Malaria: Persistent Killer: Continuing Renal Complications

Rubina Naqvi (Sindh Institute of Urology and Transplantation, Dow Medical College, Karachi.)

Not long ago, the world seemed poised for victory in its age-old struggle against malaria. Armed with new drugs and potent pesticides, the World Health Organization (WHO) declared in 1955 that the disease would soon be eradicated. Obviously, things have not gone according to plan. Today, the number of cases are increasing worldwide. There are about 500 million cases per year and 2.7 million deaths, predominantly in young children in sub-Saharan Africa. The pathophysiological disturbances and symptoms of malaria are attributable solely to the asexual erythrocytic forms. Schizogony releases endogenous pyrogens. Endotoxins are absorbed from the gut as a result of altered splanchnic blood flow. Most of the acute symptoms are the result of pyrogen release and hemolysis. Massive intravascular hemolysis is seen in hyperparasitemia. Stagnant anoxemia may be the cause of cerebral malaria and ischemia of other organs including kidney. Erythmocytes parasitized by certain strains of P. falciparum develop knob like protrusions on their surface containing malarial antigens. These adhesions bind to endothelial ligands including thrombospondin. Damage to vascular endothelium may also contribute to cytoadherence. Tumor necrosis factor (TNF) concentrations are also reported to be increased during malarial illness with variations in IL-1 known to synergize with TNF.

The renal involvement in malaria varies widely. The spectrum includes transient glomerulonephritis, nephrotic syndrome in few cases, mild proteinuria with urinary sediment changes, abnormal serum electrolytes, haemoglobinuria and acute renal failure, associated with heavy parasitemia or with intravascular hemolysis with or without O-6PD deficiency. Delay in treatment due either to misdiagnosis, infection with resistant strain, lack of positive smears, or patients’ failure to seek medical help, besides dehydration resulting from high grade fever all contribute to development of renal failure. Malaria may present in an atypical fashion with symptoms suggestive of hepato-biliary disease, gastrointestinal disease or hepatitis and there is often clinical overlap between pneumonia and malaria. P. vivax, P. malaria and P. ovale should be treated with chloroquine unless high grade resistance develops for P. vivax. Relapsing malarias should be treated with primaquine to reduce risk of relapse from dormant liver stages, provided the patient is not G6PD deficient. P. falciparum (which is causative parasite in most cases of acute renal failure) remains a management challenge. Choice of anti-malarial therapy depends on knowledge of the sensitivity pattern of parasites in the area where the infection was acquired and severity of illness. Quinine is the mainstay of therapy for severe malaria and cures about 85-90% of infections when used with a second drug viz: pyrimethamine sulphadoxine or tetracycline. For multidrug resistant P. falciparum the choice of treatment is mefloquine, halofantrine or artemether. In managing sick patients, strict attention should be given to prevention of hypoglycemia, assessment and maintenance of adequate hydration and early institution of dialysis or haemofiltration if acute renal failure develops. Fresh blood transfusion may be required for anaemia or coagulopathy and broad spectrum antibiotics should be given for suspected bacteremia of gastrentestinal or respiratory origin. In malaria associated acute renal failure the factors associated with morbidity are reported to be cerebral involvement and coagulation abnormalities. In brief, despite global efforts to eradicate malaria, it is still causing significant morbidity, complications and mortality. Infection with resistant strains is increasing. Newer therapeutic agents still deserve research. Better understanding of factors that determine the pathogenesis of complications and long-term and continuous efforts towards prevention of disease remains a serious issue to be addressed.
References