LEARNING OBJECTIVES

1. Recognize rare, but potentially serious side effects of glycoprotein IIb IIIa inhibitors
2. Work up and management of severe thrombocytopenia in post Percutaneous Coronary Intervention patients

CASE
A 61 yo man with a past medical history of ulcerative colitis controlled on sulfasalazine, remote tobacco use, and a family history of coronary artery disease was transferred directly to the cardiac catheterization lab from an outside hospital with evidence of inferior and posterior ST elevation myocardial infarction on ECG after presenting to the emergency room with acute onset severe substernal chest pain at rest. He received 5000 units of heparin intravenously, 325mg of aspirin, and 600mg of clopidogrel prior to transfer. Baseline labs from the outside hospital revealed a hemoglobin of 15, hematocrit of 45, platelets 325, creatinine 0.8, INR 0.9, and PTT of 24. Coronary angiography revealed the culprit lesion to be an occluded proximal left circumflex artery along with significant three vessel disease. Intervention of the 100% stenosis in the proximal left circumflex artery was performed after the patient received an additional 1000 units of intravenous heparin and a bolus of eptifibatide with aspiration thrombectomy followed by stenting with a bare metal stent.

Shortly after the stent was deployed, the patient was noted to be hypoxic with oxygen saturation of 84% and hypotensive with a blood pressure of 70/40mmHg. The patient was aroused easily and was alert without complaints. Epinephrine was administered and blood pressure increased promptly to 130/90mmHg. Nasal cannula was replaced by a rebreather mask with 40% FIO2 with improvement in oxygen saturation. However, the patient rapidly decompensated shortly thereafter, and was intubated. The anesthesiologist noted a significant amount of blood in the oropharynx prior to intubation and suctioned 250cc of blood from the ET tube after easy intubation. Eptifibatide infusion was discontinued. Echo revealed hypodynamic left ventricle with no pericardial effusion and aortogram was negative for dissection.

HOSPITAL COURSE
At the CVCU, he had a platelet count of 26 with hgb of 11.4; groin site did not reveal any hematoma. Repeat platelet count was 65 and peripheral smear confirmed thrombocytopenia. DIC panel, PF4 antibodies were sent along with integrin antibody. Bronchoscopy was performed and revealed active alveolar hemorrhage. Patient received 4 units of packed RBCs. PF4 was negative for HIT. Fibrinogen and INR were within normal limits though D dimer was mildly elevated at 5.4. Hematology was also suspected Integrin induced hemorrhage patient. Patient’s platelet count rose to 103 and was given 2 units of platelets, but post transfusion platelet count was now 82. His dimer was found to be above 34, PT was now 16.2, PTT was prolonged at 40.2 and fibrinogen was now low at 119 giving a DIC score of 6. He also went into renal failure with rise in creatinine to 2.2 from baseline of 0.9 and liver enzymes elevated to 140 for ALT and AST 472.

On HD 2, his dimer continued to remain elevated above 34 and platelet count was in the twenties to thirties with hgb of 11.0 despite 4 units of blood transfused. Peripheral smear revealed shistocytes and nucleated platelets were transfused and temporarily rose to 100, but again by HD 3 dipping down to the fifties. An attempt at a Swan Ganz Catheter was made, but the patient went into subsequent cardiac arrest and the procedure was aborted. Echo was repeated and revealed worsening systolic function. An intra-aortic balloon pump (IABP) placed for cardiogenic shock while on pressure support. Patient died 1 out of 2 blood cultures positive for strep viridans by day three with fever of 101.3 and was started on zosyn. By HD 4 with lack of improvement in clinical course while the patient in multi organ failure, he was made comfort care only and passed away.

DISCUSSION

IIB IIIA Induced Thrombocytopenia:
- Glycoprotein IIB-IIIa inhibitor induced thrombocytopenia (GIT) is a rare, serious complication, occurring in roughly 0.3-0.7% of patients usually happening within minutes to hours of administration. Counts can be as low as <30,000. Other causes such as pseudothrombocytopenia, DIC, HIT should be ruled out.
- Treatment includes supportive care including platelets for active bleeding. Platelet counts should otherwise return to normal in three to six days.

Diffuse Alveolar Hemorrhage:
- Diffuse Alveolar Hemorrhage has become an increasingly reported event related to IIB IIIa inhibitors with recent analysis suggesting an occurrence in 0.2-0.3% of cases.
- Symptoms can include hypoxia, anemia, hemoptysis (though one third do not have this symptom), and new chest infiltrates.
- Treatment includes discontinuation of IIB-IIIa inhibitors and other anticoagulation and supportive treatment to maintain O2 sat.

Reflection:
- This is a unique case in that our patient developed not only GIT with DAH, but subsequently developed DIC within hours leading to persistent thrombocytopenia and multi-organ failure.

TAKE HOME POINTS
IIB IIIa Inhibitor induced thrombocytopenia and diffuse alveolar hemorrhage are rare, but potentially life threatening events. With greater use, the clinician must be aware of these complications in order to recognize and properly treat. If platelet counts do not improve on their own after discontinuation, further pathology including DIC should be investigated.

REFERENCES:
