Images in Neurology

Extensive Cerebral Microhemorrhages Caused by Acute Disseminated Intravascular Coagulation Secondary to Sepsis

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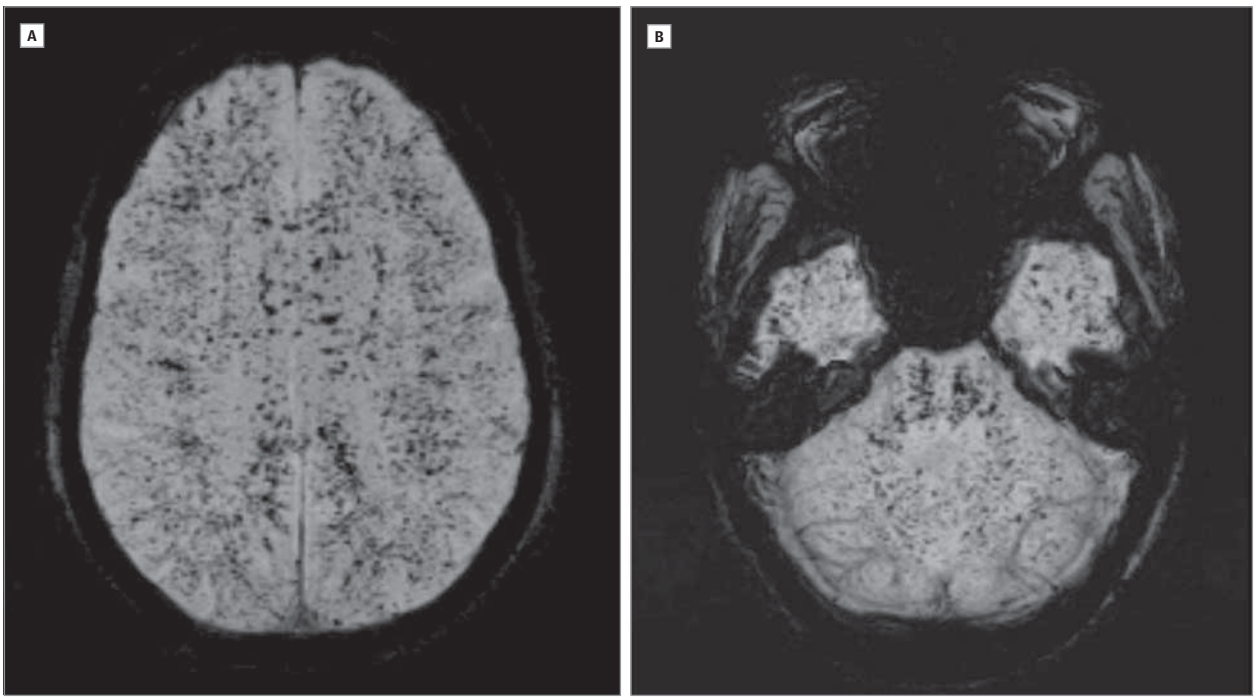
A 55-year-old man with a history of sickle cell disease (sickle cell hemoglobin C) but no history of sickle cell crises was seen with an episode of acute abdominal pain and fever. He had no significant medical history, including no antecedent neurologic problems. He became unresponsive during 24 hours, necessitating intubation and ventilation. On examination before intubation, he was febrile, anarthric, and quadraparetic. In addition, he was unable to track objects in his visual fields or follow simple commands. His blood test results were consistent with disseminated intravascular coagulation (9.9 g/dL hemoglobin level, 35 $\times 10^3$/μL platelet count, 1.7 international normalized ratio, 5928 U/L lactate dehydrogenase level, 42 000 μg/mL D-dimer level, and 100 mg/L C-reactive protein level), which was thought to have occurred secondary to sepsis (to convert hemoglobin level to grams per liter, multiply by 10.0; platelet count to $\times 10^9$/L, multiply by 1.0; lactate dehydrogenase level to microkatals per liter, multiply by 0.0167; D-dimer level to nanomoles per liter, multiply by 5.476; and C-reactive protein level to nanomoles per liter, multiply by 9.524). Results of initial brain computed tomography before and after intubation were normal, but subsequent brain magnetic resonance imaging (MRI) (T2-weighted * susceptibility-weighted imaging [SWI]) revealed extensive microhemorrhages supratentorially and infratentorially (**Figure**). He was managed supportively in the intensive care unit for a few weeks. Although initially following discharge from the intensive care unit he was anarthric and intermittently responsive to external stimuli, he had a significant cognitive and physical recovery during 2 months, following which he was discharged to a specialist center for further cognitive rehabilitation.

Discussion

The brain MRI demonstrated widespread brain microhemorrhages affecting the cortex, subcortex, brainstem, and cerebellum, establishing the cause of this patient's neurologic presentation. We postulate that the microhemorrhages were a consequence of disseminated intravascular coagulation and not of his underlying sickle cell condition[1] because sickle cell disease is typically associated with a large-vessel occlusive vasculopathy in adults, which can be silent.[2] In addition, the absence of neurologic and, in particular, cognitive dysfunction[3] before his presentation would strongly suggest that the MRI changes were acute and not long-standing.

Figure. Extensive Microhemorrhages



Demonstrated on susceptibility-weighted imaging supratentorially (A) and infratentorially (B).

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To date, the only case report[4] of cerebral microhemorrhages in a patient with sickle cell disease was a consequence of fat embolism following an acute coronary syndrome.[4] Indeed, the prevalence of cerebral microhemorrhages in the context of sepsis or other systemic illness is unknown. The advent of SWI-MRI has enabled the evaluation of brain microhemorrhages.

It is speculative whether the observation reported herein is underrecognized. The increasingly widespread use of SWI-MRI may assist in determining the prevalence of acute cerebral microhemorrhages with systemic illness. Whether individuals with sickle cell disease are more susceptible remains unknown.

In conclusion, this case highlights the importance of detailed MRI in patients with unexplained neurologic and cognitive dysfunction in the context of systemic disease. In particular, SWI is useful to evaluate brain microhemorrhages.

ARTICLE INFORMATION

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