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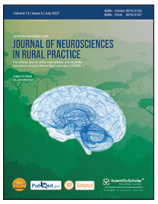

Case Report

Enterococcus faecium meningitis: A challenging pathogen in the central nervous system

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ABSTRACT

Enterococcus faecium meningitis is typically linked to procedures, devices, or severe immunosuppression. This report discusses a 66-year-old man with chronic obstructive pulmonary disease (COPD) who developed *E. faecium* meningitis without conventional risk factors, suggesting a shift in regional microbiological trends. The patient was previously hospitalized for an acute COPD exacerbation and developed a catheter-associated urinary tract infection with vancomycin- and linezolid-sensitive *E. faecium*. Despite linezolid, he experienced persistent headaches and altered sensorium following discharge. On readmission, he exhibited drowsiness, neck stiffness, and a positive Kernig's sign. Cerebrospinal fluid (CSF) analysis suggested bacterial meningitis, with *E. faecium* resistant to ampicillin but sensitive to vancomycin and linezolid in culture. Magnetic resonance imaging suggested ventriculitis. Initial empirical treatment was modified to vancomycin, based on the sensitivity report, with the addition of linezolid when the second CSF culture showed persistent infection. Meningitis resolved with CSF clearance and a negative brain computed tomography scan. He was shifted out of the intensive care unit, but developed nosocomial pneumonia and died. The increasing pathogenicity of *E. faecium* in patients with chronic medical conditions or prior antibiotic use, as observed in this case, is concerning. Gut dysbiosis from antibiotic exposure and COPD may have facilitated colonization by this organism. Linezolid serves as a vital adjunct to vancomycin. This case underscores the need for due vigilance by clinicians while treating *E. faecium* infections.

Keywords: Antimicrobial resistance, Bacterial, Catheter-associated infections, Chronic obstructive pulmonary disease, *Enterococcus faecium*, Meningitis

INTRODUCTION

Enterococcus faecium meningitis, a rare but fatal condition, tends to occur in patients with epidural anesthesia, neurosurgery, devices such as epidural catheter or shunt, or occasionally hematogenous spread in the immunocompromised, like post stem cell transplantation.[1-5] Until recently, *Enterococcus faecalis* caused intensive care unit (ICU) infections, while *E. faecium* isolates from catheter cultures were dismissed as colonization. At present, urinary tract, wound, and central line-associated bloodstream infections by *E. faecium* are increasing.[5] According to the European Center for Disease Prevention and Control, *E. faecium* was the fastest-growing pathogen in Europe in 2022. Vancomycin-resistant enterococcal infections remain a serious concern, as no optimal therapy has been established. We report a case of *E. faecium* meningitis with no prior surgical history.

CASE REPORT

A 66-year-old gentleman with chronic obstructive pulmonary disease (COPD) presented with drowsiness. He had been hospitalized a month earlier for an acute COPD exacerbation. During that stay, he developed a lower urinary tract infection with grade 1 prostatomegaly and significant post-void residual urine (388 mL) on ultrasonography. *E. faecium*, sensitive to vancomycin and linezolid, grew in urine culture, for which he received IV linezolid for an unspecified duration. His medical history was otherwise unremarkable, with no history of head injury, surgery, or immunosuppression.

Following discharge, he experienced persistent headaches and was readmitted with altered sensorium. On ICU admission, his Glasgow coma scale score was impaired (E3V4M5). His sensorium worsened (E1V1M2) in the next 7 h, requiring intubation. He had severe neck stiffness and positive Kernig's sign, but no focal neurological deficits. He was afebrile, with stable vitals except for mild tachypnea.

Blood investigations and chest X-ray were unremarkable except for polymorphonuclear leukocytosis [Table 1]. Electroencephalogram and brain computed tomography (CT) scan were normal. Magnetic resonance imaging brain

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Table 1: Laboratory investigations.

Investigations	Day 1	Day 6	Day 11
Blood Culture	Negative	Negative	Negative
Urine culture	Negative		
CSF study			
Culture	*Enterococcus faecium*	*E. faecium*	Negative
TC /mm^3	400	1000	350
DC (%)	P 80 L 20	P 83 L 18	P 78 L 20
Protein**	253	39	30
Sugar*	33	72	80
Blood count			
Hb	13.5**	10.3	9
TC	15500	18900	15900
DC	P89 L6 E1 M4	P84 L9 E4 M3	P82 L12 E3 M3
Procalcitonin	0.59		
Urea*/Creatinine*	17/0.6	24/0.4	30/1.6
Liver function test			
S. Bilirubin	0.9*	0.6	0.6
AST/ALT	44/43#		
ALP	127#		
T Protein	6.4**		
Albumin/ Globulin	3.2/3.2**		

*mg/dL, **g/dL, # U/L. CSF: Cerebrospinal fluid, Hb: Hemoglobin, , TC: Total count, DC: Differential count, AST: Aspartate transaminase, ALT: Alanine transaminase, ALP: Alkaline phosphatase, P: Polymorphs, L: Lymphocytres, E: Eosinophils, M: Monocytes.

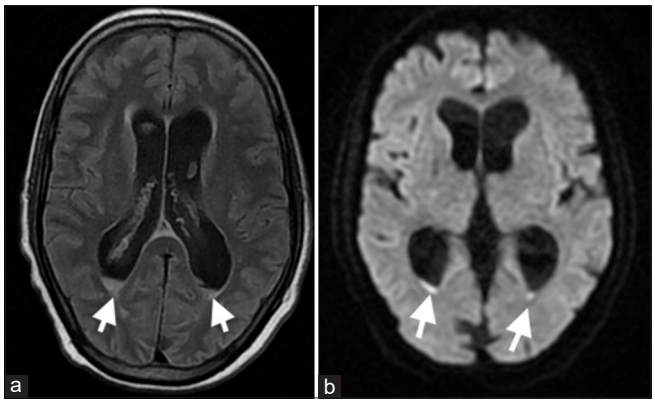

Figure 1: A 66-year-old man with chronic obstructive pulmonary disease who presented with altered mentation. (a) Brain magnetic resonance image T2-weighted fluid-attenuated inversion recovery shows hyperintense dependent signal within the occipital horns of the bilateral lateral ventricles (arrows). (b) Diffusion restriction in the corresponding regions (arrows).

on day 1 showed hyperintense dependent signal in the lateral ventricles (T2-weighted fluid-attenuated inversion recovery), with diffusion restriction suggesting ventriculitis [Figure 1]. Cerebrospinal fluid (CSF) analysis revealed total white cell count 400/mm^3 (Polymorphs 80%, lymphocytes 20%), glucose 33 mg/dL, and protein 253 mg/dL) consistent with bacterial meningitis. CSF culture grew ampicillin-resistant *E. faecium,* sensitive to vancomycin and linezolid. Empirical treatment with IV ceftriaxone and ampicillin at meningitis doses was modified to IV vancomycin. Echocardiography for vegetations, blood and urine cultures were negative. CT kidney, ureters, and bladder showed bilateral minimal perinephric fluid and fat stranding.

On Day 3, he was extubated as his sensorium improved. He continued to be drowsy but arousable to call and responsive to commands. On Day 4, he experienced temperature spikes of 102°F, tachycardia, and tachypnea. Repeat CSF cultures after 72 h grew *E. faecium*. Consequently, IV linezolid was added. Although he was responsive to commands, he was unable to cough out secretions. He was reintubated on Day 8 for airway protection.

Follow-up CSF culture on Day 10 was negative. The CT brain on Day 13 also did not show any new findings. On Day 17, tracheostomy was performed for weaning difficulty. He improved clinically, work of breathing reduced, and became afebrile. He was shifted out of the ICU, and IV vancomycin and linezolid continued. He subsequently developed nosocomial pneumonia in the ward and died on day 16 post-admission.

DISCUSSION

E. faecalis and *E. faecium,* typically gut commensals, have become a rising concern in healthcare due to an increase in invasive infections. *Enterococcus* species account for 0.3–0.4% of bacterial meningitis, with 10% of these caused by *E. faecium*. The mortality rate of Enterococcal meningitis for post-operative versus spontaneous is 12% versus 33%, respectively.[1] In addition, *E. faecium* easily acquires resistance to vancomycin and transfers it to other organisms.

In patients with severe underlying immunosuppression, i.e., spontaneous cases of *E. faecium*, bacteremia is common.[1-3] Hematological patients are at higher risk for vancomycin-resistant enterococci colonization, as they experience long periods of profound neutropenia.

E. faecium meningitis, in this case, may be due to secondary hematogenous seeding of the meninges, originating from urinary or gastrointestinal tract infections. Prior hospital stay for COPD with antibiotic use could have rendered the patient a vulnerable host. Ventriculitis is a complication of meningitis in 30% of adult cases.

Ampicillin-resistant *E. faecium* is treated with vancomycin, with intrathecal administration considered for delayed CSF bacterial clearance. Other treatment measures include IV linezolid, IV quinupristin-dalfopristin, and intrathecal

daptomycin.[5] In this case, we added IV linezolid to IV vancomycin for delayed CSF clearance. Linezolid, with excellent CSF penetration, serves as a valuable adjunct for multidrug-resistant (MDR) isolates.[4,5]

E. faecium has acquired adaptive traits with genes for antibiotic resistance and virulence, which allow them to thrive as a hospital pathogen. While infection control is important, host factors that facilitate gut colonization by organisms like *E. faecium* may also play a significant role, potentially leading to hematogenous dissemination to other body sites.[6] The absence of "colonization resistance" by gut commensals, particularly anaerobic fermenters, that is, gut dysbiosis caused by prior antibiotic exposure and COPD, may drive expansion of resistant strains of *Escherichia coli* and enterococci in the gut.

Molecular profiling methods, such as single-cell sequencing, next-generation sequencing (NGS), and metagenomics NGS, are paving the way for earlier and more accurate detection in severe infections like *E. faecalis* meningitis.[7] MDR enterococci, especially *E. faecium*, are emerging as major hospital pathogens. With rising prevalence, new therapeutic strategies are urgently needed to manage these challenging infections. Advancements in molecular pathology offer promising avenues for early detection, precise diagnosis, and effective management of such infections.

CONCLUSION

E. faecium meningitis is an emerging infectious disease with high mortality. The challenges posed are due to antibiotic resistance and the potential to cause complications in the immunosuppressed. Clinicians should be vigilant of the increasing virulence of *E. faecium* while deciding on the management of *E. faecium* positive culture reports.

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