AN UNUSUAL CASE OF HYPOALBUMINEMIA

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ABSTRACT

This report describes a female patient of 45 years with progressive pallor and malaise. Clinically patient was thin built, jaundiced, with palmar erythema and splenomegaly. Her hemoglobin was 4.2g/dL, mean corpuscular volume was 69.2f/ land serum albumin was critically low i.e. 12g/ L. Hypoalbuminemia is a feature of decompensated cirrhosis of liver however such a low level of albumin without peripheral signs of edema has not been reported to the best of the authors' knowledge. It is hereby reported as the first case of its kind.

Keywords: Albumin, Cirrhosis, Edema.

INTRODUCTION

Albumin is a major determinant of colloids osmotic pressure. Most of the osmotically active particle in the blood stream (such as sodium, urea, glucose) are relatively small and pass freely between vascular and interstitial compartments. Albumin in contrast is too large to pass freely through vascular endothelium, and remains within the bloodstream. It has been used for a long period of time as a resuscitative fluid in critically ill patients. It is an antioxidant agent and has multiple ligand binding sites for free radical trapping[1]. Hypoalbuminemia has three major potential causes, i.e. hepatic failure, protein losing enteropathy and nephropathy. Here we are sharing a case of Hepatitis C Virus (HCV) related cirrhosis with critically low serum albumin i.e. 12g/L without ascites or pedal edema.

CASE REPORT

A 45 year old lady, a case of HCV related cirrhosis admitted with complaints of pallor and malaise for 10 months and weight loss for 6 months. Clinically, a lady of thin built and height, appeared ill looking, jaundiced with blood pressure of 110/70 mmHg, pulse 80/min regular non collapsing, respiratory rate 18 breaths/min, temp 98.8°F, oxygen saturation was 94% while breathing ambient air. Lymph nodes and thyroid were not palpable, jugular venous pressure was not raised; there was no pedal or sacral edema. Bone tenderness could not be appreciated. There was angular chelosis, splenomegaly was found upto 6cm below left subcostal margin along its axis. Chest was clear to auscultation, first and second heart sounds were clearly audible and there was no murmur. Glassgow coma scale (GCS) was 15/15, cranial nerves were intact, power was 5/5 in all limbs. She had bilateral diminished ankle jerks however sensations were intact.

Laboratory analysis revealed hemoglobin 4.2g/dL, mean corpuscular volume 69.2fl, total leucocyte count 3.4x10*9/L, platelet count 281 x 10*9/l. RBC morphology revealed microcytic hypochromic picture, prothrombin time 17/14, partial thromboplastin time 50/34, serum bilirubin 10 umol/l, alanine transaminase (ALT) 23U/L, alkaline phosphatase 296 IU//L, urea 3.9 mmol/L. Anti-extractable nuclear antigen, carcinoembryonic antigen, and alpha fetoprotein levels were unremarkable. Serology for hepatitis B virus was negative. Her ultrasound abdomen revealed coarse echo texture liver with portal vein diameter upto 0.7mm (figure).

The patient was managed with red cell concentrate, iron replacement therapy, proton pump inhibitors, salt free albumin 20%, rafixamin and analgesics. Patient improved remarkably and was discharged on 17th day of

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admission with albumin 24g/L and heamoglobin up to 8.4g/L regular follow up in outdoor clinics. After 6 months follow up her hemoglobin is 9.6gm/dl, and albumin is 20g/L and there was no ascites or pedal edema.

DISCUSSION

Albumin, the major plasma protein, is produced only by the liver. Albumin consists of 585 amino acids, with a molecular weight of 69 kDa. Serum albumin contributes upto 80% of effective plasma oncotic pressure. A decrease in oncotic pressure allows fluid to leak from the interstitial spaces into the peritoneal cavity, producing ascites. It occurs at serum albumin of 20g/L. However this patient did not develop ascites despite such a low albumin. Albumin plays a vital role in the transportation of various molecules; Including bilirubin, free fatty acids, drugs, and hormones[2]. However, chronic hypoalbuminemia alone may be insufficient to induce edema. Experimental and clinical observations indicate that, patients with increased intravascular volume due to primary renal Na+ retention are likely to have glomerular filtration rate less than 50% of normal, and a plasma albumin concentration of greater than 20g/L[3].

The increased probability of poor outcomes such as mortality, prolonged intensive care unit (ICU) and hospital stay in acutely ill patients is associated with hypoalbuminemia. Considering its importance as an outcome predictor, serum albumin level has been added as one of the parameters in the Acute Physiology and Chronic Health Evaluation (APACHE) III score. The association between hypoalbuminemia and poor outcomes has motivated experts in administering exogenous albumin to all critically ill patients, and it is licensed in the United States and other developed countries[4]. Accordingly this patient received salt free albumin which improved her serum albumin upto 24g/L.

Recent studies have highlighted the efficacy of branched-chain amino acids (BCAA) in improving hypoalbuminemia in cirrhotic patients initiating appropriate nutritional interventions, such as supplementation of BCAA, in the early stage of cirrhosis may improve the prognosis[5]. For every 10g/dl fall in serum albumin, mortality increases by 67% and morbidity by 89%[6]. Another important cause of hypoalbuminemia is acute and chronic inflammatory responses, as it is negative acute phase reactant. There is strong evidence that hypoalbuminemia is associated with worse outcomes; however, evidence justifying albumin supplementation is lacking, which is in contrast to ref[3-6]. Although severe adverse events are frequently reported with administration of human serum albumin however this patient received albumin uneventfully[7].

Malnutrition is common in cirrhotic patients. It has been observed that upto 43% patients

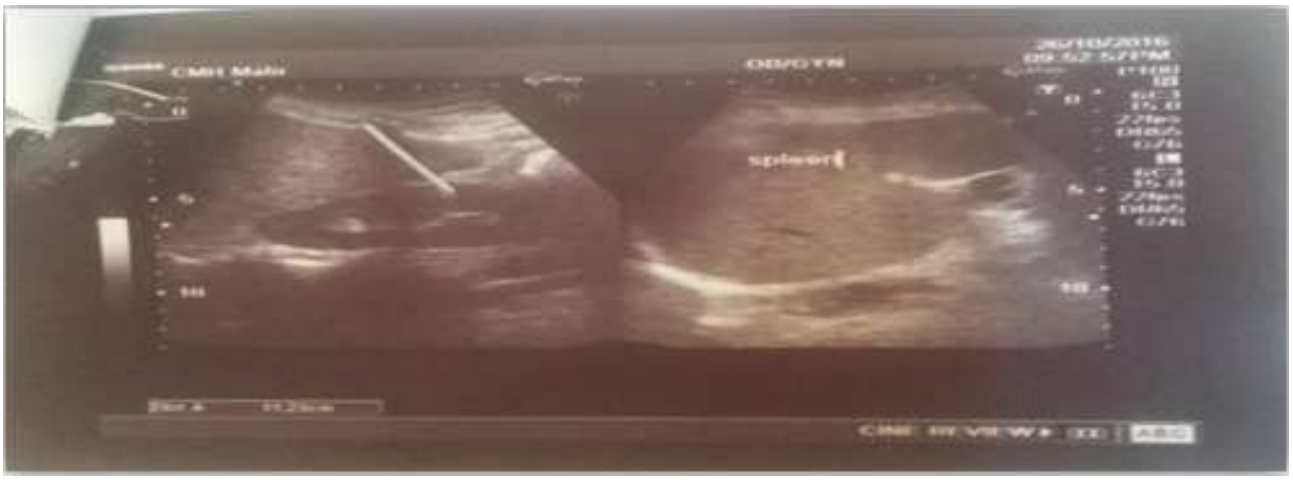

Figure: Ultra sound abdomen of the patient revealed mild splenomegaly with minimal ascites.

hospitalized with liver cirrhosis do not undergo formal dietetic assessment and intervention. While patients with hypoalbuminemia and ascites were likely to receive formal dietetic input, however neither of these factors was predictive of nutritional status[8]. This patient had reasonable appetite and food intake was appropriate, thereby ruling out malnutrition as a cause of hypoalbuminemia.

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

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