Case Report

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A case of cardiac arrest due to air embolism during scoliosis surgery

Mohamad Zaki Haji Mohd Amin[1], James Tan Boon Beng[1], Brian Teo Yian Young[1], Nur Aida Faruk Seman[2], Teo Shu Ching[3] and Wong Chung Chek[2]

Abstract

Cardiac arrest during scoliosis surgery is rare in idiopathic scoliosis. We present a case of cardiorespiratory collapse during corrective surgery in a young patient with idiopathic scoliosis. A diagnosis of venous air embolism was made by exclusion. A cardiorespiratory resuscitation was performed in supine position. Patient recovered without any sequelae and had operation completed 6 weeks later.

Keywords

cardiorespiratory arrest, scoliosis, survive, venous air embolism

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Introduction

Cardiorespiratory arrest (CRA) in a patient undergoing elective non-cardiac surgery is the worst nightmare for both surgeons and anaesthetists. According to the Scoliosis Research Society mortality and morbidity database, the risk of death is 1.8 per 1000 scoliosis surgery.[1] However, the incidence of intraoperative CRAs during scoliosis surgery is not reported. In most cases reported, the causes are due to cardiac rhythm abnormality or secondary to intraoperative complications.

We present a case of a young lady with adolescent idiopathic scoliosis who underwent an elective posterior scoliosis correction surgery and had intraoperative CRA midway during the surgery. A written consent was taken from the patient for the radiograph and the case to be reported.

Case report

The patient was a 20-year-old lady, newly diagnosed with bronchial asthma on steroidal metered dose inhaler. Her asthma was well controlled with her last attack 6 months prior to admission. She was diagnosed with scoliosis since the age of 12, but she defaulted follow-up until the age of 19 when she agreed for surgery because of the worsening deformity and axial back pain.

She had a Lenke type 3C double major scoliosis with a Cobb angle of 90° degrees and planned for fusion from second thoracic to third lumbar vertebra. We planned to insert pedicle screws on every level on the concave side and intermittently on the convex except each vertebra level at the apex for vertebral derotation procedure. The screws were inserted from caudal to cephalic with two surgeons on each side.

Her preoperative blood investigations were unremarkable. Creatine kinase level was not checked as it was not a

[1] Department of Orthopaedic, Faculty of Medicine and Health Sciences, University Malaysia Sarawak, Sarawak, Malaysia
[2] Department of Orthopaedic, Sarawak General Hospital, Kuching, Sarawak, Malaysia
[3] Department of Anaesthesiology, Sarawak General Hospital, Kuching, Sarawak, Malaysia

Corresponding author:
Mohamad Zaki Haji Mohd Amin, Department of Orthopaedic, Faculty of Medicine and Health Sciences, University Malaysia Sarawak, Jalan Datuk Mohammad Musa, 94300 Kota Samarahan, Sarawak, Malaysia.
Email: mamzaki@unimas.my

Table 1. Vital sign measurement preoperative and intraoperative.

Parameter	Preoperative	Intraoperative
Systolic BP	100–128 mmHg	89–103 mmHg
Diastolic BP	68–88 mmHg	61–74 mmHg
Heart rate	96–108 beats per min	85–99 beats per min

BP: blood pressure.

Table 2. Ventilation set up at induction.

Ventilation parameters	Measurement
FiO_2	0.35–0.40 (air/oxygen)
TV	360–400 mL
PAW	20–22 cmH_2O
RR	12 breath/min

TV: tidal volume; PAW: peak airway pressure; RR: respiratory rate.

routine preoperative investigation. Her preoperative lung function test showed FEV1/FVC: 0.78, FEV1:57% predicted and FVC: 64% predicted. The echocardiogram (ECHO) revealed left ventricle ejection fraction (EF) of 68%, with no thrombus and normal valves. Her electrocardiogram (ECG) displayed a sinus rhythm with no ischemic changes or strain pattern. Her preoperative vital signs were as below She was induced with target-controlled infusion (TCI) propofol and remifentanil, relaxed with intravenous (IV) cisatracurium 10 mg (0.2 mg/kg). The intubation was uneventful. Post-intubation, lungs were clear and air entry was equal. The cardiac rhythm remained in the sinus. IV accesses were through a 16G cannula in the left hand and 14G at the right external jugular vein. A left radial arterial line was inserted. The patient was positioned prone on two rolled pillows, leaving her abdomen free from compression. The vital parameters during the perioperative period prior to arrest were as in Table 1.

Her anaesthesia was maintained with TCI of propofol 4–5 mcg/mL and TCI remifentanil 4–5 ng/mL with no further paralysis due to intraoperative neuromonitoring. IV midazolam (2 mg) was given at induction and 1 mg after she was in prone position. Nitrous oxide was not used. She was given a total of 1500 mL of crystalloids and 500 mL of colloid over 2 h and 40 min (induction to skin incision was 1 h and 15 min) prior to the event with estimated blood loss of 400 mL. Ventilation measurement intraoperatively was as per Table 2. With relatively high TCI remifentanil, despite train-of-four count of 4, there was no sign of spontaneous breathing effort and recorded respiratory rate was 12 throughout.

Her vital signs remained stable intraoperatively (Table 1). An arterial blood gas (ABG) was taken 60-min pre-event and showed in Table 3. Replacement with a 1-h infusion of 20 mmol/L potassium chloride and 1 gram of calcium gluconate was commenced.

Table 3. Results of ABG before and after the event.

Parameter	Pre-event	Post-event
pH	7.38	6.98
PCO_2	36 mmHg ($ETCO_2$:32)	90 mmHg
PO_2	202 mmHg (FiO_2: 0.38)	56 mmHg
K^+	2.9 mmol/L	4.1 mmol/L
Ionized Ca^{2+}	0.8 mmol/L	1.37 mmol/L
Glucose	4.4 mmol/L	11.7 mmol/L
Lactate	0.6 mmol/L	5.5 mmol/L
Haematocrit	28%	27%
HCO_3^-	22.3 mmol/L	16.3 mmol/L
Base excess	−3.8 mmol/L	−10.4 mmol/L
Haemoglobin	8.7 g/dL	8.4 g/dL

An hour and 35 min into the procedure, we had already inserted 11 pedicle screws. End tidal carbon dioxide ($ETCO_2$) suddenly dropped from 31 to 19 then 0, within a few seconds. Two minutes prior to the event, blood pressure (BP) readings were 89/65 and 92/67 mmHg with the MAP of 73 and 75 mmHg, respectively. HR was recorded as 92 and 89 bpm. SpO_2 was 100% on FiO_2 0.36. The temperature was maintained at 36.2°C. However, the HR suddenly dropped to 0, BP was 28/28 mmHg. SpO_2 recorded from 100% suddenly dropped to 86% (Figure 1). Manual bagging was immediately attempted and the tidal volume (TV) of 300+ mL was achieved, but there was no $ETCO_2$ reading detectable. The surgery was immediately stopped. The wound was covered with an iodine adhesive dressing, and the patient was turned supine immediately. The first dose of IV adrenaline 1 mg was administered.

Cardiopulmonary resuscitation (CPR) was commenced immediately once in supine position, with the time taken from asystole to supine position, and CPR commencement was about 3 min. Return of spontaneous circulation (ROSC) was achieved after 10 min and IV adrenaline 1 mg was given twice during CPR. Twenty millilitres of 8.4% sodium bicarbonate, 10 mL 10% calcium gluconate, and 1 pint of packed cell were given during the resuscitation which was done in a slight Trendelenburg position. Her BP was sustained with IV adrenaline and IV noradrenaline infusion post-CPR. Post-CPR ABG is shown in Table 3. The wound was sutured on lateral position with a drain inserted, and the patient was transferred to ICU for post-resuscitation care.

There was no dynamic changes or any acute ischemic changes seen in the ECG. Computed tomography of the pulmonary angiogram (CTPA) showed no evidence of pulmonary embolism. An ECHO in ICU showed EF of 63%, with no abnormality detected in the valves or cardiac chambers. In the ICU, she was kept sedated and ventilated for 24 h for cerebral resuscitation. However, sedation was temporarily lightened on the same night to assess her neurology, and there was no neurological deficit. Her body temperature was maintained at 36°C to 37°C in the ICU.

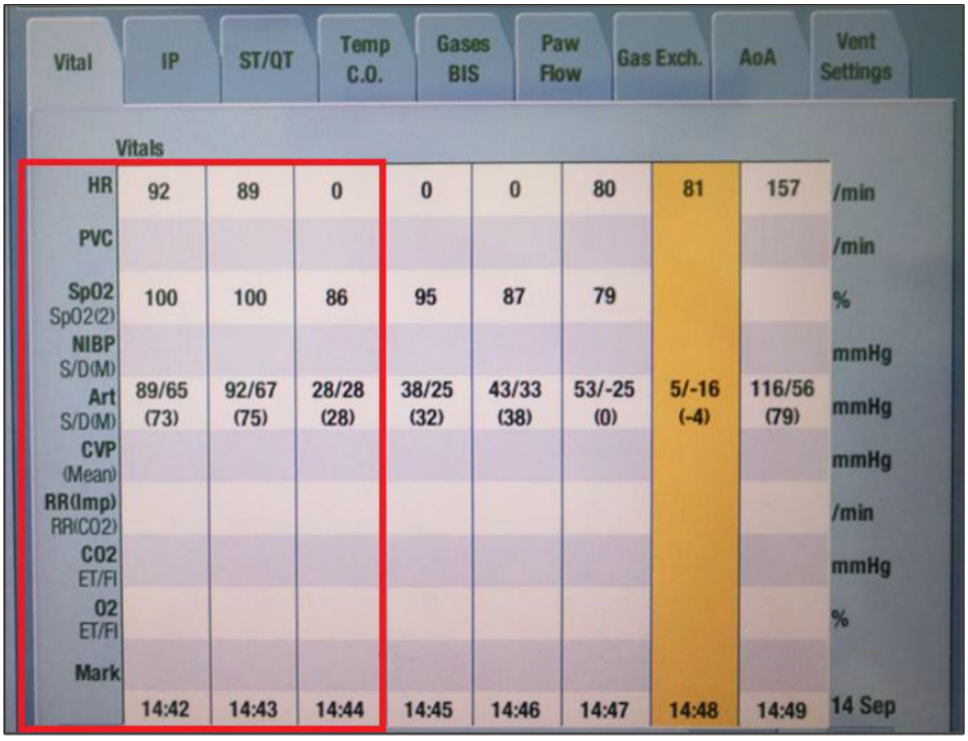

Figure 1. Screenshot of vital sign monitoring intraoperatively.

Inotropic support was weaned off by day 3, and she was extubated by day 4. Patient subsequently underwent an uneventful wound debridement and closure 1 week after her first operation and definitive surgery 6 weeks later.

Discussion

CRA following spinal surgery is rare. A Scandinavian study reports an incidence of 6.8 cardiac arrests per 10,000.[2] CRA is seen more often in complex case of spine surgery due to excessive blood loss as in neuromuscular scoliosis.[3] In idiopathic scoliosis, it is associated with cardiac rhythm abnormality, anaphylactic reaction and placement of an epidural catheter.[3,4] In one case reported, no risk factor is identified.[5]

We have considered common complications of spine surgeries in prone position following our resuscitation. The stable hemodynamic vital parameters and no significant blood loss ruled out haemorrhage-related cardiac arrest. Acute haemorrhage usually leads to tachycardia or occasionally bradycardia. Immediate asystole is less likely to occur. She is given 1500 mL of crystalloid and 500 mL of colloid over 2 h and 40 min prior to the event (with blood loss of 400 mL during that time). However, peak airway pressure (PAW) at induction is 20 cmH_2O with TV of 361 ml, compared to immediate pre-event where PAW is 22 cmH_2O and TV of 384 mL. This could rule out the element of fluid overload which is usually evidenced by lower lung compliance. Furthermore, good oxygen saturation and FiO_2 rule out this possibility.

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. Electrolyte imbalance such as hypo or hyperkalaemia usually preceded by arrhythmia, which is absent in this case. This is also ruled out by the blood result taken after ROSC, as presented in Table 3. Pulmonary embolism is considered but unlikely with a normal CTPA. 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 VAE as in our case. Hence, we are inclined to postulate that venous air embolism (VAE) is the most likely diagnosis in our case coupled with the risk factors of spine surgery and prone position.

In VAE, air can enter the venous circulation when there is a negative pressure gradient between the right atrium and the point of entrance of air from the venous channel.[6] Air embolism can be catastrophic and very likely fatal. In the prone position, when the operative site is above the level of the heart, venous entrainment of air is likely to occur.[7] Efforts to minimize abdominal compression and thus vena cava pressure in the prone position can result in an increased negative pressure gradient between right atrium and veins at the operative site. This has increased the risk of air entrainment.[8] Sudden spontaneous breathing effort from

the patient during operation may decrease the intrathoracic pressure, hence producing an effect of suction and entrain air via venous circulation. Despite almost full muscle recovery from the intubating dose of cisatracurium, high dose of remifentanil TCI managed to avoid unnecessary breathing effort from the patient.

Presence of visible bubbling at the operative site was usually the first clinical indication of VAE, but it can also happen without the visible bubbling.[9] There is no bubbling present in our case. A high index of suspicion could potentially save the life of the patient as a definitive diagnosis could be difficult.

Immediately upon suspicion, we flooded the operative field with saline to stop the entrainment.[10] We resuscitated our patient in a slight Trendelenburg position to dislodge the possible air bubble occluding the pulmonary artery.[5] We performed cardiac compressions that can be effective in pushing air from the pulmonary artery into the smaller vessels where the small bubbles are rapidly resorbed hence facilitating ROSC.[11,12]

Transoesophageal echocardiogram (TEE) has been advocated to help in the diagnosis of patients who has a cardiac arrest during non-cardiac surgery. Presence of right atrial dilatation is characteristic of a diagnosis of VAE.[13] However, in our case, we can only rely on clinical suspicion when definite diagnosis modality is not available.

CPR on a prone patient with CRA is challenging because of the difficulty in maintaining the airway and performing defibrillation.[8] Therefore, we perform the resuscitation in supine position due to limited experience with prone position resuscitation. Despite the 3-min delay for turning the patient before initiating CPR, the patient had survived the ordeal.

Conclusion

A case of intraoperative cardiac arrest from a suspected VAE during scoliosis surgery was presented. This report serves as a reminder of a potentially fatal complication in scoliosis surgery. Early detection and prompt resuscitation will help to save lives and ensure a favourable outcome.

Declaration of conflicting interests

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