Case Report

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Case of Cardiorespiratory Arrest following Removal of Cervical Cerclage

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Abstract

We report a case of cardiorespiratory arrest following the removal of a cervical suture in a non-labouring woman.

Keywords: Amniotic fluid embolism, Cervical cerclage, Cervical suture

Abbreviations: AFE: Amniotic Fluid Embolism; DIC: Disseminated Intravascular Coagulation; T2DM: Type 2 Diabetes Mellitus; eGFR: estimated Glomerular Filtration Rate; ICU: Intensive Care Unit; CPR: Cardiopulmonary Resuscitation; LVEF: Left Ventricular Ejection Fraction; CCU: Coronary Care Unit

Case Presentation

A 30-year-old gravida 5 para 1, was booked for removal of cervical cerclage as an outpatient at 37 weeks gestation at a tertiary institution in Canberra. She had three consecutive first trimester miscarriages and one preterm vaginal birth at 28+1 completed weeks of gestation. With this pregnancy, a cervical suture was placed at 13 weeks with no complications. Her past medical history is significant for methyltetrahydrofolate deficiency mutation (homozygous), poorly controlled Insulin dependent Type 2 diabetes mellitus (HbA1c of 11.5%), essential hypertension, hypothyroidism, endometriosis and polycystic ovarian syndrome. The conditions are managed with Labetalol (100 mg tds), le vemir insulin (32 units nocte), Metformin (1000 mg bd), novorapid (32 units mane 60 units midi 32 units nocte) clexane (40 mg daily), thyroixine (100 mcg). She had two previous laparoscopies under general anaesthesia in 2015 for endometriosis with no adverse outcomes. She was admitted for removal of cervical cerclage at 37 weeks, however she did not tolerate the procedure as an outpatient, and thus was booked for removal under sedation two days later.

The patient arrived in day surgery fasted, and was sedated with midazolam 2 mg, fentanyl 50 mcg and given metoclopramide 10 mg, dexamethasone 4 mg and ondansetron 4 mg. During the procedure, the patient was quite agitated, but settled subsequently. Following the procedure, she was observed routinely in Recovery, where she became acutely short of breath and hypoxic. An arterial blood gas showed PaO2 of 54 mmHg, metabolic acidosis with a pH of 7.39, HCO3 of 18.2 mmol/L, base excess of -9.2 mmol/L, lactate of 2.4 mmol/L, potassium of 5.4 mmol/L, sodium of 130 mmol/L and a troponin rise to 2055 ng/L. Coagulation studies were normal (PT of 11, APTT of 25) except for a fibrinogen of 6.1 (high). She had an associated renal impairment with a sudden drop in her eGFR from >90 to 77 and subsequently 59 with an associated rise in creatinine from 60 mmol/L to 89 mmol/L and subsequently 109 mmol/L. Chest X-ray showed diffuse opacification of both lung fields. A diagnosis of amniotic fluid embolism was suspected and decision was made for an emergency caesarean section under general anaesthesia.

On return to theatre, the patient received 80 mg of rocuronium, 180 mg of propofol for induction and despite
being ventilated with 100% oxygen, had a relative hypoxia (Sats 85-90%). The procedure was uncomplicated; the baby was delivered in good condition and the total estimated blood loss was 800 ml. She received 100 micrograms of carbetocin and 10 units of syntocinon post-delivery. During the procedure she remained haemodynamically stable, however at the end became hypotensive with a systolic blood pressure of 100 mmHg, during which time a metaraminol infusion was commenced at 5 ml/hr. Post-operatively, she was transferred to ICU intubated and ventilated. On arrival in ICU, became increasingly cyanosed despite being easily ventilated. Her oxygen saturations then dropped to 60%, she became hypotensive and bradycardic to 37 beats per minute and progressed to cardiac arrest. Cardiopulmonary resuscitation (CPR) was commenced immediately and arterial blood gas results showed hyperkalaemia with a potassium of 10 mmol/L. She received 2 cycles of CPR and the hyperkalaemia was managed with insulin, dextrose, calcium gluconate, bicarbonate, which allowed return of spontaneous circulation.

During her admission in ICU, following the cardiac arrest, the patient developed severe cardiogenic shock, was treated for suspected sepsis with IV antibiotics (Piperacillin/Tazobactam) and developed oliguric renal failure which was treated with continuous renal replacement therapy. The first bedside transthoracic echocardiogram (TTE) showed normal left ventricular size, severe global systolic dysfunction with a left ventricular ejection fraction (LVEF) of 10%, a small pericardial effusion and no valvular disease. She was subsequently commenced on a dobutamine infusion. The repeat TTE two days later showed moderate segmental systolic dysfunction with an LVEF of 35-40% and these findings were the same on the following TTE one week later. She was then discharged to the Coronary care unit (CCU) for cardiac monitoring. In CCU, the patient had a persistent tachycardia (managed with initially metoprolol and subsequently carvedilol) and pulmonary oedema (managed with frusemide). Her clinical condition improved significantly and she was discharged home day 8 post-operatively with a plan for follow-up with cardiology in 4 weeks and Gynaecology clinic in 6-8 weeks.

Discussion

We report an interesting case of cardiac arrest following removal of a cervical cerclage. Several differential diagnoses were considered including Amniotic fluid embolism (AFE), myocardial infarction, pulmonary embolism and pre-eclampsia. It is difficult to determine the true cause of the patient's sudden unexpected cardiorespiratory compromise. One theory is that the patient's long history of poorly controlled type 2 diabetes is likely to have resulted in micro and macrovascular disease which may have predisposed her to a cardiac event in the setting of a general anaesthetic and surgical procedure. Another possibility is a pulmonary embolism, although given the patient's rapid recovery without anticoagulation therapy makes this diagnosis less likely. The diagnosis of pre-eclampsia was also considered; however the patient did not experience hypertension in the antenatal period. Thus, although atypical, it is also important to consider the diagnosis of AFE.

Amniotic fluid embolism (AFE) is a rare and life-threatening obstetric complication characterized by profound cardiovascular collapse, altered mental status and disseminated intravascular coagulation (DIC). The incidence reported is between 1-12 cases per 100,000 deliveries, with a high mortality rate ranging from 13.3-40% [1]. Two large population-based retrospective cohort studies found that AFE was associated with increased maternal age (>35 years), caesarean delivery, instrumental delivery, placenta praevia, placental abruption, eclampsia and foetal distress. Other risk factors included polyhydramnios, cervical laceration, uterine rupture [1,2]. Interestingly, only one case of non-fatal AFE was reported following removal of a cervical suture [3].

Given the rarity of AFEs, the diagnostic criteria has been recently debated. The Australian diagnostic criteria for AFE can be either clinical (acute hypotension or cardiac arrest, acute hypoxia or coagulopathy in the absence of any other potential explanation of signs and symptoms) or pathologic (presence of foetal squames or hair or debris in maternal pulmonary circulation) [4,5]. Since then, Clark and colleagues, together with the Society of Maternal Fetal Medicine and the Amniotic Fluid Embolism Foundation reviewed the definition of AFE and concluded that AFE is characterized by four specific criteria that includes the following - sudden onset cardiorespiratory arrest or both hypotension and respiratory compromise, documentation of overt DIC, clinical onset during labour or within 30 minutes of delivery of the placenta and the absence of fever [6,7].

The true pathophysiology of AFE is unknown however it is thought to be caused by an abnormal activation of immunologic mechanisms by foetal antigens in the maternal circulation resulting mast cell degranulation and

complement activation and a subsequent anaphylactoid or systemic inflammatory response syndrome [8]. Evidence suggests that the haemodynamic changes in AFE result initially from increased pulmonary vascular resistance and right ventricular failure followed by left ventricular failure. Clarke and colleagues suggested that ischemic myocardial injury as well as a potential depressive effect of amniotic fluid on the myocardium as the cause of the profound and rapid left ventricular failure based on animal studies [9]. In some cases of AFE, the coagulation cascade is activated resulting in DIC. This mechanism is thought to be multifactorial - in vitro, amniotic fluid decreases clotting time and induced platelet aggregation as well as the extrinsic clotting pathway resulting in the activation of factor X with a subsequent consumptive coagulopathy [10]. Renal failure has also been widely reported in cases of AFE [11,12]. Ihara and colleagues described a case of AKI in a patient with AFE which was successfully treated with continuous renal replacement therapy [11]. The precipitating factor responsible for acute kidney injury in AFE is likely ischemic, secondary to cardiogenic shock resulting from hypoperfusion of the kidneys and subsequent acute tubular necrosis. Bilateral renal cortical necrosis (a progression of acute tubular necrosis) may develop as a result of hypotension and DIC in severe cases of AFE [13].

There is no reliable evidence on the reoccurrence rates of AFEs. A total of 9 cases of successful pregnancy have been reported following AFE with no reoccurrence of the condition [14-16]. The lack of concrete evidence makes it challenging to counsel these patients, however the traumatic nature of the clinical condition impacts the future reproductive decisions of these women [17,18].

We report an interesting case of sudden cardiorespiratory collapse following the removal of a cervical cerclage in a non-labouring woman. Her clinical deterioration was severe, sudden in onset and culminated in cardiorespiratory arrest and acute tubular necrosis which were managed with vigorous and prompt supportive therapies. Unfortunately, her cardiac function prior to discharge only improved to an LVEF of 40%. We believe that underlying microvascular disease resulting from her poorly controlled insulin dependent type 2 diabetes mellitus, would have predisposed her to a greater degree of cardiac and renal injury in the setting of this acute event. The true cause of this event is unknown although the case had several similarities to an AFE. Certainly, we appreciate that the lack of clinical evidence of DIC makes the diagnosis of AFE less likely given the recent changes to the diagnostic criteria. Biopsy examination of renal vasculature or post-mortem examination would have further aided in confirmation of the diagnosis however this was not necessitated as the patient made a full recovery following delivery and did not consent to further invasive investigations.

Conclusion

We report a case of cardiorespiratory arrest following removal of cervical cerclage in a non-labouring woman. The patient experienced cardiac arrest, hypotension, respiratory distress and renal dysfunction - all features associated with the diagnosis of AFE. However, given the lack of features of DIC and further histopathological investigation, the diagnosis of AFE cannot be confirmed.

Declarations

Ethics approval and consent to participate

The patient has given verbal consent to participate.

Consent for publication

The patient has given verbal consent for publication of this case report.

Availability of data and material

The data and clinical information were obtained while the patient was an inpatient through clinical interactions, notes and post-discharge through medical records.

Competing interests

We have no conflicts of interest to declare.

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Authors’ contributions

Collation of information and drafting of case: M. Raheem, R. Orefice.

Literature review and drafting of discussion: M. Raheem.

Critical revision: R. Orefice.

Re-edit: M. Raheem.

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