Suspected malignant hyperthermia in a patient undergoing thyroidectomy

Sir,

Malignant hyperthermia (MH) is an inherited hypermetabolic disorder of skeletal muscle, triggered by potent anaesthetic agents. The incidence is between 1:10,000 and 1:150,000 adult patients receiving a general anaesthetic.[1] Early detection and use of dantrolene has reduced the mortality from 70% to <5%.[2] We report a case of perioperative suspected MH in a patient undergoing thyroidectomy.

A 40-year-male with no significant clinical history including history of chronic medication was posted for subtotal thyroidectomy. His pulse rate was 84 beats/min and blood pressure measured 120/86 mm Hg. His airway and systemic examinations were essentially normal. Investigations including thyroid profile were within normal limits. After informed written consent, Tab. Diazepam 10 mg the night before and early morning of the day of surgery was administered. In the operation theatre after intravenous (IV) access, he was premedicated with Inj. Glycopyrrolate 0.2 mg and Inj. Fentanyl 100 μg. Monitoring included electrocardiogram, non-invasive blood pressure, oxygen saturation, temperature and following intubation, end tidal carbon dioxide ($EtCO_2$). General anaesthesia was administered using Inj. Thiopentone sodium 300 mg and endotracheal intubation facilitated by Inj. Succinylcholine 100 mg. Intermittent positive pressure ventilation was instituted using closed circuit with sodalime and anaesthesia was maintained with O_2-N_2O, 0.4% to 1% halothane and appropriate doses of IV Atracurium. Perioperatively patient was haemodynamically stable, but after 45 min, towards the end of surgery there was sudden rise in heart rate (140/min) along with a rise in blood pressure (180/120 mm Hg), $EtCO_2$ (90 mm Hg) and body temperature (108°F, 42.2°C, measured from oesophageal probe).

To rule out lighter planes of anaesthesia, halothane 2% and top up of IV atracurium were administered, with hyperventilation but there was no respite from the above changes. Blood samples were drawn and sent for thyroid profile, serum electrolytes and serum creatinine phosphokinase (S.CPK). Anaesthetic circuit was changed to Bain's circuit and patient hyperventilated. Arterial blood gases revealed severe metabolic acidosis (pH – 7.08) and marked hypercarbia (PCO_2 – 90 mm Hg). Laboratory investigations revealed marginally elevated thyroid values, gross elevation of S.CPK (3665 IU/L) and K^+ (6.2 mmol/L). Patient developed gradual hypotension with range of dysrhythmias and conduction defects, including sinus tachycardia, supraventricular tachycardia, ventricular tachycardia and ventricular fibrillation. Pharmacological cardiac support, sodium bicarbonate, use of surface cooling with ice packs and cold IV fluids etc., as also electrical defibrillation was in vain and patient succumbed after 45 min of onset of the initial derangement.

MH is a pharmacogenetic disorder of skeletal muscle that is associated with hypermetabolism secondary to uncontrolled increase of calcium in muscle sarcoplasm. It manifests in susceptible individuals when exposed to triggering agents, commonly an inhalational agent or Succinylcholine.[3,4] Speed of onset of MH reflects the rate of rise in intracellular Ca^{++} levels.[4] Mortality of

less than 5% could be achieved by early diagnosis and use of dantrolene.[2] Except for the thyroid swelling, our patient was essentially healthy before this procedure. A sudden increase in heart rate, blood pressure, body temperature and end-tidal CO_2 intraoperatively aroused suspicion of onset of thyroid storm, even though the patient was euthyroid, and not on any treatment. As the patient was (a) Not responding to appropriate resuscitative measures (b) Investigations revealed severe acidosis, marked hypercarbia, marginally elevated thyroid values and gross elevation in S.CPK and (c) We had used triggering agents like succinylcholine and halothane, we thought MH was a possible diagnosis. A clinical situation such as this can also be mimicked by thyroid storm, neuroleptic malignant syndrome (NMS) and phaeochromocytoma. Though hypertension, hyperpyrexia and cardiac arrhythmias were present, thyroid storm was ruled out as there were marginal elevations in thyroid hormones but marked elevation in $EtCO_2$ and severe acidosis. As there was no history of any chronic drug intake or the triad of sweating, episodic headache and tachycardia, the possibility of either NMS or pheochromocytoma was remote.[4,5] The gold standard diagnostic test for MH is *in vitro* Halothane Caffeine Contracture test, the facilities for which were not available at our institute. We applied MH clinical grading scale and a total score of 46 was achieved (Serum K^+ >6 mmol/L – 3 points, $EtCO_2$ > 7.5 kpa during controlled ventilation – 15 points, inappropriate rapid increase in temperature – 15 points, Inappropriate sinus tachycardia – 3 points, Arterial pH < 7.25-10 points). A total score of 46 corresponds to MH rank of D5 which represents the clinical scenario as "very likely" of MH.[6] Though appropriate measures to treat the clinical condition were instituted, our patient succumbed due to non-availability of dantrolene sodium which is the drug of choice for MH. We conclude that a high index of suspicion must be maintained about onset of MH peri-operatively and prompt recognition, early intervention with dantrolene administration can be life-saving.

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