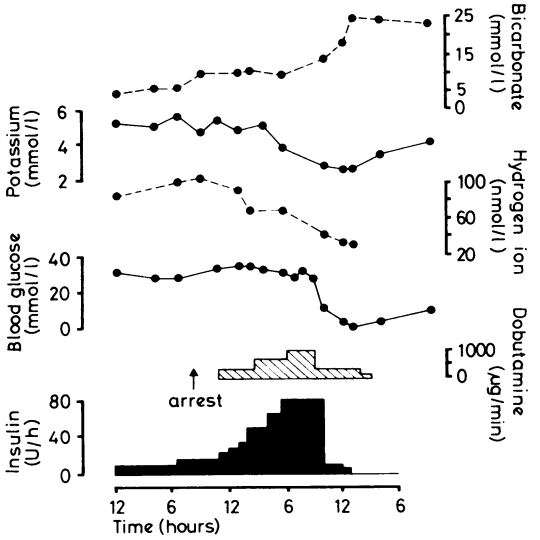
normal at this stage with blood pressure 130/70 mm Hg. Blood glucose concentration was 13 mmol/l (234 mg/100 ml) and bicarbonate 12 mmol/l.

She again became hypotensive, however, with blood pressure of 90/60 mm Hg and deteriorated over the subsequent 12 hours, despite continuation of the insulin infusion. Blood glucose concentration rose to 34·5 mmol/l (621 mg/100 ml) and bicarbonate fell to 9 mmol/l. Chest x-ray showed extensive bilateral pulmonary shadows. Her progress rapidly worsened despite treatment with gentamicin and flucloxacillin, culminating in cardiorespiratory arrest. After resuscitation she required ventilation and developed low cardiac output failure, which was treated with dobutamine. Introduction of this drug was associated with an appreciable increase in insulin requirement. Despite an infusion of 80 units of Actrapid MC hourly for five hours her blood glucose concentration remained raised at >30 mmol/l (>541 mg/100 ml). After improvement in cardiac function the dobutamine infusion rate was reduced. This was associated with an appreciable fall in blood glucose and insulin requirement. On complete withdrawal of dobutamine the insulin requirement fell to 2 units/hour (figure).



Effect of dobutamine on blood glucose, potassium, hydrogen and bicarbonate concentrations.

Conversion: SI to traditional units—Potassium: 1 mmol/l = 1 mEq/l. Glucose: 1 mmol/l ≈ 18 mg/100 ml. Bicarbonate: 1 mmol/l = 1 mEq/l. Hydrogen ion: 1 nmol/l = 0·1 ng/100 ml.

Comment

In this patient the temporal relation between the reduction in the dobutamine infusion rate and the precipitous fall in blood glucose concentration suggested a causal relation between the use of dobutamine and insulin resistance. Hypotension, acidosis, and infection probably played only a small part in the insulin resistance. Furthermore, these factors remained constant at the time of the reduction in the dobutamine infusion rate and could not account for the sudden fall in blood glucose value.

Dobutamine is commonly used as an inotropic agent; glucose intolerance during its use has not to our knowledge been reported in man. Studies in dogs demonstrate no effect on blood glucose.[4] The effect of dobutamine on the blood glucose in this patient may well have been dose dependent in view of the relatively normal insulin requirement at the lower rate of dobutamine infusion.

Other beta-adrenergic agonists (salbutamol, adrenaline, dopamine) increase insulin requirement in diabetics[1 2] and may produce hyperglycaemia in normal subjects.[3 5] In normal subjects this effect may be compensated for, as these agents also stimulate insulin release. In insulin-dependent diabetics, however, this compensatory mechanism is diminished or absent and the hyperglycaemic effect is likely to predominate. Whether or not dobutamine acts by stimulating glucose production or by other mechanisms—for example, as an insulin antagonist—remains to be investigated.

Our observations indicate the need for caution in the use of dobutamine and other beta-agonists in diabetes.

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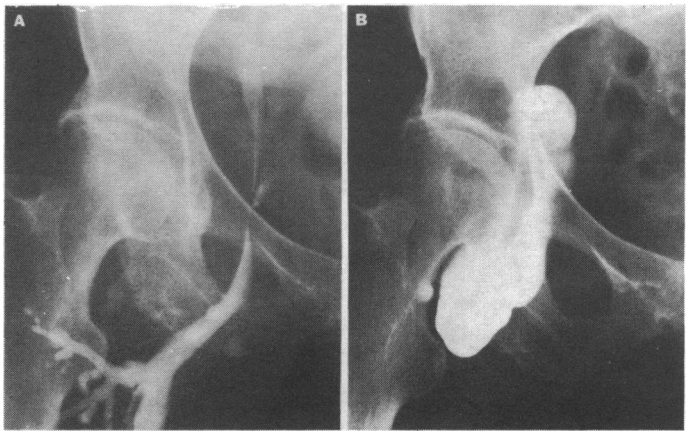
Iliopsoas bursitis in rheumatoid arthritis: an unusual cause of leg oedema

Ankle swelling is a common diagnostic problem in rheumatoid arthritis. Though popliteal bursitis is a well-recognised cause of unilateral ankle oedema, disease of the iliopsoas bursa is an uncommon cause of hip pain, still less a cause of leg oedema. We describe a case of iliopsoas bursitis which presented clinically as an iliofemoral venous occlusion and which resolved with conservative management.

Case report

A 64-year-old woman with seropositive rheumatoid arthritis of 15 years' duration was referred to the rheumatic diseases unit with increasing swelling of the right leg for three weeks. Fifteen months previously she had had a total condylar geometric arthroplasty of the right knee, after which she had noticed the slow progression of oedema of the leg and continuing pain in the knee.

On admission the right leg was grossly oedematous to the thigh, and a small tender mass was palpable in the right groin 2 cm below the inguinal ligament lateral to the femoral artery. Ascending venography showed extrinsic compression of the lateral aspect of the external iliac and femoral veins (figure). An ultrasound scan disclosed a cystic mass 8 cm long under the right iliopsoas muscle. This was aspirated and 200 ml viscous synovial fluid, sterile on bacteriological culture, was removed. Injection of contrast medium later confirmed the presence of an iliopsoas bursa which did not communicate with the hip (figure). The hip was clinically and radiologically normal. After bed rest and raising the leg the oedema resolved.



A: Venogram showing extrinsic compression of external iliac and femoral veins. B: Injection of radio-opaque contrast medium showing extent of iliopsoas bursa. There is no communication with hip.

Comment

The iliopsoas or iliopectineal bursa lies anteriorly on the iliofemoral ligament of the hip capsule beneath the psoas and pectineus muscles.

Its average size is 6 × 3 cm and it may be shown in all adults; it communicates with the hip in 14% of cases.[1] Iliopsoas bursitis may occur at any age and is most often the result of acute or recurrent trauma; it may also occur in any condition which produces a generalised bursitis or synovitis.[2] It usually presents as a mass in the groin with pain referred to the knee, and tenderness can almost always be elicited immediately below the midpoint of the inguinal ligament.[3] The pain, which is usually referred to the knee and aggravated by extension of the hip, is probably the result of entrapment of the femoral nerve in the femoral canal.[2] Differential diagnosis usually includes inguinal lymphadenopathy, femoral hernia, aneurysm of the femoral artery, and psoas abscess. Retro-peritoneal extension may occur with displacement of the bowel or ureter and, more rarely, pressure on the femoral vein may produce oedema of the leg.[4,5]

Iliopsoas bursitis is not a commonly recognised cause of pain in the hip, referred pain in the knee, or a mass in the groin, still less as a cause of a swollen leg. This collection of symptoms and signs should alert the clinician to the possibility of iliopsoas bursitis, even in the absence of hip arthritis. A careful history and clinical examination will help to confirm or exclude the diagnosis, and ultrasonography should resolve any further difficulties and obviate the need for surgical exploration.

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Scleroderma in the elderly

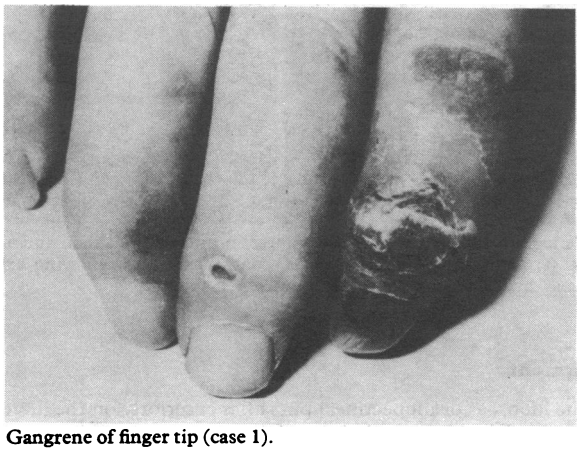
Scleroderma in the elderly is generally held to be a benign condition in which systemic disease is rare. We report on two patients, however, one of whom had widespread systemic disease and a rapidly fatal course.

Case report

CASE 1

An 80-year-old man was referred with severe proximal myopathy, Raynaud's phenomenon of recent onset, and weight loss of 19 kg over the preceding two years. There was acrosclerosis of the hands with small areas of necrosis on the finger tips, facial scleroderma and telangiectasias, and synovitis of the interphalangeal joints and wrists. Proximal muscles were weak and wasted. Haemoglobin concentration was 11·2 g/dl, erythrocyte sedimentation rate 52 mm in 1st hour, and the antinuclear factor test positive at 1 in 160. Creatine phosphokinase activity was raised at 940 IU/l, but a needle biopsy specimen of the quadriceps showed only type II fibre atrophy. A latex test was negative and thyroid function tests normal. Radiology showed moderate cardiac enlargement with interstitial changes in the mid and lower lung zones. Lung function tests showed a restrictive defect with distinct reduction of transfer factor (T_LCO 5·6 ml/min/mm Hg (predicted 24·5)).

Progressive systemic sclerosis with active myositis and fibrosing alveolitis was diagnosed and treated with prednisolone 40 mg daily. Eight months later he developed gangrene of the tips of several fingers, necessitating amputation of two fingers. Four months later his fingers had healed well, creatine phosphokinase activity was normal, and muscle power was satisfactory with prednisolone 10 mg daily. Over the next year the fibrosing alveolitis progressed with right heart failure, responding temporarily to bumetanide 2 mg daily. Muscle weakness recurred with a modest rise in creatine phosphokinase activity and multiple small ulcers appeared on his hands and ankles. He died of right-sided heart failure four years after clinical onset.



Gangrene of finger tip (case 1).

CASE 2

A 76-year-old woman with an eight-year history of Raynaud's and Sjögren's syndrome developed subacute small intestinal obstruction, which was managed conservatively. Weight loss of 19 kg over the preceding two years and oesophageal reflux had been noted at the time. Barium enema showed multiple wide-mouthed diverticula throughout the large bowel.

Two years later she developed severe scleroderma of the hands over five months, with rapidly progressive loss of hand function. Skin changes and telangiectasias of the face were noted. There was subluxation of the metacarpophalangeal joints and some ulnar deviation of the fingers. Schirmer's test was positive. Haemoglobin concentration was 11·4 g/dl and erythrocyte sedimentation rate 24 mm in 1st hour; a latex test was positive but the results of sheep-cell agglutination and antinuclear factor tests were negative. Thyroid function tests and chest radiography were normal, and there was no calcinosis in *x*-ray films of the hands.

Comment

Although the highest reported incidence of scleroderma occurs in patients over 65,[1] it has been suggested that most patients in this age group have a benign form of the disease,[2,3] often corresponding to the CRST syndrome (calcinosis, Raynaud's phenomenon, sclerodactyly and telangiectasis). Of 17 patients reported in two papers,[3,4] all were women and only one had systemic disease (pulmonary fibrosis) apart from oesophageal lesions. No deaths attributable to the disease were reported.

Both our patients developed scleroderma after the age of 65, with Raynaud's disease as an initial feature. Both had appreciable weight loss and severe progressive skin changes. The first patient was unusual in being a man and having the systemic complications of fibrosing alveolitis and polymyositis and a rapid progression of disease leading to death from right heart failure within four years of onset. The other patient had Sjögren's syndrome, oesophageal reflux, probable disease of the small and large bowel, and hand changes sufficient to cause considerable incapacity.

Scleroderma in the elderly, therefore, is not always benign. Our patients were not selected for the severity of the condition but were two consecutive patients aged over 65, confirmed as having scleroderma after referral to a rheumatologist from general practice. The mild nature of the cases in the published series may be due to a Berkson-type bias.[5]

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