

Cardiac arrest in a child following cranioplasty

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Abstract

Craniofacial surgery in paediatric patients is associated with several complications, including severe bleeding and venous air embolism. We present a case of an 18 months, 10 kilogramme weight child who had cardiac arrest after completion of surgery, but before extubation. Possibility of both haemorrhage and venous air embolism is discussed.

Keywords: Craniofacial surgery, Paediatric, Venous air embolism.

Introduction

Craniofacial surgery in paediatric anaesthesia is associated with serious morbidity. Complications like severe bleeding and venous air embolism have been described before, but in majority of cases these occur during the intraoperative period. We present a case of suspected venous air embolism that occurred after completion of surgery, but prior to extubation in a child who had undergone bifrontal cranioplasty.

Case Report

A 10kg, 18-month-old female child presented for elective bifrontal cranioplasty. She had history of trauma followed by posttraumatic seizures and had undergone bifrontal craniotomy three months earlier. She was on anti-convulsant medication.

Standard monitoring in the operating room included electrocardiogram (ECG), non-invasive blood pressure (NIBP), pulse oximetre, end-tidal carbon dioxide, temperature, precordial stethoscope, end-tidal concentration of gases and urine output. Two peripheral intravenous cannulae were inserted and anaesthetic induction was done with 10ug fentanyl, 20mg propofol and atracurium. Left radial artery was cannulated and a central venous catheter was inserted in the right femoral vein. The patient was placed in supine position.

Surgical procedure was of three-hour duration. Patient remained haemodynamically stable throughout. Intraoperative blood loss was calculated by observation

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of blood-soaked sponges, surgical drapes, suction apparatus, changes in haemodynamics, trend of central venous pressure (CVP), urine output and was estimated to be approximately 215mls. The patient received 460mls crystalloids and 220mls of packed cells.

After completion of surgery, but before reversal of the muscle relaxation, while the neurosurgical resident was applying the head bandage, we noticed a sudden fall in end-tidal CO₂ from 31mmHg to 18, 11 and 0 within few seconds. Bradycardia occurred at the same time with decrease in heart rate from 127 beats min⁻¹ to 60 beats min⁻¹. Tracheal tube was rechecked. Upon auscultation, no heart sounds were audible and chest compressions were initiated immediately and intravenous atropine 0.2mg was administered. Simultaneously it was noticed that the patient was extremely pale. Blood in both redivac drains was noted to be approximately 150mls. Ventilation with 100% oxygen was continued. Adrenaline was given in boluses of 20ug three times, each one minute apart. Five minutes later, spontaneous cardiac rhythm returned with a heart rate (HR) 135-140 beats minute⁻¹. Two hundred ml blood was also transfused with a 20ml syringe. An arterial sample for blood gas analysis was sent. Ten mEq bicarbonate was also administered. Arterial blood gas which was sent showed metabolic acidosis - pH 7.28, partial pressure of carbon dioxide (PaCO₂) 33.6, partial pressure of oxygen (PaO₂) 526.7, bicarbonate (HCO₃) 14.8, saturation level of oxygen in haemoglobin (SaO₂) 100%. Ten mEq bicarbonate was repeated along with colloid administration. Blood gas sample sent after 20min showed improvement (pH 7.38, PaCO₂ 33.6, PaO₂ 500, HCO₃ 19.7). Haemoglobin after 200mls of blood transfusion was repeated and was found to be 9.4g/dl. Further need to continue vasopressors/inotropes was abandoned as blood pressures (BP) was maintained in normal range and the reason for sudden hypotension and cardiac arrest at that time was thought to be acute blood loss in the drain.

After the patient's condition stabilised, the surgeon decided to re-explore the wound and the surgical site. No bleeding point was identified. The patient was extubated at the end of second surgery, and had spontaneous eye opening and was maintaining an end-tidal CO₂ of

40mmHg with stable vital signs (HR 148 beats minute⁻¹, BP 116/77mmHg, SO₂ 100%). She was monitored for 10 minutes in the operating room for any sudden fall of BP or any deterioration in condition and was then shifted to the paediatric intensive care unit (ICU). Patient had one episode of generalised tonic clonic seizures approximately six hours after ICU admission. This was controlled on IV midazolam (0.5mg). Patient remained stable and was discharged home after two days.

Discussion

Craniofacial surgery in paediatric patients can lead to significant morbidity. Perioperative bleeding can result in hypotension, coagulopathy, intraoperative cardiac arrest and even death.¹ Other complications like postoperative seizures, surgical site infections, facial swelling and unplanned postoperative mechanical ventilation have also been documented.² These procedures are also associated with a high incidence of venous air embolism (VAE). Faberowski et al have reported this incidence to be as high as 82.6%,³ whereas previously it was reported as 66%.² Faberowski et al justified this high incidence based on an observation that children have a large head compared to the body and even in the supine position the head maybe positioned above the heart. Use of a precordial ultrasonic probe monitoring device helps in earlier detection of this complication.⁴ In our patient the haemodynamic event occurred towards the end of the surgery and before the reversal of muscle relaxation. At the time of the event, our initial diagnosis was the sudden blood loss associated with the release of pressure in the drains by the surgeon, but later we included venous air embolism in our differential diagnosis. The point in favour of hypovolaemic shock was circumstantial evidence of loss of approximately 150mls of blood in the drains, but CVP was maintained above 10cms H₂O throughout the procedure. No evidence of bleeding was seen on re-exploration. The suddenness of the event, an acute fall in end-tidal carbon dioxide and the extreme pallor observed on exposure of child are more in favour of mechanical obstruction of stroke volume.

Venous air embolism is more likely to occur during the intraoperative period, but recently the recurrence of VAE has been reported at the end of procedure after skin closure associated with change in position at end of surgery.⁵ Movement of previously entrained air may occur at the end of surgery, while moving the patient from prone or lateral position to the supine position and this can be considered one of the etiology of unexpected

sudden postoperative cardiovascular collapse.⁶ Hypotension associated with VAE in children is also more pronounced because the volume of entrained air is greater in comparison with blood volume.³

In our patient, the head was raised at the end of the surgery to apply the bandage, and the event occurred after placing the head in supine position. One possibility could be that sudden release of blood into the redivac drains could have led to a decrease of venous pressure below atmosphere and air trapped inside the closed drain or intra-thoracic vessels might have entered the venous blood stream and the right atrium with redistribution of air bubbles. Unfortunately we do not have the facility of Doppler probe or Trans Esophageal Echocardiography (TEE) available for use. Therefore, we do not have objective evidence of this event.

Rapid initiation of resuscitation and chest compression has presumptively demonstrated efficacy for massive VAE that results in cardiac standstill.^{7,8} Cardiac massage forces air out of the pulmonary outflow tract into smaller vessels. This could also have happened in our patient.

Conclusion

VAE should be considered one of the causes of unexpected cardiovascular collapse in craniofacial surgery in children, and anaesthetists should be vigilant not only intra-operatively, but also towards the end of the surgery after wound closure.

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