CORRESPONDENCE

Malnutrition and Immunity

To the Editor—Peña-Cruz et al. [1] reported an enhancement of severity of Sendai virus infection in malnourished mice. Normal mice experienced no mortality in contrast to 71% mortality in those malnourished, and normal mice abrogated viral shedding 2 days earlier than the malnourished group. These observations parallel those made by Woodruff [2] and Katz and Plotkin [3] >20 years ago. In the Woodruff study, well-nourished mice fared much better than the malnourished ones when both groups were infected with Coxsackie B3 virus. The malnourished group experienced increased mortality and viral shedding. In our study [3], which used herpes simplex virus, in the malnourished mice viremia continued until death, encephalitis was universal, and mortality was 100%. In contrast, only 20% of the well-nourished infected controls died and the survivors abrogated viremia by 42 h. In this group, encephalitis was exceedingly rare. Of interest, normal mice became viremic 12 h earlier than the malnourished animals.

Taken together, these results affirm that malnutrition is a form of acquired immune deficiency. Considering its prevalence among the children of the third world, this form of immune deficiency substantially exceeds the currently more popular cause, AIDS.

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References


Smear-Negative Cerebral Malaria Due to Mefloquine-Resistant Plasmodium falciparum Acquired in the Amazon

To the Editor—Chia et al. [1] are to be commended for considering the diagnosis of malaria in a person returning from a malaria-endemic area with a febrile illness. The failure of physicians to consider the diagnosis of malaria is a frequent cause of morbidity and mortality among persons diagnosed in the United States [2]. The authors present evidence that their patient developed Plasmodium falciparum parasitemia while taking mefloquine prophylaxis. However, the data presented do not support the authors’ conclusion that the patient failed mefloquine treatment and developed cerebral malaria.

Chia et al. state that the patient had documented parasite clearance 1 day after receiving a 1500-mg treatment dose of mefloquine. He then experienced a recurrent febrile illness, throughout which he repeatedly had negative blood smears for malaria parasites. In the absence of persistent or recurrent parasitemia, malaria could not have been the diagnosis, and consequently, an infection with a mefloquine-resistant parasite is not an issue. Any proposition of mefloquine resistance must be supported by demonstration of Plasmodium falciparum parasitemia in addition to confirmation of an adequate drug dosage.

All accepted case definitions of cerebral malaria require the presence of Plasmodium falciparum parasitemia [3, 4]. Smear-negative cerebral malaria has been reported, but it is extremely rare and has been accompanied by the finding of Plasmodium falciparum parasites in the cerebral vasculature at autopsy [3]. Smear-negative cerebral malaria may occur in a synchronously replicating Plasmodium falciparum infection, with parasites completely sequestered in capillary beds at the time of the blood film examination. However, after rupture of the sequestered schizonts into the bloodstream, young trophozoites circulate freely in erythrocytes, and blood smears repeated at 6- to 12-h intervals will invariably reveal parasites. Parasites were never demonstrated after initial treatment in the case reported by Chia et al. [1].

A further requirement for the diagnosis of cerebral malaria is the exclusion of other causes of neurologic impairment [3, 4]. In this case the lumbar puncture results demonstrated an elevated cerebrospinal fluid protein and lymphocytic pleocytosis, both of which are inconsistent with the diagnosis of cerebral malaria and suggest other illnesses. Neither the magnetic resonance imaging results, the apparent clinical response to antimalarial therapy, nor the negative virus titers constitute adequate evidence to incriminate Plasmodium falciparum as the cause of the patient’s illness.

In conclusion, we agree that Plasmodium falciparum infection needed to be considered as a cause of the patient’s febrile illness. However, the multiple negative blood smears over 3 weeks and the abnor-