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Case Report

Fatal neonatal septicemia with meningitis due to *Escherichia coli* in a full-term baby: an unusual presentation

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ABSTRACT

Neonatal meningitis carries the risk of long-term debilitating squeal with very high mortality. With the use of antibiotics, the mortality due to neonatal meningitis has significantly reduced in developing countries, with India reporting incidence rates of 0.5 per 1000 with mortality ranging from 16-34%. Diagnosis of neonatal meningitis is based on clinical presentation, gestational age, causative agent, and geographical location. Early empiric antibiotic therapy may often obscure the diagnosis of the causative agent. We report a fatal case of neonatal meningitis in a 39-day-old baby. The child was initially treated for hyperbilirubinemia and mild fever. The child presented with a fever for 4 days and episodes of convulsion. The child was put on ampicillin and gentamicin empirically. The CSF was suggestive of pyogenic meningitis. The Gram-stained smear of the CSF sediments revealed Gram-negative bacilli suggestive of *E. coli* (Enteric-like gram-negative bacilli). *E. coli* was isolated from blood as well as CSF and was a phenotypically ESBL (CTX-M like) producer. The antibiotic was changed to meropenem and amikacin. Despite appropriate antibiotic therapy, the patient rapidly deteriorated and died on the third day of admission. Treatment of bacterial meningitis due to *E. coli* in neonates has changed over the years due to the emergence of antibiotic resistance patterns of *E. coli*. Resistance to third-generation cephalosporin has evolved in *E. coli* due to ESBL production. Neonatal meningitis due to *E. coli*, particularly in full-term babies, is reported rarely. We report a case of fatal meningitis due to ESBL-producing *E. coli* in a full-term baby.

Keywords: Neonatal meningitis, *Escherichia coli*, ESBL, Antimicrobial resistance, Late onset sepsis, Full term infant

INTRODUCTION

With the use of antibiotics, the mortality due to neonatal meningitis has significantly reduced in developing countries, with India reporting incidence rates of 0.5 per 1000 with mortality ranging from 16-34%.[1] Well-known risk factors for bacterial meningitis in neonates are preterm birth, maternal colonization due to *Streptococcus agalactiae (*Group B *Streptococcus)*, premature or prolonged rupture of membranes, and very low birth weight (VLBW<1500 gm). The incidence of early-onset meningitis has significantly decreased by screening for GBS at 35-37 gestation weeks and the use of intrapartum antibiotics targeting GBS infection in developed countries. In India, screening for GBS is not mandatory. However, GBS remains the leading cause of meningitis and neonatal sepsis, responsible for more than 40% of all such infections.[2]

The second most common pathogen, *E. coli, accounts for* approximately 30% of all early-onset neonatal meningitis and is documented as the most common cause of early-onset sepsis and meningitis in VLBW and preterm

newborns.[2,3] Despite advances in therapeutic interventions, *E. coli* infection has a rapid progression to cause severe neurological sequelae.

Bacterial meningitis is one of the most important causes of meningitis in infants. If the patient survives, they will develop severe disabilities. Bacterial meningitis can induce complications between 20% and 60%. Research has shown that meningitis accounts for 3% of all deaths of infants under five years of age.[4] Mortality rate in infants is highest in the case of bacterial meningitis. One of the predisposing factors is immaturity of their immune system, which does not reach its peak until two months. The mortality rate is 10% in developed countries and 40-58% in developing countries.[4] Up to 50% of surviving infants suffer from neurological complications, such as seizures, cognitive defects, movement problems, and hearing and vision disorders.[5] All these data show the importance of meningitis in infants and make it one of the most fatal diseases of infants.

Bacterial meningitis in <3 years of age has the highest incidence rate among all age groups. It is a devastating condition that leads to increased morbidity and mortality in newborns, particularly in the early neonatal period.

Meningitis in neonates often presents with nonspecific, varied signs in different age groups. When accompanied by septicemia, the clinical course can be fulminant, requiring immediate intervention. Certain strains of *E. coli*, particularly those possessing the K1 capsular antigen, are known to have increased virulence and enhanced ability to cross the blood-brain barrier, causing meningitis.[6]

This case report describes a full-term neonate diagnosed with septicemia and meningitis due to ESBL-producing *E. coli*. This case underscores the importance of early detection of bacterimia and aggressive management to prevent further complications like meningitis.

CASE REPORT

A 36-day-old child, weighing 2.8 kg, was brought to our hospital with a complaint of fever and convulsion. The patient was admitted to the NICU and was administered ampicillin and gentamicin as empirical therapy, considering it as late-onset meningitis due to *Streptococcus agalactae*.

The child was born at full term via normal vaginal delivery with a birth weight of 2.9 kg, had no congenital abnormalities or infections at birth, and was exclusively on breast milk. The child was apparently healthy for the first few weeks of life. On the 22nd day of life, she developed Jaundice and was admitted to the NICU for 7 days, where she received phototherapy. The patient was apparently healthy for 4-5 days. Later, she developed a fever and was treated with antipyretics at home. On the sixth day, she developed convulsions, and the patient was taken to Parul Sevashram Hospital. She was detected to have jaundice (Serum total bilirubin 14.8 mg/dl, conjugated bilirubin 7.8 mg/dl, unconjugated bilirubin 2.2 mg/dl). She had tachycardia (Heart rate: 130-140/min), tachypnea (Respiratory rate 48/min), SpO_2 98% and temperature was 99.8°F at the time of admission. Laboratory investigations included CBC, CRP, HHH, Urine routine examination, blood culture and sensitivity, CSF routine examination with matching blood sugar. CSF culture and sensitivity. CSF was pale yellow in coloured and the appearance was hazy (Figure 1). CSF routine micro shows: Total count of CSF>10000/cm^3 with neutrophilia, glucose CSF<20 mg/dl, protein CSF>240 mg/ml, suggestive of pyogenic meningitis. CBC revealed normal WBC counts with thrombocytopenia (platelet count/c/mm CRP level was 152 mg/l.

The Gram-stained smear of the CSF sediments revealed Gram negative bacilli morphologically suggestive of *E. coli* (Enteric like), some of them appeared fragmented (Figure 2). The antibiotic was changed to meropenem and amikacin after the CSF gram stain report was received. The brain USG showed mildly dilated bilateral lateral ventricle (diameter of frontal horn of right lateral ventricle: 12 mm and frontal horn of left lateral ventricle: 11 mm), suggestive of hydrocephalus. The next morning, the baby developed bradycardia (Heart rate <50/min), desaturation (SpO2 30-40%), and hypotension. She was intubated and kept on ventilator support with an inotropic support. The next day, CSF culture grew *E. coli* (Figure 3 and 4) and AST suggestive of strain that was ESBL producer (CTX-M-like), identified by VITEK-2.

E. coli, *in vitro* showed susceptibility to ongoing antibiotics (Figure 5). The blood culture bottle also flagged positive and grew *E. coli*. AST of both strains was similar.

The next day of admission X-ray chest showed patchy homogeneous opacities with air bronchograms noted involving bilateral lung fields, with relative sparing of bilateral lower lung zones (right more than left), suggesting the possibility of infective etiology. Later, the patient received a platelet transfusion in view of severe thrombocytopenia, and developed refractory hypotension with increasing inotropic requirements. On the following day, the patient succumbed to the illness.

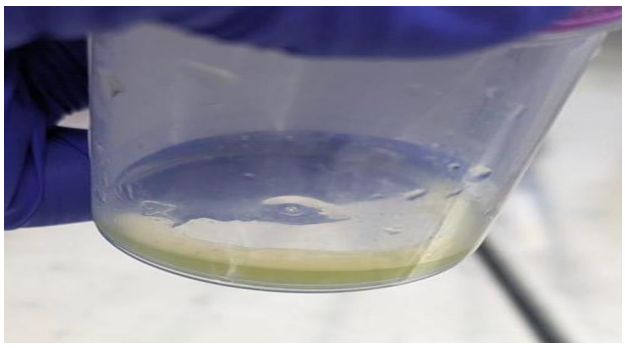

Figure 1: CSF in sterile container (Hazy and yellow).

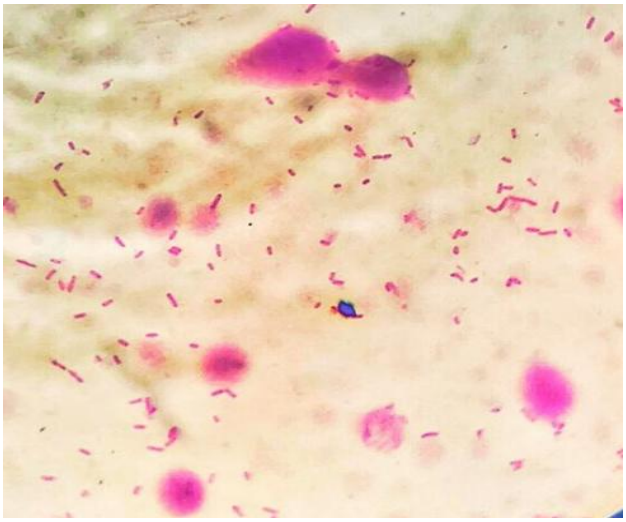

Figure 2: Gram stain of CSF (Gram negative bacilli-100× lens).

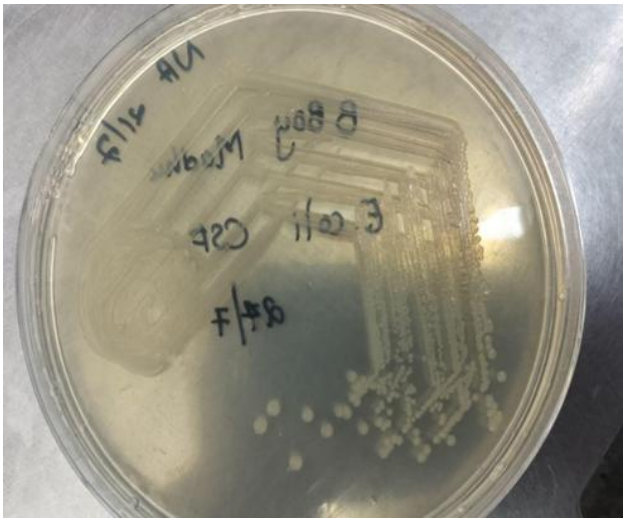

Figure 3: Growth of *E. coli* on nutrient agar.

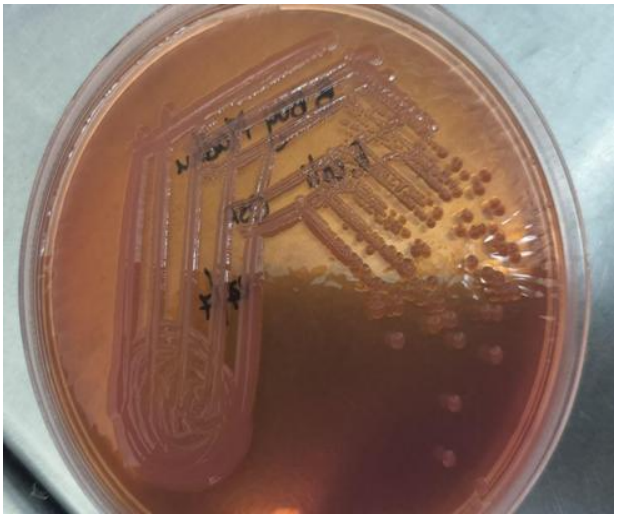

Figure 4: Growth of *E. coli* on MacConkey agar.

Organism Quantity:
Selected Oreanism : Escherichia coli
Source: CSF **Collected:**

Comments:	THE INTERPRETED MIC FOR CEFEPIMEIS SDD(Susceptible dose dependent) Please Check for new intermediate interpretive catagory introduced by CLSI 2020 that states the names of drugs which has the potential to concentrate at any anatomical site, urine or epithelial lining

Identification Information	**Analysis Time:**	4.32 hours	**Status:** Final
Selected Organism	99% Probability **Bionumber:**	**Escherichia coli** 0405610554526611	
ID Analysis Messages			

Susceptibility Information	**Analysis Time:** 7.97 hours	**Status:** Final

Antimicrobial	MIC	Interpretation	Antimicrobial	MIC	Interpretation
Piperacillin/Tazobactam	<= 4	S	Gentamicin	>= 16	R
Ceftazidime	8	I	Ciprofloxacin	>= 4	R
Cefoperazone/Sulbactam	<= 8	S	Levofloxacin	>= 8	R
Cefepime	4	SDD	Minocycline	2	S
Aztreonam	4	S	Tigecycline	<= 0.5	S
Imipenem	<= 0.25	S	Fosfomycin	<= 16	S
Meropenem	<= 0.25	S	Colistin	<= 0.5	I
Amikacin	4	S	Trimethoprim/ Sulfamethoxazole	>= 320	R

PARUL INST. OF MED. SCI
AES Detail Report

bioMerieux Customer: Printed by: LabSuper
System #:
Accession Number: 13507109863-1 Date Tested: Jul 18, 2025 21:29 IST

Selected Organism: Escherichia coli (99%) ID Confidence: Excellent identification
AES Confidence: Consistent
Card Type: AST-N406 Bar Code: 1563100504737042 Testing Instrument: ??? (???)

Phenotypes

Antibiotic Family	Detected Phenotypes
BETA-LACTAMS	EXTENDED SPECTRUM BETA-LACTAMASE
AMINOGLYCOSIDES	RESISTANT GEN (AAC(3)-I),RESISTANT GEN TOB NET (AAC(3)-IV),RESISTANT GEN TOB (ANT(2'')),RESISTANT GEN TOB NET (AAC(3)-II)
QUINOLONES	RESISTANT
TETRACYCLINES	WILD
FOSFOMYCIN	WILD
POLYPEPTIDES	WILD
TRIMETHOPRIM/SULFONAMIDES	RESISTANT

Figure 5: Identification and antimicrobial susceptibility test report given by VITEK 2.

DISCUSSION

Bloodstream infections and meningitis are the most significant risk factors after a normal vaginal delivery. Group B *Streptococcus*, followed by *E. coli*, are the commonest pathogens responsible for neonatal BSI and meningitis as they are part of the normal genital flora of females. The percentage of females harboring these two pathogens varies depending on geographical location. In developed countries, almost ¾ of females carry GBS in the genital tract. This is the reason for mandatory screening for GBS at 35-37 gestational weeks during antenatal check-up. The use of intra-partum antibiotic therapy significantly reduces the consequences of neonatal sepsis. Because of the extensive use of antibiotics, the prevalence may differ in India. The data regarding the GBS prevalence in India is scarce. Neonatal bacterial meningitis results due to hematogenous spread of this pathogen to the central nervous system, where it cross the blood brain barrier, leads to cerebral edema and raised intracranial pressure.

Neonatal meningitis can be early onset or late onset. Early onset appears within 72 hours of life, while late onset appears 3to 28 days of life.[12] GBS is the most common pathogen in early onset neonatal meningitis, and *E. coli* is

the most common pathogen in late- onset meningitis. Both of them show an increasing trend and a high risk of neurological complications and mortality.[11]

Due to that, sepsis is often complicated by late diagnosis and antibiotic resistance. In a study of developed countries, the rate of early onset sepsis dropped 2.26 from 3.25 per 1000 live births.[7] In the U.S GBS GBS-based early onset sepsis declined from 1.7 to 0.34 per 1000 live birth.[8] Newborns born to mothers who had antenatal urinary tract infection were at 3.55 times higher risk of developing neonatal sepsis. Moreover, neonates born to mothers having intra-partum fever were 3.63 times more likely to develop neonatal sepsis.[9]

The incidence rate of bacterial meningitis is around 1-2 % in full-term and 4-6% in preterm or VLBW infants.[10] According to past studies, *E. coli* meningitis has a significantly higher risk of neurological complications like subdural effusion, hydrocephalus, and brain abscess. As reported in other studies, neonatal *E. coli* meningitis may cause learning and memory impairments in adulthood, which may be related to the high incidence of *E. coli* neurological complications. As serious neurological complications, hydrocephalus and brain abscess are associated with significant long-term morbidity and mortality.[11] Similar to the findings of this study, our case report is of late-onset meningitis in a term baby due to *E. coli* complicated with the development of hydrocephalus.

A high proportion of ESBL-producing isolates in *E. coli* cases increases the serious neurological complications. The high proportion of ESBL producing *E. coli* is due to overuse and misuse of antibiotics across the world.[11] Same like this, in our case, isolated *E. coli* is an ESBL producer which is resistant to gentamicin, levofloxacin, ciprofloxacin, and trimethoprim-sulfamethoxazole.

As appreciated in this case, meningitis in these populations has a high likelihood of resulting in some amount of neuro-developmental injuries with a range of severity. In a case study, the patient experienced EOS and LOS in rapid succession, suggesting that both instances resulted from infection with the same ampicillin-resistant *E. coli*. The patient was treated with gentamicin alone and may have been predisposed to his LOS either because of a partially treated *E. coli* CNS infection or as a result of a new infection from translocation of colonizing *E. coli* in his leaky, premature gastrointestinal tract.[10] The high rates of *E. coli* resistance increasing globally raise concerns about the safety of continued empiric use of ampicillin and gentamicin in EOS. Similar to this study, our patient also developed jaundice on 22nd day of life. The patient was sent home after receiving phototherapy only. This leads to developing late-onset sepsis and meningitis. At this time, many argue that the change is not warranted and that individual risk assessment and local antibiotic resistance rates are key in the decision to broaden antibiotics in EOS and the ESBL producer.[10]

In cases of neonatal bacterial meningitis, early etiologic diagnosis and prompt targeted treatment significantly reduce the mortality and risk of long-term neurologic disabilities.[13]

CONCLUSION

Meningitis due to *E. coli* is most commonly observed in very low birth weight, preterm neonates. The targeted antibiotic treatment can only be made after proper investigations. CSF culture mostly is not yielding as a result of early empiric antibiotic therapy. Sophisticated tests like Biofire and PCR of the CSF are not available in many low resource facilities. Empiric therapy for neonatal meningitis generally targets group B *Streptococci* or Listeria monocytogenes. Possibility of late onset neonatal meningitis due to ESBL-producing *E. coli* needs to be considered as differential diagnosis. Early broad spectrum antibiotic cover in such cases may be of great help.

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