



Introduction

Out of the many causes of exudative ascites, hypothyroidism is among the least documented. We present a case of myxedema ascites diagnosed in a patient with end stage renal disease (ESRD) secondary to polycystic kidney disease.

Case Description

55-year-old man presented with six months of increasing abdominal distension and dyspnea. Associated symptoms included cold intolerance, lower extremity swelling, myalgias, generalized weakness, constipation and weight gain. Past medical history included end stage renal disease secondary to polycystic kidney disease on hemodialysis without history of liver disease, cancer, radiation, or neck surgery. Tuberculosis risk factors were present.

<u>Vitals</u> Temp 37.2°C | Pulse 54 | BP 182/85 | RR 22 | SpO2 90%

Clinical Exam

GEN: chronically ill-appearing, temporal and interosseous wasting

HEENT: anicteric sclera, periorbital edema, no thyromegaly

CV: II/VI systolic murmur and bilateral inspiratory crackles

ABD: tense and distended with shifting dullness

EXT: 3+ mixed pitting and non-pitting edema to the thighs with hyperkeratosis

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AST 28 ALT 19 ALP 322 T.Bili 0.3 Alb 2.2 Protein 4.8 INR 1.06

Bedside ultrasound: large amount of ascites

Echocardiogram: reduced ejection fraction 45-50%, no pericardial effusion

Work-up supporting a diagnosis of myxedema ascites

Ascitic Fluid Analysis Albumin 1.3 Protein 4.8 SAAG 0.9 (exudative) Gram stain: negative

Cytology: few reactive lymphocytes, no malignant cells

Cultures: bacterial, fungal and mycobacterium negative

Exudative Ascites Work-up

Ascititc adenosine deaminase negative Serum Quantiferon Gold negative

CT scan chest, abdomen and pelvis: negative for malignancy, cirrhosis, lymphadenopathy, portal venous thrombosis or portal hypertension

TSH: 89 fT4: 0.1

A Dropsical Diagnosis

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Figure 1- Abdominal ultrasound, longitudinal view of liver outlining a large anechoic area consistent with ascites. Large volume paracentesis was performed following this ultrasound and 9.4 L of ascitic fluid was removed.



Improvement in TSH after initiation of levothyroxine

Figure 2- Patient received a loading dose of IV levothyroxine followed by weight based dosing. Three months later, the patient's thyroid function normalized and his ascites significantly improved, with decrease in frequency of paracentesis.



Proposed mechanism of myxedema ascites

Low circulating thyroid hormone causes abnormal capillary permeability causing increased extravasation of plasma proteins leading to high protein in ascitic fluid¹



Discussion

Hypothyroidism is rarely a cause of exudative ascites and is termed myxedema ascites. The exact mechanism is not fully understood. Myxedema ascites characteristically has a high protein content (>2.4 g/dL) and resolves within 3-6 months after initiation of thyroid hormone.² Although this patient still has ascites, the frequency of large volume paracentesis has decreased significantly.

In patients with ESRD, the prevalence of hypothyroidism is higher compared to the normal population, perhaps because renal disease affects the hypothalamic-pituitary-thyroid axis and thyroid hormone metabolism. Over half of ESRD patients with effusions of any kind have concomitant hypothyroidism, suggesting that hypothyroidism may contribute to fluid collection.³

Cardiomyopathy has also been documented in patients with myxedema ascites, which is reversible with levothyroxine treatment.⁴ Consistent with these reports, repeat echocardiogram in the patient described showed normalization of patients ejection fraction from 45-50% to 55-60%.

The diagnosis of hypothyroidism can be obscure in ESRD as signs and symptoms are similar. This case highlights the need to consider hypothyroidism as a cause of new onset ascites in patients with ESRD prior to additional invasive and unnecessary testing.

References

- 1. Parving HH, Hansen JM, Nielsen SL, et al. Mechanisms of edema formation in myxedma increased protein extravasation and relatively slow lymphatic drainage. NEJM. 1979; 301: 406-5.
- Ji JS, Chae HS, Cho YS, et al. Myxedema ascites: case report and literature review. Journal of Korean medical science. 2006;21(4):761-764.
- Paudel K. Prevalence and clinical characteristics of hypothyroidism in a population undergoing maintenance hemodialysis. Journal of clinical and diagnostic research : JCDR. 2014;8(4):Mc01-04.
- Gotyo N, Hiyama M, Adachi J, Watanabe T, Hirata Y. Respiratory failure with myxedema ascites in a patient with idiopathic myxedema. Internal medicine (Tokyo, Japan). 2010;49(18):1991-1996.