Limb lymphoedema in rheumatoid arthritis

Sir: There is an alternative, and simpler, explanation for limb lymphoedema in rheumatoid arthritis as reported by Dacre, Scott, and Huskisson—obstruction. The report by Macfarlane and van der Linden1 hints at this; oedema in their case was associated with a leaking olecranon bursa, but ‘after injection of corticosteroid into the bursa and elevation of the arm the forearm swelling resolved almost completely’.

I have seen four cases of severe limb oedema in inflammatory arthritis.

CASE 1
A 56 year old housewife had a 15 year history of seropositive rheumatoid arthritis. This was atypical, with involvement of large and medium sized joints and complete sparing of the small joints of the hands, though there was erosive change at the wrists. Both elbows were affected, with moderate destructive change, fixed flexion deformity, and lateral instability, especially on the right. She had had both knees and hips replaced.

She developed slow onset pitting oedema of the right forearm and hand, which caused severe limitation of function and was very painful. This failed to respond to elevation of the arm and persisted for over a year despite continuing steroids by mouth. She was reviewed by the orthopaedic department, who suggested that the oedema might be due to obstruction of venous or lymphatic return at the elbow, and accordingly I injected the elbow with triamcinolone.

Over the next week the oedema resolved completely. Two years later it has not recurred, though the elbow continues to produce some pain.

CASE 2
A 72 year old man was referred with a history of sudden painful swelling of the right wrist. A previous diagnosis of seronegative rheumatoid arthritis and coincident psoriasis had been made elsewhere two years previously, and he had been treated since with 5 mg prednisolone on alternate days and gold injections; he was also taking diuretics for mild cardiac failure and hypertension. He had been free from symptoms in the joints for many months until the sudden flare in the wrist.

When I first saw him the wrist was quiet (the steroids had been increased for a week to 40 mg daily), but on review one month later the whole hand was swollen, painful, tender, and oedematous, looking almost gouty. He was unable to flex the fingers because of the oedema. After one month’s treatment with diclofenac and splinting the wrist and hand were unchanged. I injected the wrist with triamcinolone. Within a few days the oedema had disappeared. Although he has some finger stiffness, the oedema remains absent 14 months later.

CASE 3
A 67 year old women with a past history of myocardial infarction and severe peripheral vascular disease requiring bypass surgery was referred with a short history of pain and swelling in the small joints, especially the hands, and bilateral carpal tunnel syndrome. Her sister had severe rheumatoid arthritis.

Examination confirmed small joint polyarthritis. She was treated with wrist splints and triamcinolone with improvement of the carpal tunnel symptoms, but overall she was worse and treatment was started with prednisolone 7.5 mg daily. She had side effects, however, and stopped these, continuing to receive diclofenac only.

Four months after presentation she attended for routine review with a painful swollen hand. The wrist was stiff and tender, but the striking feature was severe pitting oedema of the hand. I injected the wrist with triamcinolone. The oedema disappeared within four days and three months later has not recurred.

CASE 4
A 60 year old man was referred with a short history of unilateral ankle pain and swelling. He had previous foot and knee symptoms suggestive of osteoarthritis. On examination the skin over the ankle felt very tight. There was no obvious oedema and he had no pain on movement. Both feet were flat.

The ankle was injected, and improved. He developed some foot numbness suggestive of tarsal tunnel syndrome and arch insoles were prescribed, but he then defaulted from the clinic.

He was re-referred six months later with shoulder pain, apparently due to supraspinatus tendinitis, but on review two months later had developed generalised inflammatory type symptoms affecting hands, shoulders, and knees, which responded only partly to anti-inflammatory drugs. Then quite suddenly he developed swelling of his left knee and leg. On examination he had a tense knee effusion with a popliteal cyst and severe pitting oedema of the leg and foot. The leg above the knee was normal.

The knee was injected with triamcinolone, though no fluid was aspirated. Initially, both knee and oedema subsided slightly but then worsened again and he attended the accident department and was admitted for treatment by the orthopaedic surgeons, who aspirated 80 ml turbid fluid without injecting steroid, pending my outpatient appointment 10 days later. The oedema again abated, but the knee effusion reaccumulated and the leg swelling reappeared.

The response of the oedema in each case to injection of the joint just proximal is surely not coincidence. Just as peripheral nerves can be compressed when there is soft tissue swelling, thickening, or inflammation, so can veins and the lymphatic system. It is interesting in this context that none of the above patients had nerve root compression symptoms.

Apart from Macfarlane’s case, none of the patients in Dacre’s series or in the quoted references had joint injections. I suggest it is tried more often.

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doi: 10.1136/ard.50.6.407