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# High Index of Suspicion of Malignant Hyperthermia: A Case Report

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## ABSTRACT

Lack of early detection due to either unawareness of the disease process or inadequate monitoring device, and lack of availability of dantrolene makes it difficult to manage malignant hyperthermia (MH). It has been reported that the risk of mortality rate increased by 50 % if skin temperature rather than core temperature was monitored. We hereby report the case of a 54 year old lady scheduled to undergo exploratory laparotomy for abdominopelvic mass who developed unexplained rise in end tidal carbon dioxide intraoperatively. Probably, this case appears to be one of the first to have been reported from Nepal of possible MH.

**Key words: Core temperature, Malignant Hyperthermia, Dantrolene, End tidal carbondioxide.**

## INTRODUCTION

Malignant hyperthermia (MH) is a rare autosomal dominant disorder with variable penetrance triggered by anaesthetic drugs in genetically susceptible individuals resulting in life-threatening hypermetabolic state. The incidence of MH episodes during anaesthesia has been described as 1: 5000 and 1:50,000-100,000 anaesthetics**.** Ever since dantrolene was approved by the United States Food and Drug Administration (US FDA) in the late 1970s, the mortality rate from acute MH reaction has reduced from 70 % - 80% back then to less than 5% in recent years (1). It is essential to have a comprehensive knowledge for the early diagnosis and management in order to prevent serious consequences. The first sign of MH under anaesthesia is the increase in end-tidal carbon dioxide ($Etco_2$) exponentially.

## CASE PRESENTATION

A 54 year old female (height, 150 cm: weight, 58 kg) with an abdominopelvic mass; was scheduled to undergo exploratory laparotomy under general anaesthesia with endotracheal intubation.

In the preoperative assessment, the patient had no history of systemic abnormalities and no history suggestive of hyperthyroidism. She had no history of anaesthetic exposure in the past and no family history of anaesthetic related morbidity and mortality. The remaining findings were unremarkable. Routine preoperative haematological, biochemical investigations (complete blood count, serum electrolytes, liver function tests, and renal function tests) and electrocardiogram were within normal limits.

In the operating room after establishing intravenous access and attaching standard monitoring devices, an epidural catheter was placed at $T_{11-12}$ using an aseptic technique under local anaesthesia. Inj. ceftriaxone 1gm, inj. metoclopramide 10 mg and inj. ranitidine 50 mg were administered intravenously prior to induction.

Anaesthesia induction was achieved with



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injections fentanyl 100 mcg, propofol 120 mg and intubation facilitated with rocuronium 50 mg. Maintenance of anaesthesia was done with sevoflurane with intermittent boluses of vecuronium. Vitals were normal during induction and after intubation. A mixture of inj. 0.1% bupivacaine with fentanyl 3 mcg/ml in 6 ml was given via the epidural catheter followed by infusion at the rate of 5 ml/hr.

Approximately 45 min after induction of anaesthesia, a rapid rise in $Etco_2$ from 36 to 45 and then to 54 mm of Hg was noticed, along with rise in HR of 98 bpm from the baseline of 60 bpm. In response to this, minute ventilation (MV) was increased, the breathing circuit was checked for leaks, the endotracheal tube position was checked and any bronchospasm was ruled out. In spite of our initial response, $Etco_2$ further increased to 64 mm of Hg. Other vital parameters, viz blood pressure and temperature, was within normal limits. This unexplained rise in $Etco_2$ despite increasing MV prompted us to suspect malignant hyperthermia. The inhalational agent was switched off, the propofol infusion was started, the new breathing circuit was changed, and new soda lime with canister was changed. A blood sample was sent for arterial blood gas (ABG) analysis and the report showed pH of 7.193, $Pco_2$ of 65.8 mmHg, $Po_2$ of 279 mmHg, lactate 5.2 mmol/L, and a base deficit of -4.5 mmol/L. Intravenous fluid plasmalyte 500 ml was given rapidly. Surgeon was informed and requested to complete the surgery as quickly as possible. Fortunately, at that time, the adnexal cyst had already been removed and the surgical team was in the process of closing the abdomen. There was no muscle rigidity, $Spo_2$ and BP were normal, skin temperature was not raised, urine was normal in colour. After the surgery was ended, propofol was reduced and the patient was kept ventilated on SIMV mode for another half an hour. After return of spontaneous respiration, reversal was given and the patient was observed further. After ensuring adequate tidal volume and regain of full consciousness, trachea was extubated. At the time of extubation, the patient was sweating and her skin was warm on touch with skin temperature measuring 36.5 ˚C. We, however couldn't measure core body temperature at that time which was very important if we were suspecting MH. The room temperature was adjusted to lower her temperature. A repeat ABG done after extubation of trachea showed improvement from the initial one. The patient was then shifted to the post anaesthesia care unit (PACU) for monitoring after she gained consciousness with stable vital signs. The patient was kept in PACU for further half an hour and was shifted to ward with close monitoring of HR, BP, $SPO_2$, temperature and urine colour. ABG done at 8 hours after surgery was unremarkable. On first postoperative day, the patient complained of mild whole body ache. Serum creatinine kinase was raised in first (1509 U/L) and second (3225 U/L) postoperative day with normal PR, $Spo_2$, BP, temperature and urine colour. The urine for myoglobin and plasma myoglobin was not sent due to unavailability of the service in our institute. The patient was discharged on the 4th postoperative day without any problem.

As we could not perform confirmatory tests for malignant hyperthermia due to unavailability of the test or use of dantrolene which is the drug recommended for MH, the definitive diagnosis could not be made.

## DISCUSSION

We had a high index of suspicion of possibility of MH to our patient on the basis of clinical features she developed. Although the caffeine halothane contracture test is considered the gold standard for determining MH susceptible individuals (2), DNA analysis has become increasingly important in recent years (3, 4). Because these tests are not widely available, the diagnosis of MH in such a setting can only be determined based on clinical presentation.

The clinical presentation of MH varies from a slight or moderate reaction to a fatal condition due to skeletal muscle hypermetabolism (5). Many studies have provided that slight and moderate MH reactions have improved without dantrolene administration (5-9). However in severe reaction, dantrolene is the only drug that saves a life if used early. Fortunately, the clinical presentation in our case was mild to moderate, which might have improved after rapid diagnosis and early discontinuation of the triggering agent. As long as we provide anaesthesia, MH cannot be omitted. And once MH occurs in severe condition or detected late, the only drug that improves the outcome of the patient is dantro-

lene. Hence, we have to consider the availability of dantrolene in tertiary hospitals of Nepal where a large number of operations are being performed under general anaesthesia.

The most common early sign of MH under anesthesia is an exponential increase in $Etco_2$ as reported in many literatures (5, 10-12). In our case also there was a sustained rise in $Etco_2$ with tachycardia and without any change in other vital signs. However, the patient's skin temperature was within normal limit in spite of being warm on touch and sweating. Therefore, it has been recommended that due to the reported risk of increase mortality by 50%, core body temperature rather than skin temperature be monitored (13). Because of unexplained rise in $Etco_2$, we were able to suspect MH and immediate appropriate intervention was initiated. On the scale described by Larch et al**.** for definitive diagnostic indicator of MH in 1994 (14), the total score in our case was 28 with $Etco_2$ > 55 mmHg (15 points), inappropriate sinus tachycardia (3 points) and arterial pH < 7.25 (10 points). The score in our patient falls on rank 4 (somewhat greater than likely).

**Table 1: Clinical indicators for calculating MH Raw Score in the present case (14)**

| Clinical Indicator | Points |
|---|---|
| Masseter spasm shortly following succinylcholine administration | 15 |
| Serum K+ >6 mEq.L-1 | 3 |
| Etco2 >55 mm Hg with appropriately controlled ventilation | 15 |
| Inappropriately rapid increase in temperature (in anesthesiologists judgment) | 15 |
| Inappropriately increased temperature >38.8℃ (101.8℉) in the perioperative period | 10 |
| Inappropriate sinus tachycardia | 3 |
| Arterial pH <7.25 | 10 |

To the best of our knowledge, currently we do not have the facility for testing MH susceptible individuals anywhere in Nepal for diagnosis and the availability of early access of dantrolene for management of MH.

## CONCLUSION

Although we could not confirm the diagnosis, our case was highly likely to have developed malignant hyperthermia, albeit mild type.

End tidal $Co_2$ and core body temperature rather than skin temperature monitoring is necessary when general anaesthesia is consider for early detection of MH and further deterioration to happen especially in our setup because of unavailability of dantrolene.

Identify susceptible individuals of MH and avoid agents that trigger MH as possible if you have other alternative means.

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## CONFLICT OF INTEREST

The authors declare that there is no conflict of interests regarding the study or this article.

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