Neonatal septicemia due to Listeria monocytogenes

Sahu M, Parveen M, Harish BN, Parija SC

ABSTRACT

Listeriosis caused by Listeria monocytogenes is the third most important cause of neonatal septicemia. Regretably, reports of such cases from India is scarce. In Indian setup, the common modality of treatment for neonatal septicemia is a combination of gentamicin with a third generation cephalosporin, which is unfortunately, ineffective against listeriosis. We report a case of neonatal septicemia in a term baby due to L. monocytogenes who was successfully treated with parenteral ampicillin and gentamicin, emphasizing the inclusion of ampicillin to the therapeutic regimen.

Keywords: Listeria monocytogenes, neonatal septicemia, blood culture, PCR

INTRODUCTION

Listeria monocytogenes, a Gram-positive intracellular rod, is a relatively uncommon cause of self-limiting febrile gastrointestinal illness in healthy adults.[1] Listeriosis is a food-borne disease often associated with ready-to-eat foods.[2] It usually causes mild febrile gastrointestinal illness in immunocompetent persons. However, septicemia, encephalitis and meningitis are more common in persons with deficient cell mediated immunity. Human listeriosis has a special predilection for pregnant women, neonates, immunocompromised and elderly persons.[1] It is one of the three important causes of neonatal septicemia and meningitis only next to Escherichia coli and Streptococcus agalactiae.[2] There are very few reports of perinatal listeriosis from India.[3,4] We report a case of neonatal septicemia in a term newborn baby due to L. monocytogenes emphasizing novel ways of diagnosis and treatment.

CASE REPORT

A full term newborn baby born to a primigravida by normal vaginal delivery was admitted to the neonatal intensive care unit with complaints of respiratory distress. Mother had fever with history of prolonged rupture of membrane and foul smelling meconium stained liquor. On examination features of asphyxia were obvious with a respiratory rate of 60/min and grunting. The heart rate was 130/min. The baby was provisionally diagnosed as a case hypoxic ischemic encephalopathy and started on steroids. Laboratory examination showed a total leukocyte count of 13600 cells/mm^3 with differential neutrophil count 70%, lymphocytes 25% and monocytes 5%. Cerebrospinal fluid culture was sterile. Peripheral venous blood was collected, inoculated into biphasic brain heart infusion medium, incubated at 37^{0}C for 48 hours and initial report was negative. On the 7th day, a final subculture was done on 5% sheep blood agar (SBA) and Dominguez-Rodriguez isolation agar (DRI), (Himedia, Mumbai) and incubated at 37^{0}C for 24-48 hours. On 5% SBA, beta hemolytic colonies were seen and on DRI agar greenish yellow glistening and pointed colonies surrounded by a diffuse blackening due to aesculin hydrolysis was observed. Morphologically they were verified as gram positive rods by staining. The isolate was checked for temperature dependent motility, it was motile at 25°C and non motile at 37°C by hanging drop. Other characteristics of the isolate were: catalase positive, oxidase negative, glucose fermented without gas, D-mannitol and D-xylose not fermented, Methyl red and Voges-Proskauer positive, hippurate hydrolysis positive and Cristie, Atkins, Munch-Petersen test positive with Staphylococcus aureus but negative with Rhodococcus equi. *L. monocytogenes* MTCC 1143 was grown overnight on SBA. The genomic DNA from L. monocytogenes MTCC 1143 and the test isolate were isolated using commercial Kit (MO BIO

Laboratories, Ltd.) and 5µl lysate was used as template. PCR was performed for the detection of virulence genes namely, hemolysin, phosphatidylinositol phospholipase C, and actin.[5] The amplified products were analyzed by 1.5% agarose gel with 0.5µg/ml ethidium bromide and visualized under UV (Figure 1).

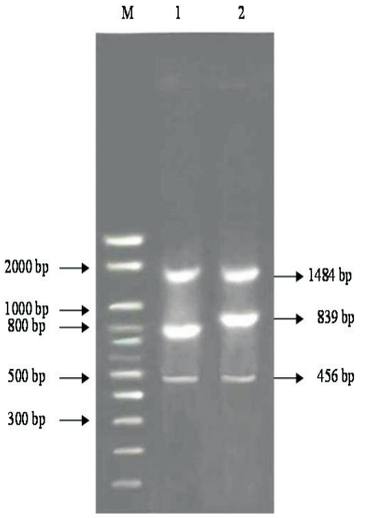

Figure 1: PCR of virulence associated genes of Listeria monocytogenes. Lane M: DNA ladder (100 bp to 3000 bp), Lane 1: Amplified products of the standard L. monocytogenes (MTCC1143) 1484 bp- plcA gene, 839 bp- actA gene, 456 bp – hlyA gene, Lane 2: Listeria monocytogenes isolate from the neonate.

Antimicrobial susceptibility testing by Kirby Bauer method showed that the isolate was sensitive to ampicillin, penicillin, erythromycin, and gentamicin. The baby was started on treatment with parenteral ampicillin and gentamicin and was discharged from hospital after fourteen days with uneventful recovery. Culture from high vaginal swab of mother was negative for *L. monocytogenes.*

DISCUSSION

L. monocytogenes principally causes intra-uterine infections, meningitis and septicemia. Neonatal listeriosis is divided into early (< 2days old), intermediate (3-5 days old) and late onset (>5 days old) disease.[6] The incidence of listeriosis among pregnant women is 20-fold higher than that among the general population.[7] The outcome of maternal listeriosis is usually benign; common presentations being mild flu-like symptoms. The outcome is variable for neonates. Listeria may infect the fetus through the infected birth canal or transplacentally. Abortion or still births are common depending upon the gestational period at the time of acquiring infection.[6] The occurrence of pathogenic *L. monocytogenes* in cases of spontaneous abortions is 3.3%; plcA gene expression was an important virulence determinant.[5] Early neonatal infection is mostly a septicemic illness, contracted in utero. In contrast, late onset infections present as meningitis and probably are due to cross infections in hospitals.[8]

Our case represents an early onset disease with septicemia where the neonate presented with respiratory distress and had history of meconium stained liquor. The incidence of neonatal listeriosis in India is 2.2% in such babies.[3] Though neonatal listeriosis is mostly seen in preterm babies, our case was a term baby. There are a few reports from India as well as other parts of world wherein term neonates presented with symptoms of early neonatal listeriosis. The paucity of reporting from India could possibly be due to a low carriage rate or may be a lack of awareness and non inclusion of selective media for Listeria in plating of blood cultures. Early diagnosis and treatment of listeriosis in high-risk patients is of paramount importance, because the outcome of untreated infection can be devastating with a very high mortality rate; especially among newborns, where it is approximately 25%–50%.[7]

Traditionally, neonatal septicemia is treated with a combination of aminoglycosides like gentamicin and third generation cephalosporins like ceftriaxone. It is pertinent here to point out that such a regime may not be effective in cases of listeriosis because *L. monocytogenes* is not sensitive to cephalosporins.[7] Hence every attempt should be made in all cases of neonatal sepsis to rule out Listerial etiology and ampicillin ought to be included in the regimen.

Pregnant women with listeriosis may have only

mild symptoms of the infection, but for those with fever or signs of sepsis, blood and urine samples should be obtained for culture and sensitivity. High vaginal swab culture is less satisfactory.[9] In pregnant women with suspected listeriosis empiric therapy, including ampicillin should be started for coverage of Listeria infection. Though the incidence may be low it's worth to look out for the possibility of Listeriosis.

AUTHOR NOTE

Manisa Sahu, Assistant Professor, Contact no. 08895436149, E-mail: nisit1978@gmail.com, **(Corresponding author)** Department of Microbiology, Hi-Tech Medical College and Hospital, Bhubaneswar, Odisha.

M Parveen, Research Scholar
B N Harish, Professor
S C Parija, Professor and Head
Department of Microbiology, JIPMER, Puducherry

REFRENCES

1. McLauchlin J, Jones D. Erysipelothrix and Listeria. In: Balows A. Duerden BI, editors. Topley and Wilson's Systematic Bacteriology. 9th ed. London: Arnold publishers; 1998. pp. 683-708.
2. Schuchat A, Broome C V. Infections Caused by *Listeria monocytogenes.* In: Kasper DL, Fauci AS, Longo DL, Brauwald E, Hauser SL, Jameson JL, editors. Harrison's principles of Internal Medicine.16th ed. New York: McGraw-Hill press; 2005. pp. 837-40.
3. Bhujwala RA, Hingorani V. Perinatal listeriosis: a bacteriological and serological study. *Indian J Med Res.* 1975 Oct;63(10):1503-8.
4. Srivastava S, Sen M R, Kumar A, Anupurba S. Neonatal Listeriosis. *Indian J paed.* 2005; 72:1058-9.
5. Kaur S, Malik SVS, Vaidya VM, Barbuddhe SB. *Listeria monocytogenes* in spontaneous abortions in humans and its detection by multiplex PCR. *J App Microbiol.* 2007; 103:1889-96.
6. McLauchlin J, Riegel P. Coryneform bacteria, Listeria, Erysipelothrix. In: Greenwood D, Slack RCB, Peutherer JF, editors. Medical Microbiology. 16th ed. New Delhi: Elsevier press; 2006. pp.189-99.
7. Bortolussi R. Listeriosis: a primer. CMAJ [serial on the internet]. 2008 Oct [cited 2008 Sept];179(8):[about 3p.] Available from www.cmaj.ca.
8. Nelson KE, Warren D, Tomasi AM, Raju TN, Vidyasagar D. Transmission of neonatal listeriosis in a delivery room. *Am J Dis Child.* 1985; 139:903-5.
9. Bhujwala RA, Hingorani V, Chandra RK. Genital listeriosis in Delhi (India): a pilot study. *Indian J Med Res.* 1973; 61:1284-88.