

## Malignant hyperthermia: An Indian perspective

Madam,

Malignant hyperthermia (MH)<sup>[1]</sup> is a life-threatening disorder triggered by various anesthetic drugs. In the year 2008, the first case<sup>[2]</sup> was reported from India, at a time when it was thought to be non-existent in our population. Unlike in developed countries, there is no national registry in India for MH; hence, case reports are the only way to know its incidence and challenges in recognising/treating it.

We recently encountered MH in an 8-year-old female child, scheduled for forearm surgery. Anesthesia was induced using propofol and morphine, and maintained with O<sub>2</sub>, N<sub>2</sub>O and isoflurane, with patient breathing spontaneously on Magill circuit via laryngeal mask airway. After 90 min of uneventful anesthesia course, sudden tachypnea with hypercapnia was noted (EtCO<sub>2</sub> 45 mmHg, RR 30/min). Within next couple of minutes tachycardia (150/min), hypertension (150/110 mmHg) and hypoxia (SpO<sub>2</sub> 88%) occurred. Patient was also febrile to touch, which was confirmed by monitoring (38.4°C), along with muscle rigidity. Diagnosis of MH was considered, inhalation agent discontinued, Bain's circuit attached, ventilation assisted with 100% O<sub>2</sub> and midazolam given. Surgeons were requested to expedite the surgery. Drapes over lower body were soaked with saline. Surgery finished within next 15 min, by when EtCO<sub>2</sub> increased to 75 mmHg, HR to 170/min, BP to 180/110 mmHg and temperature to 39.8°C. The arterial blood gas (ABG) analysis showed acute respiratory and metabolic acidosis (pH 6.99, pCO<sub>2</sub> 49.6 mmHg, HCO<sub>3</sub><sup>-</sup> 12.2 mmol/L, base-deficit 18.0 mmol/L). Upon completion of surgery whole body sponging with tepid water was initiated, trachea intubated, and controlled ventilation instituted. With continued supportive measures, EtCO<sub>2</sub> dropped to 60 mmHg, HR 150/min

and BP 150/90 mmHg over next 30 min. ABG showed metabolic acidosis, hyperkalemia (5.9 mmol/l), raised serum urea (61 mg/dl) and normal serum creatinine (0.7 mg/dl). Aggressive cooling was continued till axillary temperature dropped to 37.8°C in the next hour. Patient's trachea was extubated after 8 hours of ventilation, following which recovery was uneventful. Repeated investigations revealed normal serum K<sup>+</sup> (4.5 mmol/L), urea (31 mg/dL) and creatinine (0.3 mg/dL), with raised creatinine kinase NAC (>2000 IU/L), creatinine kinase MB (440 IU/L) and SGOT/PT (295/601 IU/L). The patient was observed for another 36 hours and then shifted to ward for further care.

MH may have a delayed onset, occurring in 2<sup>nd</sup> or 3<sup>rd</sup> hour of anesthesia, as witnessed by us also. The clinical suspicion is aroused by a constellation of seemingly common events including tachycardia, tachypnea, rise in EtCO<sub>2</sub>, unexplained desaturation, hyperthermia and combined respiratory and metabolic acidosis, as in our case. Since the gold standard for diagnosis i.e., caffeine-halothane muscle contracture testing,<sup>[1]</sup> has limited availability it is most often based on clinical criteria. The clinical grading scale of Larach *et al.*<sup>[3]</sup> scores each of the clinical indicators and then retrospectively calculates probability of the event being MH. Our patient had a score of 68 that correlates with a likelihood of almost certain event.

We noted a negative end tidal CO<sub>2</sub> to arterial CO<sub>2</sub> gradient (pCO<sub>2</sub> 49.6 mmHg with EtCO<sub>2</sub> of 75 mmHg). This reversal of gradient had been previously known to occur with MH.<sup>[4]</sup> Various explanations offered included increased cardiac output, increased CO<sub>2</sub> production. The EtCO<sub>2</sub> in such cases correlates with the venous CO<sub>2</sub>.

Treatment of MH includes discontinuation of triggering agents, ventilating with 100% O<sub>2</sub> and increased minute ventilation, use of dantrolene, active cooling till the core temperature reaches 38°C, correction of acidosis and hyperkalemia, treatment of

**Table 1: Previously published case reports from India**

Author	Year	Age/ gender	Inhalation agent	Succinylcholine used	Dantrolene used	CGS score*	Likelihood	Outcome of patient
Saxena KN <i>et al.</i> <sup>[2]</sup>	2007	28/F	Halothane	Yes	No	61	Almost certain	Survived
Gopalakrishnan C V <i>et al.</i> <sup>[7]</sup>	2010	9/M	Sevoflurane	No	No	63	Almost certain	Survived
Gulabani M <i>et al.</i> <sup>[6]</sup>	2014	16/F	Isoflurane	No	No	43	Very likely	Survived
Raut S <i>et al.</i> <sup>[5]</sup>	2016	68/F	Isoflurane	No	Intravenous	61	Almost certain	Survived
Iqbal A <i>et al.</i> <sup>[4]</sup>	2017	45/F	Halothane	Yes	No	53	Almost certain	Survived

CGS=Clinical grading scale Larach *et al.*; M=Male; F=Female, \*The score was calculated based on the clinical description given in the case report

arrhythmias and other supportive measures. Supportive measures were instituted by us and despite unavailability of dantrolene we could avoid fatal outcome, which emphasises the favourable outcome associated with early diagnosis and supportive measures.

A PubMed search revealed a total of 5 cases from India so far [Table 1].<sup>[2,5-8]</sup> Most Indian patients have been females (4/5 = 80%), isoflurane and halothane being used in 40% cases each, almost all of the cases occurred intraoperatively (4/5 = 80%) and all of them survived.

Thus, MH is not uncommon in India and attempts at wider awareness should be encouraged.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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### Conflicts of interest

There are no conflicts of interest.

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