THE MECHANISM OF FORMATION OF BONY SPURS (ENTHESOPHYTES) IN THE ACHILLES TENDON

INTRODUCTION. Bony spurs (enthophyses) are well documented at the attachments or ‘entheses’ of a wide variety of tendons and ligaments, and are especially common in older people. They appear as irregular outgrowths of varying size that extend from the bone into the adjacent soft tissue and often develop in parallel with osteophytes in articular cartilage [1]. Although practically all tendons or ligaments can develop spurs, they are particularly characteristic of the iliac crest, patella, spine and calcaneus. They can be a feature of degenerative, inflammatory and metabolic enthesopathies, and are widespread in diffuse idiopathic skeletal hyperostosis. Most spurs, however, occur in otherwise healthy individuals and are thus not necessarily an indication of disease - up to 25% of the population can develop heel spurs, with the bone extending from the calcaneus into either the Achilles tendon or the plantar fascia [2]. In a histological survey of Achilles tendons from elderly dissecting room cadavers, spurs were found in 32% [3]. Spurs can be painful, and their presence can lead to disorders of gait and posture [4]. It is commonly suggested that spurs develop as a result of microtears at an entheses, or in response to an inflammatory reaction [4]. The tears are thought to lead to a proliferation of fibroblasts, the appearance of granulomatous tissue and the onset of ossification. It has been suggested that calcaneal spurs develop in response to microtrabecular stress fractures in the underlying bone [5]. To determine the processes involved in formation of bony spurs, we describe the growth, development and ageing of the enthesis of the rat achilles tendon and compare the results with studies of aged human Achilles tendons. We provide clear evidence to show that bony spur formation in the Achilles tendon can occur by endochondral ossification of enthesis fibrocartilage, as an extension of normal growth and development.

MATERIALS AND METHODS. Histology. The insertional region of the Achilles tendon was dissected out from both limbs of 2 white Wistar rats at 2, 3, 4 and 7 weeks of age, and 3 and 12 months. Specimens were fixed for 1 week in 10% neutral buffered formol saline, decalcified in 2% nitric acid, dehydrated in graded alcohols, cleared in Inhibisol and embedded in paraffin wax. Sagittal sections were cut at 10 µm and stained with toluidine blue or Masson’s trichrome. 8 µm sagittal sections of 50 human Achilles tendons used in a previous study [3] were re-examined for detail of bony spur structure. The material was obtained from dissecting room cadavers that had been perfusion-fixed with an emulsing fluid containing 4% formaldehyde and 25% ethanol. After the tendons had been dissected out, they were refixed in 10% neutral buffered formal saline, and processed and stained as above.

Immunohistochemistry. Achilles tendons from 2 and 3 month old white Wistar rats (3 animals per group) were fixed in cold 90% methanol for 2h. Immunohistochemistry was performed using a peroxidase-conjugated anti-mouse Fab fragments (1:100; Sigma, Poole, Dorset, UK) by standard procedures for indirect immunofluorescence using FITC conjugated goat anti-mouse Fab fragments (1:100; Sigma, Poole, Dorset, UK). Control sections were incubated with non-immune mouse immunoglobulins (10 µg/ml; Dakopatts, High Wycombe, Bucks., UK).

RESULTS. Rat tendon. At 2 weeks, the calcaneus was largely composed of the cartilage of the original embryonic rudiment, with the tendon inserting into the cartilage. The calcaneal ossification centre had not yet reached the enthesis, and no blood vessels were present. The onset of fibrocartilaginous metaplasia could be seen in the tendon adjacent to the calcaneus, showing the developing enthesal fibrocartilage. By 3 weeks, the cartilage beneath the enthesis attachment was hypertrophic, with blood vessels invading the chondrocyte lacunae. By 4 weeks the original cartilage of the calcaneal rudiment had been replaced by bone; in the tendon attachment, prominent rows of fibrocartilage cells were now present. The highly vascular bone formed an irregular interface with the fibrocartilage and vascular invasion occurred simultaneously into both the Achilles tendon and the plantar aponeurosis along the rows of fibrocartilage cells. By 7 weeks, large capillaries invading from the bone marrow were seen to throw off smaller sprouts to invade several rows of fibrocartilage cells simultaneously. By 3 months, as the calcaneus continued to enlarge, small bony spurs were present growing into the Achilles tendon. Each contained a prominent central capillary surrounded by newly-deposited bone. The spurs grew along along the rows of fibrocartilage cells, with the vascular tips of actively growing spurs invading fibrocartilage-cell lacunae. Immunolabelling with laminin or type IV collagen antibodies highlighted the basement membranes of bone vessels in the bone and fibrocartilage. Strongly labelled capillaries were seen extending from the bone into the tendon along the rows of fibrocartilage cells. These capillaries were linked to a network of larger blood vessels in the bone marrow.

Human tendon. Bony spurs were frequently seen in the human Achilles tendon. Each was invariably surrounded by a metachromatic region of fibrocartilage and the smaller spurs contained a vascular core in which the long axis of the vessel matched that of the rows of fibrocartilage cells. Larger spurs had a central core of adipose tissue.

DISCUSSION. These results show that bony spurs can develop by endochondral ossification of fibrocartilage at tendon/ligament attachment sites. We have shown that small spurs can develop without any preceding microtears or inflammatory reaction, as an extension of normal enthesis development. Bony spur formation appears to be initiated by vascular invasion into the tendon by capillaries from the underlying bone marrow, without the presence of inflammatory cells. The vessels migrate along the rows of fibrocartilage cells that have developed by metaplasia from tendon fibroblasts. Each fibrocartilage cell then throws off a sprout to invade the walls of the tunnels and a bony spur is formed. Thus, the whole process is essentially an extension of normal bone growth, and is endochondral ossification through fibrocartilage, confirming earlier suggestions of the enthesis acting as a ‘growth plate’ [6]. The structure and location of the small spurs reported here in the human tendons is entirely predictable from the animal studies that show how they develop. In both, spurs are surrounded by enthesis fibrocartilage, there is always a vascular core in a growing spur, and the long axis of the contained blood vessel is parallel to that of the neighbouring rows of fibrocartilage cells.

Our findings clearly indicate that bones grow into tendons during development and not vice versa. Tendon collagen fibres do not continue into the bone lamellae themselves - they are removed during fibrocartilage remodelling, which is then followed by bone deposition. The interface that finally develops is in fact a complex interdigitation of calcified fibrocartilage and bone. This will hold bone and calcified fibrocartilage cartilage together like the interlocking pieces of a jigsaw. Bony spur development increases the surface area of the interface and thus could be an adaptive mechanism to increased mechanical traction.

The growth of bone into tendon in establishing the enthesis has important implications for directing future efforts aimed at promoting the effective surgical reattachment of a tendon or ligament to a bone. Attention should be focussed on encouraging enthesis fibrocartilage formation and its subsequent vascular invasion.


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