CASE REPORT KERALA MEDICAL JOURNAL

Anaesthetic Management of Craniosynostosis Repair

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ABSTRACT

Published on 24th December 2019

Craniosynostosis (craniostenosis) refers to a condition where one or more cranial sutures fuse prematurely leading to focal or global growth delay of the skull. Surgical intervention should be performed early in infancy to prevent the further progression of the deformity and potential complications associated with increased intra-cranial pressure (ICP). A six month old child undergoing Craniostenosis correction developed severe air embolism, and was managed successfully is discussed here.

Keywords: Craniosynostosis, Air Embolism, Paradoxic Air Embolism

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INTRODUCTION

Craniosynostosis (craniostenosis) refers to a condition where one or more cranial sutures fuse prematurely leading to focal or global growth delay of the skull. 1 in 2000 to 1 in 2500 live births may have craniosynostosis.[1] Patients include otherwise normal children with single suture craniosynostosis, undergoing cosmetic procedures or syndromic individuals with multiple abnormalities for functional abnormalities (raise in intracranial pressure (ICP), hydrocephalus, developmental delay, feeding difficulties proptosis and amblyopia).

Surgical intervention should be performed early in infancy to prevent the further progression of the deformity and potential complications associated with increased ICP. Also, the cranial vault of children younger than the age of 9 months is still very malleable, and deformations therefore are easier to correct. Current surgical technique for craniosynostosis consists of either total cranial vault reconstruction or strip craniectomy (endoscopic) followed by molding helmet therapy for 6 to 8 months postoperatively. Despite its elective nature, surgical correction of craniofacial abnormalities carries high risk of extensive blood loss (often significantly more than one circulating blood volume) and other serious Intraoperative and postoperative complications.

Intraoperative death is primarily a consequence of massive blood loss. Anesthetic considerations include associated congenital syndromes (Apert, Pfeiffer, Crouzon syndrome), difficult airway, obstructive sleep apnea, raised intracranial tension , invasive monitoring, difficult airway , considerable blood loss , massive transfusion, DIC, venous air embolism, positional injuries, corneal abrasions, hypothermia, endotracheal tube displacement, head and neck edema due to prolonged surgery.[2] Airway management may be challenging (upper airway obstruction) because of other concomitant craniofacial anomalies. In patients with a difficult airway, spontaneous ventilation should be maintained until the airway is secured. Alternative airway management techniques (e.g., laryngeal mask airway, fiberoptic bronchoscope) should be available. In the presence of increased ICP, high dose sedative premedication, ketamine, and succinylcholine should be avoided. In the presence of increased ICP, specific anesthetic measures must be considered (avoiding hypercapnia, hypoxemia, and arterial hypotension). Large-bore intravenous access is required, and arterial indwelling catheter is recommended to allow for real time blood pressure measurements and repeated blood sampling.

Requirements of blood transfusions should be based on hemodynamic parameters rather than simply on hematocrit values. For craniosynostosis repair, packed red blood cells should be available in the operating room upon skin incision. Techniques to reduce allogenic blood transfusions include cell saver, preoperative acute normovolemic hemodilution, and controlled arterial hypotension. Hypotensive anesthesia is most

Cite this article as: Madhu V, Lal SM. Anaesthetic Management of Craniosynostosis Repair. IMA Kerala Medical Journal. 2019 Dec 24;12(4):97–9.

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often achieved by using volatile anesthetics, opioids, and/or sympatho-adrenergic receptor blocking drugs. However, to maintain sufficient cerebral perfusion pressure, arterial hypotension should not be used in patients with increased ICP.

Positive-pressure ventilation should be initiated once the airway has been secured and the ventilation parameters adjusted to yield normocapnia. Suture the endotracheal tube in place to avoid accidental extubation intraoperatively, which could potentially result in a "cannot ventilate/cannot intubate" situation (due to blood and facial swelling). Facial swelling related to surgery can be quite pronounced (particularly when the surgery extends below the orbital ridge) and require postoperative mechanical ventilation.[3] Never cut the endotracheal tube short. Some surgeons prefer to suture the eyelids together rather than simply keep them closed by self-adhesive tape, which often comes loose during surgical manipulation. Normothermia should be maintained, because infusion of significant amounts of cold fluids (e.g., blood products) may contribute to a rapid drop in body temperature. The use of warmed infusions and convective forced-air warming devices is therefore recommended.

CASE REPORT

A six month old 6Kg infant presented for suturectomy of craniosynostosis. She had no other positive findings in the history or physical examination. Investigations were within normal limits. High risk informed consent was taken. Surgery was done under general anesthesia. 300 ml of packed RBCs was issued. Surgery

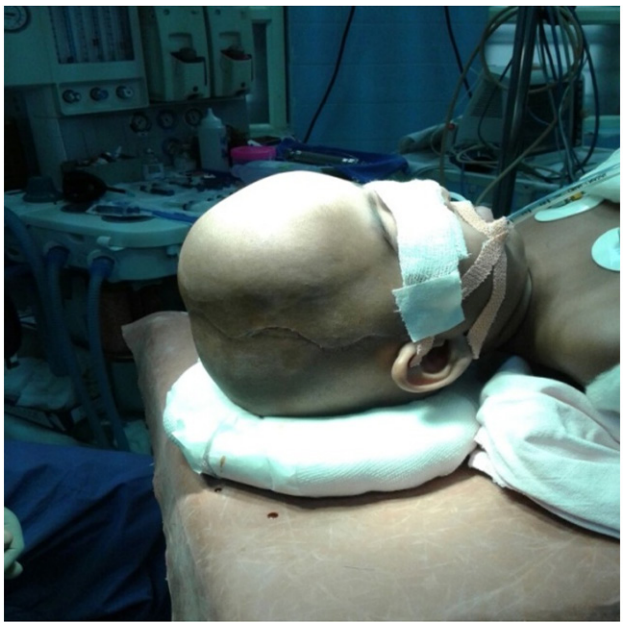

Figure 1. **Craniosynostosis before repair**

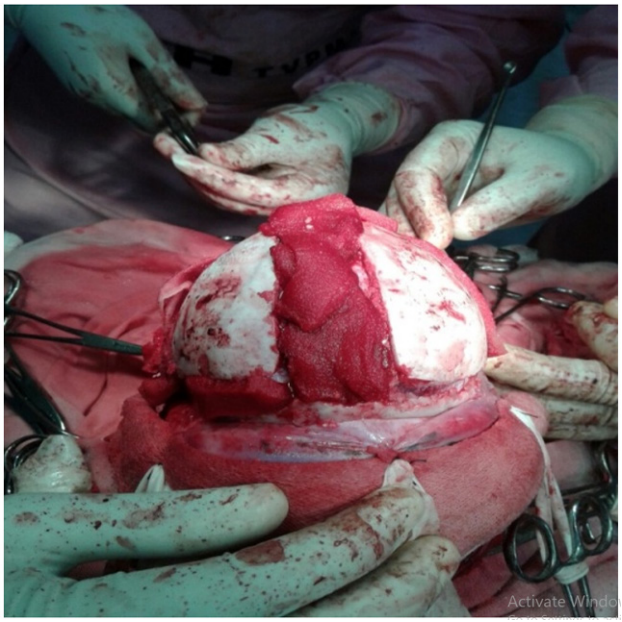

Figure 1. **Craniosynostosis intra-operative**

was done under general anaesthesia with controlled ventilation with endotracheal intubation with Right Angle Endotracheal (RAE) South pole 4mm ID tube with intravenous induction and maintenance with air, oxygen and sevoflurane. Peripheral venous access included 24 Gauge (24G) cannula in left lower limb and 20G cannula in left upper limb. Central venous access was included using 20G Cavafix catheter inserted via right internal-jugular vein and fixed at around 10cm. Intra arterial blood pressure was monitored via left radial artery cannula. Venous access included were 24G cannula in left lower limb, 20G cannula in left upper limb and 20G Cavafix in right internal-jugular vein, fixed at around 10 cm. Intra arterial blood pressure was monitored via left radial artery cannula. Intraoperatively there was a fall in EtCO2 from 35 to 18 mm Hg, and suspected venous air embolism. The surgical field was flooded with saline and baby ventilated with 100% oxygen. Around 30 ml of frothy blood was aspirated via central venous catheter. The EtCO2 slowly rose to 24 mm Hg. The SpO2 rose from 88% to 95% .There was improvement in blood pressure. Blood loss which exceeded 15% of the total blood volume was replaced. The baby was ventilated for 24 hours postoperatively and extubated in ICU on the second postoperative day.

DISCUSSION

The abrupt fall in EtCO2, intraoperative could be due to venous air embolism, endotracheal tube displacement or kinking or severe hypotension. Intraoperative venous air embolism may occur in more than 80% of cases.[4] Early detection of venous air embolism and the presence of a central venous catheter helped

in immediate and successful management of the scenario. Venous Air Embolism- airlock produced in the pulmonary circulation results in a rapid reduction in CO2 excretion (usually together with a fall in oxygen saturation). Arterial pressure decreases and cardiac arrhythmias are frequently seen.

The physiological consequences of venous air embolism depend on the volume as well as the rate of air entry, and whether the patient has a probe-patent foramen ovale (10–25% incidence). The latter is important because it can facilitate passage of air into the arterial circulation (paradoxical air embolism). Air bubbles entering the venous system ordinarily lodge in the pulmonary circulation, where their gases eventually diffuse into the alveoli and are exhaled. Small bubbles are well tolerated by most patients. When the amount entrained exceeds the rate of pulmonary clearance, pulmonary artery pressure progressively rises. Eventually, cardiac output decreases in response to increases in right ventricular after-load. Pre existing cardiac or pulmonary disease enhances the effects of venous air embolism; relatively small amounts of air may produce marked hemodynamic changes. Nitrous oxide, by increasing the volume of the entrained air, can markedly accentuate the effects of even small amounts of air.

Clinically, signs of venous air embolism are often not apparent until large amounts of air have been entrained. A decrease in end-tidal CO2 or arterial oxygen saturation might be noticed prior to hemodynamic changes. Arterial blood gas values may show only slight increases in PaCO2 as a result of increased pulmonary dead space (areas with normal ventilation but decreased perfusion). Major hemodynamic manifestations such as sudden hypotension can occur well before hypoxemia is noted. Moreover, rapid entrainment of large amounts of air can produce sudden circulatory arrest by obstructing right ventricular outflow when intracardiac air impairs tricuspid and pulmonic valve function or blocks pulmonary arterioles.[5]

Paradoxic air embolism can result in a stroke or coronary occlusion, which may be apparent only postoperatively. Paradoxic air emboli are more likely to occur in patients with probe-patent foramen ovale, particularly when the normal transatrial (left > right) pressure gradient is reversed.[6] Reversal of this gradient is favored by hypovolemia and perhaps by PEEP. Some studies suggest that a right > left pressure gradient can develop at some time during the cardiac cycle even when the overall mean gradient remains left > right. Transpulmonary passage of venous air into the arterial system has also been demonstrated and suggests that even small bubbles in intravenous infusions should be avoided in all patients.

A precordial doppler ultrasound probe may be indicated to detect intraoperative venous air embolism. Some anesthesiologists insert a long central venous catheter to evacuate air from the right side of the heart in case of significant air embolism. Nitrous oxide should be avoided.

END NOTE

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Conflict of Interest: None declared

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