

## **Casting a broad differential: Unstable Angina in the setting of Thrombotic Thrombocytopenic Purpura**

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### **Introduction**

This case describes a man with unstable angina and new thrombocytopenia, due to thrombotic thrombocytopenic purpura (TTP). It emphasizes maintaining a broad differential for chest pain along with management of unstable angina in the setting of TTP.

### **Case Description**

A 64-year-old African American male presented with new onset chest pain, awaking him from sleep. Physical exam was unremarkable with stable vital signs. Electrocardiogram showed sinus rhythm with t-wave inversions in V2-V4. Echocardiogram showed anterior wall motion abnormalities. High sensitivity delta troponin was 4. He had a normal white blood cell count (5,600/uL), mildly decreased hemoglobin and hematocrit (12.3 g/dL and 37% respectively), and low platelets (20,000/uL). Coronary catheterization was deferred due to severe thrombocytopenia.

His thrombocytopenia work-up had evidence of hemolysis (indirect bilirubin 2.8 mg/dL, lactate dehydrogenase 643 U/L, haptoglobin < 20 mg/dL). Testing revealed decreased ADAMTS13 activity (6%) with positive ADAMTS13 inhibitor, and his blood smear showed schistocytes (Figure 1). Therefore, he was diagnosed with acquired TTP.

As he was treated for TTP, he did not have further chest pain. When his platelet count reached 58,000/uL, he was started on aspirin 81mg to prevent microthrombi formation. When his platelets normalized, a left heart catheterization showed an occluded proximal left anterior descending (LAD) artery with right-left collaterals (Figure 2). Due to TTP, intervention during this angiogram was deferred. We discussed performing a single vessel coronary artery bypass graft (CABG), rather than using a drug-eluting stent, as the time on dual-antiplatelet medication (DAPT) is reduced with a CABG (1 month) as compared to a drug-eluting stent (at least one month, optimally 1 year). This could be important if his TTP reoccurred, necessitating stopping antiplatelet medications. A subsequent nuclear stress test was positive for ischemia. With continued platelet stability and signs of ischemia, he had percutaneous coronary intervention with drug eluting stents placed in the LAD and diagonal arteries (Figure 3). This was decided over CABG, as he could stop DAPT 1 month out if needed for either procedure, and stenting was lower risk. He was discharged on aspirin, clopidogrel, metoprolol succinate, atorvastatin, and lisinopril. For TTP, ten plasmapheresis sessions were performed, and he was discharged on prednisone and initiated rituximab. One month since hospitalization, platelet count remains normal, and he is continuing DAPT.

### **Discussion**

This case emphasizes (1) the importance of maintaining a broad differential for chest pain. TTP can cause chest pain by microthrombi in the coronary arteries. Before obtaining a coronary angiogram, platelets need to be normalized, and there still has to be signs of ischemia. (2) TTP affects the timing of angiography and initiation of antiplatelet medications. (3) Starting

antiplatelet medications is important for prevention of microthrombi formation. (4) Rituximab is used to prevent relapse of TTP.

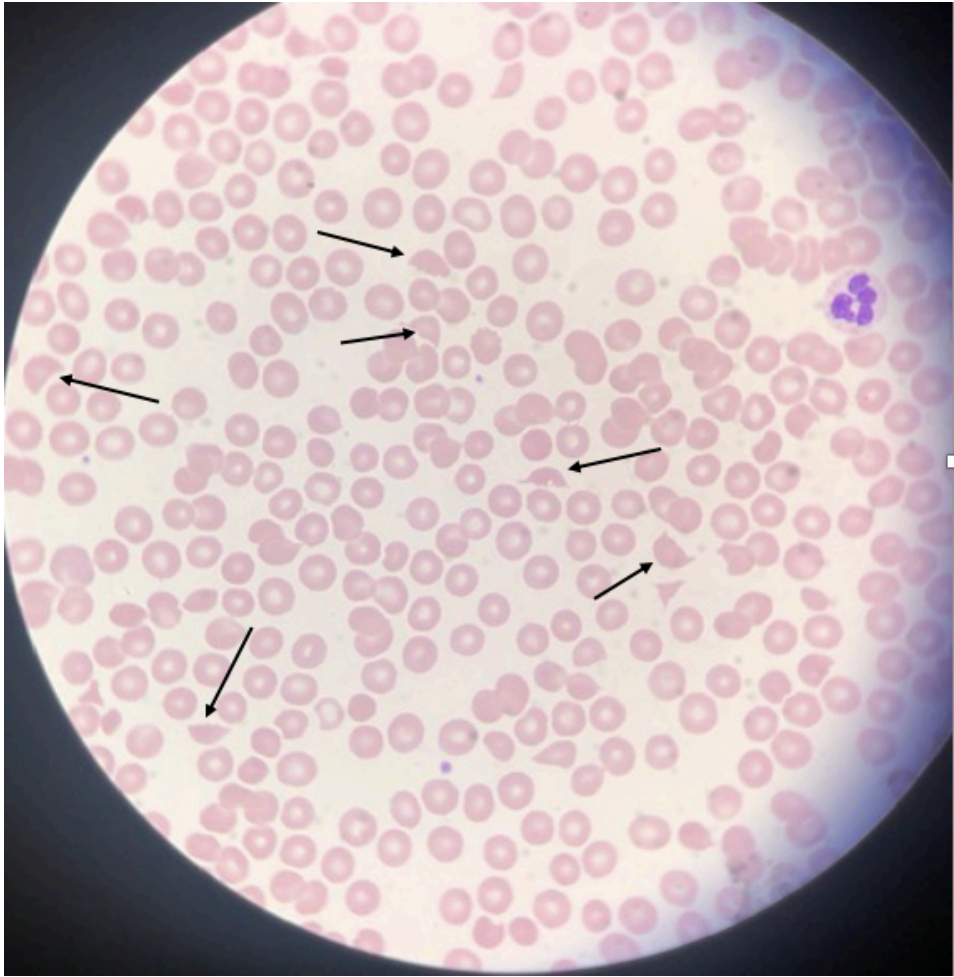


Figure 1: Peripheral smear with at least 6 schistocytes (arrows) per high power field.

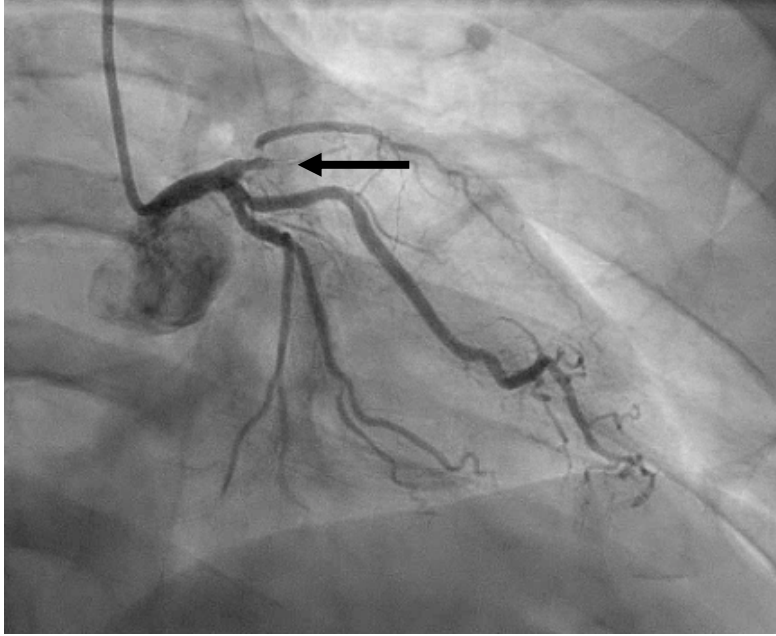


Figure 2. Occluded proximal LAD (arrow) with right to left collaterals.

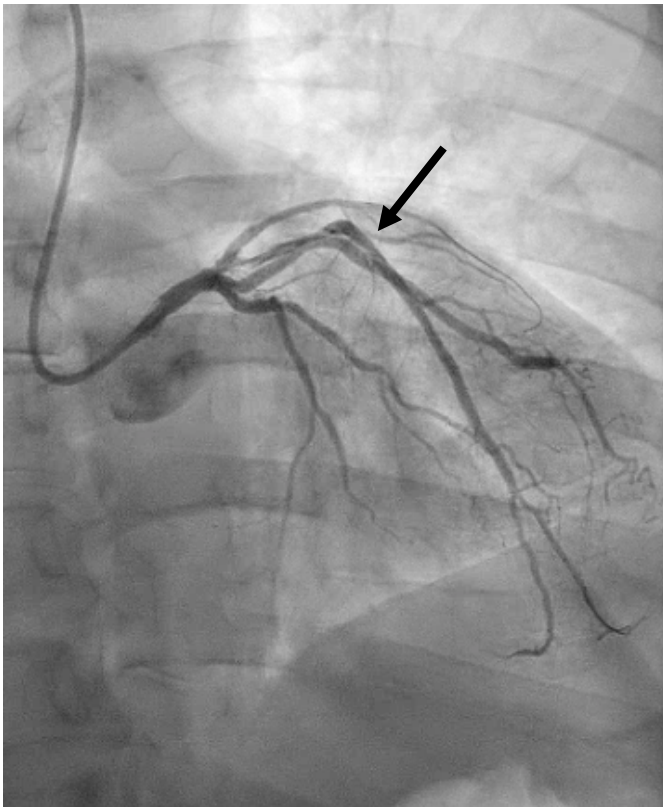


Figure 3. Coronary arteries after percutaneous coronary intervention. LAD revascularized (arrow).