

## Case Report

# Long-Term Survival of a Patient with Adenocarcinoma of the Esophagogastric Junction with a Portal Vein Tumor Thrombosis Who Underwent Palliative Total Gastrectomy: A Case Report

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## Keywords

Portal vein tumor thrombosis · Advanced gastric cancer · Palliative chemotherapy · Doxifluridine · Survival

## Abstract

Portal vein tumor thrombosis (PVTT) with advanced gastric cancer is very rare; when it occurs, it exhibits aggressive growth and carries a poor prognosis. In addition, definitive treatment has not been established due to insufficient data. Herein, we report a case of PVTT associated with an adenocarcinoma of the esophagogastric junction that was successfully controlled by means of a palliative total gastrectomy without surgical resection of the PVTT and administration of palliative continuous doxifluridine.

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## Introduction

Portal vein tumor thrombosis (PVTT) usually occurs in patients with advanced hepatocellular carcinoma (HCC); it is rare in patients with advanced gastric cancer [1]. In rare cases

of advanced gastric cancer with PVTT, the tumor exhibits aggressive growth and carries a poor prognosis [2]. The treatment of HCC with PVTT is established. It comprises hepatic resection with extirpation of the tumor thrombus, microwave coagulation therapy, ethanol injection therapy, transcatheter arterial embolization, and systemic chemotherapy [3, 4]. In contrast, because of its low incidence, there are few reports on the treatment of PVTT associated with advanced gastric cancer.

Herein, we report a case of PVTT associated with an adenocarcinoma of the esophagogastric junction without liver metastasis that was successfully controlled by palliative total gastrectomy without surgical resection of the PVTT and administration of palliative enteral chemotherapy.

### Case Report

A 71-year-old man presented with epigastric discomfort, dysphagia, and regurgitation. He had no specific medical or family history. The initial esophagogastroduodenoscopy revealed an ulcerofungating mass encircling the esophagogastric junction; pathologic examination identified the mass as an adenocarcinoma. The patient underwent a contrast-enhanced computed tomography (CT) scan of the abdomen and pelvis that revealed advanced gastric cancer at the esophagogastric junction, multiple enlarged lymph nodes along the lesser curvature of the stomach, no visible distant metastatic lesions in the other organs, no ascites, and a suspicious splenic vein thrombosis (Fig. 1). The initial carcinoembryonic antigen (CEA) level was 667.9 ng/mL and other tumor markers were within normal limits. To determine operability, the patient underwent a positron emission tomography/CT scan; this revealed advanced gastric cancer, multiple perigastric and retropancreatic lymph node metastases, and a suspicious splenic vein tumor thrombosis (Fig. 2).

In the field of operation, the gastric tumor showed no evidence of serosal invasion. A total gastrectomy with D2 lymph node dissection was performed, together with a splenectomy. Additionally, grossly enlarged retropancreatic lymph nodes were dissected out. The most difficult operative procedure was the suprapancreatic lymph node dissection as lymph nodes that had metastatic features were firmly attached to the splenic vessels and pancreas. Because of the patient's age, the surgeon decided not to perform a pancreatectomy but rather just to dissect out the suprapancreatic lymph nodes.

The Borrmann type III gastric cancer at the esophagogastric junction was 8.0 × 7.5 × 1.2 cm in size and was histologically diagnosed as a moderately differentiated tubular adenocarcinoma of the intestinal type. According to the 7th edition of the AJCC/UICC classification of gastric cancer, the case was classified as stage IV (T3, N0, with retropancreatic lymph node metastasis).

The patient's postoperative course was uneventful. He underwent chemotherapy with S-1 (tegafur, gimeracil, potassium oxonate). After 4 courses of chemotherapy, the patient complained of generalized weakness and vomiting; the CEA level had risen to 603.8 ng/mL. Hence, his chemotherapy regimen was changed to FOLFOX (5-fluorouracil, oxaliplatin, leucovorin). After 2 courses of FOLFOX chemotherapy, the patient refused further chemotherapy due to its side effects. After discussion and persuasion, the patient conditionally agreed to enteral administration of chemotherapy rather than intravenous chemotherapy – oral doxifluridine was used. The patient tolerated this chemotherapy, and the CEA level decreased 1, 3, and 4 months after taking doxifluridine to 213.3, 29.48, and 29.97 ng/mL, respectively. Thereafter, however, the CEA level increased again, rising to 46.5 and 1,257.0 ng/mL, re-

spectively, 5 and 12 months after starting doxifluridine. In addition, the CT scan performed at 12 months of doxifluridine chemotherapy revealed a tumor thrombosis in the main and right portal veins, with cavernous transformation of the portal vein (Fig. 3). As a result, the chemotherapy regimen was changed to FOLFIRI (5-fluorouracil, leucovorin, irinotecan). However, after 2 courses of FOLFIRI chemotherapy, the patient again developed severe generalized weakness, prompting a change back to oral doxifluridine chemotherapy as this regimen was the most acceptable to the patient. The patient experienced no specific side effects during doxifluridine chemotherapy, and the CEA level decreased to 5.4, 6.2, 4.2, 5.5, 4.2, 4.2, and 4.3 ng/mL, respectively, 4, 12, 16, 24, 35, 44, and 54 months after restarting doxifluridine. In addition, a follow-up CT scan performed 78 months postoperatively revealed no change in disease status compared with the previous CT scan (Fig. 4). The patient has survived for 82 months after surgery without disease progression.

## Discussion

PVTT is extremely rare in patients with gastric cancer; the reported incidence is 1.2% [5]. Because cancer cells originating in the stomach spread via the lymphatic or portal venous systems, metastases most frequently occur in the regional lymph nodes and liver. Generally, PVTT develops in cases of advanced HCC, because of the hypervascular features of this cancer, with shunt formation from the hepatic artery to the portal vein. In addition, locally invasive metastatic tumors in the liver may directly cause PVTT, i.e., PVTT is generally seen when a liver tumor is located adjacent to the involved segmental portal vein. However, no liver mass was observed in this case.

Therefore, other causes should be considered to explain the formation of PVTT in patients with gastric cancer. There are some reports that explain the pathogenesis of PVTT. First, patients with cancer are in a hypercoagulable state [6, 7]. Intact tumor cells may express procoagulant activity that can directly induce thrombin generation. In addition, normal host tissue may express procoagulant activity in response to the tumor. Second, portal flow may decrease due to compression by enlarged lymph nodes around the portal vein. Hemodynamic forces strongly influence the biochemical make-up of thrombin and the reaction pathways involved in thrombus formation [8]. However, there were no lymph nodes influencing portal flow in this case. Third, there are some reports that adhesion molecules, such as intercellular adhesion molecule 1 (ICAM-1), are strongly expressed during the invasion and/or metastasis of gastric cancer and that the expression of ICAM-1 on adenocarcinoma cells is augmented by tumor-infiltrating lymphocytes [9]. Thus, it is conceivable that adhesion and permeation of tumor cells into the portal vein may be related to the highly augmented expression of adhesion molecules on adenocarcinoma cells and the increased number of tumor-infiltrating lymphocytes in the portal vein. This remains to be investigated further in this case.

The treatment of PVTT originating from HCC comprises hepatic resection, microwave coagulation therapy, ethanol injection therapy, transcatheter arterial embolization, and systemic chemotherapy. However, these methods cannot completely control PVTT. Therefore, hepatic resection with extirpation of the PVTT has been performed to prolong survival as far as possible, even though curative resection is not possible. However, in patients with gastric cancer and PVTT, the role of surgical resection of PVTT with gastrectomy cannot be evaluated due to insufficient data. In addition, there is currently no definite chemotherapeutic regi-

men for PVTT originating from gastric cancer, although some reports indicate that combinations of S-1 plus CDDP or 5-fluorouracil plus CDDP have shown a treatment effect [10, 11].

In the present study, we report the case of an elderly patient who had adenocarcinoma of the esophagogastric junction with PVTT. After performing a palliative total gastrectomy without surgical resection of PVTT, the disease is being successfully controlled with administration of doxifluridine. This case report shows that palliative total gastrectomy with continuous doxifluridine could be a treatment option allowing for long-term survival of elderly patients with PVTT secondary to adenocarcinoma of the esophagogastric junction.

### Statement of Ethics

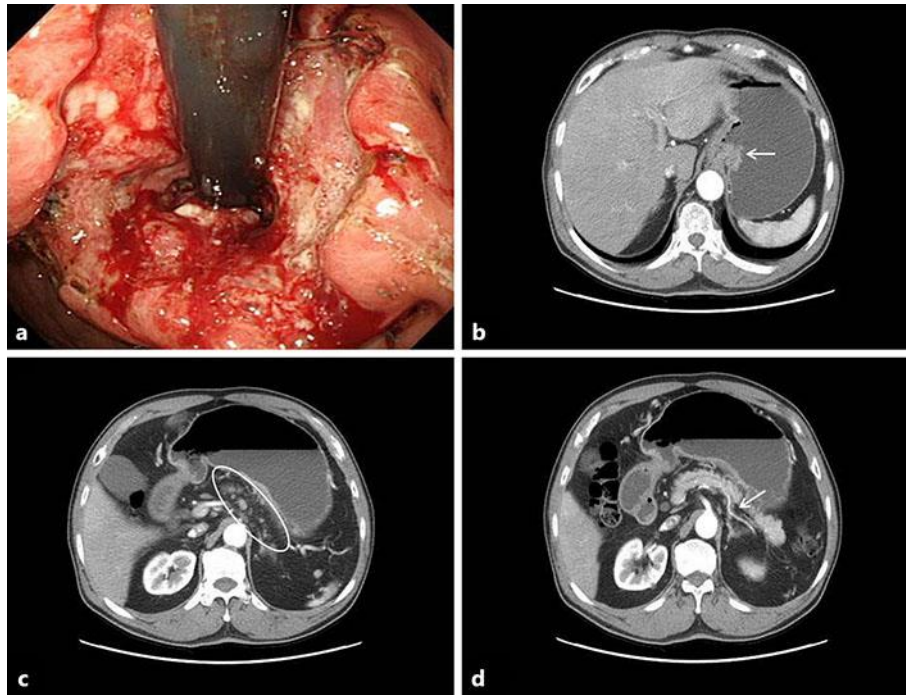
Written informed consent for publication of this paper was obtained from the patient.

### Disclosure Statement

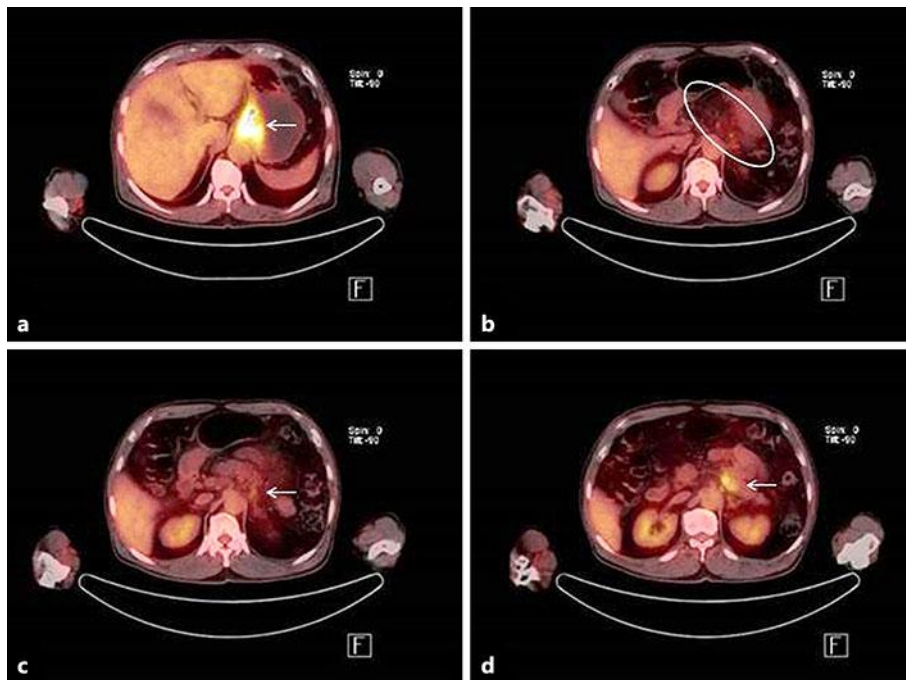
The authors have no conflicts of interest to declare.

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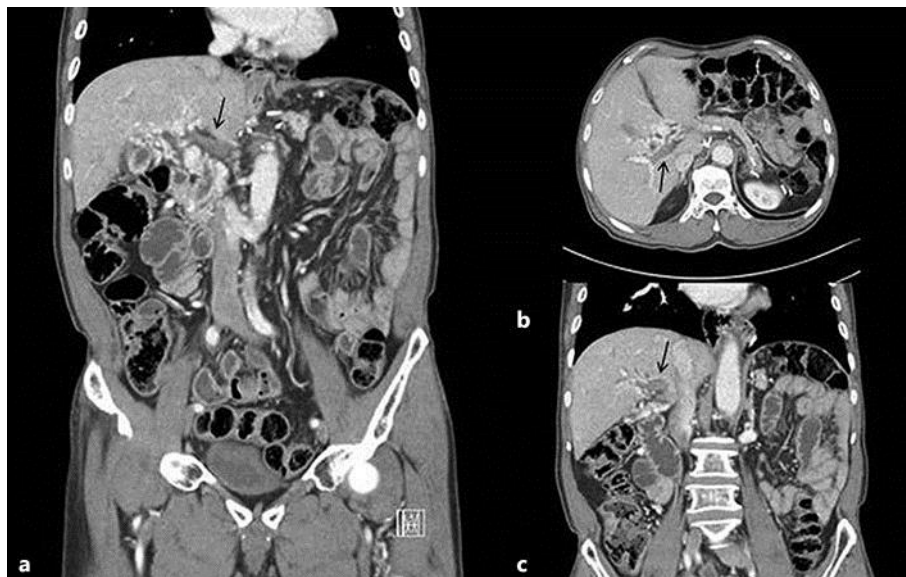
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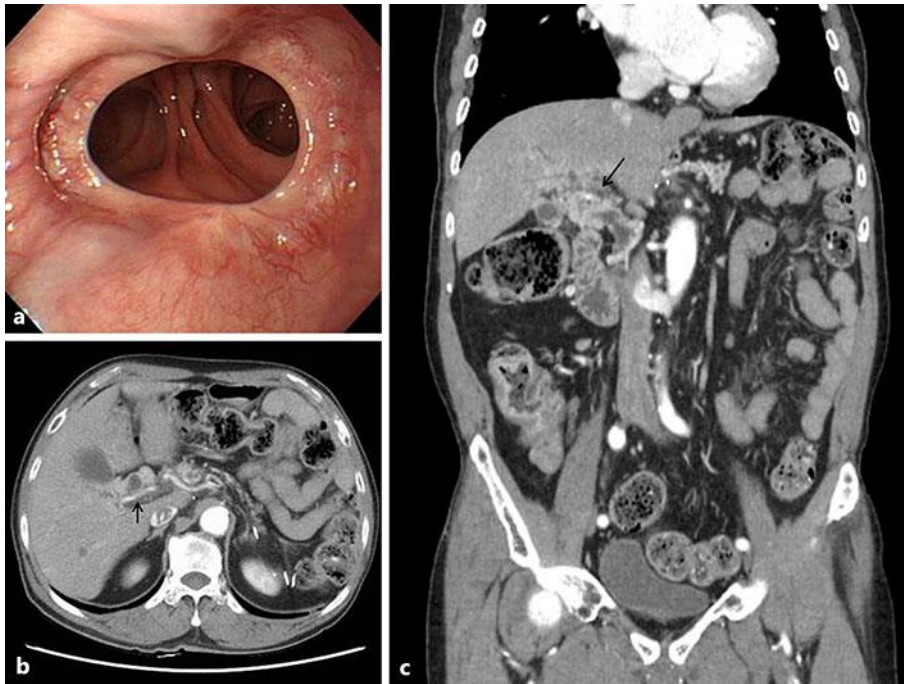
**Fig. 1.** **a** Esophagogastroduodenoscopy revealed advanced gastric cancer at the esophagogastric junction. **b** Computed tomography revealed advanced gastric cancer at the esophagogastric junction. **c** Multiple enlarged lymph nodes along the lesser curvature of the stomach. **d** Suspicious splenic vein thrombosis.



**Fig. 2.** a Positron emission tomography/computed tomography revealed advanced gastric cancer at the esophagogastric junction. b Multiple perigastric lymph nodes metastases. c Retropancreatic lymph node metastases. d Splenic vein tumor thrombosis.



**Fig. 3.** a Follow-up computed tomography images reveal tumor thrombosis in the main portal vein. b Right portal vein tumor thrombosis (axial view). c Right portal vein tumor thrombosis (sagittal view).



**Fig. 4.** **a** The most recent follow-up esophagogastroduodenoscopy revealed no tumor recurrence at the anastomosis site (esophagojejunostomy). **b** The most recent follow-up computed tomography revealed persistence of the right portal vein tumor thrombosis. **c** The main portal vein tumor thrombosis remains evident.