

## The first-ever reported primary spinal infection with *Aeromonas*

Meghena Mathew<sup>1</sup>, T P Bharadwaj<sup>2</sup>, P Senthur Nambi<sup>3</sup>, Ashwin Mani<sup>4</sup>

From <sup>1</sup>Consultant, <sup>4</sup>Senior Consultant, Department of Critical Care, <sup>2</sup>Senior Consultant, Department of Hematology, <sup>3</sup>Senior Consultant, Department of Infectious Disease, Apollo First Med Hospitals, Chennai, Tamil Nadu, India

### ABSTRACT

Back pain can be an insignificant symptom in most patients in the intensive care unit. However, a simple complaint of back pain can have a tell-tale behind it. We present the case of a 57-year-old patient who presented to us with thrombocytopenia, leukocytosis, and urinary symptoms. Investigating her back pain revealed that she had a spinal epidural abscess with a very rare organism like *Aeromonas hydrophila* with no risk factors for the same. She had no prior history of trauma or any spinal surgeries. Hence, this is a primary *Aeromonas* spinal infection with no other source of seeding from any other sites. Our understanding and review of the literature could not find us any other case reports ever reported.

**Key words:** *Aeromonas hydrophila*, Back pain, Gram-negative pathogen, Spinal *Aeromonas* infection, Spinal epidural abscess

A spinal epidural abscess can produce devastating deficits if not identified in a timely fashion. It is a rare infection with an incidence of 0.33–1.96 abscesses/10,000 hospital admissions per year [1]. Predominantly an infection caused by a Gram-positive pathogen, it can also be caused by Gram-negative pathogen. Even rarer and probably unreported is the primary spontaneous infection of the spine with *Aeromonas*. *Aeromonas* is predominantly associated with gastrointestinal, skin, and soft-tissue infections in humans [2]. All the case reports of *Aeromonas* spinal epidural abscess reported in the literature had an antecedent precipitating cause such as trauma or surgery [3].

Our review of the literature could not identify any antecedent case reports of primary spinal infection with *Aeromonas* and we believe that our case could be the first-ever reported primary infection of the spine with *Aeromonas*.


### CASE REPORT

A 57-year-old female known hypertensive for 10 years on metoprolol 50 mg once daily and telmisartan 40 mg once daily was referred to our intensive care unit (ICU) for altered sensorium and thrombocytopenia of 2 weeks duration. She was initially admitted to an outside hospital for drowsiness and urinary incontinence. Her electroencephalogram showed epileptogenic activity and the magnetic resonance imaging (MRI) brain showed small vessel ischemic changes. She had thrombocytopenia with a platelet count

of 80,000 in the outside hospital, which had dropped to 20,000/mm<sup>3</sup> on admission to our unit. Her urine culture was done at the other Center grew *Escherichia coli*, the sensitivity of which was not available. She was given steroids for worsening thrombocytopenia, the exact dose and duration of which was not known.

On examination in the emergency room in our unit, she was agitated with a Glasgow coma scale of E2V4M5, blood pressure of 130/70 mmHg, and her saturation was 98% on room air. Neurological examination revealed bilateral extensor plantar response with no focal neurologic deficits. Other system examinations were normal. She was shifted to the ICU for further evaluation.

The laboratory findings showed a hemoglobin of 13.5 g/dL, white blood cell count (WBC) of 35000 cells/mm<sup>3</sup>, with a differential count of 90% neutrophils, 6% lymphocytes, 3% monocytes, 1% myelocyte, and platelet count of 20000/mm<sup>3</sup>. The C-reactive protein (CRP) level (CRP) was 110 mg/L and procalcitonin was elevated at 2.77 mg/mL. Peripheral smear showed neutrophilic leukocytosis with myeloid left shift. Leukocyte alkaline phosphate was elevated. Liver function showed hyperbilirubinemia of 1.9 mg/dL, serum alanine aminotransaminase of 31 U/L, and aspartate transaminase of 20U/L. The urine routine showed 8–10 pus cells. After sending blood and urine for culture, she was started on broad-spectrum antimicrobials including meropenem, teicoplanin, and fluconazole. An echocardiogram showed left ventricle hypertrophy with normal left ventricle function. Ultrasound abdomen did not reveal any organomegaly and no other focus of infection. Computed tomography (CT) brain done for the altered

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**Correspondence to:** Dr. Ashwin, Mani, Senior Consultant, Critical Care Unit, Apollo First Med Hospitals, Chennai, Tamil Nadu, India. Email: ashwin.mani@gmail.com

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sensorium did not reveal any significant finding to explain the altered sensorium.

A multi-disciplinary team was involved in her care. Neurologist opinion was taken for epileptogenic activity and her anti-epileptic drugs including levetiracetam intravenously 500 mg twice daily and clobazam 5 mg once daily were continued. Her blood culture grew carbapenem-resistant *Aeromonas hydrophila* which was sensitive to amikacin (3 of 4 bottles) (Fig. 1). Subsequently, antibiotics were changed to intravenous amikacin 750 mg once daily. She was gradually more awake and responsive and was mobilized out of bed. While in ICU, she developed urinary incontinence and a fever spike on the seventh day of admission and complained of severe lower backache which was non-radiating. A repeat blood culture was sent again after 7 days of admission grew carbapenem-resistant *A. hydrophila*.

In view of recurrent bacteremia and excruciating back pain, an orthopedician opinion was obtained and an X-ray pelvis and lumbosacral spine were done and found normal. An infectious disease consultant review was obtained and a whole body positron emission tomography (PET) scan (Fig. 2) was done in view of persistent bacteremia, looking for a possible focus of infection. It showed linear FDG uptake in the body and end plates at the L4-L5 level and along the protruded disc, suggestive of infective discitis at the L4-L5 level and increased FDG uptake in the spleen and bone marrow of axial and proximal appendicular skeleton. In view of marrow uptake serum, protein immunotyping was done which showed the absence of monoclonal gammopathy. An MRI lumbosacral spine was done which showed features of spinal meningitis with long segment epidural abscess from the inferior endplate of D5 vertebrae to the L5 vertebral body with involvement of the anterior aspect of the spinal canal and spinal spondyloarthropathy (Figs. 3 and 4). A neurosurgeon's opinion

was obtained and the patient was advised to undergo CT-guided aspiration/biopsy of the abscess followed by surgical intervention. However, the family and patient were not willing to undergo any further intervention and wanted to continue intravenous antibiotics and were discharged against medical advice. A repeat blood culture done before discharge did not show any growth further. Complete blood count at discharge showed a WBC of 4500/mm<sup>3</sup> and platelet count of 60000/mm<sup>3</sup>.

**DISCUSSION**

Spinal infection is a complex condition and is a broad terminology that can involve any part of the spinal column. It can include vertebral columns or can spread inside the spinal canal and involve the spinal cord. The infection can spread to the spinal canal from a distant site through a hematogenous route, through contiguous spread from the adjacent site, direct inoculation following post-surgical intervention, or following a trauma. The most common sites for hematogenous seeding of the spinal column can occur from the urinary tract, gastrointestinal tract, oral cavity, respiratory tract, infective endocarditis, infected catheters, and skin and soft-tissue infection. Many case reports of spondylodiscitis have been reported after prostatic biopsy [4,5]. Despite this, nearly 50% of them may still not have any other identifiable primary source. Spinal epidural abscess is a rare entity and needs a high suspicion of the index. It has a reported incidence of 1.8/100,000 hospital admissions per year [6]. Predominantly a disease of the elderly, young intravenous abusers can also develop spinal epidural abscess. Epidural abscess predominantly involves the thoracic spine followed by the lumbar and cervical region [7]. Involvement at the level of the cervical cord can be disastrous as it can affect the diaphragmatic innervation which originates from C3 to C5 and can result in respiratory failure.

<b>CULTURE AND SENSITIVITY [BLOOD] : (Culture)</b>	
<b>Comments</b>	.
<b>Organism Name</b>	<b><u>Aeromonas hydrophila</u></b>
<b>Methodology Name</b>	<b>Kirby Bauer</b>
<b>Growth Observed</b>	<b>Yes</b>
<b><u>Antibiotics Name</u></b>	<b><u>Results</u></b> <span style="float: right;"><b><u>Zone Value</u></b></span>
<b>Cefotaxime</b>	<b>Resistant</b>
<b>Ceftriaxone</b>	<b>Resistant</b>
<b>Cefepime</b>	<b>Sensitive</b>
<b>Cefoperazone-Sulbactam</b>	<b>Resistant</b>
<b>Piperacillin-Tazobactam</b>	<b>Resistant</b>
<b>Aztreonam</b>	<b>Sensitive</b>
<b>Imipenem</b>	<b>Resistant</b>
<b>Meropenem</b>	<b>Resistant</b>
<b>Ertapenem</b>	<b>Resistant</b>
<b>Doripenem</b>	<b>Resistant</b>
<b>Cotrimoxazole</b>	<b>Sensitive</b>
<b>Gentamicin</b>	<b>Sensitive</b>
<b>Amikacin</b>	<b>Sensitive</b>
<b>Ciprofloxacin</b>	<b>Sensitive</b>

Figure 1: Blood culture sensitivity done at the time of admission

The abscess can involve multiple segments by the time of diagnosis and, if anteriorly located is associated with vertebral osteomyelitis. Some of the most common risk factors for spinal infections include diabetes mellitus, HIV/AIDS infection, malignancy, renal failure, hepatic cirrhosis, malnutrition, and chronic immunosuppressive therapy including steroids.

The most common microbiological etiology for a spinal epidural abscess is *Staphylococcus aureus* which attributes

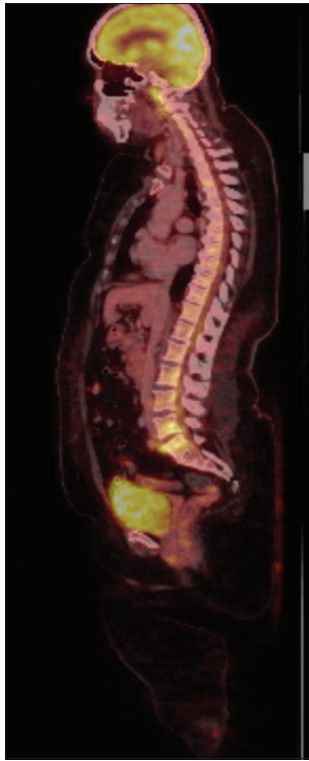


Figure 2: Positron emission tomography scan with linear FDG uptake in the body of L4–L5 vertebra

to two-thirds of all infections while approximately 5–20% of infections are due to streptococci and enterococci. *Enterobacteria* is the second leading cause of infection among which *E. coli* is the most common microorganism [8]. Spinal epidural abscess reported secondary to *E. coli* has been mostly seen in patients following urinary tract infection, prostatitis, or urological intervention. Some other Gram-negative bacteria associated with spinal infections include *Pseudomonas aeruginosa*, *Haemophilus influenzae*, and *Klebsiella pneumoniae*. Tuberculosis is an important etiology in the Indian context. Other less common pathogens associated with spinal epidural abscess include *Brucella* and fungi. Starting empirical treatment for these less common organisms is based on the index of suspicion and also depends on patient risk factors and endemic factors. Hence, invariably an opinion from infectious disease specialists in such complex sites of infections may be needed.

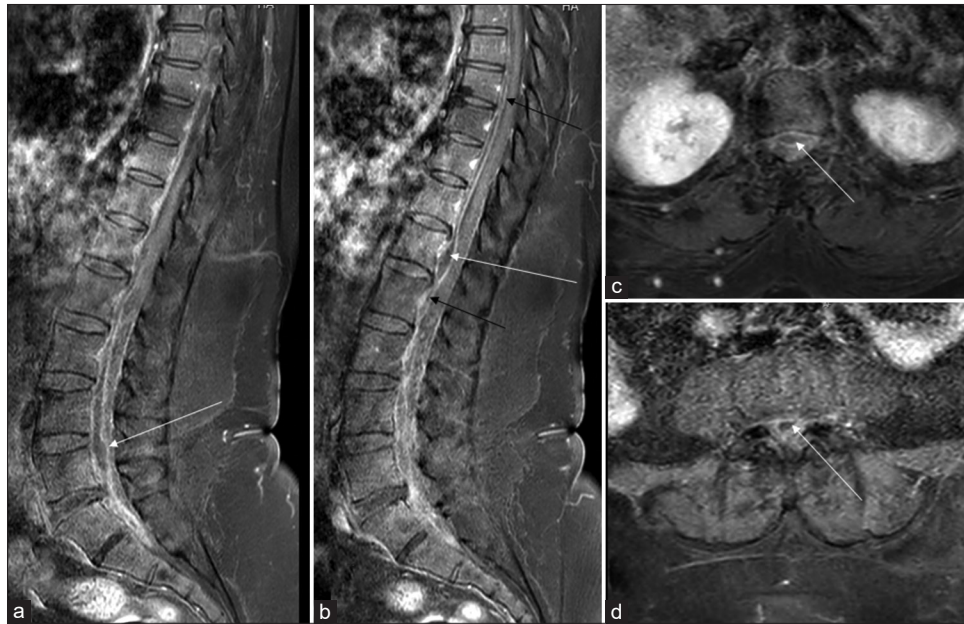
*A. hydrophila* is a motile, Gram-negative, non-spore-forming rod with facultative anaerobic metabolism. It is ubiquitous in our environment and is found very commonly in aquatic habitats, fish, foods, domesticated pets, invertebrate species, birds, ticks, insects, and natural soils. The predominant infection associated with *Aeromonas* include gastroenteritis, cellulitis and necrotizing fasciitis. There are reports of meningitis, peritonitis, respiratory infections, and ocular infections in humans. The oro-faecal route is the most common site for entry, followed by open wounds. *Aeromonas* is very frequently seen in immunosuppressed individuals, especially those with a history of neoplasm or chronic liver disease.

To the best of our knowledge, this is the first-ever reported case of primary spontaneous *Aeromonas* spinal epidural abscess. She had no prior history of trauma to the spine or surgical intervention done ever. The only other case of *Aeromonas* causing spinal infection was post-vertebroplasty reported in 2013 [3].



Figure 3: Magnetic resonance sagittal images of the dorsolumbar spine (a: diffusion imaging, b: T2 weighted, c: T1 weighted) show the presence of a long segment epidural abscess along the anterior aspect of the dorsolumbar spinal canal (white arrows) extending from inferior endplate of D5 vertebra to L5 vertebral body





**Figure 4:** Post-contrast T1 weighted FS magnetic resonance images of dorsolumbar spine (a and b: sagittal views, c and d: axial views) of the same patient show peripheral enhancement of epidural abscess (white arrows) with associated meninges enhancement (black arrows)

The most common presenting symptoms include fever, back or neck pain, and neurological deficit [9]. The absence of fever does not exclude spinal infection. Nearly 50% of patients with spinal infections can be afebrile. Back pain is a frequent complaint, and it can get difficult to differentiate mechanical causes from infection. It is mostly constant in nature and becomes worse at rest and night. The neurological manifestation can range from mild (radicular pain corresponding to a nerve root) to moderate in nature (motor weakness, sensory loss, bowel or bladder dysfunction) and can be severe resulting in paralysis. The development of paralysis is a sign of irreversible disease. Physical examination should include neurological examination, tenderness, and looking for any scars of prior surgery and obvious deformities. Since history and examination have a very low sensitivity and specificity in the diagnosis of spinal epidural abscess, diagnostic delays often occur.

The initial investigations should include white WBC, inflammatory markers such as CRP, ESR, and cultures including blood, urine, and imaging of the spine. WBC can be normal in up to 55% of patients with spinal infections. Blood cultures can be negative in 40% of the cases [10]. MRI with contrast of the whole spine is the most preferred imaging to look for an extension of the infection and for skip lesions. It has a sensitivity of about 95% and a specificity of over 90% [10]. Spondylodiscitis is a common finding seen in most of the patients with spinal epidural abscess and MRI has a good diagnostic yield even in the early stages of infection. Imaging should include the entire extent of the spine to look for skip lesions. There are concerns about performing lumbar puncture since it has a low diagnostic yield and also carries a risk of introduction of infection to subarachnoid space if performed without proper imaging done before introducing the needle. The role of FDG PET is limited since it cannot detect involvement of disc, vertebra, and spinal canal involvement distinctly. There is growing interest in other tracer-bound imaging in the field of

spinal infection, however, it has not replaced MRI in diagnostic accuracy. CT-guided aspiration when used in combination with CT or MRI has a higher yield and the aspirate should include culture sensitivity, nucleic acid amplification test, and histopathology. An open biopsy can be considered the next step if blood cultures and CT-guided aspiration are non-yielding and provided there is no immediate indication for surgical intervention.

The goals of the management of spinal infections essentially include eradication of infection and preventing neurological deterioration. While initiating empirical antibiotics for spinal canal infections the choice of antibiotics should include a third-generation cephalosporin, or fluoroquinolones, plus clindamycin, or vancomycin. This should be followed by de-escalation after culture sensitivity is available. The ideal duration of treatment for spinal canal infection is 4–6 weeks [11]. However, *Aeromonas*-related spinal canal infection will also need follow-up with spinal imaging to decide the duration of antibiotics.

Early surgical decompression should be considered along with intravenous antibiotics in case of acute neurological deficit or in situations of progressive neurological deficit. Delayed surgical decompression is mostly associated with poor neurological outcomes.

## CONCLUSION

Spinal infection is a complex condition and will demand a multi-disciplinary approach involving surgeons, infectious disease specialists, radiologists, and the physician. An early diagnosis and treatment improves prognosis. Our review of the literature does not show any prior case of spontaneous primary spinal infection due to *Aeromonas* in an immunocompetent individual. Persistent bacteremia with a rare organism like *Aeromonas* led to a detailed investigation and eventually narrowed down the source to the spinal epidural abscess.

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