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Case Report

# A Finding That Is Not Clear What Will Emerge When You See It: Hypoalbuminemia

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#### ABSTRACT

Malnutrition can cause hypoalbuminemia. Gastrointestinal malignancies are among the causes of protein-losing enteropathy. Herein, we presented a case of protein-losing enteropathy due to gastrointestinal malignancy presenting with symptoms such as hypoalbuminemia and oedema in the hands and legs.

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**Keywords:** Malnutrition, hypoalbuminemia, oedema, protein-losing enteropathy, gastrointestinal malignancy.

#### Introduction

Hypoalbuminemia can occur for many different reasons, such as chronic kidney damage, nephrotic syndrome, chronic liver diseases, malnutrition, dermatological losses. Gastroscopy, colonoscopy, and in some cases, enteroscopy can be used to demonstrate that protein loss originates from the gastrointestinal system. Protein-losing enteropathy can be caused by inflammatory bowel disease due to mucosal surface damage, gastrointestinal malignancies, lymphoma, erosive gastritis, multiple ulcerations, primary intestinal lymphangiectasia due to lymphatic obstruction, and many multisystemic causes.1 Gastrointestinal malignancy, which generally presents with bleeding, obstruction, and changes in defecation habits, may rarely present with symptoms related to hypoalbuminemia.<sup>2</sup> Herein, we presented a case of

protein-losing enteropathy due to gastrointestinal malignancy presenting with symptoms such as hypoalbuminemia and oedema in the hands and legs.

### Case Report

A 67-year-old male patient with no known history of chronic disease had come to the emergency department with an increase in swelling in both legs that had increased, soft, pitting oedema for the last

week. He applied with the complaint of swelling in both hands and legs. In laboratory examinations of the patient, albumin level was 19.7 g/L, hemoglobin 10 g/dL, MCV 82 fL, leukocyte 4.5 K/uL, platelet 220 K/uL, creatinine



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0.9 mg/dL, AST 12 IU/L, ALT 15 IU/L, and total protein 4.25 g/L. The patient had a history of colonoscopy due to dyspepsia two months ago, colonoscopy was found to be normal, and pangastritis was present in the gastroscopy. The patient was admitted to our internal medicine clinic for a hypoalbuminemia examination. The patient's echocardiography was normal (EF: 60, no valve pathology, right insufficiency and diastolic dysfunction). Bilaterally leg lower extremity venous doppler ultrasonography for oedema of both legs showed acute deep vein thrombosis in both legs. We started low molecular weight heparin at the treatment dose. Abdominal ultrasonography and abdominal tomography were unremarkable. Liver function tests requested for the possible liver disease were normal ranges. Albumin loss was not dependent on renal causes, but urea and creatinine levels were normal (urea: 32 mg/dL, creatinine 0.73 mg/dL), urinary ultrasonography was unremarkable, and 24-hour urine protein level was 0.03 mg/day.

There were no malnutrition findings in our research on malnutrition, which may be the cause of hypoalbuminemia. In evaluating the patient's malnutrition, the Nutritional Risk Screening had a low risk (as 0) in 2002. In the gastrointestinal system screening that the patient's current protein loss may be from the gastrointestinal tract, contrast-enhanced abdominal tomography showed diffuse asymmetrical wall thickness increase in a segment of approximately 12 cm extending from the hepatic flexure to the midright half of the transverse colon. Then the patient underwent colonoscopy. Colonoscopy revealed an ulcerovegetant mass in the transverse colon that almost completely occluded the lumen (Picture 1). The biopsy was taken from the patient resulted

in colon adenocarcinoma. Since there was no distant metastasis in the PET-CT taken later, we transferred the patient to the general surgery service for operation.

#### Discussion

Hypoalbuminemia is an essential clinical condition that needs to be examined from a detective perspective. In this case, who underwent colonoscopy two months ago, systematically reviewing the causes of hypoalbuminemia and excluding other reasons, and advocating the idea that the case was of gastrointestinal origin, led to the renewal of the colon screening the patient and the early detection of malignancy. Although there was another aetiology such as deep vein thrombosis that could cause peripheral oedema, examination of hypoalbuminemia also helped for the accurate diagnosis of malignancy in the patient. The pathophysiology of protein-losing enteropathy is the loss of plasma proteins entering the gastrointestinal lumen. Protein-losing enteropathy may result from lymphatic obstruction or mucosal damage. It can develop as primary or secondary.3 In acute situations, albumin infusion can increase the oncotic plasma pressure. In some cases, the use of corticosteroids or heparin may be required.

In conclusion, although there are gastrointestinal and cardiac diseases in the aetiology of protein-losing enteropathy, it can be seen as a rare complication of many diseases. Clinically, hypoalbuminemia, low total protein, diarrhoea, and weight loss can be seen. The most important approach to consider in a case of suspected protein-losing enteropathy is the exclusion of hepatic and renal causes.<sup>4</sup> Although its prognosis is not known precisely, it is crucial to correct the underlying pathology.<sup>5</sup>









**Picture 1.** Colonoscopy revealed an ulcerovegetant mass in the transverse colon that almost completely occluded the lumen.

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#### Conflict of interest

The authors declared that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

#### Authors' Contribution

Study Conception: ES; Study Design: ES; Supervision: MVK; Materials: MVK; Data Collection and/or Processing: MVK; Statistical Analysis and/or Data Interpretation: EI; Literature Review: EI; Manuscript Preparation: EI; Critical Review: ES.

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