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Case Report:

Spontaneous Bacterial Peritonitis in Subclinical Hypothyroidism

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Abstract: Hypothyroidism is an uncommon cause of ascites. Here we describe a case of a 75 year-old female patient with spontaneous bacterial peritonitis and subclinical hypothyroidism that resolved with thyroid replacement and antibiotic therapy respectively. Ascitic fluid analysis revealed a gram-positive bacterium on gram staining. A review of the literature revealed just one other reported case of myxoedema ascites with concomitant spontaneous bacterial peritonitis and no case has till been reported of spontaneous bacterial peritonitis in subclinical hypothyroidism.
Key Words: Peritonitis; Ascites; subclinical Hypothyroidism

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 SBP.[1] SBP typically presents with abdominal pain, fever and raised inflammatory markers such a C reactive protein and white blood cell count.
The case reported here describes a patient with subclinical hypothyroidism with concomitant SBP. This patient did not have other risk factors for developing ascites such as a previous history of alcohol excess and hepatitis B infection. Moreover investigations also eliminated cirrhosis of the liver, portal hypertension, malignancy, heart failure and other common causes of ascites. Subclinical 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.

Case Report:
A 75 years old woman from Jatog, Himachal Pradesh presented with 8 month history of constipation on and off and a 2 month history of increasing abdominal distension and 5 day history of increasing abdominal pain. She was non smoker and non alcoholic, non diabetic and non hypertensive.
Clinical examination revealed a moderately built well nourished lady. Pulse rate was 60/minute, regular, blood pressure was 120/90 mmHg, and respiratory rate was 18/minute. Cardiopulmonary examination was within normal limits. Abdominal examination revealed free fluid in the form of shifting dullness, diffuse tenderness and decreased bowel sounds. The white blood cell count was 7590/cumm, haemoglobin 11.2gm%, MCV 80.6fl, and platelets 178000/cumm. The C-reactive protein was positive. Renal function tests were within normal limits. The alkaline phosphatase level was 127U/L, the total bilirubin was 5umol/L, the alanine amino-transferase was 14U/L and the aspartate amino transferase was 30 IU/L. The total serum protein level was 57g/L, the albumin was 30g/L and the international normalised ratio (INR) was 1.0. Thyroid function studies revealed a thyroid stimulating hormone (TSH) of 10.92mIU/L and a T4 of 7.70micgram/L and T3 of 0.66ng/mL. Amylase was 39 IU/L. ANA was negative by latex agglutination method. Chest X - ray was within normal limits and ECG revealed sinus bradycardia.
Ultrasound of the abdomen and pelvis revealed marked ascites but there was no suggestion of liver cirrhosis or gyanecological pathology. Computer tomography of the abdomen and pelvis revealed a large volume of ascites with minimal bilateral pleural effusion but no feature of liver cirrhosis. UGI endoscopy was also normal. Echocardiogram revealed good systolic function.
A diagnostic and therapeutic ascitic drain was conducted. The ascitic fluid had a total protein level of 32g/L. The white cell count was 6400, 90% of which were polymorphs. ADA levels 9.1U/L. Gram staining of the fluid revealed gram positive diplococci. No malignant cell was found in three consecutive samples that were sent. The ascitic fluid culture

grew streptococci. It was treated with a 5 day course of pipericillin and tazobactam and levothyroxine was also started. Patient improved and on discharge only minimal ascites was there on ultrasonography.

On follow after 2 months, patient had no free fluid on USG and her TSH was 3.12mIU/L.

Discussion:

Hypothyroidism is a common clinical condition. 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 mechanism by which a patient with hypothyroidism 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 is 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 search 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 myxodemea 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 have also indicated that 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]

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