Peripartum Cardiomyopathy with Ischemic Hepatitis, Thyroid Storm and Right-sided Pneumonia in a Patient of Type 2 Diabetes Mellitus: A Case Report

SM Rezaul Irfan¹, AMB Safdar², Marufa Hossain³, AKM Mohibullah⁴

Abstract:
Peripartum Cardiomyopathy in association with Diabetes mellitus and Thyroid storm is an extremely rare. Here we describe a young aged Bangladeshi lady, who admitted with progressive breathlessness, high grade intermittent fever, productive cough and altered level of consciousness in Coronary Care Unit. She was a diagnosed case of Diabetes Mellitus and Graves ' disease but during her last pregnancy the patient voluntarily stopped taking anti-thyroid medication and delivered a healthy male baby through LUCS (two months before admission). After thorough clinical evaluation and laboratory investigations she was diagnosed as having Peripartum Cardiomyopathy, Thyroid Storm, Pneumonia and Metabolic Acidosis. Her condition improved by closely monitored therapy guided by team consisting of Cardiologists, Endocrinologists, Pulmonologists and Gynecologists. There are very few reports in the world which have depicted this unusual association.

Key words: Peripartum Cardiomyopathy, Heart failure, Diabetes mellitus, Thyroid storm.

Case Report
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Introduction:
Thyroid storm is a rare, life-threatening condition characterized by severe clinical manifestations of thyrotoxicosis. In a national survey from Japan, the incidence of thyroid storm in hospitalized patients was 0.20 per 100,000 per year.¹ The reported incidence of PPCM varies. Much of the reported discrepancy is due to wide geographical variation, with reported incidences of 1:2289 to 1:4000 live births in the United States, 1:1000 in South Africa, 1:300 in Haiti, and 1:100 in Zaria, Nigeria.²³⁻⁹ It is defined as a condition meeting four criteria:¹⁰¹¹

I. Development of heart failure (HF) in the last month of pregnancy or within five months of delivery.

II. Absence of another identifiable cause for the HF.

III. Absence of recognizable heart disease prior to the last month of pregnancy.

IV. LV systolic dysfunction (e.g. left ventricular ejection fraction [LVEF] below 45 percent or a reduced fractional shortening).

Associations of such diseases are very rare. In Bangladesh perspective there is no research data yet available, and hence such a case is reported bellow.

Case Note:
A 35 year-old Diabetic, Normotensive lady was admitted in CCU with the complaints of high grade intermittent fever with productive sputum for 7 days and progressive breathlessness along with altered level of consciousness for 1 day. She was a known case of Grave’s Disease but during her last pregnancy (eight months prior to this admission) the patient voluntarily stopped taking anti-thyroid medication and delivered a healthy male baby through LUCS (two months before...
admission). Her previous pregnancy was uneventful and there was no history of still birth or abortion.

On general examination, she was dyspnoic, restless, confused, agitated, non-cooperative, below-average body-built, having Temp- 102°F, Respiratory rate-30/min, Blood Pressure - 80/60 mmHg, rapid feeble pulse of 120 beats/min and raised JVP of 5 cm from Sternal Angle. The Thyroid gland was diffusely enlarged measuring about 5cm x 4cm, firm in consistency, non-tender, mobile, smooth surface with normal overlying skin without any bruit or any retrosternal extension. On bed side urine examination there was 1+ Sugar with no Albuminuria or Ketonuria.

On Precordium Examination, there was no visible Apical Impulse; Apex Beat was shifted in left 5th ICS 10 cm from the midline which was diffuse in character with no parasternal heave or palpable P2. On auscultation the S1 was normal with loud P2 and there was no added sound.

On Examination of Respiratory System there was tachypnea with restricted chest movement in the right lower chest along with features of consolidation.

On Neurological Examination the Patient was restless, confused, agitated with 12/15 GCS, generalized reduced muscle bulk, brisk exaggerated all deep tendon reflexes with bilateral flexor plantar response. She had bilateral exophthalmos and lid retraction with bilateral normal fundi. Her muscle power was 5/5 in all four limbs with no sign of meningeal irritation or cranial nerve palsy. Gait and Sensory function could not be assessed.

Important laboratory investigations revealed: Hb%-12.8 gm/dl, WBC- 14,420/mm³, ESR- 40 mm in 1st hour, RBS- 21.2 mmol/L, S.FT3- 9.59 mmol/L, S.FT4- 56.4 pmol/L, S.TSH- 0.10 IU/ml, Urine Pus Cell- 2-3/HPF, X Ray Chest (P/A view)- Cardiomegaly with Rt. sided consolidation and pleural effusion (Fig. 1). ECG-Sinus Tachycardia, ST-T changes in Anterior and Inferior leads. Her ABG was; PH: 7.2, PO2- 80 mmHg, PCO2- 17.8 mmHg, HCO3- 7.1 mmol/L, SpO2- 90%. Blood Urea- 30 mg/dl, S. Creat- 1.0 mg/dl, AST- 670 IU/L, ALT-540 IU/L, S. Bilirubin- 1.8 mg/dl, Blood C/S – No Growth, Na+- 142 mmol/l, K+- 4.5 mmol/l, Cl-- 105 mmol/l, TCO2- 09 mmol/l, S. BNP- 2770 pg/ml. Echocardiography- Global Hypokinesia, All Chambers are dilated, LVEF-32%, Mild Pericardial Effusion, Moderate MR with restrictive filling defect Moderate TR, Moderate Pulmonary HTN (PASP= 65 mmHg) (Fig. 2).

The patient was initially treated the patient with; I/V Normal Saline, Inj. Dopamine, Inj. Nor-adrenaline, Inj. Sodium Bicarbonate, Inj. Hydrocortisone, Regular insulin via syringe pump, Oxygen inhalation, Inj. Ceftriaxone, Inj. Levofoxacin, Inj. Frusemide, Inj. Thiamine, Tab. Carbimazole, Tab. Phenobarbitone, Tab. Propranolol and Tab. Clopidogrel. A team consisting of Cardiologists, Endocrinologists and Obstetricians regularly monitored the patient and advocated treatment strategies and other necessities from time to time. After initial treatment patients condition was improved and hemodynamically stable. The patient was transferred to general ward after a 7 day stay in CCU. The inotrope support was withdrawn along with adding of Tab. Digoxin, Combination of oral Frusemide and Spironolactone, oral Iron and Calcium supplements. The patient was discharged with proper counseling regarding outcome of future pregnancy and chances of recurrence of peripartum cardiomyopathy with essence of effective contraception and advice to follow up in Endocrine and Cardiology OPD after 6 weeks with review Thyroid Function Tests, Echocardiography and Serum BNP.
Discussion:

Although thyroid storm can develop in patients with long-standing untreated hyperthyroidism (Graves’ disease, toxic multinodular goiter, solitary toxic adenoma), it is often precipitated by an acute event such as surgery, trauma, infection, an acute iodine load, or parturition. Patients with severe and life-threatening thyrotoxicosis typically have an exaggeration of the usual symptoms of hyperthyroidism. Cardiovascular symptoms in many patients include tachycardia to rates that can exceed 140 beats/minute and congestive heart failure. Hypotension, cardiac arrhythmia, and death from cardiovascular collapse may occur. Hyperpyrexia to 104°F to 106°F is common. Agitation, anxiety, delirium, psychosis, stupor, or coma are also common and are considered by many to be essential to the diagnosis. Severe nausea, vomiting, diarrhea, abdominal pain, or hepatic failure with jaundice can also occur. There are no universally accepted criteria or validated clinical tools for diagnosing thyroid storm. In 1993, Burch and Wartofsky introduced a scoring system using precise clinical criteria for the identification of thyroid storm. This scoring system is likely sensitive, it is not very specific. The degree of hyperthyroidism is not a criterion for diagnosing thyroid storm. Other nonspecific laboratory findings may include mild hyperglycemia, mild hypercalcaemia, abnormal liver function tests, leukocytosis, or leukopenia. Full support of the patient in an intensive care unit is essential, since the mortality rate of thyroid storm is substantial (10 to 30%).

Some actions of T₃ on the heart produce clinical findings similar to those of beta-adrenergic stimulation. Cardiovascular symptoms and signs are common in patients with hyperthyroidism:

- Systolic hypertension with widened pulse pressure
- Exertional dyspnea, which is due more to respiratory and skeletal muscle weakness than cardiac dysfunction
- Angina-like chest pain, with EKG changes suggesting myocardial ischemia, which can occur especially in women; this appears to be the result of coronary vasospasm and responds to treatment with orally administered calcium channel blockers
- Increase in left ventricular mass index and left ventricular hypertrophy
- Increased ventricular irritability, especially in Amiodarone treated patients with a prior history of ventricular ectopy (often in the setting of implanted cardiac defibrillators)

Hyperthyroidism is also associated with an increased risk of atrial fibrillation (5 to 15 percent of patients), heart failure, pulmonary hypertension, and angina. Heart failure is most commonly seen as a result of coexistent atrial fibrillation. The signs and symptoms of heart failure resolve when the ventricular rate is slowed, normal sinus rhythm is restored, and the patients are rendered euthyroid. Heart failure in the absence of underlying cardiac disease or arrhythmia is thought to reflect a rate-related cardiomyopathy, which disappears when the
hyperthyroidism is treated. There is no clear histopathologic correlate of this cardiomyopathy, and treatment is primarily directed at rate control with beta-adrenergic blockade. Pulmonary hypertension can also produce signs of isolated right heart failure with a rise in central venous pressure, neck vein distension, and hepatic congestion. Hyperthyroidism is associated with an increase in N-terminal pro-B natriuretic peptide (NT-proBNP) in hyperthyroid patients without cardiac insufficiency. Despite many attempts to uncover a distinct etiology of PPCM, the cause still remains unknown and may be multifactorial. No distinct hormonal disorder has been identified in patients with PPCM, even though estrogen, progesterone, and prolactin have significant effects on the cardiovascular system. Although the etiology of PPCM remains unclear, a number of factors have been associated with increased risk, including the following: Age greater than 30 years, multiparity, African descent, Pregnancy with multiple fetuses, A history of preeclampsia, eclampsia, or postpartum hypertension, maternal cocaine abuse, Long-term (>4 weeks) oral tocolytic therapy with beta adrenergic agonists such as terbutaline.

Treatment of PPCM is largely similar to that for other types of heart failure (HF). Additional therapeutic issues include anticoagulation and arrhythmia management. Immunosuppression and immune globulin therapy have also been evaluated, although the role of these treatments in PPCM is not established. The prognosis of PPCM must take into account maternal, obstetric, and neonatal outcomes, and the effect of subsequent pregnancy. The largest series of 123 cases of PPCM cited above (23 with onset more than one month before delivery) described a cardiac transplantation rate of 4% and a mortality rate of approximately 10% at a mean follow-up of about two years. Death due to PPCM is usually caused by progressive pump failure, sudden death, or thromboembolic events.

Our patient was suffering from DMT2 and Graves’ Disease but was not adherent to the treatment advice for and with that status of her metabolic derangement she became pregnant and delivered a healthy live baby through LUCS and thereby admitted in CCU BIRDEM for high grade intermittent fever with productive sputum for 7 days and progressive breathlessness along with altered level of consciousness for 1 day. Parturition along with development of pneumonia in a previously noncompliance patient of Diabetic and Grave’s Disease lidos to the development of Thyroid storm and metabolic acidosis and she also had Peripartum Cardiomyopathy at the same setting which was responsible for these kind of critical scenario of heart failure.

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