LETTER TO THE EDITOR

Pulmonary Tumor Thrombotic Microangiopathy in a Patient of Gastric Carcinoma: A Rare Entity

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Keywords: Acute hypoxemic respiratory failure, High-resolution computed tomography chest, Malignancy, Pulmonary hypertension. *Indian Journal of Critical Care Medicine* (2022): 10.5005/jp-journals-10071-24230

Dear Sir

Pulmonary tumor thrombotic microangiopathy (PTTM) is a rapidly progressing pulmonary complication of an underlying malignant disease. Tumor embolization to pulmonary vasculature induces thrombotic microangiopathy resulting in pulmonary hypertension. Clinically, it manifests as dyspnea and hypoxemia which is usually rapid in onset and often fatal. Here, we discuss a rare entity of gastric carcinoma associated with PTTM.

A 42-year-old female admitted to the emergency department with chief complaint of shortness of breath of 10-day duration and generalized weakness for 15-20 days. On examination, she was tachypneic and dyspneic with room air saturation of 88%. Arterial blood gas (ABG) analysis showed hypoxemia with type I respiratory failure. She was initially put on NIV and later intubated and put on mechanical ventilation. Chest skiagram showed bilateral consolidation and right-side pleural effusion. Therapeutic thoracocentesis of 650 mL of straw-colored pleural fluid was done and sent for cytological and biochemical evaluation. Antibiotics and other supportive therapies were started after sending blood and tracheal aspirate culture. Laboratory investigations showed neutrophilic leukocytosis and raised D-dimer. Pleural fluid adenosine deaminase (ADA) was normal, and cartridge-based nucleic acid amplification test (CBNAAT) was also negative. Both tracheal aspirate culture and bronchoalveolar lavage (BAL) fluid culture did not show any growth.

Her echocardiography revealed dilated right atrium (RA) and right ventricle (RV) with moderate tricuspid regurgitation. However, pulmonary CT angiogram did not reveal any thrombus.

High-resolution computed tomography (HRCT) thorax showed ground-glass opacities (GGO) and septal thickening (Fig. 1) mixed with air space consolidation in both lungs, with extensive peribronchiolar and centrilobular soft tissue nodules with tree-in-bud pattern (Fig. 2: white arrow).

She had one episode of hematemesis on Day 20 of hospitalization. Esophagogastroduodenoscopy showed an ulcero-proliferative growth of approximate size of 10×10 cm in gastric body of stomach and biopsy confirmed it to be adenocarcinoma. Contrast-enhanced computed tomography (CECT) of the abdomen confirmed it to be a gastric mass with ovarian deposits. So, an antemortem diagnosis of PTTM was kept in view of pulmonary hypertension in the absence of thrombus and classical radiological findings like centrilobular nodularity with GGO and interlobular septal thickening especially in the context of underlying gastric malignancy.

Medical oncology consultation was done, and chemotherapy was planned. However, due to financial issues, relatives shifted her to a government hospital.

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How to cite this article: Choudhury S, Mishra SB, Panda S. Pulmonary Tumor Thrombotic Microangiopathy in a Patient of Gastric Carcinoma: A Rare Entity. Indian J Crit Care Med 2022;26(6):763–764.

Source of support: Nil
Conflict of interest: None

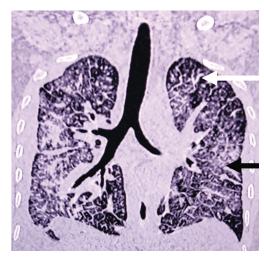


Fig. 1: HRCT coronal image of lung showing interlobular septal thickening (white arrow) and ground-glass opacity (black arrow)

This case demonstrates the diagnostic challenges due to nonspecific symptoms and absence of known cancer at initial clinical presentation. It also highlights the need for a high index of suspicion in patients with rapidly progressive respiratory symptoms and pulmonary artery hypertension on imaging especially in the context of a known or suspected malignancy. In many cases, patients present with acute symptoms of pulmonary hypertension causing shortness of breath and right heart failure leading to death in a few days.²

This disorder is found in various malignancies, but most frequently, it is associated with gastric adenocarcinoma as seen in

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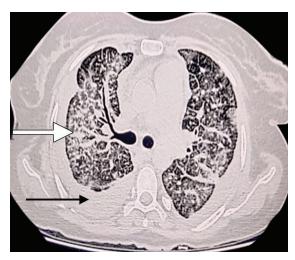


Fig. 2: HRCT axial image of lung showing tree-in-bud pattern (white arrow) and right pleural effusion (black arrow)

our patient.² The median age of patients diagnosed with gastric carcinoma is 50–70 years, but gastric cancer-associated PTTM has also been reported in the younger age-group.³ This finding is also similar in our case. The most common pulmonary manifestations among patients with cancer are thromboembolism, metastasis, pleural effusion, and lymphangitic carcinomatosis. Most frequent laboratory parameters include an increased level of D-dimer, anemia, and thrombocytopenia.⁴

It has been hypothesized that tumor cells express vascular endothelial growth factor (VEGF) and platelet-derived growth factor (PDGF) which cause activation of coagulation cascade and inflammation that leads to small vessel thrombosis and fibrocellular intimal thickening and remodeling.⁵

While early diagnosis and treatment are desirable, antemortem diagnosis is exceedingly complex. It highlights the

importance of high index of suspicion in patients with rapidly progressive respiratory symptoms and chest CT suggestive of pulmonary hypertension and classical findings of inflammatory bronchiolitis, especially in the context of a known or suspected malignancy.

Our case highlights the significance of high index of suspicion of PPTM in patients of known or suspected malignancy when present with clinical features of rapidly progressive respiratory symptoms along with radiological features of inflammatory bronchiolitis and pulmonary hypertension on CT imaging.

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