

Spinal Epidural Abscess Unmasking Streptococcus Gallolyticus Endocarditis: A Rare but Treatable Entity

CHERISHA SELVARAJ¹, RANGARAMANUJANAIDU², N VASANTHI³

ABSTRACT

Infective Endocarditis (IE) is a microbial infection of the endocardial surface of the heart, most often involving the cardiac valves, leading to the formation of vegetations composed of organisms and inflammatory debris. While many patients with IE experience joint, muscle and back pain, actual infections at these sites are uncommon. Spinal abscess formation represents an uncommon yet clinically significant complication seen in patients with IE. Among these various spinal locations, involvement of the lumbar region is particularly rare. When it does occur, it typically results from haematogenous spread of infecting microorganisms from the cardiac vegetation to the vertebral or epidural spaces. These abscesses may present with back pain, neurological deficits or signs of systemic infection, making early recognition and treatment crucial to prevent serious outcomes. Although surgical intervention is typically the mainstay of therapy for spinal abscesses, conservative management can still be considered and prove to be effective in selected cases. We present a case of a 52-year-old gentleman with complaints of low back pain over a duration of three months, who, on Magnetic Resonance Imaging (MRI) was diagnosed to have a lumbar epidural abscess caused by *Streptococcus gallolyticus* endocarditis. He was subsequently treated with an 8-week course of intravenous antibiotics. This case highlights the importance of considering spinal epidural abscess as a potential complication in patients with bacterial endocarditis, thereby underscoring the critical need to include spinal epidural abscess in the differential diagnosis when evaluating patients with bacterial endocarditis who develop new or unexplained spinal symptoms.

Keywords: Intervention, Microorganism, Subaortic vegetation

CASE REPORT

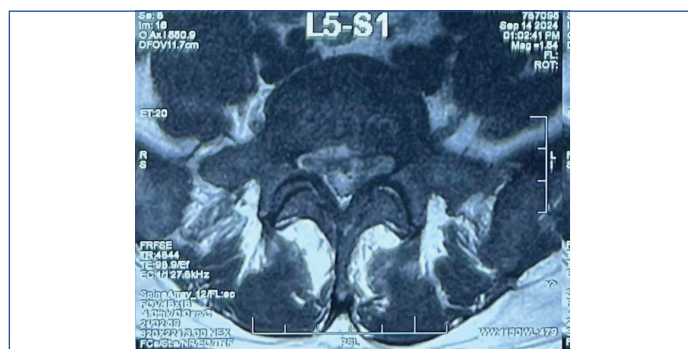
A 52-year-old gentleman, a chronic alcoholic (for about 20 years) with no known comorbidities, was admitted with severe back pain radiating to both legs associated with low-grade fever, which is intermittent in nature for the duration of three months. MRI of the lumbosacral spine showed a highly enhanced epidural abscess at the S1 level with features suggestive of infective spondylolisthesis at L5-S1. Blood culture was taken, which showed growth of *Streptococcus gallolyticus*. He was started on Injection Ceftriaxone 2 grams i.v. BD followed by oral Cefuroxime for two weeks and oral Linezolid for one week and was discharged following symptomatic improvement. Four weeks later, he further developed high-grade fever, with a temperature of ~101 degrees Fahrenheit, associated with abdominal pain, loose stools and pedal oedema for a three-day duration. On examination, he had pallor, bilateral pitting pedal oedema, with a pulse rate of 113/min and blood pressure of 140/90 mmHg. Chest auscultation revealed a diastolic murmur in the aortic area. On neurological examination, no motor weakness or sensory loss. Investigations showed total leukocyte count of 9060/ μ L, Erythrocyte Sedimentation Rate (ESR) 116 mm/hr, and C-Reactive Protein (CRP) 39 mg/dL. Liver function test revealed Aspartate Aminotransferase (AST) of 361 U/L and Alanine Transaminase (ALT) of 268 U/L. Chest X-ray showed no active lesion in the lungs or cardiac enlargement. Transoesophageal Echocardiography (TEE) depicted a thickened aortic valve, vegetations present in non-coronary cusps: approximately 0.8 cm above the aortic valve, moderate Aortic Regurgitation (AR), mild Tricuspid Regurgitation (TR) with an ejection fraction of 42%. The patient was started on conservative management with Injection Ceftriaxone 2 gm i.v. BD for nine days and Injection Vancomycin 500mg i.v. BD for nine days. After one week of i.v. antibiotics, the patient's culture reports were sterile and the patient was advised for early aortic valve replacement. Details of the follow-up aren't

available as the patient moved to another city for residence and further treatment.

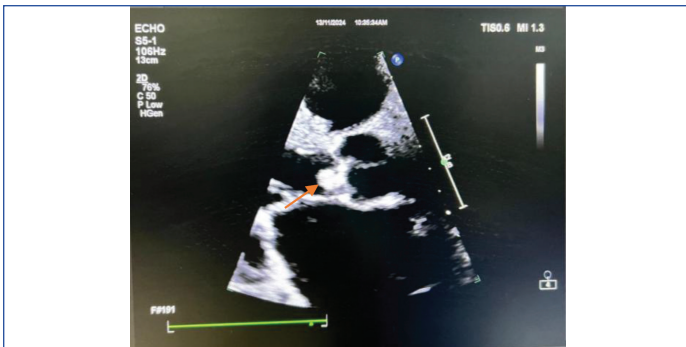
Below mentioned is the lumbar spinal epidural abscess noted on MRI at the level of L5-S1 [Table/Fig-1,2] and a two-dimensional echocardiography image in the paralongitudinal axis showing the presence of a subaortic vegetation [Table/Fig-3].



[Table/Fig-1]: Magnetic Resonance Imaging (MRI) of lumbosacral spine showing a spinal epidural abscess at the level of L5-S1.



[Table/Fig-2]: Magnetic Resonance Imaging (MRI) of the lumbosacral spine showing a spinal epidural abscess at the level of L5-S1.



[Table/Fig-3]: A 2D ECHO in the paralongitudinal axis showing a subaortic vegetation.

DISCUSSION

Spinal epidural abscess is a surgical emergency that requires immediate decompression [1]. The common causes of epidural abscesses are surgical interventions in the spinal canal and spread from nearby bone or soft tissues. Conservative management of epidural abscess is reserved for patients with poor surgical risk or patients without any neurological deficit [2]. The association between spinal epidural abscess and IE has rarely been described [3]. *S. gallolyticus* has been reported to cause SEA only on three occasions earlier-all were associated with endocarditis or colonic malignancy [4,5]. Spinal epidural abscess is a rare condition, with an estimated incidence of 0.2-1.2 cases per 10,000 hospital admissions in Western countries [6]. Common causes include spinal canal interventions (such as lumbar puncture or surgery), haematogenous spread from skin or soft-tissue infections, direct extension from vertebral osteomyelitis, abdominal abscess, mediastinal infections, and, in rare instances, IE. Around 14-22% of epidural abscess cases are linked to spine surgery or percutaneous spinal procedures [7]. Blunt trauma may precede the onset of symptoms in 15-35% of cases [8]. The most frequent pathogen, *Staphylococcus aureus*, is responsible for up to 70% of spinal epidural abscesses, followed by *Streptococcus* species, which account for approximately 7% [7]. Rare causes include *Mycobacterium tuberculosis*, fungal species, and parasitic infections. *S. gallolyticus*, previously known as *Streptococcus bovis* type 1, is a coloniser of human skin in 2-15% of the human population. The correlation between *S. gallolyticus* bacteraemia and colorectal carcinoma has been well established through the adenoma-carcinoma sequence [9]. A Danish study of 6,506 streptococcal Bloodstream Infections (BSIs) (mean age 68 years, 53% men) found an overall IE prevalence of 7.1%.

- Lowest IE prevalence: *S. pneumoniae* (1.2%) and *S. pyogenes* (1.9%)
- Intermediate prevalence: *S. anginosus* (4.8%), *S. salivarius* (5.8%), *S. agalactiae* (9.1%)
- Highest prevalence: *S. mitis/oralis* (19.4%), *S. gallolyticus* (30.2%), *S. sanguinis* (34.6%), *S. gordonii* (44.2%), *S. mutans* (47.9%). In multivariable analysis (reference: *S. pneumoniae*), all species except *S. pyogenes* showed a significantly increased

IE risk, with the highest odds for *S. mutans* (Odds Ratio [OR] 81.3) and *S. gordonii* (OR 80.8) [10].

In our case, the initial blood culture results identified *S. gallolyticus*. The three most common pathogens responsible for IE are *Streptococcus viridans*, *S. aureus*, and coagulase-negative *Staphylococci* [11]. The treatment for spinal epidural abscess typically involves decompressive laminectomy, debridement, and antibiotics [12]. However, many cases can be managed conservatively [13]. Conservative management is generally reserved for patients at high risk due to concomitant medical conditions, those with an extensive epidural abscess (spanning from the cervical to lumbar region), patients with complete paralysis lasting over three days, or those with no neurological deficit [1]. In our case, conservative management was chosen as the patient was neurologically intact, and his condition showed improvement.

CONCLUSION(S)

The IE presenting with a spinal epidural abscess, though a rare and radical condition, must be considered in any patient presenting with back pain and when the focus of infection cannot be locally ascertained. This case report elaborates on a non-specific and variable presentation of IE, a condition that can masquerade as other illnesses. The variability in the symptoms poses a diagnostic challenge for clinicians, thereby warranting thorough evaluation of the patients. With adequate therapeutic management, resolution of the infection can be achieved, helping to prevent severe complications and improve patient outcomes.

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PARTICULARS OF CONTRIBUTORS:

1. CRRI, Department of General Medicine, Pondicherry Institute of Medical Sciences, Puducherry, India.
2. Assistant Professor, Department of General Medicine, Pondicherry Institute of Medical Sciences, Puducherry, India.
3. Professor, Department of General Medicine, Pondicherry Institute of Medical Sciences, Puducherry, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Cherisha Selvaraj,
2/47, Puliady, Vadasery, Nagercoil-629001, Tamil Nadu, India.
E-mail: doctorcherisha@gmail.com

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