Stenotrophomonas maltophilia Causing Early Onset Neonatal Sepsis

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Stenotrophomonas maltophilia, a multi drug resistant non fermenting Gram negative bacillus is an increasingly common nosocomial pathogen, especially in intensive care units. Comparatively few cases of infection have been reported in neonatal population. We present two cases of early onset neonatal sepsis due to *S.maltophilia* and a brief review of documented isolation in neonates.

Key words: *Neonate, Sepsis, Stenotrophomonas maltophilia.*

Stenotrophomonas maltophilia (formerly *Pseudomonas maltophilia/Xanthomonas maltophilia*), a multi drug resistant Gram negative bacillus, is being increasingly implicated as a nosocomial pathogen, associated with significant case fatality [1]. Though a well recognized opportunistic pathogen in severely debilitated patients, comparatively few cases have been reported in neonates. We herein present two cases of early onset neonatal sepsis due to *S.maltophilia*. To the best of our knowledge, this may be the first report of neonatal bloodstream infection from Asian subcontinent and the first documented case of early onset neonatal sepsis.

CASE REPORT

***Case* 1**. A single, preterm (32 weeks) female baby, weighing 1810 grams was delivered vaginally to a 19 year old primipara following spontaneous onset of labour at a district hospital. There was no history of maternal fever or any other antenatal complication. Rupture of membranes occurred about 6 hours before delivery. At delivery, the baby had a delayed cry and poor muscle tone, with an Apgar score of 6 at 1 minute. He was provided with warmth and airway was cleared. After giving stimulation and supplemental oxygen, the baby was admitted for observational care. At 5 minutes, Apgar score was 7. The baby did not feed well and was lethargic. Test for C Reactive protein by latex agglutination was positive, band cells were seen on peripheral smear, total leucocyte count was 4800/cu mm and platelet count was 230,000/cu mm.Blood was drawn for culture. IV fluids and antibiotics (cefotaxime and amikacin) were started. However, the baby deteriorated rapidly and died on Day 2.

***Case* 2**. A single, term (38 weeks) female baby weighing 3180 grams was delivered to a 22 year old primipara by cesarean section at a tertiary care teaching hospital, indication being foetal distress. There was no history of maternal fever or any other illness. Liquor was meconium stained and the baby presented with severe perinatal asphyxia. She was immediately transferred to the NICU and ventilated. IV fluids were started and blood for culture was drawn. Sepsis screen showed positive CRP, leucopenia (2600/cu mm) and band cells >2% on peripheral smear. Perfusion was poor; baby was lethargic and went into shock. Besides management for shock, IV antibiotics (piperacillin-tazobactam and

amikacin) were started. The baby died within 48 hours.

Blood culture was done by Bactec 9050 (BD Diagnostic Systems, Sparks, MD USA). Both cultures turned positive within 8 hours. Gram stain showed presence of Gram negative bacilli. Subculture on Mac Conkey Agar and 5% Sheep Blood agar (Biomerieux, Marcy l'Etoile, France) showed growth of non lactose fermenting colonies that were catalase positive and oxidase negative. Identification was confirmed by mini API (analytical profile index-Biomerieux, La balme les Grottes, France) using ID 32 GN strip. The ID percentage was 99.9% in both cases and typicity index was 0.77 and 0.89, respectively. In both cases, maternal serology was negative for human immunodeficiency virus, hepatitis B and syphilis. Antibiotic sensitivity test was performed by Kirby Bauer disc diffusion method. Isolate 1 was sensitive to cotrimoxazole alone and isolate 2 to ciprofloxacin and cotrimoxazole. Both isolates were resistant to aminoglycosides, extended spectrum penicillins, third generation cephalosporins, carbapenems and monobactams.

DISCUSSION

The first documented case of neonatal infection due to *S.maltophilia* was in neonatal meningitis and conjunctivitis in 1984 [2]. Subsequently, it has been reported in superficial and deep infections both as a colonizer and pathogen. A surveillance in our own unit during 2006-07 showed presence of *S.maltophilia* in gastric aspirate of newborns, collected within 4 hours of birth [3]. Though, as yet, not so common in Neonatal Intensive care Units (NICU), the particular danger is because of high degree of resistance to most commonly used antibiotics including aminoglycosides and cephalosporins,as well as intrinsic resistance to carbapenems. It is usually susceptible to cotrimoxazole, but emerging resistance to this drug is being documented [3]. Fluoroquinolones have been considered a possible therapeutic option. Two cases of neonatal meningitis were treated successfully with ciprofloxacin alone or in combi-nation with cotrimoxazole [4,5]. SENTRY surveillance [6], however, reports that ciprofloxacin resistant mutants can be easily selected *in vitro*.

In the present report, both cases were early in onset, presenting with features of sepsis on the first day of life. There was no history of maternal antibiotic administration. Maternal swab, blood culture, and environmental sampling could not be done. All documented deep seated infections in neonates till now were late in onset and usually involved preterm neonates [4,7-9]. The cases reported have been both from developed and developing countries with the environmental source usually not traced [7-9]. *S.maltophilia* infection is usually associated with prolonged hospital stay, long duration of broad spectrum antibiotic therapy, or ventilation and presence of central vascular catheter. None of these risk factors were present in the two cases. In the first case, delivery occurred in an overcrowded district hospital, and there may have been lapses in asepsis. The mother also had a history of PROM for about 6 hours. It is possible that she may have been colonized and passed on the infection to the premature baby. The second baby however was of term gestation and a good weight. The primary cause of mortality was considered to be perinatal asphyxia, with sepsis being a contributory factor. However, knowing the propensity of *S.maltophilia* infection for having high case fatality, it cannot be ruled out as the primary cause of death. Asphyxia *per se*, is known to predispose to infection, both due to compromised immunity and because of the interventions required.

This communication highlights that *S. maltophilia*, a recognized nosocomial pathogen, is emerging as a cause of early onset neonatal sepsis. This may be due to colonization of antenatal women in the hospital. *S.maltophilia* is known to adhere to plastic surfaces and be involved in formation of biofilms [10], which could be one of the reasons for persistence in the hospital environment.Both clinicians and microbiologists need to be aware of this non fermenter in neonatal infections.

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Partial Monosomy 7q

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We report a case of partial monosomy 7q and partial trisomy 14q in a 4 year old male with microcephaly, prominent eyes, arched eyebrows, malformed ears and overlapping of toes. The unbalanced rearrangement resulted in monosomy of 7q33→qter and trisomy of 14q32.2→qter. The clinical phenotype was similar to the other cases of 7q deletion.

Key words: *7q monosomy, 14 q trisomy, Mental retardation, Translocation.*

Reciprocal translocation carriers are at the risk of having a mentally and physically abnormal child because of “segmental aneusomy”. The imbalance is due to duplication or deletion of the chromosome segment involved in segregation. Partial autosomal monosomies and trisomies, although associated with congenital malformations, are known to be compatible with life.

7q deletions have been reported in more than 30 cases as either an isolated deletion or in combination with other chromosomal anomalies [1]. In most of the cases the associated clinical features are highly variable, and are found to share a few common features like microcephaly, broad nasal bridge, bulbous nasal tip, auricular malformations, micrognathia and genital anomalies, which delineate a distinct phenotype as ‘7q terminal deletion