Malaria: Persistent Killer: Continuing Renal Complications

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Not long ago, the world seemed poised for victoiy 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 inyoung children in sub-Sahaian Afnca[1]. The pathophysiological disturbances and symptoms of malaria are attributable solely to the asexual erythrocytic forms. Schizogony releases endogenous pyrogens. Endotoxins are absorbed fmm 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 hyperparasiternia. Stagnant anoxemia may be the cause of cerebral malaria and ischaemia of other organs including kidney. Erythmcytes parasitized by certain strains of P. falciparum develop knob like protrusions ontheir surface containing malarial antigens. These adhesions bind to endothelial ligands including thmmbospondin. Damage to vascular endothelium may also contribute to cytoadherance[2]. Tumour necrosis factor (TNF) concentrations are also reported tobe increased dunng malarial illness withvariations in IL,I known to synergize with TNF[3].

The renal involvement in malaria varies widely. The spectrum includes transient glomemlonephritis, nephrotic syndn)me infew cases, mild pmteinuria with urinary sediment changes, abnonnal 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 billiary disease, gastro-intestinal 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 malanas should be treated with pnmaquine 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 dru& viz: pyrimethainine sulphadoxine or tetracycline[1]. For multidrug resistant P. falciparuin the choice of treatment is mefloquine, halofantrine or artemether[1,5]. In managing sick patients, strict attention should be given to prevention of hypoglycemia, assessment and maintenance of adequate hydrationand early institution of dialysis or haemofilteration if acute renal failure develops. Fresh blood transfusion may be required for anaemia or coagulopathy and bmad spectrum antibiotics should be given for suspected bacteraemia of gastmintestinal or respiratory origin. In malaria associated acute renal failure the factors associated with pmgnosis are reported to be cerebral involvement and coagulation abnormalities[5-6]. In brief, despite global efforts to eradicate malaria, it is still causing significant moibidity, complications and mortality. Infection with resistant strains is increasing. Newer therapeutic agents still deserve research. Better understanding of factors. that detennine the pathogenesis of complications and long-term and continuous efforts towards prevention of disease remains a serious issue to be addressed.

References

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