



Case Report

Anesthetic challenges involved in successful resuscitation of a child from cardiac arrest secondary to massive hemorrhage and possible venous air embolism while undergoing fronto-orbital advancement surgery for metopic craniosynostosis

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ARTICLE INFO

Article history:

Received 13-10-2020

Accepted 22-01-2021

Available online 01-06-2021

Keywords:

Air embolism

Cardiac arrest

Craniosynostosis

Pulseless electrical activity

ABSTRACT

A one year 3 month old child undergoing fronto-orbital advancement surgery for metopic craniosynostosis had severe bleeding when the surgeon attempted to remove bone flap. Head-end elevation was given at the surgeon's request to reduce bleeding. Immediately there was a drastic fall in end tidal carbon dioxide (ETCO₂) and arterial saturation (SpO₂). Considering air embolism, fraction of inspired oxygen (FiO₂) was increased to 100% and the surgeon filled the field with saline and covered the area with wet gauze. The operating table was leveled. The child continued to deteriorate with the cardiac rhythm changing to pulseless electrical activity and asystole. Incremental bolus doses of adrenaline, blood products transfusion, fluid bolus and infusion of inotropes were given. Chest compression was not done as the endotracheal tube was fixed to the chest of the patient. Tube dislodgement without access to the head-end of the patient would have been a disaster. The child became hemodynamically stable, the surgery continued and the child was extubated the next day. Other than focal seizures which responded to levetiracetam, the child had no neurological deficits.

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1. Introduction

Massive blood loss and air embolism are the two leading causes of cardiac arrest in craniosynostosis surgery.

Pulseless electrical activity (PEA) is an organized electrical activity without a palpable pulse or blood pressure. According to American Heart Association guidelines, PEA requires advanced cardiac life support.

Post cardiac arrest survival depends on the electrocardiographic rhythm, efficiency of CPR, preoperative condition and the duration of absent circulation.

2. Case Report

A 1 Year 3 month old male child of body weight 5 kg was a case of metopic craniosynostosis who

underwent fronto-orbital advancement surgery. The patient was provided with adequate insulation to maintain body temperature at 37 degree C with hair cap and body warmer. Standard ASA monitors including arterial saturation probe, electrocardiogram leads, and pediatric blood pressure cuff were attached. Induction was performed with 100% O₂ supplementation with a pediatric face mask, 4% w/w Sevoflurane, IV Glycopyrrolate 0.05 mg, IV Fentanyl 10 mcg, and IV Propofol 10 mg. IV Atracurium 5 mg was used for neuromuscular blockade. Intubation was uneventful. Following intubation, a temperature probe was kept orally and ETCO₂ probe was attached. Left radial artery and right internal jugular vein were cannulated and urinary bladder catheterized. Intermittent positive pressure ventilation was provided at volume control mode: RR: 30/min; TV: 35 ml/min; PEEP: 5; FiO₂: 50%. A baseline ABG was taken showing: P_aO₂: 180, P_aCO₂: 46.9, pH: 7.29, HCO₃⁻: 21.2, Potassium: 4.2, Hb: 9.3. The respiratory rate was increased

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to 35/min since the PCO₂ was high.

Use of a precordial Doppler monitor was deferred because of the small size of the patient combined with the likelihood of repeated dislodgement of the probe by the surgical team's intra-operative manipulations of the patient's head.

Fluid maintenance was continued at 20 ml/hour with IV Ringer Lactate. Fluid bolus was given immediately post induction. IV Tranexamic Acid infusion was started and continued at 10 mg/kg/hour (50 mg/hour). IV Atracurium infusion was started and continued at 0.5 mg/kg/hour (2.5 mg/hour).

The patient was hemodynamically stable during the first 3 hours of surgery.

Intraoperatively, severe bleeding occurred during attempted removal of the frontal bone flap. The patient's head-end was elevated at surgeons request to control bleeding, resulting in a sudden fall in EtCO₂ to 12 mmHg followed by drop in SPO₂ and systolic blood pressure (SBP) to 50 mm of Hg. Packed RBC bolus of 20 ml and 20 ml of 0.9% saline were rushed without any significant improvement in blood pressure. We suspected air embolism primarily because of the drop in EtCO₂ preceded the fall in SPO₂ and blood pressure. We increased the FiO₂ to 100% and stopped the inhalational anesthetic agent. The surgeon irrigated the field with normal saline and packed the bleeding area under the bone with wet gauze. Aspiration of the central venous catheter revealed occasional bubbles and froth which confirmed our diagnosis of venous air embolism.

In spite of all the efforts the ETCO₂, SpO₂ and BP dropped to unrecordable levels which did not respond to boluses of 10 µg of phenylephrine and 1 mg ephedrine, with sinus rhythm still present on the ECG monitor. The child developed pulseless electric activity which was managed with escalating IV adrenaline boluses (10 µg, 20 µg, 40 µg, 40 µg, and 40 µg) at 1-2 minute intervals until signs of perfusion returned 15 minutes after disappearance of the arterial tracing. We decided against administering chest compressions as the ET tube was fixed to the patient's chest and airway dislodgement would have been devastating without access to the head-end of the patient. Systolic Blood Pressure slowly improved to 40 mmHg, ETCO₂ improved from 10 mmHg to 18 mmHg and SPO₂ improved slowly to 80%. Noradrenaline and adrenaline infusions were started at 0.1 and 0.05 µg.kg⁻¹.hr⁻¹ respectively while volume status was further corrected using additional packed RBC and FFP.

During resuscitation, the child had severe metabolic acidosis and hyperkalemia (Venous blood gas analysis was done since arterial line did not have a backflow). It revealed a pH - 6.76, P_vO₂ - 31 mmHg, P_vCO₂: 91.1 mmHg, HCO₃⁻: 6.4 mmol.l⁻¹, potassium: 8.4 mmol.l⁻¹ and Hb: 7.1 g.dl⁻¹) which was managed with glucose-insulin, sodium bicarbonate and calcium gluconate and 100 ml of PRBC

transfused.

A repeat venous blood gas sample done showed a pH - 6.72, P_vO₂ - 33.9 mmHg, P_vCO₂ - 52.5 mmHg, HCO₃⁻ - 7 mmol.l⁻¹, potassium - 5.6 mmol.l⁻¹, Hb - 9.8 g.dl⁻¹, and glucose: 373 mgdL⁻¹. 2 units of plain human insulin were given IV followed by 8.4% sodium bicarbonate infusion of 17 ml over 20 minutes. IV Calcium Gluconate 30 mg/kg (150 mg) was given over 10 minutes. 50 ml more of PRBC was transfused IV.

After return of arterial tracing, arterial blood gas sampling revealed pH: 7.05, P_aO₂: 245 mmHg, P_aCO₂: 38.7 mmHg, HCO₃⁻: 10.3 mmol.l⁻¹, potassium: 2.9 mmol.l⁻¹ and Hb: 6.5 g.dl⁻¹.

As the patient was oozing continuously, a total of 300 ml PRBC and a total of 100 ml of FFP and a single unit of platelet (40ml) was transfused intraoperatively.

Once the patient's hemodynamic parameters returned to a semblance of their pre-event values (on inotropic support), the surgery continued and the torn sinus was repaired. There were no further detectable embolic events. Thromboelastography showed a normal study. Adrenaline infusion was tapered and stopped; nor-adrenaline infusion was continued in the immediate post-operative period, and the patient was electively ventilated. Before shifting to the Intensive Care Unit, arterial blood gas sampling was taken: pH: 7.34, P_aO₂: 413 mmHg, P_aCO₂: 30.1 mmHg, HCO₃⁻: 16.0 mmol.l⁻¹, potassium: 2.8 mmol.l⁻¹ and Hb: 10.6 g.dl⁻¹. 2.5 mEq Intravenous Potassium Chloride correction started at 1 mEq/hour.

Postoperatively the patient received a total of 50 ml of PRBC. The next day, the child was weaned off ventilatory and inotropic supports. Prior to extubation arterial blood gas sampling showed: pH: 7.41, P_aO₂: 225 mmHg, P_aCO₂: 24.4 mmHg, HCO₃⁻: 15.1 mmol.l⁻¹, potassium: 2.7 mmol.l⁻¹ and Hb: 9.0 g.dl⁻¹. Intravenous Potassium Chloride correction was continued. Post extubation, the patient had no neurological deficits except for focal seizures which responded to levetiracetam.

3. Discussion

This case study shows the effects of head-end elevation during surgery to reduce bleeding resulting in massive air embolism leading to PEA which responded to inotropic support.

The first recorded case of air embolism was in 1830 by Barlow.¹ Virchow coined the term embolism² and JZ Amussat outlined the treatment of embolism.² Two separate studies analyzed the incidence of air embolism in craniosynostosis surgeries as 2.6% and 83%.³ The incidence of cardiac arrest is around 0.4%-2.2% in non-syndromic craniosynostosis surgeries.⁴ The Pediatric Perioperative Cardiac Arrest Registry of North America analyzed 289 perioperative cardiac arrests and found that 5 patients had air-embolism as the cause for cardiac arrest.⁵

Elevation of the head results in a pressure difference between the right atrium and the cranial venous sinuses leading to air entrapment. 100 ml air in the venous system may trigger clinical manifestations in adults; the lethal dose is 3-4 ml/kg.⁶ Large air volumes can cause ventilation perfusion mismatch, right heart failure, myocardial and cerebral ischemia and cardiac arrest.

A devastating complication of massive venous air embolism is paradoxical air embolism where air enters the systemic circulation via a patent foramen ovale (PFO) resulting in ischemic stroke and quadriplegia. The incidence of a PFO in adults is 25% and its incidence may be similar in children.^{3,7} There are no indicators that our patient developed paradoxical air embolism since the patient did not have any significant neurological deficits.

A sudden drop in ETCO_2 and SPO_2 with hypotension is suggestive of air embolism. Capnography has moderate sensitivity and specificity for diagnosing air embolism. Precordial Doppler is highly sensitive. The transducer should be kept over the right atrium. Transesophageal echocardiography (TEE) is the gold standard for diagnosing air embolism as it can pick up even minute volumes of air (0.02 ml/kg), but it is expensive, invasive and needs expertise.⁸ TEE is not routinely used for procedures in which the surgeon may need access to the patients' airway, such as craniofacial procedures that children with craniosynostosis are subjected to. End tidal nitrogen (ETN_2) though not widely available much can detect air embolism much earlier than ETCO_2 .⁹ Nevertheless, these more sensitive monitors are useful in detecting minor embolic events and serve to warn the treating team of further events.

4. Management

During craniotomy when VAE is suspected, the neurosurgeon should irrigate the surgical field with normal saline to prevent the further entrapment of air. The patient should be provided controlled positive pressure ventilation with 100% FiO_2 . A central venous catheter needs to be placed prior to any neurosurgical procedure requiring the patient to be in sitting or semi-sitting position so that in the event of VAE, any entrapped air can be aspirated. Bilateral compression on jugular veins reduces the entry of air into the exposed sinuses but it may raise the ICP and reduce the brain perfusion. Placing the patient in Durant position helps to move the air bubbles toward the right atrium. If there is no improvement with the above measures, cardio-pulmonary resuscitation should be started.

The risk factors for perioperative cardiac arrest in children include anesthetic and surgical risks and associated comorbidities. Pediatric Advanced Life Support Protocol includes identifying the arrest rhythm (asystole, pulseless electrical activity, ventricular fibrillation or ventricular tachycardia). PEA does not respond to defibrillation therefore chest compressions, ventilatory and inotropic

support is necessary. Once ROSC is achieved, Post-Cardiac Arrest Care is initiated.¹⁰

The National Blood Authority of Australia, in their Module on Critical Bleeding/Massive Transfusion¹¹ (March 2011), has defined massive transfusion in children as transfusion of $> 40 \text{ mL blood/kg.}$. Our patient received 350 ml of packed red blood cells, 100 ml FFP, and 40 ml platelets perioperatively; qualifying massive blood transfusion for our patient. The goals in management of massive blood loss include early recognition of blood loss, maintenance of tissue perfusion and oxygenation by restoration of blood volume and hemoglobin, arrest of bleeding with early surgical or radiological intervention, and judicious use of blood component therapy to correct coagulopathy. Laboratory parameters to be aimed for during management include, core body temperature $>35^\circ\text{C}$, pH >7.2 , base excess <-6 , lactate $<4 \text{ mmol.l}^{-1}$, serum ionized calcium (Ca) $>1.1 \text{ mmol.l}^{-1}$, serum platelet $\geq 50 \times 10^9.\text{l}^{-1}$ ($>100 \times 10^9.\text{l}^{-1}$ in head injury/ intracranial haemorrhage), serum Prothrombin/APTT ≤ 1.5 times normal, and serum Fibrinogen $\geq 1.0 \text{ g.l}^{-1}$. Risks and complications of massive transfusion in children include volume overload, haemo-concentration (which can increase the risk of thromboembolic events), dilutional coagulopathy of clotting factors in plasma, Transfusion related acute lung injury (TRALI), excessive citrate causing metabolic alkalosis and hypocalcaemia and hyperkalaemia.

During the post-operative period, patients may develop neurological (focal neurological deficits, coma), cardiovascular (arrhythmias, myocardial ischemia, acute right heart failure, cardiovascular collapse) and pulmonary (pulmonary arterial hypertension, pulmonary edema, hypoxia, hypercarbia) complications. Also air in the pulmonary vasculature can trigger the release of inflammatory factors leading to coagulopathy and thrombocytopenia. CT scan is used in detecting air in the dural venous sinuses and cortical veins.

Post-operatively supplemental oxygen and inotropic supports may be required. Hyperbaric oxygen promotes nitrogen re-absorption and reduces the size of the air bubbles.

5. Conclusion

A child undergoing open craniosynostosis repair suffered cardiac arrest due to massive hemorrhage, which required massive blood transfusion, and possible venous air embolism. Rapid recognition of the cause of arrest, initiation of steps to limit further entrainment of air, and quick administration of vasoactive drugs with volume expansion (without chest compression) allowed us, in this instance, to tide over this crisis. Though air embolism may frequently be encountered during craniosynostosis surgery, episodes severe enough to cause cardiac arrest are infrequent and their management is challenging. Our patient

managed to recover from the incident with no significant or lasting neurological sequelae.

6. Conflicts of Interest

There are no conflicts of interest.

References

1. Kurnutala LN, Sandhu G, Stoicea N, Kinthala S, Wei L, Bergese SD. Semisitting Position and Venous Air Embolism in Neurosurgical Patients with Patent Foramen Ovale: A Systematic Analysis. *Int J Anesth Res.* 2016;4(8):305–12.
2. Dalen JE. Pulmonary Embolism: What have We Learned Since Virchow? *Chest.* 2002;122(4):1440–56. doi:10.1378/chest.122.4.1440.
3. Gale T, Leslie K. Anaesthesia for neurosurgery in the sitting position. *J Clin Neurosci.* 2004;11(7):693–6. doi:10.1016/j.jocn.2004.05.007.
4. Lin Y, Pan IW, Mayer RR, Lam S. Complications after craniostomy surgery: comparison of the 2012 Kids' Inpatient Database and Pediatric NSQIP Database. *Neurosurg Focus.* 2015;39(6):E11. doi:10.3171/2015.9.focus15383.
5. Morray JP, Geiduschek JM, Ramamoorthy C, Haberkern CM, Hackel A, Caplan RA. Anesthesia-related cardiac arrest in children: initial findings of the Pediatric Perioperative Cardiac Arrest (POCA) Registry. *Anesthesiology.* 2000;93(1):6–14.
6. Giraldo M, Lopera LM, Arango M. Venous air embolism in neurosurgery. *Colombian J Anesthesiol.* 2015;43(1):40–4.
7. Hubail. Diagnosing a Patent Foramen Ovale in Children Is Transesophageal Echocardiography Necessary? *Stroke.* 2011;42:98–101.
8. Schäfer ST, Lindemann J, Brendt P, Kaiser G, Peters J. Intracardiac transvenous echocardiography is superior to both precordial Doppler and transesophageal echocardiography techniques for detecting venous air embolism and catheter-guided air aspiration. *Anesth Analg* 2008 Jan;106(1):45-54. 2008;106(1):45–54.
9. Shaikh N, Ummunisa F. Acute management of vascular air embolism. *J Emerg Trauma Shock.* 2009;2(3):180–5. doi:10.4103/0974-2700.55330.
10. Disque K. PALS. Pediatric Advanced Life Support Provider Handbook 2015-2020 Guidelines and Standards. vol. 2018. Las Vegas: Satori Continuum Publishing;.
11. National Blood Authority, Australia. Patient Blood Management Guidelines: Module 1-Critical Bleeding Massive Transfusion. Cadman Editing Services Canberra; 2011.

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Cite this article: Menon G, Kumar P, Danial N, George M. Anesthetic challenges involved in successful resuscitation of a child from cardiac arrest secondary to massive hemorrhage and possible venous air embolism while undergoing fronto-orbital advancement surgery for metopic craniosynostosis. *Indian J Clin Anaesth* 2021;8(2):345-348.