CASE REPORT

Cardiovascular Collapse Under Spinal Anesthesia

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Abstract

A 20 years old ASA I full term primigravida in labor, underwent an emergency lower segment cesarean section under spinal anesthesia, the indication being fetal distress. Immediately following delivery of fetus, she complained of severe breathlessness and suffered a cardiovascular collapse. A presumptive diagnosis of high spinal anesthesia was made and she was managed accordingly. Thirty minutes later she developed angioedema which increased in severity over the next two hours and a diagnosis of anaphylactic reaction was made. The patient responded to intravenous adrenaline and recovered over next 24 hours.

The aim of this case report is to discuss cardiovascular collapse and its etiological factors, such as anaphylaxis, amniotic fluid embolism and high spinal anesthesia during cesarean section.

Keywords: Cesarean section, high spinal anesthesia, anaphylaxis, amniotic fluid embolism.

INTRODUCTION

Cardiovascular collapse under any kind of anesthesia is a dreaded complication and the diagnosis is more often than not elusive. We present one such case of a patient who suffered a massive cardiovascular collapse under spinal anesthesia, just following cesarean delivery of her baby. We have discussed the most common conditions that could lead to such a catastrophe.

CASE REPORT

A 20 years old primigravida in labor was taken up for an emergency lower segment cesarean section for fetal distress. She was a healthy young adult with no history of medical illnesses, surgical interventions or any allergy in the past. Her present pregnancy was uneventful and she had regular antenatal check-ups. She was averagely built with a weight of 60 kg. On examination she well hydrated, had a normal volume pulse rate of 85 per minute and a blood pressure of 120/78 mm Hg right arm supine. The cardiovascular, respiratory and central nervous systems were normal and per abdominal examination revealed a full term pregnancy with cephalic presentation. The fetal heart rate was 120 per minute. She was accepted as an ASA I E case.

The patient was preloaded with 500 ml ringers lactate (RL) via an 18G intravenous cannula. Following an aseptic technique, 2.2 ml of 0.5% hyperbaric bupivacaine was slowly injected using a 25 gauge Whitaker spinal needle in the Lumbar 2-3 subarachnoid space with the patient in the sitting position. She was thereafter positioned supine with a left lateral tilt and administered oxygen by face mask at a rate of 5 liters per minute. Continuous pulseoximetry and electrocardiography was monitored. Noninvasive blood pressure monitoring was performed every 3 minutes.

Surgery was started immediately and in 5 minutes a live and healthy female baby was delivered. 0.2 mg of Inj Methyl ergometrine was administered intravenously. The patients pulse was 100 per minute and arterial pressure was 98/60 mm Hg. 6% hydroxyethyl starch was started.

The patient soon complained of severe difficulty in breathing. She became intensely restless and agitated. The blood pressure was noted to be 78/40 mm of Hg. She was administered 100% oxygen. 3 mg bolus of Inj mephenteramine was given IV and the infusion rate of hydroxyethyl starch was increased. Despite repeated doses of mephenteramine and fluid infusion the arterial pressure was not recordable and the surgeon noted an extremely pale uterus.

The ECG monitor initially displayed bizarre complexes with motion artifacts while pulseoximeter readings were extremely low and unreliable. On ausculation feeble distant heart sounds were heard. Air entry was decreased bilaterally. The patient was unresponsive. She was intubated with a 7.0 mm cuffed endotracheal tube. Intubation was easy and nontraumatic. There was an increased resistance to air entry on ventilating the patient and there was decreased air entry to the both lungs. Wheezes were heard in all lung zones. The SpO_2 was between 75 and 80%. The pupils were dilated and sluggishly reacting to

light. The tongue and extremities were cyanosed. The ECG monitor displayed a heart-rate of 160 per minute. No blood pressure was recordable. A diagnosis of high spinal block was made.

The patient was placed in Trendelenberg position. 1000 ml of hemaccel and 1000 ml of RL was infused using a pressure infuser. 200 mg Inj hydrocortisone was administered IV. There was no respiratory effort for 10 minutes. Thereafter spontaneous respiration started and the patient continued to receive 100% oxygen. Inj Prostadin was given on two occasions to ascertain uterine contraction. Over the next 10 minutes the patient started to respond to verbal communication and was able to move her upper limb and head. The chest had vesicular breath sounds with no evidence of bronchospasm. The surgery had been completed and since the patient had started maintaining SpO_2 of 98% she was extubated.

It was observed that the patient had developed angioedema of lips. The patient was now conscious and responding to verbal commands. She was shifted to the ICU. Her heart-rate persisted to be around 160 per minute and the arterial pressure was not recordable. The patient had gross angioedema and discomfort in her throat. She was conscious but restless. Anaphylactic shock was considered as the possible cause. 3 ml of 1 in 10000 inj adrenaline was injected IV in divided doses. The arterial pressure increased and was recorded to be 140/90 mm Hg. The pulse rate decreased to 130 per minute. Thereafter the patients condition continuously improved and she stabilized after 4 hours.

DISCUSSION

The most common etiologies of cardiovascular collapse during cesarean section are high spinal anesthesia, amniotic fluid embolism and anaphylaxis.[1] A high spinal refers to more cephalad progression of the level of anesthesia than planned when the neuraxial block was administered or from a relative drug overdose.

Consequences of high spinal may include: severe hypotension and bradycardia. Complete diaphragmatic paralysis occurs if the C3 to C5 roots are blocked. The patient may feel nauseated and dyspneic. Aspiration of gastric contents may result due to loss of consciousness and compromise of airway reflexes. High spinal may result due to issues involving baricity and maternal position. As an example, injection of a hyperbaric solution (relative to CSF) and placing the parturient in a steep Trendelenburg position or injection of a hypobaric solution and positioning the parturient in a seated position will result in exaggerated cephalad spread.

Treatment of a high spinal requires intubation of the trachea to maintain adequate ventilation and protect the lungs from aspiration. The cardiovascular system must be supported with vasopressors and inotropes as necessary to maintain blood pressure.[2]

Anesthesiologists use a myriad of drugs during the provision of an anesthetic. Many of these drugs have side effects that are dose related, and some lead to severe immune-mediated adverse reactions. Anaphylaxis is the most severe immune-mediated reaction; it generally occurs on re-exposure to a specific antigen and requires the release of proinflammatory mediators. Anaphylactoid reactions occur through a direct nonimmunoglobulin E-mediated release of mediators from mast cells or from complement activation. Table 1 outlines the common drugs involved in perioperative anaphylaxis.[3]

Table 1: Drug involved in perioperative anaphylaxis

Substance	*Incidence of perioperative anaphylaxis (%)*	*Most commonly associated with perioperative anaphylaxis*
Muscle relaxants	69.2	Succinylcholine, rocuronium, atracurium
Natural rubber latex	12.1	Latex gloves, tourniquets, foley catheters
Antibiotics	8	Penicillin and other β-lactams
Hypnotics	3.7	Propofol, thiopental
Colloids	2.7	Dextran, gelatin
Opioids	1.4	Morphine, meperidine
Other substances	2.9	Propacetamol, aprotinin, protamine, bupivacaine

The cause of sudden cardiovascular collapse in the perioperative period can be elusive. Allergy may be overlooked as a cause. When allergy is considered, latex is often suspected. Because hydroxyethylstarch is frequently used in situations involving hypovolemia and hypotension, and because allergic reactions to it are rare, it may be overlooked as a possible allergen. A case has been reported of a patient suffering cardiovascular decompensation during four nonconsecutive perioperative periods before it was determined that she was allergic to hydroxyethylstarch. She also had a very highly positive latex radioallergosorbent test, suggesting a latex allergy.[4] Anaphylaxis has also been described in a parturient receiving hydroxyethylstarch.[5] It has been suggested that when hydroxyethylstarch is given, it should be included in the differential diagnosis of allergic reaction. Our patient had a cardiovascular collapse almost immediately following administration of hydroxyethylstarch. We however have no conclusive evidence as the patient could not be tested for allergy to the same due to loss of follow-up.

Death from anaphylaxis most commonly results from intractable bronchospasm, asphyxiation from upper airway edema, or cardiovascular collapse. The most common symptoms seen in anaphylaxis are urticaria and angioedema, which are seen in 88 percent of individuals. A patient presenting with angioedema must be assessed for signs of airway compromise if the swelling involves the tongue, uvula, soft palate, or larynx and for impending circulatory collapse if the angioedema represents an anaphylactic event.[6] Respiratory tract involvement and complaints of shortness of breath, wheezing, or laryngeal edema occur in approximately 50 percent of patients. Elevated airway resistance may result in increased peak airway pressures in intubated patients.

Respiratory failure can result from airflow obstruction, cardiogenic or noncardiogenic pulmonary edema, or the acute respiratory distress syndrome (ARDS). Anaphylactic shock occurs in 30 percent of cases. Cardiovascular collapse results from hypovolemia (due to increased vascular permeability and loss of up to 50 percent of blood volume), alterations in peripheral vascular resistance, and myocardial depression. ECG monitoring may demonstrate tachycardia, relative bradycardia, arrhythmias, or ST-T wave changes. Gastrointestinal symptoms, such as nausea, vomiting, diarrhea, and abdominal or uterine cramping, are reported by 30 percent of patients.[7]

Amniotic fluid embolism has often been linked to precipitous or tumultuous labor, uterine hyperstimulation, and the use of oxytocin. The onset of symptoms most commonly occurs during labor and delivery or in the immediate postpartum period. Rarely, AFES has been reported as late as 48 hours postpartum, or following cesarean delivery, first and second trimester abortions, or amniocentesis. Rapid cardiorespiratory collapse occurs at presentation in the majority of cases.

Nonspecific symptoms, including chills, nausea, vomiting, and agitation, may precede the onset of dyspnea and hypotension. The clinical presentation of AFE is similar to that of septic or anaphylactic shock. It has been further suggested that the name amniotic fluid embolus be discarded and the entity be renamed as the "anaphylactoid syndrome of pregnancy".[8,9]

To summarise, our patient suffered a cardiovascular collapse during cesarean section. Anaphylaxis was the most likely clinical diagnosis. There were no investigations available to substantiate our diagnosis. The patient responded to adrenaline and supportive treatment. Diagnosis of cardiorespiratory collapse under spinal anesthesia can be very challenging due to the number of clinical conditions presenting with similar clinical presentation. However emergent resuscitation and appropriate supportive care are the cornerstones of successful management of such patients.

REFERENCES

1. Riley ET, Cohen SE, Macario A, Desai JB, Ratner EF. Spinal versus epidural anesthesia for Cesarean section: A comparison of time efficiency, costs, charges, and complications. Anesth Analg 1995;80:709-12.
2. Grant GJ. Prevention and treatment of adverse effects of neuraxial anesthesia. In: Up to Date, Rose, BD (Ed), Up to Date, Waltham, MA, 2007.
3. DL Hepner, MC Castells. Anaphylaxis during the perioperative period. Anesth Analg 2003;97:1381-95.
4. Ritchey RM, Helf and RF, Irefin SA, Argalious M, Tetzlaff JE. Hetastarch allergy and positive latex radioallergosorbent test in a patient suffering cardiovascular decompensation during multiple perioperative periods. Anesth Analg 2005;101:1709-12.
5. Vercauteren MP, Coppejans HC, Sermeus L. Anaphylactoid Reaction to hydroxyethylstarch During Cesarean Delivery in a Patient with HELLP Syndrome. Anesth Analg 2003;96:859-61.
6. Bingham CO. An overview of angioedema. In: Up to Date, Rose, BD (Ed), Up to Date, Waltham, MA, 2007.
7. O'Dowd LC, Zweiman BF. Anaphylaxis in adults. In: Up to Date, Rose, BD (Ed), Up to Date, Waltham, MA, 2007.
8. Clark SL, Hankins GD, Dudley DA, et al. Amniotic fluid embolism: Analysis of the national registry. Am J Obstet Gynecol 1995;172:1158.
9. Baldisseri MR. Amniotic fluid embolism. In: Up to Date, Rose, BD (Ed), Up to Date, Waltham, MA, 2007.