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Staphylococcus Aureus Septic Shock during Labor: A Case Report

Soumaya Berrada, Soukaina Laaraj, Ghizlane Salek, Jaouad Kouach and Driss Moussaoui Rahali

Department of obstetrics & gynaecology Military Training Hospital Mohamed V, Rabat, Morocco.

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ABSTRACT

Pregnancy causes an altered immune response which predispose pregnant women to increased severity of infections. Management of septic shock requires prompt resuscitation with volume expansion, inotropic therapy and empiric antibiotic therapy. Toxic shock syndrome can be caused by *Staphylococcus aureus* or *Clostridium sordellii*. It may occur unexpectedly after an uncomplicated pregnancy and delivery. Management includes antibiotic therapy with penicillin and clindamycin and early surgical intervention. Immunoglobulins may be beneficial. We report the case of a 22 years-old primagravida who represents a septic shock during labor caused by staphylococcus aureus which the point of departure was a urinary tract infection.

Introduction

Sepsis is defined [1] as suspected or proven infection plus a systemic inflammatory response syndrome (e.g., fever, tachycardia, tachypnea, and leukocytosis). whereas septic shock is defined as sepsis with organ dysfunction and hypotension, despite adequate fluid resuscitation.

S. aureus is responsible for a multitude of infections where it is either present on the infection site or acts at a distance by secretion of toxins. [2]

Proposed revision of diagnostic criteria for staphylococcal toxic shock syndrome (TSS) includes (1) isolation of *S. aureus* from mucosal or normally sterile site; (2) production of TSS-associated superantigen by isolate; (3) lack of antibody to implicated toxin at time of acute illness; and (4) development of antibody to toxin during convalescence. [2]

Observation

This is a 22-year-old primigest, blood group A +, without ATCD, admitted in labor at 39 SA + 4j with a fever at 40 ° C dating back to 2 days.

The pregnancy was well followed with a complete and normal prenatal assessment (normal OGTT), several consultations and ultrasound revealed a macrosomia at 33 WA. The patient reported an unencrypted fever 2 days before the day of her consultation with urinary frequency and burning, for which she took paracetamol without consulting. She expressed vomiting, diarrhea and decreased active fetal movements with the onset of uterine contractions on the day of admission.

The examination of admission found a conscious patient, well oriented in time and space, in a state of severe sepsis: tachycardia, polypnea, TA = 10/06 cmHg, febrile at 40 ° C, with a sign of Giordano positive, soft calves and thighs without neurosensory signs or edema of the lower limbs.

At the obstetrical examination, the uterine height= 35cm, the fetal heart sounds not perceived, the patient contracted at 2CU / 10min with good inter phasic relaxation, the cervix was 80%, dilated at 2cm, with a flexible lower segment, a high cephalic presentation and intact membranes.

Ultrasonography confirmed fetal death without evidence of skin delamination or skull bone overlap, with an estimated fetal weight at 4000g, postero-fundal placenta without hematoma and a normal amount of amniotic fluid. Renal ultrasound found pyelic dilation without image of renal abscess or obstruction.

Emergency management was the setting up of the condition, carrying out a complete infectious assessment, with a vascular filling with 1 l of saline serum, the administration of paracetamol, a bi-antibiotherapy based on amoxicillin-clavulanate and metronidazole.

The evolution was marked one hour later by aggravation in a state of septic shock: fever at 40,1 ° C, TA = 08/04 cmHg and SaO2 = 85%, with oliguria, appearance of petechiae and mottling without metrorrhagia.

The biology found a multi-organ failure: hematologic (anemia at 7,8g / dl, leucopenia at 3600 elmts / mm3, disseminated intravascular coagulation: plq = 15000elmts / mm3, prothrombin rate = 28%, activated cephalin time lengthened to 70 sec, fibrinogen = 1.1 g / l), hepatic cytolysis and renal failure (urea = 0.46g / l, creatinine = 25 mg / l), acidosis (RA = 9 mol / l). the infectious balance showed a urinary infection with staphylococcus aureus, a positive blood culture with staphylococcus aureus and a CRP = 208 mg / l.

The patient was resuscitated by the administration of norepinephrine, the transfusion of 7 red blood cells, 9 platelet pellets and 12 fresh frozen plasmas, the bi-antibiotic treatment with ceftriaxone and tazocyline.

After hemodynamic stabilization and transfusion, under general anesthesia and oro-tracheal intubation, it was possible to perform caesarian fetal extraction of a stillborn weighing 4100g. Examination of the placenta did not find any detachment or retro-placental hematoma. Plasma samples retrieved a posteriori found bacteriology staphylococcus aureus and normal pathological examination.

Tele: 00212611298529

E-mail address: soumaya.berrada12@gmail.com

During caesarean section, and after hysterorrhaphy, the uterus remained loose without signs of apoplexy, so a triple arterial ligation was performed with Lynch technique haemostatic stitches, the placement of an intraperitoneal drain and administration of 800 µg misoprostol intra-rectal and continuous infusion of oxytocin to prevent uterine inertia.

The patient remained intubated under mechanical ventilation, with norepinephrine, heparin sodium, calcium, exacyl and vitamin K.

The post-operative evolution was marked by the stabilization of the blood pressure, the SaO2 and the diuresis during 2h then there was a relapse of the hypotension, the anuria and the progressive desaturation following a pulmonary haemorrhage complicated by a cardiac arrest not recovered.

Discussion

Sepsis, including maternal sepsis and other pregnancy-related infections, is one of the 9 leading causes of maternal death [3]. Other causes include haemorrhage, hypertension, labor anomalies, abortion, other direct causes, indirect causes, HIV and late mortality (between 6 weeks and 1 year after birth).

The maternal mortality rate globally decreased [3] and sepsis remains one of the causes of maternal death where avoidability is highest. [4]

Toxic shock syndrome can be caused by Staphylococcus aureus or Clostridium sordellii. It may occur unexpectedly after an uncomplicated pregnancy and delivery. [5]

Distribution of S.aureus infections include lower respiratory tract infections, bloodstream infections (positive blood cultures with fever or hypotension), endocarditis; infections of the urinary tract, brain, and abdominal cavity. Mortality related to S. aureus BSI is high, ranging from 20% to 30% of the cases. [2]

The contemporary epidemiology of severe pregnancy-associated sepsis syndrome (PASS), focusing primarily on hospitalizations during labor, has been studied in a cohort, on the population of Texas (2001-2010): Hospitalization during labor accounted for 38% of all PASS-related hospitalizations with 111 deaths.

The following major risk factors have been independently associated with PASS: chronic liver disease, congestive heart failure, gestational diabetes, anemia, substance abuse, lack of health insurance [vs. Private Insurance], black race, and age ≥ 35. [6]

The main predictors of mortality associated with PASS were: gestational diabetes, substance abuse, lack of health insurance, septic shock and age ≥35. [6]

Potential causes [1] of sepsis or septic shock during pregnancy and the postpartum are: Acute pyelonephritis, Pneumonia, Neglected chorioamnionitis, Endomyometritis, Wound infections including episiotomy- unrecognized or inadequately treated, septic abortion with retained products of conception, ruptured appendix, infarcted bowel, Necrotizing pancreatitis, acute cholecystitis.

Signs and Symptoms of Septic Shock [1] are: fever (>38°) or hypothermia (temperature <36° with tachycardia HR > 110 beats per minute [bpm]), tachypnea (RR >24/min), diaphoresis, clammy/mottled skin, nausea/vomiting, hypotension/shock, oliguria, pain (location based on site of infection), altered mental state (confusion, decreased alertness).

The most common biologic anomalies in patients with septic shock during pregnancy is leukocytosis (usually a white blood count >15,000/mm3); however, in cases of advanced sepsis the patient can develop leukopenia and neutropenia. In addition, patients with viral sepsis usually have leukopenia. Moreover, most patients have abnormal serum creatinine levels (>1 mg/dL).

Management

The administration of intravenous antibiotics is the cornerstone of the treatment of sepsis. It must be done intravenously. [7] Broad-spectrum antibiotics should be administered to cover a wide range of potential pathogens. This approach is supported by retrospective studies in which inappropriate antibiotic therapy was associated with higher mortality. An additional question that sometimes arises is whether antifungals should be added initially. The decision to cover broadly must be linked to the commitment to defuse antibiotics based on the results of biologic results. De-escalation therapy is safe and leads to better results. Another controversial topic is the use of combined treatment for a high index of suspicion of pseudomonas infection. Recent data suggests that this approach could lead to better results. [7]

The timing of antibiotic administration has been the subject of several studies and a recent meta-analysis. [7] Kumar showed that early administration of antibiotics, hour after hour of hypotension diagnosis, improved survival. Delaying administration beyond the first hour has had an exponential impact on mortality. After adjusting for other factors, the timing of antibiotic administration was the strongest predictor of survival in this patient population [7]. Ferrer similarly showed that a delay in antibiotic administration was associated with a worsening of the survival. Not all studies support these findings [7]. In addition, correctly identifying a "zero" time can be difficult. It has been shown that in an emergency, a significant proportion of patients (15% to 23%) with documented severe sepsis are misclassified during triage; Thus, it is discouraging to correctly decide on a specific moment of onset of shock because of the unpredictable and changing clinical course of the disease. So, effective antibiotics should be administered as soon as possible and preferably within one hour of hypotension or other organ dysfunction induced by infection.

Management of Toxic shock syndrome includes antibiotic therapy with penicillin and clindamycin and early surgical intervention. IV immunoglobulin (2 g/kg over 1 to 3 days) may be beneficial. [5]

With S.aureus, even a single positive blood culture should prompt initiation of antibiotic therapy, sampling of blood for follow-up cultures, and determination of the source and extent of infection. An important consideration in bacteremia is the risk of endocarditis and the need for echocardiography in the assessment. [2]

Definitive therapy for methicillin-susceptible *S. aureus (*MSSA) bacteremias should be a β-lactam rather than vancomycin. [8] 14-day course of antibiotic treatment is necessary if BSI is related to a removable catheter or drainable localized infection. [9] Deeper infections, such as arthritis and osteomyelitis and endocarditis, must be treated with antibiotics for 4 to 6 weeks, with or without surgery depending on individual circumstances. Empirical antibiotic treatment must take into account the probability of (methicillin-resistant *S. aureus* (MRSA), which may represent more than 50% of cases of BSI in the hospital milieu. [2]

Conclusion

Altered immune response may predispose pregnant women to increased incidence or severity of certain

infections, management of septic shock is similar to that in the non-pregnant patients necessiting emergent antibiotics with penicillin and clindamycin, early surgical intervention and IV immunoglobulin.

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