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Case Report

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Malignant Hyperthermia - A Case Report of a Paediatric Patient

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Abstract

We encountered a case of malignant hyperthermia (MH) in a 3-year-old boy during general anesthesia induction for laparotomy. It has been generally reported that sevoflurane can induce the delayed onset of MH in the absence of succinylcholine. Our case of MH was elicited after about 90 mins of sevoflurane administration with 50/50% Air/O_2 mixture. However the patient was successfully treated by early recognition of his condition and supportive treatment.

Keywords: Malignant Hyperthermia, Sevoflurane, Dantrolene

Introduction

Malignant hyperthermia (MH) is a chain of symptoms that are triggered in susceptible individuals by commonly used inhalational agents such as isoflurane and sevoflurane and also by muscle relaxants such as succinylcholine. The syndrome also occurs upon vigorous exercise and exposure to heat. Malignant hyperthermia is a hypermetabolic syndrome characterized by hyperthermia, hypercarbia, tachycardia, acidosis, muscle rigidity and rhabdomyolysis. It is known that sevoflurane and desflurane are less potent triggering agents as compare to other inhalational agents and they produce a more gradual onset of MH [1,2]. Yet we encountered a case of MH during anesthetic induction with sevoflurane, and the patient was successfully treated by early recognition and supportive measures only.

Case Report

A 3-year-old, 12 kg boy was scheduled for laparotomy for suspected intestinal obstruction. Neither the patient nor his family had any history of neuromuscular disease or a special family history. The preoperative laboratory examinations were within the normal values. The preoperative vital signs were blood pressure: 100/60 mmHg, heart rate: 106 beats/min, respiratory rate: 26/min and axillary temperature: 37.0°C. Anesthesia was induced with sevoflurane 2.5 vol% by mask ventilation in a mixture of air and oxygen (FiO_2 0.5). 10 mg of atracurium was injected during induction. About 2 min after injection of atracurium, a size 4.0 cuffed endotracheal tube was inserted without any difficulty under direct laryngoscopy. Two large bore IV lines maintained and Foley's catheter for checking the hourly urine output. Then 1 min after intubation, the heart rate of patient was increased from 106 to110 beats/min. At first, the tachycardia was considered to be due to stimulation by the tracheal intubation, but the end tidal carbon dioxide concentration ($ETCO_2$) was concurrently increased from 4.0 to 5.0 mmHg within 2 hrs. The oral temperature was increased to 36.0 to 39.1°C within 3 hrs after induction. No significant changes in the muscle tone and skin appearance were noted. We suspected MH, so we stopped using sevoflurane. The patient was hyperventilated with 100% O_2 through a new anesthetic circuit. After discontinuing the sevoflurane and we started propofol infusion TIVA as maintenance because propofol is known to be a safe anesthetic agent in patients with MH. For decreasing the body temperature 1 g paracetamol was given, active cooling was immediately initiated by cold IVF, Cold sponging, bed cooler at 10 C. This was done via external jugular cannulation for rapid infusion of cold IV fluid and central administration of drugs for resuscitation if it was need. The potassium levels in the serum were normal. 4 hrs after anesthetic induction, the patient showed an oral temperature of 39.0°C, a pulse of 180 beats/min, a blood pressure of 150/110. So, we decided to stop the general anesthesia and started on supportive treatment. Approximately 30 mins after the supportive treatment started the patient showed an oral temperature of 38.2°C, a heart rate of 140 beats/min and a blood pressure of 140/100 mmHg. Thereafter the patient maintained an oral temperature of 36.5-37.4°C and a normal blood pressure and heart rate. The patient was transferred intubated to the intensive care unit for further observation. Discharge was given on the seventh day without any sequelae.

Discussion

Malignant hyperthermia (MH) is a rare disorder, occurring in 1 per 5,000 to 1 per 50,000 – 100,000 anesthetic procedures and is usually fatal if untreated. The incidence in pediatrics is 1 event per 10 000 surgeries [1]. The pathophysiology of MH is still unclear. During an acute episode of malignant hyperthermia, intracellular calcium increases in skeletal muscle which causes spasmodic muscular contractions. It may be due to a defect in the RYR1 gene for the ryanodine receptor-a calcium channel receptor in the sarcoplasmic

reticulum. This abnormal receptor then releases excess calcium once triggered by specific agents particularly succinylcholine and the halogenated inhalational agents [1,2]. Dantrolene inhibits the release of calcium from sarcoplasmic reticulum by binding to the ryanodine receptors, thereby halting the uncontrolled muscle contractions [3]. The mortality rate from MH is reduced from 80% to less than 10% after the introduction of dantrolene. There are multiple case reports of malignant hyperthermia in the west but Denborough reported the first case in 1960 [4]. Many early signs of an acute MH episode can present in various ways and the syndrome may be confused with other conditions such as an inadequate anesthesia, hypoxia, hypercarbia and endocrine disorders like thyrotoxicosis and pheochromocytoma. Neuroleptic malignant syndrome was also ruled out as the patient was not taking any other medications [5-7]. A clinical grading scale helps to establish the likelihood of MH in specific problematic cases [8]. It is based on assigned scores for muscle tone, temperature, tachycardia, muscle breakdown, acid-base parameters, arrhythmias and the response to dantrolene. We were able to diagnose MH on the basis of the clinical symptoms and the clinical grading scale by Larach et al. Early detection and prompt supportive management can save the patient without any catastrophe [8]. There was no family history and also the preoperative laboratory studies suggested the patient was susceptible to MH. Several genes have been implicated rather than MH being a single gene defect. Genetic testing for malignant hyperthermia is not available in Pakistan presently and could not be done in this patient [9]. The halothane and caffeine contracture tests are bioassays and they currently remain the gold standard tests but they are not generally used in our country, and so a diagnostic contracture test was not performed. Recently it has been discovered that intramuscular injection of caffeine can be given and the $ETCO_2$ concentration is measured. However there is a probability of pre-cipitating an acute MH attack. Microscopic examina¬tion of the muscle biopsy is also suggestive of MH [10]. Shulman et al. first reported MH in swine caused by sevoflurane, later on it was also reported in the humans during sevoflurane anesthesia [11]. Generally, sevoflurane and desflurane have been reported to be less potent triggers but they produce a more gradual onset of MH [12,13]. However in another report from Pakistan the patient could not survive in the absence of dantrolene [14]. Although malignant hyperthermia is a rarely occurring syndrome but its consequences are grave and mortality is very high in the absence of dantrolene, therefore it must be made available in all the tertiary care set ups of Pakistan so that no patient die because of its non-availability.

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