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Research Article

Hypothyroidism and Transudative Ascites: Highlighting a Little-known Association

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Abstract

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. 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 ischaemic 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 oedematous. Haemodynamic 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 optimised 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. Ascites associated with hypothyroidism is rare and the transudative nature of the fluid is exceptional; it is generally included in myxedema. 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, Transudative Ascites, Myxedema

1. Introduction

Hypothyroidism is common in clinical practice [1]. Ascites is an accumulation of fluid in the peritoneal cavity [2]. The formation of pathological ascites results from an imbalance between fluid production and resorption at the peritoneal level [3].

Hypothyroidism can lead to altered hemodynamic parameters, resulting in pericardial or pleural effusion, or even edema, defining a picture of myxedema. This is an exceptional cause of ascites, accounting for less than 1% of etiologies. Similarly, ascites occurs in around 4% of hypothyroid-

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ism cases [4]. It is a revealing symptom of hypothyroidism that is rare in medical practice [5, 6], and is part of the myxedema framework [4].

Ascites analyses of patients with this condition generally show elevated protein levels [4, 5, 7].

Diagnosis is based on clinical presentation, hormone assays and a rapid, positive response to thyroid hormone therapy [4-6].

Exudative ascites is the characteristic feature of the fluid described in the observations we found in the literature [1, 4-9, 11], but we believe that this entity has been studied in too few cases to date to be conclusive, however no case of hypothyroidism associated with transudative ascites had been described previously.

We report the observation of a case of transudative ascites associated with hypothyroidism in a post-thyroidectomy setting.

2. 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 ischaemic heart disease for 4 months. He had been hospitalised several times, mainly for the investigation and treatment of refractory ascites, despite the optimisation 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 oedematous. Haemodynamic constants showed arterial hypotension (BP = 90/57 mmHg); HR = 67 beats/min; T = 36 ℃.

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 litres of citrine yellow fluid, cytochemical analysis of which showed a transudative fluid (protein =21g/L), sterile, poorly cellular (red blood cells <8e/mm^3, leukocytes 5 elements/mm^3, N = 03elmt/mm^3, Lym=08elmt/mm^3).

We suspected glomerular nephropathy, cardiac decompensation from ischaemic heart disease, hepatic cirrhosis and hypothyroidism with myxedema.

The electrocardiogram showed signs of necrotic sequelae in the anteroseptal-apical territory and microvoltage anteriorly.

The cardiac echo showed a segmental kinetic disorder in favour of ischaemic 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 suprahepatic veins, and normal-sized, well-differentiated kidneys.

He had normocytic normochromic anaemia (haemoglobin = 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 m^2), urine chemistry with multiparametric urine dipstick showed no proteinuria, albuminemia = 35 g/l.

Thyroid work-up revealed profound hypothyroidism (TSHus = 54.26 microgr/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 suprahepatic veins were not dilated, and the ascites persisted despite optimised 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 microgr/day and then gradually increased to 50 microgr/D.

For ischaemic 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.

3. Discussion

Ascites associated with hypothyroidism is rare, and is generally included within the framework of myxedema. However, the importance of its diagnosis lies in the fact that the use of thyroid hormone replacement leads to complete resolution. Should ascites appear, the diagnostic work-up should begin with analysis of the ascites fluid.

We have identified ten cases described in the literature, all of which showed exudative ascites. No study had evoked transudative ascites in the context of hypothyroidism.

The mechanism of ascitic fluid formation in patients with myxedema is unclear. There are two main hypotheses. The first is that low levels of circulating thyroid hormones cause increased extravasation of plasma proteins due to increased capillary permeability and the absence of a compensatory increase in lymphatic flow and protein return rate [5]. The

second hypothesis is that hyaluronic acid accumulates in the skin and produces edema by direct hygroscopic effect. However, hyaluronic acid has only been found in minute quantities in patients with myxedema ascites: not large enough to exert a direct hygroscopic effect. However, it could interact with albumin to form complexes preventing lymphatic drainage of extravasated albumin [3].

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

Ascites was a sterile exudate A. Laargane et al [10] 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, predominantly lymphocytic. In our context, the ascites was transudative and cell-poor.

A very consistent finding was the high level of total protein. Total protein levels exceeded 2.5 g/dL in almost all cases. White blood cell counts were rather low, with lymphocytes generally predominating. In our patient, the white blood cell count was 05 and the proportion of lymphocytes was 70%.

We found a normochromic, normocytic anemia. These characteristics are in line with observations found in the literature. [1, 3, 4].

Table 1. *Summary table of the characteristics of the ascites and the duration of the hormone replacement therapy, according to some case reports found in the literature [1, 5, 6, 8-10, 13-18].*

Authur	Title	characteristic of ascites		Duration
		Nature	Cell	
Jeong-Seon Ji	Myxedema ascites: case report and literature review.	Ex	84%	12 Weeks
Marc Atzenhoefer	Case report of hypothyroidism presenting with myxedema ascites	Ex	NF	4 Weeks
Imen Akkari	Hypothyroidism: a rare cause of exudative ascites	Ex	NF	NF
TITU MIAH	Hypothyroidism with ascites	Ex	20 cell/mm^3	8 Weeks
Vidhya Subramanian	Symptomatic ascites in a patient with hypothyroidism of short duration	Ex	NF	8 Weeks
Sana Riaz	Myxedema ascites complicated by ischemic colitis			
Otero Bedoya	Ascites due to hypothyroidism in a patient with alcoholic cirrhosis	Ex	L. predominance	NF
F de Castro	Myxedema ascites. Report of two cases and review of the literature	Ex	NF	NF
Naoki Gotyo	Respiratory failure with myxedema ascites in a patient with idiopathic myxedema	Ex	NF	12 Weeks
Youssoufa zafar	Hypothyroidism Manifesting as a Combination of Ascites and Malnutrition Requiring Total Parenteral Nutrition			8 Weeks
C Abby Philipps	Partha Chattopadhyay. Isolated ascites in hypothyroidism: medical and ethical issues	Ex	NF	NF
H Canehara	Myxedema ascites with an extremely elevated CA125 Level: a case report	Ex	NF	NF
R Dhingra	Myxedema Ascites: An Unusual Presentation of Uncon-trolled Hypothyroidism Cureus.	Ex	NPC0%, L 57%	NF

NF= no find; NPC = neutrophil polynuclear cell, L= Lymphocytes, Ex= Exudative.

Ascites had completely regressed after initiation of hormone therapy this is the same for all cases observed. [1-12]

4. Conclusions

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.

Once routine evaluation of ascites excludes common causes such as liver cirrhosis, malignancy, peritoneal infection and congestive heart failure, thyroid function should be assessed in any suggestive context. Thyroid function evaluation should be carried out in any suggestive context, regardless of the exudative or transudative nature of the ascitic fluid.

Conflicts of Interest

The authors declare no conflicts of interest.

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