CASE REPORT

A case of advanced rectal cancer treated with chemotherapy after developing short bowel syndrome following emergency surgery for superior mesenteric artery embolism

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Abstract : Introduction : While venous thrombosis is often associated with malignancy, arterial thromboembolisms are rare. We report a case of unresectable rectal cancer complicated by superior mesenteric artery (SMA) embolism, resulting in short bowel syndrome (SBS), but was successfully treated with chemotherapy. Case presentation : A 60-year-old woman with unresectable rectal cancer presented to our hospital with abdominal pain. Computed tomography revealed contrast defects originating from the SMA. In addition, CT demonstrated small intestinal wall disruption and free air. She was diagnosed with small intestinal ischemia and gastrointestinal perforation due to SMA embolism. Emergency small intestinal resection and jejunostomy were performed (operative time, 77 min; blood loss, 80 mL). The stoma was created 60 cm from the ligament of Treitz, resulting in the development of SBS. After stabilization with fluid management, a central venous port was placed. Chemotherapy was initiated, and the patient was discharged on postoperative day 29. The tumor shrank after completing four courses of chemotherapy. The patient received home parenteral nutrition and had nine courses of chemotherapy. Conclusions : This case suggests that the introduction of chemotherapy may be feasible and effective even in patients with SBS secondary to SMA embolism. SBS should not preclude chemotherapy in patients with malignant tumors. J. Med. Invest. 73 : 260-264, February, 2026

Keywords **:** *Superior mesenteric artery embolism, short bowel syndrome, chemotherapy, peritoneal dissemination, rectal cancer*

INTRODUCTION

Patients with cancer are prone to venous embolic complications, with an estimated 4–20% of patients experiencing venous thrombosis (1). However, the incidence of arterial embolism in patients with cancer is approximately 1.5% (2). The mechanism of venous embolism in patients with cancer is hypercoagulability due to malignancy (3). Arterial embolism is generally caused by atrial fibrillation, arterial embolism, or valvular heart disease (4). Thus, patients with cancer are more susceptible to venous embolism.

Superior mesenteric artery (SMA) embolism often requires massive small bowel resection, and many cases of short bowel syndrome (SBS) develop postoperatively. Fluid management is difficult in SBS because of digestive and absorption disorders and should be strictly controlled using total parenteral nutrition (TPN). Chemotherapy is the first choice for prolonging survival in patients with unresectable advanced cancer ; however, to date, there are no reports on its feasibility or efficacy in patients with SBS. Chemotherapy in patients with SBS may induce mucosal damage in the remaining gastrointestinal tract, requiring more rigorous fluid and nutritional management, and potentially shortening the patient's life. If chemotherapy is administered to patients with SBS, clinicians and medical staff must start and administer it with great care.

Here, we report a patient with unresectable rectal cancer with peritoneal dissemination who underwent emergency surgery for SMA embolism and subsequently developed SBS, but successfully underwent chemotherapy.

CASE PRESENTATION

A 60-year-old woman presented at our hospital with abdominal pain. There was no notable family history, but her medical history included hypertension and diabetes. Computed tomography (CT) revealed a contrast-enhanced mass in the rectum (Fig. 1A), increased density in the omentum (Fig. 1B), and a nodule around the left ovary, leading to a diagnosis of unresectable rectal cancer with peritoneal dissemination. The patient's treatment plan included a referral to our hospital for further examination and treatment. However, she presented as an unscheduled visit because she vomited at home before further examination at our hospital. She had abdominal distention and tenderness throughout the abdomen, but there was no substantial change compared to the previous few days.

A laboratory examination showed a marked increase in white blood cell count at 26400/μL and C-reactive protein at 40.23 mg/dL, but there were no other abnormal findings. Carcinoembryonic antigen and cancer antigen 19-9 levels were not elevated. Contrast-enhanced CT showed, in addition to rectal wall thickening and peritoneal nodules, contrast defects at the origin

Abbreviations :
BUN, blood urea nitrogen ; CT, computed tomography ; FOLFOX, folinic acid, fluorouracil, and oxaliplatin ; HPN, home parenteral nutrition ; POD, postoperative day ; SBS, short bowel syndrome ; SMA, superior mesenteric artery ; TPN, total parenteral nutrition ; QOL, quality of life

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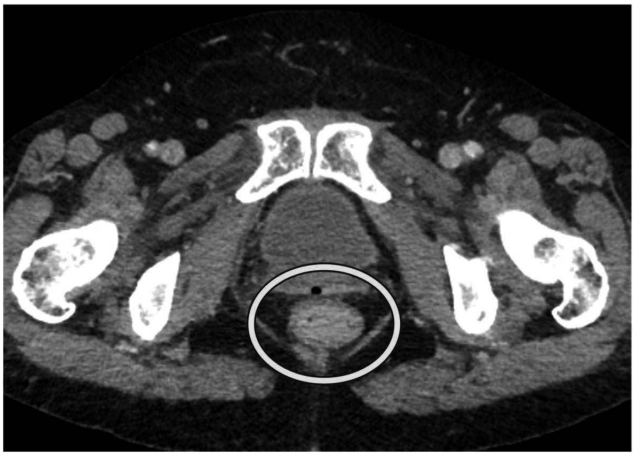

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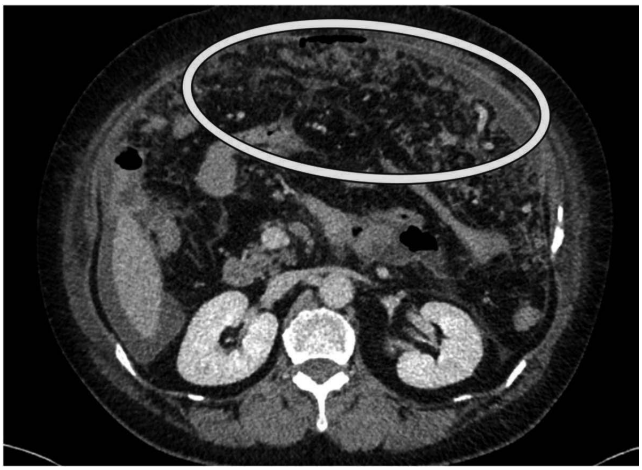

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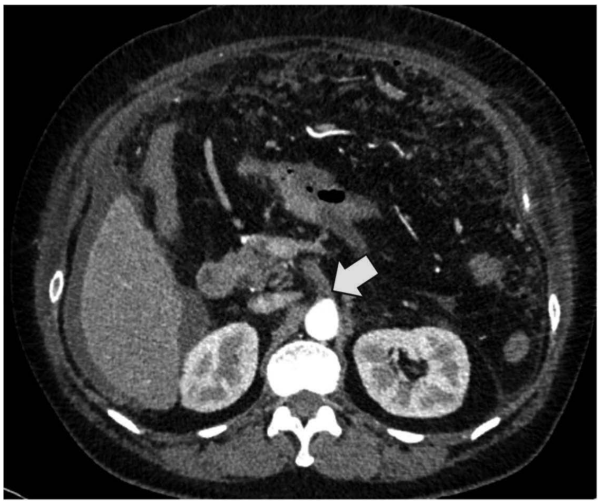
Fig 1. The findings when the tumor was discovered.
(A) Irregular wall thickening in the upper rectum.
(B) Ascites with numerous disseminated nodules.

of the SMA and poorly contrasted areas in the small intestine (Fig. 2A). Additionally, disruption of the small intestinal wall was observed in part of the small intestine, and free air was observed in the surrounding area (Fig. 2B). To investigate the potential cause of thrombus formation, an electrocardiogram was performed and demonstrated a normal sinus rhythm without any evidence of atrial fibrillation or other arrhythmias. Furthermore, contrast-enhanced computed tomography revealed no signs of advanced arteriosclerosis, such as vascular calcifications or significant atheromatous changes in the major arteries, including the SMA. The patient also had no prior history of cardiovascular disease.

Based on these findings, the patient was diagnosed with small intestinal ischemia and gastrointestinal perforation due to an SMA embolism. Emergency small intestinal resection and the jejunostomy were performed (operative time, 77 min ; blood loss, 80 mL). The stoma was created 60 cm from the ligament of Treitz. Postoperatively, to prevent the progression of SMA thrombosis, anticoagulation therapy with heparin was initiated. The drains were removed sequentially, and the patient was started on a liquid diet on postoperative day (POD) 4. Follow-up contrast-enhanced CT revealed no changes in the SMA thrombus. Given the stability of imaging findings and the absence of newly formed thrombi, the cardiologist concluded that continuation of anticoagulant therapy was not necessary. Accordingly, heparin therapy was discontinued in POD 9. The patient's baseline infusion volume was 2000 mL per day. Fluid management was performed by adding 500 mL of water to 1000 mL of fluid discharged from the stoma to ensure that the daily urine volume did not fall below 1000 mL. On POD 14, fluid management was confirmed to be possible with a regular diet and 2000 mL of intravenous fluid per day without the need for additional intravenous fluids. Although the patient developed SBS postoperatively, her condition stabilized ; therefore, the patient requested chemotherapy for the unresectable rectal cancer. Chemotherapy was administered after a multidisciplinary conference. Genetically, this case was wild-type RAS and BRAF, microsatellite-stable, and HER2-negative. On POD 16, a central venous port was created, and on POD 22, a regimen of folinic acid, fluorouracil, and oxaliplatin (FOLFOX) was introduced. The patient was discharged on POD 28 on total parenteral nutrition. After discharge, the patient visited the hospital once weekly



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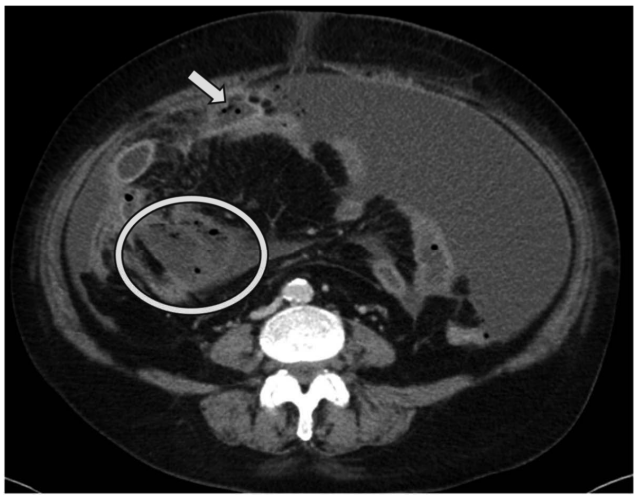

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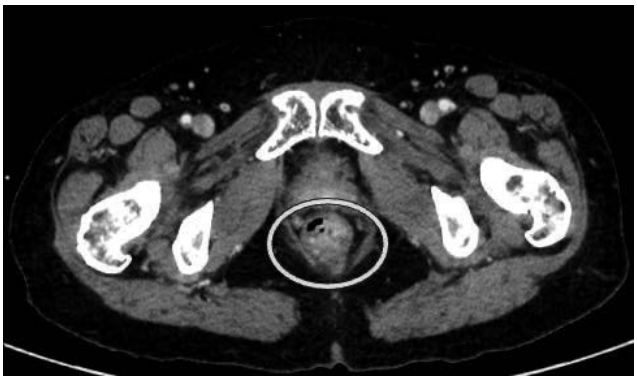
Fig 2. Intestinal ischemia and perforation
(A) Contrast-enhanced computed tomography in the arterial phase shows a contrast defect originating from the superior mesenteric artery
(B) In addition to a poorly contrast-enhanced small intestine, a small intestine with a collapsed intestinal wall or free air was also observed.

for outpatient care. Fluid management included checking the blood collection data, adjusting the TPN flow rate, and strictly managing electrolytes. Blood urea nitrogen (BUN) was particularly noted ; when elevated, the infusion rate was adjusted (40–80 mL/h). After completing 4 courses of FOLFOX therapy, the tumor shrank (partial response) (Figs. 3A, B).

When the sixth course was started, an increase in BUN level (62 mg/dL) was observed. Chemotherapy was discontinued, and the patient was urgently admitted to the hospital. Additional infusion was performed, with blood collection once every 2 or 3 days led to rapid improvement of the dehydration. Based on these findings, it was considered that the dehydration resulting in emergency admission was caused by exacerbation of intestinal mucosal injury due to chemotherapy. After resolution of the dehydration, chemotherapy was resumed at a reduced dose of 80%. After the eighth course was completed, liquid drainage from the patient's stoma decreased, and she complained of abdominal distension. The patient was diagnosed with bowel obstruction and admitted to the hospital. Contrast-enhanced CT performed at the time of admission revealed findings suggestive of bowel obstruction ; however, both the primary tumor and peritoneal dissemination had decreased in size (Figs. 4A, B). Endoscopic examination via the stoma revealed marked angulation near the stoma, leading to a diagnosis of small bowel stricture secondary to regression of peritoneal dissemination. This stricture near the stoma rendered the patient unable to tolerate oral intake. Therefore, after discussing the procedure with the patient, a percutaneous endoscopic gastrostomy tube was placed for the purpose of gastrointestinal decompression. Subsequently, a ninth course of chemotherapy was administered ; however, the patient remained unable to tolerate oral intake and showed limited improvement in overall condition. As a result, chemotherapy was discontinued, and approximately seven months after diagnosis and surgery, the patient was referred to a local home care facility.



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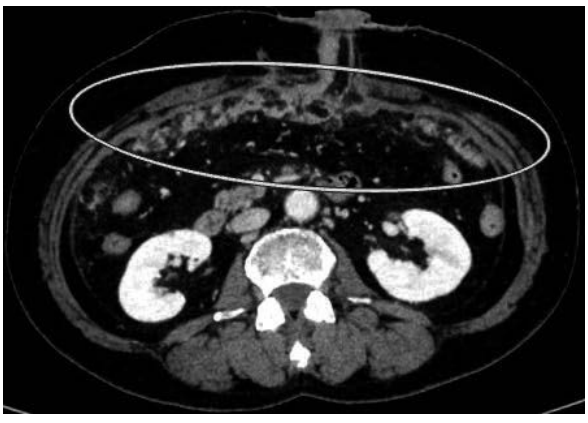

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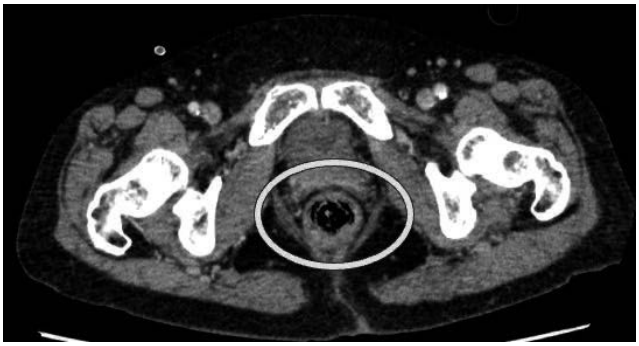
Fig 3. The effect of FOLFOX after 4 courses
(A) After 4 courses, local lesion reduction is observed.
(B) Peritoneal dissemination is also observed to shrink.



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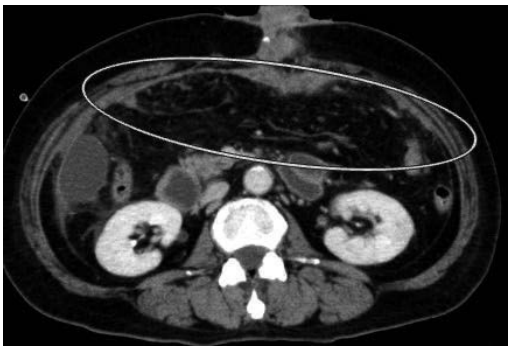

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Fig 4. The effect of FOLFOX after 8 courses
(A) After 8 courses, further reduction of the local lesion was observed.
(B) Similar findings were observed for the peritoneal dissemination.

DISCUSSION

One of the factors contributing to venous thromboembolism is cancer, a condition known as Trousseau's syndrome (1). However, reports of arterial embolism in cancer patients are relatively rare, with an incidence rate of approximately 1.5% (2). Accordingly, many clinicians remain unaware of the potential association between cancer and arterial thromboembolism. Large cohort studies have demonstrated that patients with advanced cancers, including colorectal cancer, have a significantly higher risk of developing arterial thromboembolic events compared to individuals without cancer (5). Furthermore, these events have been reported to be significantly associated with increased mortality in cancer patients (5). The factors that contribute to arterial embolism include SMA embolism, atrial fibrillation, atherosclerosis, and a history of valvular heart disease (4). Moreover, chronic inflammatory states, such as a malignant state and nonbacterial thrombotic endocarditis, can also be induced by tumor-associated inflammatory cytokines and increased coagulability, leading to arterial embolism (6). In this case, the patient had unresectable advanced rectal cancer with multiple peritoneal disseminations and no other identifiable cause of arterial embolism, suggesting that the thrombus was formed due to

hypercoagulability caused by the malignant tumor. In addition, therapeutic agents such as angiogenesis inhibitors and other molecular-targeted drugs may increase the risk of thrombosis, and attention should be paid to arterial thrombosis in patients with cancer undergoing anticancer drug treatment (6). In this case, angiogenesis inhibitors were not administered because the patient had an arterial embolism.

SMA embolisms have a poor prognosis, with a mortality rate of approximately 50% after onset. The prognosis after onset also depends on the time to starting treatment (7). Symptoms of SMA embolism include sudden abdominal pain, vomiting, and diarrhea (8), but these are common symptoms of acute abdomen and are not specific to SMA embolism. Biochemistry also shows neutrophil-predominant leukocytosis and hemoconcentration, which are nonspecific findings (8). Elevated lactate levels are associated with irreversible ischemia, but this is also nonspecific. Contrast-enhanced CT is useful in diagnosing SMA embolism and can directly show the site of occlusion (8). Prompt evaluation with contrast-enhanced CT in combination with symptoms leads to the early diagnosis of SMA embolism. In this case, in addition to vomiting, the blood samples showed a marked increase in the inflammatory response. Early contrast-enhanced CT may have saved the patient's life by enabling surgery at an early stage of the disease.

In healthy adults, the small intestine is more than 6 m in length, and the resection of more than 75% of the intestine results in severe digestive and absorption disorders. SBS is defined as a condition in which the remaining intestinal tract is less than 2 m in length (9). In this case, the stoma was initiated 60 cm distal to the ligament of Treitz, and the usable small intestine was 60 cm. In SBS, fluid and electrolyte imbalances and other problems can occur due to digestive and absorption disorders (9). TPN is used to manage fluid nutrition in SBS, and one of the indicators of fluid management is the estimation of dehydration based on urine volume and urinary constituents. Some reports suggest that a urine volume of at least 1 L/d and a urinary sodium concentration of at least 20 mEq/L are sufficient (10). In this case, dehydration was prevented by strict fluid management based on the measurement of stoma drainage volume in addition to urine output. After discharge, fluid management was performed using biochemical findings once a week. Although closure of jejunostomy might have facilitated the fluid management, the presence of severe rectal stenosis precluding endoscopic passage, and the presence of multiple peritoneal nodules raised the concern of further intestinal obstruction. Given the risk, maintaining the decompressive jejunostomy was deemed essential to ensure adequate gastrointestinal decompression and to prevent additional complications during the course of systemic chemotherapy. Recently, the glucagon-like peptide-2 analog teduglutide was recently used as a treatment for SBS, increasing the rate of TPN withdrawal by inducing mucosal proliferation and decreasing gastrointestinal motility (11) However, it has been suggested that teduglutide may promote the growth of malignant tumors (12). Therefore, after discharge, the patient received home parenteral nutrition (HPN) and was monitored for dehydration and electrolyte abnormalities at weekly outpatient visits, with close management of fluids and electrolytes during chemotherapy to continue treatment.

To date, there have been only a few reports on the administration of chemotherapy for malignant tumors with SBS. SBS can be caused by multiple bowel resections for recurrent Crohn's disease, venous thrombosis, intestinal volvulus, trauma, mesenteric artery embolism, and massive resection of the intestine following tumor resection (13). SBS is often caused by benign diseases and chemotherapy is rarely required, which is probably why there have been only a few reports to date of the introduction of chemotherapy for SBS. Since about 80% of adult patients with SBS due to benign diseases survive for a long period after the introduction of HPN (9), malignant tumors may be a prognostic factor. Given the possibility that some patients with malignant tumors may not undergo chemotherapy due to SBS, it is essential to accumulate clinical cases by reporting the considerations involved in administering chemotherapy in such scenarios, along with its safety and efficacy following treatments.

Chemotherapy is the standard treatment for unresectable colorectal cancer with peritoneal dissemination. Chemotherapy is required to prolong survival in patients with SBS after emergency surgery but with coexisting unresectable colorectal cancer. Gastrointestinal toxicity, including symptoms such as diarrhea, nausea, vomiting, and anorexia, which contribute to dehydration, is one of the adverse events of chemotherapy (14). In addition, increased risk of infection is commonly observed. Catheter related infections are well known complication in patients with SBS. Given the risk with chemotherapy, diligent catheter care and close clinical monitoring are essential components of management in these patients to minimize the risk.

Because fluid and electrolyte management is often difficult in SBS, there are more precautions required when introducing chemotherapy than in the usual setting. The adverse effects of chemotherapy require careful monitoring during chemotherapy induction. Therefore, irinotecan-based chemotherapy, which has strong gastrointestinal toxicity, is difficult to administer, and oxaliplatin-based chemotherapy may be preferable. In this case, the patient's performance status was maintained, and FOLFOX, based on oxaliplatin, was introduced with strict fluid and electrolyte control during hospitalization. Chemotherapy was continued in an outpatient setting after hospital discharge. After the introduction of FOLFOX, a total of 9 courses were administered to the patient through weekly visits to the hospital and home nursing care. Although chemotherapy was discontinued approximately seven months after diagnosis and surgery, both the primary lesion and peritoneal dissemination remained reduced in size. These findings suggest that, even in the presence of SBS, therapeutic efficacy was achieved, and the treatment may have contributed to prolonged survival and maintenance of quality of life (QOL) compared to no treatment. Previous reports have indicated that the survival duration of untreated colorectal cancer patients with peritoneal dissemination is less than nine months and the peritoneal dissemination impairs patient QOL, manifesting as abdominal pain, distension, and anorexia (15). Compared to this report, chemotherapy may have contributed to prolonging her survival and maintaining QOL. However, this comparison is indirect, and the influence of confounding factors such as performance status and tumor burden cannot be ruled out. Therefore, further accumulation of similar cases and prospective data are needed to validate the survival benefit of chemotherapy in patients with SBS. Although chemotherapy can be administered to patients with SBS, further case accumulation is needed to better understand treatment strategies and prognostic effects.

CONCLUSIONS

We encountered a case of unresectable rectal cancer complicated by SBS secondary to SMA embolism, in which chemotherapy was effective. This case suggests that chemotherapy should be considered as a therapeutic option for malignancies even in patients with SBS.

DECLARATIONS
ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Patient privacy was protected, and this study did not include any patient-identifying information.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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