A Case Report of Lethal Invasive Group a Streptococcal Infection in the Puerperium.

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Keywords: Invasive group A streptococcus; infection; pregnancy; puerperium; toxic shock syndrome; hysterectomy.

Introduction

Invasive Group A Streptococcus (iGAS) is a well-recognised infection in the peripartum period associated with high morbidity and mortality [1,2]. In the late 1980’s the maternal mortality rate from sepsis in the UK was 0.4/100 000 maternities, whereas in the period from 2006 to 2008 the maternal mortality rate increased to 1.13/100 000 [3].

Infections with virulent strains of iGAS can take an unpredictable course in which fever or mild somatic symptoms might be the only warning signs before progression to toxic shock syndrome (TSS) in 8-14% of cases [4]. We report a case of uterine infarction secondary to pelvic thrombosis associated with iGAS and TSS treated by intensive care and hysterectomy.

Case report

A previously well 29-year-old woman, para 2 presented at 33 weeks with threatened preterm labour. She went in to active labour 12 hours after admission. A live, healthy female infant weighing 2120 grams was delivered vaginally and showed no signs of infection (she was transferred to the special care unit for twenty four hours as a routine precaution due to maternal deterioration).

Two hours postpartum, the patient developed a temperature of 38.1°C, heart rate of 159 beats/minute and blood pressure of 66/21 mmHg. On examination she had diffuse lower abdominal pain and a purpuric abdominal rash. Blood, vaginal and urine cultures were taken prior to treatment with intravenous ticarcillin-tazobactam. Initial blood tests showed features consistent with infection and coagulopathy and arterial blood gas analysis showed a mixed respiratory and metabolic acidosis. She was taken to theatre for examination under anaesthesia, however, no retained products were found. As there was no clinical improvement, she was transferred four hours postpartum to the intensive care unit (ICU). A diagnosis of suspected TSS was made and her antibiotics were changed to meropenem, clindamycin and gentamicin. Continuous veno-venous haemodiafiltration (CVVHDF) was commenced to correct her acidosis.

She remained intubated and ventilated and four units of packed red cells were transfused as part of supportive care. On day two, due to lack of improvement, a subtotal hysterectomy was performed [Figure 1]. Blood cultures, placental tissue and high vaginal swab confirmed group A streptococci. CT scan of the pelvis revealed extensive pelvic and ovarian vein thrombosis. She had an inferior vena cava filter inserted and was commenced on therapeutic heparin.

A Computed Tomography Pulmonary Angiogram (CTPA) confirmed bilateral pulmonary embolism despite a therapeutic. International Normalized ratio (INR) (2 - 3). Histological examination revealed a uterus with features of acute inflammation and vascular thrombi, complete loss of endometrial tissue and areas of infarcted myometrium muscle. She recovered gradually and was discharged home on day nineteen postpartum.

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Received: July 19, 2018; Accepted: July 23, 2018; Published: July 25, 2018
Discussion

iGAS is a rare infectious disease in the puerperal period. Since the first report from Scandinavia in 1987 [1] an increase in virulent iGAS strains has been documented in the literature. The Streptococcal and Diphtheria Reference Unit (SDRU) at the Health Protection Agency Centre for Infections have observed a 66% increase in iGAS incidence in December 2008 compared with the previous year [5]. M1 and M3 subtypes occurred most commonly, accounting for 30-50% of maternal mortality [6], whilst the M28 strain is also common.

Infections due to iGAS have been attributed to a variety of risk factors including spontaneous miscarriages, vaginal or abdominal hysterectomies, caesarean sections, genital tract abscesses and intrauterine contraceptive devices [2].

There have been case reports utilising intravenous immunoglobulin (IVIG) in iGAS TSS with improvement suggesting neutralisation of toxins as a possible mechanism [2,7]. The use of IVIG has been associated with favorable outcome in 91% of cases reviewed [8].

In our case, IVIG was considered for use, but due to her clinical improvement was not given. In conclusion, we believe that severe prolonged hypotension along with toxic shock due to iGAS in a pregnant woman may have caused the uterine infarction. The uterine vasculature is unable to auto regulate blood flow in response to variations in perfusion pressure, and therefore requires an adequate blood pressure to maintain perfusion.

The combination of dilated puerperal vascular beds and septic shock leads to a syndrome of low flow related haemostasis resulting in uterine small arterial thrombosis and infarction of the uterus. Early recognition and intensive treatment for iGAS during pregnancy is recommended in women with high fever, muscular pain, hemolysis and DIC during pregnancy [9]. Failure to respond to intensive resuscituation should lead the obstetricians to suspect uterine infarction and hysterectomy should be considered to improve the outcome [10].

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References