ACCSCIENCE PUBLISHING

Tumor Discovery

CASE REPORT

Advanced gastric and esophageal malignant tumor complicated by chylous ascites: A case report

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Abstract

Chylous ascites caused by lymphatic vessel obstruction due to compression from advanced gastric and esophageal malignant tumors is relatively rare in clinical practice. It is often accompanied by malnutrition, hypoproteinemia, and a poor prognosis. We report a patient with an advanced gastric and esophageal malignant tumor complicated by chylous ascites. Management focused on symptom control and individualized nutritional support, including intravenous medium-chain triglyceride emulsion together with enteral nutritional emulsion administered through a nasoenteric tube, paracentesis for symptomatic relief, and supportive measures. Although the underlying malignancy determined the overall prognosis, and the patient died in May 2023, the interventions provided temporary improvement in symptoms and nutritional status. This case highlights the importance of early recognition of chylous ascites and of prioritizing symptom relief, individualized nutritional strategies, and palliative goals of care in patients with advanced malignancy.

Keywords: Gastric cancer; Esophageal malignant tumors; Chylous ascites; Palliative treatment

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1. Background

Chylous ascites refers to the accumulation of lipid-rich lymphatic fluid in the abdominal cavity, compression of the thoracic duct, abdominal lymphatic vessels, etc., causing them to block or rupture, resulting in lymphatic fluid spilling into the abdominal cavity, which is rare in clinic.[1,2] Under physiological conditions, lymph is drained into the thoracic duct and then enters the systemic circulation through the internal jugular and subclavian veins, where it circulates in the body through active contraction and relaxation of the lymphatic vessels and passive external pressure. Destruction or obstruction of lymphatic vessels in the chest or abdominal cavity due to malignant tumors, cirrhosis, surgery, infection, and trauma are the root causes of chylous abdominal effusion in adults.[3] Its pathological mechanisms include acquired lymphatic channel disruption and lymphatic system fibrosis.[4]

Acquired lymphatic channel interruption is mainly caused by obstruction or destruction of the thoracic duct caused by surgery or trauma, leading to abdominal lymphatic fistula.[5,6] Lymphatic fibrosis is caused by lymphoma and solid organ malignant tumor compression, which leads to lymphatic obstruction of the intestinal trunk into the cisterna chyli, subserous lymphangiectasia, and lymph extravasation into the abdominal cavity. Management focused on symptom control and individualized nutritional support, including intravenous medium-chain triglyceride (MCT) emulsion together with enteral nutritional emulsion administered through a nasoenteric tube, paracentesis for symptomatic relief, and supportive measures. Here, we report a patient with an advanced gastric and esophageal malignant tumor complicated by chylous ascites who was admitted to the Affiliated Hospital of Nanjing University of Chinese Medicine for progressive abdominal symptoms. This case highlights the importance of early recognition of chylous ascites and of prioritizing symptom relief, individualized nutritional strategies, and palliative goals of care in patients with advanced malignancy.

2. Case presentation

The patient was a 63-year-old male, 168 cm tall, weighing 62 kg, with a body mass index of 21.97 kg/m^2. He was admitted to the hospital with a 1-year history of abdominal pain. Gastroscopy revealed lesions consistent with malignancy. Histology and immunophenotype supported poorly differentiated adenocarcinoma of the gastric body and squamous carcinoma of the esophagus. He received a cycle of systemic therapy (tislelizumab 200 mg + paclitaxel 300 mg). He was admitted to our hospital for the next treatment plan. Upon admission, he presented with poor spirits, fatigue, anemia, a yellow complexion, poor appetite, poor sleep, and melena. He had a history of diabetes mellitus and no history of surgery or food or drug allergies. The abdomen was slightly distended, with no visible abdominal varicose veins. It was soft on palpation, with periumbilical tenderness more pronounced on the left side. There was no rebound tenderness. A positive fluid wave was noted. Based on clinical features such as reduced oral intake, marked fatigue, and limited physical activity, the treating team assessed the patient's Eastern Cooperative Oncology Group performance status as Grade 2.

Tumor indexes were examined after admission, including carcinoembryonic antigen (131.29 ng/mL), carbohydrate antigen 12-5 (481.20 U/mL), and carbohydrate antigen 19-9 (1200.00 U/mL). Gastroscopic examination unveiled upper esophageal mass, lower esophageal mucosal lesion, and gastric body mass. Pathologic assessment of the gastric body mass demonstrated inflammatory granulation tissue, with atypical epithelial cell clusters in small pieces of tissue, which, combined with immunophenotype, supported poorly differentiated adenocarcinoma, whereas evaluation of tissue at 35 cm from the incisor revealed poorly differentiated adenocarcinoma (Figure 1).

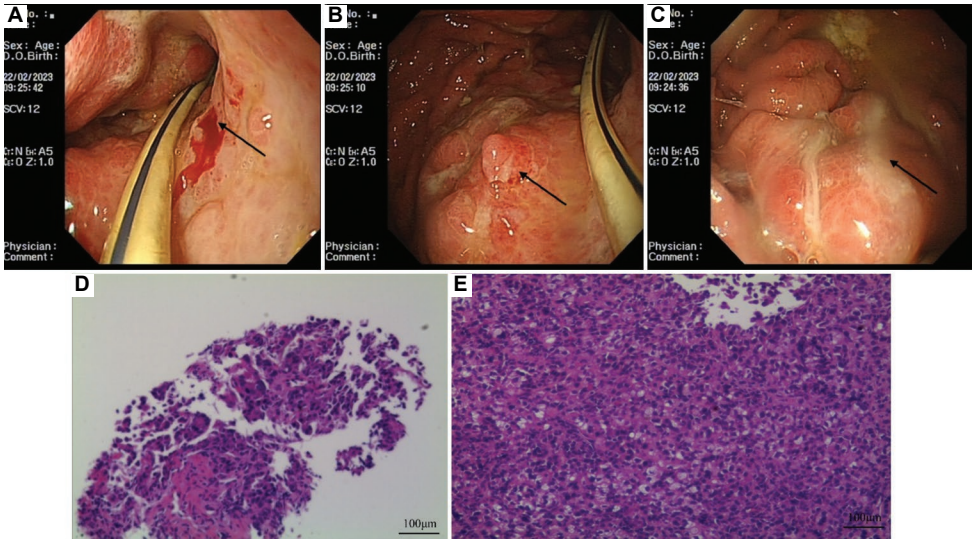

Figure 1. Gastroscopic examination. (A) The esophageal mucosa is not smooth, the new growth of the semicircle is found about 19–23 cm from the incisor, and the surface is broken (arrow). (B) A flat protuberant lesion measuring 0.8 cm in size was seen 35 cm from the incisor. (C) Endoscopic view of the gastric body and gastric angle showing diffuse mucosal hyperemia and swelling. An irregular, raised neoplastic lesion is visible on the posterior wall of the greater curvature, with the upper edge extending to the cardia and the lower edge involving the gastric angle and the junction between the antrum and body. Arrows indicate a centrally ulcerated, friable lesion with contact bleeding. (D) Hematoxylin-eosin staining of a gastric mass specimen showing invasive, poorly differentiated adenocarcinoma with pleomorphic nuclei and prominent nucleoli (magnification: ×100). (E) Hematoxylin-eosin staining of a specimen at 35 cm from incisor showing poorly differentiated adenocarcinoma (magnification: ×100).

Immunohistochemically, the tumors showed positivity for CK-P (+++), CK8/18 (+++), and p40 (+), while CK5/6, CD68, HER2, MSI, and PD-L1 were negative; NTRK fusion testing was not performed.

Chest and abdominal computed tomography (CT) scan revealed multiple enlarged lymph nodes in the abdominal, pelvic, and retroperitoneal regions, suggestive of metastasis (Figure 2). There was evidence of peritoneal and omental metastasis, along with massive ascites in the abdominal and pelvic cavities. The bilateral adrenal glands were enlarged, raising suspicion of metastatic involvement. Abdominal ultrasound confirmed a moderate volume of ascites. Abdominal puncture and drainage were performed. The puncture drainage fluid was milky white, and the drainage volume was about 900 mL within the first 24 h (Figure 3). The drainage fluid was still milky white after 2 days, and the drainage volume was not significantly reduced. The pathological examination of ascites suggested adenocarcinoma cells, which were considered to be of digestive tract origin. Immunohistochemical analysis showed that the tumor cells were positive for MOC-31, CK7, and villin, with focal positivity for CK20 and partial positivity for claudin 18, while calretinin was negative. The chylous test of pleural effusion and ascites was positive.

The patient was managed with fasting, peritoneal effusion drainage through paracentesis, and initiation of nasogastric enteral nutrition along with parenteral nutritional support. Additional treatments included albumin supplementation, electrolyte balance maintenance, and symptomatic therapy aimed at protecting gastric and hepatic function. The general condition of the patient showed improvement after the treatment, leading to his discharge from the hospital with a drainage tube. He received a further cycle of systemic therapy but experienced a progression subsequently and succumbed to end-stage cachexia and severe malnutrition.

3. Discussion

Chylous ascites refers to the accumulation of lipid-rich lymphatic fluid in the abdominal cavity, compression of the thoracic duct, abdominal lymphatic vessels, etc., causing them to block or rupture, resulting in lymphatic fluid spilling into the abdominal cavity, which is rare in the clinic.[1,2] In cases of chylous ascites, the primary underlying mechanism is lymphatic obstruction—either

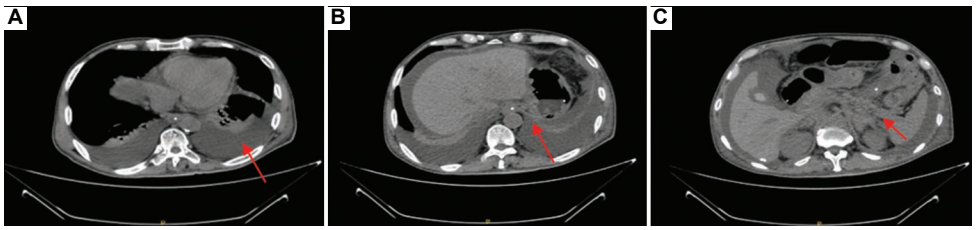

Figure 2. Chest and abdominal computed tomography (CT) scans. (A) Pleural effusion with atelectasis of the anterior lung tissue. (B and C) CT images of the abdomen demonstrating multiple enlarged retroperitoneal and pelvic lymph nodes suggestive of metastasis (red arrows). There is evidence of peritoneal and omental metastases, accompanied by massive ascites in the abdominal and pelvic cavities. Red arrows indicate the enlarged lymph nodes and areas of probable lymphatic compression/obstruction responsible for chylous leakage.

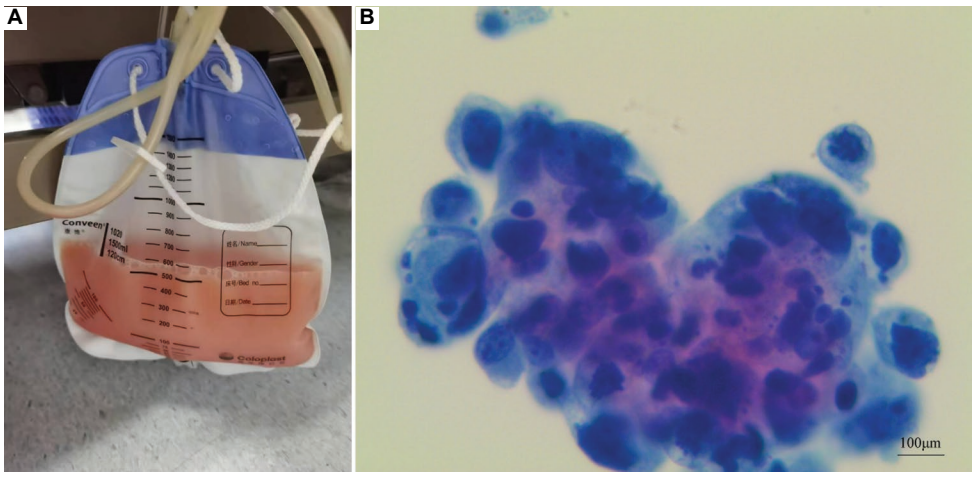

Figure 3. Abdominal cavity effusion. (A) The ascitic fluid appeared milkywhite and opaque, consistent with chyle. (B) Papanicolaou staining of abdominal fluid showing scattered malignant adenocarcinoma cells with high nuclear-to-cytoplasmic ratio and conspicuous nucleoli (magnification: ×100).

due to intraluminal fibrosis or extraluminal compression. Typically, patients with chylous ascites harbor large tumor that extensively infiltrates the retroperitoneal and mesenteric lymph nodes. Local tumor invasion and mass effect likely cause compression or obstruction of lymphatic vessels, ultimately leading to the development of chylous ascites.

The main clinical manifestations of chylous ascites include abdominal distension, abdominal pain, diarrhea, dysphagia, fatigue, malnutrition, and progressive peripheral edema, possibly accompanied by nausea, vomiting, steatorrhea, and fever.[7,8] In physical examination, the condition may present as cachexia, chest and abdominal effusion, abdominal mass, and lower limb edema.[9] Peritoneal aspiration is the preferred measure for the diagnosis and evaluation of chylous ascites. If the ascites is milky in appearance, the triglyceride level in the ascites should be measured. Laboratory tests included detection of total protein, albumin, lactate dehydrogenase, triglyceride concentration, amylase, and lipase. Typical chylous ascites has a chylous, milky appearance with a specific gravity of 1.012–1.018. After standing, it is stratified and alkaline. The Sudan III staining test revealed substantial deposition of fat droplets and lymphocytes. Further assessments include triglycerides >200 mg/dL; total protein >25 g/L, serum/ascites protein gradient <11 g/L, and lactate dehydrogenase 110–200 IU/L. Chylomicron bands can be seen in lipoprotein electrophoresis. In addition, X-ray lymphangiography is the gold standard for the diagnosis of lymphatic obstruction. Lymphangiography and radionuclide lymphoscintigraphy are important in detecting abnormal retroperitoneal lymph nodes, the presence of leakage, fistula formation, and the patency of lymphatic vessels.[10]

The effective treatment principle for chylous ascites lies in adopting conservative treatment and nutritional support on the basis of alleviating the primary disease so as to improve patient comfort, reduce adverse reactions, and prolong survival. The treatment approaches include the following: (i) Abdominal puncture drainage. It temporarily relieves symptoms, but it is not appropriate to repeatedly drain ascitic fluid in large volume. (ii) Rational use of somatostatin. Somatostatin inhibits the secretion of intestinal fluid and reduces the amount of fluid absorbed into the stroma through the intestine, thereby reducing the production of intestinal lymph. In addition, somatostatin inhibits lymph secretion by inhibiting specific receptors in the lymphatic vessels of the normal intestinal wall.[11] Retrospective and small observational studies have reported that treatment combined with nutritional interventions resulted in reductions in ascitic output and paracentesis frequency, but prospective controlled data are lacking to support such findings.[12] (iii) Fasting. Fasting keeps the gastrointestinal tract at rest and reduces lymph production and loss. When selecting nutrients, attention should be paid to the selection of short-chain and MCTs. Short-chain and medium-chain fatty acids are absorbed directly into the venous system, thereby reducing lymphatic fluid production and lymphatic pressure. This helps facilitate the healing of lymphatic leaks or disruptions.[13] Foods containing long-chain triglycerides, such as nuts, fish, and meat, should be avoided.[14-16] In selected instances, invasive lymphatic interventions—including lymphangiography with embolization or thoracic duct ligation—and peritoneovenous shunting (e.g., Denver shunts) provide more durable control of chyle leakage. Such strategies require specialized imaging, technical expertise, and patient fitness, and are most suitable when leaks are localized rather than diffuse.[17] Taken together, the evidence supports a stepwise, goal-concordant approach in malignant chylous ascites—prioritizing simple, low-burden palliative measures (judicious drainage, nutritional optimization, the use of octreotide when appropriate), reserving invasive lymphatic or shunt procedures for carefully selected patients with a realistic expectation of benefit, and de-emphasizing serial radiologic or biomarker assessments when these will not change patient-centered goals of care—an approach that aligns with the management choices made in the present case.

Malignant chylous ascites most often reflects extensive lymphatic involvement by advanced malignancy and therefore commonly signals a poor overall prognosis. In this context, the therapeutic aims should pivot away from disease-directed endeavors with low likelihood of meaningful benefit, and instead emphasize symptom control, preservation of dignity, and optimization of quality of life. For patients whose clinical trajectory and physiological reserve no longer permit toleration of cytotoxic or aggressive interventional therapies, pursuing repeated staging investigations or aggressive attempts at tumor control can impose additional harm without improving outcomes. We therefore advocate that clinicians explicitly reassess goals of care when chylous ascites develops in the setting of widely metastatic disease and move promptly toward palliative priorities when appropriate.

4. Conclusion

In the context of advanced, often inoperable malignancy with CA, management should prioritize symptom control, preservation of remaining functional status, and maintenance of quality of life. Clinical decisions should be guided by frank discussions with the patient and

family about prognosis, treatment goals, and the potential benefits and burdens of interventions. Early involvement of palliative care services is recommended to assist with symptom management, psychosocial support, and complex decision-making.

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Conflict of interest

The authors declare they have no competing interests.

Author contributions

Conceptualization: Guanwen Gong
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Supervision: Guanwen Gong
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Writing–review & editing: Xiaochun Zhang, Jialing Shi

Ethics approval and consent to participate

This study protocol was reviewed and approved by the ethics committee of Affiliated Hospital of Nanjing University of Chinese Medicine (registry number: 2022NL-129-01). Written informed consent was obtained from the patient before his participation.

Consent for publication

Written informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images. All identifying information has been removed or masked to ensure anonymity of the participant.

Availability of data

The data from the current study are available from the corresponding author on reasonable request.

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