

Back pain in a Bangladeshi worker in Iraq

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Abstract

Pyogenic Spinal Infection (PSI) is an uncommon disorder encompassing a broad spectrum of diseases including septic spondylodiscitis, osteomyelitis, epidural and paravertebral abscess formation. Presentation can be vague and highly variable but usually includes back pain and fever. Whilst predisposing factors, such as trauma and diabetes can often be identified a pathogenic organism may not be identified in up to a half of all cases leading to significant delay in both accurate diagnosis and effective treatment. Precise spinal imaging is essential and includes plain X-ray, CT and preferably MRI. The treatment of PSI can be conservative (including antibiotics); however, spinal surgery may be required for the complications in up to 50% of cases, with varying degrees of success. We present a challenging case of PSI encountered in a locally-employed 42 year-old Bangladeshi civilian working in Iraq. Despite obvious resource limitations available within a Role 2 Field Hospital, clinical suspicion coupled with repeat spinal CT was pivotal in obtaining the diagnosis. The patient was repatriated to Bangladesh for MRI and definitive surgical treatment.

Introduction

Pyogenic Spinal Infection (PSI) is an uncommon cause of unexplained fever and requires a high degree of suspicion accompanied by high quality spinal imaging – usually by CT or MRI scanning – for diagnosis. We present a case of PSI presenting to the Role 2 Field Hospital in Iraq in a locally employed Bangladeshi civilian which was diagnosed after repeated CT scanning.

Case Report

A 43-year-old Bangladeshi male, with an unremarkable past medical history, presented with a six day history of sacral pain following an accidental fall onto his back. He had been working as a caterer for 38 months for British Forces in the Contingency Operating Base in Basra (COB), Iraq and had not been home during this time.

On admission he was clinically well and afebrile with a heart rate of 80/minute and blood pressure of 101/62 mmHg. There was localized tenderness over the L5/S1 region with a minor superficial skin abrasion, but no focal neurological signs. The rest of the clinical examination including a detailed neurological examination was normal. Spinal x-ray revealed a chronic grade 1 spondylolisthesis of L5 on S1 with bilateral Pars defects, which may have explained his back pain.

Within eight hours of admission he had developed a fever (37.8°C) and tachycardia of 104/minute. His white cell count (WCC, neutrophils 90%) and C-reactive protein (CRP) were both elevated at 16.8 x10⁹ cells/L and 140 mg/L respectively. His fasting

blood glucose was normal. Urine dipstick was positive for nitrites, blood and leukocytes, but with a negative gram stain. He was commenced on oral trimethoprim for a presumed urinary tract infection, although he had no urinary tract symptoms. In the following 48 hours the patient failed to improve clinically with intermittent fevers up to 39.2°C, rigors and an elevated CRP (145 mg/L), WCC (27.2 x10⁹ cells/L) and ESR (62). The patient's midstream urine specimen grew a methicillin sensitive *staphylococcus aureus* (MSSA) which was sensitive to ciprofloxacin and gentamicin. Consequently, he was converted to oral ciprofloxacin (500mg bd) and intravenous gentamicin (5mg/kg/day). An abdominal/pelvic ultrasound was normal.

On the fourth day of admission he remained febrile with persistently elevated inflammatory markers (CRP 141mg/L, WCC 24.6 x10⁹ cells/L) and ongoing rigors. *Staphylococcus* sp was grown from his blood cultures, which was subsequently identified as MSSA, as noted in his MSU. He underwent a CT scan of his chest, abdomen, pelvis and spine. This showed no evidence of deep-seated intra-abdominal collections or infection of the lumbar-sacral spine. However, several small shotty para-aortic lymph nodes (<1cm) were noted with confirmation of the grade I anterior spondylolisthesis of L5 and S1. Consequently, he was commenced on high-dose intravenous flucloxacillin (8g/day) and rifampicin 600mg bd for a presumptive diagnosis of pyogenic spinal infection (PSI) with associated bacteraemia. Additional investigations revealed a negative malaria antigen test and a normal transthoracic echocardiogram with no evidence of cardiac vegetation or aortic root abscess.

The patient made a progressive and marked clinical improvement with a significant reduction in his CRP and neutrophilia. He was converted to high dose oral flucloxacillin and rifampicin following two weeks of intravenous therapy. The patient was jointly managed by the Orthopaedic Surgeon and Consultant

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Physician. At day 19, the patient was discharged with a CRP of 21.9 mg/L and WCC of 6.9×10^9 cells/L, on light duties with a plan of twice weekly ward review and liver function/CRP testing (Figure 1).

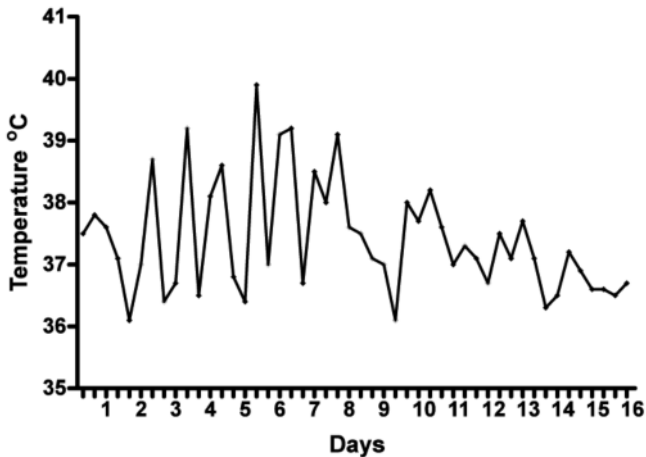


Figure 1. Eight hourly temperature chart

Unfortunately, five days following discharge, the patient was readmitted with an acute deterioration in his back pain in association with a small rise in CRP (28.8 mg/L). A repeat CT scan of his chest/ abdomen/pelvis and spine demonstrated complete destruction of his L5/S1 disc compared with his previous scan three weeks before, with an associated 3.0x1.5 cm collection lying anteriorly. The appearances were in keeping with a pyogenic discitis and an associated prevertebral collection/abscess. All other disc spaces and the sacroiliac joints were unremarkable (Figure 2).



Figure 2. Sagittal CT reformat scan on bony algorithm of the lumbar spine demonstrating marked loss of disc height at L5/S1 and the associated low attenuation prevertebral collection (arrow).

Given the need for definitive spinal surgical treatment the patient underwent urgent aeromedical transfer back to Bangladesh where he underwent urgent spinal MRI which confirmed extensive degenerative disease and a paravertebral collection (Figure 3). The L5-S1 disc space was obliterated and showed mild contrast enhancement consistent with a pyogenic discitis. At the L4-5 level

there was evidence of posterolateral osteophytosis causing bilateral neural foraminal narrowing. The patient underwent posterior decompression with an L4/5 discectomy. Histology and culture from the disc and surrounding tissue revealed no evidence of *mycobacterium tuberculosis*/MSSA on subsequent culture. The patient made an excellent postoperative recovery and was able to walk independently with only a minor limp and no neurological sequelae at six weeks post surgery.



Figure 3. Sagittal T2 weighted contrast MRI showing marked spondylololthosis of L5 & S1 vertebral bodies with obvious prevertebral collection at L5-S1 level.

Discussion

This case highlights an unusual and challenging case of PSI in a Bangladeshi civilian contractor working for UK forces. PSI is uncommon and encompasses a spectrum of pathologies which include spondylodiscitis, septic discitis, pyogenic facet arthropathy, vertebral osteomyelitis, and epidural abscess [1]. The lumbar-sacral spine is the most commonly affected site followed by the thoracic spine. Clinical studies of PSI have identified spondylodiscitis to be the most common clinical presentation [2]. *Staphylococcus aureus* is the most commonly identified pathogenic organism accounting for 30-70% of cases in the Western World [1-3]. However, globally, *mycobacterium tuberculosis* is associated with the greatest number of cases [2,3].

Predisposing factors for PSI include trauma, immunosuppression, diabetes mellitus, systemic infection (eg bacterial endocarditis), instrumentation (eg epidural anaesthesia) and intravenous drug abuse [1].

The presentation of PSI can be variable and neither a pathogenic organism nor predisposing cause is identified in up to a half of cases. This partly explains the often significant diagnostic delay, which may be up to two months from initial symptoms [1-3]. Key to the diagnosis is clinical suspicion in the first place and performing basic tests for inflammation, such as CRP and ESR with further complex imaging as indicated in any patient presenting with back pain and pyrexia.

Typical features of PSI include percussion-related focal back pain (90%), fever (60-80%) radicular signs (59%) and identifiable spinal cord symptoms (29%) [3]. It usually presents in the 5th or 6th decade with a slight male preponderance [1]. In a recent retrospective study of 79 patients with PSI admitted to the Mayo Clinic it was shown that the cumulative 1-year survival rates of microbiologically confirmed treatment failure were 100%, 89% and 56% for patients with improved, equivocal, and worse follow-up imaging findings, respectively (p=0.004) [4]. Patients with deterioration in their clinical status and/or inflammatory biomarker responses, as noted in our case, are at a higher risk for treatment failure and should undergo follow-up serial imaging. Indications for surgery, which is required in up to a half of cases,

include the need to obtain a bacteriologic diagnosis when other methods have failed, the presence of a clinically significant abscess, refractory infection despite prolonged non-operative treatment, cord compression with considerable neurological deficit, and substantial deformity or spinal instability [1,5].

The isolation of *staphylococcus aureus* in a mid stream urine specimen from a community patient without previous urinary tract instrumentation or history of lower urinary tract symptoms is highly unusual. It should always prompt investigations to identify staphylococcal bacteraemia or deep-seated occult infections. It is likely that the route of bacteriuria is via haematogenous seeding of *Staphylococcus aureus* to the kidneys and urinary tract [6,7]. In fact, staphylococcal bacteriuria has been reported in up to 27% of patients with staphylococcal bacteraemia in the absence of concomitant urinary tract symptoms or urinary tract catheterization [7]. The source of *staphylococcus aureus* infection in our patient is unknown. Given the temporal relationship of his infection to a recent mechanical fall it is difficult not to implicate this fall as the triggering event. The presence of chronic grade 1 spondylolisthesis at the site of the subsequent infection is also likely to have been a significant predisposing factor.

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