



STENOTROPHOMONAS MALTOPHILIA LUMBAR SPONDYLODISCITIS IN A CKD PATIENT

Orthopaedics

Dr Abhishek Kumar*

Fellow Spine Surgery, Bharati Hospital & Research Centre, Pune *Corresponding Author

Dr Mandar Borde

Head Spine Unit, Bharati Hospital & Research Centre, Pune

Dr Akash Mane

Assistant Professor, Spine Unit, Orthopedics Department, Bharati Hospital & Research Centre, Pune

Dr. Tanmay Datey

Senior Resident, Spine Unit, Bharati Hospital & Research Centre, Pune

Dr K.V. Menon

Consultant Spine Surgeon, Rajagiri Hospital, Kochi.

ABSTRACT

Stenotrophomonas maltophilia is an increasingly recognized cause of severe nosocomial infections, particularly in immunocompromised individuals. Community-acquired infections have also been reported. We present a case of lumbar spondylodiscitis caused by *S. maltophilia* in a chronic kidney disease (CKD) patient undergoing maintenance hemodialysis. This report highlights the diagnostic challenges and management of such infections in an immunocompromised host. The discussion also elaborates on the pathogen's biofilm-forming capabilities, antimicrobial resistance, and the importance of multidisciplinary management in achieving favorable outcomes.

KEYWORDS

Spondylodiscitis, Stenotrophomonas maltophilia, Chronic Kidney Disease, Hemodialysis, Nosocomial Infections

INTRODUCTION

Stenotrophomonas maltophilia is a multidrug-resistant, non-fermenting, gram-negative aerobic bacillus found in water, soil, and plants. While it is of low virulence, it frequently colonizes body fluids, especially in patients subjected to prolonged antibiotic exposure^[1]. *S. maltophilia* is increasingly recognized as a cause of severe nosocomial infections, particularly in immunocompromised patients^[2]. Its association with biofilm formation on indwelling medical devices complicates its management^[3]. Although rare, community-acquired infections can also occur. This case report describes lumbar spondylodiscitis due to *S. maltophilia* in a CKD patient undergoing hemodialysis.

Case Report

A 47-year-old male, hotelier from Pune presented with a one month history of low back pain, which aggravated on walking and getting up from seated position and radiating to the left lower limb. He reported neurogenic claudication over left leg (10 minutes walking time), sleep disturbances due to pain, urinary incontinence, and left footwear slippage. Examination revealed normal gait, heel and toe walking, restricted spinal flexion and painful extension. Neurological evaluation showed normal motor and sensory functions, diminished left ankle reflex, and bilateral pedal edema and positive bilateral slump test.

The patient had a significant history of multiple chronic conditions. He was diagnosed with diabetes mellitus and hypertension 15 years ago, both of which were managed with oral hypoglycemics and antihypertensive medications, respectively. Three years prior, he was diagnosed with chronic kidney disease (CKD) and had been on maintenance hemodialysis for the last 2.5 years. Two years ago, he underwent angioplasty for coronary artery disease and had been on antiplatelet therapy with aspirin since the procedure. Additionally, he was diagnosed with Hepatitis C virus infection three months before the current presentation and received treatment with sofosbuvir and velpatasvir. This complex medical history contributed to his immunocompromised status, predisposing him to severe infections.

A summary of laboratory findings is presented in Table 1.

Parameter	Value	Reference Range
Leukocyte count (μL)	7500	4000–11,000
Hemoglobin (g/dL)	10.9	13.0–17.0
ESR (mm/h)	57	<20
C-reactive protein (mg/dL)	79.67	<0.5
Blood urea (mg/dL)	30	10–50
Creatinine (mg/dL)	3.47	0.6–1.3
Total bilirubin (mg/dL)	0.9	0.2–1.2

Albumin (g/dL)	3.7	3.5–5.5
Aspartate transferase (U/L)	14	0–35
Alanine transferase (U/L)	7	0–45
Alkaline phosphatase (U/L)	92	40–150
HCV viral load (S/CO)	1.59	<1.0
Leukocyte count (μL)	7500	4000–11,000
Hemoglobin (g/dL)	10.9	13.0–17.0
ESR (mm/h)	57	<20
C-reactive protein (mg/dL)	79.67	<0.5
Blood urea (mg/dL)	30	10–50
Creatinine (mg/dL)	3.47	0.6–1.3
Total bilirubin (mg/dL)	0.9	0.2–1.2
Albumin (g/dL)	3.7	3.5–5.5
Aspartate transferase (U/L)	14	0–35
Alanine transferase (U/L)	7	0–45

Radiological investigations revealed significant abnormalities consistent with spondylodiscitis. X-ray imaging of the lumbar spine demonstrated lucency at the anterosuperior corner of the L5 vertebral body, accompanied by surrounding sclerosis, indicative of structural compromise as shown in Figure 1.



Figure 1- Radiograph showing lucency at anterosuperior corner of LV5.

Further evaluation with contrast enhanced magnetic resonance imaging (MRI) showed T2-weighted axial and T1 weighted sagittal images showing hyperintensity at the L4-L5 vertebral level, consistent with inflammatory changes typical of spondylodiscitis as shown in **Figure 2, 3** respectively. Notably, contrast enhanced coronal image showed paravertebral abscess or muscular involvement (**figure 4**), which helped in narrowing the differential diagnosis and planning subsequent management strategies.

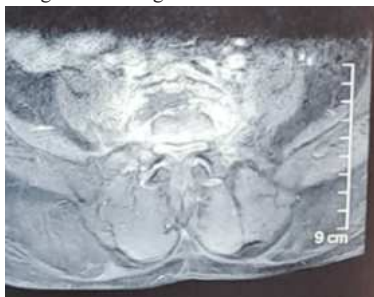


Figure 2 Contrast enhanced T2 axial MR image

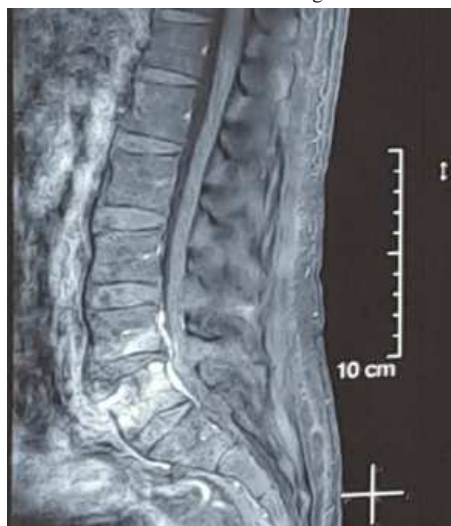


Figure 3 Contrast enhanced T1 sagittal MR image



Figure 4 Contrast enhanced coronal image

Management

The patient underwent L4 and L5 decompressive laminectomy, thorough debridement, discectomy, interbody fusion using an autologous graft and L3 - S2 AI fixation (**Figure 5, 6**). Intraoperative samples were sent for microbiological and histopathological analysis. Microbiological studies identified *Stenotrophomonas maltophilia* (**Figure 7**), which was sensitive to levofloxacin, minocycline, and trimethoprim-sulfamethoxazole^[4].



Figure 5: Post op AP radiograph of L3 - S2 AI fixation



Figure 6: Post op lateral radiograph of L3 - S2 AI fixation

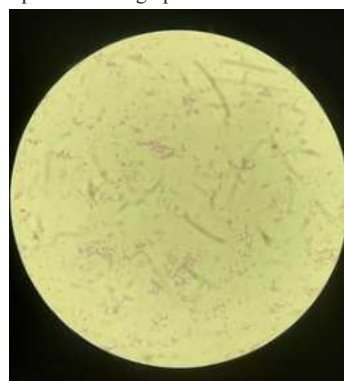


Figure 7- *Stenotrophomonas maltophilia*

Postoperative treatment included intravenous minocycline 100 mg twice daily for two days, followed by oral minocycline and trimethoprim-sulfamethoxazole for one month. The patient showed

significant clinical improvement, with resolution of symptoms and return to normal daily activities.

DISCUSSION

Stenotrophomonas maltophilia has emerged as a pathogen of clinical significance, particularly in immunocompromised populations. Its multidrug-resistant nature, coupled with its ability to form biofilms, presents challenges for effective management^[5]. Studies have demonstrated that *S. maltophilia* infections are associated with substantial morbidity and mortality in patients with predisposing conditions such as chronic kidney disease, malignancy, or those receiving immunosuppressive therapies^[6].

The pathogen's propensity for biofilm formation on medical devices, including catheters and implants, significantly complicates its eradication^[7]. Biofilms provide a protective niche against host immune responses and antimicrobial agents, contributing to persistent infections^[8]. Another study emphasized the critical role of biofilms in these infections and explored potential therapeutic strategies such as enzyme inhibitors and quorum-sensing blockers^[9].

The cornerstone of *S. maltophilia* management is antimicrobial therapy tailored to susceptibility profiles. Co-trimoxazole remains the first-line treatment, with alternative agents like minocycline and levofloxacin being vital in cases of resistance or intolerance^[4]. In this case, the combination of surgical debridement and antimicrobial therapy led to clinical resolution. Surgical intervention is often necessary to manage spondylodiscitis, particularly when structural integrity is compromised. Debridement reduces microbial load and prepares the site for effective antimicrobial penetration^[7].

Emerging literature emphasizes the importance of multidisciplinary care in managing such complex infections. Close collaboration among nephrologists, infectious disease specialists, and surgeons ensures optimized outcomes. For immunocompromised hosts, such as CKD patients on hemodialysis, vigilant monitoring and prompt intervention are critical to minimize complications and mortality^[6].

Future directions for research include exploring novel antimicrobial agents and strategies to disrupt biofilms, improving diagnostic tools for early pathogen detection, and establishing standardized protocols for managing *S. maltophilia* infections. These advancements could significantly reduce the disease burden associated with this opportunistic pathogen^[8-10].

CONCLUSION

This report emphasizes the diagnostic and therapeutic complexities of managing *Stenotrophomonas maltophilia* infections in immunocompromised patients. Multidisciplinary collaboration, early surgical intervention, and tailored antimicrobial therapy were key to the successful management of this case of lumbar spondylodiscitis due to rare organism.

REFERENCES

- Brooke, J.S. Advances in the microbiology of *Stenotrophomonas maltophilia*. Clin Microbiol Rev. 2021;34(3).
- Looney, W.J., Narita, M., Mühlemann, K. *Stenotrophomonas maltophilia*: An emerging opportunist human pathogen. Lancet Infect Dis. 2009;9:312–323.
- Adegoke, A.A., Stenstrom, T.A., Okoh, A.I. *Stenotrophomonas maltophilia* as an emerging ubiquitous pathogen: Looking beyond contemporary antibiotic therapy. Front Microbiol. 2017;8:2276.
- Falagas, M.E., Kastoris, A.C., Vouloumanou, E.K., Rafailidis, P.I. Therapeutic options for *Stenotrophomonas maltophilia* infections beyond co-trimoxazole: A systematic review. J Antimicrob Chemother. 2009;64(5):889–894.
- Maria F. Mojica, Romney Humphries, John J. Lipuma, Amy J. Mathers, Gauri G. Rao, Samuel A. Shelburne, Derrick E. Fouts, David Van Duin, Robert A. Bonomo, Clinical challenges treating *Stenotrophomonas maltophilia* infections: an update, JAC-Antimicrobial Resistance, Volume 4, Issue 3, June 2022, dlac040.
- Kuwahara M, Noma M, Sunakawa T, Shirai K, Kohama K, Miyawaki A, Hirata JJ. *Stenotrophomonas maltophilia* Bacteremia Associated With Severe COVID-19: Successful Treatment With Appropriate Antimicrobial Therapy. Cureus. 2022 Jan 30;14(1):e21750.
- Di Bonaventura G, Spedicato I, D'Antonio D, Robuffo I, Piccolomini R. Biofilm formation by *Stenotrophomonas maltophilia*: modulation by quinolones, trimethoprim-sulfamethoxazole, and ceftazidime. Antimicrob Agents Chemother. 2004 Jan;48(1):151-60.
- García, G., Giron, J. A., Yañez, J. A., & Cedillo, M. L. (2022). *Stenotrophomonas maltophilia* and Its Ability to Form Biofilms. Microbiology Research, 14(1), 1–20.
- Wang, A., Wang, Q., Kudinha, T. et al. Effects of Fluoroquinolones and Azithromycin on Biofilm Formation of *Stenotrophomonas maltophilia*. Sci Rep 6, 29701 (2016).
- Wu, J., He, S., Guo, S., et al. The role of biofilms in *Stenotrophomonas maltophilia* infections and their potential therapeutic strategies. Front Cell Infect Microbiol. 2020;10:561324.