

Citrobacter Koseri Bacteraemia Complicated by Paraspinal Abscess and Spondylodiscitis - A Case Report

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SUMMARY

Paraspinal abscess and spondylodiscitis due to *Citrobacter koseri* is a very rare condition. We report a remarkable case of *Citrobacter koseri* bacteraemia complicated by paraspinal abscess and spondylodiscitis in a patient who has successfully been treated in our hospital. Our patient demonstrates one of the common challenges in the practice of infectious disease medicine, wherein an innocuous presentation may and often underlie a serious infection. This case report elucidates to us that the diagnosis of a paraspinal abscess and spondylodiscitis requires a high index of suspicion in at risk patient presenting with compatible signs and symptoms.

KEY WORDS:

Citrobacter koseri, Paraspinal abscesses, Bacteraemia

INTRODUCTION

The clinical diagnosis of paraspinal abscess and spondylodiscitis is often quite challenging but failure to diagnose and treat this condition can be fatal. Its initial manifestations comprises of nonspecific clinical presentation such as fever, malaise and back pain. Routine laboratory studies and plain radiography are often unhelpful. Availability of MRI or CT scan has improved our ability to diagnose this condition accurately. The majority of these infections are caused by Gram positive organisms such as *Staphylococci* and *Streptococci* species. A small proportion can be due to Gram negative bacilli organism such as *E.coli*, *Klebsiella* and *Enterobacter* species. In our setting, *Mycobacterium tuberculosis* can also be a frequent pathogen for this infection. Bacteria can gain access to the epidural space via the hematogenous route, contiguously from infected site or direct inoculation during invasive spinal procedure. *Citrobacter* species have rarely been involved in paraspinal abscess and spondylodiscitis. To the best of our knowledge, this is the first report of *C. koseri* paraspinal abscess and spondylodiscitis in Malaysia.

CASE REPORT

A 75-year-old Malay lady with no significant past medical history, was admitted to our hospital in June 2010 with three weeks history of recurrent fever and persistent endurable lower back pain. There was no recent history of trauma, operations, injections or skin lesions in or around her lower back region. On examination, she was not clinically septic.

Her temperature was 39.5°C. Her renal punch was negative, there was no spinal or paraspinal tenderness, she has full power in all her limbs and there were no neurological deficits detected. The cardio-respiratory and abdominal examinations were unremarkable. Her white blood cell count (WBC) was $15 \times 10^9/L$ (normal range is $4.0-11.0 \times 10^9/L$) and C-reactive protein (CRP) was 35.8mg/L (normal range is 0.0-5.0mg/L). A lumbosacral X-ray disclosed no pathological changes. Magnetic Resonance Imaging (MRI) was performed and it revealed a spondylodiscitis at the L2/L3 level with large paravertebral and psoas abscesses (Figure 1 and 2). The spinal team was consulted and the incision and drainage of the psoas and the paravertebral abscesses was conducted. Histopathological examination (HPE) of these tissues revealed presence of inflamed granulation tissue and culture of the pus identified growth of *Citrobacter koseri* (*diversus*). The Ziehl-Nielsen staining of the pus was negative. The blood culture was also positive for *Citrobacter koseri* (*diversus*).

Two months prior to this presentation, she was admitted to a district hospital for ten days following a week history of fever and general unwellness. She denied dysuria at that juncture, but recalled having had frequency, nocturia and polyuria. She was empirically treated with intravenous ceftriaxone which was later changed to cefuroxime when her blood culture revealed growth of *Enterobacter* sp which was sensitive to cefuroxime, amikacin, ciprofloxacin and gentamicin and resistant to ampicillin. Her urine culture was negative and her chest X-ray was normal. She was discharge from the district hospital after completed one week of cefuroxime. Upon discharge, her fever had settled but her WBC and CRP were still elevated and she had started having the lower back pain. However, her back pain was not investigated further and it was attributed to her prolonged bed rest. She was referred to the physiotherapist for limb exercises.

Taking into consideration of her recent medical history, intravenous cefuroxime was started on the day of her admission to our hospital. The *Citrobacter koseri* (*diversus*) isolated from the patient's blood and paravertebral pus in our hospital showed similar sensitivity result to the *Enterobacter* sp isolated from her blood in the district hospital. Hence, cefuroxime was continued. Fortunately, our patient's fever settled after 48 hours of intravenous cefuroxime, the WBC normalized ten days later and her CRP normalized after three weeks. Her back pain has also completely resolved by the end of the fourth week of therapy. The antibiotic was continued for a total duration of 6 weeks. The MRI was not repeated at

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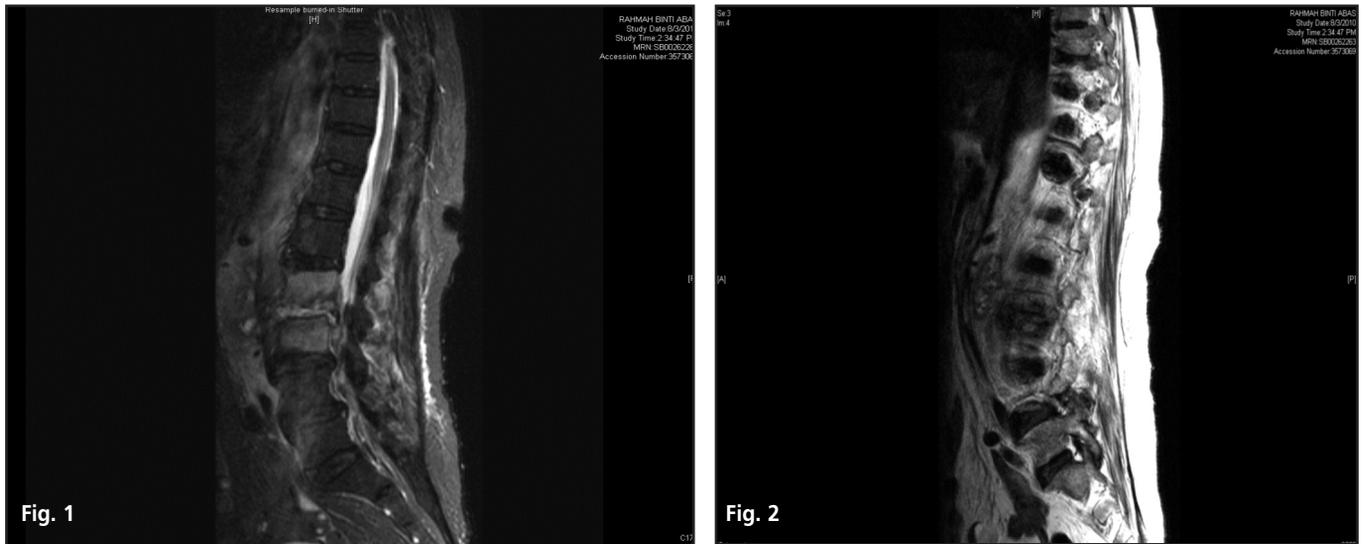


Fig. 1 & 2 : Magnetic Resonance Imaging (MRI) of the spine shows a spondylodiscitis at the L2/L3 level with large paravertebral and psoas abscesses.

end of therapy and she was discharged a few days later. She was reviewed at the outpatient clinic at three and six months after discharge and reported to us that she has completely recovered and is returning back to her normal life style.

DISCUSSION

Citrobacter species are frequently found in water, soil, food and as occasional colonisers of animals and humans gastrointestinal tract. *Citrobacter* belongs to the family of *Enterobacteriaceae* and comprises 11 different species of facultative anaerobic, motile, Gram-negative bacilli, which are oxidase-negative and typically utilize citrate as the sole carbon source. Among *Citrobacter* species, the most commonly isolated from human clinical specimens are *C. koseri* (formerly named *C. diversus*), *C. freundii*, *C. youngae*, *C. braakii*, and *C. amalonaticus*¹⁻³. Traditionally, *Citrobacter* species has been considered as a low virulence organism in human but its increasing importance as human pathogen has been described in the western literature since 1970s¹. Albeit rare, *Citrobacter* species has been linked to several types of infections such as urinary tract, intra-abdominal, wound, bloodstream and central nervous system infections¹⁻³. In particular, *C. koseri* has long been recognized to be the cause of meningitis and brain abscess in neonates. However, the literature on *Citrobacter* as a cause of spinal infection in adults is scant. In adult, *C. koseri* was most commonly isolated from the urinary tract and *C. freundii* was frequently isolated from the gastrointestinal tract, especially the gallbladder.

The risk factors for *Citrobacter* infection are not particularly known but studies of these infections have observed that it is more common in immunocompromised hosts, elderly, neonates and are frequently hospital acquired¹⁻³. Nevertheless, community acquired *Citrobacter* infections have also been documented. Patients infected with *Citrobacter* commonly have a history of invasive procedures

resulting with substantial tissue trauma or inflammation at the site of primary infection before bacteremia occurred. The incidence of *Citrobacter* infection in the western countries had been reported to be less than 1%². There is no epidemiology data on these infections in Asia countries but *Citrobacter* has been reported as a cause of significant infection with mortality rate as high as 6.8% in India³.

Despite the fact that *C. koseri* and *C. freundii* originate from the same enterobacteriaceae family, their antibiotic susceptibility patterns have been observed to be different⁴. Unlike *C. freundii*, *C. koseri* is rarely intrinsically resistant to ampicillin and first-generation cephalosporins as it lack the inducible ampC gene⁵. *Enterobacter* sp also belongs to the enterobacteriaceae family and this genus has similar characteristic to the *C. freundii* and is intrinsically resistant to ampicillin and first-generation cephalosporins due to the presence of the chromosomally encoded ampC gene⁵. In view of this patient's initial sensitivity results for the *Enterobacter* sp, it is very likely that this *Enterobacter* sp may well have actually been an unidentified *Citrobacter* considering the limited microbiology facility at the district hospital. We believed that the urinary tract infection (UTI) was the source of our patient's bacteraemia and the haematogenous spread of the uncontrolled bacteraemia has lead to the paraspinal abscess formation. Situation of our patient concord the findings in most reports on *Citrobacter* infection where UTI has been the predominant site of involvement. The only significant risk factor for *Citrobacter* infection in our patient was her advanced age.

The management of spinal infection requires input from multidisciplinary team involving the spinal surgeon, radiologist, microbiologist, infectious disease specialist and physiotherapist. The aim of treatment is to eradicate the infection, restore and preserve the structure and function of the spine and alleviate pain which is principally achieved by

surgical drainage, prolonged antibiotic therapy, adequate analgesics and physiotherapy^{6,8}. To this date, there are no uniform recommendations about duration of antibiotic therapy in spinal infection such as in the case of our patient. It is impractical and unethical to determine the optimal duration of antibiotic treatment of these infections by prospective, randomized clinical trials due to uncommonness and serious nature of the infection. Treatment recommendations are primarily based on retrospective studies, case series and expert opinions^{6,8}. In the literature, the total duration of treatment varies between 4 and 16 weeks depending on comorbidities, isolated microorganism and presence of neurological complications^{6,7}. Criteria for antibiotic discontinuation include symptoms resolution and normalization of the inflammatory markers⁷. For this reason, we have continued the antibiotic therapy in our patient for a total duration of 6 weeks.

MRI is definitely the preferred imaging method for diagnosing spinal infection. However, studies assessing the utility of MRI as a tool to monitor the response of patients with spine infection to therapy have shown that MRI lack correlation with patients' clinical features⁹. Furthermore, conducting a follow-up MRI would certainly impose a high cost in our setting. In consideration of the fact that our patient has clinically improved with normalization of her inflammatory markers, we have decided that repeating MRI at the end of her therapy is neither clinically necessary nor cost effective.

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