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Atypical Case of Recurrent Saddle Pulmonary Embolism with Associated Chronic Hypoxia

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Abstract:

A saddle pulmonary Embolism (PE) represents a blood clot within the pulmonary artery vasculature that lodges itself between both the left and right pulmonary artery. They can be described as being: massive or sub-massive. Massive PE's cause large clot burden that puts the patient risk for sudden hemodynamic collapse, while sub-massive saddle PE's typically do not cause hemodynamic collapse nor significant cardiac effects, such as right heart dysfunction. As clinicians, we need to be vigilant about whether a patient is placed into that criteria of sub-massive or massive and act quickly to start management that could be life saving [3].

Case Presentation:

A 62 year-old male with no significant pmhx presented to the ED for conversational dyspnea and unilateral left leg redness and swelling for the past three days. Patient endorsees having some dyspnea on exertion that is better when he sits and without any chest pain. States he lost his job two years ago. Denies any cardiac history and never been on a blood thinner. Patient follows up regularly with primary care for being in remission from bipolar one disorder. Patient social history is negative for smoking, alcohol use, and illicit drug use.

At presentation in the ED, patient was hypoxic required 4L nasal cannula (NC) to keep his oxygen levels stable, tachycardic 136 bpm, afebrile, blood pressure of 142/93, and a body mass index of 30.8 kg/M^2. ECG showed sinus tachycardia with some nonspecific T-wave changes in anterior leads.

Physical examination revealed non-toxic, but ill-appearing male in respiratory distress. Lung auscultation showed decreased vesicular breath sounds in bilateral lung fields. He had left lower extremity erythematous, swelling and tenderness to palpation from the posterior aspect of lower leg. Otherwise, physical examination was unremarkable.

12/30/23: CTA chest pulmonary angiography done on presentation showed saddle embolus across right and left pulmonary arteries extending into both lower pulmonary arteries (Figure 1). In addition, interventricular septum flattening, right ventricle dilation, and right atrial enlargement was noted. Deep Venous Thrombosis (DVT) Ultrasound of left leg was completed and showed extensive DVT starting in the left common femoral extending down to the femoral, popliteal, posterior tibial, and peroneal vein. Patient was immediately started on heparin drip with bolus and IR contacted for thrombectomy (Figure 2). He was then started on Eliquis 10mg po and taken to the Intensive Care Unit (ICU).

12/31/23: Patient post-thrombectomy and after downgrade to stepdown, acutely required an increase amount of oxygen through the evening maxing out on NC, non-rebreather and requiring 45L 60% FiO_2 high flow nasal canula (HFNC). Patient was immediately upgraded back to ICU.

01/01/23: Due to patient acutely becoming more hypoxic, a new CTA pulmonary angiogram was done that showed a second saddle PE. IR was immediately contact, patient switched to heparin drip and a second thrombectomy with IVC filter placement was done (Figure 3). After the procedure, patient was placed on a continuous thrombolysis catheter by IR for 24 hours. Patient still required oxygen.

Patient was placed back in ICU and oxygen requirements decreased to NC. Unfortunately, patient was unable to come off of 4L NS and remained hypoxic after treatment. Internal Jugular Swan Catheter was placed and showed unremarkable pressures in all chambers of the heart, including unremarkable pulmonary capillary wedge pressure. MUGA scan and cardiac MRI was preformed because the patient continued to be hypoxic requiring oxygen with otherwise unremarkable results. Both tests showed unremarkable results, including ejection fraction of 45-50%. Hematology workup was unremarkable, except for positive lupus anticoagulation screen. However, due to patient being on Heparin at the time of screening and the cross-reactivity, this result is invalid.

Eventually, patient was downgraded to stepdown and still continued the need for 4L NC. He was discharged on oxygen and given follow up with pulmonology and hematology outpatient for further evaluation and management.

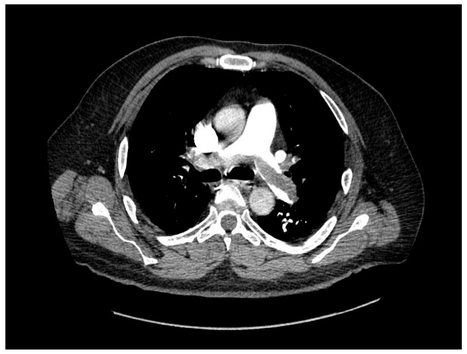

Figure 1. 12/30/2023

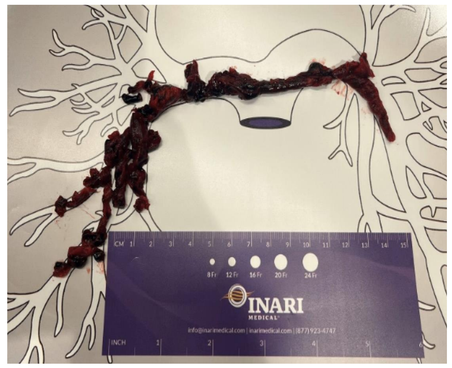

Figure 2. 12/30/2023

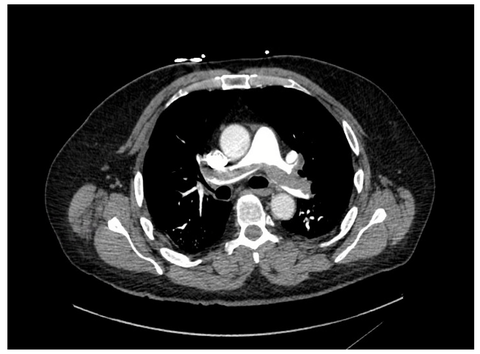

Figure 3. 01/01/2024

Figure 4. 01/01/2024

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Discussion:

PE is associated with many complications including heart failure, hypoxia, and death. The mortality of PE depends the risk type and presentation. Massive PE, which comprises hemodynamic collapse at the time of the PE has an overall mortality rate ranging from 18% to 65%. Moreover, submassive PE's have a mortality range from 5%-25% [1]. Ryu et al. pointed out that without any previous cardiopulmonary disease, in-hospital mortality rate is relatively lower and aggressive medical management may not be necessary [3]. However, this patient does not fit that mold and still continued to experience hypoxia post-aggressive management.

Diagnostic algorithms have been studied for years to determine the safest and most cost-effective way to diagnose suspected PE. The gold standard still remains CTA pulmonary angiogram for diagnosing PE. However, there has been more evidence to suggest this test has lead to more frequently suspecting PE's, with shifting to aiming to exclude PE rather than actually confirming PE [5]. These patient that present with saddle PE typically have concurrent DVT on hospital admission [6].

Management of PE is first addressed by determining risk assessment with the Pulmonary Embolism Severity Index (PESI) score. Initial medication therapy starts with anticoagulation with low molecular weight heparin (LMWH). In addition, catheter directed thrombolysis (CDT) was studied in multiple trials that shows no significant benefit on overall mortality. However, the use of CDT is still suggested in patients with persistent hemodynamic instability [7].

In acute PE, there come chronic complications that can leave patients with post-PE syndrome. During the initial stage of PE, hypoxemia and an increase in the alveolar-arterial oxygen tension gradient are the most common gas exchange abnormalities [4]. After treatment and discharge, patients may develop post-PE syndrome. Pulmonary artery pressure and right ventricular dysfunction can occur in up to 10-30% of patients after discharge. The most severe of them is chronic thromboembolic pulmonary hypertension (CTEPH) which manifests as this phenomenon of permanent changes in pulmonary gas exchange, pulmonary artery flow, and/or cardiac dysfunction. Around 0.5-4% of individuals treated for acute pulmonary embolism will develop some degree of CTEPH [8].

Conclusion:

Saddle Pulmonary Embolism is a significant cardiopulmonary and vascular disease that needs to be sought out quickly and taken action. Being aware of not only the first signs of an acute PE, but treatment and management of post-PE is just as important. Understanding the reg flag signs such as hypoxemia and acting quickly to assess these high risk patients could be live saving. The standard for imaging still remains the CTA pulmonary angiogram. The persistent or worsening hypoxia and hemodynamic changes in patients post-PE should still warrant further evaluation for possible recurrent PE. As seen in this case, a recurrent PE without significant risk factors was diagnosed efficiently by the signs of persistent and worsening hypoxia after previous intervention and clot removal. Furthermore, patient who are discharged and seen outpatient or return to the Emergency Department with continued hypoxia may be suffering from CTEPH. The understanding of post-PE syndrome needs acknowledgement for both outpatient clinical practices where previous PE patients follow up and the Emergency Department.

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