Clinical Observations

Spontaneous rupture of the quadriceps tendon and patellar ligament during treatment for chronic renal failure

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The quadriceps tendon and patellar ligament rupture rarely, even when under great stress or trauma, but can rupture spontaneously in patients with chronic diseases such as gout, rheumatoid arthritis and renal disease. Several factors probably combine to weaken the tendon, including an impoverished local vascular supply, repeated microtrauma and secondary hypoparathyroidism with osteodystrophy. In the three cases reported here, one of which was bilateral, the patients were being treated for chronic renal disease; surgical repair of the tendons led to sound healing and a return to normal function of the joints.

La rupture du tendon du quadriceps ou du ligament rotulien est rare, même quand celui-ci est soumis à un stress important ou à un traumatisme; toutefois, il peut y avoir rupture spontanée chez les patients souffrant de maladies chroniques telles que la goutte, la polyarthrite rhumatoïde et les maladies rénales. Plusieurs facteurs s'associent probablement pour affaiblir le tendon, y compris un appauvrissement de l'apport vasculaire local, des microtraumatismes répétés et une hypoparathyroïdie secondaire avec ostéodystrophie. Dans les trois cas décrits ici, dont un bilatéral, les patients étaient tous traités pour maladie rénale chronique. La réparation chirurgicale des tendons a procuré une bonne cicatrisation et un retour au fonctionnement normal des articulations.

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Rupture of the quadriceps tendon or patellar ligament is a relatively rare condition. It sometimes occurs in otherwise healthy individuals under severe stress or trauma,[1] but usually an underlying systemic disease can be found, such as lupus erythematosus,[2] rheumatoid arthritis,[3] gout,[4] hyperparathyroidism,[5] tuberculosis, vasculitis[6] or chronic renal disease.[7]

We report a case of spontaneous and simultaneous rupture of the patellar ligament in one knee and the quadriceps tendon in the other, as well as two further cases of spontaneous rupture of these tendons in dialysis patients. Bilateral rupture in dialysis patients has been reported only once before.[7,8]

Case reports

Case 1

A 27-year-old man who had suffered renal disease from the age of 2 years first required dialysis in 1975 for acute renal failure and in 1978 was introduced to long-term ambulatory peritoneal dialysis. In 1981, while undertaking light lifting, he experienced sharp pain in his knees and both gave way. He was then unable to actively extend his knees.

We found that on the right the patella was riding notably high, owing to rupture of the patellar ligament, while there was a palpable defect just proximal to the patella in his left knee, indicating rupture of the quadriceps tendon. The results of the laboratory investigations for this and the other two patients are reported in Table I.

Bilateral reconstruction of the ligament and tendon was carried out with Bunnell-type suturing, and the repairs were protected in cylinder casts for 6 weeks. Six months later the patient had regained full motion and had returned to work.

Case 2

A 32-year-old man who was known to have had glomerulonephritis since the age of 16 years had been receiving dialysis for 2 years. One evening, when simply stepping

Table I—Representative laboratory values obtained from the blood of three patients with chronic renal disease who suffered a spontaneous rupture of the patellar ligament or the quadriceps tendon or both

	Case no.		
Level	1	2	3
Hemoglobin (mg/dl)	7.8	7.4	8.9
Hematocrit (%)	17.0	–	25.0
Sodium (mmol/l)	132.0	139.0	133.0
Potassium (mmol/l)	5.8	4.9	3.8
Chloride (mmol/l)	99.0	102.0	92.0
Creatinine (mg/dl)	19.5	19.8	6.8
Urea nitrogen (mg/dl)	79.0	75.0	55.0
Calcium (mg/dl)	8.8	9.7	10.8
Phosphorus (mg/dl)	5.6	5.5	4.5
Uric acid (mg/dl)	–	75.0	–

out of bed, his left knee suddenly gave way, and he found himself unable to extend the knee or bear weight on it.

We found that his quadriceps tendon had ruptured just proximal to the patella. Following repair, a cylinder cast was applied for 6 weeks, and full recovery ensued.

Case 3

A 50-year-old woman with congenital horseshoe kidney had been receiving dialysis since 1973 and had had two unsuccessful renal transplants in 1976 and 1979. She had marked renal osteodystrophy, with bone biopsies repeatedly confirming moderately severe osteomalacia. The incident causing her injury was also relatively trivial, with her left knee simply giving way as she tried to pick up an object off the floor. She found herself unable to extend the knee or bear weight on it.

Rupture of the patellar ligament was identified upon presentation at the hospital, with a small avulsed fragment of bone showing on a roentgenogram. The defect was repaired with Bunnell-type sutures and protected with a splint for 6 weeks. During her stay in hospital a partial parathyroidectomy was performed. Nine months postoperatively the patient's knee was well mobilized, with a range of flexion of 110°.

Discussion

Conway[9] found that rupture of the quadriceps tendon was first mentioned by Galen, and that the first cases of bilateral rupture were reported by Sonnenschein[10] and Frey[11] in 1927 and 1928. Most of the early papers on this subject paid attention to the mechanical aspect of the injury and different modes of repair,[12-15] but as early as 1933 emphasis was placed on the role of concurrent systemic diseases in predisposing to rupture.[16]

As in our patients, most such injuries are suffered when the patient, standing with the knees slightly flexed, contracts the quadriceps muscles in an attempt to avoid falling or to lift something or simply in descending stairs.[9] In people under the age of 50 to 60 years the patella presents the weakest link in the quadriceps mechanism, fracturing 50 to 60 times more frequently than the tendon ruptures.[15] Indirect trauma accounts for more ruptures of the quadriceps tendon than does direct,[1] and the site of the rupture is suprapatellar in two thirds of the cases and infrapatellar (i.e., in the patellar ligament) in one third. McMaster[16] showed that normal tendon would not break even if half severed until the load reached about 10 to 15 kp/mm^2 — a load at which the belly of the muscle, its osseotendinous insertion and even the femur would fail first.[8,16]

The changes occurring in tendon in old age include tendinosclerosis, fatty degeneration of the fibres,[15] a decrease in the proportion of collagen and proteoglycan, and an increase in the proportion of elastin.[17] The wavy pattern of the collagen fibres, which are bound in fascicles, is also lost, and the fibres are replaced by linear hyalinized fibrils.[17] Increased levels of collagenase, as found in rheumatoid arthritis, may also play a role in tendon degeneration.[3]

It appears that the blood supply of tendon is of great importance to its strength. Tendon is supplied by arterioles from the attached muscle and connective tissue, the intrinsic vessels being in the intrafascicular spaces. At the bony insertion there is a network of capillaries, but the blood supply from the bone is insignificant.[17] Any ischemic episode will predispose this site to rupture. In patients with ruptured tendons there are degenerative fibrils with necrobiotic changes and coexisting inflammatory and regenerative reactions.[18] There is fibrinoid degeneration, with an infiltration of mononuclear cells, some vessels being thrombosed. Davidsson and Salo[18] speculated that this represents a state in which the tendon, having received multiple microtrauma, is in different stages of repair. Under such conditions the tendon's blood supply is jeopardized. Since, like muscle, this tissue's demand for oxygen increases during any strenuous activity, the damaged tendon is predisposed to rupture. Anzel and colleagues[1] judged that this hypothesis was the most likely explanation for the injury in athletes and workmen and observed that the quadriceps tendon was the one most likely to rupture in the lower extremity, followed by the Achilles tendon. Other changes in the tendon vessels can be important. In a case of arthritis in which a tendon ruptured, there was endothelial swelling, with a perivascular lymphocytic exudate from the small vessels, and in a case of systemic lupus erythematosus a perivascular infiltrate of mononuclear cells was seen.[2]

In chronic renal disease many factors can account for the tendon's tendency to rupture. Secondary hyperparathyroidism accompanied by osteodystrophy could lead to calcification and, possibly, to a change in the highly polymerized glycoprotein matrix.[2,5] Subperiosteal bone resorption due to secondary hyperparathyroidism could facilitate disruption, with a flake fracture, as happened in our third case. The elastosis associated with renal acidosis may also be a major factor in the tendon-weakening process. From a single case of gout it was suggested that the deposition of uric acid crystals might play a role, although the structural changes and disturbances accompanying uremia[4] may have more significance.

In some cases the degenerative changes in tendon in patients with systemic lupus erythematosus have appeared without there being any inflammatory reaction or mononuclear cell infiltrates. In all of these cases corticosteroids had been administered.[2,19] Tendon that is injected with steroids is prone to avulsion,[20] although the exact mechanism is still not known. It has been shown, however, that the repair mechanisms of the mononuclear cell reaction are suppressed where fibroblasts are active and capillary buds are sprouting. This would explain why no inflammatory changes could be found in the ruptured tendons of lupus patients who were treated with steroids. One might conclude, therefore, that steroid treatment could be a factor in tendon rupture even though the injury has not been reported in patients given rather high doses of steroids in the treatment of asthma or ulcerative colitis.[2]

Conclusion

The quadriceps tendon and patellar ligament are susceptible to rupture in patients with chronic renal disease. The principal factors contributing to this rupture include an impoverished vascular supply, repeated microtrauma and secondary hyperparathyroidism with osteodystrophy. Since early repair is essential to a good result[6,9,15] one should be aware of the diagnosis, which may be confirmed by the loss of knee extension and a palpable gap in the tendon. There are many methods of repair that enable the patient to return to normal activities within 6 months.[6,12,14,15]

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Cimetidine-resistant Zollinger–Ellison syndrome: successful management with ranitidine

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In a patient with the Zollinger–Ellison syndrome who had undergone vagotomy, pyloroplasty and, later, gastrojejunostomy, epigastric pain and stomal ulceration recurred despite the use of high doses (2700 mg/d) of cimetidine. Ranitidine, a new histamine H_2-receptor antagonist, reversed all symptoms and healed the stomal ulcer without side effects, thus obviating the need for further surgery. Ranitidine may prove to be the drug of choice in the medical management of patients with the Zollinger–Ellison syndrome.

Chez un patient atteint du syndrome de Zollinger–Ellison et qui avait subi une vagotomie, une pyloroplastie et, plus tard, une gastrojéjunostomie, il y eut récidive des douleurs épigastriques et des ulcères peptiques malgré l'utilisation de fortes doses (2700 mg/j) de cimétidine. La ranitidine, un nouvel inhibiteur des récepteurs histaminiques H_2, a fait disparaître tous les symptômes et cicatrisé l'ulcère peptique sans effets secondaires, rendant de la sorte inutile toute autre chirurgie. La ranitidine pourrait s'avérer le médicament de premier choix dans le traitement non chirurgical des patients souffrant du syndrome de Zollinger–Ellison.

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The Zollinger–Ellison syndrome, a disease characterized by intractable and potentially life-threatening hypersecretion of gastric acid, has traditionally been managed with total gastrectomy.[1] This procedure provides only symptomatic relief, though, and it carries an appreciable morbidity and mortality.[2] With the discovery of the histamine H_2-receptor antagonist cimetidine, a potent