A CASE OF ARGATROBAN REFRACTORY HEPARIN INDUCED THROMBOCYTOPENIA AND THROMBOSIS WITH LEFT VENTRICULAR THROMBUS

Joclyn A. Seiler MD[1] Adam Khan Durrani[2], Mentor Ahmeti, MD[1,3]

1. Department of Surgery, University of North Dakota School of Medicine and Health Sciences, Fargo, ND

2. Medical Student, Royal College of Surgeons in Ireland – Medical University of Bahrain

3. Department of Surgery, Sanford Medical Center, Fargo, ND

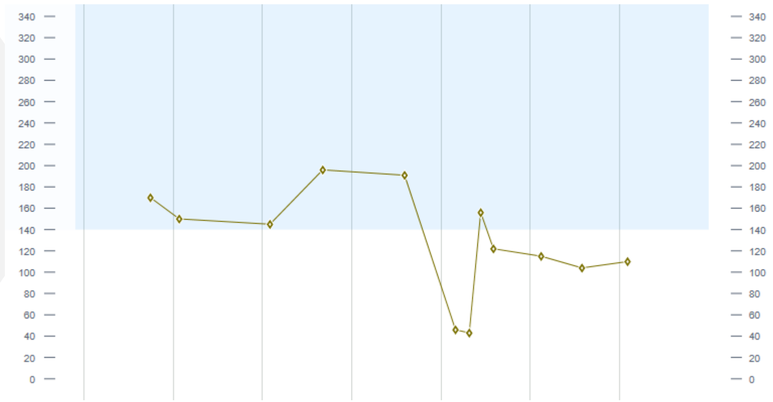
Case

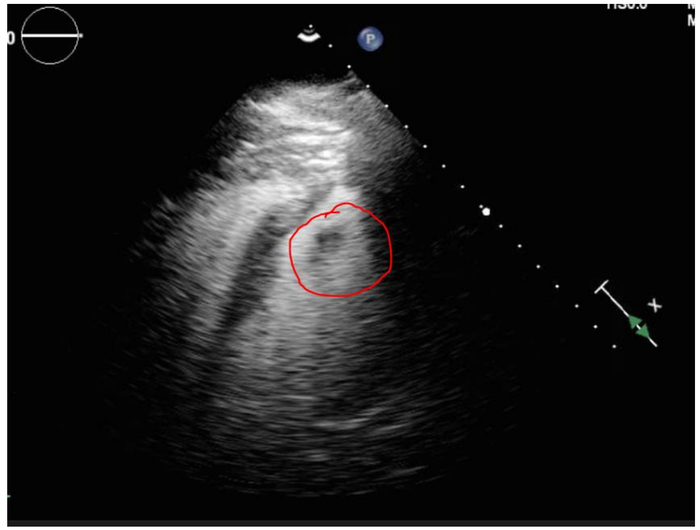
This is a report of a 69 yr old male that was initially seen for an angiogram for shortness of breath on exertion and an abnormal stress test. His cardiac angiogram demonstrated significant multivessel disease. Two days after angiogram he underwent CABG with 3 vessel bypass. The LAD was unable to be bypassed. Interventional cardiology was consulted for possible PCI of the LAD after the CABG via a hybrid procedure.

On post operative day 3 he underwent PCI of the LAD via rotational atherectomy followed by drug eluding stent placement. He was progressing well from his surgery until post operative day 6 and post procedure day 3 when he had substernal chest pain. He was taken to cath lab for angiogram. This angiogram demonstrated 100% in stent stenosis. He had a great decrease in his EF so an intra-aortic balloon pump was placed. He was transferred to the ICU for further cares and started on Ticagrelor. The IABP was removed on post procedure day 1. After removal he was noted to have a cold leg, evaluation demonstrated arterial clot. At this time there was concern for heparin induced thrombocytopenia and thrombosis, so heparin was stopped and he was started on argatroban. PF4 antibiotics were sent as well. He was taken to the OR for thrombectomy which demonstrated a white clot (consistent with HIT). After the thrombectomy he returned to the ICU intubated.

On post-operative day 1 from the thrombectomy he was transitioned to apixaban, asa, and ticagrelor. He progressed to having AKI requiring CRRT. He was restarted on Argatroban secondary to presumed malabsorption. He underwent Echocardiogram which showed EF of 40%. He has worsening hemodynamics and required additional pressors. Repeat echo showed a left ventricular clot despite being on argatroban. He ultimately expired.

Platelet Trend





Echocardiogram demonstrating left ventricular thrombus

Discussion

Historically Heparin Induced thrombocytopenia comes in two forms HIT I (milder form) and HIT II (now just HIT). HIT II comes in a delayed fashion and is associated with more severe complications.

HIT can be further separated into Autoimmune HIT(aHIT) which is characterized by delayed presentation, platelet activation even after heparinoids are stopped, more severe thrombocytopenia, more thrombotic complications, and even refractory HIT.

Treatment proposed for aHIT or refractory HIT, in the literature, is with high dose IVIG. The mechanism of action for IVIG in this setting is competitive inhibition at the platelet receptor decreasing the amount of thrombosis. IVIG is not standard of care for other types of HIT secondary to its thrombogenic nature in most cases.

References

1. Hogan M, Berger JS. Heparin-induced thrombocytopenia (HIT): Review of incidence, diagnosis, and management. *Vasc Med*. 2020;25(2):160-173. doi:10.1177/1358863X19898253
2. Newton F, Glaser K, Reeves J, Sheperd L, Ray B. Refractory Heparin-Induced Thrombocytopenia in a Patient With Subarachnoid Hemorrhage-A Clinical Conundrum. *Neurohospitalist*. 2021;11(4):360-364. doi:10.1177/1941874421995377
3. Al-Sadawi M, Haddadin M, Capric V, McFarlane SI. Argatroban-refractory, Heparin-induced Thrombocytopenia after Coronary Intervention with Radial Artery Occlusion. *Am J Med Case Rep*. 2019;7(9):184-190. doi:10.12691/ajmcr-7-9-1
4. Deen IU, Jha SA, Mustafa S. A Case Report and Literature Review on Argatroban Refractory Heparin-Induced Thrombocytopenia. *J Community Hosp Intern Med Perspect*. 2022;12(5):102-105. Published 2022 Sep 9. doi:10.55729/2000-9666.1102