Aspiration of the retrocalcaneal bursa

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SUMMARY We aspirated the retrocalcaneal bursa in cadavers to determine the characteristics of bursal fluid. A small amount of clear, viscous fluid was constantly present in the bursa. Leucocyte count was low, and the mucin clot test was good. With the same technique we aspirated the retrocalcaneal bursae of 4 patients. Three had Reiter’s syndrome; the bursal fluid was inflammatory, and symptoms promptly resolved after local corticosteroid injection. The fourth patient presented with heel pain; intracacellular, positively birefringent crystals were present in the aspirate, consistent with the diagnosis of pseudogout.

Heel pain can result from inflammation in the retrocalcaneal (or sub-Achilles) bursa, a constant structure between the Achilles tendon near its insertion and the calcaneus. While involvement of this bursa in various rheumatic diseases has been well described,1-4 the characteristics of bursal fluid remain largely unknown. We report bursal fluid findings in cadavers without rheumatic disease and the clinical application of aspiration of the retrocalcaneal bursa in 4 cases, 3 of Reiter’s syndrome and one of pseudogout.

Materials and methods

We aspirated 16 retrocalcaneal bursae and 14 joints of the lower limbs (7 ankles, 6 subtalar joints, 1 knee) of 8 unfrozen cadavers with a maximum conservation period of 24 hours. All subjects were male, ages ranged from 53 to 78, and none had a history of rheumatic disease. Bursal aspiration was performed with a no. 19 butterfly needle that was advanced perpendicular to the skin until the posterior superior calcaneal surface (above the Achilles tendon insertion) was touched. The cap of the tubing was then removed, allowing the fluid to enter the distal portion of the plastic tube. Gentle aspiration was then applied to drain the bursa fully. The exact location of the needle was ascertained by sharp dissection. Joints were aspirated by standard techniques.

Although smaller volumes were aspirated from the retrocalcaneal bursa (no fluid in 3, traces in 3, and measurable amounts in 10), the findings in bursal and joint fluids were similar (Table 1). Both were clear and highly viscous. The mucin clot test was good with the exception of 2 of 13 bursal fluids and 1 of 14 joint fluids, in which clot did not form. Leucocyte count was low. No cells were found in the haemocytometer grid in 5 of 9 bursal fluids in which count was possible and in 7 of the 14 joint fluids. The predominant cells (70 to 100%) were mononuclears. The red blood cell count was low, with the exception of a traumatic tap. Crystals were not found.

Table 1 Synovial fluid findings at necropsy

<table>
<thead>
<tr>
<th></th>
<th>Retrocalcaneal bursa (n=16*)</th>
<th>Joints (n=14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume ml, median</td>
<td>0-05</td>
<td>0-45</td>
</tr>
<tr>
<td>(range)</td>
<td>(0-0-2)</td>
<td>(0-1-3)</td>
</tr>
<tr>
<td>Viscosity</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Colour</td>
<td>Clear</td>
<td>Clear</td>
</tr>
<tr>
<td>Mucin clot test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>good</td>
<td>11</td>
<td>13</td>
</tr>
<tr>
<td>none</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>WBC x 10^9/l, median (range)</td>
<td>(0-0-3)</td>
<td>(0-0-15)</td>
</tr>
<tr>
<td>% mononuclears</td>
<td>70-100</td>
<td>100</td>
</tr>
<tr>
<td>RBC x 10^9/l, median (range)</td>
<td>0-05</td>
<td>0-1</td>
</tr>
<tr>
<td>Crystals</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

* Volume, n=16; viscosity, colour and mucin clot test, n=13; leucocyte, differential, and erythrocyte counts and crystals, n=9.

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Case reports

**Patient 1**
A 40-year-old white man was admitted on 15 July 1981 with a 2-month history of scaly rash on the soles, balanitis, asymmetrical polyarthritis, and back pain. There was a previous history of nonspecific urethritis and dysentery at the age of 20, without recurrences.

Findings on physical examination included a shallow, 1 cm diameter ulcer in the hard palate, keratoderma blenorragica on the soles, and balanitis circinata. Several joints were inflamed, all on the left side of his body. These included the 4th proximal interphalangeal joint, several metacarpophalangeal joints, wrist, elbow and shoulder. In addition, the left patellar tendon region was swollen and tender, without a knee effusion. The back of the left heel, especially the upper portion, was swollen, red, and very tender. Dorsiflexion of the foot elicited local pain, while passive plantar flexion was unimpaired. Subtalar joint motion was normal. Several cervical and lumbar interspinous ligaments were tender to palpation. Chest expansion and lumbar spine flexion were normal. Ophthalmological examination was normal. X-rays showed soft-tissue swelling in the affected areas, without bone erosions or periosteal reaction. Scarciolicaj joints were normal.

With a diagnosis of Reiter’s syndrome the patient was treated with bed rest, a wrist splint, graded exercises, indomethacin 50 mg orally t.i.d., and corticosteroid cream for the skin and penile lesions.

Corticosteroids were injected into the left retrocalcaneal bursa. After superficial infiltration with 2% xylocaine, a no. 19 needle was inserted towards the superior posterior border (above the Achilles tendon insertion) of the calcaneus. Bone contact produced mild, sharply localised pain. Following aspiration, 1 ml of Renograin (sodium and meglumine diatrizoate) 60 plus 0.4 ml of trimcinolone hexacetonide (20 mg/ml) was injected into the bursa. The bursogram obtained is shown in Fig. 1.

**Bursal fluid analysis.** The volume was 0.2 ml. The fluid was cloudy and had a low viscosity. The leucocyte count was 30·2 x 10⁹/l, with 78% polymorphonuclear cells, and erythrocyte count was 0·2 x 10⁹/l. Some leucocytes contained large homogeneous inclusions, but no macrophages containing polymorphonuclear cells or LE cells were found. There were no crystals, and culture was negative.

Three days after the intrabursal injection the heel tenderness disappeared. One week later all joints had improved except the left wrist, which was painful for an additional week. All skin lesions cleared by the patient’s discharge on 7 August 1981. Indomethacin was decreased to 50 mg orally t.i.d. Over the ensuing 17 months he remained free of articualr symptoms. The progressive reduction of retrocalcaneal soft tissue swelling is shown in Table 2.

**Patient 2**
A 67-year-old white male with crystal-proved pseudogout in the knee was seen on 16 November 1981 because of pain and swelling in his right lower leg. He was afebrile. There was marked swelling, warmth, and tenderness around the ankle, posteriorly, and in the lower leg, with moderate pitting oedema in the pretilibial region. Dorsiflexion of the foot elicited acute pain. Passive plantar flexion was unrestricted. Subtalar motion was free. X-rays of the

**Table 2 Patient 1: retrocalcaneal soft tissue thickness in mm (skin, subcutaneous tissue, and Achilles tendon)**

<table>
<thead>
<tr>
<th>Date</th>
<th>Measurements: injected side/contralateral side (normal range)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>At calcaneus</td>
</tr>
<tr>
<td>16 July 1981†</td>
<td>15·8 (8-13)</td>
</tr>
<tr>
<td>9 September 1981</td>
<td>10·9</td>
</tr>
<tr>
<td>10 December 1981</td>
<td>8·5</td>
</tr>
</tbody>
</table>

* Ref. 4.
† Date of injection.
leg revealed soft tissue swelling and calcium deposits in the Achilles tendon proximal to the bursal region.

Under sterile precautions the retrocalcaneal bursa was aspirated as described in the previous case. Traces of bursal fluid revealed multiple rhomboidal, positively birefringent crystals, some intracellular, consistent with calcium pyrophosphate dihydrate (CPPD). A Gram stain revealed no bacteria. Culture was negative. The patient responded promptly to ibuprofen 800 mg orally t.i.d.

**Patient 3**

A 27-year-old white man was seen in clinic 3 August 1982 with a 6-week history of pain in the neck, shoulders, and low back; swelling of both index fingers; and swelling of the left knee. There was a past history of injury to the neck and left shoulder.

Findings on physical examination were limited to the musculoskeletal system. There was sausage swelling of both index fingers, with tenderness most marked at the proximal interphalangeal joints. There was decreased motion of the left shoulder in all directions, with tenderness of the biceps tendon. The range of motion of the neck was normal, as was lumbar flexion. There was warmth of the left knee, with a moderate effusion.

The Westergren erythrocyte sedimentation rate was 34 mm in one hour. The urine sediment showed 25–30 leukocytes per high-power field. Latex fixation test was negative. HLA B27 was positive. Synovial fluid from the left knee was inflammatory. X-rays showed only soft tissue swelling of the index fingers; films of shoulders and the cervical and lumbar spine were normal.

Over the next several months all symptoms were well controlled with ibuprofen, physical therapy for the left shoulder, and steroid injection of the left knee.

On 28 November 1982 he was seen with a one-week history of right heel pain and inability to stand all day at his usual job as a barber. On physical examination there was marked tenderness at the insertion of the Achilles tendon and anterior to the tendon (palpated from the side) just above the calcaneus. The tendon itself was enlarged but not tender. There was mild tenderness at the insertion of the plantar fascia. The joints of the foot were all normal. X-rays of the heel showed soft-tissue swelling.

The retrocalcaneal bursa was aspirated with a no. 22 needle; 1·25 ml of pink fluid was obtained. Small pieces of clot formed spontaneously. The viscosity was decreased and the mucin clot test was fair. Cell counts were spuriously low due to clotting, but leukocytes were 1·45 × 10⁹/l, with 26% polymorphonuclear cells and 74% mononuclear cells, and the erythrocyte count was 16·5 × 10⁹/l. There were no crystals. Triamcinolone hexacetonide 10 mg was injected into the bursa.

On 2 December 1982 a walking cast was applied because symptoms persisted. Two weeks later the cast was removed. Pain and tenderness at the Achilles tendon were resolved, though enlargement of the tendon was not diminished; he was able to resume normal work. An additional finding at this visit was acute iritis of the right eye, confirmed by ophthalmological consultation.

**Patient 4**

A 36-year-old white male came to the clinic on 2 December 1982 because of pain in the right heel for 4 months, not responsive to a 2-month trial of indomethacin 50 mg orally t.i.d.

Reiter’s syndrome was diagnosed in 1972 when he presented with circinate balanitis, keratoderma blenorrhagica, arthritis of both knees, and bilateral heel pain. He was treated with indomethacin and phenylbutazone, and symptoms resolved within one and half years. Treatment was discontinued, and he remained well, working full-time as a mechanic, until the onset of his recent symptoms.

Physical examination revealed a few pseudo-pustules on the plantar surface of the left foot; diffuse swelling of the right Achilles tendon; swelling and tenderness of the back of the right heel and of the 2nd metatarsophalangeal joint; and tenderness, bilaterally, at the attachment of the quadriceps tendon to the patella.

Radiographic abnormalities were limited to the right foot. The Achilles tendon was thickened, with total soft-tissue thickness at the calcaneus, 1 cm and 2 cm above the calcaneus of 15, 17, and 15·5 mm, respectively. On plantar flexion the retrocalcaneal recess was blurred.

The patient was admitted to hospital. No weight bearing was allowed on the right foot, and ankle motion was discouraged. Indomethacin was increased to 50 mg orally q.i.d.

Following xylocaine infiltration of the skin the right retrocalcaneal bursa was entered with a no. 22 needle. After bursal aspiration triamcinolone hexacetonide 10 mg (0·5 ml) plus 1 ml of Renografin 60 (Figs 2a, b) were injected into the bursa.

Only 0·25 ml of bursal fluid was obtained. The viscosity was decreased but the mucin clot test was good. The leukocyte count was 0·8 × 10⁹/l. with 100% mononuclear cells, and erythrocyte count was 0·05 × 10⁹/l. There were no crystals, and culture was negative.

Several hours after the intrabursal injection the heel pain became unbearable, improving overnight. Symptoms resolved completely over the ensuing 7 days, with marked reduction of soft-tissue swelling.
Aspiration of the retrocalcaneal bursa

Discussion

The anatomy of the retrocalcaneal bursa and the clinical and pathological features of retrocalcaneal bursitis were well described by Rössler in 1896. A typical synovial lining can be identified only in the proximal portion of the bursa, overlying fatty lumps that protrude from the neighbouring sub-Achilles fat pad. The anterior bursal wall is composed of fibrocartilage laid over the calcaneus, while the posterior wall is indistinguishable from the thin epitenon of the Achilles tendon. The retrocalcaneal bursa can be envisaged as a disc-like cavity moulded over the posterosuperior angle of the calcaneus, filled with highly viscous fluid. The hyaluronic acid content of this bursa is higher than that of the olecranon and prepatellar subcutaneous bursae. As shown here, the normal cadaveric retrocalcaneal bursa fluid has a low cellular content, predominantly mononuclear cells, and a good mucin clot test; it is similar to joint fluids obtained from the same cadavers and the cadaveric joint fluids reported by Ropes and Bauer.

Clinically, retrocalcaneal bursitis is characterised by posterior heel pain, prominence of the Achilles tendon just proximal to its insertion, soft tissue bulging at both sides of the tendon (in acute cases the swelling is diffuse), and pain with dorsiflexion of the foot, which compresses the bursa between tendon and bone. The differential diagnosis between Achilles tendinitis and retrocalcaneal bursitis can be difficult, and they often coexist. Diagnosis is confirmed by standard radiography and xeroradiography. Failure to demonstrate the retrocalcaneal recess (a radiolucency between the calcaneus and the Achilles tendon in lateral roentgenograms of the heel obtained in plantar flexion) is an early and reliable indication of retrocalcaneal bursitis. The typical xeroradiographic finding is anterior indentation of the Achilles tendon at the bursal site. Finally, erosion of the posterosuperior surface of the calcaneus is late but unequivocal evidence of retrocalcaneal bursitis.

To these useful, noninvasive radiological procedures we are now adding diagnostic aspiration of the retrocalcaneal bursa. Since this procedure can be painful, we believe that it should be performed only when the bursa is to be punctured for instillation of therapeutic agents, or when the cause of the bursitis is uncertain. The rapid improvement of bursitis and Achilles tendinitis in our patients with Reiter's syndrome suggests that corticosteroid injected into the retrocalcaneal bursa benefits the inflamed Achilles tendon. Although the intracellular crystals in our second patient were not studied by x-ray diffraction owing to the small sample, the morphology by polarising microscopy was consistent with CPPD,

Fig. 2 Patient 4: bursogram. (a) after injection of 0.5 ml of contrast material; (b) after injection of 1.5 ml of contrast material.
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and indicated that CPPD deposits in the Achilles tendon can be shed into the bursa, causing acute inflammation. An alternative source for CPPD crystals is the fibrocartilage overlying the calcaneus in the anterior bursal wall, but deposits at this site remain to be shown.

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