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Case Report

Peripartum meningoencephalitis complicated by transient stress-induced cardiomyopathy: a case report

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ABSTRACT

Acute meningoencephalitis in late pregnancy is a rare, life-threatening emergency. We report a complex case of peripartum meningoencephalitis associated with severe, transient left ventricular (LV) dysfunction and postpartum delirium. A 30-year-old G2A1 at 37 weeks gestation presented with a one-week history of fever, headache, and thrombocytopenia. Her condition rapidly deteriorated into irritability, neck rigidity, and altered sensorium. MRI brain revealed features of meningoencephalitis with a right hippocampal infarct; CSF analysis showed pleocytosis. Following an emergency cesarean section, the patient developed acute heart failure with an Ejection Fraction (EF) of 20% and elevated Troponin-I (1200.7 ng/ml). She required mechanical ventilation and inotropic support with dobutamine. Management included intensive care and broad-spectrum antimicrobials (Ceftriaxone, Vancomycin, Doxycycline, and Acyclovir). Remarkably, her EF recovered gradually to 45% and 60% within 48 and 72 hours respectively, suggesting stress-induced (Takotsubo) cardiomyopathy secondary to the neurological crisis. Despite a postoperative generalized seizure and transient delirium, she showed significant recovery and was successfully extubated on post operative day 4. Follow-up CSF analysis was negative for tuberculosis and bacterial panels. She was discharged on postoperative day 15 on cardiac and antiepileptic medications. This case highlights the "perfect storm" of physiological stress triggering transient cardiac dysfunction secondary to a primary neurological insult. It underscores the necessity of a multidisciplinary approach in managing peripartum neurological emergencies, where severe cardiac impairment may be fully reversible with prompt stabilization and delivery.

Keywords: Bacterial meningitis, Takotsubo cardiomyopathy, Pregnancy third trimester, Decapitated meningitis

INTRODUCTION

Bacterial meningitis and meningoencephalitis during the peripartum period represent some of the most daunting challenges in modern obstetrics. While the incidence of central nervous system (CNS) infections in pregnancy is low—estimated globally at approximately 0.1 to 0.4 per 100,000—the associated morbidity and mortality for both the mother and the fetus remain disproportionately high.[1,2] In the third trimester, the physiological and immunological landscape of pregnancy creates a unique vulnerability; the relative suppression of cell-mediated immunity, necessary for fetal tolerance, increases maternal susceptibility to specific intracellular and opportunistic pathogens.[3,4]

The diagnostic dilemma and mimicry

The classic clinical presentation of meningitis—the triad of fever, nuchal rigidity, and altered mental status—is notoriously inconsistent in the obstetric population,

appearing in only a fraction of confirmed cases.[5] In the third trimester, identifying these symptoms is further complicated by their significant overlap with pregnancy-specific hypertensive disorders. Severe headaches, vomiting, and seizures are hallmark features of preeclampsia and eclampsia, which are highly prevalent in the Indian clinical setting.[6] This "diagnostic mimicry" often leads to a dangerous clinical bias, where neurological deterioration is initially attributed to toxemia, resulting in critical delays in performing a lumbar puncture and initiating life-saving antimicrobial therapy.[1-6]

Epidemiology and the Indian context

In India, the burden of meningitis in pregnancy is influenced by specific socio-economic factors and variations in community pathogen exposure. A significant challenge in this landscape is the phenomenon of "decapitated meningitis"—where patients receive sub-therapeutic or empirical antibiotic doses at the primary care level before reaching a tertiary center.[7] As seen in recent case reports, delays in referral and the administration of prior antibiotics can mask the early progression of CNS involvement, complicating the identification of the causative organism.[2]

Microbiology and pathogenesis

The microbiology of meningitis in the third trimester deviates from the general adult population. While Streptococcus pneumoniae remains a primary concern, the pregnant state specifically predisposes women to pathogens like Listeria monocytogenes due to its affinity for the placenta.[4] Furthermore, recent literature has highlighted the emergence of rare, food-borne or commensal organisms, such as Lactococcus lactis, which can breach the blood-brain barrier when host defenses are modulated by the hormonal shifts of late pregnancy.[2] Otogenic sources, such as chronic or acute otitis media, also remain a critical pathway for bacterial entry into the CNS.[8]

Maternal and fetal implications

The consequences of a CNS infection in the third trimester are twofold. Beyond the immediate risk of maternal mortality, severe neurological stress can precipitate secondary cardiac crises, such as stress-induced (Takotsubo) cardiomyopathy.[9] For the fetus, systemic maternal infection frequently triggers the inflammatory cascade leading to preterm labor, while vertical transmission poses a high risk of neonatal sepsis and meningitis, while maternal hypoxia and hemodynamic instability can result in severe fetal distress or meconium aspiration syndrome.[1-4]

CASE REPORT

Mrs. X, a 30-year-old woman (G2A1) at 37 weeks and 1 day of gestation, presented with a complex 12-day history of systemic symptoms. The illness began with a moderate-grade, intermittent fever that had finally abated two days prior to her admission. Following the resolution of the fever, she developed generalized myalgia and a maculopapular rash across her body. Her primary complaint on admission, however, was a persistent headache. The headache was gradual in onset, generalized, and dull in character, yet it eventually escalated to a severe intensity. Notably, the pain was significantly exacerbated by light and noise (photophobia and phonophobia), though at the time of initial evaluation, it was not associated with nausea, vomiting, visual disturbances, or seizure activity.

The patient's history was further complicated by a moderate left-sided earache of three days' duration. A community practitioner had previously diagnosed her with an acute perforation of the left tympanic membrane, for which she had completed a three-day course of Faropenem. Upon arrival at the tertiary center, Mrs. X was an unbooked case of low socioeconomic status. She did not have any other comorbidities and had no significant personal or family history. Her obstetric history was otherwise unremarkable, with normal dating, anomaly, and growth scans through the first and second trimesters.

Day 1: admission and initial stability

On the first day of hospitalization, Mrs. X appeared clinically stable with a Glasgow Coma Scale (GCS) score of 15/15. On Physical examination, the patient was afebrile and was confirmed to have the presence of the generalized maculopapular rash and grade 2 pitting pedal edema. Initial blood investigations revealed mild anemia (Hb 9.9 g/dl) and a reduced platelet count (1.27 lakh/ul) with a TLC of 4860/mm^3. An otolaryngology consultation was done, which found a mild congestion of the external acoustic meatus, but no active signs of the previously reported eardrum perforation, with she was started on a regimen of intravenous ceftriaxone and analgesics; A fever workup was done which gave negative results. However, despite this, she began to experience new-onset nausea and a worsening of her cranial pain.

Day 2: acute neurological crisis and intervention

The clinical trajectory changed dramatically on the morning of the second day of admission. At approximately 10:00 am, Mrs. X underwent a rapid neurological decline, manifesting as severe irritability, restlessness, involuntary limb movements, and a transition into a drowsy state where she was no longer able to follow commands. Clinical signs of meningeal irritation, specifically neck rigidity, were present, along with anisocoria (unequal pupil size).

To ensure airway protection, she was emergently intubated and sedated. A stat MRI of the brain indicated findings consistent with meningoencephalitis, though it ruled out dural venous thrombosis. Subsequent cerebrospinal fluid (CSF) analysis via lumbar puncture supported a central

nervous system infection, showing a low glucose level (41 mg/dl), elevated protein (88.4 mg/dl), and a pleocytosis of 30 cells/cumm (55% lymphocytes, 45% neutrophils). HSV PCR and ADA were reported to be negative after 2 days of sending the CSF analysis. A bacterial panel was sent which was reported after 7 days to be sterile.

Table 1: Laboratory trends (day 1 of admission to post operative day 5).

Date	Hb (g/dl)	WBC (x103)	Platelets (x103)	Albumin (g/dl)
On admission	9.9	4.86	127	2.36
POD0	9.5	6.03	130	2.20
POD1	8.8	11.36	138	2.12
POD2	7.7	11.53	85	2.49
POD3	7.2	12.32	88	-
POD5	8.9	8.66	89	-

Given the critical maternal state, the medical team opted for an immediate emergency LSCS. A preoperative 2D Echo was done which was normal with an Ejection fraction (EF) of 60%. The procedure revealed Grade 3 meconium-stained liquor. Mrs. X delivered a 2.7 kg female baby who required NICU admission for respiratory distress, meconium aspiration syndrome, metabolic acidosis and Severe Pulmonary artery Hypertension.

Post-operative management and complications: day 3 to day 5

Following delivery, Mrs. X was moved to the MICU, where she developed features of Congestive Cardiac Failure. On Post-Operative Day (POD) 1, an ECG showed T inversions in V2 - V4 and poor R wave progression, and her Troponin I was significantly elevated at 1200. A 2D echocardiogram revealed global hypokinesia with severe left ventricular systolic dysfunction with an ejection fraction (EF) of only 20%–25% which indicated an acute stress induced Cardiomyopathy.

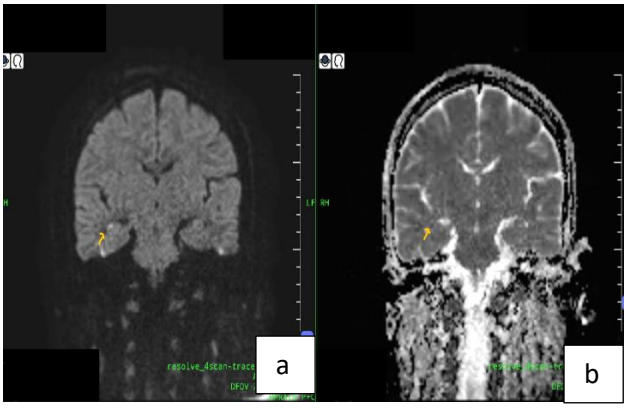

Figure 1 (a and b): MRI images on post operative day 3.

Management included Dobutamine to support cardiac output and furosemide, once her blood pressure stabilized. On POD 2, she received 20% albumin to treat low serum levels (2.12 gm/dl). On POD 3, she experienced a generalized tonic clonic seizure, necessitating a loading dose of Levetiracetam 3 gm IV followed by 1 gm IV three times a day. A follow-up MRI on this day showed leptomeningeal enhancement along the cerebral cortical sulci with acute to subacute nodular infarct in the right hippocampal region, suggestive of infective vasculitis changes. However, her cardiac function began to recover, with her EF improving to 40%–45%. Once her BP improved and stabilized, dobutamine was stopped and she was started on Tab ramipril, Tab bisoprolol, Tab spironolacton and Tab torsemide. By POD 4, her cardiac function normalized completely (EF 60%), and she was successfully extubated, conscious, oriented and obeying commands.

Stabilization and discharge: day 6 to day 15

Mrs. X was stable on room air by POD 5 and received a blood transfusion for low hemoglobin (7.2 g/dl). She was transitioned to oral cardiac medications and moved to the postoperative ward on POD 6. Although she complained of a diffuse headache on POD 7, a repeat lumbar puncture was done and subsequent tests (CBNAAT and ADA) were negative for tuberculosis, leading to the continuation of her antibiotic regimen (Ceftriaxone) for a total of 14 days. In the final stages of her recovery (POD 13–14), she experienced "bad dreams" and was diagnosed with a transient delirium disorder, which was managed with clonazepam. Mrs. X achieved a complete neurological and cardiac recovery and was discharged in stable condition.

DISCUSSION

The clinical course of Mrs. X highlights the diagnostic complexity of central nervous system (CNS) infections in late pregnancy. While the initial presentation was vague, her rapid neurological collapse underscores the aggressive nature of peripartum meningoencephalitis. This case is particularly instructive regarding the interpretation of physical findings after the administration of outpatient antibiotics and the necessity of biochemical differentiation in meningitis.

The paradox of normal ENT findings and otogenic infection

A pivotal point in this case was the discrepancy between the patient's history and the clinical examination. Mrs. X

reported a recent diagnosis of an acute tympanic membrane perforation; however, the ENT evaluation on admission revealed a normal tympanic membrane. In a medical-legal context, it is vital to understand that a normal ear examination does not rule out an otogenic source of meningitis. The most likely explanation is the patient's prior three-day course of Faropenem. This oral carbapenem is highly effective against common middle-ear pathogens. The antibiotic treatment likely initiated "surface healing" of the tympanic membrane and suppressed local inflammation, leading to the normal physical exam findings. However, the bacteria had likely already breached the tegmen tympani or traveled via retrograde thrombophlebitis into the meningeal space prior to the commencement of faropenem. Once the bacteria sequestered within the subarachnoid space, the oral antibiotic—which has limited penetration of the blood-brain barrier—was insufficient to halt the intracranial infection.[8] This resulted in a "masked" or decapitated meningitis, where the primary site of infection appears resolved while the CNS assault continues to progress.[7]

Table 2: Differential diagnosis of meningitis in Mrs. X.

Feature	Bacterial meningitis	Viral meningitis	Tubercular meningitis	Mrs. X's findings
Onset of crisis	Acute (hours to days)	Acute (days)	Subacute (weeks)	Acute (sudden crash on day 2)
CSF glucose	Low (<40 mg/dl)	Normal	Low (<40 mg/dl)	Low (41 mg/dl)
CSF protein	High (>100 mg/dl)	Mildly elevated	Very high	Elevated (88.4 mg/dl)
Predominant cell	Neutrophils	Lymphocytes	Lymphocytes	Mixed (45% neutrophils)
CSF appearance	Turbid/cloudy	Clear	Clear/cobweb	Not specified (likely clear due to partial treatment)
Prior history	Often ear/sinus infection	Often viral prodrome	Weight loss/night sweats	History of earache
Specific tests	Culture/gram stain	PCR for viruses	CBNAAT / ADA	Negative ADA/CBNAAT

Biochemical differentiation and diagnosis

The medical team faced the challenge of differentiating bacterial meningoencephalitis from viral and tubercular causes, given the prevalence of all three in the Indian population.[10]

Bacterial vs. Viral: The definitive evidence for bacterial etiology was the CSF glucose level of 41 mg/dl. In viral meningitis, glucose levels remain normal. This significant hypoglycorrhachia, combined with the acute clinical "crash" on Day 2, strongly pointed toward a bacterial pathogen.[5]

Exclusion of tuberculosis: While TB meningitis is endemic in India, it typically follows a subacute course. The acute onset and negative results for ADA and CBNAAT in the CSF, along with the lack of classic basilar enhancement on MRI, effectively ruled out tuberculosis.[10]

Stress-induced cardiomyopathy (Takotsubo syndrome)

The development of severe left ventricular dysfunction (EF 20–25%) post-delivery was a secondary crisis. The rapid recovery of the ejection fraction to 60% within four days suggests this was not peripartum cardiomyopathy, but rather stress-induced (Takotsubo) cardiomyopathy.[9] This was likely triggered by the "catecholamine storm" resulting from the acute neurological insult and the physiological stress of the emergency LSCS.[11] This "brain-heart axis" interaction requires clinicians to monitor cardiac function closely during any acute neurological deterioration.

CONCLUSION

Mrs. X's survival was due to a high index of clinical suspicion that looked beyond a normal ear examination and a "partial" antibiotic history. By prioritizing the biochemical evidence in the CSF, the team was able to provide the aggressive multidisciplinary care required to rescue the "pregnant brain under siege."

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