Basal Ganglia Infarction Complicating Basilar Meningitis: Visualization by Serial Magnetic Resonance Imaging

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Dear Sir,

We report a 10-year-old with a 10-day history of headache and photophobia. Four days prior to admission to the Children's Hospital, fever above 39°C persisted. There was marked neck stiffness. Neurologic examination was otherwise normal. There was no postnasal drip. Laboratory evaluation showed a peripheral white blood cell count of $17 \times 10^9/l$, 18.0% band forms, 63% segmented neutrophils. The erythrocyte sedimentation rate was 72 mm/h. Cerebrospinal fluid (CSF) analysis revealed a white blood cell count of 4,567/mm³ with 4,050 polymorphonuclear and 517 mononuclear cells. CSF glucose was 0.3 mmol/l (1.6–3.9 mmol/l), CSF protein was 1.51 g/l (<0.44 g/l), CSF pressure was normal. No organisms were identified on gram stain. Antimicrobial therapy with flucloxacillin, ceftriaxone and ornidazole was initiated. Despite the therapy, fever persisted and MRI showed left-sided sphenoid sinusitis and a temporal subdural empyema with meningeal enhancement (fig. 1a). In the left basal ganglia, enhancement was noted (fig. 1b). Further imaging showed progressive evolution. During hospitalization, the patient became lethargic and sleepy but was responsive, never comatose; there were never any focal neurological deficits and

signs. The temperature remained between 39 and 39.5°C until the 10th day of the therapy and the patient was discharged home after 23 days of parenteral antimicrobial therapy. No surgical intervention was required. Neurologic examination on discharge was normal. The diagnosis on discharge was: basilar meningitis with cerebral vasculitis and stroke following sphenoid sinusitis. A follow-up MRI 39 days after hospital admission demonstrated regression of the temporal empyema and resolution of the sphenoid abnormality. Subacute left basal ganglia infarction was visualized (fig. 2a, b). The patient was no longer lethargic. MRI visualizes the location, extent and intensity of intracranial inflammatory processes. After the initiation of antimicrobial therapy, a temporary increase in inflammatory signs is often demonstrated by MRI. This is not disease progression, but probably represents the inflammatory host reaction mediated by antigen release induced by the antimicrobial therapy. The inflammatory stimulus leads to an increase in the water content, mediated by disruption of the blood-brain barrier and direct toxic effects. Even though the increase in the water content rarely exceeds 10–20%, it allows the visualization of inflammation by MRI [1].

The increase in the water content is more pronounced in the white matter than in the cerebral cortex. Two mechanisms may have been implicated in the basal ganglia lesion in the case presented here: (a) an infection with spread of bacteria along the vessels, or (b) an infarction secondary to vasculitis [2]. The latter possibility appears more likely because the pattern of enhancement corresponds to that seen with luxury perfusion. Cerebrovascular complications of meningitis include vasculitis, e.g. in tuberculosis [3], vasospasm, venous and arterial thrombosis [4–6] affecting both small and distal branches of cerebral vessels [7], Haemophilus infection [1], *Candida albicans* [8], and coccidioid meningitis [9]. The presence of persistent focal neurological deficits secondary to a stroke is associated with poor outcome [3]. The case presented here illustrates the clinical utility of MRI in the early visualization of cerebral ischemia and infarction complicating inflammatory damage to supplying blood vessels in the basilar subarachnoid space. Indeed, this report shows that when confronted with fever and slight lethargy but no focal deficits, intracerebral inflammation can be detected and monitored and not simply documented as before.

Fig. 1. **a** Coronal contrast-enhanced T₁-weighted image showing left-sided sphenoid sinusitis as well as the presence of a temporal subdural empyema on the left side with accompanying basal meningeal enhancement. **b** The presence of an enhancement in the left basal ganglia close to the third ventricle. The subdural empyema is now smaller.

Fig. 2. **a** At 1 month, the contrast-enhanced T₁-weighted MRI shows regression of the temporal empyema as well as of the basal ganglia changes; there is only an area of hypointense infarction. **b** On T₂-weighted imaging, the left temporal white matter is now also of normal intensity.

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