



Quinine induced disseminated intravascular coagulopathy

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CASE REPORT

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Abstract

There are only a handful of case reports of Disseminated Intravascular Coagulopathy (DIC) as a result of quinine use however it is important to recognise this early especially when quinine has not been prescribed for managing malaria. Off licence use of any medicine may result in serious complications, quinine induced DIC is one of them.

Key Words

Quinine, DIC

Background

It is important to recognise quinine induced DIC early in the clinical setting, as this diagnosis affords a better prognosis than other adult forms of DIC as is evident in this case. The clinical expression of DIC varies and may be manifested by laboratory abnormalities alone or in combination with haemorrhagic and thrombotic complications. A Food and Drug Administration (FDA USA) advisory in 2006 warned against the off-label use of quinine sulfate and its derivatives in the treatment of muscle cramps, however it is commonly prescribed in a general practice setting in Australia, many times as a first line agent for leg cramps.

Case details

A 67-year-old lady, presented with episodic right-sided sharp chest pain radiating from her right shoulder. There was history of recent nose bleed. Her past medical history included asthma since childhood, non-insulin dependent diabetes which was diet controlled for the last eight years,

controlled hypertension for the last few years, gastro oesophageal reflux disease with no evidence of ulcers on recent upper gastrointestinal scope, and a hydatid cyst in the liver for the last five years.

Her medications included pantoperazole 40mg once daily, nifedipine 30mg once daily, ventoline inhaler as needed, paracetamol 1gm four times per day, and quinine 300mg started one day ago by her GP for leg cramps

She was haemodynamically stable with normal general physical and systemic examination except petechias at the shoulder blades posteriorly.

Investigations revealed haemoglobin 13.6g/dl, white cell count 3.1, neutrophil count 0.9, platelets 98, band forms 1.4, lymphocytes 0.4

Blood film showed increased band forms, poikilocytosis, and decreased platelets, with CRP < 6, ESR 45 APTT 63 sec, INR 4.2, and D dimers >3.2 mg/l (raised D dimer) FDP were also raised. Urea, creatinine and liver transaminases were normal. LDH was 2135 mg/l and Troponins <0.15 mg/l. Her ECG, chest X ray and CTPA were normal. CT of chest abdomen and pelvis revealed no evidence of rupture of hydatid cyst. Repeated blood cultures and urine cultures showed no growth

The serology for autoimmune discrepancies, HIV, hepatotropic viruses, respiratory viruses, atypical organisms, and fungi was negative.

She was admitted to ICU and treated conservatively. Her APTT and INR became normal on the second day. Platelet count, recovered after four days. Neutrophil count recovered after three days. LDH showed a downward trend. Platelet associated antibodies and quinine dependent platelet antibodies were not detected.

She remained stable and asymptomatic and was discharged home after seven days of hospitalisation. There was no clinical evidence of malaria, with no travel history or history of suggestive clinical features, so she was not investigated for malarial parasites. Her recovery and clinical picture was



also against her having malaria.

Patient consent

No patient identifiable material has been used

Discussion

Quinine generally causes drug-induced immunologic thrombocytopenia by initiating antibodies, however quinine induced DIC is a distinct clinical entity, which may present as unexplained thrombocytopenia, coagulopathy, or renal failure.¹

Central to the pathogenesis of DIC is the unregulated and excessive generation of thrombin which results in the consumption of coagulation factors, such as fibrinogen, factor V, and factor VIII.

DIC has been previously reported with quinine² which is characterised by laboratory evidence of consumption and proteolytic degradation of haemostatic components. The clinical expression varies and may be manifested by laboratory abnormalities alone or in combination with hemorrhagic and thrombotic complications.

It is important to recognise quinine induced DIC early in the clinical setting, as this diagnosis affords a better prognosis than other adult forms of HUS or DIC.¹

Variability in the production of antibodies and their pattern in a patient may be responsible for the diverse clinical presentation from mild isolated transient thrombocytopenia to intravascular haemolysis, acute kidney injury or coagulopathy.

A FDA USA advisory in 2006 warned against the off-label use of quinine sulfate and its derivatives in the treatment of muscle cramps³, however it is commonly prescribed in a general practice setting in Australia, many times as first line agent for leg cramps.

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CONSENT

The authors declare that

1. They have obtained informed consent for the publication of the details relating to the patient(s) in this report.
2. All possible steps have been taken to safeguard the identity of the patient(s).
3. This submission is compliant with the requirements of local research ethics committees.