

Case report

Multiple tendon rupture in systemic lupus erythematosus: case report and review of the literature

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SUMMARY Tendon rupture in systemic lupus erythematosus (SLE) is a rare complication that appears to occur in patients receiving corticosteroid therapy. A case is presented with sequential bilateral rupture of Achilles tendon and unilateral rupture of a patellar tendon. Six more published cases are reviewed. Tendon rupture in SLE has affected both males and females between the ages of 24 and 63 years. It occurred exclusively in the weight-bearing tendons: in 6 instances the patellar tendon was severed, in 2 the Achilles, and in one the quadriceps. All 7 patients were on corticosteroid therapy at or just before the time of injury. The deleterious effect of this treatment in loosening connective tissue is discussed.

Rupture of tendons in systemic lupus erythematosus (SLE) is rare. Only a few cases have been published.¹⁻⁶ The rupture seems to occur exclusively in those tendons which bear great weight. Some reports described the rupture of the patellar tendon,^{1 3-6} while one other described tears in the Achilles tendon.² The common factor in all the cases apart from the SLE that most probably caused the rupture was the administration of corticosteroids.

This report concerns an unusual case of sequential bilateral rupture of the Achilles tendons and unilateral rupture of a patellar tendon in a patient with SLE.

Case report

A 24-year-old female diagnosed as suffering from SLE 5 years ago also had symptoms of arthritis and nephritis. In the initial phase of her illness she was treated with steroids and azathioprine, which produced a remission for one year. Subsequently she developed arthritis of both knees, which did not improve on treatment with the first-line anti-inflammatory drugs. A programme of 60 mg per day of prednisone was started, with gradual tapering.

Three months later (while the patient was off steroids) she was admitted to hospital with pleuritis and nephritis. At this stage 30 mg per day of prednisone was administered. A month later pain and swelling in the left knee necessitated plaster fixation for about 3 weeks.

One week after the plaster was removed she felt a sharp pain in the left ankle while standing on her toes. Instinctively she moved her weight to the right leg and felt the same pain in the right ankle. Despite the pain and malfunctioning of both ankles she did not consult a physician for one month. Examination at our clinic revealed bilateral wasting of the area of the Achilles tendons and incapacity to stand on her toes. She was transferred to the Orthopedic Department, where on operation a bilateral tear of the Achilles tendons was noted, with both proximal and distal degeneration of the tendon remnants. About 3 cm of tissue was missing on each side. The tears were repaired. Histopathological examination of both edges showed marked proliferation of blood vessels, with perivascular mononuclear infiltrate and in the adjacent muscle vacuolar myopathy and degeneration. Recovery following operation was uneventful and treatment with corticosteroids was not renewed.

Eighteen months later she was seen at the outpatient clinic complaining of abdominal pain and fever. Physical and laboratory findings were consis-

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tent with nephritis. Treatment with 60 mg of prednisone per day was started, with gradual tapering to 15 mg. On this dose while alighting from a bus 3 months later she felt a sharp pain in her right knee and was unable to stand on her right leg. Examination revealed a 'high-placed' patella which moved proximally on contraction of the quadriceps. The patient was unable to perform straight leg-raising of the right leg. On the same day she underwent surgery under spinal anaesthesia. During the operation the patellar tendon was found to be severed for 90% of its width. Repair was accomplished by suturing the proximal remnant of the tendon to the tibial tuberosity with wire. The leg was put in a plaster cast and the patient was released.

Discussion

Spontaneous rupture of a tendon is rare. McMaster, and later others, showed that an artificial tear of 75% of a tendon's width would be unlikely to cause a complete rupture unless some other pathological process exists.⁷⁻⁸ Collagen disease and corticosteroid therapy, either singly or together, provide suitable conditions for such a rupture.

Reports of tendon rupture caused by corticosteroid therapy have appeared since the advent of these agents. In a few cases the corticosteroid was injected directly into the tendon,⁹ while in others it was given by mouth.¹⁰ In addition, corticosteroids have caused derangements in collagen tissue following topical absorption through the skin.¹¹ They affect

collagen tissue at 3 levels: they exert an antimittotic effect on fibroblasts¹²; have an antisynthetic effect on fibroblasts when topically applied¹³; and they stimulate collagenase.¹⁴ Experimental evidence exists showing that hydrocortisone injected into rabbits' tendons or muscle tissues resulted in breakage of collagen fibres within a short time.¹⁵⁻¹⁶ In one experiment¹⁵ injection of hydrocortisone into the rabbits' Achilles tendon was accompanied by loss of periodic binding, and the collagen fibrils became kinked, shrunken, and finally a structureless mass. Several athletes treated with intratendinous corticosteroid injection to relieve pain experienced rupture of the tendons.⁹⁻¹⁷⁻¹⁸

Six cases of tendon rupture in SLE have been previously reported.¹⁻⁶ Some of their clinical aspects are shown in Table 1.

The ages of the patients varied from 24 years (in our case) to 63 years.⁴ In most instances some kind of stress was placed on the tendon and caused it to tear. The stress might have come from the simple act of walking in the case of an overweight patient, jumping a rope, or descending from a bus.

Accurate data on the time lapse from starting corticosteroids to tendon rupture were not available for all reported cases. However, in one case it was 3 days after cessation of treatment,² while in another it followed 10 years continuous treatment.⁴⁻⁵ In 6 out of 7 cases one or 2 of the patellar tendons were ruptured. In only one instance,² a case with discoid lupus, were both Achilles tendons torn. The present case is the only one with sequential rupture of both the

Table 1 Tendon rupture in systemic lupus erythematosus

Reference	Age/sex	Tendon(s)	Inciting event	Treatment at time of rupture	Pathological findings
Martin <i>et al.</i> ¹	31/M	Patellar (B)*	Jump	25mg/day prednisone	Degeneration, mononuclear infiltration
Cowan and Alexander ²	52/M	Achilles (B)	Walking	16/ mg/day triamcinolone	—
Twining <i>et al.</i> ³	46/M	Patellar and quadriceps	Walking	60 mg/day prednisone	Degeneration, no inflammation
Strejček and Popelka ⁴	63/F	Patellar (B)	Descending from bed	5 mg/day prednisone	—
Wener and Schein ⁵	40/F	Patellar	Walking (overweight)	30 mg/day prednisone	Neovascularisation, chronic degeneration and inflammation
Rascher <i>et al.</i> ⁶	45/F	Patellar (B)	Rope jumping	5 mg/day prednisone	—
Potasman and Bassan	24/F	Achilles (B) and patellar	Tiptoeing, descending from bus	15 mg/day prednisone	Perivascular mononuclear infiltrate, neovascularisation, degeneration, vacuolar myopathy

*B=bilateral.

Achilles and patellar tendons. In both instances our patient was taking oral corticosteroid, so that this type of treatment in SLE patients previously suffering a tendon rupture evidently deserves to be reconsidered.

Biopsy specimens in 4 reports showed the following conditions either alone or in combination: degeneration,³ mononuclear infiltration,¹ neovascularisation,⁵ and vacuolar myopathy (in the present case). From these findings and some experimental evidence.^{11 15 18} it is clear that corticosteroids exert a harmful effect on the microarchitecture of tendons and collagen tissue. The question arises as to the contribution of SLE proper to the degenerative-inflammatory process which led to the tear. The histological preparation in our case showed among other findings a perivascular mononuclear infiltrate, which was probably due to SLE.

From the current state of knowledge of tendon rupture in SLE it is impossible to predict which patients with SLE will develop this disabling but surgically curable complication. However, one should be extremely careful not to administer unnecessarily large doses of corticosteroids to patients with SLE.

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