Unique case of cocaine induced TMA and renal infarction mimicking atypical HUS

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BACKGROUND

- Cocaine use has known association with fatal medical complications, including; myocardial ischemia, cerebrovascular accident, and kidney injury secondary to ischemia, rhabdomyolisis, and malignant hypertension.
- However, cocaine induced Thrombotic microangiopathy (TMA) and renal infarction is a rare entity that has not been described in the literature except in few case reports.

CASE PRESENTATION

- 24-year-old female with a past medical history of alcohol and cocaine abuse;

- **Chief complain:** abdominal pain radiating to the back, decrease urinary output
- **Laboratory studies:** creatinine 7 mg/dl (baseline of 0.7–0.9 mg/dl), thrombocytopenia (PLT 40), schistocytes on peripheral smear, a drop in hemoglobin (from 9 to 5.6 g/dl), with elevated total bilirubin 3.2 g/dL and LDH 3400
- **Initial diagnosis:** alcoholic induced acute pancreatitis acute & anuric renal injury.

HOSPITAL COURSE

- She developed microangiopathic hemolytic anemia, thrombocytopenia, and neurologic symptoms including headache and blurry vision which were concerning for TMA versus atypical hemolytic uremic syndrome (HUS).
- She was started on plasma exchange and IV mehtylprednisone for presumptive diagnosis of Thrombotic thrombocytopenic purpura (TTP). ADAMTS13 level was 86 therefore TTP was ruled out.
- Complement level was low shifting the diagnosis toward atypical HUS and the patient was started on Eculizumab
- **Further workup:** immunologic work-up including ANA, CCP, RF, CRP, C3/C4, myeloperoxidase AB, and proteinase-3 AB which were all unremarkable. Lupus anticoagulant was positive.
- Finally the patient underwent kidney biopsy once her platelet count improved and the final report showed evidence of ATN, ischemic necrosis, focal interstitial hemorrhage, and infarcts most likely due to cocaine induced TMA.

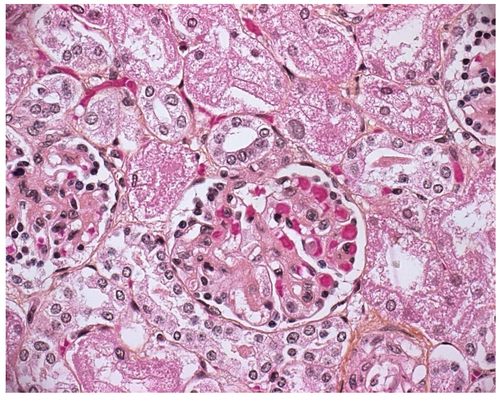

Figure1. Acute thrombotic microangiopathy with glomerular ischemic wrinkling, segmentally accentuated, foci of glomerular ischemic necrosis, and focal interstitial hemorrhage with infarct. Acute tubular injury/necrosis

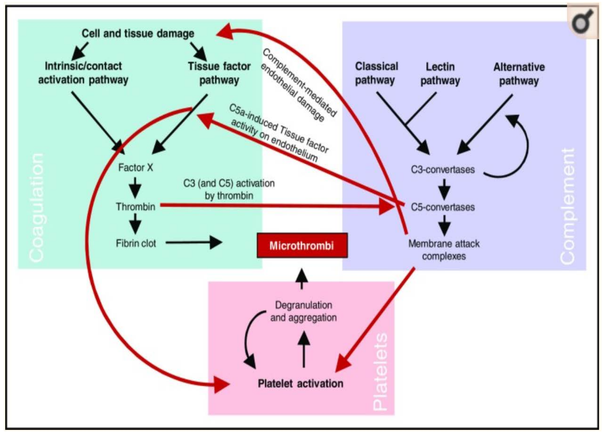

Figure 2. Schematic presentation of the main links between the complement and coagulation systems and platelets in formation of microthrombi in aHUS

DISCUSSION

- The pathophysiological basis of cocaine-related renal injury is multifactorial and involves one or a combination of changes in renal haemodynamics, changes in glomerular matrix synthesis, & degradation, oxidative stress and induction of renal atherogenesis
- Malignant hypertension-associated thrombotic microangiopathy following cocaine use has been reported in the literature in multiple rare case reports
- Previous kidney biopsies revealed typical features of thrombotic microangiopathy with fibrinoid necrosis of arterioles and glomeruli, vascular sclerosis and glomerulosclerosis

CONCLUSION

- Cocaine induced renal TMA should be suspected in patients with heavy use of cocaine who presents with a new onset Acute Kidney Injury.
- There is also emerging evidence that cocaine can activate complements and causes hypo-complementemia seen in our patient.

REFERENCES

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