Gastroesophageal tumor embolization to the popliteal arteries

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Arterial tumor embolization is an unusual cause of acute arterial occlusion and is reported primarily in the oncologic literature. We report a case of acute bilateral popliteal artery emboli from adenocarcinoma of the distal esophagus. Arterial tumor emboli are infrequent but should be considered in the differential diagnosis by the vascular surgeon caring for patients with malignancy. (J Vasc Surg 1997;26:333-6.)

Acute arterial occlusion as a result of embolization from a malignant tumor is rare. When it does occur, however, it is most commonly from bronchogenic carcinoma through direct invasion of pulmonary veins[1-4] or from benign cardiac myxomas.[5-8] Tumor arterial emboli have also been described in conjunction with osteogenic sarcoma [9] and testicular teratoma.[10]

Reports of arterial embolization of tumors have appeared mainly in the oncologic literature. We recently treated a patient with adenocarcinoma of the distal esophagus who underwent a transhiatal esophagectomy and, in the postoperative period, was found to have acute bilateral lower extremity ischemia. Subsequent embolectomies were performed on both popliteal arteries, and the embolic material removed was consistent with the gastric adenocarcinoma. It is the purpose of this report to alert vascular surgeons to this clinical problem.

CASE REPORT

The patient was a 68-year-old man whose chief complaint was dysphagia of 4 months duration. He lost 10 pounds of weight because of his symptoms. His history was significant for non–insulin dependent diabetes mellitus and amputation of the left first great toe for osteomyelitis. He appeared as a thin, pale, elderly man in no acute distress. The physical examination was unremarkable except for the rectal examination, which revealed stool that was positive for occult blood. He had a regular cardiac rhythm.

The patient's hemoglobin level on admission was 8.9 g/dl. Results of liver function tests were within normal limits. Esophagogastroduodenoscopy revealed a tumor of the distal esophagus, and a biopsy was performed. The pathologic diagnosis was gastric adenocarcinoma. A computed tomographic scan revealed a large tumor mass of the distal esophagus with indistinct tissue planes between the pericardium and the mediastinum. There appeared to be preservation of tissue planes between the pericardium and the pulmonary hilum.

After this evaluation, the patient underwent transhiatal esophageal resection with cervical esophagogastrostomy. The tumor was noted to be adherent to the pericardium, and the celiac nodes were enlarged. There was extensive manipulation of the tumor at the time of surgery. The estimated blood loss for the procedure was 1000 ml. Subsequent pathologic diagnosis confirmed T_4, N_1, and M_0 stage III adenocarcinoma, with two of nine regional nodes testing positive for tumor. The tumor was a large lesion that measured approximately 9.6 cm in length and was completely circumferential. The high-grade adenocarcinoma penetrated through the muscularis into the surrounding fat. Lymphatic channels and extramural veins contained tumor (Fig. 1).

The patient initially appeared to be doing well, but on the third day after surgery he complained of pain in his left foot. The left leg at that time was noted to be acutely and chronically ischemic with pallor, atrophy of all secondary skin appendages, and a prior great toe amputation. The right calf was also noted to be swollen and tender. Motor function and sensation to light touch was intact bilaterally. The femoral pulses were palpable but no distal pulses were palpable. Monophasic Doppler signals were present in the right and left dorsalis pedis arteries. The initial clinical impression was acute exacerbation of chronic arterial oc-

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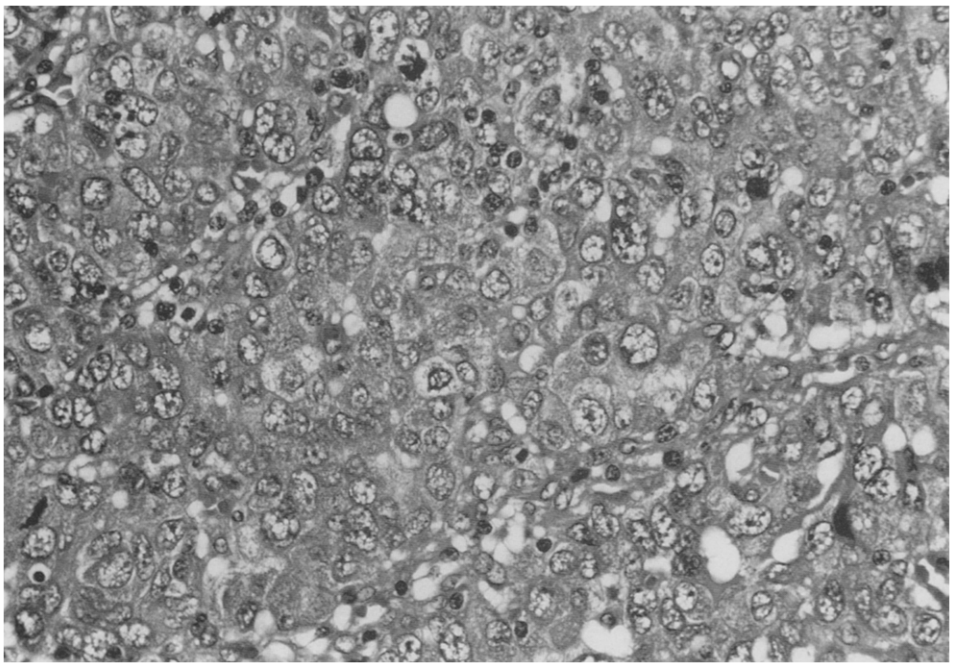

Fig. 1. Representative area of poorly differentiated adenocarcinoma of the esophagus (hematoxylin and eosin; original magnification, ×400).

clusive disease in the left leg and deep venous thrombosis in the right leg. The patient was given heparin, and a venous duplex ultrasound of the legs was obtained. The results of the study were negative for deep venous thrombosis in both the right and left legs. Angiography of the legs was obtained, and this revealed bilateral popliteal thrombosis that appeared acute in nature. The patient was taken immediately to the operating room, and bilateral popliteal artery exploration was performed. Each of the vessels of the popliteal trifurcation was explored, and thrombus was extracted from the distal popliteal artery as well as each of the trifurcation vessels. The thrombus itself appeared grossly unremarkable and was submitted for pathologic review. A four-compartment fasciotomy was performed in the right leg, and bilateral completion angiograms demonstrated patency of the trifurcation vessels into the feet.

After surgery, the left foot demarcated progressive gangrene of three of the four remaining toes. A subsequent transmetatarsal amputation was performed, and this healed primarily. On the right side, the fasciotomy wounds were debrided and subsequently closed with split-thickness skin grafts. The remainder of the patient's hospital course was uneventful. The patient was placed on warfarin therapy for 6 months. The patient died of metastatic carcinoma with liver metastasis 18 months after surgery. There was no evidence of further emboli. An autopsy was not obtained.

Pathologic examination of the thrombotic material removed from both the right and left popliteal arteries at the time of surgery demonstrated adenocarcinoma. The thrombus contained some degenerative cells as well as collections of viable tumor cells. Immunohistochemical stains for cytokeratin were performed on the primary tumor and the tumor within the blood clot. Both stained positive. The combination of cytokeratin positivity and the histologic similarity of the tumor cells on hematoxylin and eosin staining was convincing evidence that the tumor in the blood clot removed from the popliteal arteries was the same as the esophageal carcinoma (Fig. 2).

DISCUSSION

Arterial embolization of tumors is rare. Embolization from benign cardiac myxomas is well described,[5,6,11,12] and the cerebral circulation is a frequent endpoint location.[7,8] Pulmonary malignancies may embolize to the peripheral circulation during surgical excision. To our knowledge, our report represents the only example of gastroesophageal tumor embolization to the lower extremities reported in the literature. Chandler,[2] in 1993, reviewed 60 cases in the literature of documented arterial tumor emboli, excluding atrial myxomas. He noted that the most common source of arterial tumor emboli was from primary lung tumors (41%), followed by sarcomas (28%). The most common endpoint locations of tumor emboli were to the lower extremities (29%), carotid and cerebral circulation (24%), aorta (23%), visceral vessels (6%), coronary vessels (5%), and upper

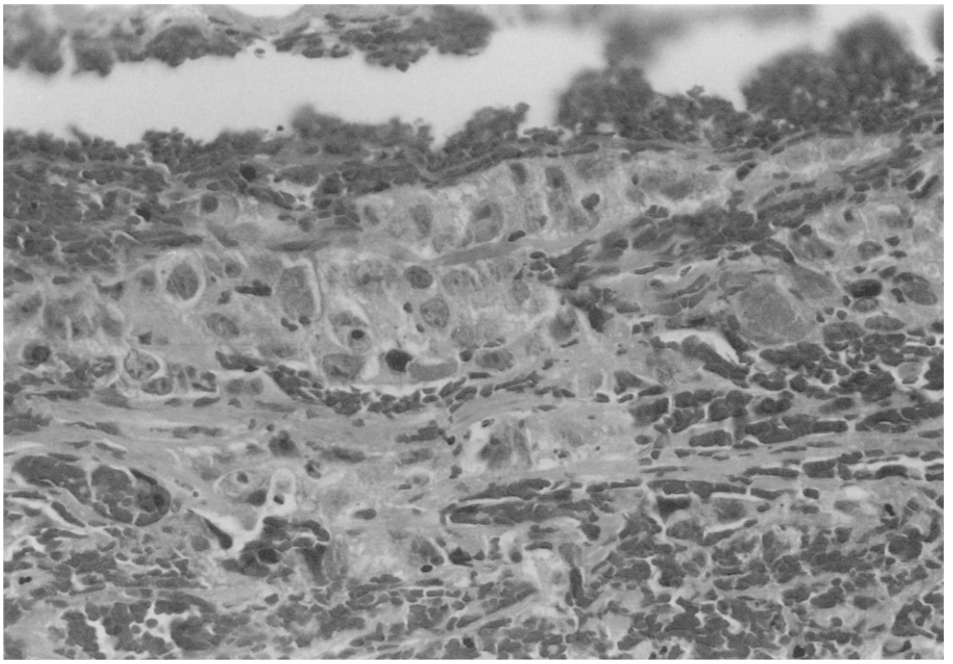

Fig. 2. Blood clot from popliteal artery contained tumor cells similar to the esophageal adenocarcinoma. (hematoxylin and eosin; original magnification, ×400).

extremities (5%).[2] Manipulation of the lung and tumor-involved pulmonary veins may lead to release of malignant tissue into the systemic circulation.[1,2] In addition, sarcomas have been reported to cause arterial emboli.[11] Balas et al.[12] described a colonic adenocarcinoma that invaded the aorta and caused occlusion by either direct extension or embolization. Wilms' tumor may cause venous emboli in pediatric patients.[13] The growth of these tumors by intravascular extension may lead to embolization spontaneously or at the time of resection.

The extension of tumor into veins and subsequent embolization is more commonly reported.[14] Examples of renal cell carcinoma[15] and Wilms' tumor[16] extending into the renal veins, vena cava, and right side of the heart have been described. Sarcomas are also known to metastasize through venous invasion. Both have produced pulmonary embolism.[2,14]

The source of the embolic tissue in our patient remained elusive. Based on the physical findings at the time the patient was seen by the vascular surgeon (swelling of right calf muscle), the emboli undoubtedly occurred before the third postoperative day and most likely at the time of the esophagogastric resection. We have no data to indicate the exact time. The computed tomographic scan showed no evidence of pulmonary or cardiac involvement, and the preoperative chest roentgenogram and echocardiogram were unremarkable. Alternative explanations include direct invasion of the left ventricle, pulmonary venous invasion,[1,2,17] or a paradoxical embolus.[10] We have no evidence for which of these mechanisms was the cause in this patient. An aortogram may have shown direct aortic invasion but was not obtained. With the healing esophagogastric anastomosis, transesophageal echocardiography was contraindicated after surgery. A two-dimensional transthoracic echocardiogram performed before surgery showed normal ventricular function and no intracardiac masses. There was no pericardial effusion observed. Arterial emboli from malignancies are infrequent but must be considered especially in the context of resection of tumors that may encroach on the pulmonary veins. If this clinical entity is considered early in the differential diagnosis, prompt treatment can lead to limb salvage and a successful clinical outcome.

It is the purpose of this report to alert vascular surgeons to this unusual cause of acute arterial occlusion in the postoperative period of a patient who has undergone operation for malignancy. This entity is more frequently described in the oncologic literature, and citations in vascular journals are remarkably sparse. Although arterial tumor embolism is rare, the vascular surgeon must have a high index of suspicion. Computed tomographic scans of the lungs and two-dimensional echocardiography should be considered

to delineate the more common sources for arterial tumor emboli.

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