Corticosteroids for Watershed Infarction in Acute Schistosomiasis

TO THE EDITOR—Acute schistosomiasis may be associated with watershed infarction. Corticosteroids alone are rapidly effective, as illustrated in this report.

A 19-year-old French man was admitted with a 15-day history of high fever (39°C), 15-kg weight loss, cough, arthralgias, and urticaria. Two weeks earlier he had returned from Kenya, where he bathed in Lake Victoria every week during a 3-month period. Blood eosinophils were high (4800/mm³); Schistosoma serology was positive (Fumouze indirect hemagglutination assay and Bordier Affinity Products enzyme-linked immunosorbent assay). Schistosoma eggs were not found in urine or feces. Electrocardiography showed diffuse T-wave inversions, and troponin levels were elevated (1 μg/L; normal level <0.1), suggesting myocarditis. However, cardiac magnetic resonance imaging (MRI) was normal. Within 8 days the patient recovered spontaneously, with the disappearance of urticaria, cough, and fever. His eosinophilia decreased to 3100/mm³, inverted T-waves resolved, and troponin levels returned to normal limits.

However, 4 days later he presented with ataxia, confusion, and right-sided dysmetria. His eosinophil count increased to 11 950/mm³. Cerebrospinal fluid was normal. Brain computed tomography was normal. Brain MRI revealed multiple high-signal lesions on T2 fluid-attenuated inversion recovery (FLAIR) and restricted diffusion, predominantly distributed in the border zone of the cerebral arteries (Figure 1), suggesting watershed infarction.

Prednisolone (2 mg/kg/day) was administered and led to full clinical recovery within 5 days. The prednisolone was tapered gradually and discontinued within 1 month. Eggs of Schistosoma mansoni appeared in the stools 4 months after the beginning of the fever. Praziquantel (4.8 g on day 1, repeated on day 7) was then prescribed and effectively eradicated the infection.

Watershed infarction is a very rare complication of acute schistosomiasis: 7 cases have been described mostly among tourists in Mali (Schistosoma haematobium) and Madagascar (S. mansoni) [1–5]. Patients present with signs of encephalopathy (confusion, ataxia, dysphasia), sometimes with urticaria or myocarditis. Eosinophilia is high, and cerebrospinal fluid analysis is normal. MRI notes multiple high-signal lesions on T2-weighted and FLAIR images, predominantly distributed in the border zone of the cerebral arteries, suggesting watershed infarction. Corticosteroids have a rapid effectiveness (a few days), as in the case of our patient.

One patient presented with encephalopathy signs a few days after a praziquantel treatment [1], suggesting a role
of Schistosoma lysis with immunological reaction. Likewise, clinical worsening just after praziquantel treatment during acute schistosomiasis has been described in 40% of patients [6].

Eosinophils are toxic for endothelial cells, and this effect is impaired by methylprednisolone [7]. Moreover, eosinophils promote thrombosis by impairing thrombomodulin function.

Interestingly, other diseases with hypereosinophilia (trichinellosis, idiopathic hypereosinophilic syndrome [8]) are also complicated with watershed infarction. Cerebral biopsy in neurotrichinosis revealed distal cerebral vasculitis (with deposits of eosinophils [9]) and arteriolar fibrinocruoric thrombi [10].

Therefore, a conjunction of eosinophil-associated vasculitis and micro-thrombi could probably explain the watershed infarction. Nevertheless, most patients improve rapidly with corticosteroids alone (without antiaggregant or anticoagulant therapy), suggesting that distal vasculitis is probably the most important phenomenon.

Notes

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