CASE REPORT

Metastatic spinal abscesses from diabetic foot osteomyelitis

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SUMMARY

A 66-year-old man with long-standing type 2 diabetes, nephropathy and neuropathy was admitted acutely with an infected left big toe neuropathic ulcer, with underlying osteomyelitis. His condition rapidly deteriorated with sepsis and right lobar pneumonia. Microbiology grew methicillin-sensitive *Staphylococcus aureus*. Shortly into his admission, he developed flaccid paraparesis, and an MRI showed multiple epidural abscesses with likely cord infarction, not amenable to surgical intervention. His sepsis resolved, but his paraparesis remained severe, requiring spinal rehabilitation.

BACKGROUND

Foot ulceration is a common reason for admission among people with diabetes, and a common cause of amputation due to infection or gangrene. Diabetic foot ulceration may, however, be a source of metastatic infection. We describe an unusual presentation of foot ulceration and osteomyelitis leading to bacteraemia and epidural abscesses.

CASE PRESENTATION

A 66-year-old man was admitted via the emergency department with a 2 day history of pain, redness and swelling of his left big toe following wearing new footwear. He had a 25-year history of type 2 diabetes, complicated by proteinuria, mild renal impairment and peripheral neuropathy. Five years earlier, he was treated for right big toe osteomyelitis with antibiotics and surgical debridement. He was otherwise well, and completely independent prior to his admission. On examination, he was febrile (38.4°C), tachycardic (110 regular) but haemodynamically stable. Foot examination showed reduced fine touch and vibration sensation to the knees bilaterally, but normal dorsalis pedis and posterior tibial pulses. A small discharging ulcer with pus was noted at the tip of the left hallux. Intravenous co-amoxiclav and clarithromycin were commenced, and blood cultures grew methicillin-sensitive *Staphylococcus aureus*.

On the second day of admission, he became more breathless and required high flow oxygen to maintain oxygenation. BP dropped, and his condition necessitated admission to the high dependency unit (HDU) for close monitoring and inotropic support.

On day 3, his respiratory and cardiovascular state stabilised, but he reported of back pain and leg weakness. He was noted to have grade 1 power in the left leg and grade 0 power in the right. Reflexes were diminished, but his sensory signs had

not changed. He had normal perianal tone and sensation.

INVESTIGATIONS

Admission blood revealed a leucocytosis ($28.0 (4.0-10.0) \times 10^9/L$) acute kidney injury (creatinine 171 (62-106) $\mu\text{mol/L}$) and C reactive protein of 431 (<5) mg/L. Urine dipstick showed +proteinuria, but no evidence of infection. Chest radiograph showed evidence of right lower lobe consolidation. Foot radiograph showed osteomyelitis of the left distal phalanx. HIV serology was negative.

On the second day of admission, he had a significant rise in troponin T, and inverted T waves laterally on ECG, suggesting a non-ST elevation myocardial infarction. Echocardiogram showed poor left ventricular systolic function with ejection fraction of 35% and a small pericardial effusion, but no evidence of infective endocarditis.

As his neurological signs developed, and urgent whole spine MR scan revealed abscesses within the posterior elements of C1-C2 and T1-T3 with extensive epidural abscess from the cervical to the lower thoracic spine with cord compression and possibility of cord infarction.

DIFFERENTIAL DIAGNOSIS

The initial impression was that of osteomyelitis of the left big toe. Subsequent deterioration suggested a community acquired pneumonia, and possible sepsis syndrome secondary to a bacteraemia, later revealed to be due to *S. aureus*. The subsequent back pain and lower limb weakness suggested discitis or spinal abscess.

TREATMENT

Following microbiological advice, antibiotics were changed to fusidic acid and flucloxacillin. The neurosurgical team reviewed the patient following his MR scan and discussed his case at the neurosurgical multidisciplinary team meeting. A number of neurosurgeons felt that surgical intervention would be extremely difficult, and as there was evidence of spinal cord infarction, it was felt that surgery was unlikely to improve the neurological outcome. In addition, his poor clinical state and recent likely myocardial event meant that he was a very high-risk surgical candidate.

OUTCOME AND FOLLOW-UP

His sepsis improved and he was stepped down from HDU within 4 days. He continued intravenous antibiotics for a total of 6 weeks, and repeat MRI showed resolution of the spinal abscesses. His

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foot ulcer improved without surgical debridement. His paraparesis was slow to improve, and by week eight, he had grade 2 power in both legs. He was transferred to a spinal rehabilitation unit for ongoing rehabilitation.

DISCUSSION

Spinal epidural abscess (SEA) is rare, occurring in 3.0/10 000 hospital admissions/ year.¹ Diabetes appears to be a predisposing factor.² The condition presents classically with a triad of back pain, fever and motor weakness. Positive blood cultures and raised inflammatory markers are characteristic, and diagnosis is confirmed by MR scanning. Spinal cord injury occurs due to mechanical compression of the cord or vascular occlusion, which may eventually lead to spinal infarction. The formation of an SEA requires bacteria to enter the epidural space, often from haematogenous spread from a distant site (commonly skin or soft tissue) or local contiguous spread. In around a third of the cases, no source is identified. Management of SEA requires urgent spinal neurosurgical input, which may require surgical decompression of the spine. Medical therapy alone may lead to full recovery of the neurological function.

Cases of infected diabetic foot ulceration leading to metastatic abscesses have been reported.^{3–8} Sites of metastatic infection include psoas muscle, kidney, eye, joints and epidural space. *S.aureus* is the most commonly identified pathogen, and reported cases have had variable outcomes with some requiring surgery, or responding to medical therapy.

Patient's perspective

I have had diabetes for many years, and a foot ulcer in the past. I noticed the ulcer on my foot, and saw the chiropodist straight away, and was admitted to hospital. I never realised that a foot ulcer could lead to paraplegia. I am improving slowly and steadily. I hope by sharing this case other doctors will learn about it and it will help other people like myself.

Learning points

- ▶ Diabetes is a common cause of foot ulceration.
- ▶ Acute diabetic foot ulceration requires urgent multidisciplinary input to prevent amputation.
- ▶ Diabetic foot ulceration and osteomyelitis may occasionally lead to metastatic infections such as spinal abscesses.

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Competing interests None.

Patient consent Obtained.

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