From the clinic

Safe relief of rest pain that eases with activity in achillodynia by intrabursal or peritendinous steroid injection: the rupture rate was not increased by these steroid injections

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Histological and macroscopic appearance can confirm an inflammatory lesion in the paratenon and a degenerative lesion with myxoid, mucoid, and hyaline changes in the tendon.^{1,2} On this basis, the inflammatory lesion in the paratenon can be called a peritendinitis and the degenerative lesion a tendinopathy. Some patients with achillodynia give a history that predominantly includes pain on first rising in the morning but which eases as they become mobile. This may return when they have been sitting but again eases with walking. An athlete may have pain on commencement of activity but this eases during activity to return afterwards when the athlete is resting.

A second type of history is predominantly of no pain in the morning, but as walking and activities are introduced so the pain ensues. There is no increase in pain after sitting, and athletes are free of pain at the start of activity but during the activity, pain starts, getting worse as the activity is prolonged.

A third group of patients have elements of both of these histories.

Read and Motto³ postulated that rest pain that eases with activity is a presenting clinical history of an inflammatory lesion and should therefore improve with treatment with anti-inflammatories such as cortisone injections. This paper reports further on this. A literature search by Mahler and Fritschy,⁴ previous experience with peritendinous steroids,³ and prospective trials^{5,6} had not shown any increase in rupture rate with the use of peritendinous steroids, which was therefore deemed an appropriate treatment for this source of inflammation. However, patients with activity induced pain, which was postulated to be a tendinopathy, did not receive steroid injections, as it was thought to be unethical to inject an anti-inflammatory into a degenerate lesion which could be weakened.⁷

Method

A retrospective analysis of 81 consecutive patients referred to one private clinic between 1984 and 1995 as achillodynia was carried out. Diagnosis was made on a history of pain at rest that improved with activity or a pain that worsened with triceps surae activity, and signs of pain

on resisted plantar flexion and tenderness to palpation over the Achilles tendon or the retro Achilles/superficial Achilles bursa. Crepitus was an unusual finding as most patients had previously had physiotherapy and taken non-steroidal anti-inflammatory drugs, and all had been having problems for more than four weeks. The symptoms of these patients also had to fit the following criteria: no pain on talar and subtalar joint movement and ligament stressing; no pain on the calcaneal tibial compression test (passive whipped plantar flexion of the ankle to force impingement of the calcaneum on the posterior aspect of the tibia); pain free palpation and resisted testing of the posterior tibialis and peroneals. The Thompson/Simmonds test and prone and flexed knee attitude of the foot were negative except in the five (8%) total ruptures diagnosed.

The following patients were excluded from analysis: eight with posterior tibialis tendinitis (10%), five with calcaneotibial compression test positive ankles (6%), one with lumbar disc, one with lipoma, two with gout, and the five with total rupture. Six (7%) retro Achilles bursae (two were HLA B27 positive and one was rheumatoid factor positive), four (5%) superficial Achilles bursae and one combined retro and superficial Achilles bursae (total 13%) were diagnosed.

Because the bursal pain also could be worse at rest and because one rupture occurred after retro Achilles bursal injection on two occasions several months apart, these were also included in the analysis.

Further investigations such as magnetic resonance imaging were only instituted if therapeutic progress was not being maintained and then only to define the intratendinous lesion, the tendinopathy.

The remaining 64 were questioned about morning, rest, and activity pain and asked to grade the pain using a visual analogue score (VAS) on a sliding scale of "no pain" to "worst pain ever". The reverse side, unseen by the patient, gave a scaled reading from 0 to 10 which was recorded. The 64 were then divided into two groups. Those in group 1 (35 of the 64; 55%) had a history of rest pain that improved with activity; they were treated with peritendinous or

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Accepted for publication
21 September 1998

intrabursal injections of 1 ml hydrocortisone acetate (Hydrocortistab; Knoll Ltd, Nottingham, UK; 25 mg/ml) with 1 ml 1% xylocaine (Astra Pharmaceuticals Ltd, Kings Langley, UK). For the peritendinous injections a bent needle was inserted along the tendon in a longitudinal direction. Normal thumb pressure was applied to the plunger and the needle withdrawn until the solution flowed. This was repeated around the painful area until the dose was administered. Cross training on a bike or rowing machine or in water was permitted but no other training activities until the review one to two weeks later. Group 2 (29 of the 64; 45%) comprised patients with a history of rest pain that improved with activity, who did not want an injection, and patients without such a history of pain but who had pain on activity which got better with rest; they were treated with appropriate orthotics,^{3 8 9} adjustment of the shoes,^{3 10} and controlled rehabilitation,³ as were those in group 1 once morning/rest pain had settled. The two groups were not matched for age or sex as they were consecutive patients, whose grouping was defined by their history at presentation. Their ages ranged from early 20s to late 60s, and their sports ranged from track and field athletics and road running to leisure golf and tennis.

The patients were reviewed after 7–14 days and their VAS was recorded. A further analysis of the ruptures was recorded over the following year but long term results were thought to be dependent on the extent and nature of the tendinopathy and not the inflammatory lesion and were not monitored.

Results

The patients in group 1 had a mean morning/rest pain VAS reading of 5.5 (range 1–9) before the injection and 0.2 (range 0–3) after the injection. Those in group 2 had a mean morning/rest pain VAS reading of 2.5 (range 0–9).

RUPTURES

After exclusion of the five presenting cases (none of whom had received steroid injections), two ruptures (6%) occurred in group 1 and two (8%) in group 2.

Discussion

Steroid injections around the Achilles tendon have been used with varying success, and Da Cruz *et al*⁶ suggest that they produce no overall improvement in the rate of healing. If histological examination shows a degenerate tendinopathy, logic would suggest that an anti-inflammatory drug would have no effect on the healing rate. However, many patients are inhibited by the morbidity of the pain. The peritendinitis is an inflammatory lesion^{1 2} and as such should benefit from treatment with an anti-inflammatory and this should be placed in or around the paratenon. It will, however, treat only the peritendinitis and not the tendinopathy. This

report has looked at whether one element of the pain can be reduced by peritendinous steroid injection, and whether the rupture rate is increased by this treatment.

A reduction in mean pain VAS from 5.5 to 0.2 in 7–14 days is a significant reduction in morbidity, especially as this pain occurred at rest. Many patients were then happy to take the time required for rehabilitation as this different pain appeared to start during exercise rather than at rest.

Anti-inflammatory injections should only be used to treat the inflammatory lesion not the tear or degenerative lesion. As the morning/rest pain that eased with activity was improved by anti-inflammatory injections, it would appear that this may be an inflammatory lesion as postulated by Read and Motto.³ Local infiltration of hydrocortisone acetate around the paratenon should therefore help the pure peritendinitis and the combined peritendinitis and tendinopathy, but only the peritendinitis element not the tendinopathy. When the results of Subotnick and Sisney⁸ are further analysed, from a series of 356, a rupture rate of 3.5% for those not given steroids and one of 3.7% for those receiving steroids emerges. These results, together with the corresponding values of 8 and 6% in this report and a 3% rupture rate in patients given steroids in the report of Read and Motto,³ suggest that achillobiodynia may well have an attrition rate of 3–8% irrespective of treatment. It may be that the extent of the underlying tendinopathy is the risk factor not the steroid injections.

SUMMARY

A history of morning and rest pain that eases with activity was found to improve after anti-inflammatory injections around the paratenon or within the Achilles bursae. The reduction in pain morbidity was significant, and the peritendinous steroid injections did not increase the rupture rate.

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Take home message

The morbidity of morning/rest pain is high but it may be safely relieved by peritendinous or intrabursal steroid injection.