A 4 kg male baby was born to a healthy 35 years old G2P1A0 by normal vaginal delivery at 36 weeks gestation. There were no problems during pregnancy other than gestational diabetes controlled by insulin. An antenatal echography and routine antenatal blood test were normal. Vaginal exudates culture for Streptococcus agalactiae (GBS) was negative, serology tests results for toxoplasmosis, rubella; HBV were negative.

The mother was admitted one hour prior to delivery with abdominal pain and early contractions. Due to late and variable deceleration seen on the cardiotocograph, her membranes were artificially ruptured. Meconium stained fluid was seen.

The born infant was vigorous with a 9/10 Apgar score; oral and nasal suction was performed. The baby was transferred to the nursery. At hour 1 of life he started to have poor suck, hypotonicity, grunting with retractions and 38.5°C fever. He was transferred to the intensive care unit.

Upon admission to the neonatal intensive care unit the baby was ill looking, hypotonic, had continuous grunting with subcostal and intercostal retractions, as well as nasal flaring. He had abnormal jerky movements in the lower limbs bilaterally and bilateral eye twitching.

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Cardiac auscultation was normal. The examination of other organs showed no significant findings.

Laboratory tests taken upon admission showed: • Leucocytes: 14.5 x 10^3/μL with left shift • Normal red blood cell and platelet counts • Electrolytes, glucose, calcium, magnesium and phosphorus within normal limits • C-reactive protein 240 mg/l (normal < 10 mg/l) • Negative urine testing.

Cerebrospinal fluid analysis showed: • Turbid lightly pink fluid • Xantochromia testing positive • Protein: 270 mg/dl • Glucose: 29 mg/dl • WBC: 600 cells/mm^3 (N: 70%, L: 30%) • RBC: 10000 cells/mm^3 • Gram stains: positive for gram + diplococcus.

In addition, blood and urine, CSF samples were taken for culture.

Chest radiography showed mild bilateral in-filtrate.

During the first few hours after admission, the patient presented a declining in his general condition. He required 2L/min oxygen by nasal canula, with abnormal movement in the limbs bilaterally. Due to the unavailability of brain echography, a CT scan was done and showed symmetric expansion of the lateral ventricules with bilateral occipital corn hemorrhage mainly on the left (Figure 1A).

Empiric intravenous antibiotic treatment (ampicillin, Claforan and Amikin) was initiated with antipyretic and phenytoin intravenous after the consultation of neurologist. At this stage, our differential diagnosis was severe meningitis with intracerebral hemorrhage.

On the third day of admission, he had improvement in his respiratory symptoms, no more need for oxygen, saturation was 98% on room air, no more abnormal movements except mild neck spasticity, and he had good suck. Follow-up head circumference was stable. The culture results came back negative for urine, while the CSF and blood cultures was positive for S. pneumoniae (sensitive to Amikin and Claforan). Follow-up labs showed decreasing in C-reactive protein (CRP) to 37 mg/l and leucocytes: 11.2 x 10^3/μL (N: 56%, L: 26.7%). He was started on PO feeding.

The baby was diagnosed to have early onset neonatal sepsis due to Streptococcus pneumoniae (serotype not done).

He was discharged from the hospital at day 15 of life after completing antibiotic treatment with normal physical examination on valproic acid PO. Follow-up CT brain scan one month later showed mild improvement of the intraventricular hemorrhage and 6 months later it was normal (Figure 1B,1C). Depakin was tapered gradually then discontinued at age of 8 months. At this age the baby had normal physical exam with normal milestones.

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 7 days 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, Esherichia coli, Haemophilus influenza, 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 most common microorganisms associated with late-onset neonatal sepsis are coagulase negative staphylococci, Staphylococcus aureus, E. coli, Klebsiella spp., 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 upper 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 neonate is transmitted via the ascending route on passage through the birth canal. *S. Pneumoniae* is not part of the resident normal 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 gynecological surgery) [3]. The colonization 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].

Recent reviews and studies have concluded that gestational age, 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 neonatal sepsis has no specific features to differentiate it from other causes of neonatal sepsis. Various forms of presentation include bacterial meningitis, bacterial pneumonia, disseminated intravascular coagulation, septic arthritis, osteomyelitis and otitis media. Invasive *S. Pneumoniae* infection in neonates has also presented with leucopenia/neutropenia, but this does not predict poor prognosis [1].

In order to try to prevent neonatal streptococcal pneumonia, the WHO (1998 Geneva) advised the need to establish whether it would be beneficial to vaccinate the mothers during pregnancy or vaccinate the newborn. Newborn vaccination schedules could substantially reduce the impact of pneumococcal disease in immunized children, but does not have an effect on the morbidity and mortality of infants less than three months of age. Pneumococcal vaccination during pregnancy may be a way of preventing pneumococcal disease during the first months of life [7]. Until 2018, two types of pneumococcal vaccine were available: the pneumococcal polysaccharide vaccine (PPSV23) and the pneumococcal conjugate vaccine PCV13 [7]. The 23-valent vaccine was safe and immuno-nogenic in pregnant women and transplacental transmission of vaccine-specific antibodies was efficient [8]. Infecting serotypes reported include 19, 9, 3, 18, 1, 6, 14, 5, and 12 [1]. Serotypes responsible for 26% of invasive *S. Pneumoniae* infections in neonates are 1, 3, 5 and 12. PPSV23 covers 23 serotypes including 1, 3, 5, and 12. But there is insufficient evidence to support whether pneumococcal vaccination during pregnancy could reduce infant infections. A study was conducted in July 2014 in the Cochrane Pregnancy and Children Group’s Trials Register to assess the effect of pneumococcal vaccination during pregnancy for preventing infant infection [7]. The study is a randomized controlled trial that compare the effect of streptococcus infection in infants in two groups. In the first one, mothers received pneumococcal vaccine (PPSV23); in the second, mothers received placebo. This study showed that there is no evidence that pneumococcal vaccination during pregnancy reduce the risk of neonatal infection [7]. More studies are needed to confirm whether pneumococcal vaccination during pregnancy is effective in reducing infant infections.

The rarity of vaginal carriage of pneumococcus 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 [9]. *S. Pneumoniae* should always be considered as a cause of neonatal sepsis [10]. *S. Pneumoniae* should be specifically sought in swabs taken from the vagina of a pregnant mother and from the newborn, and if isolated, even in the absence of symptoms, antibiotic therapy should be strongly considered for the mother and the baby [11].

Mothers of infants affected by early onset pneumococcal antibody levels run the risk of subsequent babies being similarly affected and vaccination should be considered to prevent recurrence [12]. 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 [13]. Streptococcal pneumonia produces serious disease in neonates. Because the increasing prevalence of penicillin-resistant pneumococci, the relationship between the percentages of mothers colonized with pneumococci and neonatal infections should be determined to develop new prevention and treatment strategies in newborn infants [14].

CONCLUSION

*S. Pneumoniae* remains a rare but important cause of neonatal sepsis and can mimic early onset group B streptococcal sepsis. It is unclear whether current infant or adult pneumococcal immunization programs might influence its incidence in the neonatal period. We should screen for *S. pneumoniae* by swabs culture; if result is positive we should treat even in the absence of symptoms. Till now there is insufficient evidence to support whether pneumococcal vaccination during pregnancy could reduce infant infection, and more studies are needed to assess the effect of pneumococcal vaccination during pregnancy for preventing infant infection.

REFERENCES

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