Casting a Broad Differential: Unstable Angina in the Setting of (Thrombotic (Thrombocytopenic Purpura (

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History of Presenting Illness

- A 64-year-old African American male presented complaining of new onset, "sharp", left sided chest pain. It initially occurred in the evening while he was walking in his backyard, but improved with rest. However, another episode occurred about five hours later that woke him up from sleep. The pain went away on its own, nothing made it worse.

- *Past medical history*: hyperlipidemia
- *Family history:* no history of heart " disease "
- *Social Hx:* recreational marijuana use; remote history of cocaine and tobacco abuse (quit smoking 30 years ago). Does not exercise.
- *Home medications:* aspirin 81mg, atorvastatin 40mg

Physical Exam

Vitals: T: 36.1 °C, HR 56, BP 138/88, RR 16, SpO_2: 97% on room air

General: muscular build, not in acute distress
Cardiac: normal S1/S2 cardiac sounds with no murmurs, no chest pain on palpation
Pulmonary: clear breath sounds bilaterally
Abdominal: no abdominal tenderness and normoactive bowel sounds
Musculoskeletal: full strength and sensation in extremities
Neuro: no focal deficits
Skin: no evidence of rash or petechiae

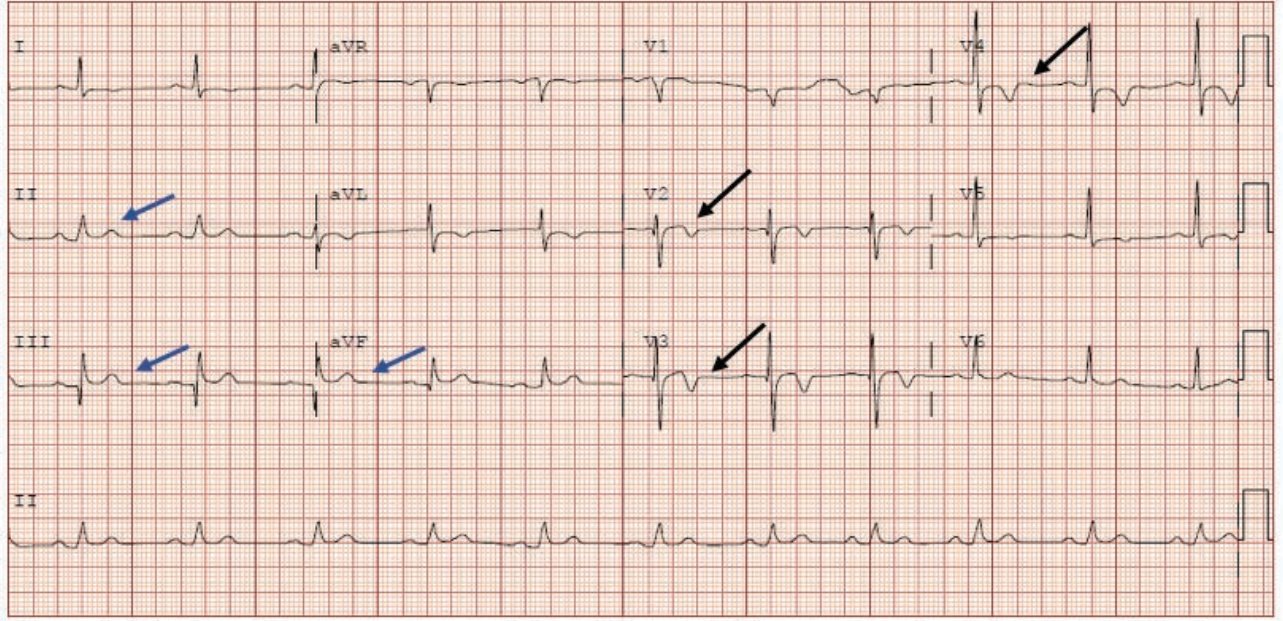

Figure 1. Electrocardiogram on presentation. Abnormal T-waves in anterolateral leads (black arrows). Minimal ST elevation in inferior leads (blue arrows).

Table 1. Pertinent Lab Results, *abnormal results are in blue*

Lab test	Value	Normal
0 hour troponin	14	0-21 ng/L
3 hour troponin	18	0-21 ng/L
Delta troponin	4	0-11
White blood cell count	5,600 / uL	4,200-9,100 / uL
Hemoglobin	12.3 g/dL	13.7-17.5 g/dL
Hematocrit	37%	40-51%
Platelets	20,000 / uL	150,000-330,000 / uL
Total bilirubin	3.2 mg/dL	0-1.2 mg/dL
Indirect bilirubin	2.8 mg/dL	0.1-1.0 mg/dL
Lactate dehydrogenase	643 U/L	118-225 U/L
Haptoglobin	<20 mg/dL	30-200 mg/dL
Prothrombin time (PT)	12.2 seconds	10-12.9 seconds
International normalized ratio (INR)	1.1	0.9-1.1
Activated partial thromboplastin time (aPTT)	31.6 seconds	25.8-37.9 seconds
Fibrinogen	377 mg/dL	172-409 mg/dL
D-dimer	2.08 ug/mL FEU	0-0.5 ug/mL FEU
ADAMTS13 activity	6%	>70%
ADAMTS13 inhibitor	Titer 1.6	<0.4

- **Echocardiogram:** anterior wall motion abnormalities consistent with the distribution of the left anterior descending artery

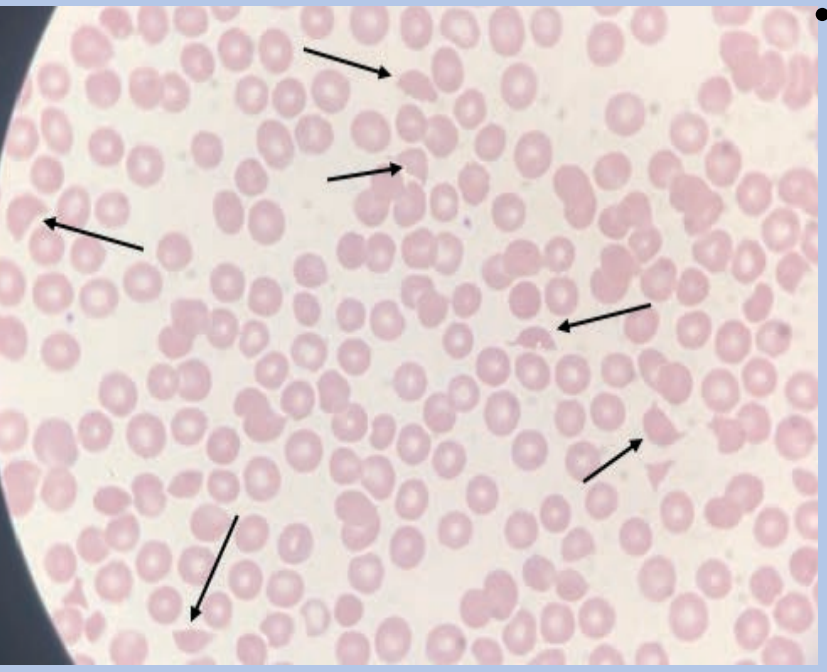

Figure 2. Peripheral smear **showing schistocytes** (black arrows)

Clinical Decision Making '

Unstable angina

-EKG

-Echo

-Negative delta troponin

Thrombocytopenia

-Blood smear shows schistocytes (Figure 2).

-ADAMTS13 inhibitor positive (Table 1).

-Deferred plan for coronary angiogram.

Plasmapheresis

-10 sessions total over 15 days

-Started aspirin 81mg when platelets >50,000/ uL

Coronary angiogram

-Occluded proximal left anterior descending artery with right-left collaterals (Figure 3A).

-No coronary intervention

-Discussion about single vessel CABG vs. PCI vs. optimized medical therapy

Nuclear stress test

when thrombocytopenia resolved

-28% ischemia in left anterior artery distribution

*Continued ischemia with normal platelet count

Percutaneous coronary intervention

-Drug eluting stents placed in left anterior descending and diagonal arteries (Figure 3B).

Discharge medications

TTP: prednisone, initiation of rituximab

CAD: aspirin, clopidogrel, metoprolol, lisinopril, atorvastatin

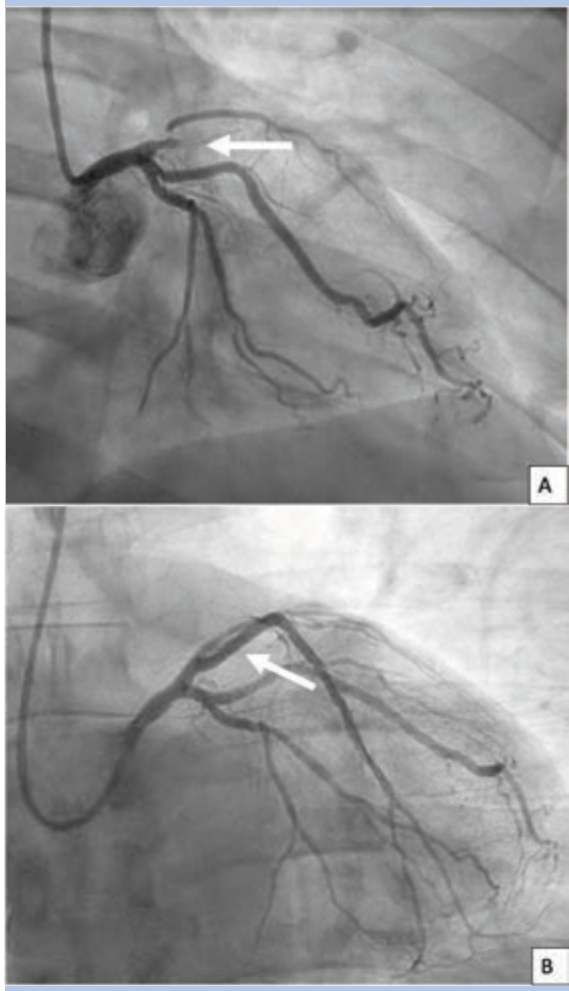

Figure 3. Coronary angiography, (A) percutaneous coronary intervention (B).

Thrombotic Thrombocytopenic Purpura (TTP) *

- Decreased activity of ADAMTS13, a protease that cleaves von Willebrand factor.
- Acquired TTP is due to an autoantibody against ADAMTS13.
- Cardiac involvement[1,3,4,5]
  - Hypertension
  - Myocardial infarction
  - ST elevation myocardial infarction (STEMI) and non-ST elevation myocardial infarction (NSTEMI)
  - Atrial fibrillation
  - Congestive heart failure
- Most common cause of death associated with TTP is due to cardiac arrest or myocardial infarction, thought to be due to *microthrombi*.[6,1]
- Plasma exchange removes the anti-ADAMTS13 antibodies.[1]
- Other therapies include glucocorticoids, rituximab, and caplacizumab in certain high-risk patients.[1]

Pentad: [1]

1. Fevers
2. Altered mental status
3. Thrombocytopenia
4. Reduced kidney function
5. Microangiopathic hemolytic anemia (MAHA)

Conclusions

- Our patient had TTP with concomitant obstructive coronary artery disease.
- Maintain a broad differential for unstable angina
  - include TTP as a cause for chest pain, due to microthrombi in the coronary arteries.
- Before obtaining a coronary angiogram, *platelets should be normalized*, and there still has to be signs of ischemia.
- TTP affects the *timing of angiography* and *initiation of antiplatelet medications*.
- Starting antiplatelet medications is important for *prevention of microthrombi formation*.
- Rituximab is used to reduce the risk of exacerbation and relapse of TTP.

References

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