Successful Anesthetic Management of a Ruptured Aorta Complicating Aortic Dissection following Emergency Cesarean Section: A Case Report

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Abstract
A 30-year-old woman who underwent emergency cesarean section due to fetal distress, in our hospital, was admitted to the intensive care unit for close observation because of tachycardia and suspected sepsis. In the intensive care unit, the patient was reintubated and resuscitation started due to unstable hemodynamics and clinical picture of hypovolemic shock and suspected septic shock. Emergency laparotomy, with a multidisciplinary team of obstetric, general, and vascular surgeons, revealed a large retroperitoneal hematoma and a big 3–4 cm long tear with a ruptured infrarenal abdominal aortic wall. Vascular surgeons resected the dissected and ruptured part of the aorta and repaired it with a synthetic graft. The anesthetic management included massive blood and blood product transfusion, invasive hemodynamic monitoring, management of hypotension, hypertension, and transient pulmonary edema due to aortic clamping and massive transfusion. The patient was further managed in the intensive care unit and underwent multiple surgeries due to anterior abdominal wall wound and sequelae. She was finally discharged home, after 66 days, in stable condition.

Introduction
Cardiac disease was the most common overall cause of maternal death in the Confidential Enquiry into Maternal and Child Health report [1]. The main cardiac causes of death are myocardial infarction, aortic dissection (AoD), and cardiomyopathy [1]. Incidentally diagnosed ascending aortic aneurysm and/or dissection in pregnancy is a rare but potentially fatal event [2]. According to the International Registry of Acute Aortic Dissections, being in the peripartum period of pregnancy confers a risk for AoD of 0.2% [3]. The diagnosis of this condition can be difficult during pregnancy and puerperium due to the wide variety of the symptoms, which is further compounded by pregnancy-related symptoms.
We present a case of nontraumatic spontaneous AoD in a previously healthy pregnant patient. We also reviewed the clinical literature regarding the evaluation and management of a pregnant patient presenting with maternal collapse and suspicion of AoD.

**Case Report**

A 30-year-old woman, Gravida 2, Para 1, had an emergency cesarean section under general anesthesia due to premature rupture of membranes and clinical presentation suggestive of placenta abruption and leaking. The ASA status was 3E, and standard general anesthesia was induced and maintained. Intraoperatively, the patient remained stable hemodynamically with an estimated blood loss of 700 mL, which was replaced with 500-mL packed red blood cells (PRBCs). Postoperatively, the patient was extubated and shifted to the post-anesthesia care unit. She was awake but pale and had tachycardia with normal blood pressure. In the post-anesthesia care unit (ICU), hemoglobin on the arterial blood gases was 9.8 g/dL.

Preoperative investigations revealed a plasma lactic acid of 4.2, high CRP and procalcitonin, and an acceptable coagulation profile. Triple antibiotic regimen was started.

After 2 hours in the ICU, the patient became pale, centrally cyanosed, and drowsy. Over a few minutes, the patient became vitally unstable, tachycardia with heart rate >145, hypotensive with blood pressure (BP) around 85/55, oxygen saturation not recordable. She had cold clammy skin and poor perfusion with capillary refill for more than 3 seconds. On examination, the abdomen was tense with no external bleeding. Immediate resuscitation with crystalloid 1,000 mL bolus followed by 2 units of PRBCs was initiated. Invasive arterial blood pressure monitoring was started and arterial blood gas revealed a drop in hemoglobin with high lactate.

A few minutes later and due to the deteriorating clinical condition, the patient was intubated, a triple lumen central venous catheter was also inserted into the right internal jugular vein, and a 2-Liter normal saline was started as a bolus along with the third unit of PRBCs. An urgent arterial blood gas showed a drop in hemoglobin. Inotropes infusions were adjusted to manage the hypotension.

An urgent bedside ultrasound of the abdomen was done which showed a mild to moderate amount of free fluid in the left adnexa. No clear abdominal wall collection could be detected. The fluid around the spleen was measuring 10 × 1 cm (Fig. 1, 2).

By that time patient was sedated with an infusion of midazolam 5 mg/h, 4th unit of PRBC was started. The patient was shifted to the operation theatre for exploratory laparotomy at around 15:00 hours. ASA classification was 5E due to morbid condition and maternal collapse in the ICU. In the operation theatre, general anesthesia was induced, the patient was on controlled mode ventilation, and maintained with a titrated dose of inhalational anesthetics sevoflurane, oxygen, and nitric oxide 50% each. Analgesia was achieved with fentanyl boluses and later added morphine at the end of the surgery and relaxation was achieved by bolus doses of cisatracurium.

A team of general surgeon and obstetric surgeon, joined shortly after by a vascular surgeon, did exploratory laparotomy. On exploration, 500 mL of free blood was found in the peritoneal cavity. There was a large 10 × 15 cm retroperitoneal hematoma on the left pelvic side under the sigmoid colon. The surgical teams present suspected a ruptured aorta that might be due to dissection or aneurysm. The hematoma was expanding; therefore, the obstetric and general surgeons applied manual pressure on it until the vascular surgeon arrived and the equipment for aortic cross-clamping dispatched.

The first round of massive blood transfusion protocol was activated and transfusions of blood and blood products by rapid infusion started. Hypotension was treated with boluses of phenyl-
ephrine and ephedrine with a titrated dose of dopamine infusion. After improving the hemodynamic parameters, the vascular surgeon explored the retroperitoneum above the aorta. Further rounds of massive blood transfusion protocol were activated and transfused according to clinical need and hemoglobin levels on the arterial blood gas.

The hematoma was evacuated and a big tear around 3–4 cm long with a ruptured aortic wall on the left side of the aorta, just above the iliac bifurcation extending to both iliac arteries, was found. The aorta was clamped just below the left renal vein. Both common iliac arteries were exposed and the bleeding was controlled. The dissected and ruptured part of the aorta was resected and sent for pathology. The pathology report showed membranous pieces showing hemorrhage, scattered acute inflammation, necrosis, and disrupted elastic and collagen fiber in keeping with the clinical history of dissection. Scattered calcification deposits are also seen.

Heparin was started and the aortic cross-clamping lasted for approximately 141 min. The aortic graft was unavailable at our hospital, and a vascular graft was brought from another network hospital. During this period, severe hypertension was managed by labetalol blouses and a titrated dose of glyceryl trinitrate infusion. Metabolic acidosis was managed by sodium bicarbonate. Tranexamic acid was administered in addition to blood and blood products replacement and multiple doses of calcium gluconate for hypocalcemia.

During aortic clamping, the patient developed pulmonary edema characterized by high airway pressure, low oxygen saturation, bilateral pulmonary crepitation on auscultation of the chest, and pink frothy secretion from the endotracheal tube. The pulmonary edema was treated by managing hypertension, high oxygen concentration, high PEEP, and furosemide. The patient responded and improved clinically within 20 min.

Surgical repair was done using an axillobifemoral graft which was the only available one and it was a lifesaving procedure. The graft was 8 mm at the proximal diameter, so the aorta and the graft were fashioned for the proximal anastomosis and used as Cobra head end to end. In the distal part, anastomosis was done on both common iliac arteries as end to end anastomosis using the 2 legs of the graft. The ruptured anterior wall of the aorta was resected. Heparin was reversed at the end of the procedure by protamine sulfate.

Total estimated blood loss during the procedure was 12,000 mL with a urine output of 2,500 mL. Factors contributing to the massive blood loss were the initial uncertain diagnosis of the cause of the shock following cesarean section and also, the lack of CT scans and the immediate unavailability of vascular surgeons or general surgeons in the hospital. The obstetrician started the laparotomy and then the general surgeon and minutes later the vascular surgeon arrived and started the repair. Blood loss was optimally replaced by 20 units of PRBCs, 14 units of plasma, 4 pools of platelets, 30 units of cryoprecipitate.

Postoperative, the patient was shifted to the ICU in stable condition with low dose dopamine and producing adequate urine output. In the ICU, she was sedated by midazolam and fentanyl infusions and ventilated using a controlled mode of ventilation with
continuous invasive monitoring. The next day, the patient was shifted to the ICU in the general hospital under the care of vascular surgeons. Postoperative CT aortogram showed good contrast opacification noted within the aortoiliac bypass graft with no evidence of thrombosis (Fig. 3, 4).

The histopathology report of the aortic wall biopsy showed membranous pieces showing hemorrhage, scattered acute inflammation, necrosis, and disrupted elastic and collagen fiber in keeping with the clinical history of dissection. Scattered calcification deposits were also seen. Further course of the patient included multiple surgeries including relook laparotomies, wound debridement, and negative pressure dressings under general anesthesia without any adverse events.

Finally, the patient was discharged stable after 66 days of hospitalization, fully awake, oriented, on room air, ambulatory, without any neurological or renal sequelae. She reunited with her baby who was discharged from our hospital’s NICU in stable condition.

Discussion

AoD and aortic rupture are rare but life-threatening conditions that can result in death if not treated promptly. Most AoDs occur due to essential hypertension. Pregnancy-related vascular emergency is uncommon but a potentially fatal complication [4]. In such patients, studies often reflect underlying prevalent but undiagnosed or undocumented connective tissue disorders, or they may indicate that the physiologic changes of pregnancy may trigger aortic injury even in otherwise healthy women. The mechanism of pregnancy-related aortic complications is uncertain. Pregnancy and the postpartum period are associated with hemodynamic changes, such as increased heart rate, stroke volume, cardiac output, and left ventricular dimensions that may affect the forces acting on the aortic wall. This may be exaggerated by the increased outflow resistance in the distal aorta due to the compression by the gravid uterus [5, 6]. AoD usually occurs in the third trimester due to the hyperdynamic circulation and the hormonal effect on the vessels. It was also found to occur in all stages of pregnancy and in the weeks following delivery [5]. A cohort crossover analysis in which the risk of AoD or rupture for each pregnant woman was compared to her risk during the equivalent period 1 year later revealed that the risk of AoD or rupture is elevated during pregnancy and the postpartum period. This study also found that the absolute risk of AoD or rupture attributable to pregnancy was approximately four per million pregnancies [4]. Patients with acute AoD, aortic rupture, ruptured aortic aneurysms, thoracic aortic trauma, and acute lower limb ischemia are at high risk of perioperative morbidity and mortality [7]. Vascular emergency patients are particularly challenging to the anesthetist especially due to the inadequate time to evaluate and optimize their preoperative state. If possible, a focused preanesthetic evaluation should include checking for allergies, medications, and cardiac history in addition to routine blood tests, arterial blood gases, and ECG. According to the American College of Cardiology/American Heart Association 2007 perioperative evaluation and care for noncardiac surgery guidelines [8] and the 2009 update [9], major vascular surgery is ranked as high risk even in the elective setting.

Minimal intraoperative anesthesia monitoring includes noninvasive blood pressure, pulse oximeter, and an ECG. Invasive arterial blood pressure monitoring is optimal but should not delay the repair. In emergency cases, it can be obtained after the aorta is cross-clamped [7]. Central venous access is decided based on each case. Evidence from random controlled trials examining the risk-benefit of pulmonary artery catheters found no added mortality and equally no benefit [10].

Major blood loss is a complication in most open operations on the aorta. This blood loss is secondary to aortic rupture, disruption of retroperitoneal vessels, and coagulopathy. Coagulopathy can occur due to hypothermia, acidosis, massive blood loss, and fibrinolysis caused by the aortic cross-clamping.

In a review article by Ellard and Djaiani [7], they recommend for managing patients with vascular emergency operations, to maintain a hemoglobin concentration more than 9 g/dL, INR of less than 1.5, and platelets more than $10^9/L$ [10]. Thromboelastography can be used in addition to coagulation tests. End-organ damage after aortic cross-clamping is caused by hemodynamic changes in preload, afterload, and cardiac output as well as humoral response caused by the release of mediators which cause an ischemia-reperfusion injury [11].

Anesthesia management of open aortic repairs should include strategies to offset the hemodynamic as well as humoral effects of aortic cross clamping and unclamping. Hypertension following aortic cross-clamping can be managed using venodilators like nitroglycerin. Venodilators reduce preload and allow fluid loading before unclamping. Before unclamping, communication between surgeons and anesthetists is vital. Venodilators should be stopped; anesthetists should optimize volume replacement and treat acidosis. Hyperventilation, calcium, bicarbonate, vasopressors, and fluids are all tools for managing the effects of unclamping [11, 12]. Gradual release of the cross-clamp reduces the effects of reperfusion injury [12]. Postoperative ICU admission is usually recommended.
for hemodynamic and ventilatory management as well as treatment of other complications like coagulopathy and end-organ support.

**Statement of Ethics**

A written informed consent was signed and obtained from the patient to publish the case and any images. Ethical approval is not required according to the Dubai Health Authority committee policies.

**Conflict of Interest Statement**

The authors have no conflicts of interest to declare.

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**References**

1 Confidential Enquiry into Maternal Deaths (Great Britain). Saving mothers’ lives: reviewing maternal deaths to make motherhood safer—2003–2007: the seventh report of the confidential enquiries into maternal deaths in the United Kingdom. Confidential enquiry into maternal and child health. 2007.

2 Elkayam U, Rose J, Jamison M. Vascular aneurysms and dissection during pregnancy. In: Elkayam U, Gleichen N, editors. Cardiac problems in pregnancy. New York, NY: Alan R. Liss; 1990. p. 215–29.

3 Isselbacher EM, Eagle KA. Epidemiology of thoracic aortic aneurysms, aortic dissection, intramural hematoma, and penetrating atherosclerotic ulcers. In: Baliga RR, Nienaber CA, Isselbacher EM, Eagle KA, editors. Aortic dissection and related syndromes. New York, NY: Springer; 2007. p. 1–15.

4 Kamel H, Roman MJ, Alex Pitcher, Devereux RB. Pregnancy and the risk of aortic dissection or rupture. A cohort-crossover analysis. *Circulation*. 2016;134(7):527–33.

5 Immer FF, Bans R, Immer-Bansi AS, McDougall J, Zehr KJ, Schaff HV, et al. Aortic dissection in pregnancy: analysis of risk factors and outcome. *Ann Thorac Surg*. 2003;76:309–14.

6 Ohlson L. Effects of the pregnant uterus on the abdominal aorta and its branches. *Acta Radiol Diagn*. 1978;19(2):369–76.

7 Ellard L, Djaiani G. Anaesthesia for vascular emergencies. *Anaesthesia*. 2013;68(Suppl 1):72–83.

8 Fleisher LA, Beckman JA, Brown KA, Calkins H, Chaikof E, Fleischmann KE, et al. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: executive summary: a report of the American College of cardiology American heart association task force on practice guidelines. *Circulation*. 2007;116:1971–96.

9 Fleischmann KE, Beckman JA, Buller CE, Calkins H, Fleisher LA, Freeman WK, et al. 2009 ACCF/AHA focused update on perioperative beta blockade. A report of the American College of Cardiology Foundation/American heart association task force on practice guidelines. *Circulation*. 2009;120:2123–51.

10 Sandham JD, Hull RD, Brant RF, Knox L, Pinoe GF, Doig CJ, et al. A randomized, controlled trial of the use of pulmonary-artery catheters in high-risk surgical patients. *N Engl J Med*. 2003;348:5–14.

11 Zammert M, Gelman S. The pathophysiology of aortic cross clamping. *Best Pract Res Clin Anaesthesiol*. 2016 Sep 1;30(3):257–69.

12 Gelman S. The pathophysiology of aortic cross-clamping and unclamping. *Anaesthesiology*. 1995 Apr;82:1026–57.