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Between A Rock and A Hard Place: Thrombotic Thrombocytopenic Purpura Induced ST-Elevation Myocardial Infarction

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Between A Rock and A Hard Place:

Thrombotic Thrombocytopenic Purpura Induced ST-Elevation Myocardial Infarction

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BACKGROUND

 Thrombotic Thrombocytopenic Purpura (TTP) is a rare and life-threatening coagulation disorder caused by antibodies against ADAMTS13. Loss of ADAMTS13 activity can lead to organ ischemia and thrombi formation secondary to depletion of hemostatic factors. It is reported that approximately 15-41% of TTP cases are complicated with a myocardial infarction (MI).

CASE PRESENTATION

- A 57-year-old female presented to the ER with complaints of hematuria and abdominal pain.
- Labs demonstrated Hgb 8.0 g/dL, Platelets 9.0 thou/cmm, LDH 9342 u/L, Direct bilirubin 2.0 mg/dL (Total bilirubin 6.6 mg/dL), INR 1.9, SCr 2.46 mg/dL (baseline <1.0 mg/dL), and Troponin 8.41 ng/L.
- Peripheral smear demonstrated schistocytes.
- The patient was clinically diagnosed with TTP.

INTERVENTIONS

REFERENCES

blood-2017-04-636431

 The patient was administered FFP and started on plasmapheresis to reduce antibody mediated destruction of ADAMTS13.

Figure 2: Courtesy of CMC EKG Masters - https://www.cmcecg.com

Figure 3 and 4: Blood 2017 130:1181-1188; doi: https://doi.org/10.1182/

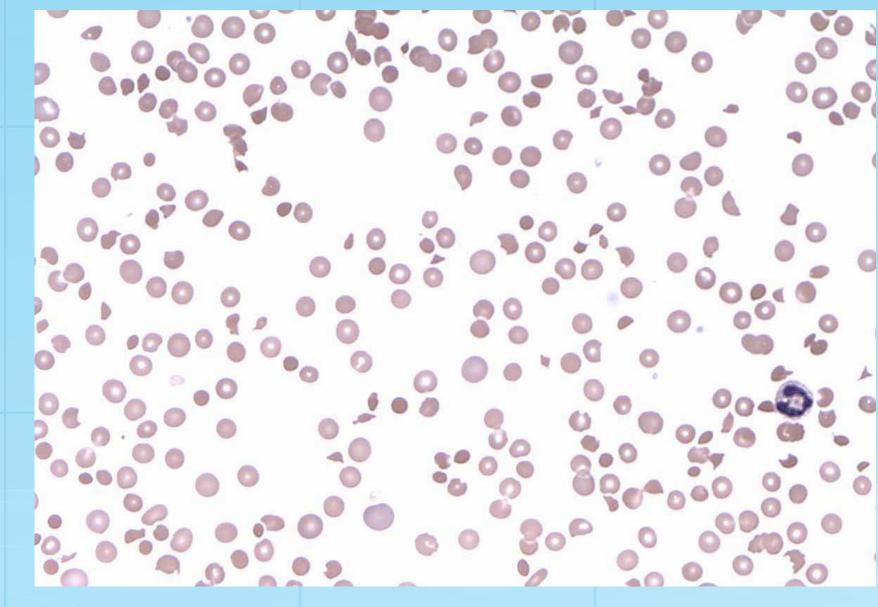


Figure 1: Peripheral smear demonstrating schistocytes in TTP.

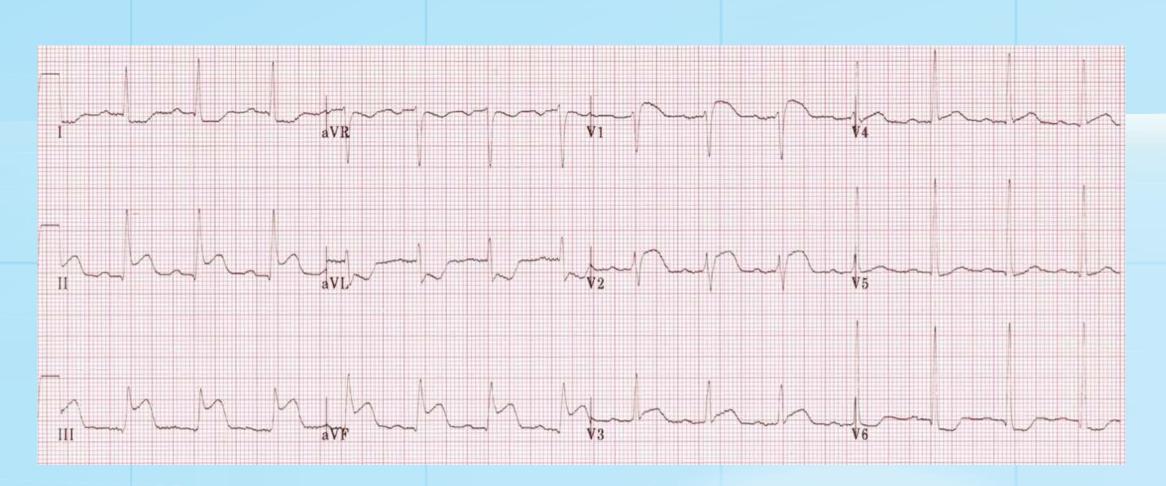


Figure 2: EKG demonstrating acute ST elevations in inferior leads (II, III, avF) and anterior leads (V1, V2).

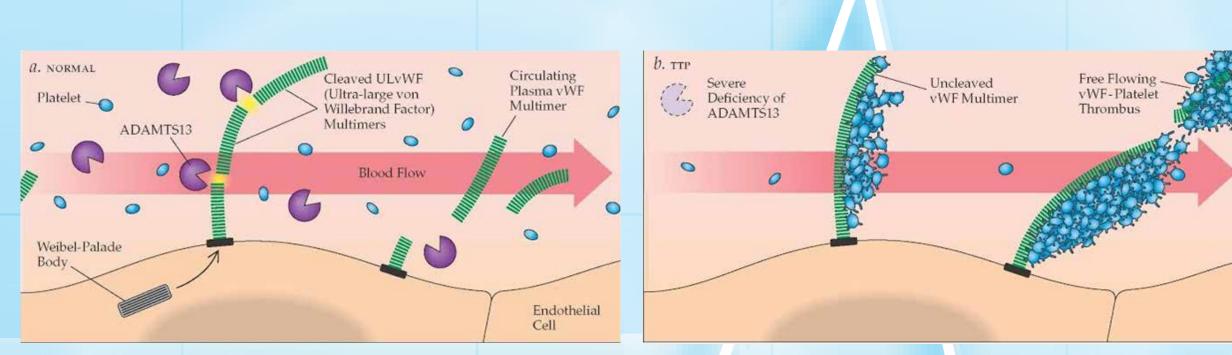


Figure 3: a. Physiology of ADAMTS13 and b. Pathology of ADAMTS13 in TTP.

Plasma Recombinant ADAMTS13 Caplacizumab B cell Plasma cell Bortezomib N-acetylcysteine

Figure 4: Therapies targeting the pathophysiology of TTP

RESULTS

- ADAMTS13 activity was obtained which resulted at 2.0% (normal >66%).
- Unfortunately, the patient went into PEA arrest with EKG revealing STEMI in leads 2,3, avF and V1-V2. Catheterization (PCI) was deemed too high risk for bleeding and renal failure considering the patient's AKI and thrombocytopenia. Resuscitative measures were stopped after 5 rounds of CPR.

DISCUSSION

- MI is a well described complication of TTP; however, subsequent management remains controversial.
- Recently, studies have shown a potential role for corticosteroids and Rituxan in order reduce thrombi formation; neither of which were used in our patient.
- This presentation illustrates the importance of acknowledging MI as a common complication of TTP and brings to attention the role of immunosuppressive agents as possible therapies in cases where PCI is contraindicated.



Figure 1: Courtesy of Dr. Erhabor Osaro