RUPTURE OF PATELLAR LIGAMENT AFTER STEROID INFILTRATION
Report of a Case

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A man aged 25 years, a high jumper, developed pain over the left patellar ligament in March 1966. In August 1966 there was tenderness over the ligament but no swelling. He was given short-wave diathermy, but when this failed to relieve his symptoms the ligament was infiltrated with 75 milligrams of hydrocortisone with 2 millilitres of 2 per cent lignocaine in September 1966. This gave considerable relief, but the pain returned and persisted in milder degree, becoming worse whenever he was training. In October 1966 he was given triamcinolone with Xylocaine injected into the area around the insertion of the ligament. Relief of pain was again temporary, but he continued jumping. He was seen again in the following year with the same complaint. Infiltration was repeated on August 11, 1967, and on August 23, 1967.

In November 1967 during a training session he had cleared 1·84 metres and was attempting to clear 1·89 metres when an accident occurred. He had planted his left foot for take off at the end of his run and was on the point of lifting his right leg up when the left foot slipped. He felt something give in his left knee and fell on to his buttock. He had severe pain and he was unable to get up or to extend his knee.

The mechanism by which the patellar ligament ruptured was the forceful stretching of the quadriceps, which was contracting in order to thrust his weight up over the bar. Because of the slipping of the left foot, the altered centre of gravity caused the body weight to flex the knee against the muscular pull of the quadriceps, thus causing the rupture.

On examination, he was in pain and unable to move the left knee. Passive movement caused severe pain. The patella was riding high above the knee and was abnormally mobile from side to side and upwards. Radiographs showed a high patella: there was no sign of fracture of the patella or of any break in the periosteal continuity of the tibial tubercle.

At operation, the patellar ligament was found to be ruptured through its substance about two and a half centimetres above its insertion. The tear extended laterally through the joint capsule and the anterior part of the lateral ligament. The patellar ligament was sutured end-to-end with chromic catgut. The joint capsule and lateral ligament were repaired separately.

The leg was immobilised in plaster in full extension. The plaster was removed six weeks after the operation and active movements were begun. A month later he had a range of 20 to 110 degrees. Six months after operation there was a full range of movement, with no extension lag, but the quadriceps was still wasted. He walked without a limp but had not resumed jumping.

DISCUSSION

Rupture through the substance of the patellar ligament as distinct from rupture at the insertion is rare. In a review of the literature we have not been able to find any instance of rupture of a patellar ligament after infiltration with steroids.

Disruption of the extensor mechanism of the knee most often occurs at the musculo-tendinous junction above the patella or its insertion to the tibial tubercle. With sudden flexion of the knee against a contracting quadriceps, it is usually the patella that gives way across the femoral condyle. McMaster (1933) in a review of the literature could not find a single case
which he considered to be a rupture through the patellar ligament. He noted that Cotton (1910) mentioned the rare possibility, and that Wagner (1927) described a case of complete rupture of the infrapatellar ligament and adjacent capsular ligament which apparently was a tearing of the ligament at its insertion.

In his experiments with rabbits McMaster noted that approximately one-half of a tendon’s fibres had to be severed before rupture occurred with severe strain. He concluded that normal musculo-tendinous systems do not rupture when subjected to severe strain, but that a tendon insertion could be pulled away, the muscle belly ruptured, the musculo-tendinous junction severed or even a fracture sustained.

Anzel, Covey, Weiner and Lipscomb (1959) described fifty-four disruptions of the quadriceps femoris collected from the files of the Mayo Clinic for the period 1945 to 1954. Of these 40.1 per cent were due to indirect stress and 38.9 per cent were due to direct trauma. Slightly more than one-third of these disruptions were in the patellar ligament but there is no indication in his report that any of them occurred through its substance.

Mayer (1961) reported a case of spontaneous bilateral rupture of the calcaneal tendon in “a perfectly fit woman of forty-six.” The rupture occurred about five centimetres above the insertion of the tendons.

Corticosteroids act primarily on tissues of mesenchymal origin. In a healing wound corticosteroids prevent the ground substance precursor of collagen from being sulphated (Walter and Israel 1965). Histologically, the fibroblasts as well as blood vessels are scarce and maturation of fibrous tissues is delayed (Florey and Jennings 1962).

Experimental evidence is conflicting. Gonzalez (1953) and Berkin (1955) found that tendon union was not affected by steroids; while Wrenn, Goldner and Markee (1954) in experiments on dogs found that the tensile strength of sutured tendons treated with cortisone was 40 per cent less than controls.

Several cases of rupture of the calcaneal tendon in patients on oral corticosteroids therapy have been reported (Cowan and Alexander 1961, Lee 1961, Smaill 1961, Melmed 1965). Lee (1957) described a case of rupture of the calcaneal tendon following local infiltration with hydrocortisone and argued that the injections may have hindered repair.

In the case described the cause of the pain in the first instance is not known, but the provisional diagnosis of “tendinitis” was made. The use of corticosteroids may have reduced the tensile strength of the patellar ligament to the extent that linear tension caused it to rupture through its substance.

**SUMMARY**

1. A case of disruption of patellar ligament is described.
2. An association with previous steroid infiltration is present in this case.

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**REFERENCES**


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