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Malignant hyperthermia following elbow fixation surgery: A case report

[1]Amin Hassanzad[*], [1]Seyed Ali Mousavi Mohajer, [2]Mohammad Ahmadi, [3]Elham Kashani, [2]Kazem Kazem Nejad and [4]Azar Azadmard

[1]Department of Anesthesiology, Golestan University of Medical Science, Gorgan, Iran, Student Researchs Committee Golestan University Medical of Science, Iran
[2]Department of Anesthesiology, 5th of Azar Hospital, Golestan University of Medical Science, Gorgan, Iran
[3]Department of Gynecology & Obstetrics, Golestan University of Medical Sciences, Gorgan, Iran
[4]Department of Basic Science, 5th of Azar Hospital, Golestan University of medical science, Gorgan, Iran

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ABSTRACT

Malignant Hyperthermia is a rare life threatening condition, which characterized by a very high temperature, an increased heart rate and breathing rate, increased carbon dioxide production, increased oxygen consumption, acidosis, rigid muscles, and rhabdomyolysis, that develops under general anesthesia. Also, it is an inherited muscle disorder triggered by certain types of anesthesia that may cause a fast-acting life-threatening crisis. We present a case of seizure who was delivered for an elbow fixation surgery in a 21year-old male, Persian ethnicity, who suffered malignant hyperthermia during general anesthesia. Patient was treated with carbamazpin and had never been underlying disease or any drug sensitivity. Induction was performed thiopental sodium, succinylchpline and fentanyl. The patient was diaphoresis, increase plod pressure and heart rate after 30 minute that malignant hyperthermia occurs. Regarding life-threatening feature, the occurrence of malignant hyperthermia is probable during surgery even in patient with previous non-complication surgeries. Therefore, continues monitoring of patient, appropriate history and side effect reducing are necessary.

Key words: Malignant hyperthermia, Elbow fixation

INTRODUCTION

Malignant Hyperthermia (MH) or malignant hyperpyrexia was the name given to a type of severe reaction under general anesthesia that was firstly described in 1960. Another disorder was Malignant Hyperpyrexia [1]. In 1977, the Danish Malignant Hyperthermia Register was established. One of the purposes of this unit was to collect information about MH [2]. Malignant hyperthermia (MH) is an autosomal dominant neuromuscular disease involving defects in calcium release, triggering in as many as one in 15,000 anesthetic procedures, thus can be fatal if not treated promptly [3,4].

Malignant hyperthermia (MH) is an inherited pharmacogenetic disorder characterized by an accelerated skeletal muscle metabolism, muscle rigidity; rhabdomyolysis [5], rapidly rising body temperature and defect appears closely associated with the ryanodine receptor, the protein that forms the SR Ca release channel [3]. The gene frequency has been estimated to be as low as 1 in 14,000 [5]. Attendant hypermetabolism, blood salt, and pH imbalance can result

in neurological, liver, and kidney damage and are potentially fatal unless the conditions are reversed [4]. There is no accurate noninvasive diagnostic test for this disorder yet. However, MHS can be diagnosed by evidence of a perioperative episode of malignant hyperthermia or halothane/caffeine contracture testing on a skeletal muscle biopsy specimen in vitro. Recent reports have suggested a single genetic locus for MHS that is closely linked to polymorphic DNA markers on chromosome 19q13.1 [5,6].

MATERIALS AND METHODS

Case reported was patient aged 21 years-old to M.H (Malignant Hyperthermia) associated ASA status 2 .his medical history showed seizures and had been drug treatment with carbamazepine. The patient had been never underlying disease and sensitivity certain drug but patient was came into operation room, two day before main surgery to put pins in the forearm and had received anesthetic volatile with mask. The patient was referred to operation room 5Azar hospital for fixation elbow. The anesthesia caused by thiopental sodium, fentanyl and Succinylcholine.

After 2 minutes the patient was laryngoscope and was placed under ventilator and was used anesthetic drug isoflurane, nitrous oxide and oxygen and atracurium. Patient was used to monitor factors including pulsoximeter, ECG (ElectroCardioGraphy) and blood pressure. Sever diaphoresis was started during surgery and disconnected isuflorane from patient and continue anesthesia with propofol. Patient vital sign was conversional that included Spo2 between 92% to 99%, heart rate between 110 to 120 and blood pressure between 110/70 to 140/95. Anesthesiologist was injected opoid and after that decreased his diaphoresis but could not solve.

Blood glucose was checked and 290 reported. Anesthesiologist had been injected 5^{u} insulin but the patient vital sign has been changed. Patient's heart rate was zero that for first time anesthesiologist guess that it might has been insulated from patient electrocardiograph but after receiving assurance from the electrocardiograph, anesthesiologist was understood cardiac arrest patient. they started CPCR (Cardiopulmonary-Cerebral resuscitation) associated with other anesthesiologist that after 10 minute patient return to initial state and after that they got ABG (Arterial Blood Gases) from patient that had been represented Pao_2:180 and Pco_2:120. Echocardiography was obtained from patient during echo, patient was cardiac arrest again, after an hour and 30 minute of CPCR, it was not successful and he was dead. After re-examination of the problem, it had been showed that anesthesia machine existence was closed and the patient was repeatedly exhaled.

Table-1: Vital sign before Surgery

Temperature	37
Heart rate	82
Respiratory	18

Table-2: Lab data before Surgery

WBC	7700
RBC	4.76
HBG	14.1
HCT	41.9%
PLT	212
FBS	110mg/dl
Creatinine	1mg/dl
BUN	10mg/dl
Sodium	135mEq/L
Potassium	4mEq/L

Table-3: ABG after Surgery

Pao_2	180
Pco_2	120
PH	7.10

RESULTS AND DISCUSSION

MH is a pharmacogenetic disorder of skeletal muscle that presents a hypermetabolic response to potent volatile anesthetic gases such as halothane, sevoflurane, and the depolarizing muscle relaxant succinylcholine, and rarely, in

humans, to stress such as vigorous exercise and heat. Early MH signs include masseter muscle rigidity, arrhythmia, acidosis metabolic and respiratory, raise of CPK (Creatine Phosphokinase) and electerolyte disorder [7, 8, 18]. Succeeding signs includes- de an increased body temperature, which is the origin of the term "MH", and ventricular arrhythmia. As late signs of MH, continuous muscle contractions may provoke generalized muscle rigidity, which is typically followed by rhabdomyolysis when inappropriately treated(18).Malignant hyperthermia have been diagnose with 3 risk factors:

1. Increase temperature $1^{o}C$/5 minute
2. Bas excess > 5 mEq
3. Pco2 > 60 ml

There have been three points in each patient. malignant hyperthermia pathophysiology has related to Ion homeostasis disorder in skeletal muscle [7, 9] and malignant hyperthermia maybe susceptible to genetic disorder and mutation in receptor ryanodine gene [5, 10] that located on chromosome19 [5,6,7,11] but 3-75% of patient have malignant hyperthermia, They have not genetic disorder and mutation in receptor ryanodine gene. So it seems a genetic disorder is associated not only with malignant hyperthermia but also with other factors [12] Strazis and co-workers was reported malignant hyperthermia about 503 patient that they were not complicated with pervious anesthetic [13].

Britt and colleagues showed the absence complication in pervious anesthetic, that was not rejected malignant hyperthermia [14]. The patient had gotten halothane about 25 minute in pervious anesthetic and they got halothane about 75 minute in recently anesthesia, so halothane and succinylcholine is two main factors in causing malignant hyperthermia. Changes calcium Ion metabolism of succinylcholine may be severe with inhalation of halothane that dose explain why malignant hyperthermia is caused after 75 minute. Increase blood pressure and heart rate can be related to increase calcium ion metabolism. Differential diagnose in this case was included Malignant hyperthermia, Sepsis, Hypoventilation and Malignant neuroleptic syndrome. The result of blood culture negative rejected septicemia diagnosis, Patient did not get drugs that could benefit malignant neuroleptic syndrome and also hypoventilation was rejected for mechanical respiratory.

Hyperkarbi and acidosis as well as increase temperature are cause of malignant hyperthermia [15]. Although diagnosis MH is only with muscle biopsy but this test is unnecessary in definitive diagnosis [15, 16]. The goal of treatment malignant hyperthermia is stopped operation, adjusted temperature and electrolyte disorder. Dantrolene has main role for treatment of MH with prevent of calcium ion release that was used in this patient [17]. MH is autosomal dominant disease [18] a case of MH was a inherited disease that there were no problem with halothane in previous surgery, but in his recently surgery due to long-time succinylcholine and halothane consumption leaded to Malignant hyperthermia. In this case, interview and full awareness about MH susceptible family perhaps prevent and reduce complication due to MH. MH is not only influenced by genetic factors and other factors are also involved. Therefore, the absence complication in previous anesthetic, it does not rule out disease.

Regarding life-threatening feature, the occurrence of malignant hyperthermia is probable during surgery even in patient with previous non-complication surgeries. Therefore, the continue monitoring of patient, appropriate history and side effect reducing are necessary.

Consent Section: Written informed consent was obtained from the patient's next of kin for publication of the case for publication of this manuscript and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Authors' contributions: AHZ analyzed and interpreted the patient data regarding arterial blood gases and was a major contributor in writing the manuscript. SAM analyzed and interpreted the patient data regarding vital sign before surgery. MA and KKN analyzed and interpreted the patient data regarding laboratory for before surgery.

REFERENCES

[1] P.M. Hopkins, *Br J. Anaesth,* **2000**, 85:118-128.
[2] Helle Ording, *Anesth Analg*, **1985**; 64: 700-4.
[3] Michael Fill, Roberto Coronado, James R. *J. c Biophysical Society,* **1990**, 50: 471-475.

[4] Elizabeth F, Gillard Kinya OTSu, Junichi FuJii. *Genomics*, **1991**, 11: 751-755.
[5] R. C. Levitt, A. Olckers, S. Meyers. *Genomics*, **1992**, 14: 562-566.
[6] A. Urwyler, T. deufel, T.McCarthy. *Br J of Ananesthesia*, **2001**, 86(2): 283-297.
[7] Geonert G.A, Antogini J.E, Pessah I.N. In: Miller RD, editor. Anesthesia, 6th ed. *Philadelphia: Churchill livingstone Inc*, **2005**, 1169-1191.
[8] Larach MG, Localio AR, Allen GC, Denborough MA. *Anesthesiology*, **1994**; 80: 771-779.
[9] Lopez JR, Allen PD, Alamo L, Myoplasmic free Ca2+ during a malignant hyperthermia episode in swine. Muscle nerve, **1988**; 82-88.
[10] Fill M, Cponodo R, Mickelson JR. *Biophys J*. **1990**; 57: 471-475.
[11] MC carthy TV, Healy JMS, Heffron JA. *Nature*. **1990**; 343: 562-564.
[12] Kikuchi H, *Nihon Ijishinpo (Jpn)*, **1997**; 38(12): 11-16.
[13] Koduh A, Kikuchi A,Wake yama S. *J Anesth*, **1998**; 13: 181-184.
[14] Britt BA, Malignant hyperthermia. *Can Anesth Soc J*. **1985**; 32: 666-667.
[15] Jorkat Rott K, MC carthy T, Lehman Horn F. *Muscle Nerve*, **2000**; 23: 4-17.
[16] Rosenberg H, Reeds S. Invivo contracture tests for susceptibility to malignant hyperthermia. Anesth analg, **1983**; 62: 412-420.
[17] Britt BA. Dantrolen, *Can Anesth Soc J*. **1984**; 31: 61-75.
[18] Kenji Yamada, Tadao Okada, Shohei Honda, Post-Operative Malignant Hyperthermia Complicated by Rhabdomyolysis: Case Report. Surgical Science, **2012**, 3, 89-92.
[19] Ameh EG., Akpah FA. *Adv. Appl. Sci. Res.* **2011**, 2(1): 33.
[20] Ogbonna O., Jimoh WL., Awagu EF., & Bamishaiye EI. *Adv. Appl. Sci. Res.* **2011**, 2(2): 62.
[21] Yadav, S. S., Kumar, R. *Adv. Appl. Sci. Res.***, 2011**, 2 (2), 197.
[22] Sen, I., Shandil, A., Shrivastava, V. S. *Adv. Appl. Sci. Res.* **2011,** 2(2): 161.
[23] Levine, D. M., Sulkin, S. D. *J. Exp. Mar. Biol. Eco.* **1984,** 81: 211-223.
[24] Gupta N., Jain U. K. *Der Pharmacia Sinica* **2011,** 2(1): 256-262.