CASE REPORT

Post-partum Invasive Group B Streptococcus Infection With Fatal Outcome: A Case Report

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ABSTRACT

Group B streptococcus (GBS) is generally known to cause severe disease in the neonate and immunocompromised adults. GBS in the pregnant mother is rare and can potentially be fatal. Clinical presentation can be as mild as an uncomplicated urinary tract infection or serious invasive disease in the form of bacteremia, chorioamnionitis, endometritis and septic abortion. We report a case of a 46-year-old Para 3 lady, post-partum day 12, whom was found dead at home. Prior to her death, she had intermittent fever and abnormal lochia. Autopsy findings indicate GBS endometritis and bacteraemia. She was never screened for GBS. The cost-effectiveness of universal GBS screening needs to be explored to reduce maternal and neonatal morbidity due to GBS.

Keywords: *Streptococcus agalactiae*, Maternal mortality, Endometritis, Autopsy

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INTRODUCTION

Group B streptococcus (GBS) or *Streptococcus agalactiae* is a gram positive bacteria that is widely known to cause neonatal meningitis and sepsis which results in significant morbidity and mortality. Although GBS colonization is high in pregnant mothers, approximately 30% in Malaysia, only 0.3% of infants of carrier mothers develop sepsis and 3 out of 10,000 of these infants die (1). However, invasive maternal GBS is extremely rare, accounting for 1 case per 100,000 births (2). Invasive GBS in pregnancy may manifest as bacteremia, chorioamnionitis, endometritis and to a lesser extent, septic abortion. Herein we report a case of invasive maternal GBS leading to mortality.

CASE REPORT

We report a case of a 46-year-old lady who presented to the hospital casualty with no signs of life after being found unconscious at home. She had recently given birth to her third child, 12 days prior, vaginally with no complications. Her last child birth was 6 years prior to this pregnancy. She had two previous pregnancies with uneventful spontaneous vaginal deliveries at term. She was also not known to have any significant medical illness. During the third pregnancy, her antenatal booking was at 13 weeks gestation with subsequent regular follow-ups at a local district clinic. Gestational diabetes mellitus was detected near term and controlled with dietary modification. She had spontaneous onset of labour at 39 weeks of gestation and was admitted to a district hospital. There was no leaking liquor or any episode of fever prior to labour or during intrapartum period. The labour was not prolonged and she delivered vaginally a healthy baby girl of 3.28 kg with Apgar scores of 9 and 10 at one and five minutes respectively.

She was discharged home the next day with postpartum visits planned at her local district clinic. Since discharge from the hospital, she reported experiencing abnormal lochia. The lochia was described as heavy in amount, blood-red in colour although non-foul smelling. It was associated with intermittent fever, lethargy and poor appetite. All of the symptoms occurred throughout the postpartum period. In addition, she also developed one episode of breathing difficulty one day prior to her death.

Medico-legal autopsy was requested on the same day of her death by the investigating police officer to ascertain her cause of death. Unnatural cause of death was ruled out as no suspicious injuries were found on the body. Apart from oedematous lower limb and hyperaemic inner mucosal wall of vagina, external examinations were unremarkable. Internal examinations revealed pale and non-contracted uterus weighed 650 grams. Uterine

walls appeared thin while cervix was oedematous and haemorrhagic (Fig.1a). The endometrial surface was entirely haemorrhagic with the presence of punched-out areas interspersed with multiple adherent clumps of haemorrhagic necrotic tissue and blood clots (Fig. 1b and 1c). Both ovary and fallopian tubes were unremarkable. Grossly, the lungs, liver and spleen were enlarged and congested while the kidney appeared pale. The brain and heart showed no significant morphological changes. Blood taken during the autopsy (approximately 8 hours after the time of death) showed a raise in total white cell count (22.3 x109/L) and prolonged bleeding profile (PT : >120 seconds, APTT : > 180 seconds). Blood and tissue from spleen and liver isolated Group B Streptococcus which was sensitive to ampicillin, penicillin, erythromycin, gentamicin and clindamycin while resistant to tetracycline and co-trimoxazole. Microscopic examination of the uterus (Fig.2) were suggestive of endometritis. The adherent endometrial clots (Fig.3) contained bacterial colonies pointing to an infection. Other interesting microscopic findings are follicular formation of the spleen, foci infiltration of heart interstitium and liver parenchyma by neutrophils and lymphoplasmacytic cells, generalized hepatocyte infarction, congested and proteinaceous fluid-filled of lung alveoli, and also foci of tubular infarction of renal. Presence of follicular formation or reactive change therefore supports that infection plays an important role in the disease process or even death like in this case.

The brain was devoid from significant pathologic changes. There was no evidence of pulmonary embolism. Autopsy findings together with the culture results were suggestive of GBS endometritis. Her baby was never admitted to hospital for sepsis and is well and alive at home.

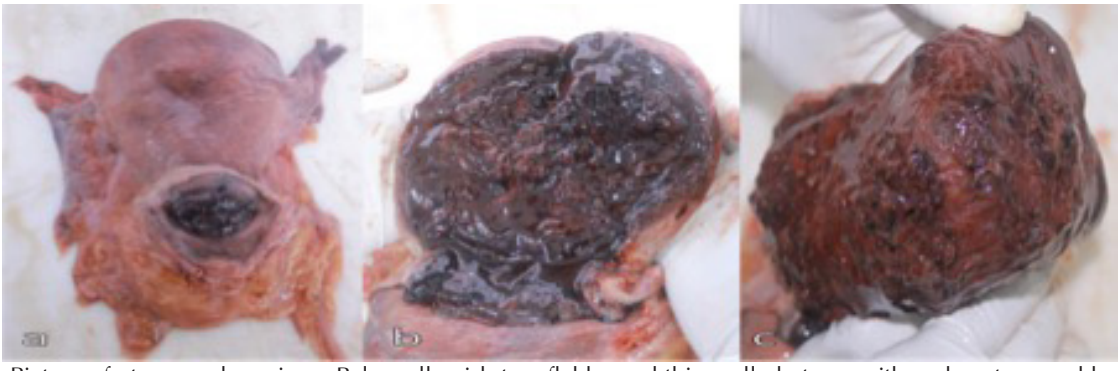

Figure 1: Picture of uterus and cervix. a: Pale, yellowish-tan, flabby and thin walled uterus with oedematous and haemorrhagic cervix. b-c: Cut section of uterus before and after cleansed from haemorrhages. Haemorrhagic endometrial surface with presence of punch-out areas interspersed

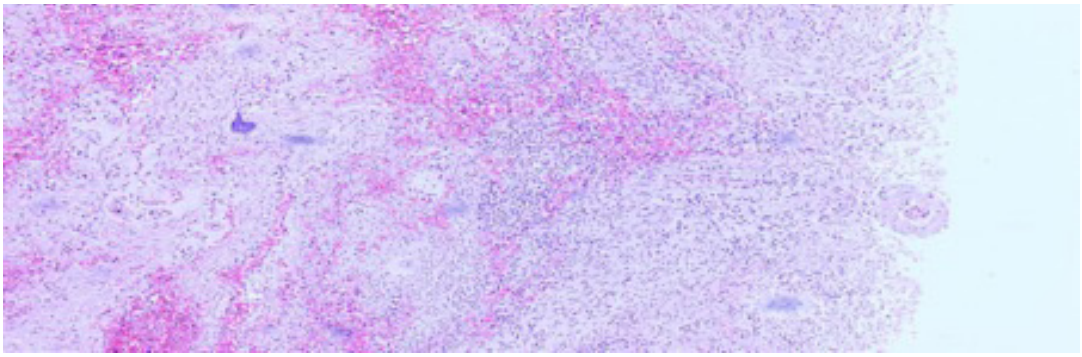

Figure 2: Section from uterus showing haemorrhagic endomyometrium with cellular infiltration of mixed neutrophils and lymphoplasmacytic cells; Presence of bacterial clumps seen (4x H&E).

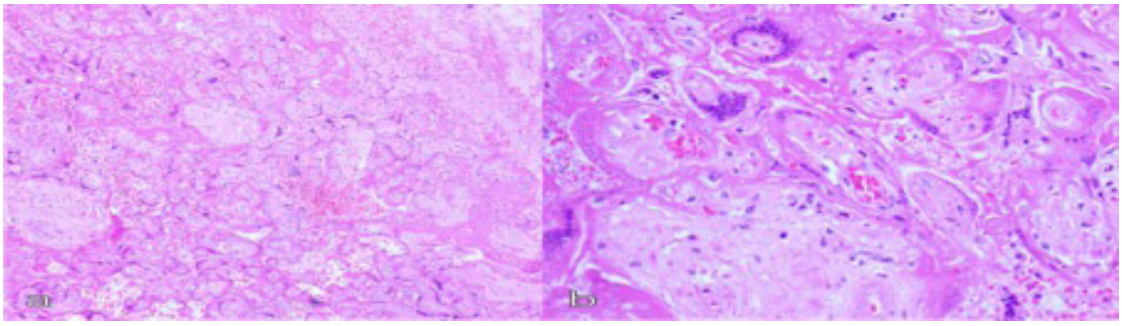

Figure 3: a: Section from adherent endometrial clots contained haemorrhagic fibrinous necrotic exudates admixed with prominent chorionic villi of various sizes and shapes (4x H&E). b: Chorionic villi with degenerated syncytio-trophoblasts (20x H&E).

DISCUSSION

There are 10 serotypes of GBS characterized after its capsular polysaccharide which also contributes to its virulence. Antibodies to capsular polysaccharide confers protection against invasive disease. Serotype Ia, III and V are commonly associated with invasive disease although geographical variations exist. Local studies have proven these 3 serotypes are prevalent within GBS isolates in Malaysia (3). Though there is a lack of local data on the incidence of pregnancy-related GBS. GBS frequently colonizes the human genital and gastrointestinal tracts. The pathogenesis of chorioamnionitis is by retrograde or ascending infection from the lower genital tract to the chorioamnion and/or umbilical cord of the placenta. Haematogenous, iatrogenic infection as a complication of amniocentesis and anterograde infection from peritoneum via the fallopian tubes is also possible though less common (4).

Routine GBS screening during the antenatal period is still controversial. The Royal College of Obstetricians and Gynaecologists recommends clinical risk-based screening strategy in the UK whereas the US and the CDC guidelines recommend a universal swab-based screening strategy. Local Malaysian protocols practice clinical risk-based screening as the benefits and cost-effectiveness of early detection is still unclear. Therefore, this patient was never screened for GBS throughout her 3 pregnancies.

GBS frequently colonizes the human genital and gastrointestinal tracts. It is an important cause of infection in 3 populations: Neonates, pregnant women and non-pregnant adults. GBS in the pregnant lady usually presents as a urinary tract infection. However, the infection may become invasive and manifest as bacteremia, chorioamnionitis, endometritis and to a lesser extent, septic abortion (5).

Fever and unusual amounts of lochia even if non-foul smelling should raise alarm in a post-partum mother. However, it is unknown why this patient did not seek medical attention. Previous studies have noted that infants of mothers with severe GBS sepsis are at increased risk of developing sepsis themselves and were more likely to have been delivered preterm (2). In our patient, her baby was born term, remained aseptic and was well post-delivery which led to low suspicion of GBS in the mother.

It is important to promptly diagnose GBS colonization in order to initiate immediate intrapartum antibiotic therapy. Studies have demonstrated that use of intrapartum antibiotics significantly reduces maternal and fetal complications of chorioamnionitis (4). Clinical consensus recommends intravenous ampicillin every 6 hours and gentamicin every 8-24 hours until delivery. However, the optimal antibiotic regimen has not been well-studied (4).

CONCLUSION

We present a rare case of maternal mortality due to invasive GBS. Studies on incidence of pregnancy-related GBS are needed to determine the cost-effectiveness of universal screening in pregnant mothers.

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