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Short Communication

When should you think about the Thyroid? Refractory Transudative Ascites

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Abstract

Introduction:
Hypothyroidism is a frequent condition in medical practice and clinical forms with ascites are exceptionally rare. After a review of the literature, we found that the exudative nature of the fluid is the main feature associated with hypothyroidism, however no case of hypothyroidism associated with transudative ascites has been reported. We report a case of transudative ascites associated with hypothyroidism in a post thyroidectomy setting.
Observation: This 72-year-old patient underwent total thyroidectomy 3 years ago, without supplementation with synthetic thyroid hormone. He had been treated in cardiology for compensated ischemic heart disease for 4 months. He presented with apathy, significant physical asthenia and a hoarse voice. His general condition was altered, with a rounded, puffy face and infiltrated eyelids. The feet were edematous. Hemodynamic constants revealed arterial hypotension. Ascites aspiration yielded a sterile, pauci-cellular, citrine-yellow, transudative fluid. After ruling out renal, glomerular and hepatic causes, the ascites persisted despite optimized treatment of his heart disease, making cardiac ascites unlikely. Signs of hypometabolism and myxedema, together with a very high TSH (TSHus = 54.26 microgr/L) and disappearance of ascites after thyroid hormone supplementation, supported the hypothesis of transudative ascites associated with hypothyroidism.
Discussion: Ascites associated with hypothyroidism is rare and the transudative nature of the fluid is exceptional; it is generally included in myxedema.
Conclusion: Hypothyroidism is rarely manifested by ascites but can be considered after ruling out common causes. Hormonal treatment allows complete regression of ascites within a few weeks and constitutes a therapeutic test.

Keywords: hypothyroidism; thyroidectomy; myxedema

I-Introduction

Hypothyroidism is common in clinical practice [1].

Situations with serous fluid effusion are exceptional. Ascites is a rare manifestation of hypothyroidism [2].

Exudative ascites is characteristic of the fluid described in the observations that we found in the literature, however no case of hypothyroidism associated with transudative ascites has been evoked. We report a case of transudative ascites associated with hypothyroidism in a post thyroidectomy setting.

II- Observation:

This is a male patient, 72 years old, who 3 years ago underwent a total thyroidectomy, without synthetic thyroid hormone supplementation, for a goiter that had been developing for 5 years, of undocumented cause and characteristics. In addition, he had been undergoing cardiological treatment for ischemic heart disease for 4 months. He had been hospitalized several times, mainly for the investigation and treatment of refractory ascites, despite the optimization of medical treatment for his heart disease.

In this context, he was admitted to our department for evaluation of ascites, which had been developing for 6 months, without signs of portal hypertension or hepatocellular insufficiency, or other signs related to right heart failure.

He presented with apathy, marked physical asthenia and hoarse voice.

The physical examination revealed a deterioration in general condition that would be classified as WHO **stage 3.** The face was round and swollen with infiltrated eyelids. The feet were edematous. Hemodynamic constants showed arterial hypotension (BP = 90/57 mmHg); HR = 67 beats/min; T = 36°C.

The abdomen was distended with no collateral venous circulation, a positive float sign and no palpable mass. The heart sounds were muffled, the rhythm

regular, with no murmur. Peripheral pulses were felt, and there was no turgidity of the jugular veins or cardiac liver or hepatojugular reflux. Lung examination was unremarkable. The rest of the physical examination was normal. An exploratory and evacuation ascites puncture was performed, yielding 3 liters of citrine yellow fluid, cytochemical analysis of which showed a transudative fluid (protein =21g/L), sterile, poorly cellular (red blood cells <8e/mm3, leukocytes 5 elements/mm3, N = 03elmt/mm3, Lym=08elmt/mm3).

We suspected glomerular nephropathy, cardiac decompensation from ischemic heart disease, hepatic cirrhosis and hypothyroidism with myxedema. The electrocardiogram showed signs of necrotic sequelae in the anteroseptal-apical territory and micro voltage anteriorly. The cardiac echo showed a segmental kinetic disorder in favor of ischemic heart disease and Coronary angiography was not performed due to lack of funds, Abdominal ultrasound showed abundant free ascites, a normal-sized homogeneous liver, non-dilated prehepatic veins, and normal-sized, well-differentiated kidneys. He had normocytic normochromic anemia (hemoglobin = 8.3g/dl), low PT (62%), ASAT= 22 UI ALAT= 10UI, normal renal function (urea= 0.44; creatin = 7.9 mg/l GFR = 117 ml/min/1.73 m²), urine chemistry with multiparametric urine dipstick showed no proteinuria, albuminemia = 35 g/l.

Thyroid work-up revealed profound hypothyroidism (Thus = 54.26 microgram/L). A glomerular cause was ruled out in the absence of proteinuria. Renal disease was ruled out on the basis of normal renal function and renal ultrasound. The ascites was isolated, the prehepatic veins were not dilated, and the ascites persisted despite optimized treatment of his heart disease, making a cardiac cause unlikely.

In view of the signs associated with hypometabolism and myxedema, as well as the very high TSH and disappearance of the ascites after supplementation, the hypothesis of ascites associated with hypothyroidism was confirmed. He progressed on L- thyroxine initially at 12.5 microgram/day and then gradually increased to 50 microgram/D. For ischemic heart disease, he was on Aspirin 100mg/d, atorvastatin 20mg/d, and ACE inhibitors and beta-blockers were only introduced after a return to normal blood pressure. The evolution was marked by considerable regression of ascites after 3 weeks of supplementation, followed by complete disappearance without subsequent reconstitution.

III- Discussion:

Ascites associated with hypothyroidism is rare, and is generally included in myxedema. We have listed about ten cases described in the literature, all of which observed exudative ascites. No study had suggested transudative ascites in the context of hypothyroidism.

Our patient was being followed in cardiology for ischemic heart disease with impaired LVEF, diagnosed after the onset of ascites. Ascites persisted despite optimization of medical treatment for his heart disease. After 3 weeks of treatment with thyroid hormone, we obtained complete and definitive regression of the ascites.

Akkari et all [1] in Sousse (Tunisia) in six cases, found a progressive deterioration in general condition and a progressive increase in the volume of the abdomen. These observations were the same as those seen in our case study.

Ascites was a sterile exudate A. Largan et al [2] in Rabat about a case, identical to the observations of A. Grati et al [3] in 2016 about 3 cases in Tunis, I. Akkari et al [1] also evoked an exudative, sterile ascites, with lymphocytic predominance. In our case, the ascites was transudative and cell-poor.

We found normochromic, normocytic anemia; these characteristics are consistent with observations in the literature. [1 ;3 ;4]

The ascites had completely regressed after the introduction of hormone therapy; this was the case in all the cases observed. [1 ;2 ;3 ;4]

Conclusion

Hypothyroidism rarely manifests as ascites, but can be considered after ruling out common causes.

Hormonal treatment leads to complete regression of ascites within a few weeks and constitutes a therapeutic test.

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