

Antibiotic prophylaxis in cesarean section causing anaphylaxis and intrauterine fetal death

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Abstract

Intrauterine fetal death and maternal shock occurred as a result of a type-1 hypersensitivity reaction following antibiotic prophylaxis in a cesarean section. Amniotic fluid embolism may mimic the condition. The ability to diagnose and treat such an event as early as possible is necessary in all maternity centers. The selection of antibiotic regimen and the type of anesthesia should be individualized depending upon the existing facilities and the patient's profile, especially in a resource-scarce developing country.

Key words: anaphylaxis, antibiotic prophylaxis, cesarean section, intrauterine death.

Introduction

The reduction of endometritis by two thirds to three quarters and a decrease in wound infections necessitates the use of preoperative or intraoperative antibiotic prophylaxis in cesarean sections.¹ Intra or preoperative anaphylaxis caused by antibiotics or anesthetic drugs may cause significant maternal and perinatal morbidity. Although it is difficult to differentiate clinically between drug-induced severe anaphylaxis with low platelet counts and prolonged clotting time and amniotic fluid embolism, early recognition and prompt diagnosis of anaphylaxis, and aggressive resuscitation including immediate parenteral administration of epinephrine, is necessary to prevent untoward maternal and perinatal outcome. The ability to diagnose and treat is also a requisite in resource-scarce developing countries, as in developed countries.

Case Report

This is a rare case where a 35-year-old, non-diabetic, non-hypertensive (gravida 3, para 2, previous 2 lower

segment cesarean section [LSCS]) woman, with a largely uneventful antenatal period, except a history of occasional wheezing during her antenatal period, was admitted for an elective cesarean section. As per the hospital protocol, one dose of preoperative antibiotic prophylaxis (cefotaxime 1 g IV after test dose) was administered preoperatively, 1 h before the elective surgery. Slow intravenous infusion with plain dextrose solution was also started. After approximately 30 min, mild cough and dyspnea was noted, without any apparent signs of urticaria or angioedema. She had one bout of vomiting. Her condition deteriorated rapidly, with systolic blood pressure falling to less than 70 mmHg, SPO₂ below 90%, and marked fetal bradycardia was noted on fetal Doppler. Immediate active resuscitation was started with rapid infusion of fluids, foot-end elevation, steroids and 100% oxygen via the nose. Epinephrine solution was prepared for intravenous infusion keeping in view the rapid fall in blood pressure. Her condition improved and mean arterial pressure of >90 mmHg was recorded within 20 min of beginning the active resuscitation. Incidentally, the fetal heart disappeared within the next 5–10 min. Lower segment cesarean section was deferred pending

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coagulation profile. Platelet count of less than 70 000 and prolonged clotting time was noted even 12 h after the event. Immediate coagulation profile was not available. Lower segment cesarean section was deferred until just after platelet level increased. However, there was no evidence of any bleeding or ecchymosed patches. She was operated on and her postoperative period was uneventful and normal.

Discussions

At times in clinical presentation, the side-effects of a drug causing a severe anaphylactic reaction can create an emergency situation where it can be difficult to differentiate between the anaphylactic reaction caused by the drugs and amniotic fluid embolism (AFE).² Moreover, in resource-scarce developing countries laboratory diagnostic tests, such as serum mast cell tryptase, sialyl Tn antigen or C3/C4 level to differentiate AFE from drug anaphylaxis, are generally not available.³

Anaphylaxis or any other anaphylactoid reaction, such as AFE, induces sudden severe hypotension, hypoxemia, tissue hypoxia and acidosis. Pathophysiological processes involve intravascular fluid volume depletion, myocardial suppression and increased airways resistance. The mediators such as histamine, tryptase, leukotrienes, platelet activating factor, nitric oxide, inflammatory prostaglandins, and bradykinins produce such cardiopulmonary and hematological effects.⁴ Low platelet level, hemostatic disturbances and/or disseminated intravascular coagulation (DIVC) is seen following severe anaphylactic shock in humans, as well as in rats and rabbits models.^{5,6} Acidosis also impaired coagulation by depleting clotting factors, inhibiting thrombin generation, and affecting clot strength and stability in pigs.⁷

Explanations for sudden intrauterine fetal deaths or early neonatal deaths generally include acute fetal tissue hypoxia, oxidative stress and acidosis and fetal bradycardia. Additionally, in fetal lambs, hypoxia is found to increase the pulmonary vascular resistance at birth by augmenting the synthesis of platelet activating factor (PAC).⁸ Intrauterine fetal hypoxia also triggered coagulation disturbances in asphyxiated neonates.⁹

Thus, the challenge is not only to stabilize the mother as early as possible but also expediate the delivery of the hypoxic and distressed fetus.¹⁰ The situation may become more difficult if the platelet count in the mother starts falling below a critical limit initiating DIVC, a serious condition.¹¹

In addition to initiating active resuscitative measures, prompt intramuscular administration of 0.3–0.5 mL of 1:1000 epinephrine is important to prevent prolonged hypotension, thus reducing the chances of severe maternal morbidity and intrauterine fetal death. The intravenous route can be reserved for extreme emergencies in severely ill patients with inadequate circulation.

Regional anesthesia and intraoperative antibiotic prophylaxis soon after the cord clamping appears to be safer. This not only limits the number of general anesthesia drugs, but the presence of an emergency set up and anesthetist in the operating theatre and postoperative recovery room also ensures greater safety, especially in resource-scarce developing countries. If anaphylaxis occurs where a preoperative antibiotic prophylaxis regimen is followed, an emergency labor room/induction room/operating theatre set up, rapid resuscitative measures, epinephrine infusion¹² and, if it is necessary to expedite the delivery, a crash induction anesthesia, should be made available. The availability of fresh whole blood and blood products such as cryoprecipitate, fresh frozen plasma, and platelet concentrate is also a requisite. The Cochrane review suggested a need for an appropriately designed randomized trial to test the optimal timing of administration (immediately after cord clamping versus preoperative) for its efficacy in preventing the morbidity.¹

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