Infective Discitis Mimicking Infective Endocarditis and Osteoarthritic Back Pain

N Talsania, O Ogundipe

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Abstract

The case of a 76 year old lady with bacteraemia and persistent back pain is presented. Due to the presence of notable co-morbidities, she was initially managed as a case of possible infective endocarditis and severe osteoarthritis of the lumbar spine but subsequently found to have infective discitis. Both infective endocarditis and infective discitis represent conditions that require a high index of suspicion, early diagnosis and institution of appropriate management so as to prevent the development of potentially serious complications. A discussion on the evaluation of patients with infective discitis is presented highlighting some potential pitfalls and diagnostic cautions for the clinician.

INTRODUCTION

Back pain is a common problem in the adult population. Most cases are of benign causation. In older patients, intervertebral disc desiccation, bony degenerative changes and osteoporosis-related fractures are not uncommon contributory causes to the development of back pain. The need to identify potentially more serious causes in a timely fashion and thus prevent adverse sequelae is apparent, but poses a continuing challenge in routine clinical practise.

The case of an older patient who developed a potentially serious cause of back pain, in the form of infective discitis following bacteraemia is described.

CASE REPORT

A 76 year old lady presented to hospital with a history of tiredness, fatigue and poor appetite of five weeks duration. She complained of urinary frequency, vomiting and low back pain.

She had a history of rheumatic heart disease in childhood which subsequently resulted in severe tricuspid regurgitation (TR). Other history included pulmonary tuberculosis in childhood, chronic atrial fibrillation (AF), hypertension, recurrent cystitis and bilateral hip replacements for osteoarthritis (OA). She did not have diabetes mellitus and was not on immunosuppressive therapy.

More recently, she had a closed manipulative reduction of a dislocated left prosthetic hip five days prior to the current admission. A temporary urethral catheterization had been performed prior to the hip manipulation as she was noted to be in acute urinary retention. Inpatient stay at this time was brief, and the catheter was removed prior to discharge without recurrence of urinary retention.

Her medications were warfarin, digoxin, bumetanide, amiloride, atenolol, ramipril, doxazosin, pravastatin, nizatidine and lactulose.

On presenting again to hospital, her temperature was 37.5°C with recorded temperatures spikes over the course of the next day, peaking at 39.3°C. No peripheral stigmata of infective endocarditis were noted. She had bipedal oedema, bibasal crackles in her lungs, an elevated jugular venous pressure (JVP) and a murmur consistent with the known TR. Central nervous system examination was unremarkable. Features of osteoarthritis were noted and there was a pressure ulcer to the left gluteal area.

Laboratory tests showed a neutrophilia of 11.8 $x10^9$ cells/L, an elevated C-reactive protein (CRP) of 192mg/L and erythrocyte sedimentation rate (ESR) of 112mm/h. Haemoglobin was 11.5g/dL(normocytic normochromic), sodium 123mmol/L, potassium 4.6mmol/L, urea 22.5mmol/L and creatinine 192mmol/L.

Urine dipstick was suggestive of infection and midstream urine was sent for culture and sensitivities. ECGs showed rate-controlled AF and CXR findings were unremarkable. Plain radiographs of her pelvis, hips and lumbar spine

showed marked degenerative bone changes.

Treatment was started empirically with intravenous (IV) cefuroxime on suspicion of a urinary tract infection (UTI) with possible bacteraemia/septicaemia. The back pains were attributed to OA and analgesia prescribed.

Urine cultures grew Escherichia coli (E. coli). Three serial sets of blood cultures (first set prior to commencing antibiotics) all grew Staphylococcus aureus. The left gluteal ulcer swab grew Staphylococcus aureus and methicillin resistant Staphylococcus aureus (MRSA).

As she remained unwell, the possibility of infective endocarditis (IE) was revisited. Antibiotics were reviewed based on sensitivities and further microbiological advice to IV flucloxacillin and cefuroxime.

A trans-thoracic echocardiogram showed severe TR secondary to rheumatic heart disease. A trans-oesophageal echocardiogram showed severe TR. The other valves were unremarkable. No obvious vegetations were noted on either scan. A myeloma screen and an auto-antibody screen returned negative.

On advice from microbiologists, cefuroxime was discontinued after five days; flucloxacillin was continued for four weeks IV and a further two weeks orally.

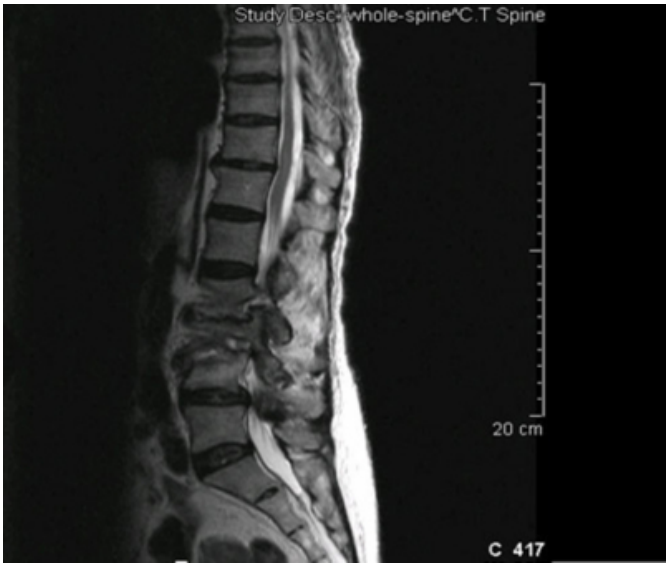
Following six weeks of antibiotics, the CRP and ESR remained elevated at 186mg/L and 126mm/h respectively. Six sets of blood cultures returned negative following the flucloxacillin therapy.

Clinically, the fever had resolved and she felt better in herself but the back pain had not improved. Repeat neurologic examinations showed no evidence of deficits.

An isotope bone scan showed increased uptake at L2/3 and L3/4 vertebral levels with evidence of bone destruction. In comparison to earlier plain films, the current lumbar views now showed marked disc space narrowing at L2/3 and L3/4 vertebrae. There was subluxation of L3 on L4 and evidence of some bone destruction at the adjacent end plates of L3 and L4. The features were interpreted as being highly suggestive of infective discitis. A magnetic resonance imaging (MRI) of the spine was recommended and results were compatible with infective discitis at L3/4 as depicted in Figure 1.

Figure 1

Figure 1: MRI spine showing abnormal high signal within the disc and oedema within the adjacent vertebral bodies at L3/4 level. Bony destruction of vertebral bodies is seen with some forward subluxation of L3 on L4 as well a spinal stenosis at this level.



IV vancomycin (through a Hickman line for central venous access) and oral fusidic acid were administered for six weeks. Her back pain gradually abated and her inflammatory markers showed a steady decline, reducing to an ESR of 37mm/h and a CRP of 30mg/L. She was discharged home with a three month course of oral linezolid subject to satisfactory haematological reviews.

DISCUSSION

Infective discitis refers to an infection of the intervertebral disc.[1,2] It is most often, though not exclusively of bacterial origin[2,3] and has been described as arising following direct inoculation (post-instrumentation/surgery), through haematogenous spread, or from contiguous spread from structures around the disc space.[2,4,5,6] The commonest organism isolated is Staphylococcus aureus [1,3,6,7] and the commonest site affected is the lumbar spine.[6] Isolated reports of discitis following urinary tract infections or genito-urinary instrumentation /procedures have been described.[2,8]

Infective discitis remains an uncommon, but potentially serious cause for back pain in adult and older patients.[1,3,9,10,11] Delayed diagnosis can occur and a high index of suspicion remains paramount to early diagnosis and the institution of appropriate management.[5,8,12]This is especially the case in the presence of pre-existing co-

morbidities.[2,3,4,6]

Delays in diagnosis of up to five months have been reported.[5] In this patient, definitive diagnosis was delayed by up to eight weeks because of distracting co-morbidities in the form of severe OA and the clinical suspicion of IE.

A temporary catheter had been inserted for acute urinary retention prior to reduction of a dislocated hip five days prior to presentation. With the noted abnormal urine dipstick, a genito-urinary portal of entry was considered the cause for a septicaemia.

Antibiotic prophylaxis for endocarditis is generally recommended for certain at-risk patient groups who are to undergo specific procedures.[13,14,15,16] According to the current European Society of Cardiology Guidelines of 2004, urethral instrumentation is accepted as one of the conditions likely to result in bacteraemia.[14]Prophylactic antibiotics against endocarditis are recommended in susceptible persons, as is the case of individuals with acquired valvular heart disease. [13,14,15,16] The need for this becomes more apparent if infected urinary tract tissue is involved.[14]In retrospect, this patient should probably have received endocarditis prophylaxis prior to her urethral catheterization given the history of recurrent cystitis and the presence of a severe acquired valvular heart disease.

Regardless, the UTI turned out to be of E. coli causation and the blood cultures were Staphylococcal in origin. This suggested that the urine was not the source of the bacteraemia but rather the left gluteal wound infection. The diagnosis of possible right sided valvular Staphylococcal endocarditis was entertained and treatment was given as guided by microbiological sensitivities. A ‘definite' diagnosis of IE would require the presence of varying combinations of major and/or minor criteria as set forth in the Duke criteria; and by further diagnostic modifications proposed by Durack et al and Li et al.[17,18] The criteria for a ‘definite IE' diagnosis were not satisfied in full in this patient. However, she met the diagnostic criteria for ‘possible IE' on the basis of one major Duke clinical criterion (three serial positive blood cultures) and two minor criteria (fever >38°C and a predisposing heart condition). The high associated morbidity and mortality in this condition if left untreated was borne in mind.[14]

Despite the delayed diagnosis, this patient suffered no neurological deficits as a result of the infective discitis. It must be reiterated that this is not the norm and severe, permanent deficits can occur.[8,10]Early recognition and treatment is still advocated as this can prevent the onset of neurological deficits. The latter more commonly presents following spread of the infection to the epidural space, or following collapse of vertebral bodies resulting in root or cord lesions, either by direct compression and/or vascular compromise.

The marked degenerative changes on plain lumbar radiography had been rightly attributed to severe osteoarthritis. However, the co-existence of osteoarthritis with infective discitis has been described.[6] It is equally noteworthy that plain radiographs may remain normal or unremarkable till later stages of the disease.[6,12] Typically in osteoarthritis, it is worthwhile to recall that the inflammatory markers tend be normal or non-significantly elevated. Though non-specific,[1,3,12]an aid to the diagnosis of infective discitis includes the markers of inflammation such as CRP and ESR which are usually raised.[3,4,10]In this case, the persistent back pain and continually elevated inflammatory markers despite prolonged antibiotics prompted further evaluation.

Bone scintigraphy can be used in cases where the exact site of pathology is not evident from clinical examination or radiographs. Although very sensitive, bone scans are not specific for infection.[6,12]

MRI is considered the best radiological investigation[1,2,3,4,6,19]though it is not infallible (particularly in early stages of the disease process).[20]

There is a definite role for invasive testing in form of diagnostic biopsy and cultures of vertebral disc material, particularly if diagnosis remains uncertain with initial workup. [2,4,12]

Blood cultures are valuable but negative cultures do not rule out the possibility of infective discitis. [4,8,10,12]Aerobic and anaerobic cultures must be ensured.[21]Even when blood cultures are negative, cultures from disc samplings may return positive and would further aid in guiding appropriate antibiotic choices.[4,12]Mycobacteria, fungi and rare organisms should be borne in mind, particularly in immunocompromised patients.[7,11,21]

There is no universal consensus on the type, duration or mode of administration of antimicrobial agents. A common approach is to give prolonged antibiotic treatment based on culture and sensitivity results till the inflammatory markers

normalise and symptom resolution is noted.[6,11] Empirical treatment may be required based on a consensus regime drawn up in liaison with microbiologists, especially when positive culture results are lacking.

Surgical decompression may be required on occasions to safeguard neurological viability (if irreversible deficits have not yet ensued), or as an option for the management of intractable pain.

CONCLUSION

This report hopes to serve as a timely reminder that co-morbidities can mask the presentation of infective discitis, thus resulting in delayed diagnosis. Infective discitis remains an important, albeit uncommon cause of persistent back pain. Early diagnosis requires a high index of suspicion and is best confirmed by MRI. The institution of appropriate management is best guided by antimicrobial sensitivities of relevant culture specimens, and could forestall the development of severe neurological deficits.

CORRESPONDENCE TO

Dr OA Ogundipe ola_ayodele@hotmail.com

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Author Information

N. Talsania, MB, BS
Senior House Officer in General (Internal) Medicine & Geriatric Medicine, Whiston Hospital

O. A. Ogundipe, MRCP (UK)
Specialist Registrar in General (Internal) Medicine & Geriatric Medicine, Whiston Hospital