

[\[Download PDF\]](#)**TROPICAL NEPHROLOGY**

Year : 1994 | Volume : 40 | Issue : 3 | Page : 132-4

Renal involvement in malaria.**MM Rajapurkar**

Department of Nephrology, Muljibhai Patel Urological Hospital, Nadiad.

Correspondence Address:

M M Rajapurkar

Department of Nephrology, Muljibhai Patel Urological Hospital, Nadiad.

How to cite this article:

Rajapurkar M M. Renal involvement in malaria. J Postgrad Med 1994;40:132-4

How to cite this URL:

Rajapurkar M M. Renal involvement in malaria. J Postgrad Med [serial online] 1994 [cited 2018 May 26];40:132-4

Available from: <http://www.jpgmonline.com/text.asp?1994/40/3/132/542>**Full Text****:: Introduction**

Malaria, a complex disease caused by plasm odia, affects almost all organ systems. After the initial success of malaria eradication programmes through vector control by DDT spraying and chemoprophylaxis, there has been a resurgence of the disease as well as a change in the virulence worldwide[1].

Malaria affects kidneys leading to both tubulointerstitial damage as well as glomerulonephritis. In India, two species of plasmodia namely Faiciparum and Vivax cause malaria. Renal lesion are commonly caused by P. falciparum; however, Vivax malaria also causes renal lesions, various authors have reported a wide variation in the incidence of malaria and the incidence of renal lesions in malaria.

Mahakur *et al*[2] reported 2.5% of all medical admissions to be due to malaria; out of which 1.08% were associated with acute renal failure. One of their patients of acute renal failure had P. Vivax[2]. In endemic areas, particularly from rural regions, most of the physicians when confronted with a febrile illness will treat with chloroquine before investigation. It is therefore difficult to detect malaria on peripheral smears by the time the patient reaches an institution.

:: (a) acute renal failure (arf) due to acute tubular necrosis (atn) in faiciparum malaria:

The most common renal lesion of malaria is acute renal failure due to acute tubular necrosis. This is seen in around 1 % of all P. Falciparum infected patients[3]. The incidence of acute renal failure rises to as high as 60% in patients having heavy parasitaemia (More than 10% P. falciparum infested RBCs). Incidence of Falciparum malaria during 1990, 91 and 92 in Gujarat is given in [Table:1].

From January 1988 to June 1992, in our hospital 797 (5.69%) episodes of malaria were diagnosed on peripheral smear in 14006 patients. Out of these 730 (91.59%) were P. Vivax and 67 (8.40%) were P. Falciparum malaria. 12 (1.6%) out of these 730 vivax cases had abnormal renal function; 11 (12.79%) out of the 86 P.Falciparum had acute renal failure. The pathophysiology mechanisms of acute renal failure in malaria are due to either; direct effect of the parasite on the erythrocyte (RBC) or due to non-specific effect of the infection. These are discussed briefly.

(i) Changes In RBCs due to Parasitization: The entry of merozoites of P. falciparum into the RBC depend on a specific receptor which is thought to be thrombospondin a glycoprotein. The parasite attaches to this receptor and then is taken into the RBC by invagination; it then matures in the RBC. This leads to alteration of RBC membrane and to formation of electron dense protrusions or knobs on the RBC surface.

The number of knobs increases as the parasite matures[5]. This reduces the deformability of the RBC. It also enhances adherence of the RBC to endothelial cells. These changes lead to increase in clearance of RBCs in the RE system and to occlusion of microcirculation in organs. This adherence of parasite infested RBCs to vascular endothelium is seen with only P. falciparum but has not yet been demonstrated in P. Vivax. This interference with microcirculation leads to ischemia in peritubular vessels in the kidney. This is an important factor in the causation of ischemic acute tubular necrosis. The loss of deformability also adds to ischaemia by causing sluggishness of blood flow and increase in viscosity, which is also contributed to by an increase in acute phase proteins such as fibrinogen.

(ii) Changes in the blood volume: Various studies have demonstrated hypo, hyper and normal blood volume in patients of P. falciparum. Hypovolemia is a common feature in our patients, especially those with heavy parasitaemia due to excessive insensible fluid loss in patients with hyperpyrexia. Hypovolemia is also due to increased capillary permeability particularly in very serious patients with associated bacterial endotoxins[6].

Hypovolemia is also contributed to by inadequate fluid replacement and vomiting in patients receiving chloroquine and metronidazole. In patients in the early stages hypervolemia due to shift of fluids into blood compartment from intracellular compartment secondary to vasodilatation and inappropriate ADH secretion, has been demonstrated[7]. Caution should therefore be exercised in fluid replacement in such patients lest pulmonary oedema should develop. Use of furosemide IV drip along with IV Dopamine is shown to be beneficial.

(iii) Next important factor contributing to development of acute tubular necrosis is Intravascular Haemolysis. The most common cause for haemolysis is drug induced oxidant stress in patients having glucose-6 phosphate dehydrogenase deficiency. Other factors, which contribute to intravascular hemolysis are: changes in RBC membrane[8], immunologic injury[9] and interference with RBC ATP and ATPase[10].

(iv) Catecholamine excess: Increased catecholamine activity has been demonstrated in both animal and human malaria. Improvement in urinary indices have been shown on administration of phenoxybenzamine in patients having early ATN in P. falciparum by Sitprija[11].

(v) Severe jaundice often associated with malaria contributes to causation of acute tubular necrosis.

(vi) Disseminated intravascular coagulation has been demonstrated in patients with both P. falciparum and P. Vivax. 14.3% patients of P. vivax in a series reported from Safdarjung Hospital, New Delhi presented with DIC like Syndrome[12].

DIC like Syndrome, cerebral signs, acute respiratory failure and acute renal failure is well known in severe malaria[13]. These patients carry very high risk of mortality.

Summary of Pathophysiologic mechanisms causing Acute Renal Failure in malaria is given in [Table:2].

:: (b) biochemical changes in malaria:



Apart from acute tubular necrosis malaria causes several biochemical alterations see [Table:3].

(i) Hyponatremia is not uncommon though clinically insignificant. The causes include inappropriate ADH secretion, hypervolemia and replacement with only glucose containing IV solutions. (ii) Hypokalemia occurs because of hyperventilation due to hyperpyrexia and respiratory alkalosis is common. (iii) Hyperkalemia occurs in patients who have marked intravascular hemolysis and/or well-established acute tubular necrosis. (iv) Severe hypoglycemia due to quinine induced secretion of insulin in patients having heavy parasitemia is common. Other factors, which contribute to hypoglycaemia are large glucose requirements of malaria parasite and exhaustion of hepatic glycogen reserve[14].

Continuous peritoneal dialysis with Tenckhoff catheter has been reported to be beneficial in preventing hypoglycaemia and providing adequate dialysis to patients with *P. falciparum*[15].

:: (c) malaria in renal transplant recipients:



Malaria is associated with severe morbidity in immunocompromised renal transplant recipients. 107 episodes of malaria were diagnosed in 467 transplant recipient from January 1988 till June 1992 at Muljibhai Patel Urological Hospital, Nadiad. 24 (22.42%) out of these 107 had *P. Falciparum* and 83 (77.57%) had Vivax Malaria. Fever was seen in all 107 (100%) and splenomegaly was present in 86 (80.37%) Rise of serum creatinine by more than 20% from baseline was noticed in 63 (58.87%). Graft tenderness was noticed in 23 (21.49%). Therefore malaria forms an important differential diagnosis of acute graft rejection crisis in endemic areas. 32 graft biopsies were done prospectively at our unit in smear positive patients. These biopsies revealed no change in histology in 11 (32.35%); tubular necrosis in 16 (47.05%); interstitial nephritis in 3(8.82%) and cortical necrosis in 4(11.76%). Cellular rejection was associated in 8 (23.52%) of these biopsies[4].

:: (d) glomerulonephritis in malaria:



Malaria nephropathy presenting as nephrotic syndrome has been reported inpatients having *P. malaria* infection from Nigeria, Uganda and Yemen etc. Plasmodium malaria infection is not seen in India hence will not be discussed here.

Transitory acute glomerulonephritis occurs often in association with *P. falciparum* infection. Microscopic haematuria, mild proteinuria usually less than 1 g in 24 hours is encountered in as many as 25-50% patients. Hypertension, oedema and drop in GFR however is rather uncommon. Renal biopsy done in early stages shows fine and coarse granular deposits of IgM and C3 in the mesangium and along capillary walls. Occasionally IgG deposits have also been demonstrated. Light microscopy shows widening of mesangium and endothelial cell proliferation. Pigment laden macrophages, parasite infested RBCs may be seen. Platelet fibrin thrombi and patchy necrosis of tubules may be associated. The glomerulonephritis usually resolves with proper treatment of the malarial episode.

Glomerulonephritis has not been reported in patients with *P. vivax*. Glomerulonephritis and nephrotic syndrome is a very common condition in malaria endemic regions, where both *P. falciparum* and as well *P. vivax* individually or in combination occurs. More work need to be done to ascertain the contribution of malaria to glomerulonephritis, and to ascertain if *P. vivax* causes glomerular lesions. It is my feeling that glomerulonephritis is caused by *P. vivax* and it

also contributes to morbidity of patients with glomerulonephritis in our region. At present however there is no evidence to support these statements.

References

- 1 Wyler DJ. Malaria □ Resurgence, Resistance and Research (first of two parts). N Engl J Med 1983; 308 (15):875□878.
- 2 Mahakur AC, Panda SN, Nanda BK, Bose TK, Satpathy SR, Misra Y. Malarial Acute Renal Failure. J Assoc Phys India 1983; 31:633□616.
- 3 Sakhuja V, Chugh KS. Int J Art Org 1986; 9:391-392.
- 4 Rajapurkar MM. (Abstract) XIIth Int Cong of Neph Jerusalem, Israel: 1993.
- 5 Sitprija V. NePhropathy in faiciparurn malaria. Kid Int 1988; 34:867□867.
- 6 Clark IA. Does endotoxin cause both the disease and parasite death in acute malaria and babesiosis? Lancet 1978; 2:75□77.
- 7 Malloy JP, Brooks MH, Barry KG. Pathophysiology of acute falciparurn malaria. Am J Med 1967; 43:745□750.
- 8 Balcerak SP, Arnold JD, Martin DC. Anatomy of red cell damage by plasmodium falciparurn in man. Blood 1972; 40:98□104.
- 9

Saturday, May 26, 2018

[Site Map](#) | [Home](#) | [Contact Us](#) | [Feedback](#) | [Copyright and disclaimer](#)