An unusual cause of renal failure: I took quinine for my leg cramps

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Abstract
We present the case of a 57 years old female who developed a Thrombotic Microangiopathy and renal failure after a single dose of quinine. She had plasma exchange and was started on immunosuppressive treatment. She slowly improved and is now no longer dialysis dependent.

Case
A 57 years old female presented to hospital due to bruising and lethargy. She reported a fever and had passed less urine over the last 3 days. She was a smoker and was on salbutamol and seretide inhalers.

Physical examination revealed mild pitting oedema, fever and a rash.

Her blood results were grossly abnormal. Haemoglobin was 87, Platelets 9, CRP >500, eGFR 9, pH 7.27, Bilirubin 137 and LDH 1912. Clotting screen and Chest Xray were normal. A blood film had schistocytes which confirmed haemolysis.

A diagnosis of thrombotic microangiopathy (TMA) was made. At that point, sepsis of unclear source was the probable diagnosis and antibiotics were given to treat for disseminated intravascular coagulopathy (DIC).

She became anuric and was commenced on haemodialysis. Other causes such as Thrombotic Thrombocytopenic Purpura (TTP) and Haemolytic Uraemic Syndrome (HUS) were considered as there was no clear focus of infection.

There was no history of diarrhoea which excluded HUS. She did not have any neurological symptoms and ADAMTS13 levels were normal, which excluded TTP.

Immunology, Virology and Microbiology screen were negative and CT PET did not show any malignancy. She did not improve and the diagnosis of sepsis was again put into question.

A full collateral history was taken from the husband, and he reported that she was scratched by a cat recently and had taken quinine for leg cramps one day prior to the start of symptoms.

A diagnosis of quinine induced TMA was made. She had plasma exchange and was immunosuppressed with prednisolone and mycophenolate. She gradually improved and is now no longer dialysis dependent.

Discussion
Quinine induced TMA is immune mediated, and can happen after one single dose. The treatment is immunosuppression, plasma exchange and drug avoidance.

Drug induced TMA is rare. It has been noted to occur after exposure to other drugs such as gemcitabine and quetiapine.

It has an abrupt onset. Patients can present with a myriad of symptoms and it often leads to anuric acute kidney injury.

Drug-induced TMA can be especially challenging to diagnose. There may not be a readily available specific laboratory tests to identify a drug etiology such as quinine antibodies.

The role of the potentially implicated drug may not be clear. The information may not be volunteered by the patient especially if exposure is via a beverage such as tonic water.

Management of a patient with suspected drug induced TMA may be problematic as the diagnostic criteria overlaps with other serious conditions such as HUS, TTP and DIC.

Judicious screening should be done to exclude these other diagnoses such as low fibrinogen in DIC, E.coli and Shigella toxins in HUS and ADAMTS13 enzyme level in TTP.

Neurological disorders are common in TTP while AKI tends to be rare.

Learning points
While this is a very rare presentation, this case highlights the importance of a careful and systematic approach to investigation especially when the diagnosis is unclear.

Collateral history has proven crucial to this case. The patient has been advised to never take quinine and tonic water.

This is of particular relevance to Acute Medicine Physicians as we are the ones assessing the patient first and it is paramount that the correct investigations are requested to ensure timely diagnosis and hence management of patients.

References