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A CLINICAL REPORT OF SUCCESSFUL RESUSCITATION OF PROLONGED DELAYED EMERGENCE FROM GENERAL ANAESTHESIA DUE TO SEVERE RESPIRATORY ACIDOSIS, HYPERCARBIA, HYPOTHERMIA AND HIGH ICP

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ABSTRACT This report describes the case of a 12 year old boy posted for cranioplasty surgery under general anesthesia. Patient had a past history of craniotomy 6 months back for traumatic head injury which was uneventful from anaesthesia point of view. On day of surgery, patient was conscious and mobile (GCS 15) WITH no signs of raised ICP . After induction of the patient using regular institutional protocol , for next 15 minutes his vitals were stable. Suddenly his etco2 started rising to levels of 70 mmHg from initial reading of 32 mmHg. This was followed by bulging of brain from the previous craniotomy defect . Even after Increasing Respiratory Rate the ETCO2 did not come to normal. We changed the work-station (with fresh sodalime) but that too did not help in reducing ETCO2. Giving IV Mannitol did not help. ABG showed severe respiratory acidosis (pH 7.23, PACO2 of 62 mmHg, PAO2 of 168 mmHg, HCO3 of 26 mmol/lit) so surgery was postponed and it was decided to shift the patient to PICU in intubated state and continue ventilation . In PICU the patient was kept on controlled ventilation with VCV mode. Close monitoring of ETCO2 was done and regular ABGs were taken. In the second hour ETCO2 started reducing and the child started gaining the consciousness. Gradually the ventilator was weaned. The child was extubated after ABG was normal, and full consciousness was gained . He was shifted to ward the next day and discharged 1 day later. He was given a future date for surgery which went smoothly . The prompt recognition of developing hypercarbia and respiratory acidosis during surgical period led to the rapid management and recovery of the patient.

KEYWORDS : Delayed emergence, Cranioplasty, Hypercarbia, Respiratory acidosis, Paediatrics

INTRODUCTION

Delayed emergence from general anesthesia (GA) is a rare but serious complication that can occur in pediatric patients undergoing neurosurgical procedures. It is medically defined as a state of unresponsiveness from which the patient cannot be aroused.[1] Delayed emergence from general anaesthesia is often multifactorial and the main cause can be due to physiological causes (hypoglycaemia, hyperglycaemia, dyselectrolytaemia, hypothermia, cerebral hypoxia, intracerebral event) or pharmacological causes (opioid/benzodiazepine over dosage, central nervous system depressants, residual neuromuscular blockade).[2] In addition, the occurrence of delayed emergence usually relates to an underlying undiagnosed condition or medical error.[3] Recognizing organic conditions is important as they require prompt surgical intervention to reverse serious sequelae, especially in head surgery, as this this type of surgery may be associated with serious postoperative complications. Of course, accurately collecting information on patient interview and checklists to ensure that all information exists can also reduce the risk and harm to patients. This current case report describes a paediatric male patient with delayed emergence and respiratory acidosis from general anaesthesia during cranioplasty surgery.

CASE REPORT

A 12-year-old boy with a past history of craniotomy six months back presented in pre-anaesthesia checkup clinic in Parul Sevashram hospital for cranioplasty surgery under general anesthesia. He had undergone craniotomy six months back for traumatic head injury. Preoperatively, the patient was fully conscious and mobile with a glasgow coma scale (GCS) score of 15. No other significant past history was there.Patient was taking oral anticonvulsant drugs regularly. He was not taking any other medications.

Physical examination revealed a surgical scar in scalp (due to previous surgery - Right fronto-tempoaral craniotomy) and a post surgical defect in the scalp . Neurological examination was normal. His body weight was 35kg.Preoperative blood investigations and electrocardiogram were within normal limits. Preoperative CT Scan of Brain revealed post-operative defect involving Right fronto-parietal bones. No other significant finings were noted. Hence a Cranioplasty was planned under General Anaesthesia. The Skull bone to be placed back was kept safe in sterile environment (ETO sterilized).

On the day of surgery, the patient received morning dose of anticonvulsants with sip of water. High risk consent for surgery and post operative ICU stay with ventilatory support ws taken. Premedication was given with Inj Glycopyrrolate and Inj Ondansetron according to body weight.

His initial Blood Pressure was 109/76 mmHg, heart rate was 89 beats/min and peripheral oxygen saturation SpO2 was 98%.

Anaesthesia was induced with 70 mg Propofol (slow single intravenous injection) and 35 mcg fentanyl (slow single intravenous injection) Intravenously. Rocuronium 30 mg was administered to facilitate endotracheal intubation (single intravenous injection) and intermittent positive pressure ventilation was commenced . 6mm ET tube was inserted. Balanced anaesthesia was maintained with an Sevoflurane in 60% Oxygen with air. Intermittent Rocuronium was given to maintain the level of muscle relaxation . Intraoperatively, end-tidal carbon dioxide concentration, volatile anaesthetic concentration, minimal alveolar concentration, fraction of inspired and expired oxygen concentration, and degree of neuromuscular blockade were monitored. Initial value of ETCO2 was 34mmHg. After induction, the surgical position was given. Surgical team was asked to start preparations for painting and draping the part to be operated.

Figure-1: The Bulge Seen At Site Of Surgery

Fifteen minutes after induction of anesthesia, the patient's end-tidal carbon dioxide (ETCO2) levels started rising rapidly, reaching 70 mmHg within 20 minutes . Heart rate ,Blood pressure also started rising.Bulging of Brain (from the previous Craniotomy defect) was evident [Figure-1]. So Neurosurgical team was called upon and it was discussed to wait for some time before starting the surgery. Meanwhile we changed the anaesthesia workstation with freshly filled soda lime, changed ventilatory settings to assist in reducing the ETCO2 (i.e Increasing respiratory rate to 24 from initial setting of 18; increased I:E ratio from 1:2 to 1:3). To reduce the ICP , we gave Inj Mannitol 25grams over 15-20 minutes. Initial ABG sample was sent for blood gas analysis. ABG revealed pH of 7.23, PCO2 62mmHg, PO2 168mmHg, HCO3 26mmol/L. This was suggestive of severe respiratory acidosis . To avoid any serious neurological damage due to high ICP it was decided to postpone the surgery and primary focus was now to correct the respiratory acidosis and normalize the ETCO2.

Hence the patient was shifted to the pediatric intensive care unit (PICU) in an intubated state for continuation of ventilation and monitoring. While shifting the patient to PICU, CT Brain and HRCT thorax were also done . CT Brain was sugestive of minimal herniation from the surgical site (as compared to previous CT Brain). HRCT thorax was normal.

In the PICU, the patient was placed on controlled mechanical ventilation with volume-controlled ventilation (VCV) mode. Continuous monitoring of ETCO2 , Heart Rate, Blood Pressure, SPO2 was done. ABG was taken again which revealed worsening of respiratory acidosis. (pH 7.13, PCO2 85mmHg, PO2 173 mmHg, HCO3 28 mmol/L). This increase can be due to use of Bains circuit during shifting.Ventilatory settings were re-adjusted. We continued ventilation for some time. Throughout the resuscitation his vitals were monitored . The relatives were also counselled and reassured time to time. After continuous ventilation for an hour, the ETCO2 started to settle down to 55-60 mmHg range. His vitals were also improving. In the meanwhile Pulmonologists and Neurophysicians were consulted . Pulmonologist advised to continue the ventilation and repeat ABG after 1 hour. Neurophysician team advised to continue the ventilation . Gradually the ETCO2 settled down to range of 45-48mmHg. At this time another ABG was sent which revealed pH 7.44, PCO2 37mmHg, PO2 147mmHg, HCO3 27 mmol/L. We continued ventilation with same settings. Gradually, patient started to gain consciousness , and ETCO2 levels subsided. He was then put on SIMV mode to assist ventilation. In next 1 hour he gained consciousness and his muscle tone also improved. So a trial for CPAP mode was given which was tolerated well by the patient. After 3 hours of CPAP trial a t-piece support was kept and preparations for extubation were started. A final ABG was sent for before extubation. It revealed pH 7.42 PCO2 36 mm Hg , PO2 95mmHg, HCO3 26 mmol/L. After discussion with paediatric intensivist we extubated the patient. Post extubation he was kept in 15 degree head up position throughout the night with oxygen support of 2L/min via nasal prongs.

Next day the patient was shifted to the room and kept under monitoring. He was discharged a day after. He was given a future date for surgery. The surgery was conducted 1 month after this event and it went uneventfully.

The study was approved by the Ethics Committee of Parul Sevashram Hospital, Affiliated to Parul University. Vadodara ,India . The patient was informed of the anaesthesia method that would be used before the operation and provided written informed consent for anaesthesia. Written informed consent was also obtained from the patient for the publication of this report and any accompanying images.

DISCUSSION

At present, with the widespread use of short-acting anaesthetic agents, patients usually awaken quickly after General Anaesthesia. If disturbance of consciousness persists, anaesthesiologists must pay more attention to the accurate diagnosis of the underlying cause, especially in head surgery, as this type of surgery can carry a potential risks. Cranioplasty is an established procedure for post-craniotomy patients . It is generally an uneventful surgery with minimum blood loss if ICP remains within normal range. If due to some reason ICP rises, it can cause a potential havoc during surgery and even after surgery as high ICP gets transmitted to brain tissue after Cranioplasty is done. Unfortunately, the current patient gradually developed high ETCO2 and raised ICP following induction of anaesthesia. The detailed medical history inquiry by the anaesthesiologists did not reveal any positive finding.[4]

Delayed recovery from anaesthesia is feared by all anaesthesiologists. It is generally believed that the main cause of delayed awakening following anaesthesia is an overdose of anaesthetic agents administered in the perioperative period or hyopventilation causing CO2 narcosis. In our case the main cause seems to be with the ventilation . Although we tried to change the workstation, it didn't help. Other cause could have been hypothermia. Since it is a paediatric case, they are more prone to hypothermia. Hypothermia was taken care of by using warmer at 40 C throughout the procedure which might have been inadequate.

The changes in pharmacokinetics and pharmacodynamics of paediatric patients result in a diminished rate of elimination, hence the drug dose administered should be reduced especially opioids and benzodiazepines. Studies have shown that the demand for opioids should be strictly titrated to body weight in paediatric patients. Careful titration of the dose based on the individual response to reduce adverse effects of general anaesthetics,opioids and benzodiazepines is especially important in paediatric patients. In current case, Anaesthesia was induced with 70 mg Propofol and 40 mcg Fentanyl. Inj Rocuronium 30 mg was administered to facilitate endotracheal intubation and intermittent positive pressure ventilation was commenced . Balanced anaesthesia was maintained with an sevoflurane in 60% oxygen with air. Intermittent Rocuronium was given to maintain the level of muscle relaxation . For a 12 year old child, weighing 35kg weight, doses could have been reduced further by titrating the effect.[5,6]

Some underlying physiological or metabolic disorders such as hypoglycaemia, hyperglycaemia, dyselectrolytaemia, hypothermia, cerebral hypoxia and intracerebral events may also be responsible for delayed recovery following anaesthesia. In current case preoperative investigations were normal hence above causes were ruled out.

In The Current Case, The Following Errors Might Have Occurred:

1. Overdose of anaesthetic agents may have been administered.
2. Htpothermia could have been dealt in a better way.
3. Hypoventilation might have caused CO2 narcosis
4. Larger diameter ET tube could have helped in better ventilation (6.5mm instead of 6mm)

Any single error in a procedure could be mitigated by other procedures, but all the above errors eventually led to the final adverse event. The delayed recovery in this case was likely multifactorial, involving severe respiratory acidosis, hypercarbia, hypovolemia, hypothermia, and high ICP. The rapid rise in ETCO2 indicated inadequate ventilation, possibly due to impaired lung function, increased metabolic rate, or inadequate ventilator settings.

In clinical anaesthesia, anaesthesiologists often face the problems of high workload and a highly effective flow process that affect the safety of anaesthesia, as well as problems of staff shortage, unskilled staff, equipment failure, careless shift handover and poor communication. Each of these may lead to the occurrence of adverse events in the perioperative period [4,6] .The current case highlights the importance of the careful titration of the anaesthetic dose in paediatric patients based on an individual's response.. In addition, multiple checks of the patient information, surgical safety checklist and medical history by anaesthesiologists, surgeons and nurses can minimize the chance of an adverse outcome.

Key points

Delayed recovery from general anesthesia in pediatric patients undergoing neurosurgical procedures is a rare but serious complication. Prompt recognition and management of contributing factors such as respiratory acidosis, hypercarbia, hypovolemia, hypothermia, and increased ICP are crucial in preventing adverse outcomes. Close monitoring and appropriate interventions, including controlled ventilation, are essential in managing delayed recovery and ensuring patient safety.

CONCLUSION

This case highlights the successful resuscitation of prolonged delayed recovery in a pediatric patient undergoing cranioplasty surgery under general anesthesia. Prompt recognition of contributing factors and

appropriate management, including controlled ventilation and supportive care in the PICU, led to the patient's successful recovery and discharge.

REFERENCES

1. Hambrecht-Wiedbusch VS and Mashour GA. Accelerated Recovery of Consciousness after General Anesthesia Is Associated with Increased Functional Brain Connectivity in the High-Gamma Bandwidth. Front Syst Neurosci 2017; 11: 16.
2. McCarthy G, Szenohradszky J, et al. Influence of chronic phenytoin administration on the pharmacokinetics and pharmacodynamics of vecuronium. Anesthesiology 2004; 100: 626–633.
3. Joshi SA, Misal US, and Shaikh MM. Delayed recovery from anesthesia: A postgr aduate educational review. Anesth Essays Res 2016; 10: 164–172.
4. Liu Y, Huang H. Clinical report of delayed recovery after general anaesthesia in elderly patients with cervical spine surgery: A case report. J Int Med Res. 2022 Oct;50(10):3000605221134462. doi: 10.1177/03000605221134462. PMID: 36300317; PMCID: PMC9619878.
5. Frost EA. Differential diagnosis of delayed awakening from general anesthesia: A review. Middle East J Anaesthesiol. 2014;22:537–48. [PubMed: 25668997]
6. Sinclair R, Faleiro RJ. Delayed recovery of consciousness after anaesthesia. Contin Educ Anaesth Crit Care Pain. 2006;6:124–8