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Protein-Losing Enteropathy in an Operated Gastric Cancer Case

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Introduction

Of all cancers, Gastric Cancer (GC) is the third leading cause of cancer mortality and the fifth prevalent malign tumor to be diagnosed (except skin cancers) [1]. Adenocarcinomas, which develop from the glands of the stomach's mucosaor superficial layer, account for themajority of stomach cancer cases [2]. Environmental and genetic variables both play a part in the complex etiology of GC. While some of these risk factors, like age and sex, cannot be changed, others, like smoking and H. pylori infection, may be changed [2]. Today's treatments have made this disease more manageable.Surgery-treated stage I tumors have a 60% to 80% five year survival rate. However, depending on the data set, individuals with stage III cancers who have surgery have a five year survival rangingfrom 18% to 50% [3].

Protein-Losing Enteropathy (PLE) is an uncommon illness-characterized by excessive protein loss into the gastrointestinal system as a result of mucosal integrity impairment [4]. Symptoms, history, and physical examination are all used to make the diagnosis in the majority of patients. However, PLE can be confirmed if necessary by using functional imaging or by measuring **Alpha-1-Antitrypsin** (A1AT) in a stool sample [4]. In this study, we presented the case of PLE as a cause of ascites developing years later in an operated GC case.

Case Presentation

A seventy-four-year-old male patient who had been complaining of nausea, vomiting, and increased difficulty swallowing liquids for a year was admitted to Kocaeli City Hospital, Medical Oncology clinic. Fourteen years ago, it was learned that he had undergone subtotal gastrectomy and Roux-en Y bypass surgery for stage 2 gastric cancer and had undergone adjuvant chemoradiotherapy treatment. On physical examination, bilateral pretibial edema was present. Laboratory tests showed total protein: 50.7 g/L, albumin: 21.3 g/L, sedimentation rate: 46 mm/h and c-reactive protein: 110.53 mg/L, with no additional findings. In contrast-enhanced abdominal computed tomography; an increase in concentric wall thickness in the anastomosis line, diffuse free fluid and edematous appearance in the mesentery were observed (**Figure 1**).

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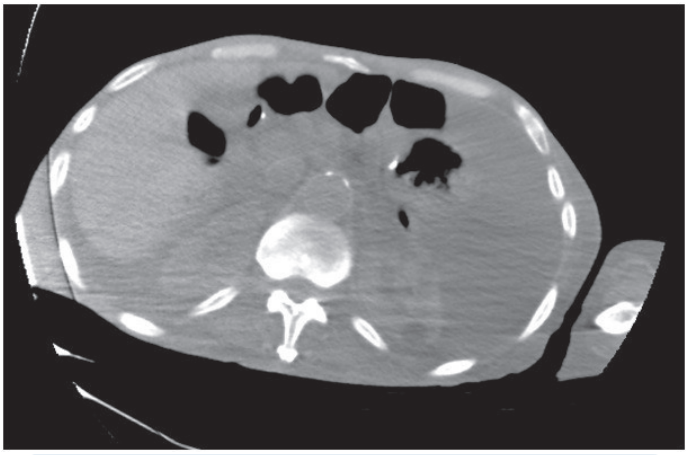

Figure 1: An increase in concentric wall thickness in the anastomosis line, diffuse free fluid and edematous appearance in the mesentery were observed in the contrast-enhanced abdominal computed tomography section.

Reactive mesothelial cells were seen in diagnostic paracentesis cytology taken three times. Serum acid albumin gradient was 1.02. In esophagogastroscopy, the entire gastric and anastomosis line was observed to be hyperemic and edematous, the relative stenosis area could not be passed at a distance of about 8-10 cm in the jejunum. A dilatation was performed on this region endoscopically (**Figures 2 & 3**). Multiple biopsies carried out from the anastomotic line revealed no evidence in favor of malignancy.

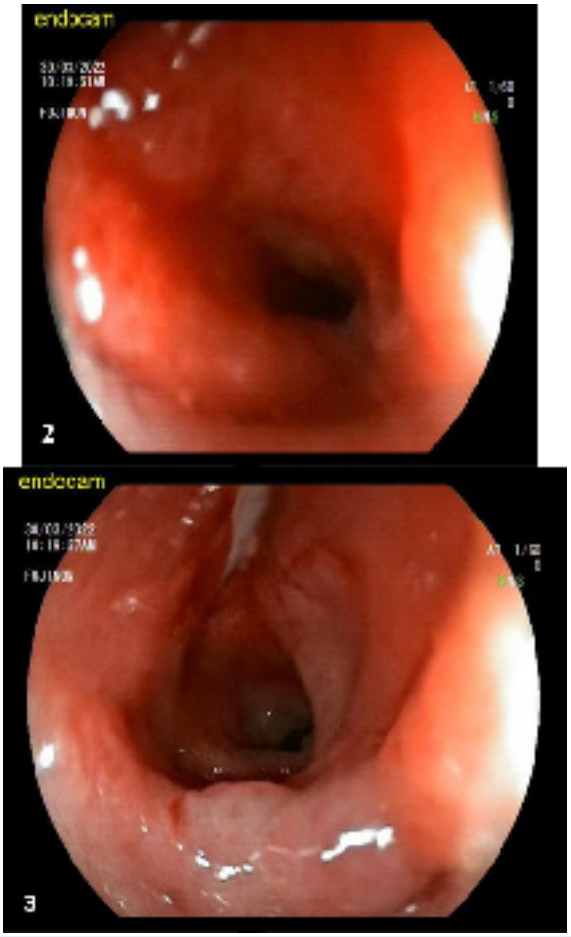

Figure 2&3: Endoscopic stenosis and edema.

A1AT in the stool was determined as 3.1 mg/g. In the patient with nonportal ascites and no evidence of malignancy, hypoalbuminemia and secondary ascites due to PLE were considered because other causes were excluded and A1AT was found to be high. The patient was started on ceftriaxone 2x1 gr with albumin replacement. Multivitamin-mineral supplementation was given with a diet rich in protein and medium-chain fatty acids. After 1 week of antibiotic therapy, 8 mg of dexamethasone was administered intravenously for 10 days to reduce inflammation in the mesentery. The patient whose complaints regressed, who did not need paracentesis and whose albumin level increased to 3 g/L was discharged.

Discussion

PLE is a disease that can develop in many different diseases due to mucosal inflammation and impaired lymphatic circulation [5]. The pathophysiological mechanism of PLE includes erosive/ulcerative mucosal diseases, non-erosive mucosal diseases, and disorders where lymphatic/interstitial pressure increases [6]. Particularly in terms of its clinical appearance, PLE is a complex disease. PLE frequently shows signs of generalized edema and hypoproteinemia. Other, less common signs and symptoms have been reported in the literature, including malnutrition, pleural and pericardial effusion, macular edema with reversible blindness, anasarca or unilateral edema in situations of lymphangiectasia [7].

Firstly, it is important to exclude hepatic, renal, and cardiac causes of decreased protein in the blood. Simple, routine diagnostic procedures (such as abdominal ultrasound and echocardiographia or blood and urine tests) should be carried out [8]. When diagnosing PLE, imaging studies are used to evaluate the gastrointestinal mucosa, followed by endoscopic procedures to directly evaluate the intestinal mucosa, allowing the exact location of protein loss throughout the gastrointestinal system to be determined [8]. Measurement of intestinal clearance of A1AT is the most typical test used to diagnose PLE. A1AT normally excretes less than 13 ml per 24 hours, and a clearance of more than 27 ml per day implies excessive protein loss from the gastrointestinal tract [9]. The gold standard test is 51Cr-labeled albumin clearance. Although they have a high level of sensitivity, this test is expensive, difficult to obtain, and rarely used [9].

An excessively high protein diet is advised for all PLE patients. Age-dependent protein requirements are calculated as 0.66 g/kg/day for adults, but this amount can be increased to 1.5-3 g/kg/day to ensure positive protein balance in the PLE [10]. It is recommended to follow a low-fat diet enhanced with Medium-Chain Triglycerides (MCTs) [10]. Octreotide therapy might be used in PLE due to primary lymphangiectasis. Besides, someresearches reported that steroid therapy is beneficial in secondary lymphangiectasia or autoimmunity and inflammation-induced PLE [5] [11]. However, it should not be forgotten that the real success of the treatment depends on the treatment of the underlying chronic disease.

Conclusions

PLE is a disease in which protein excretion is increased with feces and is thought to be due to intestinal lymphangiectasia, mucosal inflammation and epithelial damage, the pathophysiology of which is not fully elucidated. The basis of treatment is the treatment of the underlying pathology. In addition, it is important to regulate the diet. In our case, inflammation starting from the anastomosis line and extending to the mesentery after gastric operation led to epithelial damage and protein loss. When edema and ascites are detected in gastrointestinal cancer cases with a history of surgery, patients should be investigated for PLE.

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