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Case Report

Neonatal *Listeria innocua* sepsis

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ABSTRACT

Neonatal listeriosis is a potentially life-threatening infection, usually caused by *Listeria monocytogenes*. It has a high case fatality rate and can cause severe neurological sequelae among survivors. Early-onset listeriosis is caused by vertical transmission through transplacental route, inhalation of infected amniotic fluid or through ascending infection from the vaginal colonization. We report fatal neonatal listeriosis in a 5 day old female infant caused by *L. innocua*. *L. innocua* is considered as non-pathogen and only few cases were reported in an immunocompromised individual.

Keywords: Listeria innocua, Fatal, Neonatal

INTRODUCTION

Neonatal listeriosis usually caused by *L. monocytogenes* is a serious life-threatening infection having high mortality and morbidity.[1] Listerial species other than *L. monocytogenes* are generally considered non-pathogenic and few cases causing fatal sepsis to have been reported in immunocompromised individuals. We report a fatal early-onset *L. innocua* sepsis in a neonate. To the best of our knowledge, this is the first case of neonatal *L. innocua* sepsis.

CASE REPORT

A 5 day old male baby was admitted with a refusal of feed and poor activity for 2 days. There was a history of prolonged rupture of the membrane for more than 24 hours. He was born by emergency cesarean section with a birth weight of 2.56 kg with the normal transition. He was born of non-consanguineous marriage to gravida 3 abortion 1 woman. Postnatally he was noticed to have a low-grade fever on day 2 of life, treated as dehydration fever and discharged on the next day. He was admitted on day 5 of life with 10% weight loss, depressed sensorium and poor perfusion. Saturation in room air was 75%. The abdomen was grossly distended with significant, brown-colored gastric aspirate He was intubated immediately and fluid boluses were given. He responded only to painful stimuli, moro s reflex was absent. Investigation showed severe thrombocytopenia (Platelet count-14,000/cu mm) and positive C-reactive protein (95 mg/L). Blood ammonia was elevated (170 µg/dl). Blood gas showed pH of 7.426, PCO2 of 49 mmHg, bicarbonate of 31.6 mmol/L.

Shock improved with inotropes. Liver function test showed low albumin, elevated enzymes (AST more than ALT) and prolongation of both prothrombin time (PT) and activated thromboplastin time (APTT). He received 2 platelet transfusions, a plasma transfusion and a packed red blood cell (PRBC) transfusion.

Neurological examination showed depressed sensorium, hypotonia and presence of light reflex at admission. Blood ammonia was elevated to 471 µg/dl on day 2 of admission. He was treated for hyperammonemia with oral arginine and oral sodium benzoate. He had features of raised intracranial pressure. Neurosonogram showed

dilated ventricles with intraventricular strands suggestive of ventriculitis (Figure 1). He received a single dose of mannitol and oral acetazolamide as antiedema measures. He developed an unequal pupil and later pupil became dilated with absent light reflex.

Sensorium worsened on day 2 of admission despite treatment for raised intracranial pressure and hyperammonemia. Serial blood ammonia level and CRP showed a decreasing trend and platelet count showed improvement whereas sensorium deteriorated. Blood glucose was 117 mg/dl, urine ketone was negative. Lactate was 15.16 mg/dl. Tandem mass spectroscopy for 52 metabolic conditions was negative. He received intravenous meropenem, amikacin and fluconazole at admission which was changed to intravenous ampicillin and gentamicin on day 3 of admission based on the blood culture report. He was deeply comatose, hence his parents consented to withdrawal of life support. Blood culture grew *L. innocua*.

Isolation and identification

The specimen was inoculated on blood agar plate and Macconkey agar plate aseptically then incubated at 37ºC for 48 hours. The obtained colonies were sub-cultured and gram staining was done after the incubation period. Identification was done using PMIC-84 panel of BD Phoenix M50 according to the manufacturer's instructions. The results were interpreted using epicenter data management software (BD diagnostic systems) after 12 hours of incubation.

We isolated non-hemolytic, creamy white color colonies on blood plate. Gram positive rod shaped, non-spore forming bacteria were seen under the oil immersion microscope. *L. innocua* was identified using PMIC-84 panel of BD Phoenix M50 after 12 hours.

L. innocua was sensitive to amikacin, gentamicin, ampicillin, azithromycin, ciprofloxacin, linezolid, vancomycin, resistant to cefazolin and cefipime.

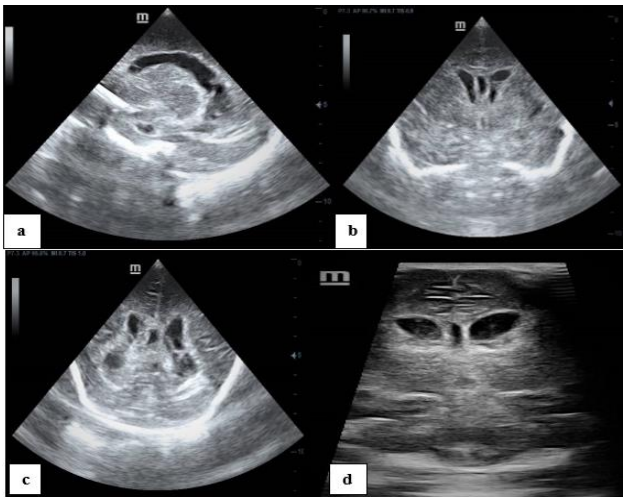

Figure 1: Ultrasound cranium (a) parasagittal view showing irregular ventricular margin and intraventricular strands; (b) coronal view at the level of 3rd ventricle; (c) coronal view posterior view; (d) on day 3 of admission, coronal view showing dilated ventricles.

Table 1: Laboratory parameters.

	D1	D2	D3	D4	D5
Hb (g/dl)	10				
PCV	26.9				
White blood cell (cells/cu mm)	15500				
Platelet (cells×10^5/cu mm)	0.14	0.13	0.11		1.23
Blood urea (mg/dl)	41				
Serum creatinine	0.4				
C-reactive protein (mg/L)	95				27
Calcium (mg/dl)	8.5				
Total serum bilirubin (mg/dl)	8.5				
Direct bilirubin (mg/dl)	1				
Total protein (g/dl)	6.9				
Albumin (g/dl)	2.5				
SGOT (U/l)	132				
SGPT (U/l)	45				40
Alkaline phosphatase (IU/l)	256				
Activated partial thromboplastin time	100/37.9	75/37.9			
Prothrombin time	20/14.6	17/14.6			
Sodium (meq/l)	138				
Potassium (meq/l)	3.4				

Continued.

	D1	D2	D3	D4	D5
Chloride (meq/l)	97				
Bicarbonate (meq/l)	23				
Ammonia (µg/dl)	176	471	300		220
Ketone		Negative			
Lactate (mg/dl)	15.16				

DISCUSSION

Listeriosis is serious and sometimes lethal in immune-compromised individuals most commonly transmitted by contaminated food. Among listeria species, *L. monocytogenes* is considered to be a human pathogen, commonly affecting pregnant women and fetuses and older individuals.[2] Listeriosis is 18 times more common in pregnant women than in the general population.[3]

Neonatal listeriosis is usually caused by *L. monocytogenes*. *L. innocua* causing early-onset neonatal sepsis has not been reported till now. *L. innocua* is a facultatively anaerobic, motile gram-positive rods found in soil and food.[4] *L. innocua* is considered non-pathogenic, hence the name innocua (innocuous). It is similar to *L. monocytogenes* except it is non-hemolytic.[5]

Perrin et al in 2003 was the first to report fatal bacteremia caused by *L. innocua* in a 62 year old otherwise healthy woman.[6] Favaro et al in 2014 reported *L. innocua* meningitis in an immune-compromised adult with an unfavorable outcome.[7]

The mode of transmission in neonatal listeriosis includes transplacental, inhalation of infected amniotic fluid or ascending infection from vaginal colonization.[8] In our case, the mother was asymptomatic with a history of consumption of meat from a restaurant, 5 days before delivery. The risk factor for early-onset sepsis was the prolonged rupture of the membrane for more than 24 hours.

L. innocua sepsis causes rapid progression and irreversible damage to the brain. Patocka et al demonstrated localized encephalitis in suckling mouse after intracerebral injection of Welshimer strain of *L. innocua*.[9] Cerebral listeriosis due to *L. innocua* was reported in a bull.[10] We can speculate that *L. innocua* is a neurotrophic organism based on the animal case reports and from our case.

CONCLUSION

This case concludes that neonatal listeriosis can also be caused by *L. innocua*, multiorgan dysfunction with irreversible damage to the central nervous system. We also concluded that delay in the diagnosis and treatment of this pathogen is fatal.

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Ethical approval: Not required

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