

Prevalence and possible pathological significance of calcium phosphate salt accumulation in tendon matrix degeneration.

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Abstract

OBJECTIVES: To investigate the prevalence of calcium phosphate mineral salt accumulation in degenerative supraspinatus 'tendinitis' compared with a normal sample of human tendons, and to determine whether there is an association of calcium salt deposition with pathological changes in the tendon extracellular matrix. **METHODS:** Cadaver tendons (supraspinatus and common biceps tendons, n = 96) and fragments of supraspinatus tendons obtained during shoulder surgery (n = 31) were analysed for calcium content by atomic absorption spectroscopy, phosphorous content using a spectrophotometric assay, and matrix composition (collagen, glycosaminoglycans and DNA) using standard biochemical techniques. **RESULTS:** We established baseline values of calcium concentration in macroscopically normal cadaver tendons (mean 1.1 (SD 0.35) micrograms/mg dry wt, n = 60) and found that 33% (nine of 27) of ruptured tendons from patients with 'degenerative tendinitis' contained an excess of calcium (more than 2SD greater than the normal sample mean). Five of these specimens had increased concentrations of phosphorous and calcium:phosphorous (molar) ratios consistent with a variety of possible calcium crystals, including calcium pyrophosphate, hydroxyapatite, and tricalcium phosphate, in addition to mixed or amorphous calcium phosphate deposits. Four of these specimens contained normal concentrations of phosphorous, consistent with deposits of calcium oxalate or calcium carbonate, although this was not confirmed biochemically. In contrast, surgical specimens (n = 4) from patients with 'calcifying tendinitis' (radiographically detected calcium deposits) all contained salts with a mineral composition consistent with hydroxyapatite. The presence and identity of crystal deposits was subsequently confirmed in five specimens by radiographic microanalysis. Analysis of the tendon matrix demonstrated a number of significant differences between normal and degenerate (ruptured) tendons, including a reduction in collagen content, an increase in sulphated glycosaminoglycans (predominantly dermatan sulphate) and an increase in DNA (cellular) content. However, there were no significant differences between degenerate tendons that were 'calcified' and those degenerate specimens that contained normal concentrations of calcium. **CONCLUSIONS:** Although there was a relatively high prevalence of calcium salts in degenerate tendons, which might contribute to the pathological process (such as increased matrix collagen degradation), these data are consistent with the hypothesis that 'dystrophic calcification' of degenerate tendon matrix is a pathological entity distinct from cell mediated 'calcifying tendinitis'. Calcification is probably one possible outcome (or end point) of chronic tendon injury, although the possibility exists that in many cases, the presence of calcium salts may contribute to the tendon matrix degeneration.

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