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Avulsion of the triceps tendon in secondary hyperparathyroidism

A case report

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We report a case of avulsion of the triceps brachii tendon in a patient with secondary hyperparathyroidism due to hemodialysis. A convulsion led to a stress rupture of the tendon, already weakened by degenerative changes. After surgical repair the patient regained nearly normal range of motion and strength.

There are 26 well-documented cases of triceps tendon avulsion in the English literature, 4 of which were secondary to hyperparathyroidism. Preston & Adicoff reported in 1962 a triceps tendon avulsion as a complication of hyperparathyroidism. Others have pointed out the risk of such injuries in patients with renal failure (Preston 1972, Cirincione & Baker 1975, Lotem et al. 1978, Fery et al. 1978, Farrar & Lippert 1981). We describe a case with impairment in strength of a calcified degenerated tendon where the rupture was successfully treated.

Case report

A 66-year-old man was admitted to our clinic because of pain and loss of active extension of his right elbow after an avulsion. He suffered from a tuberculous-induced renal insufficiency treated by hemodialysis. Three weeks before admission, his parathyroid glands had been resected because of secondary hyperparathyroidism, and a part the gland was implanted in the right forearm.

Examination of the right elbow revealed ecchymosis and a subcutaneous soft-tissue defect just proximal to the olecranon. Flexion and extension in the horizontal plane were normal, but active extension against gravity was impossible. Radiographic examination showed an irregular olecranon and calcification of the tissues about 3 cm more proximally. These calcifications were considered to be avulsed cortical bone from the olecranon.

At surgery the triceps tendon was found to be completely avulsed from the olecranon, but with no cortical defect. The distal end of the tendon showed degeneration and widespread calcification. The tendon was repaired with a thick nylon strip placed through drill holes in the olecranon and through the triceps tendon 2.5 cm proximal to the rupture. At follow-up 6 months later, the range of motion had normalized, with only 5 degrees of restricted extension. Histologic examination showed degeneration of the tendon with calcification, vascular ingrowth, and an irregular pattern of collagen fibers.

Discussion

Anzel et al. (1959) analyzed 1,014 miscellaneous muscle and tendon disruptions. There were 170 stress ruptures in which the triceps tendon was involved in 1.2 per cent. In most cases of tendon rupture associated with secondary hyperparathyroidism, the quadriceps tendon is involved (Preston & Adicoff 1962, Murphy & McPhee 1965, Preston 1972, Cirincione & Baker 1975, Lotem et al. 1978, Fery et al. 1978). Only five triceps tendon avulsions in 4 patients (Preston & Adicoff 1962, Murphy & McPhee 1965, Lotem et al. 1978, Farrar & Lippert 1981) and one triceps tendon

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rupture (Searfoss et al. 1976) have been documented in association with hyperparathyroidism. Murphy & McPhee (1965) described one of these cases with extensive changes of the triceps tendon due to secondary hyperparathyroidism. They found proliferation of elastic fibers, and attributed the tendon weakness to this elastosis as a consequence of chronic metabolic acidosis. In our patient no increase in elastic fibers was found. Preston & Adicoff (1962) suggested that the tendons may be weakened by calcium deposition associated with elevated serum calcium and phosphate levels. Lotem et al. (1978) assumed that changes occur in the protein polysaccaride complex that binds collagen fibers. Weakness of this bone may allow a sliding of collagen fibers to take place.

Preston (1972) suggested that subperiosteal bone resorption may weaken the fibro-osseous junction between tendon and bone. Probably there are changes in the tendon, as well as in the subperiosteal bone (Farrar & Lippert 1981). In most cases a fall upon the outstretched hand is the cause of the triceps tendon rupture (Murphy & McPhee 1965, Pantazopoulos et al. 1975, Levy et al. 1978, Farrar & Lippert 1981), sometimes in combination with a fracture at the radial head (Levy et al. 1978, Levy et al. 1982).

A triceps tendon rupture caused by an epileptic seizure and in combination with secondary hyperparathyroidism has been previously described only once (Searfoss et al. 1976).

The diagnostic value of avulsed bone on the lateral roentgenogram has been emphasized in the literature (Bennett 1962, Tarsney 1972, Pantazopoulos et al. 1975, Farrar & Lippert 1981, Levy et al. 1982). In our patient, however, the calcification was located in the tendon and the cortical bone of the olecranon was undamaged.

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