ABSTRACT

Aotus monkeys infected with quartan malaria (*Plasmodium brasilianum*) and injected iv with radiolabeled anti-*P. malariae* IgG and/or normal IgG (no malarial antibodies) showed (a) increased *in vivo* binding of malarial antibodies into abnormally insoluble form when precipitated by 7.5% polyethylene glycol, (b) faster disappearance of this antibody from circulation, and (c) its increased deposition in renal glomeruli. Similar results were obtained in monkeys infected with *P. falciparum* and injected with anti-*P. falciparum* IgG but the difference against controls was much less pronounced. Serum levels of complement components (C3, C3-PA, and C4) rose after infection, and the highest levels coincided with peaks of parasitemia. Decrease to abnormally low levels was detected when antibody was produced.

In Nigerian patients with nephrotic syndrome, results similar to the quartan malaria model in monkeys were found, i.e., increased binding of anti-*P. malariae* IgG with sera *in vitro*, its faster disappearance from circulation after iv injection, and increased deposition in kidney tissue IgG. These data indicate the binding of iv-injected specific antibody to soluble antigens and/or soluble immune complexes of excess antigen type (ñ*in vitro* complexingò) and their increased depositions in kidney (glomerular) lesions. They also strengthen the important role of *P. malariae* in the pathogenesis of chronic progressive nephropathies occurring in malarial areas.