The Structure and Histopathology of the "Enthesis Organ" at the Navicular Insertion of the Tendon of Tibialis Posterior

BERNHARD MORIGGL, TSUKASA KUMAI, STEFAN MILZ and MICHAEL BENJAMIN

ABSTRACT. ***Objective.*** To investigate the structure, histopathology, and molecular composition of tissue specializations of the tibialis posterior enthesis. They collectively reduce stress concentration at the insertion site and are part of an "enthesis organ." This has implications for understanding the basis of enthesopathy.

Methods. Fifty-two specimens of tibialis posterior and the associated superomedial part of the calcaneonavicular ligament taken from cadavers were sectioned longitudinally and examined by routine histology (42 samples) or immunohistochemistry (10 samples). Serial sections of formalin fixed material were stained with Masson's trichrome, toluidine blue, or hematoxylin, eosin and alcian blue. A panel of antibodies against collagens, glycosaminoglycans, and proteoglycans was used to immunolabel methanol fixed material.

Results. The enthesis organ consists of the enthesis itself, the superomedial part of the calcaneonavicular ligament (which may fuse with the tendon), the tendon sheath, and associated accessory bones. The accessory bones lay in a region of fibrocartilage that was present even in specimens where the bones themselves were absent. Degenerative changes were seen at the enthesis, around the accessory bones, and in the walls of the tendon sheath. The navicular and accessory bone entheses, together with the calcaneonavicular ligament, were all rich in fibrocartilage. This immunolabeled for aggrecan, link protein, type II collagen, and versican.

Conclusion. The complexity of the enthesis organ, and the diversity of sites showing histopathological changes, suggest that enthesopathy may not be located precisely at the osteotendinous junction. It could target a number of adjacent locations, in accord with what happens at other entheses; e.g., in patients with spondyloarthropathy. The prominence of fibrocartilage in the enthesis organ, and the degenerative changes to which it is subject, support the view that spondyloarthropathy has an underlying biomechanical basis. (J Rheumatol 2003;30:508–17)

Key Indexing Terms:
ENTHESIS ENTHESOPATHY TIBIALIS POSTERIOR
SPONDYLOARTHROPATHY HISTOPATHOLOGY

The region where a tendon, ligament, or joint capsule attaches to bone is well known to rheumatologists as an enthesis. However, in the Achilles tendon, there is also a collection of related structures close to the enthesis itself that additionally serve to reduce the risk of wear and tear at the tendon-bone junction. Thus, the tendon dissipates stress concentration away from the enthesis by pressing against the superior tuberosity of the calcaneus in a dorsiflexed foot[1,2]. Here, the tendon and bone are protected from compressive forces by 2 fibrocartilages — a sesamoid fibrocartilage (SF) near the deep surface of the tendon and a periosteal fibrocartilage (PF) on the superior tuberosity[1]. The free movement of the tendon relative to the bone is promoted by the retrocalcaneal bursa and protruding into this bursa is a variable space-filler — the retromalleolar fat pad[3]. The whole assembly of tissues, together with the enthesis itself, constitutes what has recently been called an "enthesis organ"[2], and the complexity of the region has led Canoso[3] to refer to the insertion of the Achilles tendon as "the premiere enthesis."

The recognition that there is more to entheses than simply the tendon-bone junction is of considerable clinical significance to rheumatologists, for it explains the diffuse nature of imaging abnormalities that are commonly reported close to entheses, but not precisely at the osteotendinous

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junction[4-7]. We argue that such changes should also be regarded as indicative of enthesopathy, for they affect the enthesis organ if not the enthesis itself[2].

Although we know a good deal about the structure of many entheses in addition to the Achilles tendon — and this has helped in understanding the anatomical basis of the seronegative spondyloarthropathies (SpA)[2], little attention has been paid to enthesis organs other than that of the Achilles tendon. Our purpose is to describe the structure and histopathology of an elaborate enthesis organ associated with the attachment of the tendon of tibialis posterior (TP) to the tuberosity of the navicular. This tendon and the navicular bone to which it attaches are well documented sites of disease in patients with SpA[8,9], but are also implicated in overuse injuries in athletes — especially those with an accessory bone in the tendon close to the enthesis[10,11]. As degenerative changes in tendons increase with age[12] and as evidence of enthesopathies is common in elderly dissecting room cadavers[1,13], we have again used this material for the present study. The use of biopsies from patients with enthesopathies should be subject to ethical considerations.

MATERIALS AND METHODS

Routine histology and histopathology. Forty-two specimens of the TP tendon and the associated superomedial part of the calcaneonavicular (CN) ligament were taken from 37 embalmed dissecting room cadavers at Munich and Cardiff Universities. The cadavers had been perfused with a solution containing formaldehyde and alcohol, as described[1,14]. Because of the link between the presence of accessory bones and an increased frequency of enthesopathies[10,11], the specimens were not randomly selected, but deliberately chosen (after radiographic examination and preliminary palpation) to include 50% that had accessory bones. However, no selected cadaver showed any gross abnormalities of the feet. The mean age of the cadavers was 76.4 years (range 53–95). Twenty-five specimens came from females (14 right, 11 left) and 17 from males (10 right, 7 left).

The tendon was initially cut transversely in the region of the medial malleolus so that a good length of it could be easily grasped with forceps, enabling saw cuts to be made into the tuberosity of the navicular. Two cuts were made into the bone, parallel to the long axis of the tendon, and the terminal part of the tendon with its enthesis was removed. All the specimens also included the underlying superomedial part of the CN ligament, but the plantar fibers of the tendon that inserted elsewhere were cut distal to the tuberosity. All specimens were postfixed for 1 week in 10% neutral buffered formal saline, decalcified in 5% nitric acid, and processed for routine histology as described[1]. Longitudinal sections of the enthesis organ were cut at 3–8 µm at 1 mm intervals throughout the block and 6 sections collected at each sample point. Adjacent sections were stained with toluidine blue, Masson's trichrome, and hematoxylin-eosin, with or without alcian blue.

Immunohistochemistry. Specimens were removed as described above from 10 cadavers at the University of Munich within 28 h of death. The mean age of the cadavers was 84 years (range 64–98). Seven specimens came from females (3 right, 4 left) and 3 from males (2 right, 1 left) and all were fixed for 24 h in 90% methanol at 4°C. Specimens were then stored in methanol at –20°C until required. The bone was decalcified with 5% EDTA, the specimens rinsed in phosphate buffered saline (PBS), infiltrated for 12 h with 5% sucrose in PBS, and sectioned at 12 µm on a Microm cryostat (HM500 OMV). Sections were immunolabeled with monoclonal antibodies directed against glycosaminoglycans (chondroitin 4 sulfate and chondroitin 6 sulfate, dermatan sulfate, and keratan sulfate), proteoglycans (aggrecan and versican), collagens (types I, II, III, VI), tenascin, and link protein. Details of the sources of the antibodies, together with their dilutions, and any essential pretreatments were as described[15]. The activity of any endogenous peroxidase was blocked by pretreating the sections for 30 min with 0.3% hydrogen peroxide in methanol, and nonspecific binding of secondary antibodies was minimized by blocking sections with serum for 40 min. Control sections were produced by either omitting the primary antibody or incubating the sections with nonimmune mouse immunoglobulins (10 µg/ml). Antibody binding was detected with a Vectastain ABC Elite avidin/biotin/peroxidase kit.

RESULTS

Normal histology of the components of the enthesis organ

We define the enthesis organ of tibialis posterior (Figure 1) as consisting of (1) the enthesis itself, (2) the superomedial part of the calcaneonavicular (CN) ligament with which the tendon may fuse at its insertion, (3) the tendon sheath (the functional equivalent of a bursa), and (4) any accessory bone present near the osteotendinous junction. The accessory bones lay in a region of fibrocartilage that was present even in enthesis organs where the bones themselves were absent. There are 2 types of accessory bones (Figure 2) — type I bones that lie entirely within the substance of the tendon (i.e., they are sesamoid bones), proximal to the enthesis itself, and type II bones that lie very close to the navicular tuberosity and form a joint with it[11].

Tibialis posterior enthesis. In all cadavers, the enthesis was fibrocartilaginous, with zones of calcified and uncalcified fibrocartilage that were separated from each other by one or more tidemarks (Figure 3A). Both the zone of calcified fibrocartilage and occasionally the subchondral bone could be absent locally so that blood vessels in the bone marrow contacted the zone of uncalcified fibrocartilage directly (Figures 3B, 3C). Elsewhere, this fibrocartilage was avascular. Where a type II accessory navicular was present, the tendon attached both to this bone and to the navicular itself. In addition, the accessory navicular bones articulated with the parent navicular via a small synovial joint lined on both sides by articular fibrocartilage (Figures 2F, 2G).

Two forms of entheses could be recognized. (1) Entheses where there was a significant attachment of tendon fibers to the tuberosity (Figure 3B) via a typical region of enthesis fibrocartilage (EF) with relatively few fibers passing distally at this point (15 cases). The EF was clearly visible microscopically (Figure 3C), but was less obvious with the naked eye. (2) Entheses where the tendon had a "bipartite" structure (Figures 3D, 3E), with superficial and deep parts discernible to the naked eye (27 cases). The superficial part of the tendon did not attach to the tuberosity at all, but continued towards the other tarsal bones. Although this part of the tendon was fibrocartilaginous, its fibrocartilage cells were arranged in longitudinal rows between parallel collagen fibers (Figure 3F). The deep part of the tendon consisted of a layer of EF that was clearly demarcated from

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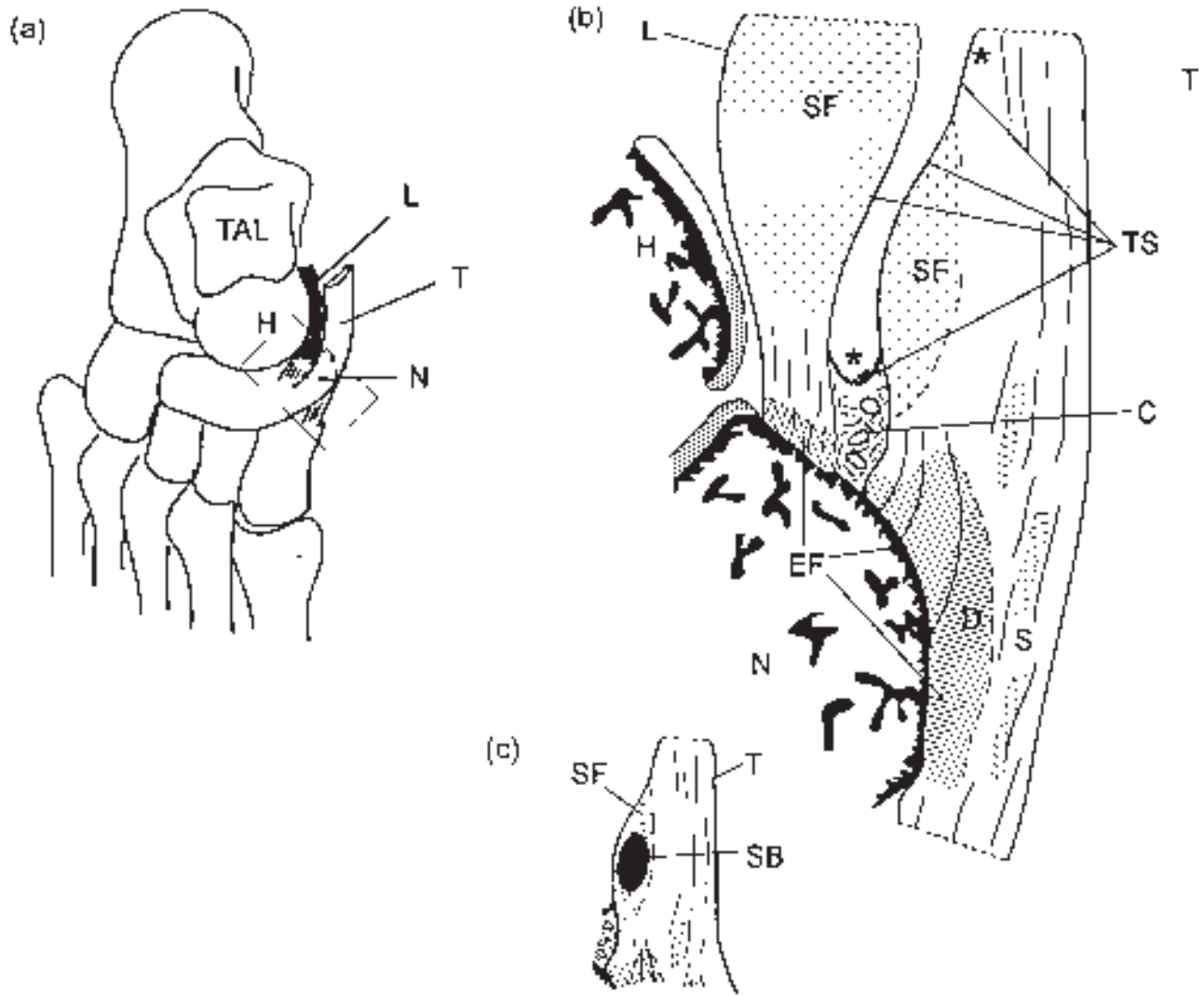

Figure 1. The enthesis organ associated with the attachment of the tendon (T) of tibialis posterior to the navicular tuberosity (N). (a) The position of the enthesis organ in the hind foot. Note that the superomedial part of the calcaneonavicular ligament (L) lies between the tendon (T) and the head (H) of the talus (TAL). (b) Detailed structure of the enthesis organ: enlargement of the region enclosed in the rectangle in (a). Note that in the region of the navicular tuberosity, the tendon may have a bipartite structure with distinct superficial (S) and deep (D) parts. Both regions are fibrocartilaginous, although fibers in the superficial part pass over the tuberosity without attaching to the bone, whereas those in the deep part contribute to a thick layer of enthesis fibrocartilage (EF) that attaches the tendon to the bone. Immediately proximal to the EF, the tendon contains a sesamoid fibrocartilage (SF) of variable size. Between the tendon and the head of the talus (H) is the superomedial part of the calcaneonavicular ligament (L). In this region, the ligament is dominated by SF that gives the whole structure a disc-like character. The ligament is also attached to the bone by EF and may either be fused with the tendon (not shown) distal to the SF or separated from it (as illustrated here) by the deep part of the tendon sheath (TS) and by loose connective and adipose tissue (C). Note that some parts of the TS are lined by synovial membrane (*), but others by the SF of the tendon and ligament. (c) If a true sesamoid bone (SB) is present as part of the enthesis organ, it is embedded within the region of tendon SF, shown in (b).

the remainder of the tendon and which could be very thick (Figure 3D). Histologically, the deep EF contained some regions (typically proximally) where the collagen fibers were parallel to each other and the cells arranged in longitudinal rows. In other regions (typically more distally), the collagen fibers were interwoven in basketweave fashion and there was no regular orientation of the cells.

Accessory bones and sesamoid fibrocartilages. Our radiographs (Figures 2A-D) suggested that one or more type I accessory navicular bones were present within the tendon in 19 specimens, and that a type II accessory bone was present in 3 further specimens. However, histological examination (Figures 2E-H) showed that one of the bones that was classed radiologically as a type II accessory navicular was actually type I — i.e., it was entirely embedded within the tendon and connected to the parent navicular purely by fibrous tissue (Figure 2H). All the accessory bones were characterized histologically by prominent zones of calcified

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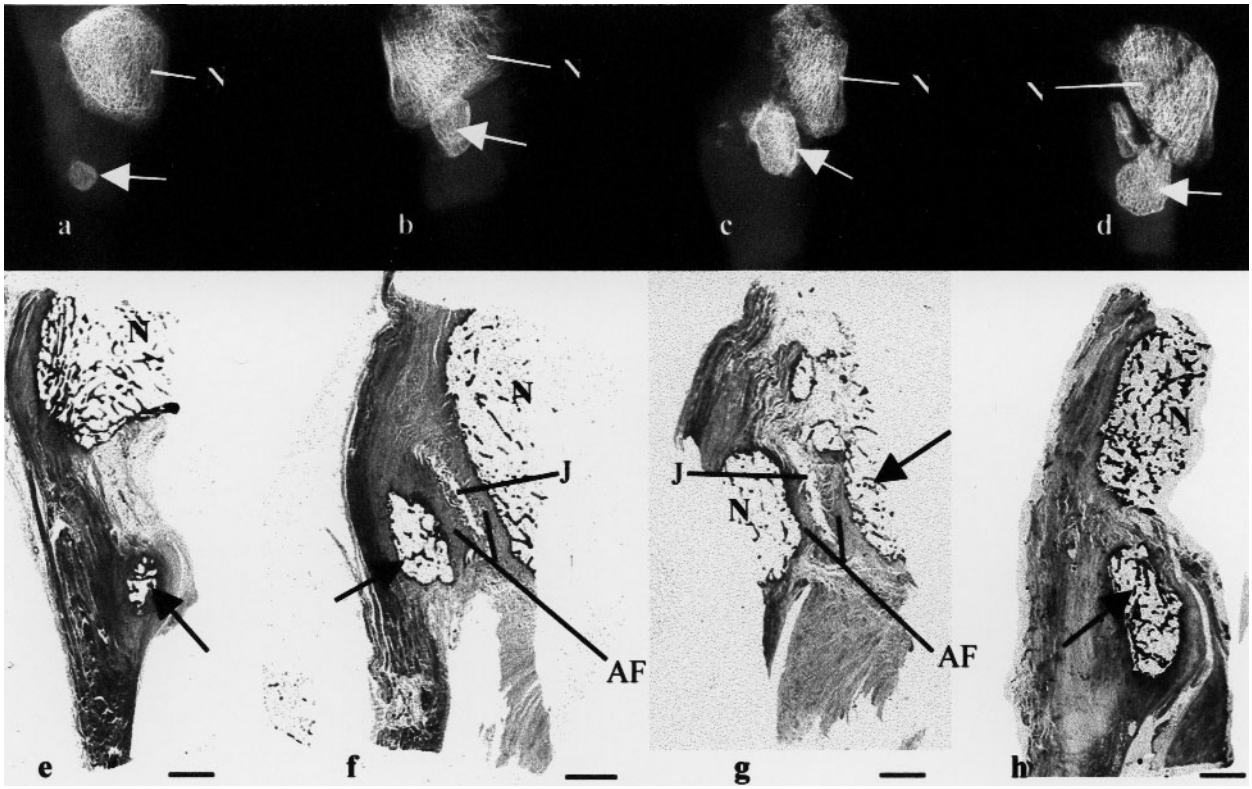

Figure 2. Radiological images (a–d) shown alongside histological sections of the same specimens (e–h) from cadavers in which an accessory bone (arrows) is present in the tendon of tibialis posterior near the navicular tuberosity (N). The bones in (a) and (e) and (d) and (h) are true sesamoid bones, but those in (b) and (f) and (c) and (g) are type II accessory navicular bones in which there is a small synovial joint (J) lined with articular fibrocartilages (AF). Note that it is impossible to determine radiologically that the ossicles in (b) and (c) are type II accessory bones rather than sesamoids (scale bars = 3 mm).

fibrocartilage at the bone-tendon interface — especially on the proximal side of the bone (Figure 3G). Remnants of calcified fibrocartilage were also common in the underlying bone (Figure 3G). The sesamoid bones lay within a region of SF that was generally present within the enthesis organ even when an accessory bone was absent.

Where a SF only was present, it was located in the deep part of the tendon, facing the fibrocartilaginous region of the CN ligament (see below) and varied greatly in size. The largest of the fibrocartilages created a distinct bulge on the deep surface of the tendon that corresponded to a concavity in the ligament (Figure 3I). Fibrocartilage cells were conspicuous, the extracellular matrix (ECM) was highly metachromatic and characterized by a typical basketweave arrangement of collagen fibers (Figures 3J, 3K).

Associated ligament. In the region deep to the tendon, the proximal segment of the superomedial part of the CN ligament is a SF that broadly resembled an intraarticular disc (Figure 3I). It has a metachromatic ECM with a basketweave arrangement of collagen fibers (Figure 3L). In several specimens, this SF greatly increased the thickness of the ligament proximally, so that the adjacent region that typically lay between the tendon and the talar head was biconcave (Figure 3I). The distal region of the ligament was more fibrous but generally attached to the navicular via a fibrocartilaginous enthesis, separated by small areas of adipose tissue (Figure 3H). Regions of vascular loose connective tissue and fat also served as a space-filler between the distal part of the ligament and tendon, although occasionally the tendon and ligament were fused more proximally. The ligament was always separated from the tendon by the deep part of the TP tendon sheath even when the tendon and ligament were partly fused. In some regions, the tendon sheath was lined by a synovial membrane (Figure 3M), but in others by the adjacent fibrocartilaginous regions of the tendon or ligament (Figure 3N).

Immunohistochemistry of the enthesis organ

A wide variety of collagens and glycosaminoglycans were present in the different regions of the enthesis organ (Table 1; Figure 4). In particular, however, labeling for aggrecan, link protein, and type II collagen was especially intense at all hard tissue interfaces, including those associated with accessory bones (Figures 4A-D). At "bipartite entheses" (see above), aggrecan and type II collagen were prominent in the superficial part of the attachment, so that an extensive

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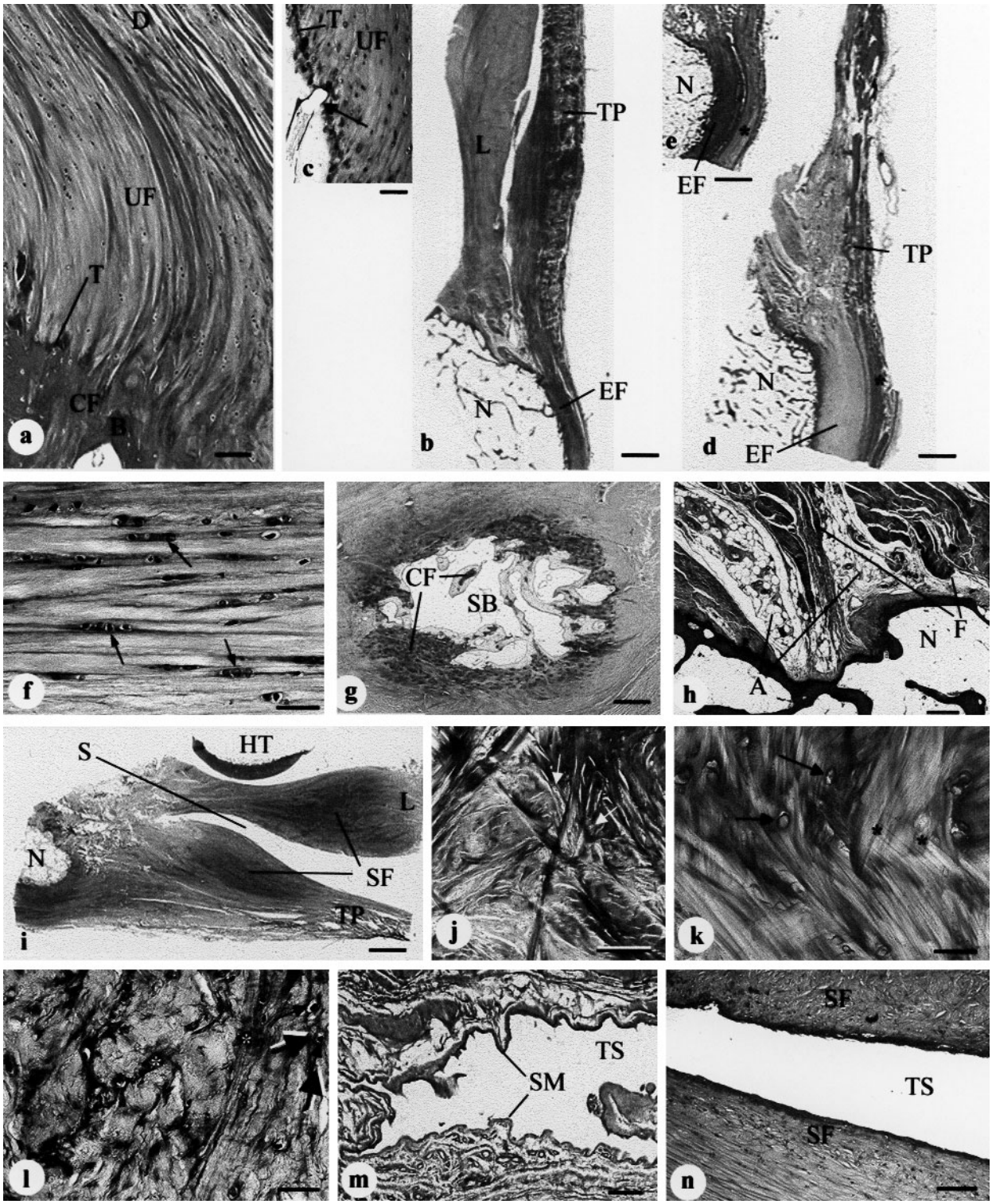

Figure 3.

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Figure 3. Routine histology of the enthesis organ. (a) The 4 zones of tissue at the fibrocartilaginous enthesis of the tendon of tibialis posterior: dense fibrous connective tissue (D), uncalcified fibrocartilage (UF), calcified fibrocartilage (CF), and bone (B). UF and CF are separated from each other by a tidemark (T) (Masson's trichrome; scale bar = 100 µm). (b) A macroscopic view of the attachment of tibialis posterior (TP) in which most of the tendon fibers attach to the tuberosity of the navicular (N) via an enthesis fibrocartilage (EF). L: superomedial part of the calcaneonavicular ligament (toluidine blue; scale bar = 2 mm). (c) A higher power view of the enthesis shown in (b). Note the presence of a blood vessel (arrow) in the underlying bone marrow that lies in direct contact with the zone of uncalcified fibrocartilage (UF) in a region of the enthesis in which bone is locally absent. T: tidemark (toluidine blue; scale bar = 100 µm). (d) A macroscopic view of the attachment of tibialis posterior (TP) in which the tendon is bipartite near the tuberosity of the navicular (N). The extensive deep part of the tendon attaches to the bone via a region of enthesis fibrocartilage (EF), but the smaller superficial part (*) continues beyond this bone to other sites of attachment in the foot (toluidine blue; scale bar = 2 mm). (e) A bipartite tendon in which roughly half the thickness of the tendon is attached via enthesis fibrocartilage (EF) to the navicular tuberosity (N) and half passes to other tarsal or metatarsal bones (*) (toluidine blue; scale bar = 2 mm). (f) Conspicuous rows of fibrocartilage cells (arrows) in the part of the tendon that passes beyond the navicular tuberosity [region indicated by * in figures (d) and (e)] to other sites of attachment (toluidine blue; scale bar = 50 µm). (g) Prominent zones of calcified fibrocartilage (CF) both at the bone-tendon interface of a sesamoid bone (SB) and in the core of its cancellous bone spicules (toluidine blue; scale bar = 500 µm). (h) Characteristic regions of adipose tissue (A) intervening between the bundles of collagen fibers (F) at the attachment of the superomedial part of the calcaneonavicular ligament to the navicular tuberosity (N) (Masson's trichrome; scale bar = 300 µm). (i) A macroscopic view of the enthesis organ of the tendon of tibialis posterior (TP) in the region of the navicular tuberosity (N). Note that the ligament (L) is biconcave because of a bulging sesamoid fibrocartilage (SF) within it, that it is separated from the tendon by the synovial cavity (S) of the tendon sheath, but then merges with the tendon at its enthesis. A prominent sesamoid fibrocartilage also bulges from the tendon in the region where the ligament is biconcave (scale bar = 2 mm). (j) The characteristic basketweave arrangement of collagen fibers (arrows) in the sesamoid fibrocartilage of the tendon of tibialis posterior (scale bar = 500 µm). (k) A higher magnification view of the sesamoid fibrocartilage within the tendon of tibialis posterior showing large fibrocartilage cells (arrows) amidst a basketweave arrangement of collagen fibers (*) (toluidine blue; scale bar = 50 µm). (l) The sesamoid fibrocartilage in the superomedial part of the calcaneonavicular ligament, showing fibrocartilage cells (arrows) in a metachromatic ECM (asterisk) that contains a basketweave of collagen fibers cut in longitudinal and cross section. Similar metachromasia was seen in the sesamoid fibrocartilage of the tendon (toluidine blue; scale bar = 50 µm). (m, n) Although synovial membrane (SM) was present in the deepest recesses of the tendon sheath (TS), it was absent more proximally in regions where sesamoid fibrocartilage (SF) was present (toluidine blue; scale bars = 100 µm).

area of the tendon was positively labeled (Figures 4E, 4F). Aggrecan, link protein, and type II collagen also characterized the SF of both the tendon and ligament (Figure 4G). Versican was present in all regions investigated (Figures 4H, 4I).

Histopathology of the enthesis organ

Histopathological changes were seen in 10 specimens. They were associated not only with the main tendon attachment itself, but also with the presence of sesamoid and accessory navicular bones and the deep part of the TP tendon sheath. However, the associated ligament showed little evidence of degeneration.

At the tendon enthesis, there were ample signs of fissuring and chondrocyte clustering (Figures 5A, 5B), together with soft tissue calcification and associated necrosis. Cell clusters were also seen near the osteotendinous junction of sesamoid bones, where microcysts, horizontal splits near the hard/soft tissue interface, and soft tissue calcification were conspicuous features (Figures 5C, 5D). In all specimens where a type II accessory navicular was present, there was clear evidence of osteoarthritis in the fibrocartilaginous synovial joint between this bone and the parent navicular (Figures 5E, 5F). The degenerative changes affected the articular surfaces on both sides of the joint. Further signs of degeneration (fragmentation and calcification) were seen in the deep part of TP tendon sheath (Figure 5G).

Table 1. Summary of the immunohistochemical labeling patterns at TP enthesis organ.

	Tendon					Ligament	
Antigen Detected	EF at Tuberosity*	SF	EF at Sesamoid Bones	SF Around Sesamoid Bones	Proximal Tensional Region	EF	SF
Collagen I	+	+	+/–	+	+	+	+
Collagen II	++	+/++	++	++/+	–/+	+/++	+/++
Collagen III	+	+	+	+	+/–	+	+
Collagen VI	+	+	–	–/+	+	+	+
C4S	+/++	+	+/++	+/++	+/–	+/++	+
C4S and DS	++	+	+	+/++	+/–	++/+	+
C6S	+/++	+	+/++	+/++	+/–	+	+
KS	+/++	+	+/++	+	+	+/++	+
Versican	+	+/++	+	+	+	+	+/++
Tenascin	+	+	+/–	+	+	+	+
Aggrecan	++/+	+	+	+/++	–/+	++	+
Link protein	++/+	+	+/++	+/–	–/+	++	+

Note: the entire enthesis fibrocartilage was included in bipartite entheses. ++ strong labeling; + moderate labeling; – no labeling. C4S: chondroitin 4 sulfate; C6S: chondroitin 6 sulfate; DS: dermatan sulfate; KS: keratan sulfate. EF: enthesis fibrocartilage; SF: sesamoid fibrocartilage.

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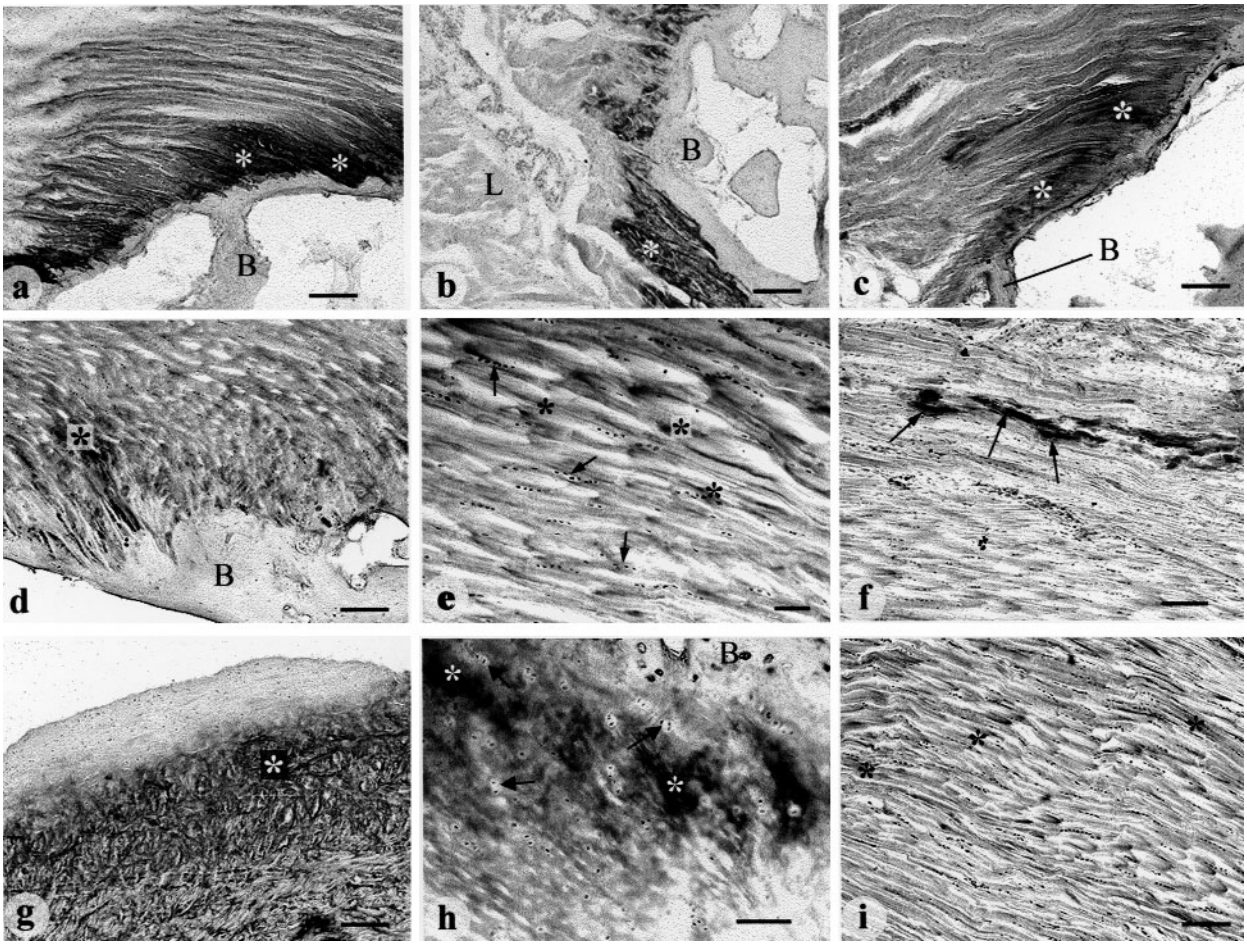

Figure 4. Immunohistochemical labeling of the enthesis organ of tibialis posterior. (a) Type II collagen (*) in the enthesis fibrocartilage at the attachment of tibialis posterior to the accessory navicular. B: bone (scale bar = 100 µm). (b) Type II collagen (*) in the enthesis fibrocartilage of the superomedial part of the calcaneonavicular ligament (L). B: bone (scale bar = 100 µm). (c) Aggrecan labeling (*) in the enthesis fibrocartilage of the tendon of tibialis posterior. B: bone (scale bar = 100 µm). (d) Labeling for link protein (*) in the enthesis fibrocartilage of the tendon of tibialis posterior in the region of its attachment to the tuberosity of the navicular. B: bone (scale bar = 100 µm). (e) Type II collagen labeling (*) in the superficial part of the tibialis posterior tendon that wraps over the navicular tuberosity to other sites of attachment in the foot. Arrows: fibrocartilage cells (scale bar = 100 µm). (f) Patches of aggrecan label (arrows) in the superficial part of the tibialis posterior tendon that wraps over the navicular tuberosity to other sites of attachment in the foot (scale bar = 100 µm). (g) Type II collagen labeling (*) in the sesamoid fibrocartilage within the tendon of tibialis posterior. Note, however, the absence of labeling from a band of tissue near the deep surface of the tendon (scale bar = 100 µm). (h) Labeling for versican (*) in the enthesis fibrocartilage of the tendon of tibialis posterior in the region of its attachment to the tuberosity of the navicular. B: bone. Arrows: fibrocartilage cells (scale bar = 50 µm). (i) Labeling for versican (*) in the superficial part of the tibialis posterior tendon that wraps over the navicular tuberosity to other sites of attachment in the foot (scale bar = 100 µm).

DISCUSSION

Our report highlights an unheralded complexity in the structure of an "enthesis organ" associated with the tendon of tibialis posterior in the region of the tuberosity of the navicular. Stress concentration is accentuated here because of the multiple functions of the muscle as an invertor and plantarflexor of the foot (i.e., it promotes movements in 3 dimensions) and of the tendon as a structure that helps to support the medial longitudinal arch. The EF of the tendon probably serves to dissipate bending and narrowing forces away from the navicular tuberosity (see reference 2 for a review of enthesis fibrocartilage function), and the neighboring ligament is also an integral part of the enthesis organ that we suggest acts as an articular disc contributing to load dissipation. It may fuse with the tendon at its enthesis. Because of the increased surface area that it provides for tendon contact, stress is dissipated away from the tendon-bone junction itself. The compression is resisted by a complementary pair of SF within the ligament and the opposing part of the tendon that are functionally comparable to fibrocartilages associated with the enthesis organ of the Achilles tendon[1]. Parts of the fibrocartilages contribute directly to lining the tendon sheath in a manner comparable to that in the retrocalcaneal bursa[1]. The ligament also helps to limit the range of motion and prevent the talar head from collapsing or slipping medially[16,17]. Functional insufficiency

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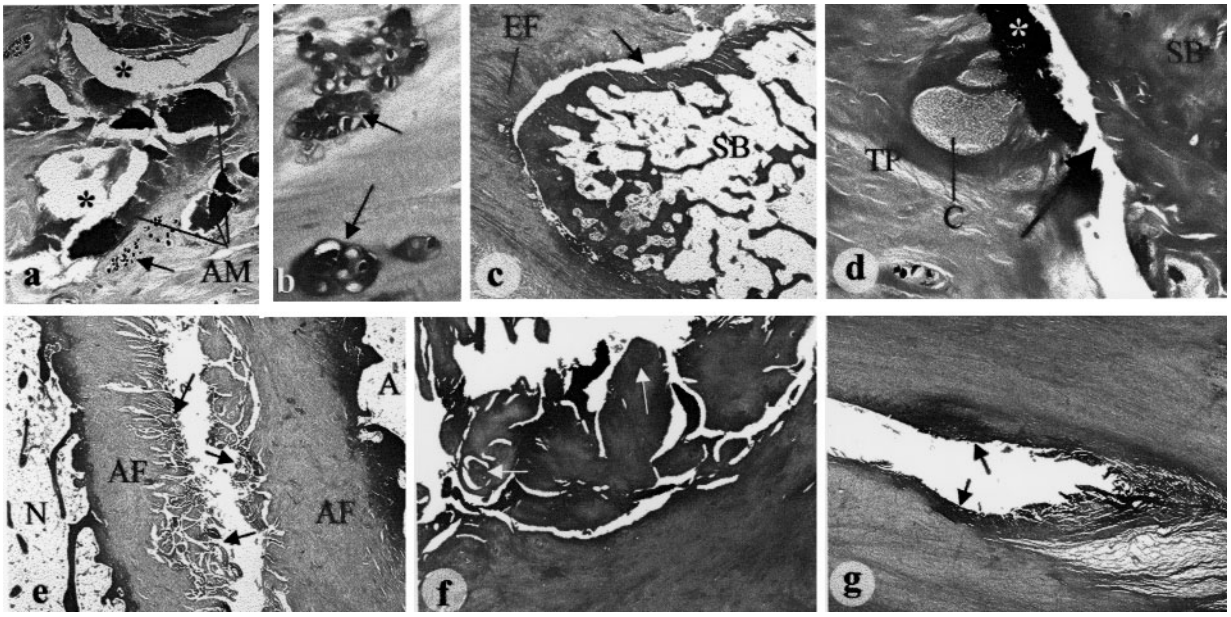

Figure 5. Histopathology of the enthesis organ of tibialis posterior. (a) Fissures (*) containing strongly staining amorphous material (AM) and associated with fibrocartilage cell clusters (arrows) in the enthesis fibrocartilage of the tendon at its attachment to the tuberosity of the navicular (H&E stain; scale bar = 100 µm). (b) High power view of the large clusters of fibrocartilage cells (arrows) found at the tendon enthesis (toluidine blue; scale bar = 50 µm). (c) An extensive tear (arrow) at the interface between the tendon and a sesamoid bone (SB). EF: enthesis fibrocartilage (Masson's trichrome; scale bar = 500 µm). (d) The presence of strongly staining material (*) within the region of a tear (arrow) between a sesamoid bone (SB) and the tendon of tibialis posterior (TP) together with the immediately adjacent cysts (C) suggests that the tears are not artefacts of histological processing (Masson's trichrome; scale bar = 50 µm). (e) A type II accessory bone (A) that forms a true synovial joint with the parent navicular (N). Note the prominent signs of fissuring and fragmentation (arrows) of the articular fibrocartilages (AF) on both sides of the joint that are suggestive of osteoarthritic degeneration (Masson's trichrome; scale bar = 100 µm). (f) A high power view of the degenerative changes in the articular fibrocartilage of a type II accessory navicular bone. Note the fissuring and the presence of cartilage cell clusters (arrows) (toluidine blue; scale bar = 100 µm). (g) Signs of calcification (arrows) in the walls of the deep part of the tendon sheath of tibialis posterior (toluidine blue; scale bar = 500 µm).

occurs in the pathogenesis of pes planus[17]. Finally, adipose tissue is constantly present between the attachment of the tendon and ligament. As fat cells are dominated by their central lipid droplet that is liquid at body temperature, we suggest that the tissue serves both to resist compression and promote independent movement between parts of the enthesis organ.

The prominence of the EF varies greatly according to whether the tendon is predominantly attached to the navicular tuberosity or has many fibers that pass over this protuberance toward other attachment sites in the foot. When the latter happens, the tendon has a bipartite structure with a large fibrocartilage at the osteotendinous junction. We suggest this serves a dual role, not only as an enthesis fibrocartilage that dissipates bending and narrowing forces near the tuberosity, but also as a structure partly equivalent to a periosteal fibrocartilage that protects the bone from pressure that comes from the rest of the tendon that wraps around the navicular tuberosity en route to other tarsal bones. The structure of the tendon here is similar to that near the medial malleolus — another region where degenerative changes in the tendon of tibialis posterior are well documented in the SpA[8,9]. Both regions are fibrocartilaginous, and together with sesamoid fibrocartilages elsewhere in the enthesis organ, they contain type II collagen and aggrecan[18,19]. They could thus be significant targets for an autoimmune response against a fibrocartilage derived antigen — a theory that has been proposed to account for the diversity of anatomical sites targeted in the SpA (see reference 20 for a review). Antigen access to fibrocartilage may be promoted by local defects in the subchondral plate. However, the presence of fibrocartilage at sites commonly targeted in the SpA also signifies a particular pattern of mechanical loading in regions where a combination of tensile, compressive, and shear forces are acting[2]. Thus, it may be that mechanical factors, in combination with microbes, could trigger immune activation and disease in genetically susceptible individuals. Although we lack data on the precise loading pattern of the TP enthesis, the histology and histopathology of the tendon in this region suggest that this target site fits the "biomechanical onset" theory of SpA[2]. Whether the TP enthesis organ is targeted more frequently in patients with SpA where anatomical variations dictate that the majority of the tendon attaches to the navicular tuberosity rather than to other tarsal bones is an intriguing possibility that merits further study. Where there are significant slips of the TP

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tendon attaching to other tarsal bones, it seems reasonable to suggest that biomechanical forces acting specifically at the navicular tuberosity are reduced.

It has long been known that the tendon of TP often has an accessory bone embedded within it and that this can be associated with foot pain[21-23]. We argue that if the accessory bone lies close to the tendon-bone junction, it is part of the enthesis organ, for it contributes to modifying stress distribution as discussed above. Although the distinction between an accessory navicular and a true sesamoid bone is problematical, the majority of clinicians call any bone close to the attachment of the TP tendon an "accessory navicular" and classify them as types I and II according to their size, location, and relationship to the parent bone[10,11,24]. The primary distinction often relies heavily on radiology, but our study shows how a bone that can appear in a radiograph to be a type II accessory navicular may actually turn out to be a sesamoid (i.e., embedded entirely within the tendon and having purely fibrous tissue between it and the parent navicular) if the material is examined histologically. Our study further shows that type II accessory bones can form true synovial joints with the parent navicular — as observed previously[25-27]. Indeed both the type II accessory bones seen in this study showed obvious signs of osteoarthritic degeneration on both sides of the joint. We suggest that such changes are likely to contribute to the spectrum of enthesopathies associated with the presence of an accessory navicular. Whether the existence of accessory bones increases the likelihood of tarsal enthesopathies in patients with SpA is unclear. However, it is obvious from the work of Burgos-Vargas and colleagues[28,29] that the navicular region of the tarsus is a common site for enthesopathy (including large enthesophytes and complete bony bridges) in patients with juvenile onset disease.

Histopathological changes, however, were by no means restricted to specimens showing an accessory navicular. We saw evidence of degeneration at many parts of the enthesis organ (including the osteotendinous junctions of true sesamoids), and suggest that any of the sites could be associated with clinically recognizable enthesopathy. In particular, we draw attention to changes noted in the fibrocartilaginous parts of the TP tendon sheath. The altered staining properties, projecting strands of tendon sheath wall, and the fragmentation of the lining tissues strikingly recall similar features described in the SF and PF of the retrocalcaneal bursa[1]. If small fragments of fibrocartilage detach into the tendon sheath, it is conceivable that they could irritate those parts of the sheath walls that are lined by synovium and trigger inflammation. Thus, it is possible that even where degenerative changes are linked to the presence of an accessory navicular, this does not exclude the possibility that other parts of the enthesis organ could also show signs of wear and tear, and thus cause pain.

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