

Case Report

Graves' Disease Hemodynamically Masquerading as Pheochromocytoma during Thyroidectomy

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Abstract: Graves' disease of thyroid occasionally requires surgical thyroidectomy for cure. In a well controlled (euthyroidised) patient with antithyroid drugs and beta blockers, it is a straight forward operation with uneventful outcome when managed by an experienced surgeon-anesthetic team. But, unlike non-toxic goiters, minor cardiac and hemodynamic events are a norm than exception during surgery for Graves' disease. In this case report, we highlight a turbulent intraoperative course with hypertensive crisis in a 43 year old patient, who was well controlled and non-hypertensive pre-operatively. We also discuss the probable underlying pathophysiology and management options through this interesting case.

Keywords: Graves' disease; Thyroidectomy; Hypertension; cardiac arrhythmia; Goiter.

INTRODUCTION:

Surgical thyroidectomy for Graves' disease is one of the challenging operative procedure for surgeon, anaesthesiologist and patient alike. Sudden severe hypertensive surges and cardiac arrhythmias during any elective surgery in a non-hypertensive patient often entails abandonment of procedure, for work up of hypertension due to intraoperative suspicion of a missed secondary hypertension (mostly either due to Pheochromocytoma hypercatecholaminemia or renal or vascular causes). Graves' disease often requires elective surgical thyroidectomy, when associated with large goiter, exophthalmos or drug non-compliance. But, anaesthesia and surgery for Graves' disease is challenging due to cardiovascular and metabolic effects of the disease. We highlight perioperative management of a typical situation, wherein Graves' disease behaved like pheochromocytoma hemodynamically. This report also emphasizes the extra caution to be taken routinely in the hitherto management of Graves' disease.

CASE REPORT:

A 43 year old non-hypertensive gentleman presented with goiter and features of hyperthyroidism for 6 months. He had no other co-morbidities such as diabetes; chronic pulmonary, cardiac, hepatic, renal

disease; no history any drug intake/ abuse; not a chronic smoker. On clinical and biochemical evaluation, he was diagnosed as Graves' disease (GD) and started on empirical dose of 30 mg/day of carbimazole and 60 mg/day of propranolol in three divided doses. His total body weight was 65 kg. Initial serum free T3 was 11.5 mcg/dL (1.8-5.4), free T4 was 13.3 mcg/dL (4.5-11.7) and TSH was <0.01 mIU/L (0.4 - 4.2). It was up-titrated till 50 mg/day of carbimazole and euthyroidism was attained in 4 weeks time (FT3 = 4.8 mcg/dL; FT4 = 9.6 mcg/dL). We have not given Lugol's Iodine as it was not in our departmental protocol. He underwent total thyroidectomy under general inhalational anaesthesia with nitrous oxide and oxygen (67:33). 130 mg (2 mg/kg) of Propofol and 180 mg (3 mg/kg) of succinyl choline were used for induction of anaesthesia. For anticholinergic action, only buscopan 20 mg was given (glycopyrrolate, fentanyl, midazolam were not needed). Premeditated with 0.6 ug/kg of dexmedetomidine and vecuronium was the muscle relaxant. Intubation was uneventful. But intraoperatively, dramatic hypertensive episodes occurred whenever thyroid gland (TG) was handled for mobilization. The maximum blood pressure (BP) was 230/ 180 mm Hg with ventricular ectopics twice. We thought of abandoning the case suspecting an

underlying pheochromocytoma, due to severe hemodynamic disturbance. But, in view of experience with managing pheochromocytoma cases and expert anaesthesiologist availability, we continued surgery. Isoflurane at a set dial concentration of 4 %, 300 mg of amiodarone as bolus, 30 mg/kg of magnesium sulphate as bolus and 0.6 ug/kg/hr of dexmedetomidine as infusion were required to control intraoperative hypertensive surge and arrhythmias till conclusion of thyroidectomy. Post-operatively, the dismemberment of thyroid gland from body induced dramatic hypotension within 10 minutes followed by sustained norm tension.

Captured Multi-parameter monitor readings of hypertensive, hypotensive and normotensive phases with corresponding operative time are displayed in Figures 1, 2A and 2B respectively. Estuation and recovery were smooth with no signs of strider, hypocalcaemia or features of hyperthermia (Infrared thermometer surface body temperature was 97°F). This dramatic hemodynamic response typical of Pheochromocytoma (PH), prompted us to evaluate serum metanephrine level postoperatively, which was normal ruling out PH. At 3 months follow-up, he is asymptomatic on thyroxine replacement.

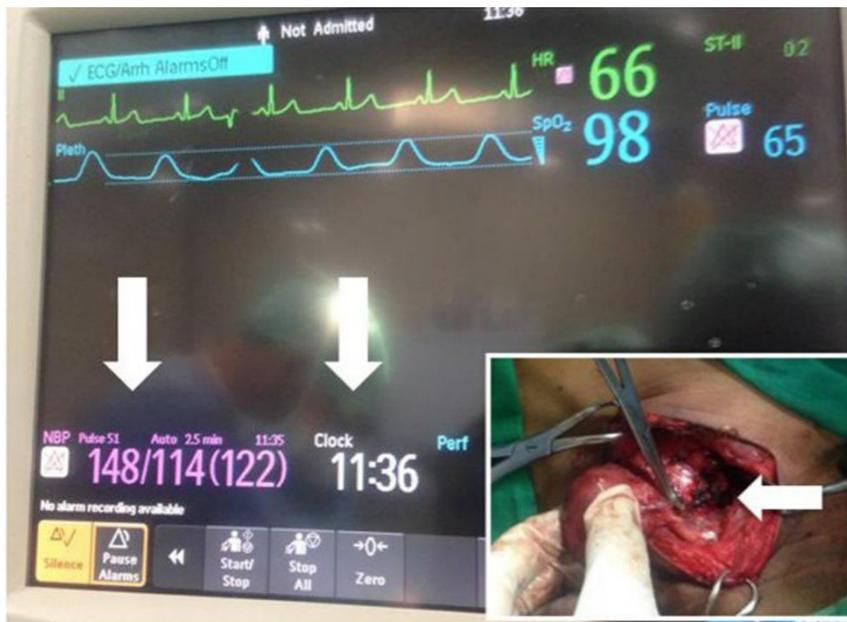


Figure 1: Multi-para monitor showing hypertensive surge corresponding with TG mobilization (Inset) at 11.36 AM [left arrow pointing BP and right arrow pointing TG]

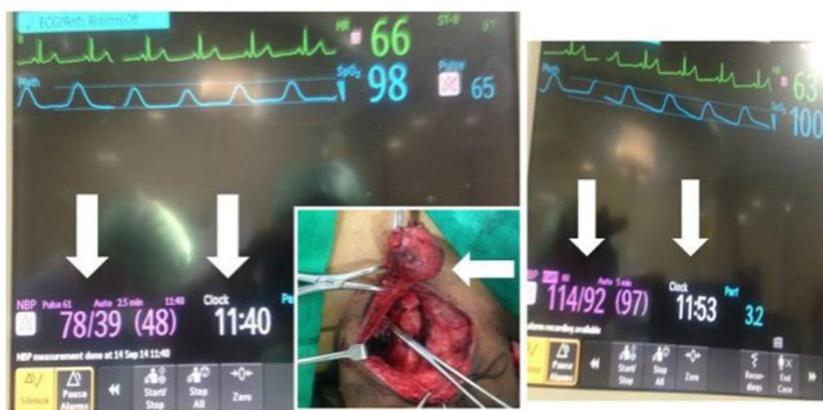


Figure 2: A) Multi-para monitor showing hypotensive episode corresponding with removal of TG (Inset) at 11.40 AM [[left arrow pointing BP and right arrow pointing TG]; B) Monitor showing normotension post-operatively at 11.53 AM [left arrow pointing BP]

DISCUSSION:

GD is well known for its hyperdynamic circulation and cardiovascular effects [1]. But, well controlled (euthyroidised) glands do not precipitate

thyrotoxic storm or hypertensive crises at surgery. In this case, the peri-operative hemodynamic responses typically simulated a case of PH, characterized by severe hypertension during gland mobilization followed

by severe hypotension after its removal. We even thought of abandoning the surgery, with the suspicion of undetected PH. But, we managed BP surges with appropriate vasoactive and cardioprotective agents as employed in PH. The hypothetical mechanisms of this labile cardiovascular phenomenon are heightened catecholamine receptor hypersensitivity for thyroid hormone or catecholamines; thyroid hormone surges during surgical manipulation of TG; increased sympathetic tone [1,2]. The hyperadrenergic state in GD due to action of thyroid hormones on its target organ receptors [2]. This hypothesis can be partly justified by mechanism of drugs used to maintain intraoperative hemodynamic stability. Dexmedetomidine, we used is a highly selective alpha-2 adrenergic receptor agonist, which peripheral catecholamine release, thus reducing sympathetic response to surgical stimuli [3]. Magnesium sulphate again is a blocker of calcium channel and N-methyl-D-aspartate (NMDA) receptors leading to sympatholysis [4]. Amiodarone is a proven anti-arrhythmic agent. Thus, all the drugs used directly/indirectly lead to reduced sympathetic responses and blunting of autonomic nervous system. We found only anecdotal case reports of synchronous association of GD and PH but no syndromic associations in literature [5]. But, the real challenge is to find pre-operative indicators of intraoperative hemodynamic instability, which are lacking as of now. Till then, as highlighted in this case, surgical-anaesthetic team should manage even a apparently well controlled GD with as much care and monitoring as for PH with vasoactive and cardioprotective drugs as needed. We also wanted to highlight through this case, that though hemodynamic events are quite frequent during thyroidectomy for GD it is not often emphasized duly to trainees of anaesthesia and surgery. Finally, unlike in other elective surgeries, wherein hypertensive surges of this magnitude warrants abandonment of procedure, but in GD we may continue with the surgery as surges should be expected and duly managed even in non-hypertensive well controlled cases.

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