

Fatal Cerebral Vasculitis Following Streptococcus Pneumoniae Meningitis

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Clinical Image

A 68-year-old man was admitted to the Emergency Department for fever and altered consciousness (Glasgow Coma Scale (GCS) score 12/15, E4V2M6). Neurological examination revealed neck stiffness, aphasia, dysarthria and left-sided hemiplegia. Lumbar puncture yielded cloudy cerebrospinal fluid (CSF) with white blood cells (WBC) 282/mm³, neutrophils 92%, protein 857 mg/dL, glucose < 3 mg/dL, lactate 27.2 mmol/L. Contrast-enhanced brain computed tomography (CT) was unremarkable. Blood and CSF culture isolated penicillin-sensitive Streptococcus pneumoniae. The patient remained hemodynamically stable. Treatment with ceftriaxone (2000 mg twice a day) was started for a total duration of 14 days, in association with dexamethasone 10 mg four times daily over the first 4 days. Neurological improvement was noted within the first 48 hours, with a regression of aphasia and hemiplegia. An initial brain magnetic resonance imaging (MRI)

on day 6 failed to reveal significant ischemic lesions even on diffusion-weighted views. However, neurological status gradually worsened from day 7 and a follow-up brain MRI was performed on day 12 when GCS was 3/15 and revealed meningeal thickening and extensive ischaemic injury of the brain parenchyma (Figure 1). Palliative care was decided. Delayed cerebral ischemia is an uncommon complication of S. pneumoniae meningitis which may occur during the second week of treatment in some patients who showed initially a clinical improvement after the initiation of the antimicrobial therapy [1]. Several processes have been hypothesized to explain the condition, including cerebral vasculitis, immune-mediated microangiopathy, rebound effect of the primary inflammatory reaction which had been initially suppressed by dexamethasone, and hypercoagulability [2,3]. The reintroduction of corticosteroids is followed by inconstant results and the final neurological prognosis is usually poor.

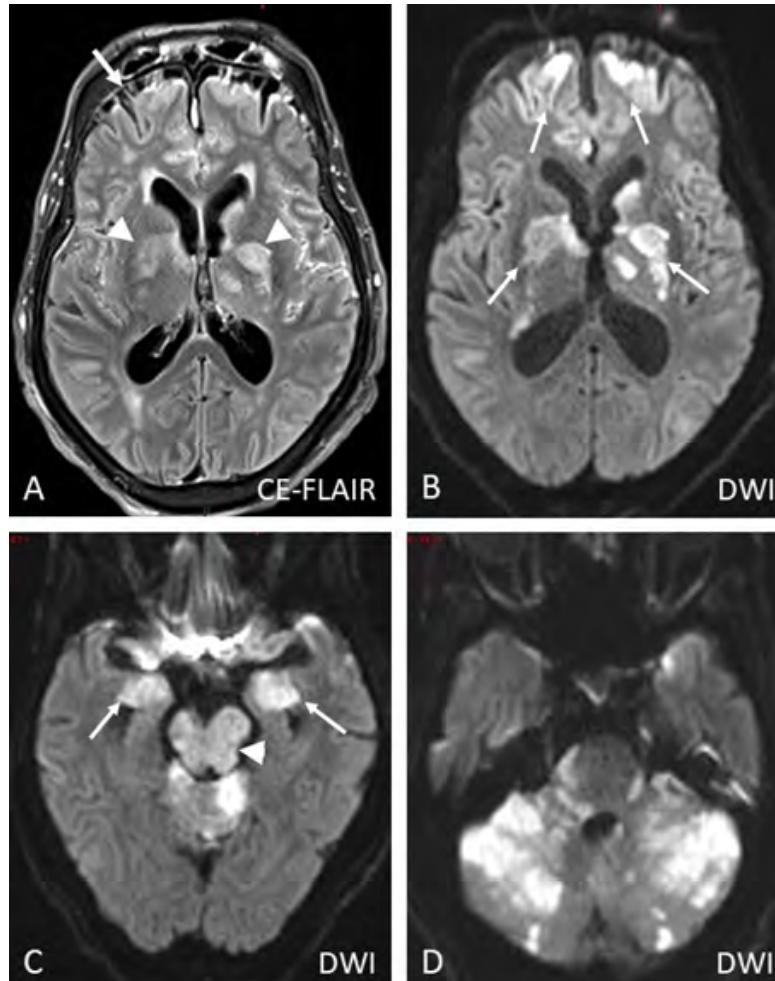


Figure 1: Magnetic resonance (MR) examination at critical phase

Figure 1A: transverse contrast enhanced (CE) fluid-attenuated Inversion-Recovery (FLAIR) view showing strongly abnormal enhancement of pial bridging veins (arrow) and ischaemic injury of central grey nuclei (arrowheads).

Figure 1B: transverse diffusion-weighted (DW) view in similar slice location as A best depicting ischaemic cytotoxic oedema within frontal cortex and central grey nuclei (arrows)

Figure 1C: transverse DW view revealing complete infarction of the mesencephalon (arrowhead), together with that of temporal amygdalian gyri (arrows) and of upper part of the cerebellar vermis (not arrowed)

Figure 1D: transverse DW view revealing huge ischaemic injury of both cerebral hemispheres and at a lesser degree of the pons

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