**CASE REPORT**

**SURVIVAL OF A MALIGNANT HYPERThERMIA PATIENT**


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**ABSTRACT**

Malignant hyperthermia is an inherited autosomal life threatening skeletal muscle disorder usually triggered by certain general anesthetic drugs like volatile anesthetics and depolarizing muscle relaxants (succinylcholine). Only early recognition and prompt accurate treatment can save life in an otherwise fatal situation. We present a case of a young boy in whom malignant hyperthermia was recognized within 10 minutes of induction of anesthesia and life was saved by prompt treatment even in the absence of dantrolene.

**CASE REPORT**

On 14th Dec 2011, an 8 year old boy weighing 24 kg reported to CMH Malir for pre anaesthesia assessment. He was scheduled for orchidopexy. On history and general physical examination he was found to be a little lethargic with bilateral ptosis. His siblings also had a similar problem. He was referred to a paediatrician in accordance to hospital protocol for evaluation of any musculoskeletal disease which can increase the susceptibility of malignant hyperthermia (MH), but no such disease was found. It was difficult to manage a young child under regional anesthesia and high suspicion of MH was not appreciated. Surgery under general anesthesia was planned employing safe drugs and careful monitoring on 28th Dec 2011. Routine monitoring was attached (NIBP, ECG, SpO₂, temp capnography). After pre-oxygenation, induction was done with injection propofol 2 mg/kg. Non-cuffed endotracheal tube was passed after injection atracurium 0.5 mg/kg. Endotracheal tube was checked bilaterally and secured. Anesthesia was maintained with 1 MAC isoflurane 50% oxygen + 50% air.

After about 10 minutes of induction, the patient started developing masseter spasm and biting ETT. At the same time ETCO₂ was noticed to be rising and tightening of abdominal muscles was noticed by the surgeon. Malignant hyperthermia was suspected. All anesthetics were stopped, 100% oxygen started, anesthesia machine and circuit were changed. Guedel’s airway was introduced with effort to prevent blockage of ETT due to masseter spasm. Surgeon was requested to stop surgery. Soon the body temperature started rising and muscle spasm extended to whole body.

Temperature lowering measures were started like surface cooling, room air conditioning, intravenous cool normal saline, intravenous paracetamol infusion 15 mg/kg during first hour then 15 mg/kg after 4 hours, (then repeated 6 hourly during 24 hours). Paracetamol suppository 250 mg was placed in rectum 8 hourly on second day. Esophageal temperature was monitored, 7.5% sodium bicarbonate 1 mEq/kg intravenous given and repeated after 1 hour to counter acidosis.

Glucose infusion of 25 gm with 10 units insulin was given to counter hyperkalemia. Utmost effort was made to acquire dantrolene injection from all available sources but was not available.

During the treatment, the temperature rose up to 100° F but started lowering after 2 hours of extensive temperature lowering measures. ETCO₂ rose up to 80 mmHg and after 2 hours lowered to 45 mmHg. Heart rate went up to 185 / min and came down to 160/ min with 1 mg intravenous lopressor.

After 3 hours of treatment, the temperature was 100° F, blood pressure 125/ 90 mmHg, heart
rate 140/ min, oxygen saturation 98%, ETCO₂ 40 mmHg, muscle spasm was improved. During treatment urinary catheter was passed and output was monitored. Injection frusemide 50 mg intravenous was given.

During the course of treatment 12 lead ECG was done which showed sinus tachycardia of 160/ min. Investigations sent during this period showed TLC = 20.5, blood glucose random = 10.6 mmol/ l, serum potassium = 5.1 meq/ l, serum CK = 8852 u/ l, serum ALT = 61 U/ l, serum urea/ creatinine and calcium were within normal limits. The patient was kept on ventilatory support with continuous monitoring of ETCO₂, esophageal temperature, ECG, NIBP, pulse oximetry.

After 24 hours he was weaned off from the ventilator. He was fully conscious and oriented, his temperature was 99°F, with blood pressure 115/ 65 mmHg, heart rate 112/ min and oxygen saturation 98%. Facility for muscle fiber test was not available. The patient was kept in ICU for another 3 days and continuously monitored for vital signs and blood chemistry. Vital signs remained within normal limits. Serum CK and ALT remained raised while rest of blood chemistry was within normal limits. Serum CK and ALT after 4 weeks were nearly normal. The family was counseled about the future risk of malignant hyperthermia to the patient and siblings if exposed to general anesthesia. The patient was also provided a warning card for MH.

DISCUSSION

Malignant hyperthermia (MH) is an inherited autosomal life threatening skeletal muscle disorder usually triggered by certain drugs of general anesthesia like volatile anesthetics and depolarizing muscle relaxants (succinylcholine)¹.


Dantrolene remains the only drug known to be effective in the treatment of MH. In suspected patients “caffeine halothane contracture test” is done which has 97% sensitivity and 78% specificity. Genetic testing is also being performed to determine susceptibility to MH¹, in people with a family history of MH, analysis for RYR1 mutations maybe useful²,³.

CONCLUSION

Although dantrolene is a specific drug for treatment of malignant hyperthermia which should be made available, however even in the absence of dantrolene, standard monitoring (temp., ECG, NIBP, Pulse oximetry, ETCO₂) during general anesthesia, early recognition and prompt symptomatic treatment can save the life of a patient.

REFERENCES