COMMENT

Spontaneous rupture of tendons may result from pyogenic infection, tuberculosis, gonorrhoea, syphilis, gout, or tumours (Boyd, 1938), and is seen not infrequently as a part of the clinical picture of rheumatoid arthritis. In the latter condition the tendons undergo fibrinoid degeneration, a characteristic of the collagen diseases. It is possible that in the present case the pain prior to the final tendon rupture was due to minute tears in the degenerating tendon, and that normal healing was inhibited by the steroid therapy. In an experimental study Wrenn, Goldner, and Markee (1954) showed that, though end-to-end healing of ruptured tendons in animals on cortisone therapy was not prevented, the tendons were 40% weaker than control tendons. The cortisone delayed the maturation of the fibrous tissue. The ease with which the tendons ruptured in the present case and the relative lack of pain at the time would indicate that they were grossly degenerated.

A similar picture is seen at the shoulder, where again a large tendon mass is involved. In the older age-group rupture of the supraspinatus tendon occurs with relatively little trauma and minimal pain. In a slightly younger age-group chronic tendinitis is seen which is painful and may be the sequel to a partial tear (Apley, 1959). This would perhaps correspond to the pain in the present case during the four months before the tendons ruptured.

I am grateful to Mr. E. A. Devenish for his helpful criticism.

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REFERENCES

Apley, A. G. (1959). *A System of Orthopaedics and Fractures*, p. 121. Butterworth, London.
Arner, O., and Lindholm, Å. (1959). *Acta chir. scand.*, Suppl. 239.
Boyd, W. (1938). *Surgical Pathology*, 4th ed. Saunders, Philadelphia.
Wrenn, R. N., Goldner, J. L., and Markee, J. L. (1954). *J. Bone Jt Surg.*, **36A**, 588.

Simultaneous Bilateral Rupture of Achilles Tendons Due to Triamcinolone

Cramps, muscle-wasting, and myopathy are well-known complications of corticosteroid therapy. They occur most commonly with triamcinolone (Dubois, 1958; Kendall and Hart, 1959; Neustadt, 1959), but have also been described after cortisone (Perkoff *et al.*, 1959), prednisone (Harman, 1959), fludrocortisone (MacLean and Schurr, 1959), and dexamethasone (Duvenci *et al.*, 1959; Golding and Begg, 1960). The following case is of interest, as the patient developed simultaneous bilateral rupture of the Achilles tendons as a sequel to triamcinolone therapy, given for chronic discoid lupus erythematosus (chronic L.E.).

CASE REPORT

A 52-year-old scaffolder was first seen in the department of dermatology in October, 1957, with a 14-year history of chronic L.E. of both cheeks. He was treated initially with chloroquine, 400 mg. daily, but some activity of the lesions remained; and by August, 1958, he was complaining of blurred vision and "spots in front of the eyes." Hydroxychloroquine sulphate was substituted for chloroquine in daily doses of 800 mg., and this was subsequently increased to 1,200 mg. and then 1,600 mg. daily, as response was not complete.

A lichenoid eruption of the legs was noticed in December, 1958, while he was on hydroxychloroquine only. In view of this, and since the chronic L.E. was still active, the hydroxychloroquine was stopped and triamcinolone begun in doses of 4 mg. twice daily and increased to four times daily two months later. Both the discoid lesions and the lichenoid eruption had cleared completely by March, 1959; however, he then complained of night sweats.

Clinical and chest *x*-ray examinations were negative, and the dose of triamcinolone was reduced to 4 mg. three times daily. On April 28 he was seen in the orthopaedic department because of pain in both Achilles tendons which interfered with his climbing ladders. No abnormality was found either in the tendons or in the ankle-joints. At this time he could walk fairly well and was treated by strapping both ankles. On May 12 he complained of cramps in both calves, worse on the left, and because of this triamcinolone was gradually reduced during the next six days and mepacrine, 300 mg. daily, substituted. On May 21, three days after triamcinolone had been stopped, he noticed pain in his left ankle, and a short time afterwards his right ankle was also painful when standing on a ladder. He had not exerted himself unduly. Walking was now almost impossible, since he could not plantar-flex his feet properly. He was taken to hospital, where a diagnosis of rupture of the Achilles tendons was made. Surgical repair was carried out a fortnight later by Mr. R. G. Taylor under A.C.T.H. cover, and recovery was uninterrupted. He has regained normal functions of both tendons.

Because of a relapse of his skin condition, oral prednisolone was started in June, 1960, and by December he was receiving 5 mg. twice daily; the chronic L.E. was inactive again and there were no untoward effects.

COMMENT

Since simultaneous rupture of both Achilles tendons is very rare, it seems probable that it was caused by triamcinolone, and that it was not due to the lupus erythematosus. The patient complained of cramps in his calves and tendon pain four weeks before treatment was stopped. It is probable that partial rupture was present at this stage, since it has been shown (Anzel *et al.*, 1959) that a tendon will continue to function apparently normally when 75% of its fibres are ruptured. Arthritis, tenosynovitis, syphilis, and tumour are no longer regarded as being significant aetological factors in this condition; however, there must be some abnormality of the tendon before rupture can occur. Characteristic degenerative and necrobiotic changes have been found in tendon tissue examined within 24 hours of rupture, which presupposes that they must have been present before (Arner *et al.*, 1959). It has been suggested that these changes are ischaemic and are secondary to hypertrophy of the media and narrowing of the lumina of medium-calibre blood-vessels. Triamcinolone almost certainly aggravated, if not initiated, these changes in our case.

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REFERENCES

Anzel, S. H., Covey, K. W., Weiner, A. D., and Lipscomb, P. R. (1959). *Surgery*, **45**, 406.
Arner, O., Lindholm, Å., and Orell, S. R. (1959). *Acta chir. scand.*, **116**, 484.
Dubois, E. L. (1958). *J. Amer. med. Ass.*, **167**, 1590.
Duvenci, J., Chodosh, S., and Segal, M. S. (1959). *Ann. Allergy*, **17**, 695.
Golding, D. N., and Begg, T. B. (1960). *Brit. med. J.*, **2**, 1129.
Harman, J. B. (1959). *Lancet*, **1**, 887.
Kendall, P. H., and Hart, M. F. (1959). *Brit. med. J.*, **1**, 682.
MacLean, K., and Schurr, P. H. (1959). *Lancet*, **1**, 701.
Neustadt, D. H. (1959). *J. Amer. med. Ass.*, **170**, 1253.
Perkoff, G. T., Silber, R., Tyler, F. H., Cartwright, G. E., and Wintrobe, M. M. (1959). *Amer. J. Med.*, **26**, 891.