J Vector Borne Dis 47, December 2010, pp. 264

Correspondence

Plasmodium vivax with acute glomerulonephritis in an 8-year old—Queries?

We read with interest the recent case report on '*Plasmodium vivax* with acute glomerulonephritis in an 8-year old' by Sanghai *et al*[1], and have the following comments to offer:

(1) The patient described in the case was having severe manifestations of vivax malaria. In addition to severe thrombocytopenia, he also had renal dysfunction, hypotension and hemolysis. The authors have diagnosed glomerulonephritis on the basis of presence of hematuria and hypertension. However, hematuria in the patient could have been due to severe thrombocytopenia or malaria-induced renal ischemia and acute tubular necrosis. The presence of brown or cola coloured urine with red blood cell casts (RBC) and deformed RBCs confirms the glomerular origin of hematuria[2]. Although the presence of heavy proteinuria suggests a glomerular origin, it would have been helpful if the authors had also commented on the presence of red blood cell casts (RBC) and deformed RBCs in the urine.

(2) Vivax malaria can lead to renal dysfunction as a result of renal ischemia and acute tubular necrosis. The patient also had hemolysis, hypotension, vomiting and hyperbilirubinemia which can predispose and contribute to the pathogenesis of acute renal failure[3,4]. Increased renin secretion in acute renal failure could have caused hypertension in the patient described[2].

References

1. Sanghai SR, Shah I. *Plasmodium vivax* with acute glomerulonephritis in an 8-year old. *J Vector Borne Dis* 2010; *47:* 65–6.
2. Davis ID, Avner ED. Clinical evaluation of the child with hematuria. In: Behrman RE, Kliegman RM, Jenson HB, Stanton FB, editors. *Nelson textbook of pediatrics*, 18th edn. Philadelphia: WB Saunders 2008; p. 2168–70.
3. Prakash J, Singh AK, Gujrati S, Maheshwari A. Acute renal failure in malaria: changing trends. *Indian J Nephrol* 2002; *12:* 113–7.
4. Das BS. Renal failure in malaria. *J Vector Borne Dis* 2008; *45:* 83–97.

Syed Ahmed Zaki & Preeti Shanbag
Department of Pediatrics
Lokmanya Tilak Municipal General Hospital
Sion, Mumbai-400 022
drzakisyed@gmail.com

Author's reply

The above authors have pointed out that the hypertension and hematuria could be due to severe thrombocytopenia or malaria-induced renal ischemia and acute tubular necrosis. We would like to point out that when the child had acute manifestations of malaria, urine only showed 4–6 RBCs. He developed hypertension and cola coloured urine after the fever had subsided and the urine did show RBC casts suggestive of glomerulonephritis. The authors have also pointed out that the patient had hemolysis, hypotension, vomiting and hyperbilirubinemia which can predispose and contribute to the pathogenesis of acute renal failure. For hemolysis to have caused renal failure, it would require massive hemolysis. However, in this child, Coombs test was negative. The hyperbilirubinemia was also predominantly direct jaundice suggestive of liver involvement rather than hemolysis.

Ira Shah
B.J. Wadia Hospital for Children, Mumbai, India
E-mail: irashah@pediatriconcall.com