

## Undetected hypothyroidism and unexpected anesthetic complications

Sir,

A 56-year-old man with carcinoma tongue was posted for radical surgery and primary reconstruction. He gave history of neoadjuvant chemotherapy with methotrexate 6 months preoperatively. His vitals and laboratory reports were within normal limit except for a slight increase in serum creatinine of 1.7 mg/dl. Electrocardiogram showed low-voltage complexes and no other significant abnormalities.

Patient was premedicated with alprazolam 0.5 mg, ranitidine 150 mg, and metoclopramide 10 mg orally on the night before and on the morning of surgery. Preoperative vitals were within normal limits. Fentanyl 100 mcg, glycopyrrolate 0.2 mg, and midazolam 1 mg were administered intravenous (IV) and general anesthesia was induced with propofol 100 mg IV. Vecuronium 6 mg IV was given to facilitate nasotracheal intubation and muscle relaxation. Immediately after positioning, in the thyroid position, there was a drastic fall in blood pressure to 50/30 mm of Hg. Peripheral oxygen saturation was 97%; electrocardiogram (ECG) showed regular sinus rhythm with a heart rate of >160/min. The peripheral pulses were not palpable and soon the central pulses also became impalpable and heart sounds were not audible. ECG showed pulseless electrical activity. Cardiopulmonary resuscitation (CPR) was started and continued after giving IV adrenaline 1 mg till peripheral and carotid pulsations reappeared and blood pressure improved to 120/80. Blood sample was sent for arterial blood gas (ABG) and electrolyte estimation. ECG pattern changed to stable ventricular tachycardia with a rate of 190/min. IV amiodarone 150 mg was given slowly over 10 min, followed by its infusion at the rate of 1 mg/min.

After a few minutes, peripheral and central pulses again became feeble and then impalpable. CPR was restarted and vasopressin 40 mg was given intravenously. ECG showed monomorphic ventricular tachycardia and a 150 joules

defibrillatory shock (biphasic) was given twice. BP improved to 130/90 mmHg and ECG showed normal sinus rhythm at a heart rate of 86/min. ABG revealed a combined metabolic and respiratory acidosis with electrolytes within normal limits. A few minutes later, BP again fell to 80/60 mmHg. Dopamine infusion was started at 5 mcg/kg/min to maintain BP to >120/80.

Patient showed no signs of spontaneous respiration and did not display pharyngeal, laryngeal, or corneal reflexes even after 2 h. Pupils were sluggishly reacting bilaterally. He was shifted to the intensive care unit and placed on ventilatory support. After 3 h, spontaneous respiratory effort returned. During this period, his hemodynamics were stable. Over another hour, he became awake, conscious, and regained full muscle power.

Blood was sent for thyroid function test (TFT) and troponin T estimation which revealed severe hypothyroidism with T3 < 5 ng/dl, T4 1.1 mcg/dl, and TSH 57.4 miu/l. Troponin T was strongly positive (1.75 ng/ml). Repeat ECG showed sinus rhythm with T wave inversion and q wave in lead 3. He was treated with IV hydrocortisone, subcutaneous fraxiparine along with oral clopidogrel, atorvastatin, amiodorone, and thyroxine. Thyroxine dose was initially given 25 mcg twice daily, taking his cardiac event into consideration, and it was gradually increased to 100 mcg twice daily over 2 weeks. The patient responded well to this therapy and was discharged home in 2 weeks. His surgery was deferred for 6 months in view of the myocardial event. He was advised to undertake chemotherapy in the meantime.

Cardiac arrest was secondary to severe myocardial depression due to routine doses of drugs used to induce general anesthesia in a background of severe hypothyroidism. Patients with apparently asymptomatic hypothyroidism (increased level of thyroid stimulating hormone, but a normal level of thyroxine) have an increased risk of congestive cardiac failure.<sup>[1]</sup> Increased sensitivity to cardio-depressant effects of anesthetics in hypothyroidism is due to decreased intravascular volume, decreased preload, blunted baroreceptor response, and decreased cardiac output. The only suggestive preoperative finding for hypothyroidism in this patient was an ECG with low-voltage complexes. This was over looked in preanesthesia check up, as the patient had no other symptoms and signs of hypothyroidism and no other significant changes in ECG.

Preoperative chemotherapy could have caused hypothyroidism. Patient's T3 level was low (<5 ng/dl). He had repeated cardiac arrests and was difficult to revive. The degree of hemodynamic instability correlates well with the degree of thyroid dysfunction.

Severe hypothyroidism should be managed with IV T3 and T4, but if they are not available oral T3 is the mode of choice. We used oral thyroxine to treat the patient.<sup>[2]</sup> Hypothyroidism was a late diagnosis and by the time TFT results were obtained patient was awake. The chance of recovering from primary cardiac event even before the hormone levels get normalized is quite rare. There were no clinical symptoms of adrenal dysfunction which could give rise to similar picture.

This report emphasizes that a high index of suspicion is necessary to detect subtle thyroid dysfunction in cancer patients. Undetected hypothyroidism should be ruled out at the earliest when unexpected hemodynamic compromise on induction of anesthesia occurs in an otherwise normal patient. Disorders of thyroid gland in cancer patients may occur due to malignancy, antineoplastic therapy<sup>[3-5]</sup> or due to causes not related to malignancy.

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