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Letters to the Editor

Hypoglycaemia and cerebral malaria

Sir,

We read with interest Dr Kiire's paper[1] but beg to disagree with him on two points.

It is difficult to see how 44 ring forms of *P. falciparum* per 300 white blood cells could be considered as a strongly positive result, and therefore imply a heavy infection. A leucocyte count over 10,000/μl occurs in under 3% of malaria cases.[2] Nevertheless, if the white blood cell count of Dr Kiire's patient were of this magnitude, his parasite count would be but 1,466 asexual blood forms per microlitre; while this can obviously be associated with a lethal outcome, it cannot be considered as a high, or even medium, degree of parasitaemia in falciparum malaria.

We do not discern the basis for Dr Kiire's assertion that 'This probably represented yet another example of chloroquine failing to save life'. As the patient had apparently vomited most of the chloroquine that had been administered before admission, and as he died 3 hours after admission, there is no firm reason to postulate a lack of effectiveness of chloroquine in this case. By the same token, the author was probably not implicating chloroquine as a cause or co-factor of the hypoglycaemia, although this drug is known to inhibit the degradation of insulin,[3] and has even been used to reduce the dose of insulin employed in a case of severe insulin resistance.[4] However, chloroquine has not been known to cause hyperinsulinaemia in malaria.[5]

Hypoglycaemia in falciparum malaria occurs in two groups of patients: those with severe disease, and/or women who are pregnant, or who have recently delivered,[5] usually (but not always) associated with the therapeutic administration of quinine.[6–9] Cases however have been reported where both quinine and pregnancy were absent; these instances of hypoglycaemia seem to be associated with renal failure, cerebral malaria, or both.[8]

The finding of asexual parasitaemia in a patient with impaired consciousness does not seal the diagnosis of cerebral malaria,[10] and other conditions have to be excluded. The examination of the cerebrospinal fluid is not sensitive or specific enough to diagnose or exclude many causes of coma.[11] Therefore, it is unfortunate that, although an autopsy was performed in this case, no details were given of the central nervous system condition in it.

Falciparum malaria resistant to chloroquine has been reported from Zimbabwe since 1984,[12] and although its extent is presently unknown,[13] the author is probably right in that quinine by infusion should be used in severe malaria cases in areas of resistance to the 4-aminoquinolines.[14] The absence of this is in the circumstances of the case reported, however, cannot be construed as the reason for the patient's death.

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This letter has been shown to Dr Kiire who replies:

Sir,

It is dangerous to extrapolate clinical findings from one area of the world to another. For instance, a leucocyte count over 10,000/μl in malaria cases is not uncommon in this part of the world. This patient had a leucocyte count of 25,000/μl giving a parasite count of 3665 asexual blood forms/μl.

The second point of disagreement is my assertion that this patient's death was yet another example of chloroquine failing to save life. It is my contention that chloroquine would have been inappropriate even if the patient had not vomited or had lived more than three hours after admission. The basis for advocating quinine is that we have chloroquine