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Treatment and nursing care of a patient diagnosed with malignant hyperthermia after general anesthesia: a case report

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Abstract

Malignant hyperthermia (MH), characterized by severe myoclonus, pyrexia, tachycardia, hypertension, elevated muscle enzymes, and hypercapnia, often occurs in patients with congenital deformities or genetic disorders. Although the reported incidence rate is as low as 1:5000 to 1:100,000, patients with MH exhibit rapid aggravation and an elevated mortality rate. Thus, MH is associated with substantial perioperative risk. Successful treatment of patients with MH largely depends on early diagnosis and timely effective treatment. This clinical report provides a detailed description of a patient with newly diagnosed MH who developed a rapid rise in body temperature, end-tidal carbon dioxide, and heart rate during maxillary osteotomy. After successful rescue, the patient recovered smoothly during the postoperative period, indicating the importance of intraoperative monitoring, early diagnosis, effective treatment, and postoperative monitoring. This case is expected to serve as a reference for future interventions and healthcare practices in managing other patients with MH.

Keywords

Malignant hyperthermia, perioperative period, nursing care, general anesthesia, case report, dantrolene sodium, *RYR1* gene

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Introduction

Malignant hyperthermia (MH) is an infrequent autosomal dominant genetic disorder triggered by volatile anesthetics such as sevoflurane and isoflurane or depolarizing muscle relaxants such as succinylcholine. Prolonged muscular rigidity results in substantial energy expenditure and rapid elevation of body temperature,[1] thereby increasing metabolic activity. Despite its low incidence rate, MH induces rapid disease aggravation and an elevated mortality rate.[2] Timely diagnosis and effective treatment can significantly decrease the mortality associated with MH.

The clinical features of MH include pyrexia, tachycardia, hypertension, elevated levels of muscle enzymes, and hypercapnia. MH is also characterized by muscle rigidity, spasm, and rhabdomyolysis.[3] Susceptibility to MH typically arises from congenital deformities or genetic disorders, and *RYR1* gene mutations account for approximately 50% to 70% of cases.[4] Patients with MH exhibit abnormal functioning of the RYR1 protein, which leads to prolonged opening of calcium ion channels and excessive release of calcium ions from the sarcoplasmic reticulum when exposed to certain drugs. Impaired recycling and release mechanisms of calcium ions result in intracellular calcium overload, leading to persistent and intense contractions of skeletal muscles. MH generally manifests during or after surgery and may appear during anesthetic recovery.[5] Therefore, timely and effective treatment throughout the perioperative period is essential for successful management.[6]

Our hospital effectively managed a patient with MH and provided comprehensive support throughout the patient's recovery period. The detailed analysis of this case presented herein can serve as a valuable reference for the treatment and care of future patients with MH. All patient details have been removed to ensure patient privacy, and written informed consent was obtained from the patient prior to treatment. Because all patient details were de-identified, written informed consent for publication was not required. In addition, because of the nature of this study (case report), formal ethics committee approval was not required. The reporting of this study conforms to the CARE guidelines.[7]

Clinical information

General information

A man in his mid-20s weighing 66 kg was diagnosed with maxillary retrognathism, mandibular protrusion, and cleft lip and palate. The patient had no history of drug allergy or infection and no family genetic history, and no obvious abnormalities were found in the preoperative examination. On 5 January 2024, tracheal intubation was performed under general anesthesia, and Lefort type I osteotomy of the maxilla was performed with elongation of the midline of the maxillary palate and formation of a fascial tissue flap.

Pathogenesis and rescue process

The patient underwent surgery at 12:20 on 5 January 2024. The intraoperative anesthetic drugs included inhaled sevoflurane, fentanyl hydrochloride injection, propofol emulsion injection, and furosemide injection. Succinylcholine was not administered. Near the end of the operation at 13:25, the patient's end-tidal carbon dioxide suddenly increased from 45 to 85 mmHg, his body temperature rose to 38.4°C, and his heart rate increased to 138 beats/minute. The sevoflurane inhalation was immediately stopped, and propofol was intravenously injected. The anesthesia machine and respiratory circuit were replaced with new ones, and a venous channel was established for

drug delivery. Physical cooling measures such as ice bags and alcohol wipes were undertaken. After clinical consultations with several departments, the patient was diagnosed with MH. Catheterization was performed at 13:30, and 70 mg of dantrolene sodium was intravenously injected at 13:56. At 14:05, dantrolene was continuously injected intravenously at a rate of 20 mg/hour. Blood gas analysis was performed four times, showing that the pCO_2 initially increased and then returned to normal at 13:42 (Table 1). The patient's body temperature dropped to 38°C at 14:35 and then to 37°C at 15:00.

The patient regained consciousness at 15:22 and was transferred to the postanesthesia care unit at 15:40. His Steward resuscitation score was 6, body temperature was 36.8°C, SpO_2 level was 100%, heart rate was 89 beats/minute, blood pressure was 138/84 mmHg, and respiratory rate was 18 breaths/minute. Both pupils were equal in size, round, reactive to light, and approximately 3 mm in diameter. Dantrolene sodium was continuously pumped into a right lower limb vein at a dose of 20 mg/hour for a duration of 24 hours. The patient reported no symptoms upon regaining consciousness. Emergency examinations including measurement of the urine creatine kinase level, serum myoglobin level, cardiac injury markers, cardiac enzyme profile, and complete blood count were performed according to medical orders but revealed no significant abnormalities.

Treatment and nursing care

The Malignant Hyperpyrexia Clinical Grading Scale (MHCGS) was developed in the United States as a standardized tool for accurately assessing susceptibility to MH.[8] Patients suspected to have MH can be evaluated using the MHCGS scoring system, which encompasses six aspects: muscle rigidity, muscle breakdown due to elevated creatine kinase levels, respiratory acidosis, increased body temperature, cardiac involvement, and family history.[9] Application of the MHCGS can help in early diagnosis of MH and increase treatment success rates. The MHCGS score for our patient was 63, indicating that MH was almost certain. The score provides valuable clinical context regarding the severity of MH susceptibility and its implications for anesthetic management. To ensure timely and effective implementation of MH management, healthcare professionals should receive training on MH-related knowledge and engage in clinical simulation exercises that cover early identification of MH as well as the use of dantrolene sodium, cooling measures, and necessary supportive care.[10]

Emphasis on preoperative assessment and collection of historical data

In recent years, there has been a notable increase in both the number and proportion of patients undergoing general anesthesia, consequently leading to an increased

Table 1. Record of intraoperative blood gas analysis.

Time	pH	pCO_2 (mmHg)	pO_2 (mmHg)	Hct (%)	Na^+ (mmol/L)	K^+ (mmol/L)	Ca^{2+} (mmol/L)	THbc (g/L)	$cHCO_3$ (mmHg)	BE (mmol/L)
13:32	7.28	56.0	486	42	141	4.5	1.19	143	26.2	−2
13:42	7.35	43.6	474	33	141	4.4	1.14	112	23.9	−2
14:54	7.41	34.7	474	31	138	4.5	1.09	105	22.7	−1
14:12	7.40	36.5	511	33	137	4.9	1.11	112	23.0	−1

Hct, hematocrit; THbc, total hemoglobin concentration; $cHCO_3$, concentration of bicarbonate; BE, base excess.

incidence of MH. The incidence rate of MH is estimated to range from 1:5000 to 1:100,000. Among children aged <15 years, approximately 52% of MH cases are attributed to general anesthesia, with a significantly higher risk in male than female patients (ratio of 2:1).[3] A majority of patients diagnosed with MH present with bodily deformities or possess a family history. During preoperative visits, particular attention should be given to identifying any congenital disorders such as idiopathic skeletal and muscular deformities, scoliosis, strabismus, ptosis, and inguinal and umbilical hernia[11,12] to facilitate early detection and prevention of MH.

Intraoperative monitoring

Each patient's medical history and previous experiences with anesthesia should be comprehensively assessed before surgery. For high-risk patients, anesthetic drugs that may trigger MH should be avoided. Sufficient cooling materials such as ice bags, ice caps, iced saline, and special medications such as dantrolene sodium, sodium bicarbonate, blood pressure boosters, and diuretics should be provided.

Intraoperative discovery of an abnormal sharp increase in body temperature and end-tidal carbon dioxide should be considered to indicate potential MH. Moreover, immediate monitoring of the arterial blood pressure, central venous pressure, blood gas analysis, biochemical indices, myoglobin concentration, and creatine kinase concentration can aid in early diagnosis of MH. Once MH has been diagnosed, inhaled anesthetics and succinylcholine should be discontinued while replacing the anesthesia machine and breathing circuit with new ones along with providing intravenous medication.

Treatment and nursing care during postoperative recovery

For patients diagnosed with MH, appropriate management during the post-resuscitation period is particularly important. When the patient is transferred from the operating room to the postanesthesia care unit, the department should promptly implement the following specific measures.

1. First, during postoperative resuscitation, healthcare professionals should have a comprehensive understanding of the etiology of MH, early clinical symptoms, confirmed key monitoring indicators, and treatment process for MH. The following emergency principles after the occurrence of MH should be followed: prompt detection, accurate assessment, elimination of triggers, timely and comprehensive symptomatic treatment, and prevention of complications.[13]
2. Second, after clinical diagnosis of MH, early clinical manifestations primarily include a rapid increase in body temperature, elevated end-tidal carbon dioxide level, arrhythmia, and muscle stiffness. Subsequent symptoms such as an increased blood potassium level, myoglobinuria, and acidosis caused by hyperrespiration and hypermetabolism may occur. Other life-threatening complications include disseminated intravascular coagulation, congestive heart failure, intestinal ischemia, and limb compartment syndrome caused by severe muscle edema, which eventually leads to multiple organ dysfunction and cardiac arrest in severe cases.[14]
3. Third, once MH has been confirmed, intravenous administration of dantrolene sodium and implementation of effective physical cooling measures are among the methods used for successful management.[15] During implementation of physical cooling, caution should be exercised to avoid frostbite when using ice packs. The alcohol concentration of the scrubbing solution should be 35% to 40%, and the solution should cover the superficial large vessels while avoiding the

precordial area, plantar area, and other contraindicated regions. If necessary, hemodialysis should be conducted to control the body temperature. When the patient's body temperature drops and is maintained below 38°C, the aforementioned cooling measures should be immediately stopped to prevent complications such as shivering.

4. Fourth, to enhance the accuracy of temperature measurement during monitoring, it is necessary to monitor the core temperature instead of the skin temperature. In addition, attention should be paid to whether the patient complains of any symptoms, and stable ventilation and normal urinary excretion should be ensured. Laboratory examinations should be performed as per physician guidance, including measurement of the urine creatine kinase concentration, serum myoglobin concentration, myocardial injury markers, myocardial enzymes, and routine blood parameters, along with dynamic blood gas analysis to facilitate timely intervention measures.[16]
5. Fifth, because MH tends to recur within 24 to 48 hours after onset,[17] close monitoring of patients' vital signs is necessary to maintain their physiological needs and ensure their safety during the perioperative period.
6. Our patient was successfully rescued after general anesthesia and smoothly recovered during the postoperative period. This outcome indicates the crucial role played by intraoperative monitoring, early diagnosis, effective treatment, and postoperative surveillance.

Further discussion

The available epidemiological data on MH remains limited. First, there are challenges in implementing large-scale diagnostic tests for MH. Second, susceptible individuals often exhibit atypical clinical symptoms when exposed to triggering factors. Therefore, relying solely on clinical manifestations for diagnosing MH remains a subject of controversy. More comprehensive epidemiological information needs to be gathered from multiple perspectives.

From a diagnostic perspective, the in vitro contracture test (IVCT) serves as the gold standard for diagnosing MH.[18] However, because of the operational challenges associated with its implementation and the absence of an overt suggestive family history and past medical records, IVCT is not typically employed as a preoperative diagnostic indicator. Nevertheless, given its widespread recognition and high reliability as a diagnostic tool, the IVCT still holds significant value in investigating the epidemiological characteristics of MH and identifying clinically applicable diagnostic criteria.

From an etiological perspective, the hereditary nature of MH underscores the pivotal role played by genetic factors in the development of this disease. Consequently, molecular genetic studies can facilitate a comprehensive analysis of the genetic characteristics associated with MH, enable assessment of familial risks, and enhance both diagnostic and preventive strategies for MH.

Summary

As a disease characterized by rapid progression and high mortality rates, MH significantly contributes to perioperative safety risks. The successful treatment of our patient highlights the significance of enhancing the medical profession's comprehension of MH, including its pathogenesis, clinical manifestations, and efficient rescue procedure. Prioritizing preoperative consultations and reinforcing perioperative monitoring are imperative for high-risk populations. In addition, it is essential to establish and improve department-specific

rescue protocols as pivotal factors in early detection, prompt diagnosis, and effective management, all of which are key factors in improving the success rate of MH treatment and nursing care.

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Author contributions

HL, LT: protocol/project development, data collection and management, data analysis, and manuscript writing. XZ, LL, JW: project development and manuscript writing. XP: protocol/project development, supervision, and manuscript writing.

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors without undue reservation.

Declaration of conflicting interests

The authors declare that there is no conflict of interest.

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