JETIR.ORG ISSN: 2349-5162 | ESTD Year : 2014 | Monthly Issue
JOURNAL OF EMERGING TECHNOLOGIES AND INNOVATIVE RESEARCH (JETIR)
An International Scholarly Open Access, Peer-reviewed, Refereed Journal

Unexpected sudden intraoperative cardiac arrest during Lumbar spine fixation surgery: A case report

Ramanand Gupta(resident), Rajnish Bajwa(consultant), Ruby Srivastav(consultant), Shipra Agarwal(consultant), Dasari Praveena(resident)

Department of Anesthesia, Regency Hospital, Kanpur, Uttar Pradesh, India

Abstract

Intraoperative cardiac arrest during surgery is a grave concern to anesthetist and surgeon. We are reporting the unanticipated cardiac arrest in an elective lumbar spine fixation surgery. A diagnosis of Takutosubo Cardiomyopathy was made by exculsion. A cardiorespiratory resuscitation was performed in supine position.Patient recovered without any sequele on day-7 of event.

Keyword – cardiorespiratory arrest, asystole, prone position, takatsubo cardiomyopathy.

Introduction

Cardiorespiratory arrest (CRA) in a patient undergoing elective non-cardiac surgery is the worst nightmare for both surgeons and anaesthetists, particularly when there is no obvious underlying cause of risk(1,). Overall, the incidence of perioperative cardiac events is 6.7 per 1000 lumbar spine procedures, but only a minority of those have cardiac arrests(2,3,4). In recent years, the incidence of intraoperative and perioperative CRA associated with noncardiac surgery has decreased markedly with the advent of improved technologies and changing clinical practices(5,6).

We present a case who underwent an elective lumbar fixation surgery and had intraoperative CRA. A written consent was taken from the patient for the case to be reported.

Case report

A 67-year old male (height,150cm; weight 70kg; body mass index (BMI) ,31kg/m2) was scheduled to undergo L4-L5 posterior fixation and fusion for L4-L5 spondylolysthesis. Patient had history of chronic alcoholic and smoker since 30years, no other chronic medical illness, no known drug allergy and no active system infection. He had a functional capacity of > 4 METS. He had a heart rate of 80/minute, respiratory rate of 16/minute, blood pressure of

120/70mmhg, and oxygen saturation (SpO_2) of 98%with room air. Physical examination of airway,respiratory,cardiovascular system was normal. Preop investigations, including complete blood count, random blood sugar, liver function test, urea, creatinine, and electrolytes, ecg and 2d echo were all within normal limits. The patient fasted for 8 hours prior to the operation, as per hospital protocol.The patient was connected to standard monitoring as per the American Society of Anesthesiologists (ASA) guidelines. After preoxygenation with 100% O_2, anaesthesia was induced with 100 µg fentanyl, 2mg midazolam,100 mg propofol, and 8mg vecuronium. The tracheal intubation was performed uneventfully. Anaesthesia was maintained with 50% O_2 in N_2O, with an isoflurane MAC of 1.0%. All patient and ventilator parameters were normal. The patient was proned on two rolled pillows, leaving his abdomen free from compression. The vital parameters during the perioperative period, prior to arrest were as in Table 1.

Parameter	**preoperative**	**intraoperative**
Spo2	100% on room air	100% with Fio2-50%
Systolic Blood pressure	122-130mmhg	100-114mmhg
Diastolic Blood pressure	86-74 mmhg	60-70 mmhg
Heart rate	80-94 bpm	70-80bpm
ETCO2		30-40mmhg

He was given a total of 1000 mL of crystalloids over 1 h and 30 min prior to the event with estimated blood loss of 200 mL.

Ventilation parameters intraoperatively was as per Table 2

Table .2 ventilations setup at induction

Ventilation parameters	**Measurement**
Fio2	0.40-0.50
Tidal volume	450-500ml
Peak airway pressure	22-26cmH20
Repiratory rate	16breath/min

An hour and 30 min into the procedure, blood pressure (BP) readings were 100/65mmhg with the MAP 70 mmHg, HR was recorded as 70 bpm. SpO_2 was 100% on FiO_2 0.50 and ETCO2 was 35mmhg. The OT ambient temperature was maintained at 20°C and Bair hugger was applied from begning of surgery.However, the HR suddenly dropped to 45bpm, BP was 60/40 mmHg. Three doses of Atropine sulfate 0.5 mg was then administered still

bradycardia was persisting. The surgery was immediately stopped. The wound was covered with an iodine adhesive dressing, and the patient was turned supine immediately.

Effective cardiopulmonary resuscitation (CPR) was started with 100%Fio2, and cardiac compressions as per ACLS protocols.

Checking of the equipment, machines, and breathing system, as well as verification of the endotracheal tube, were completed with no defects reported. Hypoglycaemia was ruled out. Adrenaline was given intravenously at 1 mg every 3–5 minutes, up to a total dose of 10 mg, 30ML of 8.4% sodium bicarbonate. Intravenous 150 mg amiodarone were administered. At this point ABG sample taken .

Return of spontaneous circulation (ROSC) was achieved after 50min of effective CPR. An epinephrine infusion was commenced at 0.2–0.3 µg/kg/min and a right internal jugular central line access was established. He remained in sinus rhythm with HR between 110 and 120 beats per minute and no cyanosis noted during event. His MAP was maintained around 60–70 mm Hg with epinephrine and norepinephrine infusions 0.2–0.3 µg/kg/min.Both pupils are equal in size reactive to light post-CPR. Urine output at that point was 120ml.The wound was sutured on lateral position with a drain inserted, and the patient was transferred to icu for post-resuscitation care.

Cardiologist was also called in OT and ECHO after ROSC showed EF of 15-20%, global hypokinesia, IVC full with no abnormality detected in the valves or thrombus.ECG post ROSC shows RBBB with LAHB, IVCD, T inversion in inferior preocordial leads with sinus tachycardia. Toponin I- 0.86,BNP-99.6 .

In the ICU, he was kept sedated and ventilated for 24 h. However, sedation was temporarily lightened on the same night to assess his neurological status, and there was no neurological deficit.

ABG during event and 2hour after event

Table-3

ABG	During event	2hours after event
PH	7.35	7.37
PCO2	**45mmhg**	**52**
P02	**16mmhg**	**92**
sodium	**136mmol/l**	**138**
Potassium	**3.1mmol/l**	**3.6**
Glucose	**133mg/dl**	**146**
Lactate	**4.2mmol/l**	**1.3**
Hemoglobin	**14.8g/dl**	**12.1**
Base excess	**-1.1mmol/l**	**3.8**
Bicarbonate	**24.8mmol/l**	**30.1**
Chloride	**103mmol/l**	**104**
calcium	**1.02mmol/l**	**1.16**

His body temperature was maintained at 36°C to 37°C in the ICU., and he was extubated on day 2. Inotropic support was weaned off by day 3.On day-7 ECHO done shows EF-60%,no regional wall motion abnormality,normal valvular apparatus,no thrombus ,IVC normal.

DISCUSSION

In this case we have considered , common causes of cardiopulmonary arrest during spine surgeries in prone position.

The stable hemodynamic parameters and no significant blood loss ruled out haemorrhage-related cardiac arrest. Acute haemorrhage usually leads to tachycardia. He was given 1000 mL of over 1h and 30 min prior to the event (with expected blood loss of 200 mL during that time).

Dislodgement of the endotracheal tube (ETT) usually leads to the sudden disappearance of $ETCO_2$, followed by a declining saturation. In most cases, hemodynamic parameters should be unaffected until a significant period has lapsed without corrective measures taken.Hence disconnection of ETT was ruled out.

Electrolyte imbalance usually preceded by arrhythmia, which was absent in this case. This was also ruled out by ABG result taken after ROSC, as presented in Table 3.

Medication errors are also ruled out from the anaesthetic records.

From multiple case reports, a precipitous drop in BP, $ETCO_2$, and SpO_2 is a common occurrence in venous air embolism(VAE).But in our case ETCO2 was maintained so VAE ruled out.(7)

There was no traction of duramater at this point of surgery so vasovagal response is ruled out.

Pulmonary thromoembolism (PTE) can present with hypotension, sudden decrease in $EtCO_2$, bulging of the jugular vein, and changes in ECG (arrhythmia, ST depression, or right bundle branch block). Wheezing or decreased lung sounds can be heard in both lungs. Extremely high pulmonary artery pressure and central venous pressure may be observed(8). In our case after ROSC ,ECHO shows no emoli in pulomary artery and no dilatation of right atrium so PTE ruled out.

Alcoholic cardiomyopathy (ACM) is a cardiac disease caused by chronic alcohol consumption. It is characterized by ventricular dilation and impairment in cardiac function. ACM represents one of the leading causes of non-ischemic dilated cardiomyopathy(9). Alcoholic cardiomyopathy can present with signs and symptoms of congestive heart failure(10). In our case ,preoperative evaluation showed no signs of congestive heart failure and no ventricular enlargement on 2dEcho. Hence alcoholic cardiomyopathy unlikely.

Takotsubo cardiomyopathy(TTC) is a life threatening condition characterised by transient left ventricular (LV) wall motion abnormality occurring in the absence of coronary artery disease(11).). The incidence of perioperative TTC is estimated to be 1/6700 cases The most common cause is a catecholamine surge causing a ‘stunning’ of the myocardium, leading to transient LV dysfunction. TTC is generally precipitated by exposure to endogenous or exogenous stress (12). It is hypothesised that elevated levels of catecholamines result in changes to intracellular signal trafficking, leading to negative inotropic effects seen maximally at the apex of the myocardium(13.Diagnosis is typically based on meeting the clinical criteria as outlined by the Mayo clinic in 2008 (14).

MAYO CRITERIA FOR DIAGNOSIS OF TAKOTSUBO CARDIOMYOPATHY(PRASAD etal)
1.Transient hypokinesia ,akinesis/dyskinesia of left ventricular mid segments, with or without apical involvement.
2.Absence of obstructive coronary disease.
3.New ECG abnormalities>ST elevation/ T inversion or modest increase in troponin.
4.Absence of pheochromocytoma and myocarditis.

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Perioperative TTC is more likely to present with evidence of heart failure, arrythmias or cardiac arrest, the presentation is similar to an ACS. However, recognising TTC intraoperatively and differentiating it from ACS can pose quite a challenge(15,16). Use of cardiac biomarkers can assist diagnosis in this regard.Compared with ACS, TTC tends to have a much higher peak NT-proBNP and a lower TnT rise.Management is generally

supportive aimed at managing myocardial ischemia with oxygen, anticoagulation (until a diagnosis of coronary artery disease is excluded) and beta-blockers.

In our case ,a 12-lead ECG after rosc showed T-wave inversion in inferior leads with sinus tachycardia . A bedside echocardiogram revealed EF of 15-20% and global hypokinesia . Vagally mediated cardiac collapse, autonomic dysregulation due to smoking, and chronic emotional stress may have contributed to this event .The patient had no neurologic deficits following the event.. Hence most probable diagnosis was Takotsubo cardiomyopathy .

Summary

This report examines a case of intraoperative cardiac arrest in a patient with no previous cardiac disease.Common causes include excessive hemorrhage, electrolyte imbalance, venous air embolism, anaphylactic shock, Takotsubo cardiomyopathy(TTC).TTC characterised by rapidly reversibleventricular dysfunction without any prior coronary arterydisease, it can imitate a myocardial infarction and lead to death if not managed appropriately.

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