Diuretic-Resistant Edematous Syndrome in Heart Failure: Look for Hypothyroidism

1 Ramiandrisoa Lahatriniaivo Ritchy, 2 Randriamihangy Narindrarimanana Avisoaa, 3 Miandrisoa Rija Mikhaël, 4 Rakotoarimanana Solofoàrina
1 Intensive Care Unit of Cardiology, Befelatanana University Hospital, Antananarivo, Madagascar
2 Department of Cardiology, Mahavoky Atsimo University Hospital, Mahajanga, Madagascar
3 Department of Cardiovascular Disease, Soavinandriana Hospital, Antananarivo, Madagascar

Abstract: Postpartum thyroiditis occurs in women after delivery and can cause both thyrotoxicosis and hypothyroidism. We report the case of a 29 years old female patient who presented with dyspnea, ascites and asthenia two months after delivery. It was a heart failure with a reduced left ventricular ejection fraction (LVEF = 36%) and a mild systolic pulmonary artery hypertension (sPAP = 37.3 mmHg) contrasting with very significant and persistent congestive signs in spite of diuretic treatment. Cardiovascular risk was low. The thyroid tests showed hypothyroidism (TSH = 8.1 mU/l, T4L = 2.9 pmol/l). Improvement in general condition of the patient and congestive signs were obtained five days after a levothyroxine therapy. Thyroid biological test should be part of the etiological research of heart failure in the peripartum period, especially when the patient’s cardiovascular risk is low.

Keywords: cardiomyopathy - heart failure – hypothyroidism.

INTRODUCTION

Postpartum thyroiditis (PTT) is thyroiditis that occurs in women after delivery and can cause both thyrotoxicosis and hypothyroidism (1). In postpartum thyroiditis, thyrotoxicosis occurs first (two months postpartum) followed by hypothyroidism (third up to sixth month in postpartum) (1). It typically happens during the first year following childbirth (1). Thyroid hormone has a great effect on the heart and vascular system (2). The heart is sensitive to changes in thyroid hormones and cardiac disorders are commonly associated with both hyper- and hypothyroidism (3-4). Dilated cardiomyopathy is a rare presentation of hypothyroidism (5).

Our goal was to report a cardiomyopathy due to hypothyroidism in the post-partum period.

CASE PRESENTATION

We report a case of a 29 years old female patient (Pregnancy=3, Parity=3, Abortion=0), who presented with an abdominal distension, a difficulty in breathing and asthenia two months after delivery.

The abdominal distension progressively increased with lower limbs swelling. Difficulty in breathing was of gradual onset, progressively increasing in severity, initially on exertion, later even at rest requiring the use of up to 2 pillows while sleeping with occasional nocturnal dry cough. There was no chest pain or history of fever. The patient didn’t have hypertension or pregnancy induced hypertension. She was not diabetic and had no history of cardiopathy. There was no difficulty or hemorrhage during delivery.

On day of admission, the patient was orthopneic, respiratory rate was 28 breaths/min, oxygen saturation was 98% with bilateral bibasal crepitations. The blood pressure was 109/67mmHg. Cardiac auscultation found a heart rate of 107 beats/min, a B3 sound, a grade 3 pansystolic murmur radiating to axilla suggesting a mitral regurgitation and a tricuspid regurgitation murmur. The patient presented with ascites, infiltration of abdominal subcutaneous tissues and a severe edema of the lower limbs up to the root of the thighs. There was no axillary or pubis depilation.

Blood investigations showed the following results: white blood cells (K/UL): 6.6, hemoglobin (g/dl): 9.4, platelets (K/UL): 229, albumin (g/L): 32, Na+ (mmol/l): 132, K+ (mmol/l): 3.9, Cl- (mmol/l): 93, creatinine (μmol/l): 98, eGFR 65mls/min. Chest x-ray showed cardiomegaly without any image of pneumopathy. Twelve-lead ECG showed sinus tachycardia (110 beats/min), low voltage on frontal derivations and planed R wave from V1 to V3. Echocardiography showed a dilated left ventricle (57 mm in telediastole), a dilated left atrium (28 cm²), a global hypokinesia with ejection fraction of 36% (Simpson biplane). Left ventricular filling pressures was high (E/Ea = 16.5). There was a mild systolic pulmonary arterial hypertension (sPAP = 37.3 mmHg) and a moderate pericardial effusion.

Initial diagnosis was a NYHA IV acute decompensated heart failure secondary to a peripartum cardiomyopathy. The patient was treated with intravenous furosemide 200 mg daily, ramipril 2.5 mg twice daily, carvedilol 3,125 mg daily and potassium supplementation. With this treatment, respiratory status improved but peripheral congestive signs persisted with unsatisfactory weight loss although renal function was correct.

We suspected an associated myxedema because of the severe and diuretic-resistant edemas contrasting with a mild pulmonary arterial hypertension. Thyroid investigations showed an increase of the thyroid stimulating hormone or TSH (8,1 mU/l for a normal range of 0,3 - 4,5 mU/l) and a decrease of free thyroxine (2,9 pmol/l for a normal range of...
8.6 - 25 pmol/l). Ultrasound showed a normal sized but hypoechogene thyroid. A.M. plasma cortisol was checked to assess other pituitary hormone lines and it was normal. Hormone replacement therapy was started with levothyroxine 100 µg tablet a day. Then oedematous syndrome markedly decreased and the general condition of the patient greatly improved five days later.

**DISCUSSION**

PTT is an autoimmune disease, characterized by the presence of antimicrosomal antibodies according to Neal DM et al. Histologic examination of PTT-affected thyroid glands reveals destructive lymphocytic thyroiditis (6).

Thyroid hormones act on cardiac myocytes and peripheral vasculature. The genomic and non-genomic effects of thyroid hormone are related to the cardiac function and cardiovascular hemodynamics (4). To explain their possible genomic effects on the cardiovascular system, it has been proposed that thyroid hormone is involved in the regulation of the messenger ribonucleic acids (mRNA) transcription of genes associated with the contractile system (2). They have a non-genomic effects on the ionic channels of cardiomyocyte's membrane (4). So, apart from autoimmunity mechanism, hypothyroidism is associated with decreased cardiac contractility, increased systemic vascular resistance and decreased cardiac output (7). Moreover, patients with hypothyroidism are at increased risks of developing atherosclerosis and ischemic heart disease (8-9). It has been reported that the cardiac manifestations are associated with alterations in the expression of T3-mediated genes in patients with thyroid dysfunction (4-10). According to Curotto et al, patients with chronic heart failure and hypothyroidism significantly improve their physical performance when normal TSH levels were reached (11).

The main differential diagnosis in this situation is Sheehan syndrome which is due to a panhypopituitarism following a postpartum hemorrhage (12). The cause of this panhypopituitarism is thought to be ischemic necrosis of the anterior pituitary secondary to postpartum hemorrhage (12). Cardiac abnormalities have been reported in patients with hypopituitarism, most of these being linked to growth hormone deficiency, hypothyroidism and/or hypocortisol state (13-14). That syndrome is rare but should be sought (12). In our case, the absence of postpartum hemorrhage, the presence of axillary and pubic hair and the normality of other pituitary hormone especially the cortisol were against that diagnosis.

**CONCLUSION**

Our case highlights that clinicians should consider the possibility of hypothyroidism as a cause of diluted cardiomyopathy in the postpartum period, when edematous syndrome is resistant to diuretics, especially when the patient’s cardiovascular risk is low. Peri-partum cardiomyopathy is a diagnosis of exclusion.

**RÉFÉRENCES**


