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PARTIAL EMPTY SELLA SYNDROME WITH PAN HYPOPITUITARISM LEADING TO PERICARDIAL EFFUSION.

Endocrinology

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ABSTRACT

There have been number of leading causes of pericardial effusion but secondary hypothyroidism has rarely been described as a cause of pericardial effusion. Here we present a case of 35 years female who presented with anasarca, shortness of breath and easy fatiguablity. Her echocardiography revealed pericardial effusion with impending cardiac temponade. There was evidence of secondary hypothyroidism with decreased free T3 and T4 and normal TSH levels. Further detailed workup history revealed history of post-partum hemorrhage few years back with loss of menstruation and lactation following it. Suspicion of pituitary disorder was made which was confirmed by decrease FSH, LH, prolactin levels. MRI brain confirmed diagnosis of partial empty sella turcica syndrome. This case indicated the need to consider hypothyroidism, even central, as a possible etiology in patients with unexplained pericardial effusion. Since Sheehan's can present at a variable duration after the last pregnancy it has to be considered even if history of childbirth is remote.

KEYWORDS

Consumer, Consumer right, Consumerism, Anti-consumerism, Consumer tolerance, Unfair trade practices,

Introduction

Sheehan syndrome (SS) is caused by postpartum necrosis of the pituitary gland, associated with significant hemorrhage during or after delivery[1]. It is rare in developed countries, but still frequent in underdeveloped ones. Usual presentation of this syndrome is mainly lactation failure and amenorrhea, fatigue, weakness, hair loss, fine wrinkles around face, loss of libido, polyuria, hypoglycemia and hypotension etc.[2] This is because of higher blood supply needed by pituitary as a result of physiological hypertrophy during pregnancy. One of the unusual manifestation is Pericardial effusion. Although common in primary hypothyroidism it is rare in central hypothyroidism.[3] In such cases tamponade can develop slowly and may mimic heart failure with features of dyspnea, orthopnea, tender hepatomegaly.

Evidence suggests a role of autoimmune response to sequestered antigens released as a result of pituitary necrosis in the causation of Sheehan's syndrome. This ongoing autoimmune phenomenon explains the late presentation of Sheehan's in the form of circulatory collapse (hypocortisolism and hypothyroidism) occurring long after the initial manifestations of acute loss of pituitary function (agalactia and amenorrheic). In fact it has been seen that a higher percentage of patients with Sheehan's syndrome had pituitary antibodies as compared to controls.[6] Here we present a case with a rare case of pericardial effusion which led to delay in diagnosis as Sheehan syndrome is a rare entity per se.

Case report

A 44 years female presented to our emergency with history of anasarca from last eight years. It was gradual, progressing from bilateral lower limb to face. It was associated with shortness of breath of same duration initially NYHA II to NYHA III from last one month. It was accompanied by myalgia and decreased appetite.

On examination her pulse rate was 48 beats per minute, blood pressure of 80/60 mm Hg in right arm supine position. Her JVP was raised and other physical examination attributes included facial puffiness, bilateral lower limb non pitting type of edema, thick coarse skin. Systemic examination revealed muffled heart sound with delayed types of deep reflexes. Other systems were within normal limits.

Patient was investigated. Her routine blood revealed haemoglobin of 9.2mg/dl, TSH on lower side 0.412 and echocardiography suggestive of mild to moderate pericardial effusion with impending cardiac temponade.

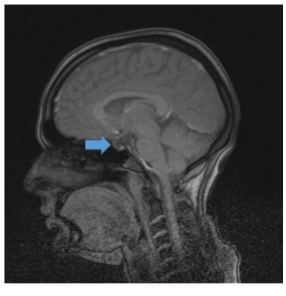
Since it was a long drawn process possibility for chronic pericarditis was considered.It included Chronic infection, Myxedema, Neoplastic, Uremia, Collagen vascular diseases, Chronic anemia, Chyloperi cardium.

Further work up was done. Her pericardiocentesis sugar 91mg/dl, total cells 345 cells/mm^3, total protein of 40mg/dl and no growth on routine and culture. The above findings were insignificant. Patient was evaluated for connective tissue disorder. Her antinuclear antibody test was negative. Thyroid profile was repeated. Her T3 was below 1 pg/ml, Free T4- below 0.4 ng/dl And TSH- 2.9 uIU/ml.This led to clinical suspicion of secondary hypothyroidism leading to pericardial effusion. So were we dealing with some pituitary disorder?

Patients history was revised and further detailed workup revealed patient had obstetric history of post partum haemorrhge eight years back leading to massive blood loss. Patient received multiple blood transfusions then. This event further led to failure to lactate and Amenoorhea.

We made the possibility of Sheehan syndrome. Her FSH and LH were checked which were on lower side. Growth hormone could not assessed because patient already received Intravenous steroid. Her prolactin levels were within normal range which after provocative test of injecting metoclopropamide did not rise.

Figure 1: MRI Brain with pituitary. Arrow represents sella filled with CSF compressing pituitary gland.



MRI brain with pituitary was done which revealed pituitary sella filled with CSF compressing pituitary and above findings were suggestive of partial empty sella syndrome.

So our final diagnosis at time of discharge Partial empty sella syndrome with panhypopituitarism leading to pericardial effusion. Patient was started on oral steroids along with thyroxine replacement done. Patient is being followed up regularly and is in improving state.

Discussion

Cardiac manifestations of hypothyroidism are pericardial effusion, increased left ventricular dimension with left ventricular dysfunction, wall motion abnormality, asymmetric septal hypertrophy and mitral valve prolapse[4]. Pericardial effusion in hypothyroidism is occasionally large and cardiac tamponade is rare [5].

It is attributed to an increased capillary permeability with protein extravasation, combination of sodium and water retention and slow lymphatic drainage[6] .

In Sheehan's syndrome the presence of low cortisol may result in low-pressure tamponade. In low-pressure tamponade, usually occurring in hypovolemia, the features can be subtle and easily missed. Very few cases of Sheehan's syndrome leading to pericardial effusion has been reported earlier [7-9]

In such cases tamponade can develop slowly and may mimic heart failure with features of dyspnea, orthopnea, tender hepatomegaly.

The management consists of management of anemia, hypotension, hypoglycaemia and any other complication. Adequate replacement of deficient hormones should be done. It is important to start steroids prior to thyroxine replacement because thyroxine replacement can lead to adrenal crisis by enhancing the metabolism of glucocorticoids and increasing the metabolic needs of the body.[10]

This case indicates the need to consider hypothyroidism, even central, as a possible etiology in patients with unexplained pericardial effusion. Since Sheehan's can present at a variable duration after the last pregnancy it has to be considered even if history of childbirth is remote. Therefore follow-up of patients of postpartum haemorrhage for development of lactational failure may help in preventing more serious late complications like hypothyroidism and hypocortisolism.

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