NEONATAL MELIOIDOSIS

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ABSTRACT

We report a neonate with melioidosis. The presentation, progress and treatment are described, followed by a discussion on melioidosis.

Keywords: *melioidosis; neonatal; Pseudomonas pseudomallei*

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INTRODUCTION

Melioidosis is a tropical disease caused by *Pseudomonas pseudomallei*. This organism is mainly distributed in soil and water[1]. The disease is more prevalent in the adult population. Melioidosis is a seasonal disease and is more common during rainy seasons. The disease has a wide spectrum ranging from asymptomatic infection to frank septicemia[2,3]. There have been many reports in adults, but experience in children is limited. In the last decade, with improvement of microbiological techniques, we have witnessed an increase in the number of cases diagnosed[4]. Neonatal melioidosis has recently been highlighted in the literature. The first case was reported in 1971 from USA, followed by five cases from Thailand[5,6]. Though cases were observed in adult, to the best of our knowledge there has been no reported cases of neonatal melioidosis in Malaysia. We report here one fatal case of neonatal melioidosis.

CASE REPORT

A male infant weighing 3400 gm was delivered at term to a 26-year-old healthy mother at a district hospital. Since it was a normal delivery, both infant and mother were discharged home within a few hours of delivery. While at home, in his first 30 hours of life, the baby developed laboured breathing, became lethargic and sucked poorly. Just prior to the admission, the baby had one cyanotic attack. He was admitted to the Neonatal Intensive Care Unit of the Hospital University Sains Malaysia at 40 hours of age with clinical evidence of sepsis and bronchopneumonia. The total white blood cell count on admission was 6300/mm^3, with 7% band forms, 47% segmented neutrophils, 36% lymphocytes, 8% monocytes and 2% basophils. Lumbar puncture was traumatic but the differential cell count in the CSF was unremarkable. The urine showed moderate proteinuria with no growth on culture. The chest X-ray revealed bilateral patchy opacities. He was initially treated with penicillin and gentamicin. The condition of the baby deteriorated with the appearance of fits. A cranial ultrasound revealed no abnormality. On the third day of life, the baby's condition suddenly deteriorated. At this time the blood culture done on admission had become available and revealed growth of *Acinetobacter species* sensitive to ticarcillin and gentamicin. A repeat septic workup was performed and penicillin was substituted by ticarcillin. He continued to deteriorate and subsequently developed diffuse macular rash all over the body. These rashes over a few hours developed a central pallor without any vesicles. At the same time, the arterial blood gas revealed severe metabolic acidosis. He subsequently slipped into a state of septic shock and respiratory failure and died on the 5th day of life. Post-mortem intra-cardiac blood culture was done. The second blood culture (done on 3rd day) and the intra-cardiac blood culture grew *Pseudomonas pseudomallei* sensitive to cefoperazone, cefotaxime, ceftazidime and resistant to the aminoglycosides. The cause of death was thus confirmed as melioidosis.

DISCUSSION

Pseudomonas pseudomallei is a gram negative, bipolar staining bacillus. It is prevalent in the tropics[2]. Neonatal melioidosis is a relatively rare condition as evidenced by few literature reports[5,6]. The most common presentation of this infection in neonates is sepsis. In the report from Thailand[6], four out of five cases presented as sepsis including one with meningitis. The fifth baby presented as congestive cardiac failure secondary to opening of the ductus arteriosus. Two babies out of five died suggesting that this is a fulminant illness. The skin lesions described in melioidosis usually range from superficial pustules to cellulitis or deep abscesses in septicemic form of the disease[2]. However, this child had macular rashes associated with central pallor and there was no evidence of cellulitis. In this patient the blood culture grew *Acinetobacter species*. It is possible that the *Acinetobacter species* was a contaminant. Inspite of the antibiotic therapy the baby deteriorated.

Transmission of the disease occurs primarily through contamination of the wound or by inhalation[7]. Other modes of transmission include laboratory acquired[8], venereal, perinatal[9,10] and vectors related transmission[11]. The possible mode of acquisition of the infection in neonates could be nosocomial and perinatal[5,6]. In our case the infection was probably nosocomial as the evidence of melioidosis was present

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only after the third day of life.

With regard to antibiotic therapy, there is no uniform consensus. A recent study recommended the use of ceftazidime, which was able to reduce the mortality by half[12]. No clear guideline is available for the duration of therapy. Dance in his review recommended treatment for at least two weeks and six weeks if large abscesses were present[2]. There are however no guidelines available for neonatal melioidosis.

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