Haemolytic anaemia and acute kidney injury: think beyond the obvious

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DESCRIPTION

A 34-year-old woman was admitted with symptoms of easy fatiguability, decreased urine output and jaundice for 1 week. She had similar history of weakness and jaundice 3 months ago that lasted for a week for which she consumed herbal medicines. Laboratory evaluation revealed anaemia with haemoglobin 30 g/L that was normocytic normochromic without any fragmented red cells, total leucocyte count 6.7×10^9 /L, platelet count 70×10^9 /L, serum creatinine 9 mg/dL, serum bilirubin of 1.3 mg/dL (unconjugated 0.7 mg/dL), lactate dehydrogenase 700 IU/L, plasma haemoglobin 60 g/L. Vitamin B12 and folate levels were normal. ANA was 1+homogeneous with normal complements and negative DsDNA. Urine dipstick showed 1+protein with 2+positive for blood and microscopy was normal. Direct Coombs test (DCT) was negative. Ultrasonography abdomen showed mild hepatosplenomegaly. After 2–3 sessions of haemodialysis and blood transfusion a renal biopsy was performed on day 5 of admission. Biopsy showed tubular damage with deposition of brown pigment in tubular epithelium that gave a blue colour on Perl's stain suggesting haemosiderin (figure 1A,B). There was no significant interstitial fibrosis and glomeruli were normal. Immunofluorescence was negative for immunoglobulins and complements. Flow cytometry done on peripheral blood granulocytes showed a paroxysmal nocturnal haemoglobinuria (PNH) clone deficient in CD59 suggestive of PNH. Patient was managed with dialysis, diuretics and folate supplementation with advice for bone marrow transplant in future. Her serum creatinine at discharge was 1.1 mg/dL with haemoglobin of 70 g/L.

Figure 1 (A) H&E stain of kidney biopsy showing dilated tubules with flattened epithelium and golden-brown haemosiderin pigment in tubular epithelium (40x). (B) Perl's Prussian blue stain showing strong positivity for haemosiderin in tubules (40x).

Learning points

- ▶ One should suspect haemoglobinuria in the presence of dip stick urine positivity for heme and negative urine red blood cell on microscopy.
- ▶ One should have a high index of suspicion and evaluate for paroxysmal nocturnal haemoglobinuria in a patient with haemolytic anaemia and acute kidney injury.

Lupus and thrombotic microangiopathy are often considered in the differentials of a patient with renal failure, haemolysis and thrombocytopenia. However, PNH is also a rare cause that should be suspected in a patient with DCT negative intravascular haemolytic anaemia and after ruling out microangiopathy. PNH can lead to massive haemoglobinuria leading to pigment nephropathy and acute tubular necrosis as in our case and in long term can lead to chronic kidney disease due to hemosiderosis and interstitial scarring.¹ Intravascular haemolysis can lead to acute kidney injury via multiple mechanisms including formation of intratubular casts and indirect proinflammatory effects via the activation of neutrophils and monocytes.² Allogenic stem cell transplant is the only curative treatment; however, eculizumab has also shown benefit to improve renal function and retard haemolysis in PNH.¹ The classic triad of haemolysis, thrombosis and thrombocytopenia with cola coloured urine may not be present in each case of PNH and it requires a high index of suspicion to diagnose it in early stage.

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