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Can a normal Achilles tendon rupture? 4 cases in rugby players examined with UTC

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It is a clinical axiom that spontaneous Achilles rupture is caused by overload of a poor quality (degenerative) tendon. This view is based on biopsies of the Achilles tendon taken immediately after spontaneous rupture; authors have reported degenerative changes such as hypoxic degenerative tendinopathy, mucoid degeneration, tendon lipomatosis and calcific tendinopathy, in almost all cases [1-3]. These pathologies are not acute—hence the conclusion that spontaneous rupture arose in a region of Achilles tendon that was degenerated and thus weakened and predisposed to rupture. However, this histological description is based on the examiners (pathologist) subjective evaluation--not on a validated and standardized description of tendon pathology. Consequently, even though it is tempting to believe that the described changes have been longstanding, there are no scientific proofs backing up that statement. Might these changes be a result of the rupture?

We followed 191 professional rugby players (382 Achilles tendons) over a four-year period using Ultrasound Tissue Characterization (UTC). UTC provides an objective and reliable evaluation of tendon matrix integrity in the Achilles tendon [4]. Contiguous transverse ultrasound images are collated at even distances to create a 3D volume block of ultrasound data. UTC algorithms derived from histopathological samples in equine tendons quantify the stability of grey-scale changes into different echo-types related to matrix integrity. Using this method tendons range in appearance from ('intact' to 'completely disorganized fibrillar matrix') [4]. Tendon structure on UTC is classified as normal if there is less than 5% disorganized tendon structure in a free-standing Achilles tendon. Based on UTC appearance, 274 Achilles tendons were normal and 108 were abnormal.

From this study group we documented 4 cases (2 Caucasian and 2 Afro-American) where the UTC examination demonstrated a normal Achilles tendon prior to a total rupture in the Achilles midportion. The ruptures occurred 6 months (3 players) and 12 months after the scans. In these cases, UTC showed less than 1% disorganization in 3 players and less than 4% disorganization in one player. In our series there were no ruptures among the abnormal Achilles tendons.

Our longitudinal study challenges the dogma that tendon degeneration is a prerequisite for rupture.

We acknowledge that the 4 players in our series may have developed tendinosis in the 6-12 months between the UTC study and the Achilles tendon rupture, but we believe that is unlikely as we have a material on professional rugby players who had UTC scans several times during a 3-year period and among the normal tendons there were only very minor changes over time. We believe larger longitudinal studies of at-risk populations using UTC may help identify whether a normal Achilles tendon can rupture.

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