Neonatal Streptococcus Pneumoniae Sepsis: Rare But Fatal

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Abstract

This is a case report of an infant who was born with severe Streptococcal Pneumoniae sepsis and died within hours of birth as a result. There are very few case reports of this illness in neonates. Although it is a rare occurrence in neonates it can be potentially fatal.

CASE REPORT

A 2038g male infant was born to a healthy 22 year old primigravida by normal vaginal delivery at 36 weeks gestation. There were no problems during pregnancy other than, the mother smoked during pregnancy and slight concerns regarding foetal growth at 34 weeks gestation. An antenatal scan at 34weeks showed reduced head circumference. Previous antenatal scans were normal. All routine antenatal blood tests were also normal. The mother was admitted one hour prior to delivery with abdominal pain. Due to late de-accelerations seen on the cardiotocograph, her membranes were artificially ruptured. Meconium stained liqour was seen. The infant was delivered with the umbilical cord tightly tied around the neck. Oral and nasal suction was performed. No meconium was seen in the vocal cords. The infant appeared white, floppy and had poor respiratory effort. The heart rate was below 100/min. Bag and mask ventilation was given after which the heart rate picked up by one minute. At 10 minutes the infant was breathing spontaneously, however continued to remain pale with poor perfusion and poor muscle tone. Apgars were 6 at 5minutes and 7 at 10minutes. Cord gases revealed an arterial pH of 7.232 and a venous pH of 7.337.

On admission to the Neonatal Unit the infant's oxygen saturation dropped to 53%. After initial bag and mask ventilation Conventional ventilation was started in 99% oxygen, with high pressure of 26/4 and a rate of 60/min. On examination the infant was hypotonic, had poor peripheral perfusion, and minimal spontaneous movements. 3 boluses of normal saline and 1 of 10% dextrose were given. Curosurf was administered. Blood culture was performed and intravenous Penicillin and Gentamicin were started. Chest radiograph showed complete white-out and small lungs. High frequency oscillation was soon commenced as oxygen saturations remained poor. Saturation improved to 92% on a FiO_2 of 100% and MAP of 17cm H_2O, although the infant continued to have both respiratory and metabolic acidosis despite of 2 boluses of Sodium bicarbonate. The echocardiogram revealed Patent Foramen Ovale and Patent Ductus Arteriosus with left to right shunt. His cranial ultrasound scan was normal. The full blood count (FBC) revealed a Haemoglobin (Hb) of 11.5g/dl, white cell count (WBC) of 4.9×10^9 /l (Lymphocytes=3.68x 10^9/l ,Neutrophils= $0.39x10^9$/l) and platelets of $93x10^9$/l . C reactive protein (CRP) was 32mg/dl. At this stage, our differential diagnosis was severe Sepsis, severe Hyaline Membrane Disease or Meconium Aspiration Syndrome.

At 6 hours of age there was a sudden deterioration with a drop in oxygen saturation to 53% and heart rate to 60/min. Cardiopulmonary resuscitation (CPR) was commenced and a dose of adrenaline was given. Right sided Pnuemothorax was suspected on cold light examination. A butterfly needle was inserted into 2nd right intercostal space followed by a chest drain. Saturation improved slightly up to 60% and then dropped again. A further chest drain was inserted. Chest x-ray revealed large Pneumothorax on the right with small lung volume on the left. In spite of continuous vigorous efforts to resuscitate with adrenaline, dopamine and fluid bolus, the infant continued to deteriorate. After two and a half hours of struggling, intensive care was withdrawn. The postmortem examination revealed Streptococcus Pneumoniae Septicaemia.

DISCUSSION

The incidence of neonatal sepsis in the developed world is 2 per 1000 live births. The incidence is increased with low birth weight or prematurity. Neonatal sepsis can be either early onset which occurs in the first 7days of life or late-onset which occurs at 7-90 days of life. In early onset the cause is usually infection ascending from the maternal genital tract, or, less commonly, via the placenta. The microorganisms most commonly associated with early onset infection are group B Streptococcus, Escherichia coli, Haemophilus influenzae, and Listeria monocytogenes. In late-onset the organisms may be acquired from the external (e.g. hospital) environment when organisms initially colonize superficial sites and the upper respiratory tract and progress to cause widespread sepsis. The common microorganisms associated with late-onset neonatal sepsis are Coagulase-negative Staphylococci, Staphylococcus aureus, E coli, Klebsiella, Pseudomonas, Enterobacter, Candida and group B Streptococcus.

Streptococcus Pneumoniae is an alpha haemolytic Gram positive diplococcus. It is present in more than 50% of the healthy population in the respiratory tract. Streptococcus Pneumoniae infections in neonates are relatively unusual events (1-11% of all neonatal sepsis) but are associated with substantial morbidity and mortality[1]. Neonatal materno-fetal infection is rare but serious[2]. The mortality in neonates is up to 60%[2]. S. Pneumoniae in neonates is transmitted by the ascending route on passage through birth canal. S. Pneumoniae is not a part of the resident vaginal flora but in some women it can be a transient part of the vaginal flora, and pelvic infection can occur especially if a predisposing condition exists (e.g. use of an intrauterine contraceptive device, recent birth or gynaecological surgery)[3]. The colonisation of the female genital tract is possibly by contaminated obstetric instruments or by oro-genital sexual practices[4]. An increased number of cases of neonatal sepsis by S. Pneumoniae have been reported in recent years but no increase in the relative incidence among neonatal infection has been noted[5]. Changes in sexual practices during pregnancy and improvement in isolation and differentiation techniques of S. Pneumoniae from other alpha haemolytic Streptococci could potentially account for the recent increase[6].

Recent reviews and studies have concluded that gestational ages, low birth weight and prolonged rupture of membranes do not appear to be risk factors for neonatal S. Pneumoniae infection[6]. Most reports suggest that babies infected are likely to be greater or equal to 38 weeks gestation[1]. Most mothers found to carry this organism were asymptomatic at the time of delivery. Early onset S. Pneumoniae neonatal sepsis has a worse prognosis and higher mortality than late onset sepsis[6]. Death in early onset sepsis usually occurs within 36 hours of presentation. Presentation of S. Pneumoniae neonatal sepsis has no specific features to differentiate it from other causes of neonatal sepsis. Various forms of presentation include bacterial meningitis, bacterial pneumonias, DIC, septic arthritis, osteomyelitis and otitis media. Invasive S. Pneumoniae infection in neonates has also presented with leucopenia/neutropenia, but this does not predict poor outcome[1].

In order to try to prevent Neonatal Streptococcal Pneumoniae, the WHO (1998 Geneva) advised the need to establish whether it would be beneficial to vaccinate mothers during pregnancy or vaccinate the newborn. There are 2 vaccines available. These are the Plain vaccine which is a 23 valent unconjugated vaccine and the new conjugate vaccine which is 7 valent. The 23-valent vaccine was safe and immunogenic in pregnant women and transplacental transmission of vaccine-specific antibodies was efficient[7]. Infecting serotypes reported include 19,9,3,18,1,6,14,5,and 12[1]. Serotypes responsible for 26% of invasive Steptococcus Pneumoniae infections in neonates are 1,3,5,and 12 which are not included in the 7-valent pneumococcal vaccine[1].

The rarity of vaginal carriage of Pneumonoccus suggests that this organism carries a higher invasion to colonization ratio than Group B Streptococcus and maternal carriage or neonatal colonization should be more aggressively treated[8]. S. Pneumoniae should always be considered as a cause of neonatal sepsis[9]. S. Pneumoniae should be specifically sought in swabs taken from the pregnant mother and newborn, and if isolated, even in the absence of symptoms, antibiotic therapy should be strongly considered for the mother and the baby[10]. In areas where S. Pneumoniae resistance is a significant problem serious consideration should be given to adding Vancomycin and / or large dose Cefotaxime to the antibiotic regimens if S. Pneumoniae is being considered as the cause of the neonatal sepsis[6].

Mothers of infants affected by early onset pneumococcal sepsis that have low pneumococcal antibody levels run the risk of subsequent babies being similarly affected and vaccination should be considered to prevent recurrence[11]. There is also concern that increasing efforts to prevent Group B Streptococcus neonatal disease may lead to an

increase in neonatal organisms due to resistant organisms[12]. Streptococcal Pneumoniae produces serious diseases in neonates. Because of increasing prevalence of penicillin-resistant pneumococci, the relationship between the percentage of mothers colonized with pneumococci and neonatal infections should be determined to develop new prevention and treatment strategies in newborn infants[13].

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