Cutaneous metastasis of esophageal adenocarcinoma presenting as telangiectatic metastatic carcinoma demonstrating intravascular proliferation of tumor emboli

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INTRODUCTION
Cutaneous metastases from esophageal adenocarcinoma are rare, often presenting as painless nodules and tumors.1-3 Here, we describe an unusual case of cutaneous metastasis from esophageal adenocarcinoma presenting as a telangiectatic metastatic carcinoma, which must be differentiated from similar-appearing conditions such as vascular or lymphatic malformations. This case provides histopathologic corroboration for inflammation associated with cutaneous metastases and provides insight concerning the habits of cutaneous metastases from esophageal adenocarcinoma.

CASE PRESENTATION
A 66-year-old man with esophageal adenocarcinoma metastatic to the duodenum and lymph nodes of the neck and left axilla presented to dermatology clinic with a rash on the anterior aspect of the left shoulder. The rash had increased in size during the last 6 months and was initially not pruritic or painful. A pink to violaceous annular smooth plaque measuring approximately 15 × 12 cm was present over the anterior aspect of the left shoulder, with irregular borders and telangiectases on dermoscopy (Fig 1, A). In the left axilla, multiple grouped and contiguous pink to red vesicular papules were present, similar to the “frog spawn” appearance of lymphangioma circumscriptum (Fig 1, B). Differential diagnosis included acquired venous or lymphatic malformation versus cutaneous metastasis. A 4-mm punch biopsy was obtained. Histopathology revealed telangiectatic metastatic carcinoma consistent with cutaneous metastasis of esophageal adenocarcinoma (Fig 1, C). The lesion demonstrated metastatic emboli within the dermis and focal areas of lymphocytic aggregation. Tumor emboli were present within dilated blood vessels (Fig 1, D), suggesting hematogenous dissemination. The patient proceeded to develop intense pruritus at the site of his cutaneous metastasis during the ensuing months. Adequate trial of a high-potency topical corticosteroid was ineffective. The patient began receiving topical lidocaine and petroleum jelly, which has been helpful to mitigate his pruritus.

The patient had received a diagnosis of metastatic esophageal adenocarcinoma 31 months before after being admitted for a pulmonary embolism. After imaging, multiple partially occlusive pulmonary emboli and prominent cervical lymph nodes were identified. The cervical lymph nodes were biopsied and exhibited moderately differentiated metastatic adenocarcinoma of unknown primary origin. Immunohistochemical analysis of nodal metastases was positive for cytokeratin 7 and 20, weakly positive for caudal-related homeobox 2, and negative for thyroid transcription factor 1, napsin, and GATA binding protein 3, suggesting a primary of gastrointestinal origin. Subsequent endoscopy revealed invasive, moderately differentiated

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esophageal adenocarcinoma and mucosal angiolymphatic invasion of the small bowel. The patient was treated with folinic acid, 5-fluorouracil, and oxaliplatin and trastuzumab. Follow-up imaging demonstrated initial therapeutic response. Tumor was found to be human epidermal growth factor receptor 2 positive, and after progression of disease, the patient was treated with 5-fluorouracil and trastuzumab.

**DISCUSSION**

To our knowledge, this is the first report of cutaneous esophageal adenocarcinoma presenting as telangiectatic metastatic carcinoma, although squamous cell carcinoma of the esophagus has been reported with a similar-appearing papular presentation. Furthermore, our patient developed intense pruritus at the site of his cutaneous metastasis, which is unusual. Although uncommon, this presentation is an important consideration in patients with a history of esophageal adenocarcinoma.

Some tumor emboli did not extravasate but contained mitotically active cells (Fig 1, D). The cascade of events precipitating a metastatic colony is a topic of ongoing discussion. One model describes a process during which single tumor cells or aggregates circulate, lodge in organ capillaries, extravasate, and proliferate to establish a metastatic colony. Cells within the present tumor embolus formed mitotic division figures (Fig 1, D), demonstrating that proliferation of disseminated tumor cells may...
occur without extravasation. This observation lends support for schemas in which proliferation of tumor cells and establishment of a metastatic colony may occur preceding or in the absence of extravasation. Clinically, this could support rationale for cell-cycle-inhibiting antitumor agents in preventing proliferation of disseminated but clinically undetectable cancer.

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