International Journal of Biomedical And Advance Research

ISSN: 2229-3809 (Online)
Journal DOI:10.7439/ijbar
CODEN:IJBABN

Case Report

Hypothyroid patient undergoing Coronary bypass surgery- A nightmare, perioperative management challenge

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Abstract

The management of hypothyroid patients for coronary artery bypass grafting (CABG) surgery has remained challenging. The patient will have depressed cardiac function with unpredictable response to the routine inotropes, depressed adrenergic response and baroreceptor reflexes, as well as increased systemic vascular resistance (SVR).Though there have been reports about the successful management of such patients, the risk is definitely higher. We hereby describe how we managed a grossly hypothyroid patient with unstable angina, using Levosimendan as the first choice inotrope.

Keywords: Hypothyroid, coronary artery bypasses grafting, cardiac function, systemic vascular resistance, levosimendan

1. Introduction

The management of hypothyroid patients before CABG is very challenging. Important consideration in hypothyroid patients for surgery include, increased sensitivity to depressant drugs, hypodynamic cardiovascular system with reduced heart rate (HR) & Cardiac Output (CO), slow metabolism of drugs, impaired ventillatory response, hypothermia, unresponsive baroreceptor reflex, primary adrenal insufficiency, decreased number and sensitivity of β receptors. Preoperative thyroid hormone therapy may precipitate angina in patients with ischemic heart disease and may contribute to myocardial infarction, although this seems to be an infrequent event.[1] Also, there have been few reports of incidences of heart failure in hypothyroid patients undergoing cardiac surgery.[2] On the contrary, some of the studies indicate that untreated hypothyroid patients tolerate cardiac surgery with few significant perioperative complications, rendering aggressive preoperative thyroid hormone replacement unnecessary. Many of these studies and case reports of CABG in hypothyroid patients are usually on patients with mild to moderate hypothyroidism.[3]

Evaluations of the impact of cardiac surgery and cardiopulmonary bypass (CPB) on thyroid hormone levels have yielded conflicting results. The potential causes of decreased thyroid hormone levels during and after CPB are varied and include hypothermia, reduced peripheral conversion of T4 to T3, hemodilution, nonpulsatile blood flow, the suppressive effect of cytokines and tumor necrosis factor on thyroid function, iodine skin preparations and cortisol-induced effects on TSH secretion.[4]

We report a case of iatrogenic hypothyroidism (secondary to radiotherapy) with unstable angina, where treating physicians were worried about triggering coronary event preoperatively by thyroid supplementation to correct the thyroid status. We had to contemplate with managing a grossly hypopthyroid patient with unstable angina, as well as deal with stress induced thyroid crisis in this patient**.**

2. Case Report

Here was a male patient aged 58 yrs posted for CABG, mainly indicated for triple vessel disease with unstable angina. He had a history of being diagnosed for glottic carcinoma, which was evaluated and excised 5 years back, following which he underwent radiotherapy around the neck region. Post radiotherapy, the patient developed iatrogenic hypothyroidism. The patient was not complying with the thyroxine medications regularly. Echo findings - Normal LV function, Normal chamber dimensions, No regional wall motion abnormality. His other significant investigation findings were hypercholesteremia, thyroid profile TSH 93 µIU/ml (0.45 - 4.5 µIU/ml), Free T3 0.02 pg/mL (2.3- 4.2 pg/mL), Free T4 0.04 ng/L (0.8 - 1.8 ng/L). The treating physician's opinion about patient's thyroid status (hypothyroidism) was mainly because of noncompliance with the thyroid supplementation and were also of the view that trying to normalize the thyroid profile by supplementing thyroxine, may trigger ischemia and possibility of infarction. Hence it was planned to get the patient undergo CABG at the earliest under high risk followed by normalization of the thyroid status.

Patient had a depressed mental status, restricted mouth opening with interincisor distance of around 4 cm. Neck extension was restricted probably because of radiotherapy he received of the neck following Glottic carcinoma excision. He came under the difficult airway category. His preoperative medications included thyroxine 100 µg, isosorbide, atorvastatin and aspirin.

Perioperative steroid supplementation was instituited in anticipation of any adrenal insufficiency that could be associated with hypothyroidism. After establishing invasive arterial pressure, awaken fibreoptic intubation done through nasal route using minimal sedation with fentanyl 100 µg and midazolam 2 mg. Patient was induced with combination of midazolam, sevoflurane and rocuronium as muscle relaxant. We supplemented tablet thyroxine 100 µg through Ryle's tube post induction. Patient was haemodynamically stable during induction. Pulmonary artery Catheter was inserted in right internal jugular vein and along with other parameters, intermittent CO was also monitored. During sternotomy, patient had a sudden fall in the blood pressure (BP) 60/40 mm Hg with HR around 86 beats/min, not responding to fluid. Adrenaline boluses of 5 µg were given intravenously twice. BP kept fluctuating from 180/100 mmHg to 60/40 mmHg, with ECG showing stable ST's with cardiac index between 2.0 to 2.2 L/min/m². We went on CPB at the earliest. Three venous grafts were anastamosed to left anterior descending, obtuse marginal and posterior descending arteries, with a total Pump time of 134 min with aortic cross clamp time 85 min.

While coming off CPB, a loading dose of levosimendan 10 µg/kg over 10 min followed by its infusion at 0.1µg/kg/min and adrenaline 0.05 µg/kg/min were instituted. His cardiac index was noted to be 2.1 L/min/m2 and SVR was on the lower side, after which we started infusion of noradrenaline 0.05 µg/kg/min. Patient was having fluctuations in BP during this phase as well, but ECG was stable. Intra Aortic Balloon pump was kept as standby for any eventuality.

In postoperative intensive care unit (ICU) also, haemodynamics were volatile and also had an episode of ventricular fibrillation (VF), which was reverted immediately by defibrillation. Patient was continued with steroid and thyroxine supplementation postoperatively. Patient's haemodynamics stabilized by 8 to 10 hrs post shifting to ICU. Patient was gradually weaned and extubated 18 hrs after shifting to ICU. Levosimendan infusion was tapered and stopped.

Otherwise uneventful postoperatively, patient was shifted to stepdown ICU and then ward by 4th post op day. Steroid supplementation was gradually tapered and stopped, while thyroxine supplementation was continued. His thyroid profile was rechecked before his discharge which was still on hypothyroid side but better than preoperative values.

3. Discussion

The most important adverse effects of hypothyroidism that may predict a bad surgical outcome are those affecting cardiac function. Routinely, inotropes which exert their effect are dependent on β adrenergic receptors, which may not be that effective in these types of patients and probably an inotrope that is independent of β adrenergic receptors for its effect may prove advantageous. Among many cardiovascular abnormalities described in hypothyroid patients is impaired cardiac contractility with decreased cardiac output and increased peripheral vascular resistance. Alterations in calcium handling seen in the cytoplasmic reticulum and a depression of the myosin ATP-ase activity contribute to the observed decrease in myocardial contractility.[5]

An increased peripheral vascular resistance is believed to be due directly to the deficiency in thyroid hormone. T3 seems to exert a vasodilatory effect by a direct action on the smooth muscle of the blood vessels and an effect on endothelial function.[6] The decrease in oxygen demand of peripheral tissues associated with hypothyroidism may also play a role in the increase of systemic vascular resistance, which in turn causes an increase in the cardiac afterload. Levosimendan, which exerts its positive inotropic effect by increasing calcium sensitivity of myocytes by binding to cardiac troponin C in a calcium-dependent manner, and also having a vasodilatory effect, by opening adenosine triphosphate(ATP)-sensitive potassium channels in vascular smooth muscle, is potentially an effective drug in these circumstances. The decreased plasma volume due to increased capillary permeability and associated shift of water and albumin into the interstitial space as well as loss of the cardiovascular responses to acute increases in intrathoracic pressures often seen in the setting of hypothyroidism could have contributed to the volatility in haemodynamics seen in our patient postoperatively.[7]

Also, most of the reports are on patients with mild to moderate hypothyroid undergoing CABG. In our case, we had to deal with patient who was grossly hypothyroid. A complex interaction between thyroid hormones and catecholamines seems to exist. Hypothyroid patients have a depressed adrenergic tone, which is not caused by decreased levels of catecholamines. On the contrary, catecholamine levels are increased and possible explanations for this paradoxical phenomenon include a down regulation of β-adrenergic receptors, loss of a direct ‘‘catecholamine-like” action of thyroid hormone or an increase of intracellular levels of inhibitory guanine nucleotide-binding (G) protein.[5] These patients are better served by perioperative supplementation of steroid for a more effective response to catecholamines. Since levosimendan is not dependent on β receptors for its effects, it may prove beneficial in these situations as it was similarly experienced by using amrinone in a case reported by Whitten *et al*.[8] A variety of electrocardiographic abnormalities have been reported in hypothyroid patients, particularly in the perioperative period, with bradycardia being most common, but other more severe abnormalities have also been documented with some frequency, including the ventricular tachyarrhythmia, as in our case as well, we had an episode of ventricular fibrillation, which could be influenced by other factors as well.[9]

4. Conclusion

From our experience, we would rather prefer to preoperatively supplement the patient with thyroxine , so that patient’s thyroid status approaches near normal or is mildly hypothyroid, than operating the patient when grossly hypothyroid. Levosimendan could be a good choice of inotrope to counter the adverse effects of reduced levels of thyroid hormones, mainly reduction in cardiac contractility and increased SVR, and also, since it doesn’t depend on the β adrenergic receptors for its action. Further study is required in order to understand more about its effectiveness in such situations.

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