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HYPOTHYROIDISM WITH ASCITES

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Abstract:

Primary hypothyroidism is a common clinical condition. Ascites caused by hypothyroidism is rare. So its diagnosis is often delayed and patients frequently receive unnecessary procedures such as liver biopsies and exploratory laparotomies. We report a male person of 58 years with hypothyroidism with ascites who responded well with thyroid hormone replacement therapy with complete resolution of ascites. Analyses of ascites from patients in this condition usually shows exudative ascites with high protein (>2.5 g/dL) and SAAG < 1.1 gm/dl. High index of suspicion is required to reach at such diagnosis. Though it is a rare but prognosis is excellent with replacement therapy.

Key words: *Ascites, Hypothyroidism.*

Introduction:

The development of ascites in hypothyroid patients is a well-known, but rare occurence. This condition was first described in 1886 and occurs in <4 % of cases.[1] In a review of eight cases of ascites due to hypothyroidism, the median duration of ascites before a diagnosis of hypothyroidism was made was approximately 8 months. Many of these patients had undergone multiple paracentesis, liver biopsy, and exploratory laparotomies.[2] Therefore,when any patient presents with ascites of uncertain etiology, hypothyroidism should be considered as a differential diagnosis.

Here we report a male person of 58 years with hypothyroidism with ascites who responded well with thyroid hormone replacement therapy with complete resolution of ascites.

Case Report:

A 58 years old male, by profession a peasant, presented to us with the complaints of gradual distension of abdomen and weakness for 3 months. He also had loss of appetite, constipation, decreased libido and cold intolerance .Previously he had been diagnosed as a case of decompensated chronic liver disease and was on treatment without improvement.

There was no history of significant weight loss, fever, yellow coloration of eyes or urine. He gave no history of cough, chest discomfort, and shortness of breath on exertion or in recumbent posture. He had no difficulty or pain during swallowing or in rising from sitting position. His urine volume was adequate. He gave no history of headache, vomiting, convulsions or loss of consciousness. He is non diabetic, normotensive,non asthmatic, non alcoholic and there was no history of TB or TB contact. On examination, he was mildly anaemic with mild ankle edema, coarse skin, sparse body hair, and loss of lateral third of both eye brows. Alimentary system reveled ascites with no other abnormality. Nervous system examination revealed delayed relaxation of ankle jerk.

On investigation, his Haemoglobin was 9.2 g/dl and erythrocyte sedimentation rate was 50mm in 1st hr. Peripheral blood film showed macrocytic anaemia. ECG showed sinus bradycardia. Ultrasonogram of whole abdomen revealed moderate ascites with normal liver,spleen, pancrease, kidneys . Ascitic fluid study showed 20/ cu mm lymphocyte, 3.2 gm/dl protein and 60 mg/dl sugar. SAAG was 0.9 gm/dl. Urine routine examination, 24 hours UTP, fasting blood sugar, serum creatinine, s.bilirubine, SGPT, SGOT, alkaline phosphatase, serum albumin, A:G ratio, Pprothrombin time, fasting lipid profile, stool for OBT, s.electrolytes, FT_3 0.3 nmol/L, FT_4 2.3 nmol/L, TSH 95mIU/L, X-ray chest P/A view, echocardiography, endoscopy of upper GIT, colonoscopy, barium follow through X-ray of small gut were normal. HBsAg, Anti-HCV and MT test were negative.

On basis of the above findings we drew the conclusion this is a case of hypothyroidism.Then we performed USG of thyroid gland, thyroid scan and Thyro peroxidase Ab. All were found normal.

Thyroid hormone replacement therapy was started with gradually increasing doses of levothyroxine, from 25 microgram to 150 microgram daily. On 8 weeks later he had a good general wellbeing and was found euthyroid with abatement of ascites. So the maintenance dose of levothyroxin (150 microgram daily) has been continued.

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Table-I

Characteristics of reported patients with myxedema ascites

	Number of patients	Mean	Ranges	Remarks
Ascitic protein (gm/dl)	49	3.9	1.8—5.1	Forty-eight patients(g/dL) (98%) showed ascites protein levels >2.5 g/dL
SAAG(gm/dl)	11	1.5	0.8—2.3	Because of the smallnumber of patients,the characteristics were unclear
Ascites WBC count (per micro L)	48	60	10—4000	Predominance of(per L) lymphocytes(mean 81%)
Duration of ascites	51	8 months		1 year to 8 years
Response to treatment	51			Regression of ascites

SAAG, serum-to-ascites albumin gradient; WBC, white blood cell.

Discussion:

Hypothyroidism is a relatively rare cause of ascites. This is a potentially curable condition with thyroid hormone replacement therapy unlike chronic liver and renal disease where little can be offered to the patient. In evaluation of new cases of ascites, ascitic fluid study and SAAG may cut short the spectrum of different expensive and invasive investigations. SAAG demonstrates the transudative and exudative ascites. SAAG more than 11 gm/l (1.1 gm/dl) indicates transudative ascites. [3] Chronic liver disease, portal hypertension, congestive cardiac failure, chronic kidney disease, nephrotic syndrome, malnutrition and hypoalbuminaemia of any cause, all produce transudative ascites. Whereas SAAG less than 11 gm/l (1.1 gm/dl) indicates exudative ascites.[3] Of the various causes, peritoneal malignancies, tuberculous peritonitis, pyogenic peritonitis and pancreatic ascites can all lead to high-protein ascites. Here hypothyroidism ascites is rare possibility but it should be kept in mind during evaluation of a exudative ascites There has been a suggestion that the SAAG may exceed 1.1 in patients with myxedema ascites, based on a review of eight patients[1]. Because so few cases have been studied and portal hypertension or heart failure do not seem to be the mechanisms causing ascites in patients with myxedema, we cannot conclude that a high SAAG is a typical feature in this disease[4]. Moreover, the patient reported here showed a low SAAG. Portal hypertension secondary to liver cirrhosis is the leading cause of ascites (more than 80% of cases) and peritoneal involvement in patients with malignant diseases is the second at about 10% [5]. In case of malignancy demonstration of malignant cell and /or peritoneal biopsy should be considered. [6] If there is any evidence of portal hypertension clinically with transudative ascitic fluid, ultrasonography and endoscopy of upper GIT should be done to detect portal hypertension, splenomegaly and oesophagial varices or congestive gastropathy respectively. As in this case ascitic fluid was exudative we performed thyroid function tests which proved decisive.

A review of the literature turned up 51 well-documented cases of myxedema ascites (Table 1) [7, 8, 9-19]. A very consistent finding was the high total protein level (>2.5 g/ dL) [6]. Total protein levels exceeded 2.5 g/dL in almost all cases, with a mean of 3.9 g/dL. The mean SAAG was 1.5 g/ dL with a range of 0.8-2.3 g/dL. White blood cell counts were rather low, usually with a predominance of lymphocytes; the mean white blood cell count was 60/ L with a mean of 81% lymphocytes. In our patient, white blood cell count was 81/microL and lymphocyte proportion, 84%.

The mechanism of ascites 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 because of abnormal capillary permeability and the lack of a compensatory increase in lymph flow and protein return rate [20]. The second hypothesis is that hyaluronic acid accumulates in the skin and produces edema by a 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 that prevent the lymphatic drainage of extravasated albumin [21].

The cause of pseudo-obstruction is also poorly understood. The most commonly proposed mechanism is that hypothyroidism causes an alteration in impulse transmission at myoneural junctions by deposition of myxedematous infiltrate between the muscular fibers and plexus. Wells et al [22] proposed that intestinal poor function was related to autonomic polyneuropathy.

In conclusion hypothyroidism with ascites is rare but easy to treat. Treatment with thyroid hormone replacement therapy leads to complete regression of the ascites. High index of clinical suspicion is required to diagnose such cases. On rutine evaluation of ascites investigations should be done to find the common causes such as chronic liver disease, peritoneal malignancy and infection, congestive heart failure, chronic kidney disease, pancreatic ascites and along with thyroid function tests specially on patients with exudative ascites.

References:

1. de Castro F, Bonacini M, Walden JM, Schubert TT. Myxedema ascites. Report of two cases and review of the literature. J Clin Gastroenterol 1991; 13(4): 411-4.
2. Gastroenterology Grand Rounds. Constipation and ascites: Diagnostic. 2003 [Accessed 2009 May 8]. Available from: URL: http://www.bcm.edu/gastro/DDC/grandrounds/BCM/12-18-03/D-LAYOUT.HTM
3. Boon NA, Colledge NR, Walker BR, Hunter JAA. Davidson's principles and practice of Medicine. 20th ed. New Delhi: Elsevier; 2006. p. 948.
4. Otero J, Landeira G, Corino M, Tamashiro A, Fassio E. Ascites due to hypothyroidism in a patient with alcoholic cirrhosis. Acta Gastroenterol Latinoam. 2001 ;31(2):77-81.
5. Runyon BA. Care of patients with ascites. N Engl J Med. 1994; 330(5): 337-42.
6. Myxedema Ascites: Case Report and Literature Review. J Korean Med Sci 2006; 21: 761-4
7. McDonough CH, Lee L, de Beur SJ, Arai S, Vogelsang GB. Myxedemaascites in the posttransplant setting: case report. Am J Hematol. 2002; 71(3): 216-8.
8. De Feudis L, Scudieri M, Orlando D, Traisci G. Ascites as preeminent manifestation of primary hypothyroidism: clinical case. Ann Ital Med Int. 1999;14(4): 294-7
9. Chiprut RO, Knudsen KB, Liebermann TR, Dyck WP. Myxedema ascites. Am J Dig Dis. 1976; 21(9): 807-8.
10. Hazard J, Merot J, Klotz A, Potel M. Ascites revealing thyroid deficiency.Ann Med Interne 1969; 120(11): 755-9.
11. Kinney EL. Myxedema ascites. Am Fam Physician. 1987; 36(2): 134.
12. Kocen RS, Atkinson M. Ascites in hypothyroidism. Lancet. 1963; 91(7280): 527-30.
13. Leung FW, Nortman DF, Shinaberger JH. Myxedema ascites in a patient undergoing chronic hemodyalisis. Dyalisis Transplant 1982; 11: 708-9.
14. Madenberg F, Byfield GV, Baker LA. Occurrence of ascites in myxedema. Arch Intern Med 1954; 93: 787-95.
15. Desrame J, Mathurin P, Rozov R, Sabate JM, Poynard T, Opolon P, Denis J. Isolated ascites revealing a hypothyroidism: study of 2 cases. Gastroenterol Clin Biol 1998; 22: 732-5.
16. Skinner J. Mixedema with thyroid antibodies presenting with ascites. Proc R Soc Med 1962; 55: 997.
17. Turner JA, Rapoport J. Mixedema ascites. Postgrad Med J 1977; 53: 343-4.
18. Von Knorring J, Friman C. Mechanism of myxedema formation. N Engl J Med 1980; 302: 469.
19. Alberti LE, Lopez-Gomez A, Alberti-Flor JJ. Spontaneous bacterial peritonitis in a patient with myxedema ascites. Digestion 2003; 68: 91-3.
20. Parving HH, Hansen JM, Nielsen SL, Rossing N, Munck O, Lassen NA. Mechanisms of edema formation in myxedema increased protein extravasation and relatively slow lymphatic drainage. N Engl J Med 1979; 301: 460-5.
21. Bonvalet JP, David R, Baglin A, Hatt PY. Myxedema with inappropriate antidiuresis and hyperaldosteronism. Ann Med Interne 1970; 121: 949-55.
22. Wells I, Smith B, Hinton M. Acute Ileus in Myxedema. BMJ 1977; 211-212.