CASE REPORT

A case presentation of thyroid storm in a case of complete hydatidiform mole

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INTRODUCTION

Hydatidiform Mole (H. Mole) occurs in 1:1000 pregnancies worldwide. Incidence is higher in Asian countries. Prevalence of hyperthyroidism during complete molar pregnancy is as high as 7%[1]. This is attributed to excess of Human Chorionic Gonadotropin (HCG), which has a weak intrinsic thyroid stimulating activity. Clinical hyperthyroidism due to trophoblastic disease is cured by evacuation of molar tissue. Here we present a case of thyroid storm in a case of complete H. Mole.

CASE REPORT

A 35 year old woman, gravida 5, para 4, presented to Emergency room with history of 2.5 months of amenorrhoea and complaints of intermittent vaginal bleeding. On examination, she had fever (101 degreees Fahrenheit), tachycardia (146 bpm) and a blood pressure of 122/84 mm Hg and tachypnea with respiratory rate of 30 breaths per minute. There was no exophthalmos and her extraocular movements were normal. The thyroid gland was palpable and of normal size. Cardiovascular examination revealed sinus tachycardia without murmurs, rub or gallops. Breath sounds were equal bilaterally, without rhonchi or wheezes. Pallor was present. There was no peripheral edema. Per abdomen examination revealed uterine size of 24-26 wks (much more than her history of amenorrhoea). There was bleeding p/v, with passage of white grape-like vesicles.

Ultrasonography of her enlarged uterus revealed that the uterine cavity was significantly distended and filled with an echogenic soft-tissue mass that had small cystic components, most compatible with complete molar pregnancy. Theca lutein cysts were present in both ovaries.

Her investigations revealed an haemoglobin level of 7.3 gm/dl, total count, differential leucocyte count and platelet count were normal, RBS was 63 mg/dl, Beta-HCG >10000 and a positive urine pregnancy test. Her Thyroid Function Tests revealed a S.TSH of 0.009 (normal value 0.27-4.20 microIU/ml), fT4 5.59 (normal value 0.8-2.0 ng/dl) and T3 465 (normal value 40-180 ng/dl). Chest X-ray was normal.

As it was a case of thyroid storm, patient was shifted to ICU and hyperthyroidism was controlled by giving T. Propranolol (40) 1-0-1 and T. Carbimazole (10) 1-1-1. Supportive treatment was given in the form of intravenous fluids, injectable antibiotics, antipyretics and Inj. Dexamethasone (8 mg) 8 hrly. Anaemia was corrected by giving Inj. PCV 3 units (1 unit/day). On her 2nd day in ICU, her S. Potassium was found to be low (2.81) (normal value 3.8-5.6 mEq/L). Hence potassium correction was done by giving Inj. KCl 2 amp in 500 ml NS over 6 hrs.

When patient was stabilized on 3rd day, suction-evacuation was carried out under sedation (Inj. Propofol 150 mg + Inj. Fentanyl 100 microgram) and paracervical block (Inj. Lignocaine 2% 10 ml). Post-operatively, patient was given intravenous fluids and injectable antibiotics. T. Propranolol was stopped on 1st post-op day. T. Carbimazole was continued. Dexamethasone was tapered and stopped in the next 4 days. She was discharged on 7th day with advice to follow-up regularly with beta-HCG levels and to use barrier contraceptives until the beta-HCG levels returned to normal.

DISa

Abnormal proliferation of trophoblast leads to formation of H. Mole. Since trophoblast produces HCG, there is excess of HCG in such patients. Gestational Trophoblastic Disease (GTD) with thyrotoxicosis is a rare clinical scenario, but thyroid hyperstimulation by HCG can have severe clinical consequences. Complete H. Mole most commonly presents with vaginal bleeding occurring at 6 to 16 weeks of gestation in 80 to 90% of cases[2]. Tisne et al reported the first case of clinical hyperthyroidism in a patient with hydatidiform mole in

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1955[3]. The glycoprotein hormone HCG is a specific tumour marker for trophoblastic diseases. The analogy in the structure between HCG and TSH can cause cross-reactivity with their receptors. It has been shown that the homology in the HCG and TSH molecules, as well as in their receptors, is likely to be responsible for the cross-reactivity of HCG with the TSH receptor[4]. Glinoer has estimated that for every 10000 mIU/ml increase in serum HCG, free T4 increases by 0.1 ng/dl and TSH decreases by 0.1 mIU/ml[5]. Molecular variants of HCG found in molar pregnancies have increased thyrotropic potency[6]. Use of both general[7] and spinal[8] anaesthesia has been reported for evacuation of the mole. General anaesthesia maybe the preferred technique in hypotensive bleeding patients scheduled for emergency evacuation. In stable patients, spinal anaesthesia is preferable due to its non-tocolytic properties and safety in hyperthyroid patients. Intravenous fluids and blood must be administered judiciously as these patints have a propensity to develop pulmonary edema. Thyroid storm is an extreme accentuation of thyrotoxicosis. Marked tachycardia, arrhythmias, pulmonary edema and congestive cardic failure may occur[9]. If unrecognised, the condition is invariably fatal. Combined use of propylthiouracil, iodide and dexamethasone restores serum T3 concentration to within normal range within 24 to 48 hours. In the absence of cardiac insufficiency, beta blockers to ameliorate symptoms should be started preoperatively.

CONCLUSION

Women with a history of molar pregnancy are at a risk of further molar pregnancies. Much has been reported in the literature about the incidence, recurrence and persistence of H. Mole. Thyroid Function tests should be mandatory in all women with hydatidiform mole and these women should be stabilized with beta blockers an anti-thyroid medication prior to induction of anaesthesia for their surgical evacuation. Vigilant montoring and intensive care should be extended into the post-operative period because there is a likelihood of occurrence of cardiopulmonary complications, thyroid storm and disseminated intravascular coagulation.

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