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Case Report

Plasmodium vivax with acute glomerulonephritis in an 8-year old

Saket R. Sanghai & Ira Shah

Department of Pediatrics, B.J. Wadia Hospital for Children, Mumbai, India

Malaria is one of the leading causes of acute renal failure in south-east Asia especially India and Vietnam, and Africa[1]. *Plasmodium vivax* is the predominant malarial parasite in India accounting for 50.4–56.4% cases in the last five years[2]. Falciparum malaria is the more severe variant of malaria in the region commonly associated with renal complications. Renal manifestations described in falciparum malaria have mainly been in the form of electrolyte disturbances, acute tubular necrosis, interstitial nephritis, acute glomerulonephritis and acute renal failure. Acute glomerulonephritis is a rare manifestation of *P. falciparum* malaria but has never been described in vivax infections[3]. We report the first case of *P. vivax* presenting as acute glomerulonephritis.

Case report: An 8-year old boy born of non-consanguineous marriage presented with fever with chills and rigors for three days and non-projectile, non-bilious vomiting for two days. He was passing dark coloured urine for two days. There was no dysuria, oliguria, breathlessness or bleeding. On examination, he was febrile, had tachycardia (pulse =124/min) with hypotension (blood pressure = 88/68 mm of Hg). He had splenomegaly. Other systems were normal. Investigations showed haemoglobin of 6.9 g/dl (MCV= 73.5 fl, MCH = 25.3 pg and MCHC = 34.3%) with, WBC count of 12,200/mm^3 (37% polymorphs, 42% lymphocytes) and platelet count of 6000/mm^3 with reticulocyte count of 0.8%. His bilirubin was 2.3 mg% (direct = 1.2 mg%) and creatinine was 1.1%. Prothrombin time and partial thromboplastin time was normal. Peripheral smear showed ring forms of *P. vivax.* OptiMal test for *P. falciparum* was negative. Blood culture did not grow any organism. Urine examination showed 3+ albuminuria with 4–6 RBCs/high power field. Leptospira and dengue tridot were negative. Coomb's test was negative. His serum electrolytes were normal. Ultrasonography of abdomen showed mild splenomegaly. ASLO was negative and serum C_3 was elevated [230 mg/dl (Normal = 110–120 mg/dl)]. Liver transaminases were normal and 24 h urine albumin was >4 g/day. He was treated with Artesunate, platelet and blood transfusion and iv fluids. Subsequently, he developed hypertension. He also developed frank hematuria which subsided on its own. Hypertension responded to oral nifedipine. The child recovered within one week and hematuria, albuminuria and hypertension resolved. His creatinine decreased to 0.6 mg% and platelet count normalized. He was given 14-days of primaquine after ruling out G-6-PD deficiency.

Plasmodium falciparum, P. vivax and mixed infection were reported to cause acute renal failure (ARF) in 16, 3 and 5 patients respectively in an Indian study from Mumbai[4]. Ahmed *et al*[5] reported that 66% of ARF was due to falciparum and 33% due to vivax. Children are at an increased risk of acute glomerulonephritis associated with falciparum malaria[6]. Glomerular lesions were described in 18% cases of renal failure due to falciparum malaria in an autopsy study[7]. These lesions are immune complex mediated and associated with a transient hypocomplimentemia (decreased C3). Usually the disease is mild, transient and overshadowed by other complications which make the interpretation of exact incidence difficult.

Vivax malaria complicated with renal failure commonly presents with fever, encephalopathy, jaundice,

hypotension, intravascular hemolysis, diffused intravascular coagulation (DIC), sepsis and thrombocytopenia[3]. Our patient also presented with fever, icterus, hypotension, splenomegaly, thrombocytopenia and hematuria. As a result, other possible diagnoses such as leptospirosis, dengue, post-streptococcal glomerulonephritis, falciparum infection and sepsis had to be ruled out. He was then treated as complicated vivax according to WHO guidelines[8]. A histological diagnosis was not required as clinical manifestations were unequivocal, peripheral smear was positive for vivax and it would not affect the management plan. He had an uneventful recovery which further substantiated our diagnosis. The elevated C3 levels did not confirm to the usual picture in acute glomerulonephritis due to *P. falciparum*[3].

There is a need for further research in the mechanism and the manifestations of acute glomerulonephritis in vivax malaria. However, this case gives enough evidence to suggest that it must be considered as a possible diagnosis in children and must be treated as a case of complicated malaria.

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Corresponding author: Dr Ira Shah, 240-D, Walkeshwar Road, Malabar Hill, Mumbai–400 006, India.
E-mail: irashah@pediatriconcall.com

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