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Lesson of the Week

Impaired healing of neuropathic foot ulcers due to neuropathic bladder distension in a patient with diabetes

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Occult distension of the bladder may cause oedema and persistence of neuropathic ulcers in patients with diabetes

Foot ulcers in patients with diabetes have several well recognised causes, including somatic neuropathy, autonomic neuropathy, abnormal stress patterns, and vascular disease.¹ Peripheral oedema, whether neuropathic or from another cause, may also play a part.^{1,2} Overdistension of the bladder can cause pressure on the iliac veins and lead to oedema of the legs,³ and distension of the bladder due to autonomic neuropathy in diabetes has been reported to cause oedema of the legs mimicking deep venous thrombosis.⁴ We report on a patient who had bilateral Charcot's arthropathy, oedema, and massive foot ulceration that was resistant to treatment. Correction of occult distension of the bladder led to resolution of the oedema and healing of the ulcers.

Case report

A 69 year old woman had had non-insulin dependent diabetes for 18 years, which was treated with oral chlorpropamide. She presented to the clinic in 1987 with a three year history of bilateral plantar foot ulceration and a five year history of Charcot's arthropathy. She also had severe proliferative retinopathy requiring laser photocoagulation. Serum creatinine concentration was normal (90 µmol/l), but she had persistent albuminuria (++ on dipstick testing). She had chronic leg oedema that was not associated with cardiac failure, hypoalbuminaemia (serum albumin concentration 38 g/l), or obvious local venous abnormality. Treatment with frusemide was only partly successful in reducing the oedema.

Over the next two years her foot ulcers persisted despite treatment with bed rest and daily dressing by the district nurse. Attempts at split skin grafting and total contact casting were unsuccessful. During this time she spent nearly six months as an inpatient. In 1989 the ulcers each measured 8×8 cm. Her serum creatinine concentration was 202 µmol/l, and she had occasional urinary incontinence. After catheterisation

1500 ml residual urine was obtained. Ultrasound scans confirmed considerable distension of the bladder with associated hydronephrosis. A neurogenic bladder was diagnosed.

On discharge she was fitted with a long term catheter and her leg oedema, which had been present for at least four years, resolved over the next few weeks. The foot ulcers healed over the next five months without further specific treatment.

Discussion

Leg oedema as a result of overdistension of the bladder has been reported in non-diabetic³ and diabetic patients,⁴ with rapid resolution after urinary catheterisation. The mechanism is thought to be compression of the common iliac veins in the pelvis. In this case the distension of the bladder, presumed to be due to autonomic neuropathy, may have been present for several years before its diagnosis. The resolution of the oedema after catheterisation suggests that the distension caused the oedema. Oedema of the foot has been suggested to be an important predisposing factor in foot ulceration regardless of its aetiology, and increased venous pressure may retard healing.

Diabetic patients with neuropathic foot ulcers resistant to treatment should have ultrasonography of their pelvises to exclude distension of the bladder, particularly if leg oedema is present.

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