Case Report

Metastatic Esophageal Tumor from Cecal Carcinoma

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A 55-year-old man developed progressive dysphagia 14 months after palliative colectomy and subsequent systemic chemotherapy for advanced cecal cancer with carcinomatosis peritonei. Radiologic and endoscopic examinations suggested a submucosal tumor in the lower esophagus causing a severe luminal stricture. A self-expanding metal stent was placed for palliation. The prosthesis was effective for several months, but ingrowth of the tumor caused re-stricture of the esophagus. Since his general condition was quite good without any evidence of recurrence of the cecal cancer, we performed bypass surgery for palliation. The pathological appearance of the tumor was compatible with the metastasis of cecal cancer. Our case suggests that a surgical approach can be considered as a therapeutic method for metastatic esophageal tumor, even in patients with advanced cancer, as long as the primary tumor is satisfactorily controlled.

Key words: chemo-GI tract – endoscopy-upper GI – GI surgery

INTRODUCTION

The incidence of metastatic esophageal tumor from a distant primary lesion is low. In particular, esophageal metastasis from colorectal carcinoma has not been documented in detail except for two short reports suggesting metastasis from rectal cancer (1,2). In autopsy series, the incidence of secondary esophageal metastasis of various primary neoplasms has been reported as 0.3–6.1% (3–5). In most of these cases, however, such lesions were accompanied by multiple metastases at other sites or mediastinal carcinomatosis, and thus aggressive local treatment is not usually performed for metastatic esophageal tumors.

Here, we present a case of stage IV cecal adenocarcinoma in which esophageal stricture developed due to metastasis to the lower esophagus 14 months after palliative colectomy and systemic chemotherapy. In this patient, however, the cecal cancer had been well controlled by chemotherapy for more than 2 years, and malignant dysphagia was successfully treated by surgical bypass as well as an endoscopic prosthesis even though he had peritoneal metastasis at the initial operation.

CASE REPORT

A 54-year-old man was diagnosed with adenocarcinoma of the cecum and underwent colectomy in August 2004. At laparotomy, carcinomatosis peritonei with lymphatic involvement was found and palliative ileocecal resection was performed. Pathological examination revealed moderately to well differentiated adenocarcinoma (pSi pN2 sH0 pP1 cM0). For stage IV colon cancer, he received six cycles of systemic chemotherapy with 500 mg/m² 5-fluorouracil and 250 mg/m² leucovorin. He was free from symptoms for 6 months with no additional chemotherapy. In November 2005, he presented with progressive dysphagia. Esophagography showed marked dilatation of the upper esophagus with severe stricture of the lower esophagus (Fig. 1A). Computed tomography (CT) showed marked thickening of the esophageal wall around the stricture (Fig. 2A). Endoscopic study showed a severe stenosis 35 cm from the incisors but did not show any mucosal...
irregularity (Fig. 1C). Mucosal biopsy specimens showed normal squamous epithelium without tumor cells, suggesting luminal stenosis due to a submucosal tumor, although the histological diagnosis was not confirmed. Because the prognosis of his cecal cancer was considered poor, we performed endoscopic placement of a self-expanding metal stent for palliation. His symptoms disappeared and he was healthy for another 10 months without any sign of cancer recurrence. In August 2006, however, he developed dysphagia again and barium study and CT scan demonstrated enlargement of the esophageal tumor, causing severe stenosis at the location of the prosthesis (Figs 1B and 2C). Esophagoscopy revealed a circumferential stricture around the esophageal stent that hampered passing of the endoscope (Fig. 1D). Pathological examination of multiple biopsy specimens also showed normal esophageal mucosa. Positron emission tomography (PET) scan showed marked uptake by the esophageal tumor, but suggested no recurrence of cecal cancer at other sites. Laboratory data revealed a normal level of carcinoembryonic antigen (CEA) (4.1 U/ml) and a slightly elevated level of CA19—9 (39 U/ml) Since his general condition was excellent at this time point, we decided to perform surgery for palliation. At laparotomy, no recurrence of cecal cancer was apparent in the abdominal cavity. The tumor of the lower esophagus markedly invaded the pericardium and descending aorta. Partial resection of the lower esophagus was performed and a gullet was constructed from the stomach. Via a left neck incision, the cervical esophagus was explored and the gullet was used to bypass the remaining thoracic esophagus through a subcutaneous tunnel route. Pathological study showed that the resected specimen was moderately differentiated adenocarcinoma (Fig. 3), suggesting a metastatic lesion from the cecal cancer. Excised lymph nodes were negative for metastasis. He was discharged on October 2006, followed by systemic chemotherapy with FOLFOX4 regimen (600 mg/m² 5Fu, 200 mg/m² leucovorin, 85 mg/m² oxaliplatin). He was well with no apparent symptoms 6 months after surgery.

DISCUSSION

Since Gross and Freedman first reported a case of metastatic esophageal cancer from the prostate (6), many reports have shown the esophagus to be a frequent metastatic site from a variety of malignancies such as breast (7), lung (8), ovary (9), liver (10) and others (11–14). Autopsy studies have shown that the overall incidence of esophageal metastases in cancer patients is 3–6.1%, with breast and lung being the most common primary tumor-bearing organs (3,5). However, the rate of esophageal metastasis in patients with colorectal cancer has not been documented in spite of the high incidence of colorectal cancer. In a literature search, esophageal metastasis from rectal cancer has been described only in two brief reports (1,2). These two cases were pathologically diagnosed as mucinous and poorly differentiated adenocarcinoma. Our case was diagnosed as moderately differentiated type, suggesting that esophageal metastasis from colorectal cancer, especially differentiated type, is extremely rare.

The route of esophageal metastasis is considered to be lymphatic or hematogenous spread from a distant primary
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This patient had peritoneal metastasis and nodal metastasis at the initial colectomy. However, the nodal involvement was localized and was completely resected by conventional D2 lymphadenectomy. Moreover, lymph nodes in the mediastinal area are rarely involved in colorectal cancer without evident metastases in the abdominal para-aortic nodes (15). In our case, therefore, it is most likely that the cecal cancer developed a secondary metastasis in the esophageal wall through hematogenous spread.

Metastatic esophageal cancers are usually located in the submucosal area causing progressive dysphagia (5,16). Esophagography and endoscopy show the presence of severe luminal stricture with normal overlying mucosa, often making histological diagnosis difficult (16). CT scan demonstrates concentric thickening of the esophageal wall over the stricture without an apparent extrinsic mass (16,17). In our patient, biopsy specimens showed normal esophageal mucosa without malignant cells, but he showed all of the above radiologic and endoscopic features, suggesting the typical type of metastatic esophageal cancer.

Standard treatment for metastatic esophageal cancer has not yet been established. However, as the majority of such patients already have advanced malignant disease or metastases at multiple sites, systemic chemotherapy and/or local radiation is usually considered the first choice. This patient had advanced cecal cancer with peritoneal metastasis and metastatic or other types of submucosal tumor were suspected from the radiological findings. However, a pathological diagnosis was not obtained with repeated biopsy examinations and we could not determine the most suitable chemotherapeutic regimen.

Since he had a strong desire to eat, we attempted endoscopic placement of an esophageal stent at the initial treatment for palliation. Recent endoscopic placement of various stents has shown great potential to palliate dysphagia due to a malignant lesion in the esophagus (18–21). In our case, we used a self-expanding metal stent that we have used most frequently, since, even at this time, we considered that his prognosis was not so good. The stent was effective for 10 months, but ingrowth of the tumor caused re-stricture of the esophagus. Recent reports have suggested that a membrane-covered expandable metal stent significantly decreases the rate of tumor ingrowth (21,22). Therefore, the use of a covered rather than a bare stent may bring better palliation to prolong the period of esophageal patency.

Fortunately, the growth of peritoneal metastasis of cecal cancer was completely suppressed for 2 years without additional chemotherapy, and PET scan showed no apparent metastasis in the peritoneum. This suggests a complete response of peritoneal metastasis to chemotherapy, which is not so unusual in colorectal cancer (23,24). Then, we chose surgical palliation rather than re-stenting. At the second laparotomy, the esophageal tumor was pathologically diagnosed as adenocarcinoma which was consistent with metastasis of cecal cancer. Although no recurrence was evident at this time point, the tumor markedly invaded the surrounding tissues. Since total resection of the tumor was expected to cause unacceptable stress to the patient, we performed bypass surgery for palliation.

In retrospective consideration, complete resection of the esophageal metastases might have been successfully performed instead of stent placement in our case. Surgical resection of metastatic lesions from other organs cannot generally be justified in patients with esophageal metastases because of the lack of evident clinical benefit as well as high morbidity. However, recent reports have shown that esophagectomy can provide excellent palliation and long-term survival in certain cases without metastases to other sites (5,7–9,11,25–28). The prognosis of these patients varied from 7 months to 14 years after esophagectomy, and appeared to be dependent on the biological characteristics of the primary malignancy. From these results together with our experience, aggressive surgery might be considered as a therapeutic procedure for metastatic esophageal cancer, as long as the primary tumor is satisfactorily controlled.

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References


