A case of suspected malignant hyperthermia

Sir,

Malignant hyperthermia (MH) is a hypermetabolic disorder of skeletal muscle that is triggered by inhalation anaesthetics and succinyl choline in susceptible individuals. Unrecognised and untreated, it carries a high mortality rate.

A 2-month-old 5 kg male child with congenital talipes equinovarus was posted for bilateral percutaneous tenotomy. There was no significant medical history or history of previous anaesthetic exposures. Birth, growth, developmental history and family history were unremarkable. Child was otherwise healthy; premedication consisted of injection atropine 0.1 mg and midazolam 0.25 mg, both intravenous (IV). After pre-oxygenation, child was induced and maintained with oxygen, nitrous oxide and halothane, with assisted ventilation through the facemask, on Jackson-Rees circuit, without intubation. Acetaminophen rectal suppository (80 mg) was placed and incision site was infiltrated with 2 ml of 0.25% bupivacaine and bilateral procedure was completed in 20 min. Monitoring consisted of precordial stethoscope, pulse-oximetry (SpO_2), electrocardiogram (ECG) and end tidal carbon dioxide ($EtCO_2$) connected to mask. Fluids were maintained as per Holliday and Segar's formula with paediatric maintenance solution (PMS). Intra-operatively persistent tachycardia (≅160/min) was noted, with adequate clinical depth of anaesthesia, with normal oxygen saturation and $EtCO_2$ of 40 mm Hg. The baby was shifted to the recovery room after Aldrette recovery score (modified) of 9 was reached in 5 min. Tachycardia persisted and after an hour in the recovery room, the heart rate (HR) increased to 220/min with respiratory rate of 24/min, SpO_2 of 95% and $EtCO_2$ (connected via a nasal cannula) of 48 mm Hg. Axillary temperature was 39.5°C. Oxygen supplementation was started via Hudson's mask. With in next 15 min, SpO_2 and HR started falling, (SpO_2 to 87% and HR, 90/min), $EtCO_2$ was 60 mm Hg and the child was getting drowsy, hypotonic and further hypoxic. Child was intubated and ventilated with 100% oxygen, with assisted ventilation. Mechanical ventilation was instituted with vecuronium induced paralysis. Mean while, body temperature reached 40°C which was treated with cold IV fluids, tepid sponging and paracetamol 25 mg IV. As the tone of the heart sounds decreased with further bradycardia (45-50 bpm), bolus of 50 ml of PMS and dopamine infusion was started. Chest X-ray was normal. Other investigations (1 h post-operative samples) showed creatinine phosphokinase (CPK) of 15,800 IU/L, sodium 131 mmol/l, potassium 5.4 mmol/l and calcium 1.01 mmol/l, arterial blood gas values of pH 7.19, pCO_2 54 mm Hg, HCO_3 −5.9 mmol/l and base excess of −1.7 mmol/l and fall in haemoglobin (7.3 mg/dl). Cardio respiratory and fluid support was continued for next 24 h; 100 ml of packed red blood cells was administered. However, there was persistent hyperthermia (39-40°C), hyperkalaemia (6-6.5 mmol/l) and acidosis (pH 6.98) with pCO_2 at 64 mm Hg (despite hyperventilation). A probable clinical diagnosis of MH was made using Larach's *et al*. raw score of 63 favouring MH diagnosis. (Elevated Creatine kinase >10,000 IU/L after anaesthesia without succinyl choline = 15, P_{EtCO_2} >55 mm Hg with controlled ventilation = 15, inappropriately increased temperature >38.8°C in peri operative period = 10, sinus tachycardia = 3, arterial base excess more negative than –8 mEq/L = 10, arterial pH < 7.25 = 10, totaling 63 and MH rank of 6, which almost certainly represents MH).[1] As dantrolene was not available we continued with conservative management. There was gradual fall in temperature and HR over next 2 hrs along with an increase in serum creatinine and acute renal failure, not responding to dialysis. Child went into severe bradycardia and cardiac arrest; resuscitation attempts were futile and the child died.

MH is a potentially fatal subclinical myopathy, which is unmasked on exposure to volatile anaesthetics like halothane or succinylcholine. Described first in 1960 by Denborough and Lovell, MH syndrome is characterized by generalised muscle rigidity unexplained CO_2 production, metabolic acidosis, rhabdomyolysis, elevated CPK level, hyperkalaemia and hyperpyrexia.[2,3] No history of exposure to general anaesthesia or family history of MH was obtained in the present case. There was no history or evidence of any myopathies, cardiac or neurological diseases associated with the club foot. The initial tachycardia after induction with halothane was attributed to atropine. Analgesia was adequate as local infiltration and acetaminophen suppository were used. The development of hypovolemia, hyperpyrexia along with tachycardia and tachypnea with elevated $EtCO_2$ is also found in neuroleptic malignant syndrome, but was ruled out in the present case. The use of halothane and the presence of hyperpyrexia, severe intractable

acidosis, persistent hyperkalaemia, raised CPK with hypovolemia suggested the possibility of MH.[4,5] Though the gold standard diagnostic test for MH is *in vitro* Halothane Caffeine Contracture test, a Larach *et al.* raw score of 63 indicates almost certain diagnosis of MH. Early dantrolene administration may decrease morbidity rate by 35%.[6] Availability of dantrolene would have probably saved the life of the child.

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