Intraoperative Cardiac Arrest during Cesarean section – A case report

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ABSTRACT

Objective: Cardiorespiratory arrest can occur suddenly even in healthy patients with unknown co-morbidity. Pulmonary embolism is a known though rare complication of cesarean section. Massive intra operative pulmonary embolism complicated with disseminated intravascular coagulation poses a challenge to the clinician. This report describes the resuscitation and subsequent management of a patient who suffered an intra operative cardiac arrest. She was operated and managed post operatively at GMCH

Material and Methods: A 36 years lady underwent emergency caesarean section under spinal anaesthesia, the indication being decreased fetal movements.

Surgical and medical history were not significant. Preoperatively her general and physical examination as well as her laboratory parameters did not reveal any abnormality. Within a minute of the baby extraction the patient developed pulseless electrical activity. Cardiopulmonary resuscitation was initiated immediately and continued as per the AHA guidelines. Return of spontaneous circulation occurred after 3 minutes and post resuscitation care was continued.

A provisional diagnosis of pulmonary embolism with DIC was made. Echocardiography and CT scan confirmed the presence of thrombus in the right main pulmonary artery and the right atrium. The immediate post operative blood reports revealed severe anaemia with thrombocytopenia and deranged coagulation profile. Doppler study of the lower limbs revealed thrombus at the right saphenofemoral junction with superficial thrombophlebitis

Result: Post operatively, she received mechanical ventilation, inotropic support and blood component therapy. She was extubated on Day 2. By this time her blood counts and coagulation profile had returned to normal values. On Day 11 she was discharged to the ward and then discharged home.

Conclusion: Outcome from cardiac arrest depends on immediate diagnosis and initiation of resuscitation. Post resuscitation period complicated by DIC requires multispecialty involvement. The key points in management are prompt diagnosis, timely intervention and supportive therapy with blood components.

Key words: Intra operative cardiac arrest; cesarean section; DIC; pulmonary embolism; management;
INTRODUCTION

Intraoperative sudden cardiac death in patients undergoing elective non-cardiac surgery is rare. Immediate diagnosis of the cause is mandatory for successful resuscitation and management of these patients. Acute pulmonary embolism, though a major cause of maternal mortality, is a relatively rare complication during the perioperative period. Massive pulmonary embolism (PE) is known to be an eliciting factor for disseminated intravascular coagulation (DIC)\(^1\). This clinical scenario of massive pulmonary embolism complicated by DIC with the initial presentation of cardiac arrest in the intraoperative period presents the treating physician with difficult management decisions and clinical dilemmas since the traditional treatment options of thrombolysis and embolectomy, both pose a threat to the patient.

CASE REPORT

A woman 36 years old, 39 weeks pregnant, with BMI of 34.8 kg/m\(^2\) diagnosed as G\(_2\)P\(_1\) with mild Pregnancy induced Hypertension (PIH) was admitted for induction and delivery. She was subsequently scheduled for emergency cesarean section due to decreased fetal movements. Her surgical history included thyroidectomy 17 years earlier and later a diagnostic laparoscopy. Both procedures were carried out under general anaesthesia and were uneventful. She was diagnosed with mild PIH in the present pregnancy for which she was not on any medication. She had history of dust allergy for which she took puffs as and when required, the last episode being about one year back. Her thyroid function tests done every 6 months were all normal.

Her preoperative evaluation revealed her vitals, general examination and investigations to be within normal limits. Written explained consent for spinal anaesthesia was obtained and patient was taken for lower segment cesarean section.

The surgery proceeded well till the extraction of the baby, within a minute of which she suddenly became unresponsive. ECG monitor showed a normal sinus rhythm with a heart rate of 52/minute. At that instant the carotid pulse was absent and there was no breathing. A diagnosis of Pulseless electrical activity (PEA) was established and cardiopulmonary resuscitation (CPR) was initiated immediately as per the AHA 2010 guidelines\(^2\). Return of spontaneous circulation occurred after 3 minutes of CPR, and post cardiac arrest care was provided which included mechanical ventilation and noradrenaline infusion.

Urgent Intraoperative transthoracic echocardiograph revealed D shaped interventricular septum, moderate tricuspid regurgitation, mild pulmonary hypertension, with end systolic pulmonary artery pressure(ESPAP) 46mmHg (Normal valve <25mmHg) and a possible mobile right atrial(RA) mass. The most likely cause of the cardiac arrest was thus established to be acute pulmonary embolism (PE) with RA thrombus. Meanwhile, the patient started bleeding excessively at the surgical site indicating the possibility of disseminated intravascular coagulation (DIC).

Postoperatively, an immediate CT scan chest with contrast confirmed the presence of embolus in the right main pulmonary artery as well as one of its distal branches. Left third order pulmonary artery branches emboli were also visualized.

Mechanical ventilation and inotrope infusion were continued. The immediate post operative blood reports showed anaemia with thrombocytopenia and deranged coagulation
profile. The arterial blood gases indicated metabolic acidosis with hypoxia and hypokalemia. D-dimer levels were markedly increased (1600ng/ml). Coagulopathy was managed by transfusion of blood products. Within the first 12 hours the patient had received 8 units each of packed cells, fresh frozen plasma (FFP) and platelet concentrates and by 24 hours 14 packed cells, 21 FFP and 16 platelet concentrates had been administered. Inotropic support was gradually withdrawn on Day 2 and having met the extubation criteria, she was extubated. Within 36 hours her blood counts, coagulation profile and blood gases too had returned to normal values (Table 1).

<table>
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<th>24/3 Post-op</th>
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<th>25/3 6:00p</th>
<th>26/3</th>
<th>27/3</th>
<th>28/3</th>
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Subsequent investigations included Transoesophageal echocardiography (TEE) which showed the presence of finger like freely mobile right atrial thrombus with intact interatrial septum (Figure 1). Lower limb Doppler study revealed presence of thrombus at right saphenofemoral junction with superficial thrombophlebitis.

![Transoesophageal Echocardiography](image)

**Figure 1**: Trans-oesophageal Echocardiography on Day 1 showing finger like freely mobile right atrial mass

By Day 3 the patient was comfortable and maintaining normal parameters on room air. She was allowed a soft diet. Infusion of low dose (500 units/hour) unfractionated Heparin was initiated and gradually increased over the next 3 days to 1300 units/hour.
On the 3rd day she had an episode of partial seizure which resolved spontaneously. Simultaneously her blood pressure too increased. A presumptive diagnosis of Pre-eclampsia/Eclampsia was established and she was prescribed Phenytoin and Magsulf bolus followed by infusion for 24 hours. Antihypertensives, namely Labetalol and Adalat were also introduced. CT brain did not exhibit any significant abnormality.

After this the patient showed a steady recovery. Phenytoin and Labetalol were discontinued after 3 days. Bisoprolol and Nifedepine were prescribed. Anticoagulant therapy with low molecular weight heparin (enoxaparin) was initiated on the 9th day postoperatively. On Day 10 Transthoracic echocardiogram showed complete resolution of the thrombus and normal pulmonary pressures with good left ventricular function (Figure 2). The lower limb Doppler on the same day also showed resolution of the previous thrombi.

The patient was then ambulated and transferred to the ward the following day. There she continued to show good progress and was discharged home on the 15th postoperative day. She attended the medical outpatient clinic a week after discharge and was doing well. She has since travelled to her native country and lost to follow up.

**DISCUSSION**

The incidence of venous thromboembolism during pregnancy and post partum ranges from 0.5 to 3.0 per thousand pregnancies and that of PE is 0.5 per 1000 deliveries\(^3,4\). Cesarean section has 10 times higher incidence of PE than vaginal delivery\(^5\). This is attributed to various physiological changes of pregnancy including increased procoagulant activity, enhanced platelet turnover and aggregation, resistance to protein S and protein C as well as increased concentration of fibrin degradation products. Other factors which place the obstetric patient at risk are venous stasis, vascular trauma, separation of placenta and multiple gestation.

Sudden tachycardia, chest pain, cough, unexplained loss of consciousness and/or haemoptysis raise the suspicion of PE, while hypoxaemia, haemodynamic instability, syncopal episode and/or cyanosis are characteristic of massive PE\(^6,8\).

Massive pulmonary thromboembolism is the term used to signify the presence of a clot in the pulmonary vascular tree that causes right ventricular failure, and constitutes
10% of all the variety of presentations of venous thromboembolism. It is defined as acute PE with sustained hypotension (systolic BP< 90mmHg) for atleast 15 minutes or requiring inotropic support (not due to a cause other than PE, such as arrhythmia, hypovolemia, sepsis, or left ventricular dysfunction), pulselessness, or persistent profound bradycardia (heart rate < 40bpm) with signs and symptoms of shock. In our patient PE presented as circulatory collapse and cardiopulmonary arrest which was indicative of a massive embolus.

Regarding diagnosis of acute PE, electrocardiogram and chest X-ray are of limited value, often being normal. D-dimer testing is a very sensitive measurement of fibrinolytic activity but not specific enough to be diagnostic of pulmonary embolism. Transthoracic echocardiography is of particular use in identifying patients with a large PE, in whom the pressure of the pulmonary circulation is elevated (90% sensitivity). In these patients we can see dilatation of the right ventricle and hypokinesis of its free wall, while at the same time the shape of the left ventricle changes, because of the displacement and flattening of the interventricular septum, adopting a D shape on its short axis during both systole and diastole. A characteristic sign is the maintenance of mobility of the apical region of the right ventricle known as McConnell sign, in contrast to the hypokinesis of the entire free wall that is observed in chronic pulmonary hypertension. Echocardiography performed in this patient too demonstrated the classical D-shaped septum and increased ESPAP.

Transoesophageal echocardiography (TEE) is a useful diagnostic modality for patients suffering cardiac arrest in non-cardiac surgery. Being a bedside examination, it is considered especially useful in patients with haemodynamic instability where moving them away from the unit in order to perform CT, MRI, etc., is difficult. Apart from the aforementioned findings, transoesophageal echocardiography can reveal the existence of thrombus in the main trunk or the pulmonary arteries in 80% of cases with massive PE. It also helps to identify surgically correctible conditions and therefore initiate the therapeutic interventions immediately after surgery. We too found TEE to be an extremely helpful tool for establishing diagnosis of RA thrombus and PE in this patient.

Perfusion lung scanning is a basic non-invasive examination for the diagnosis of PE and is classified as high probability, intermediate probability and normal.

While pulmonary angiography has been the gold-standard for establishing the diagnosis of acute pulmonary embolism for decades, it is rarely done nowadays.

Spiral computed tomography (CT) of the lung, with intravenous infusion of radiopaque medium, has been widely used in recent years for the diagnosis of PE and has good sensitivity and specificity in the identification of emboli in the large pulmonary arteries (80% and 90%, respectively), but is less successful in the peripheral vascular tree— beyond the third level of branching in the pulmonary circulation. The new, multi-slice tomographic devices seem to be a great improvement in this respect. In a recent multi-centre study that used multi-slice CT, the sensitivity and specificity were substantially better (83% and 96%, respectively). A good quality CT Angiogram that is negative for acute pulmonary embolism essentially rules out the diagnosis and specificity is excellent.
Magnetic resonance pulmonary angiography with gadolinium may prove to be particularly safe and useful in the future, since apart from anatomical characteristics it provides information about right ventricular wall motion.

Recent data suggest that mortality due to acute pulmonary embolism is higher in the setting of residual deep venous thrombosis, so that evaluating the legs in acute PE is another important investigation.

In summary, the diagnosis of acute pulmonary embolism requires an integrated approach, often involving more than one test and at least one imaging modality.

As seen in our patient a vast majority (95%) of acute pulmonary embolism cases originate from thrombi in the leg or pelvic veins, although emboli may arise from other sources such as the axillary subclavian system or the renal veins. Death from acute pulmonary embolism is caused by right ventricular failure. When thrombosis propagates from the calf veins to the larger more proximal veins, or originates more proximally, the likelihood of embolization, as well as the impact on the lungs, increases. As the embolic burden increases, right ventricular afterload increases and there is right ventricular dilation and hypokinesis associated with the increased pulmonary vascular resistance. When the clot burden reaches a critical threshold, the right ventricle is unable to generate enough force to achieve an adequate cardiac output and fails, resulting in hypotension and cardiac arrest.

DIC is characterized by systemic activation of coagulation, resulting in the generation of fibrin clots, concomitant activation of platelets and coagulation factors. The depletion of clotting factors and acute thrombocytopenia may lead to bleeding, while the fibrin clots and damage to microvasculature may result in organ failure. Several reports have previously described the correlation between massive PE and DIC.

Leitner et al have hypothesized that patients with massive PE develop a state of consumptive coagulopathy, in particular in PE leading to cardiac arrest. They have further hypothesized that parameters that are known to decrease in DIC, such as platelet count and fibrinogen, would be decreased in resuscitated PE patients as compared with PE patients without CPR, while D dimer and prothrombin time would be increased.

An alternate theory has been proposed by Gando. This states that the cardiopulmonary collapse and resuscitation as a consequence of embolus rather than the pulmonary embolism itself could be responsible for activation of coagulation. This may be due to systemic inflammatory response resulting from ischaemic reperfusion in patients with circulatory arrest/hypoxia.

Anticoagulation is the initial management for hemodynamically stable patients whereas thrombolytics would be the preferred modality in unstable patients.

Current guidelines restrict surgical embolectomy to situations when thrombolysis has failed or in extremely compromised patients who are not candidates for thrombolytic therapy, either due to their risk of bleeding or to their critical state. A variant of this procedure is the thromboendarterectomy which may be performed in patients with recurrent PE, but both these procedures are associated with very high mortality.

RA thrombus occurs in 4-8% of patients with acute PE. These may be categorized as Type A which consists of long, thin, worm like mobile thrombi associated with high early mortality. Type B consists of immobile, non specific thrombi with absence of associated PE in 60% of cases and low early mortality. Type C constitute a
small proportion of thrombi which are intermediate in character, being mobile but not worm like in shape and have the potential to obstruct right atrial or ventricular outflow\textsuperscript{39,40}. Thrombolysis is suggested for Type A, while type B may be treated by anticoagulation alone and surgical embolectomy is the optimal treatment for Type C thrombus. If this is not available anticoagulation alone is suggested as a reasonable approach\textsuperscript{41}. Our patient exhibited a type A thrombus which ideally would need thrombolysis. But the coexisting DIC was a contraindication for use of thrombolytics, and thus after correcting the coagulopathy we initiated low dose heparin infusion for our patient and later replaced by low molecular weight heparin.

Providing appropriate treatment depends largely on risk stratification of pulmonary embolism. Though in the extreme settings of pulmonary embolism presenting with circulatory collapse or respiratory failure, as in our situation, risk stratification may simply consist of proof of pulmonary embolism and documentation of significant hypotension. Treatment combines symptomatic interventions to reverse hemodynamic instability and respiratory failure and treatments designed to decrease pulmonary vascular obstruction rapidly\textsuperscript{22,26}.

Even though several guidelines on management of acute pulmonary embolism have been published\textsuperscript{10,33,34}, guidance for various scenarios which challenge the physician in management of acute PE are often not easily accessible in the guidelines. There are no well defined guidelines for the management of patient having concurrent massive PE and DIC. Massive PE leading to cardiopulmonary collapse becomes an indication for thrombolytic therapy but that was not considered here in this case due to underlying coagulopathy; but the institution of IV Heparin in a controlled setting led to dramatic improvement. We need further studies to explain the exact mechanisms leading to DIC in such patients and to guide appropriate therapy.

REFERENCES


