Rapid progression of Barrett's esophagus into adenocarcinoma in a combined lung and kidney transplant recipient

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The incidence of cancer in solid organ transplant recipients is significantly greater than that of the general population. This increase is related to the intensity and duration of the immunosuppressive therapy. We herein present a rare case in which Barrett's esophagus (BE) rapidly progressed into invasive adenocarcinoma after a patient with scleroderma underwent combined lung and kidney transplantation.

CLINICAL SUMMARY

A 56-year-old male patient with scleroderma and pulmonary fibrosis (IPF) was referred to our lung transplant program. The patient presented with progressive dyspnea over a 2-year period. While on treatment, his condition deteriorated progressively until he became oxygen dependent.

The pretransplant workup revealed a forced expiratory volume in 1 second of 53%, forced vital capacity of 47%, and diffusing capacity for carbon monoxide of 13%. Computed tomography (CT) scan showed pulmonary infiltrates consistent with IPF. An upper gastrointestinal endoscopy showed the presence of BE at 29 cm. Biopsies confirmed BE with metaplasia but no evidence of dysplasia. The renal function was moderately impaired with a serum creatinine of 141 and a creatinine clearance of 36 mL/min. The patient had a thorough evaluation by the transplant nephrologists, who confirmed the renal insufficiency. The patient was thus listed for a combined double lung and kidney transplantation. While on the waiting list, the chest CT scan was repeated 3 times at 6-month intervals and remained unremarkable.

After a 21-month wait, the recipient underwent a sequential double-lung transplant on cardiopulmonary bypass. Once stabilized hemodynamically, a heterotopic kidney transplant was performed.

Postoperatively, immunosuppression consisted of basiliximab for induction and methylprednisolone/prednisone, tacrolimus, and mycophenolate mofetil as maintenance therapy. After being extubated on postoperative day 4, the patient developed infiltrates in both lungs and consolidation of his left lower lobe, and he became ventilator dependent. On postoperative day 14, a tracheostomy was carried out and the patient was then successfully weaned from the ventilator. Seven weeks postoperatively, blood was found in the nasogastric tube. Endoscopy revealed a large mass at the distal esophagus, which on biopsy was confirmed to be a poorly differentiated adenocarcinoma. CT scan showed an 8-cm mass of the distal esophagus (Figure 1). Due to the patient's overall condition and the apparent aggressiveness of the esophageal cancer, only comfort measures were instituted. The patient died 8 weeks after his transplantation.

DISCUSSION

The incidence of cancer in transplant recipients is increased secondary to the immunosuppressive therapy. This is especially noteworthy in patients with premalignant conditions. Reports have shown that in renal transplant recipients, the incidence of skin tumors is increased 30-fold,[1] and posttransplant lymphoproliferative disorder predominates in lung transplant recipients, most likely as a result of the more intense immunosuppression.[2]

BE is a well-documented premalignant lesion. Endoscopic surveillance is generally recommended every 18 to 24 months, with follow-up at shorter intervals if there is a concern for dysplasia on histology. Once high-grade dysplasia develops, patients should be considered for esophagectomy, unless they are poor surgical candidates.

Immunosuppression may play an important role in the malignant transformation of BE into adenocarcinoma as

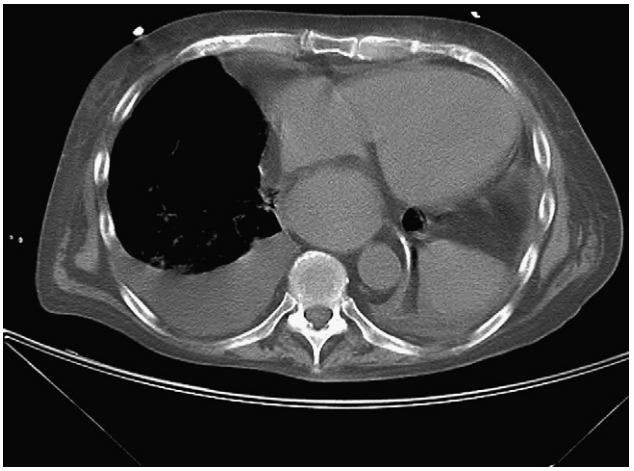

FIGURE 1. Computed tomography scan at the level of the lower chest showing large mass arising from the distal esophagus.

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suggested by Oka and colleagues.[3] The authors found that patients with BE have significant suppression of T-cell and B-cell function as well as interlukin-2 production. In addition, Hsu and associates[4] reported a case of metastatic esophageal cancer arising in BE in a patient with chronic lymphocytic leukemia with documented B-cell dysfunction. In the present report, the use of induction therapy followed by intense immunosuppression certainly placed the BE at increased risk for malignant transformation.

It is possible that the metaplasia in this patient may have progressed into high-grade dysplasia prior to the transplantation, taking into consideration the 22-month gap between the diagnosis of metaplasia and the transplant. Endoscopic surveillance during the waiting period would have permitted an early diagnosis; however, this was not possible in view of the patient's poor pulmonary status.

Only one case of rapid progression of BE to adenocarcinoma has been reported after liver transplantation,[5] and to our knowledge, the present case is the first reported after lung transplantation.

It becomes essential to ask whether the diagnosis of BE in pretransplant patients precludes them from being transplanted. Although there is no clear answer, there is no doubt that these patients should undergo upper gastrointestinal endoscopy on a regular basis prior to their transplant. Close surveillance after transplantation is certainly warranted to detect signs of dysplasia. Should high-grade dysplasia occur, adjustment of the immunosuppressive therapy is necessary and ablation or resection of the dysplastic mucosa should be considered.

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Nonobstructive ectopic accessory mitral valve tissue in association with left ventricular apical aneurysm

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Accessory mitral valve tissue is a rare congenital cardiac anomaly, an uncommon cause of left ventricular outflow tract (LVOT) obstruction, and usually diagnosed during childhood. We report a case of a nonobstructive accessory mitral valve in a 33-year-old woman who was evaluated for systemic embolization that resulted in right hemiplegia. A mass lesion was diagnosed in the apical region of the left ventricular (LV) cavity by means of transthoracic and transesophageal echocardiography. A cardiac magnetic resonance image (MRI) showed a mass lesion in the LV cavity and LV apical aneurysm. The patient had undergone successful excision of the mass lesion, which was accessory mitral valve tissue, and linear repair of the LV aneurysm.

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CLINICAL SUMMARY

A 33-year-old woman who recovered from right hemiplegia was referred to our center with a diagnosis of an intracavitory mass lesion of the left ventricle. On physical examination, she had a regular pulse, with a rate of 80 beats/min and a blood pressure of 126/80 mm Hg. Her first and second heart sounds were normal, and there was no murmur. An electrocardiogram showed sinus rhythm. She was evaluated with transthoracic and transesophageal 2-dimensional echocardiography and Doppler scanning, which showed a mass in the LV cavity, and there was no gradient across the LVOT. A cardiac MRI demonstrated a mass lesion in the LV cavity attached to the distal interventricular septum and LV apical aneurysm (Figure 1, *A*). A coronary angiogram showed normal epicardial coronary arteries.

She underwent excision of accessory mitral valve tissue and linear repair of the LV aneurysm through a standard median sternotomy during cardiopulmonary bypass. The