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Anaesthetic management of multi gravida woman with preeclampsia with severe features and hyperthyroidism complicated by thyroid storm in the latent stage of labour

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Abstract

Thyroid storm is an acute, life-threatening, hypermetabolic state and is rare in pregnancy. In contrast, pulmonary hypertension and heart failure from cardiomyopathy caused by the profound myocardial effects of thyroxine is common in pregnant women. The pregnant woman with thyrotoxicosis has a minimal cardiac reserve, and decompensation is usually precipitated by preeclampsia, anaemia, sepsis, or a combination of these. Severe features of preeclampsia include an SBP of 160 mm Hg or greater or DBP of 110 mm Hg or greater on two separate occasions at least 4 hours apart while on bed rest; thrombocytopenia; impaired liver function with twice normal concentrations of liver enzymes. The latent phase of labour begins with the onset of regular uterine contractions and extends to the beginning of the active phase of cervical dilatation. The duration of the latent phase averages 6.4 hours in nulliparous and 4.8 hours in multiparas. The latent phase is abnormally prolonged if it lasts more than 20 hours in nulliparous or 14 hours in multiparas. The cervix usually remains at 4 cm or less but is completely effaced. The etiology is likely ineffective contractions without a dominant myometrial pacemaker.

Keywords

thyroid storm; preeclampsia; toxic multinodular goitre; anaesthesia

Introduction

Thyrotoxicosis is a clinical state characterized by complex of signs and symptoms related to the excess of thyroid hormones, which is either a sequel of its overproduction in thyroid gland or disturbed peripheral metabolism of thyroid hormones or dysfunction of receptors of those hormones or, eventually, overdosing of drugs containing thyroid hormones [1].

Thyroid storm is a life-threatening illness in a patient whose hyperthyroidism has been severely

exacerbated by illness or surgery. Thyroid storm is characterized by hyperpyrexia, tachycardia, and striking alterations in consciousness. It clinically manifests in a manner similar to that of malignant hyperthermia, pheochromocytoma, and neuroleptic malignant syndrome. No laboratory tests are diagnostic of thyroid storm, and the precipitating (nonthyroidal) cause is the major determinant of survival [2].

Medical treatment of hyperthyroidism relies on drugs that inhibit thyroid hormone synthesis (propylthiouracil, methimazole), prevent hormone release (potassium, sodium iodide), or mask the signs of adrenergic overactivity (propranolol). In addition, although β-adrenergic antagonists do not affect thyroid gland function, they do decrease the peripheral conversion of T 4 to T 3 [3].

Preeclampsia is defined as hypertension (SBP > 140 mm Hg or DBP > 90 mm Hg) after 20 weeks of gestation associated with proteinuria. Preeclampsia is diagnosed when urine protein is greater than 300 mg/day or, alternatively, there is a protein/creatinine ratio of at least 0.3. Severe features of preeclampsia include an SBP of 160 mm Hg or greater or DBP of 110 mm Hg or greater on two separate occasions at least 4 hours apart while on bed rest; thrombocytopenia; impaired liver function with twice normal concentrations of liver enzymes; right upper quadrant pain; progressive renal insufficiency with serum creatinine greater than 1.1 mg/dL or a doubling of serum creatinine without other known renal diseases; pulmonary edema; and new-onset cerebral or visual abnormalities [3].

Case Report

A 30-year-old, gravida V, para IV woman whose last menstrual period (LMP) was unknown but claimed to be amenorrheic for the last 9 months. She had regular ante natal clinic (ANC) follow up and the pregnancy was uneventful. Currently, she presented with a chief complaint of pushing down pain and leakage of liquor of 14-hour duration. She has also the history of anterior neck swelling of 14-year duration with associated history of heat intolerance, palpitation and nocturnal cough for which she was given propylthiouracil 100 mg per os(PO) 3X per day since 4 months back. She also has a history of hypertension since a 12 years back but not on any antihypertensive medication.

Physical examination revealed her weight as 67.5kg, height as 158 cm, temperature of 36.8°C, blood pressure of 180/90 mm Hg, heart rate of 120 beats/min, and respiratory rate of 24 breath/min. She has anterior neck mass with multiple nodules with the largest measure 4x5 cm. The fetal HR was 132 to 150 beats/min. The regular uterine contraction was present. Vaginal examination showed cervix-3 cm dilatation, clear amniotic fluid, and ultrasound revealed an EFW (estimated fetal weight) = 3 kgs, AGA (Appropriate to gestational age) =38 weeks

Preoperative thyroid function test revealed elevation of triiodothyronine (T3) 8.15nmol/L (normal range: 0.92–2.33), and thyroxine (T4) 262.62nmol/L (normal range: 60–120), free triiodothyronine (FT3) 13.81pmol/L (normal range: 0.5–3.5), free thyroxine (FT4) 37.22pmol/L (normal range: 0.85–6.5), thyroid-stimulating hormone (TSH) 0.005mIU/L (normal range: 0.55–4.78), and thyroid globulin antibody (TGAb) 173.8IU/mL (normal range: <60). Echocardiogram has shown concentric left ventricular hypertrophy, grade II diastolic dysfunction, and normal systolic function with left ventricular ejection fraction (LVEF) of

81%, no wall motion abnormality, normal valves, no thrombus and normal pulmonary pressure.

With this history, P/E and investigative modalities, she was diagnosed to have the latent first stage of labour with superimposed preeclampsia and thyrotoxicosis. A patient is likely to develop concentric left ventricular hypertrophy as she was not treated for the last 12 years owing to lack of awareness of medical treatment. Chronic hypertension with superimposed preeclampsia is defined when a parturient with a prepregnancy diagnosis of chronic hypertension develops sudden new onset of proteinuria or sudden increase in the levels of blood pressure or when the symptoms of severe preeclampsia develop [4].

At labour ward she was administered with magnesium sulphate of 50 % 4 gm loading dose diluted in 100 ml fluid intravenously over 10 minutes, hydralazine 5mg intravenously, propylthiouracil-100 mg PO bid and propranolol 40mg PO TID according to the protocol.

After 3 hours at labour ward, she developed shortness of breath, agitation, sweating and abnormal body movement and progressive loss of consciousness. On Physical examination, the temperature of 39.1°C, blood pressure of 200/110 mm Hg, heart rate of 148 beats/min, respiratory rate of 60 breath/min, and spo_2 of 18% with face mask ventilation. Fetal heart rate became 80-100beats/min.

With the above evaluation, she was diagnosed to have thyroid storm and fetal bradycardia.

Immediately, anaesthesia team was consulted, and the patient was intubated on her couch when she was transferred to the operating theatre, ventilated with Ambubag for emergency caesarean section, and maintenance of general anaesthesia was provided with thiopentone 250 mg, suxamethonium 75 mg, fentanyl 50 µg, isoflurane 1% and 2 litre oxygen per minute ventilation. The 3.2 kilogram alive male neonate was delivered 3 min. After incision, with Apgar scores of 5, 6, and 7 in the 1st, 5th and 10th minutes respectively. Oxytocin 5 international unit was given intramuscularly. The neonate was resuscitated and transferred to neonatal intensive care unit for better care. The operation ended at 22 minutes. Intraoperative blood pressure of 190/100 mmHg, with pulse rate of 130 per minute, peripheral oxygen saturation (Spo_2) of 98% and temperature of 38.2 °C. Treatment of thyroid storm was continued while the patient was on mechanical ventilator of the anaesthesia machine. The patient was treated with 1gm of propylthiouracil given via a nasogastric tube and continued in 100-mg doses every 6 hours. Paracetamol 1gm was given with cold compresses intermittently. Propranolol 40 mg PO, hydrocortisone 100 mg every 6 hours and hydralazine 5mg as to the response of the patient given intravenously. Urine output was adequate.

The patient was kept on mechanical ventilator of the anesthesia machine due to the absence of ICU bed with morphine 5 mg, vecuronium of 3mg, and diazepam 5mg PRN

Thyroid storm medications continued throughout the night.

The following day the ICU bed was obtained and the patient was transferred to the ICU and the same management was continued.

On her 6th post op. day, her vital sign was, BP 140/90 mmHg, pulse rate of 90/minute, respiratory rate of 16/minute, Spo_2 of 98%, the temperature of 37.1°C and within the normal range of laboratory investigations (Table 1). She was then extubated and discharged with medications for thyrotoxicosis and hypertension.

Table 1: Laboratory results in intensive care unit

	Operation day	Day 2	Day 3	Day 4
WBC(cell/ul)	12.2x10^3/ul	17.9 k	14 k	10.5 k
NEUT	77.9%			70.9%
LYM	16.6%			
HGB(GM/DL)	15.2/dl	12.28/dl	9.8/dl	10.9/dl
HCT %	43.3%	38.1%	42.9%	
PLT(CELL/ul)	132 k	267k	299k	268 k
MCV	93.79 fl			77.8
HEPATIC				
SGOT	7	31	37	38
SGPT	2	3	16	43
ALP	531	483	300	258
RENAL				
Cr	0.7	1.3	1	0.4
BUN	14	34		
N+		144	144	
K+		4.3	4	
Cl -		115	108	

Discussion

Thyroid storm (crisis) is a medical emergency that carries a 10% to 50% mortality rate. It is usually encountered in patients with poorly controlled or undiagnosed Graves' disease [3].

Precipitating factors include (1) the stress of surgery and anaesthesia, (2) labour and delivery, (3) severe infection, and, rarely, (4) thyroiditis 1–2 week following administration of radioactive iodine. Manifestations usually include mental status changes (irritability, delirium, or coma), fever, tachycardia, and hypotension. Both atrial and ventricular arrhythmias are common, particularly atrial fibrillation. Congestive heart failure develops in 25% of patients [3].

Hypertension that often precedes hypotension, heat intolerance with profuse sweating, nausea and vomiting, and diarrhoea may be prominent. Initially, hypokalemia is present in up to 50% of patients. Levels of thyroid hormones are high in plasma but correlate poorly with the severity of the crisis.

The sudden exacerbation of thyrotoxicosis may represent a rapid shift of the hormone from the protein-bound to the Free State or increased responsiveness to thyroid hormones at the cellular level. Our treatment is directed toward reversing the crisis and its complications. Large doses of corticosteroids inhibit the synthesis, release, and peripheral conversion of thyroxine (T 4) to the more active triiodothyronine (T 3). Corticosteroids also prevent relative adrenal insufficiency secondary to the hypermetabolic state. Hence hydrocortisone 100mg every 6 hours was given for this patient. Propylthiouracil is administered to inhibit synthesis of thyroid hormone, but iodide was not given owing to its absence. Propranolol was given to antagonize the peripheral effects of the thyrotoxicosis. Combined β 1 - and β 2 -blockade is preferable to selective β 1 -antagonism (esmolol or metoprolol) because excessive β 2 -receptor activity is responsible for the metabolic effects. β 2- receptor blockade also reduces muscle blood flow and may decrease heat production [3,2,5]

Supportive care is an important part of the multisystem therapeutic approach to thyroid storm [1,6].

Surface cooling (cooling blanket), alcohol sponging, ice packs, acetaminophen (paracetamol), andintravenous fluid replacement were carried out. Salicylates should be avoided in thyrotoxicosis because salicylates can decrease thyroid protein binding, causing an increase in free thyroid hormone levels [7].

Hyperthermia also responds well to external cooling with alcohol sponging, cooling blankets, and ice packs

Thyroid storm can be precipitated by systemic insults such as surgery, trauma, myocardial infarction, pulmonary thromboembolism, diabetic ketoacidosis, parturition, or severe infection [8,9].

Electrocardiogram manifestations of thyrotoxicosis most commonly include sinus tachycardia and atrial fibrillation. Sinus tachycardia occurs in approximately 40% of cases, whereas atrial fibrillation occurs in 10% to 20% of patients who have thyrotoxicosis, with a tendency to occur more commonly in patients older than 60, who are more likely to have underlying structural heart disease [3,10].

Our patient had enlarged thyroid gland, sweating, and anxious appearance, together with both fever and tachycardia, suggesting thyrotoxicosis [3,11].

There was no chest x-ray to rule out whether the patient has acute pulmonary edema during our rapid intervention but clinically we could not detect any findings of pulmonary edema which is infrequently encountered in patients with severe pre-eclampsia without associated medical, surgical or obstetric complications [12].

Conclusion

Early recognition of thyroid storm should be made in obstetrics with hyperthyroidism which requires aggressive management and monitoring. Thyroid storm poses a critical diagnostic and therapeutic challenge to the anaesthetist. Rapid use of the antithyroid medications aimed at stopping the thyrotoxic

process at every step is crucial to successful management. Treatment relies on stopping the synthesis of a new hormone, the release of stored thyroid hormone, conversion of T 4 to T 3, and provision of systemic support of the patient were done successfully to save the life of both mother and newborn.

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