MYXOEDEMA ASCITES WITH CONCOMITANT SPONTANEOUS BACTERIAL PERITONITIS

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ABSTRACT

Hypothyroidism is an uncommon cause of ascites. Here we describe a case of a 71 year-old female patient with myxoedema ascites and subclinical spontaneous bacterial peritonitis that resolved with thyroid replacement and antibiotic therapy respectively. Ascitic fluid analysis revealed a gram-positive bacterium, specifically enterococcus faecium that was sensitive to vancomycin. A review of the literature revealed just one other reported case of myxoedema ascites with concomitant subclinical spontaneous bacterial peritonitis.

Key words: Peritonitis, Ascites, Hypothyroidism, Myxoedema.

Conflict of interest: None.

INTRODUCTION

Spontaneous Bacterial Peritonitis (SBP) is commonly associated with alcoholic and non-alcoholic liver cirrhosis. A literature search revealed only one other reported case of myxoedema ascites associated with SPB [1]. SPB typically presents with abdominal pain, fever and raised inflammatory markers such as C reactive protein and white blood cell count.

The case reported here describes a patient with myxoedema ascites with concomitant subclinical SBP. This patient did have other risk factors for developing ascites such as a previous history of alcohol excess and hepatitis B infection. However investigations eliminated cirrhosis of the liver, portal hypertension, malignancy, heart failure and other common causes of ascites. Hypothyroidism was determined to be the most likely cause of her symptoms. Treatment with thyroid replacement therapy led to complete regression of the ascites and antibiotic therapy successfully treated the SBP.

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CASE REPORT

A 71-year-old Caucasian woman presented with a 3 month history of increasing shortness of breath and increasing abdominal girth. Her past medical history included hypothyroidism for which she was non-compliant with treatment and intermittent claudication. She was a heavy smoker with 100 pack-years of exposure and a history of alcohol excess. She had stopped drinking alcohol 3 months prior to presentation but had drank 2 bottles of wine per day for 10 years.

Clinical examination revealed a cachectic woman with a temperature of 36.0°C. Blood pressure was 127/79mmHg, regular of pulse 80/minute and respiratory rate of 20/minute. Cardiopulmonary examination was unremarkable. Abdominal examination revealed a non-tender tense ascites. The white blood cell count was 7.3x10^9/L (4.0-11.0x10^9/L), haemoglobin 113g/L (115-165x10^9/L), MCV 100fl (76-98fl) and platelets 378x10^9/L (150-450x10^9/L). The C-reactive protein was 12mg/L (0-5mg/L). Despite the previous history of alcohol excess the alkaline phosphatase level was 62U/L (35-104U/L), the total bilirubin was 6umol/L (4-17umol/L), the alanine amino-transferase was 17 IU/L (7-32U/L). The total serum protein level was 65g/l (63-83g/L), the albumin was 43g/L (34-48g/L) and the international normalised ratio (INR) was 1.0. Thyroid function studies revealed a thyroid stimulating hormone (TSH) of 136.70mIU/L (0.3-5.0mIU/L) and a free T4 of 1.0pmol/L (12-22pmol/L). Amylase was 39 Hepatitis core total antibody was positive indicating infection with hepatitis B virus at some time in the past.

Ultrasound of the abdomen revealed marked ascites but no indication of liver cirrhosis. Liver biopsy showed that the hepatic parenchyma showed no evidence of steatosis or liver cell necrosis. There was no evidence of fibrosis, inclusion, granulomas, dysplasia or malignancy. Computer tomography of the abdomen and pelvis revealed a large volume of ascites with a pericardial effusion and no feature of liver cirrhosis. Echocardiogram revealed good systolic function and a pericardial effusion.

Tumor markers included CA19-9 of 8U/L (0-27U/L), alpha fetoprotein of 5.4U/mL (<5.8U/mL), CA15-3 of 32U/L (0-25U/L), carcinoembryonic antigen of 5μg/L (0-4μg/L) and Ca125 which was 750U/ml (0-34U/ml). A trans-vaginal ultrasound and the computer tomography of the abdomen and pelvis found no abnormalities. The Ca125 was repeated when the thyroid function had improved and the ascites resolved and had decreased to 42U/ml.

A diagnostic and therapeutic ascitic drain was conducted. The ascitic fluid had a total protein level of 45g/L, an albumin level of 30g/L and a glucose level of 5.2mmol/L. The white cell count was 323x10^9/L, 90% of which were polymorphs. The ascitic culture grew enterococcus faecium that was sensitive to vancomycin. This asymptomatic spontaneous bacterial peritonitis was treated with a 5 day course of 2g of vancomycin twice a day. Thyroid treatment was also recommenced and at time of discharge thyroid stimulating
hormone was 16.03mIU/L and free T4 was 14.8pmol/L. At 2 month follow up in clinic she remains free from ascites.

DISCUSSION

Hypothyroidism is a common clinical condition and is complicated by myxoedema ascites in less than 4% of cases. Ascites caused by hypothyroidism is also rare accounting for less than 1% of new onset ascites [2]. De Castro et al’s review of 18 reported cases of myxoedema ascites indicated that symptoms resolve with thyroid replacement therapy [3]. Therefore myxoedema ascites is an easily treated and preventable condition with careful thyroid replacement therapy.

The mechanisms by which a patient with myxoedema develops ascites is unknown. There have however been several hypotheses proposed. The first hypothesis proposed that the oedema was produced by a direct hygroscopic effect due to hyaluronic acid accumulation in the skin. However this unlikely to be significant in myxoedema ascites as only minute quantities of hyaluronic acid are present. However albumin hyaluronic acid complexes could form preventing drainage of extravasated albumin [4]. The second hypothesis proposed increased capillary permeability in myxoedema that returns to normal with thyroid replacement therapy [5]. The third hypothesis proposed that in the hypothyroid state, there is a reduced rate of albumin synthesis and catabolism, an increased rate in the transcapillary escape of albumin and an increase in the extravascular mass of albumin [6].

SBP is a common complication of alcoholic and non-alcoholic liver cirrhosis, however it is a rare complication of myxoedema ascites. Literature searches of MEDLINE and EMBASE only yielded one previous reported case of SBP in a patient with myxoedema ascites [1].

It has been suggested that low protein concentrations in ascitic fluid predisposes to SBP [7]. De Castro et al’s review of 18 reported cases of myxoedema ascites revealed that a feature of the condition is a high protein ascitic fluid content [3]. Therefore high ascitic protein levels may partially account for low levels of SBP in patients with myxoedema ascites. In this case described above the ascitic protein level was high so other factors must have predisposed the patient to SBP. Hypothyroidism has been proposed to suppress cell mediated immunity, Animal studies indicate that severe clinical hypothyroidism depresses lymphocyte function and treatment with thyroid replacement therapy reverses this effect [8]. These studies have not been verified in humans however Schoenfeld et al suggest this as a cause for bacteraemia in a male patient with severe hypothyroidism [9]. Runyon et al suggest dilution of crucial antimicrobial proteins below a threshold predisposes to SBP [10].

In conclusion we described a case of myxoedema ascites complicated with subclinical SBP. Other causes for ascites such as liver cirrhosis, malignancy, pancreatitis, renal disease and cardiac disease were excluded. Full recovery
was made with antibiotic and thyroid replacement therapy and ascites has not
recurred.

REFERENCES