Heparin-Induced Thrombocytopenia Complicated by Pulmonary Embolism: A Diagnostic and Therapeutic Challenge

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Background

- Heparin-induced thrombocytopenia (HIT) with thrombosis is an immune mediated drug reaction that can lead to life-threatening venous thrombosis in the presence of heparin that can lead to pulmonary embolism (PE).
- PE may cause ≤300 000 deaths per year in the US, ranking high among the causes of cardiovascular mortality, behind myocardial infarction and stroke
- The annual incidence rates for PE ranges from 39–115 per 100 000 population.
- We present a case of 35-year-old female admitted for polytrauma, who was placed on prophylactic subcutaneous heparin and developed HIT. Shortly after, she suffered from bilateral PE of the main pulmonary arteries and right ventricular failure.

Case Summary

- 35 year old female with no past medical history presented with polytrauma after car accident.
- Stabilized from the trauma with stable hemodynamics
- Sudden onset acute hypoxic respiratory failure requiring endotracheal intubation.
- CT PE protocol showed bilateral acute pulmonary emboli in main pulmonary arteries (PA) bilaterally with extension into multiple lobes, with right ventricle strain.

Decision Making

- After multidisciplinary discussion with intensivist, interventional cardiologist, decision was made to intervene with EKOS and tissue plasminogen activator (tPA) infusion.
- Due to refractory hypoxia during the case, she was cannulated for veno-arterial extracorporeal membrane oxygenation (VA-ECMO) and started on heparin drip for anticoagulation.
- She underwent bilateral venous doppler that was poor quality, essentially non-diagnostic
- Five days after ECMO decannulation, a right atrial thrombus was identified, requiring mechanical circulatory support removal.
- IVC filter was placed in setting of superficial femoral artery bleed was required.
- Patient started on full dose Lovenox, however on surveillance doppler she developed new acute DVT.
- She underwent another mechanical thrombectomy and EKOS.
- Due to ongoing thrombus formation despite anticoagulation, testing for HIT was pursued.
- Platelet factor 4 (PF4) antibodies returned positive, while serotonin release assay (SRA) was negative. The patient was transitioned to Apixaban.

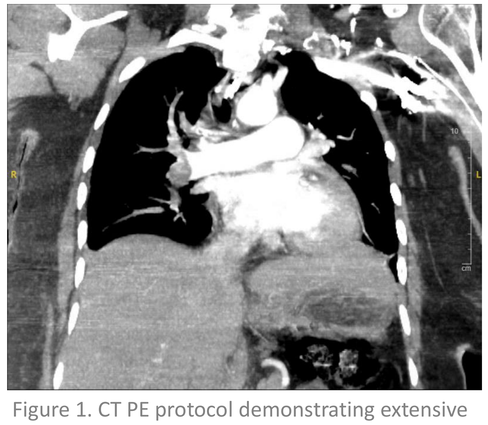

Figure 1. CT PE protocol demonstrating extensive PE in left main PA, segmental, and subsegmental branch with PA dilation. Not pictured is patients RV strain and right PA clot

Discussion

- **Heparin-induced thrombocytopenia leads to clot formation** through a cascade of cellular and molecular events.
- The American College of Chest Physicians highlights that HIT is distinguished by the formation of IgG antibodies against platelet factor 4 (PF4)–heparin complexes, which bind to Fcγlla receptors on platelets, triggering platelet activation and the release of procoagulant microparticles.
- This process results in marked thrombin generation, driving both venous and arterial thrombosis
- **Leads to thrombus formation despite a falling platelet count**, with thrombosis occurring in up to 50% of affected patients
- The risk is highest with unfractionated heparin, especially in surgical patients.

Conclusion

This case increases awareness and shows the complexity of decision making in a case of heparin induced thrombocytopenia with thrombosis leading to several pulmonary embolisms necessitating multiple interventions.