and plantar (P) regions of the enthesis that is associated with the presence of interweaving bundles of collagen fibers (TS) in the former. (Panel a) Prior to spur formation, degenerative changes occur in the dorsal enthesis fibrocartilage, near the bony interface. These changes are characterized by the appearance of clusters of cartilage cells (C) and longitudinal fissures (F). (Panel b) The development of a small spur (S1) is associated with subchondral bone sclerosis, which typically starts on the plantar side of the spur (arrow). (Panel c) In larger spurs (S2), the sclerosis is more obvious and is present on both its dorsal and plantar sides (arrows). The tip of the spur (particularly its plantar side) is covered with a pad of fibrocartilage (FC) and between the pad and the plantar fascia itself are neurovascular bundles (N).

plantar fascia is comparable to articular cartilage in a weight-bearing joint. It is this unusual, weight-bearing role of the plantar fascia enthesis, together with the insertional angle changes that occur as the arch of the foot flattens, that probably explains why the enthesis is so highly fibrocartilaginous. Direct compression on the enthesis could also account for the basketweave arrangement of its collagen fibers near the dorsal side. Such an interdigitation of fibers is common in the fibrocartilaginous "wrap-around" regions of tendons and ligaments that change direction by pressing against bony pulleys[27-29].

The suggestion that heel spurs routinely seen in many elderly people are not traction outgrowths from the calcaneus is supported by the work of Tountas and Fornasier[30]. They showed that spurs can reform after surgical release of the plantar fascia in attempts to relieve heel pain. It also means that these spurs differ from those previously documented in the human Achilles tendon[24]. Such spurs may indeed be traction spurs, for they are embedded in the most inferior (i.e., superficial) part of the Achilles tendon enthesis. We view spurs on the deep surface of the plantar fascia either as adaptive responses to changes in the loading patterns on the fascia that result from wear and tear, or conversely as an attempt to change the loading patterns on the fascia so that further damage may be minimized. Either view is in agreement with the general suggestion that both osteophytes and enthesophytes represent skeletal responses to stress[31,32]. Further, both views are compatible with the suggestion that heel spurs develop in response to calcaneal stress fractures as a means of buttressing the bone against developing microcracks[32], or that there is an association between spur formation and bone marrow edema in the calcaneus[33]. Finally, they fit easily with the idea that obesity is correlated with an increasing incidence of spurs, for in overweight individuals, the heel obviously experiences increased weight-bearing[2,3,10,34]. Heel spurs are probably common in our study because we have used material from elderly cadavers and such spurs are a normal manifestation of aging[2,3,13,14,26]. However, the incidence may also be high because of the sensitivity of the techniques we have used to identify them. Few authors have looked at spurs histologically and none has performed correlative radiographs on such small bony fragments, isolated *ex vivo*.

The spurs show evidence of intramembranous and chondroidal ossification. Although the latter is less well known than the former, the concept of cartilage that changes directly into bone by metaplasia is not new[35] and chondroid bone has been described at numerous locations including entheses[35,36]. In contrast to traction spurs in the rat Achilles tendon[18], typical signs of endochondral ossification were not seen, and the differences may relate to different locations of the spurs. Not all spurs develop by the same mechanism or in response to the same stimuli, and whether plantar fascial spurs in patients with seronegative spondyloarthropathy

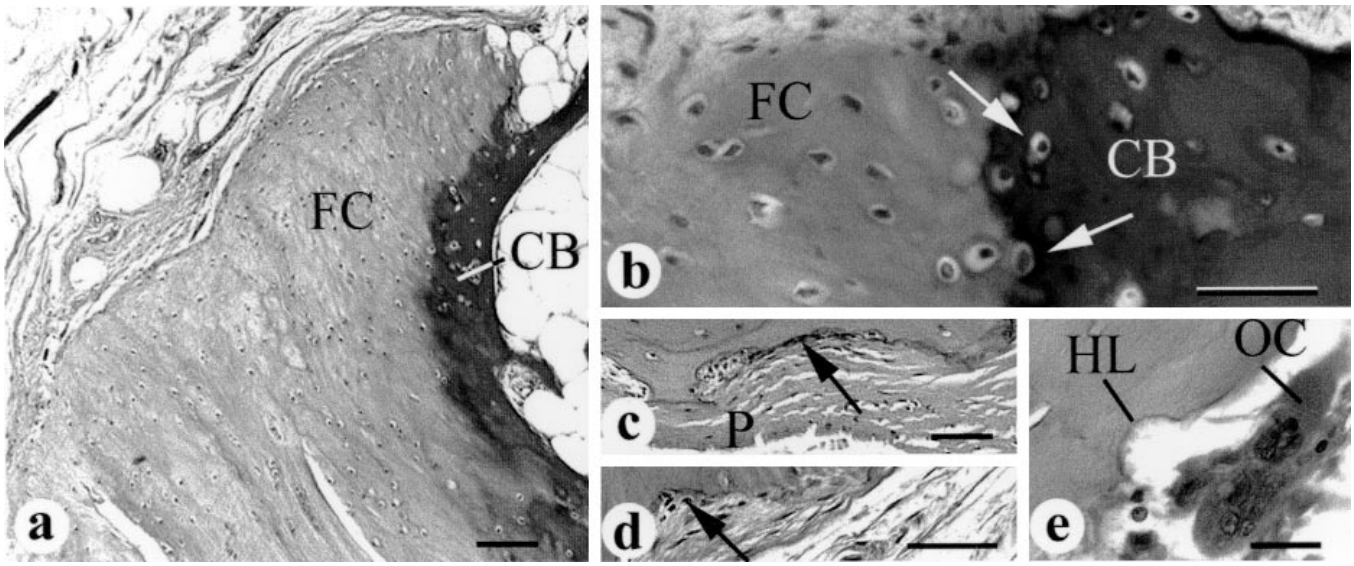

Figure 3. Evidence of tissue remodeling associated with the bony spurs. (a) The tip of a spur showing the presence of chondroid bone (CB). This tissue is associated with a thick pad of fibrocartilage (FC) on the plantar surface of the spur and there is no sharp boundary between the 2 tissues (Masson's trichrome). Scale bar = 100 μm. (b) High power view of the interface between chondroid bone (CB) and fibrocartilage (FC) to show the presence of cartilage cells (arrows) in the bone matrix. Note the absence of calcified fibrocartilage (Masson's trichrome). Scale bar = 50 μm. (c–d) Intramembranous ossification on the dorsal side of a spur, suggested by the presence of osteoblasts (arrows) that have developed from the deep layer of the periosteum (P) (Masson's trichrome). Scale bars = 100 μm (c) and 50 μm (d). (e) An osteoclast (OC) and a Howship's lacuna (HL) on the marrow cavity side of a small spur. The spur is similar in size to that illustrated in Figure 3b (Masson's trichrome). Scale bars = 20 μm.

(SpA) develop in a fashion similar to that reported here is unclear. Radiological evidence suggests that some spurs in patients with SpA are embedded in the plantar fascia, but that others lie deep to it[11,37]. Further, in both these patients and those with RA, the spurs develop after episodes of bony erosion triggered by inflammation — i.e., they reflect periods of "reactive ossification"[9,11,37].

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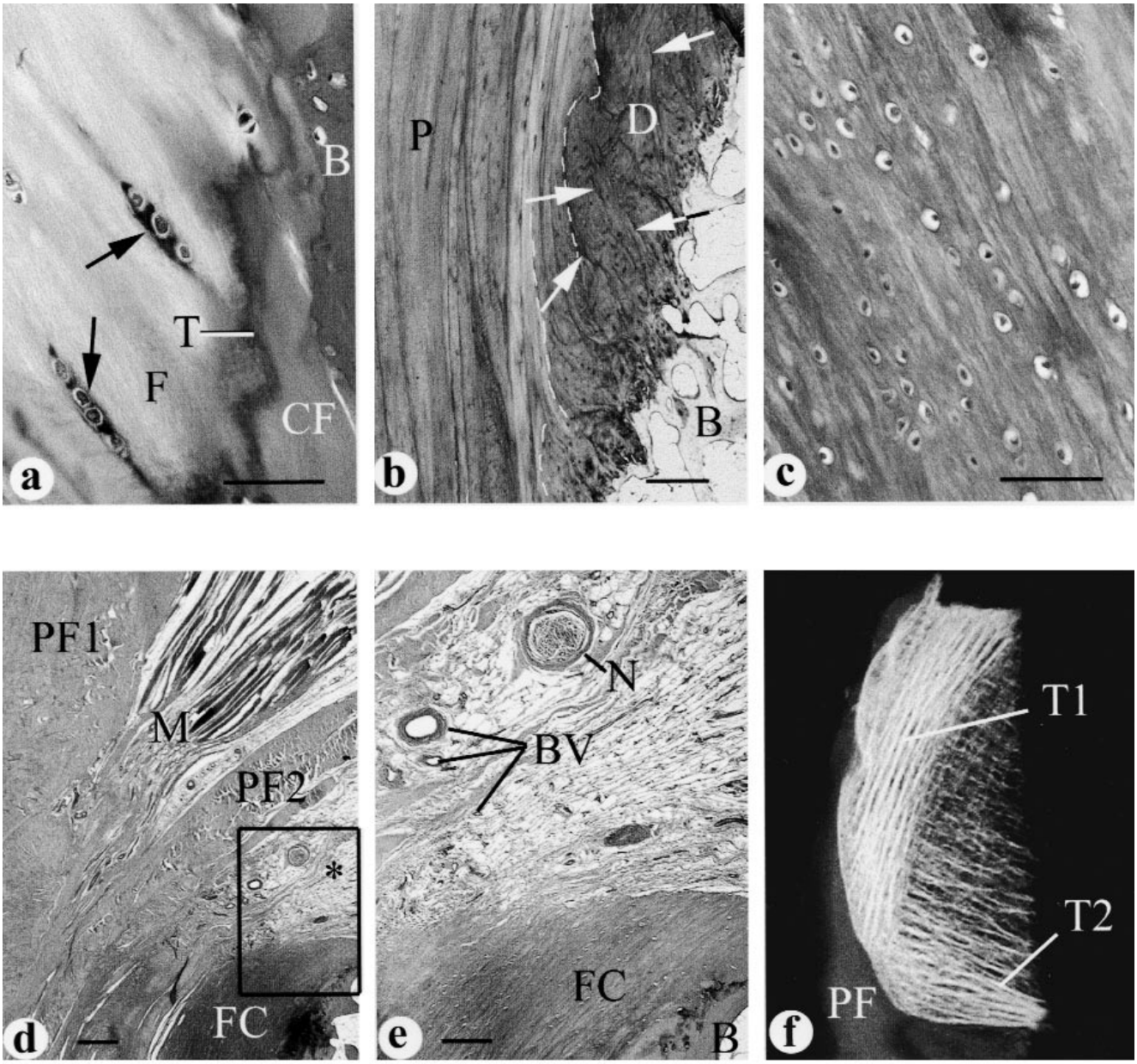

Figure 4. Aspects of enthesis structure pertinent to understanding the histopathological changes at the plantar fascia and the formation of heel spurs. (a) The fibrocartilaginous character of the interface between hard and soft tissues. Rows of cartilage cells (arrows) surrounded by a small quantity of strongly-staining proteoglycan-rich matrix characterize the zone of uncalcified fibrocartilage. The rows of cells are separated from each other by parallel collagen fibers (F). A thin zone of calcified fibrocartilage (CF) covers a thin shell of subchondral bone (B), immediately beneath a strongly-staining tidemark (T) that signifies the outer limit of calcification (Masson's trichrome). Scale bar = 100 μm. (b) A conspicuous difference in fiber arrangement allows a distinction to be made between dorsal (D) and plantar (P) regions of uncalcified fibrocartilage. In the former (which is more deeply placed), bundles of fibers that are cut in different planes are separated by thin sheets of loose connective tissue called endoligament (arrows). This suggests the bundles have a basketweave arrangement. In the latter, the collagen fibers largely run in the sagittal plane and are thus cut longitudinally (Toluidine blue). Scale bar = 500 μm. (c) The deeper, dorsal zone of fibrocartilage is generally highly cellular (hematoxylin & alcian blue). Scale bar = 100 μm. (d) Immediately distal to the enthesis, muscle fibers (M) belonging to flexor digitorum brevis, attach to the dorsal side of the plantar fascia (i.e., to its deep aspect), partly splitting this fascia into 2 layers (PF1 and PF2). Adjacent to these muscle fibers is a layer of loose connective tissue (*), filling the space between them and the tip of a bony spur. This spur is covered by a prominent pad of fibrocartilage (FC) on its plantar side. Note that the plantar fascia does not attach to the tip of the spur (hematoxylin & alcian blue). Scale bar = 500 μm. (e) High power view of the region enclosed in the rectangle in (d) to show the presence of small blood vessels (BV) and nerves (N) in the connective tissue adjacent to the spur. B: Bone; FC: periosteal fibrocartilage on the spur (hematoxylin & alcian blue). Scale bar = 200 μm. (f) Typical radiograph of a specimen that lacks a bony spur. There are 2 prominent collections of trabeculae in the calcaneus. Those labelled T1 link the enthesis of the Achilles tendon (not shown) to that of the plantar fascia. This fascia is visible in the soft tissue relieve (PF). T2 trabeculae run vertically at the anterior margin of the bone.

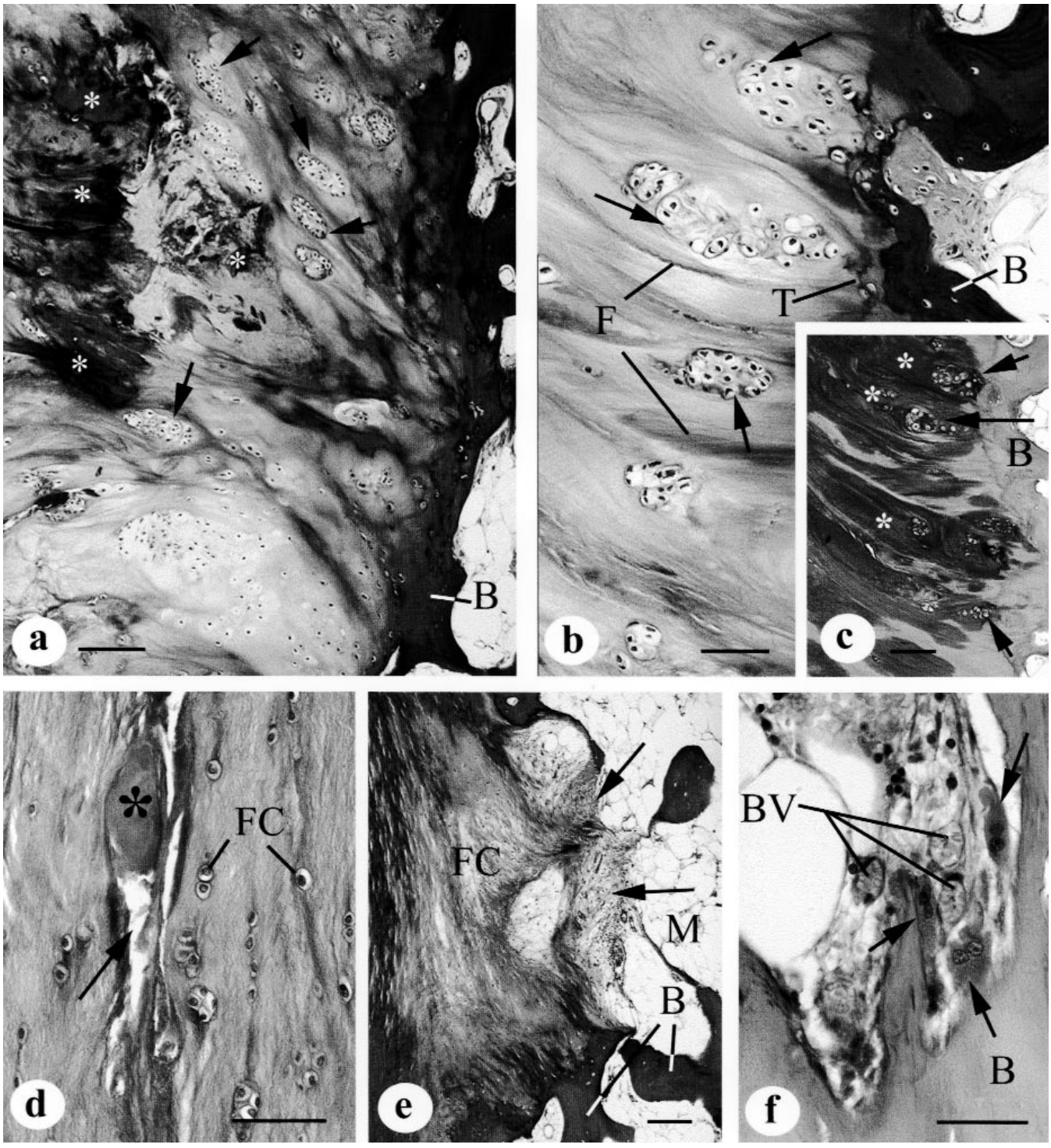

Figure 5. Histopathological changes in the plantar fascia enthesis. (a) Low power view of the plantar zone (see Figure 1b) of uncalcified enthesis fibrocartilage, showing the extensive formation of cartilage cell clusters (arrows) and prominent regions of soft tissue calcification (*). B: bone (Masson's trichrome). Scale bar = 200 μm. (b) High power view of several cartilage cell clusters (arrows, separated by bundles of collagen fibers (F) that pass towards the tidemark (T). B: bone (Masson's trichrome). Scale bar = 100 μm. (c) Cell clusters (arrows) are surrounded by a matrix that stains intensely with alcian blue (*), suggesting that it is rich in proteoglycans (hematoxylin & alcian blue). Scale bar = 200 μm. (d) A longitudinal fissure (arrow) in the plantar region of uncalcified enthesis fibrocartilage, containing a strongly-staining amorphous matrix (*). FC: fibrocartilage cells (hematoxylin & alcian blue). Scale bar = 100 μm. (e) Part of the enthesis where the subchondral bone (B) has been completely eroded and a layer of loose connective tissue (arrows) lies between the bone marrow (M) and the uncalcified fibrocartilage (FC) (Masson's trichrome). Scale bar = 200 μm. (f) Osteoclasts (arrows) eroding the subchondral bone on its marrow side, in a region richly endowed with blood vessels (BV) (hematoxylin & alcian blue). Scale bar = 50 μm.

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