Successful Treatment of Protein-Losing Enteropathy After Superior Mesenteric Artery Occlusion without Surgery

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Conflict of interest: None declared

Patient: Male, 77-year-old
Final Diagnosis: Protein-losing enteropathy • superior mesenteric artery occlusion
Symptoms: Abdominal pain • diarrhea
Medication: —
Clinical Procedure: —
Specialty: Surgery

Objective: Management of emergency care

Background: Protein-losing enteropathy as a complication of superior mesenteric artery occlusion is extremely rare and severe, and sometimes requires intestinal resection. However, the ideal treatment strategy has not yet been determined.

Case Report: A 77-year-old man with underlying hypertension and diabetes was admitted to the Emergency Department with acute abdominal pain after eating. Contrast-enhanced computed tomography revealed complete occlusion of the superior mesenteric artery with thrombosis, and superior mesenteric artery occlusion was diagnosed. It was successfully treated with interventional therapy, followed by continuous intra-arterial prostaglandin E1 infusion and continuous intravenous heparin infusion. However, the patient developed hypoproteinemia and diarrhea about 10 days after the interventional therapy. Colonoscopy and X-ray studies did not reveal any abnormal findings; however, technetium-99m-labeled human serum albumin scintigraphy indicated protein-losing enteropathy. With total parenteral nutrition and protein-rich oral nutrition, with protein intake at twice the amount in a standard diet, serum albumin improved from 15 g/L to 32 g/L after treatment. Additionally, we administered diuretics to avoiding edema related to the hypoproteinemia. The patient recovered from the hypoproteinemia and diarrhea without complications.

Conclusions: Protein-losing enteropathy is an extremely rare but critical complication of superior mesenteric artery occlusion. Treating the underlying pathology is the mainstay of protein-losing enteropathy and dietary modifications also play a critical role. Our patient was successfully treated with strict nutritional therapy, combined oral protein-rich nutrition and total parenteral nutrition, which avoided surgery.

Keywords: Congenital Disorder of Glycosylation Type 1B • Mesenteric Ischemia • Technetium

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/931114
Background

Protein-losing enteropathy (PLE) is a condition in which excessive loss of protein occurs through the gastrointestinal tract due to various etiologies, resulting in hypoproteinemia [1]. PLE occurs rarely as a complication of superior mesenteric artery (SMA) occlusion (SMAO) [2]. In Japan, all known cases of PLE after SMAO have required bowel resection [3,4]. However, the ideal treatment strategy has not yet been determined. Here, we present a case of PLE after SMAO, which was treated successfully without surgical intervention.

Case Report

A 77-year-old man who presented with a 10-h history of acute abdominal pain after eating was transferred to our Emergency Department from another hospital. He had a history of hypertension and diabetes but no other diseases, including heart disease. Upon admission, his body temperature was 36.6°C, blood pressure was 196/103 mmHg, and his heart rate was 90 beats/min. He had upper abdominal pain but no rebound tenderness. Blood laboratory testing result revealed total protein of 78 g/L, serum albumin of 43 g/L, white blood cell count of 9.35×10⁹/L, and lactate of 3.3 mmol/L. Contrast-enhanced computed tomography revealed complete occlusion of the SMA with thrombosis at the root and middle SMA; however, intestinal enhancement was maintained (Figure 1). We performed interventional therapy and initiated continuous intra-arterial prostaglandin E1 (PGE1) infusion and continuous intravenous

Figure 1. Contrast-enhanced computed tomography revealed that the root of the superior mesenteric artery (SMA) was completely occluded with a thrombus (arrow).

Figure 2. (A) Before interventional therapy, the superior mesenteric artery (SMA) was completely occluded (arrow). (B) After the treatment, the main thrombus in the root of the SMA was removed, but the SMA branch was still occluded (arrow). (C) After prostaglandin E1 and heparin therapy, the blood flow to the SMA was completely restored.
heparin infusion after the interventional therapy, which resulted in successfully restored blood flow (Figure 2).

Ten days after admission, the patient developed severe diarrhea and hypoproteinemia, and his serum albumin and total protein concentrations decreased to 15 g/L and 37 g/L, respectively, even with sufficient total parenteral nutrition (TPN) and oral nutrition. Technetium-99m-labeled human serum albumin scintigraphy revealed protein leakage into the intestinal wall and a thickened small intestine consistent with edema, on computed tomography (Figure 3). There were no other abnormal findings from colonoscopy and X-ray studies. We diagnosed PLE and continued the patient on passive TPN and oral nutritional treatment, combining a standard diet with an elemental

Figure 3. (A) Technetium-99m-labeled human serum albumin scintigraphy revealed protein leakage through the small intestinal wall. (B) Computed tomography showed that technetium-99m accumulation corresponded to edematous intestine.

Figure 4. The patient’s clinical course and changes in total protein (TP) and serum albumin (Alb) concentrations. PGE1 – prostaglandin E1; TPN – total parenteral nutrition.
diet, while carefully monitoring his serum albumin concentration. We maintained protein intake at twice the amount in a standard diet. Moreover, we administered diuretics and controlled the water balance to avoid edema related to the hypoproteinemia. Serum albumin improved 4 weeks after treatment, and the diarrhea resolved. He was discharged from our hospital 33 days after treatment, with a serum albumin concentration of 22 g/L and a total protein concentration of 54 g/L. He experienced no further complications or symptoms after discharge. When the patient returned to the outpatient clinic 48 days after treatment, his serum albumin concentration had improved to 32 g/L (Figure 4).

Discussion

PLE is a relatively rare condition characterized by loss of protein through the intestines, which causes hypoalbuminemia, hypoproteinemia, edema, and diarrhea. PLE is diagnosed by intestinal clearance of alpha 1-antitrypsin and 51Cr-labeled albumin clearance, and with scintigraphy using technetium-99m-labeled human serum albumin scintigraphy, which has high sensitivity [5].

The etiology of PLE can be divided into 3 main groups, primary erosive or ulcerative gastrointestinal disease, nonerosive or nonulcerative gastrointestinal disease, and disorders causing increased interstitial pressure or lymphatic obstruction [1,6].

In our case, because of the interventional treatment, the small intestinal blood flow was maintained, and ischemia was limited to the mucosa. As a result, the mucosal epithelium desquamated, causing continued protein loss.

Treating the underlying pathology is the mainstay of PLE treatment, and dietary modifications also play a critical role. A protein-rich diet (2-3 g/kg/d), high in medium-chain triglycerides and low in fat is considered the best diet. Additionally, diuretics are an option to treat the symptoms of fluid overload, and surgical resection is sometimes needed [1,7].

PLE as a complication of SMAO is extremely rare, and almost all cases required surgery because of severe hypoproteinemia [3,4]. However, there are problems with surgery, such as short-bowel syndrome and difficulty determining the range of bowel resection because PLE cannot be visualized intraoperatively.

We successfully managed SMAO and PLE in our patient without surgical intervention. First, we treated the diminished blood flow using interventional therapy and then initiated continuous intra-arterial PGE1 and continuous intravenous heparin, thereby treating the underlying pathology. Second, we diagnosed the patient with PLE early. Other reports of cases involving intestinal dissection took longer than 1 month to diagnose; therefore, treatment was delayed [3,4]. Finally, it was important to continue the oral protein-rich nutrition and TPN to allow the mucosal membrane to recover from the protein loss and to control edema using diuretics.

We believed that oral nutrition was the key in our patient, and we used an elemental diet added to the standard diet to maintain protein intake, which we maintained at twice the amount of that in a standard diet. We also continued TPN, considering losses from diarrhea, until the serum albumin concentration increased. Finally, we carefully monitored the patient’s general condition to determine whether the nutritional treatment should be continued.

Conclusions

PLE after SMAO, although extremely rare, often leads to severe illness; however, the condition can be treated by strict nutrition and fluid control, without surgery.

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Conflict of Interest

None.
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