Case report

Insidious bilateral infrapatellar tendon rupture in a patient with systemic lupus erythematosus

LEO M. COONEY Jr., JOHN M. AVERSA, AND JAMES H. NEWMAN

From the Department of Internal Medicine, Yale University School of Medicine, New Haven, Connecticut, USA

SUMMARY A patient with systemic lupus erythematosus developed insidious bilateral infrapatellar tendon rupture initially diagnosed as steroid myopathy. Simultaneous loss of extension at the knee due to quadriceps or infrapatellar tendon ruptures is reviewed.

Simultaneous or near simultaneous rupture of both quadriceps or intra-patellar tendons is an unusual occurrence characterised by the sudden loss of knee extension. The majority of the cases reported have had 'connective tissue diseases'1–8 or hyperparathyroidism9–12 and have responded well to surgical repair. We report here a patient with systemic lupus erythematosus whose 'steroid myopathy' was due to insidious bilateral infrapatellar tendon rupture.

Case report

The patient was a 51-year-old Caucasian female with an 18-year history of arthritis. Three years before admission to hospital the diagnosis of systemic lupus erythematosus was made on the basis of fever, pleuritis, pericarditis, alopecia, neutropenia, deforming nonerosive arthritis, and a positive antinuclear antibody and LE preparation. Two sisters were known to have idiopathic thrombocytopeptic purpura. Prednisone 20 mg per day was started, with improvement of the above signs and symptoms. Six months before admission the patient noted slowly progressive generalised weakness, which was more pronounced in the lower legs. She had great difficulty going from sitting to standing positions.

The patient was admitted to the hospital because of an infected olecranon bursa due to Staphylococcus aureus. Weakness led to the diagnosis of steroid myopathy, and prednisone was slowly tapered down. During her hospital course she developed high fevers and pulmonary infiltrates, which after extensive tests were attributed to lupus pneumonitis.

On transfer to the rehabilitation service physical examination revealed a chronically ill white woman with normal vital signs. A severe dermatomyositis infection was present over the hands, feet, and nails. The mucosae were normal. There was no lymphadenopathy. The chest was clear. Cardiac examination was normal. The abdomen was normal, without enlargement of any organ. Neurological examination gave normal results. Musculoskeletal examination was remarkable for reducible swan neck and ulnar deviation deformities of both hands. There was marked proximal muscle wasting of both the upper and lower limbs. There was grade 4 out of 5 muscle strength of the shoulder abductors and arm flexors. Examination of the lower limbs revealed high riding patellae bilaterally with marked atrophy of both quadriceps muscles. Contraction of the quadriceps muscles resulted in movement of the patellae but no active extension of the legs.

Laboratory examinations gave a haemoglobin of 8.2 g/dl, haematocrit of 25.4, a white blood cell count of 4.8 × 10⁹/l (64% polymorphonuclear leucocytes, 3% bands, 24% lymphocytes, 3% monocytes, 2% eosinophils), platelets 620 × 10⁹/l, and an erythrocyte sedimentation rate (Wintrobe) of 46 mm/h. Urine analysis was normal. The creatinine was 1.0 mg/dl (normal range 0.6–1.5; SI: 88 μmol/l), blood urea nitrogen 25 mg/dl (normal range 8–18; SI: 4 mmol/l), potassium 5.2 mEq/l (normal range 3.6–4.6; SI: 5.2 mmol/l), sodium 140 mEq/l (normal range 134–144; SI: 140 mmol/l), chloride 104 mEq/l (normal range 100–106; SI: 104 mmol/l), bicarbonate 21.5 mEq/l (normal range 25.1–30.5; SI: 21.5 mmol/l), calcium 8.8 mg/dl (normal range

Accepted for publication 16 November 1979.
Correspondence Leo M Cooney Jr., MD, Yale University School of Medicine, Department of Internal Medicine, Section of Rheumatology, 333 Cedar Street, 8 East Memorial Unit, New Haven, CT 06510, USA.
9·1–10·6; SI: 2·2 mmol/l), phosphorus 4·3 mg/dl (normal range 3·1–4·5; SI: 1·4 mmol/l), magnesium 2·4 mg/dl (normal range 1·8–3·0; SI: 0·99 mmol/l), creatinine phosphokinase 12 (range 0·6–1·5).

The antinuclear antibody was positive at a titre of 1:1020 in a homogeneous pattern. The DNA binding protein was 6·0%, C3 was 64·0 (normal of 78 to 150), and C4 20·0 (normal of 21 to 50). The VDRL test was nonreactive and the rheumatoid factor test was negative. X-rays of the knees revealed subchondral demineralisation of the distal femurs, patellas, and proximal tibias, vertical dislocation of the patellas, and gross contraction and deformity of the main quadriceps tendons. Superficial appearances are shown in Figs. 1 and 2.

**FINDINGS AT OPERATION**

Both knees had 20° of passive hyperextension and 145° of flexion. The cruciate and collateral ligments were stable. The patellae rode superiorly to the femoral condyles and were easily reducible to their anatomical positions. The patellar tendons were in continuity with the inferior poles of the patellae and the tibial tubercles, and their fibres were attenuated in appearance, with scar interspersed. The attenuated tendons were imbricated on themselves, with chronic catgut sutures holding the patellae in their anatomical positions. The knees were immobilised in a neutral position in cylinder casts.

**Discussion**

We have found 33 reported cases of bilateral quadriceps tendon rupture occurring simultaneously or sequentially within a few days. The onset in all previous cases was acute and characterised by sudden inability to extend the lower legs at the knees. Fifteen of these patients were otherwise healthy, while the remainder had either systemic lupus erythematosus, rheumatoid arthritis, or primary hyperparathyroidism.

The 15 healthy patients had all had injuries to the legs while the knees were slightly flexed. Pain was severe, and effusions of blood in the knee joints were frequent. Failure to extend at the knees, ability to flex the knees after the legs had been extended passively, and suprapatellar depressions were always present on physical examination. At surgical exploration the quadriceps tendon above the patella at the musculotendinous junction was always involved. The response to surgery was always described as good, with return to full ability to change position and walk.

With our patient 7 have now been reported with systemic lupus erythematosus and bilateral rupture of the infrapatellar tendons. They are compared with the healthy group in Table 1. The ruptures of the tendons in these patients always occurred in the infrapatellar regions at the tendino-osseous junction. Physical examination showed high-riding patellae which moved on active quadriceps contraction and a groove along the proximal anterior tibia.

Pain, although present in all patients, was not prominent in 4 of 7. Rupture occurred in the absence of appreciable trauma in 5 of the 7 patients.

All of these patients were on chronic corticosteroids in varying doses. There are, however, no reports of infrapatellar tendon rupture in patients on high-dose corticosteroids because of asthma, skin disease, lymphoproliferative disease, or other conditions. Most observers therefore believe that the rupture of the tendon is due to the disease process itself or to the corticosteroids added to the basic disease process.

The remainder of the patients reported with bilateral quadriceps tendon rupture included 2 wit
primary hyperparathyroidism, 6 with secondary hyperparathyroidism, 2 with rheumatoid arthritis, and 1 with gout. They all had similar clinical presentations. The ruptures were always acute and pain was prominent at presentation. Injury was always associated with these ruptures. They occurred in both the suprapatellar and infrapatellar tendons. Calcification of the tendons occurred in several of the patients with primary or secondary hyperparathyroidism.

Quadriceps tendon rupture is now being recognised more frequently in patients with systemic lupus erythematosus on chronic steroid therapy. It causes marked disability because patients are unable to change from a sitting or standing position and have great difficulty with walking. This lesion is quite remediable to surgical repair, and all reported patients have had good results with return to good function of the lower limb.

The diagnosis has not been difficult in previously reported cases, as the patients had sudden onset of pain, inability to extend their knees, and high-riding patellae. Our patient's insidious course shows the value of careful examination of patients with muscular weakness of the lower limbs to identify this surgically correctable cause of quadriceps dysfunction. This insidious tendon rupture should be included in the differential diagnosis of patients with systemic lupus erythematosus who present with profound weakness of proximal muscles. The high-riding patellae on physical examination and on x-ray, the good movement of the patellae with quadriceps contraction without extension of the lower leg, and the grooves noted on physical examination of the infrapatellar tendon should all alert the clinician to this diagnosis.

References


Insidious bilateral infrapatellar endon rupture in a patient with systemic lupus erythematosus

22 Wetzler S H, Merkow W. Bilateral, simultaneous and spontaneous rupture of the quadriceps tendon. JAMA 1950; 144: 615.