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Case Report

Cardiac arrest in a child during a combined general epidural anesthesia procedure

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An increased risk of perioperative cardiac arrest in children, in comparison to adults, has been recognized. A number of factors associated with perioperative cardiac arrest have been identified, including young age, comorbidities, and emergency surgery. Since anesthesia-related cardiac arrest is uncommon, a multi-related database is required to understand the mechanisms of cardiac arrest and to develop preventive strategies. Most cardiac arrests occur during induction (37%) or maintenance (45%) of anesthesia, usually following one or more of the following antecedent events, i.e., bradycardia (54%), hypotension (49%), abnormality of oxygen saturation as measured by pulse oximetry (48%), inability to measure blood pressure (25%), abnormality of end-tidal CO_2 (21%), cyanosis (21%), or arrhythmia (18%). In 11% of cases, cardiac arrest occurred without recognized warning.[1] There are only few reports in the literature, and in Kariadi Hospital, none has ever been reported. The aim of this report is to identify and discuss possible causes of cardiac arrest and to anticipate its complications.

Report of the case

A 4.5 year old, 14 kg boy was undergoing an elective herniotomy to correct his inguinal hernia. His medical history was non-specific; vital signs were normal. On physical examination the lungs were clear and heart sounds were normal. Everything was normal except for a borderline hyperkalemia (K=5.1 mEq/l). The anesthetic management consists of an inhalation induction and atropine sulfate 0.02 mg/kg as preanesthetic medication, with midazolam 0.1 mg/kg and ketamine 3 mg/kg, coadministered in one syringe intramuscularly before induction. As the patient was calm, he was taken to the operating room for induction of anesthesia.

In the operating room, the patient was on electrocardiography (ECG), pulse oximetry, and automated blood pressure monitor prior to anesthetic induction. Vital signs prior to induction showed a blood pressure of 95/46 mmHg, heart rate (HR) of 98 beats/minute (bpm), and respiratory rate of 20 breaths/minute. Preoxygenation was initiated prior to induction.

Anesthesia was induced with the initial inhalation of 50% nitrous oxide and 50% oxygen at a total flow of 6 l/minute. Halothane was introduced at 0.5% and was increased in 0.5% increments approximately every three breaths. The induction progressed

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smoothly. The halothane vaporizer concentration had reached 3.5% and was decreased to 2% upon intravenous (IV) catheter placement. The blood pressure was then 102/54 mmHg and the ECG revealed a normal sinus rhythm of 75 bpm. Atropine 0.02 mg/kg was administered intravenously. Heart rate increased to 145 bpm. After using xylocaine spray on the oropharynx, tracheal intubation was facilitated by deepening the anesthesia. The trachea was intubated using a non-cuff Portex® endotracheal tube. After the breath sounds in both lungs were confirmed, the tube was then fixated and an oropharyngeal pack/mouth pack was inserted. While maintaining the anesthesia with spontaneous ventilation, we tried to initiate caudal block for caudal analgesia in the left lateral position. While identifying the caudal epidural space using a 23 gauge needle, bradycardia with HR of 54 bpm and hypotension with BP of 62/45 mmHg occured. Soon oxygen saturation decreased until it was immeasurable; so did the blood pressure. Heart rate was aproximately 30-40 bpm. The patient then became pulseless for about 2 minutes. As the bradycardia and hypotension occured, we abandoned the caudal block attempt, turned the patient back to supine position, immediately administered atropine 0.5 mg IV, and performed fluid challenge and resuscitation, with no response. Spontaneous ventilation was still present. Immediately after discovering that the patient was pulseless, we performed CPR with the patient in a supine, slightly head-down position. Adrenaline was given every 3 minutes while continuing CPR, starting from 10 mg/kg (1:10,000) and increased up to 100 mg/kgBW (1:1000). We also gave defibrillatory shocks of 25 joules, 25 joules, and 50 joule, consecutively, because a ventricular fibrillation ECG pattern was seen, followed again by adrenaline and CPR. After this, we gave three defibrillatory shocks of 50 joules, but the ECG showed an asystole pattern. After an hour of CPR, we stopped and decided to declare it as failed resuscitation.

Discussion

Medication-related problems are the most common cause of cardiac arrest, accounting for 37% of all cases. Cardiovascular depression from the administration of halothane, halothane plus an intravenous medication (fentanyl, bupivacaine, lidocaine, propranolol, sufentanil, or thiopental), or sevoflurane was regarded as the primary cause in 71% of all medication-related cases. The median age for the 37 cardiac arrests related to halothane (either alone or in combination with an intravenous medication) was 8 months (range 5 days-7 years). These patients were in American Society of Anesthesiologists (ASA) physical status 1-2 and 3-5 (because of congenital heart disease); but had an unrecognized cardiomyopathy. The median halothane concentration was 2%; some patients received 3-3.9%, and others received 4% or more halothane just prior to arrest. Sixty-six percent of halothane-related arrests occurred during induction, and 34% occurred during the maintenance phase. The most common antecedent events were bradycardia and hypotension and abnormality in oxygen saturation as measured by pulse oximetry. The most common associated factors were assisted or controlled ventilation and difficult intravenous access requiring multiple attempts. Patients were successfully resuscitated and recovered without permanent injury. Epinephrine alone, atropine alone, or epinephrine plus atropine were the drug treatments usually associated with the return of adequate circulation.[1,2,3]

Arrests related to cardiovascular depression from sevoflurane occurred during induction of anesthesia in ASA physical status 3 children, preceded by hypotension and bradicardia. Some children were successfuly resuscitated and survived without sequelae.[4,5]

In some cases, the associated factors contributing to cardiovascular depression by halothane were the use of assisted or controlled ventilation, which accelerated the rise of halothane concentration in the blood and myocardium or reduced the cardiac output by impeding venous return. Similarly, difficulty in intravenous access may have resulted in prolonged exposure to high concentrations of inspired halothane. Patients with significant underlying disease such as congenital heart disease may tolerate poorly any change in cardiac output caused by halothane-induced reductions in heart rate or myocardial contractility.

It remains unclear at this time whether the increasing popularity of sevoflurane as an induction agent in children will have an impact on the number of reports of cardiac arrest attributed to inhalation

agents. It has been reported that compared to halothane, sevofurane has less potential for producing bradycardia and myocardial depression. Because denominators are not available, the incidences for halothane and sevoflurane-related cardiac arrests cannot be calculated.

Most cases of accidental intravascular injection of local anesthetics occurred during attempts at caudal injection despite negative aspiration and absence of response to a test dose. Incremental dosing of local anesthetic into the epidural space has been recommended over bolus injection to allow earlier detection of intravascuiar injection, although this has not been studied systematically. Alternatively, use of local anesthetics with less myocardial toxicity than that associated with bupivacaine might be recommended.[1,3,6]

Regional anesthesia and nerve blocks are now used widely in children, mostly in conjunction with general anaesthesia. They provide part of the anaesthetic and significantly improve patient comfort in the postoperative period. Many advances have been made since Curwen in Durban, South Africa presented an article in 1930 on 99 caudal blocks in neonates. He concluded his article wondering if he had done something worthwhile or was it a technique that would be regarded as ridiculous.

In Melbourne, caudal block has been used regularly in children for 30 years. Adaptations from adult practice, particularly epidurals with catheters, and more recently spinals for premature infants, development of block techniques resulting from anatomical studies, and better understanding of the drugs used and their safety through measurement of blood concentrations, have led to improvements in clinical practice.[8,9]

Sudden cardiac arrest during anesthesia in apparently healthy children may occur in the absence of anesthetic overdose due to various preexisting conditions that may not be readily clinically apparent. Arrhythmias during anesthesia induction may be caused by factors such as hypoxia, hypercarbia, and stimulation during inadequate anesthesia. When undiagnosed congenital cardiac anomalies, such as mitral valve prolapse, are exacerbated by anesthetic induction, arrhythmias and cardiovascular collapse may result. Ventricular bigeminy, multifocal premature ventricular contractions, and ventricular tachycardia have been reported during anesthesia in a pediatric patient who was subsequently diagnosed with mitral valve prolapse.[1,2,10]

The association of ventricular arrhythmias with halothane, hypercarbia, and atropine cannot be ignored in the present case. Ventricular extrasystoles are more common in children receiving intravenous atropine during halothane anesthesia with spontaneous ventilation than in those who did not receive atropine. However, in a more recent study of intravenous atropine administration during halothane anesthesia, no ventricular extrasystole was noted. In this study, ventilation was often assisted or controlled. In a study of cardiac arrhythmias in pediatric patients, there were significantly more ventricular arrhythmias in children whose airways were managed with a mask than in those managed by endotracheal intubation. Additionaly, there was a strong relationship between ventricular arrhythmias and hypercarbia in children whose airways were managed using a face mask. Unfortunately, the timing of the arrhythmias and the use or nonuse of atropine were not reported.[1,10]

Cardiac arrest during anesthesia is usually preceded by bradycardia. The most common causes of arrest in infants and children are anesthetic overdose and failure to ventilate and oxygenate.

Non-ventricular fibrillation or tachycardia

Profound bradycardia or asystole is the most common dysrhythmia associated with cardiac arrest in infants and children. Profound bradycardia should therefore be treated in the same way as asystole. The treatment consists of an initial dose of adrenaline of 10 mg/kg IV or by the intraosseous route (or 10 times this dose via a tracheal tube if venous access has not been established). Recent studies in animals and children have suggested the benefit of a higher dose of adrenaline for the unresponsive asystolic child. Therefore, should the child not respond to the initial dose, another dose of 100 mg/kg is recommended. Further studies have shown that if the child does not respond to this higher dose, the eventual outcome is likely to be poor. In reviewing the results of these studies, no children survived to discharge of those given more than two doses of adrenaline.

When there is a cardiac rhythm but no cardiac output (electromechanical dissociation/pulseless elec-

trical activity) it is also necessary to treat any of the underlying reversible causes of the arrest. Hypoxia, hypovolemia, tension pneumothorax, cardiac tamponade, drug overdose, hypothermia, and electrolyte imbalance are all treatable, and resuscitation attempts should not be abandoned until a reasonable attempt has been made to correct these potentially reversible causes of cardiac arrest [4,5,7].

Ventricular fibrillation and tachycardia

These rhythms, although common in adults, are relatively rare in infants and children. Although one study reported an incidence of ventricullar fibrillation of 23% in children, most other studies have reported an incidence of 0-10%. Nonetheless, the physician must always be aware of the occasional need to treat ventricular fibrillation in children by defibrillation.

The recommended sequence is to give two rapid defibrillatory shocks of 2 J/kg, followed by a single shock at 4 J/kg. All further defibrillation attempts should then be made at 4 J/kg in a rapid repeated series of three shocks. After the first cycle of three defibrillation attempts, adrenaline 10 mg/kg should be given and another dose of 100 mg/kg after the second cycle of three shocks and between all subsequent cycles. When ventricullar fibrilation occurs in children there is often an underlying cause and correction of hypothermia, drug overdose (tricyclic antidepressant overdose) and electrolyte imbalance (hyperkalemia) should be considered [4,5,7].

Halothane produces a dose-dependent reduction in cardiac output that principally reflects decreases in stroke volume owing to reductions in myocardial contractility. Depression of myocardial contractility produced by nitrous oxide is about one half that produced by a comparable concentration of a volatile anesthetic. As we know, the ability of volatile anesthetics to reduce the dose of epinephrine necessary to evoke ventricullar cardiac dysrhythmias in greatest with halothane. Halothane also slows the rate of sinoatrial node discharge and prolongs Purkinje fibre and ventricullar conduction time. Indeed halothane has negative inotropic, vasodilating, and depressant effects on the sinoatrial node which are similar to the effects produced by calcium entry blockers [11].

The cardiovascular effects of ketamine when administered in the presence of halothane may results in hypotension. Halothane, by decreasing the release of norepinephrine, could direct allow the cardiac depressant effect of ketamine to manifest. Ketamine causes a dose-dependent decrease in halothane anaesthetic requirement (MAC). Halothane prolongs the duration of action of ketamine by delaying redistribution and metabolism [12].

The cardiotoxic effects of excessive potassium or hyperkalemia are very important. Impaired excitability, automaticity, conductivity, and contractility may result. Signs and symptoms of increased serum potassium are mental confusion, sense of weakness, and, most importantly, bradycardia and irregular rhythms, peripheral vascular collapse and hypotension, muscular paralysis, and cardiac arrest [13].

Since the attempt of caudal block had been stopped at the time of identification of the caudal epidural space, we presumed that hypotension, bradycardia, and cardiac arrest with ventricular fibrillation and ventricular tachycardia during CPR may be related with medication and cardiovascular pathology of undetermined etiology and hyperkalemia.

Conclusion

The cardiotoxic effect of hyperkalemia and general anesthesia may have caused the cardiac arrest in this case. Furthermore, the cardiodepressant effect of ketamine was augmented by halothane and nitrous oxide.

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