STEMI due to Presumed Paradoxical Embolism Diagnosed on CT in a Patient with Metastatic Pancreatic Adenocarcinoma

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Introduction

Arterial thromboembolic events have been described in patients with pancreatic cancer, with an estimated incidence of 2-5%[1]. The etiology of these events is usually nonbacterial thrombotic endocarditis (NBTE), tumor cell emboli, or paradoxical embolus from deep venous thrombosis (DVT) through a patent foramen ovale[2]. While these events are rare, when they do occur in patients with pancreatic cancer, they can present as myocardial infarction. Traditionally, clinical diagnosis of a myocardial infarction (MI) is confirmed by a rise in cardiac troponin values accompanied by new ischemic EKG changes. Diagnosis can also be supported by evidence of new loss of viable myocardium including new regional wall motion abnormality in a pattern consistent with an ischemic etiology[3]. While CT is not part of the usual workup of MI, acute MI has presented as hypoattenuation of the myocardium on CT scans performed for other indications, including pulmonary embolism and aortic dissection[4].

Case Description

A 50-year-old man with a past medical history of metastatic pancreatic cancer s/p chemotherapy and biliary stenting was hospitalized for treatment of *Klebsiella pneumoniae* bacteremia. He also had extensive bilateral lower extremity deep venous thromboembolism, for which he was being treated with Eliquis 5 mg twice daily. On day 7 of hospitalization, the patient developed acute onset left upper quadrant pain that was reproducible on palpation, accompanied by a worsening leukocytosis (WBC 23,700/uL from 16,400/uL two days prior). CT chest/abdomen/pelvis with contrast was performed to assess for abdominal abscess vs. hematoma. CT incidentally demonstrated mid-ventricular, inferior septum, and inferior papillary muscle hypoattenuation representing myocardial infarction. Additionally, there were hypoattenuating lesions of the spleen and liver compatible with infarction, as well as right upper lobar and middle lobe segmental pulmonary thromboembolism seen in the pulmonary arterial phase. Stat EKG was performed which revealed ST segment elevations in the inferior and lateral leads, with reciprocal changes in the anterior leads. High-sensitivity troponin was 5,956 pg/mL (< 20 pg/mL). At this time, the patient was hemodynamically stable without chest pain or dyspnea. Given unknown timing of infarct (patient asymptomatic), his hemodynamic stability, and poor oncological prognosis, cardiac catheterization was deferred. He was treated medically with transition from apixaban to therapeutic heparin infusion and dual antiplatelet therapy. Echocardiogram was performed with bubble study positive for a right to left atrial shunt, consistent with patent foramen ovale. Given the patient's extensive venous clot burden and evidence of multiorgan infarction now suspected to be due to paradoxical embolism in setting of DOAC failure, an IVC filter was placed, and the patient was transitioned to enoxaparin subcutaneously.

Discussion

While paradoxical coronary artery embolism is rare, it is known to cause myocardial infarction, especially in patients with hypercoagulable state due to malignancy, in the absence of other major atherosclerotic coronary artery disease risk factors[5], and this case adds to that literature. In addition to STEMI, this patient also had evidence of arterial thromboembolism causing infarction in multiple other organs, a clinical picture that should raise suspicion for PFO. Presumptive diagnosis can be made by echocardiogram demonstrating veno-arterial communication, with identification of a venous source of embolus and lack of thrombi in the left heart[6]. Furthermore, this case illustrates the ability of CT to detect acute MI. CT scan has a sensitivity and specificity possibly as high as 83% and 95%, respectively,

for detecting acute MI[7]. This modality is often used in the workup of atypical chest pain and detection of ischemia can prompt timely workup and treatment.

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