
Neuroimaging Highlight

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Reversible Parinaud Syndrome Following Intraventricular Thrombolysis

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A 57-year-old daycare educator presented as a drowsy but oriented individual with a history of sudden and severe headache associated with vomiting. She had no previous medical or neurological history. Examination showed no focal signs and routine laboratory studies were unremarkable. Head computed tomogram (CT) revealed a Fisher grade IV subarachnoid hemorrhage in the posterior fossa with extensive intraventricular hemorrhage (Graeb 8/12, see Figures 1A and 1B) which was shown to originate from a left Posterior Inferior Cerebellar Artery (PICA) aneurysm on CT angiography and treated

successfully with endovascular embolization. Five days later she deteriorated her level of consciousness (Glasgow coma scale [GCS] 8/15). The CT scan showed moderate hydrocephalus and a ventricular drain was placed. She improved clinically but remained disoriented with slowed information processing skills.

On the 6th day of admission, her neurological examination revealed a dorsal midbrain syndrome, or Parinaud's syndrome: she presented with impaired upgaze (loss of voluntary saccades and pursuit movements with preservation of the vestibulo-ocular reflex), lid retraction (or Collier's sign, see Figure 2), and

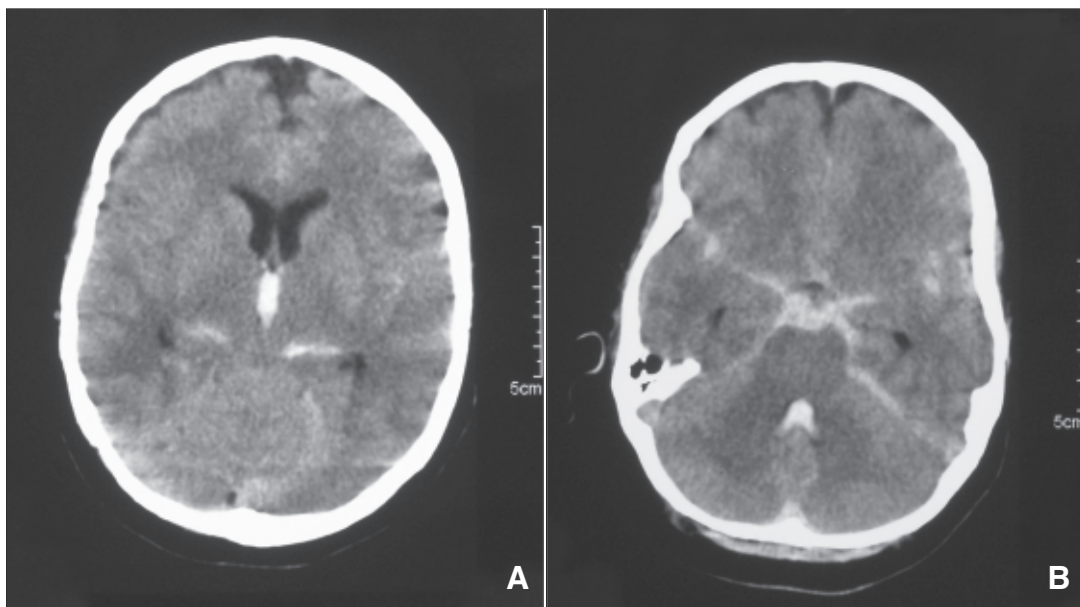


Figure 1: A. Axial non-enhanced head CT showing a Fisher grade IV subarachnoid hemorrhage with intraventricular hemorrhage and mild hydrocephalus. B. Axial non-enhanced head CT showing fourth ventricle hemorrhage.

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Figure 2: Collier's sign characterized by lid retraction.

moderate pupils with light-near dissociation (loss of light reaction with preservation of pupilloconstriction in response to a near target).¹ There was no convergence-retraction nystagmus although examination of this sign was suboptimal. These neurological signs suggested a lesion of the dorsal midbrain (including the posterior commissure), a bilateral lesion of the pretectal region, or a large unilateral tegmental lesion. Although in her age group this syndrome could have been due to stroke, an arteriovenous malformation, or multiple sclerosis, our main hypothesis was a 3rd ventricle compression due to hydrocephalus and her ventricular drain was opened to 0 cm H₂O. On day nine, and in light of a persistent Parinaud with low normal intracranial pressures (0-2 mm Hg) and stable values from her open ventricular drain, magnetic resonance image (MRI) was performed to eliminate a vascular dorsal midbrain lesion. The origin of her Parinaud is shown in Figure 3. Indeed, a T1 and T2 hyperintensity compatible with a large blood clot (2 cm long by 0.8 cm transverse by 0.6 cm thick) was visualized immediately

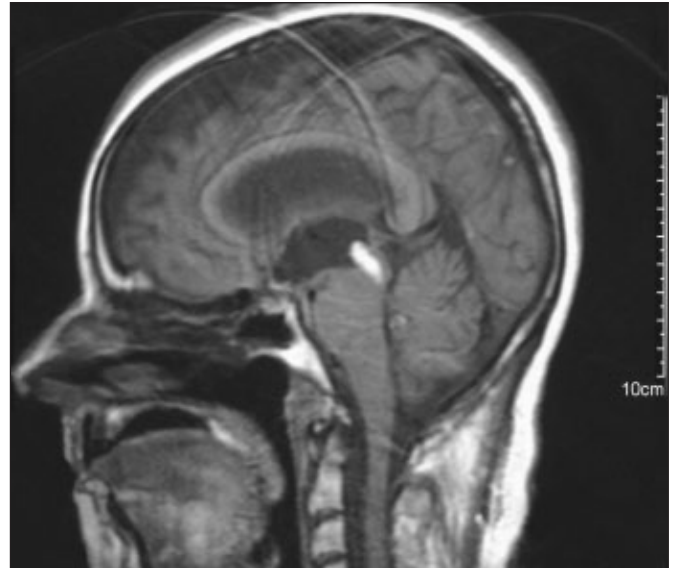


Figure 3: Non-enhanced sagittal T1-weighted MRI showing hyperintense blood clot in the third ventricle at the entrance of the cerebral aqueduct and mild hydrocephalus. There was no sign of ischemia.

at the entrance of the cerebral aqueduct, likely compressing the midbrain tectal plate. There was no sign of ischemia. We proceeded to intraventricular thrombolysis using recombinant tissue plasminogen activator (rt-PA) administered over three consecutive days (total dose of 5 mg). Her Parinaud's syndrome

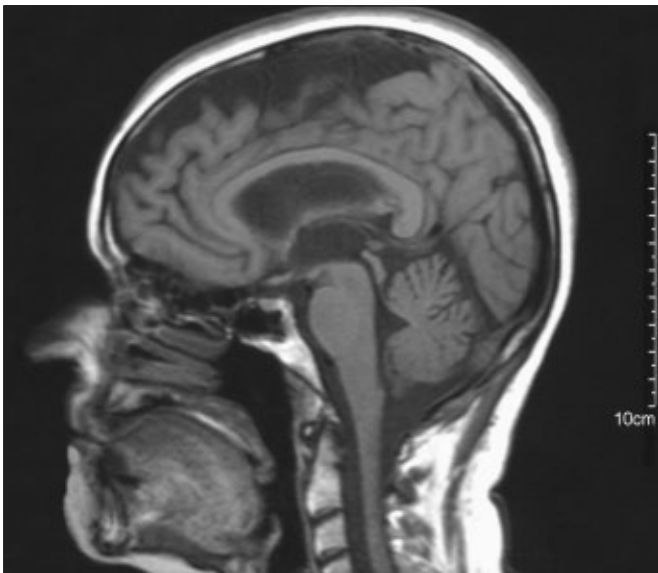


Figure 4: After intraventricular thrombolysis, non-enhanced sagittal T1-weighted MRI shows complete regression of the clot and partial regression of hydrocephalus.

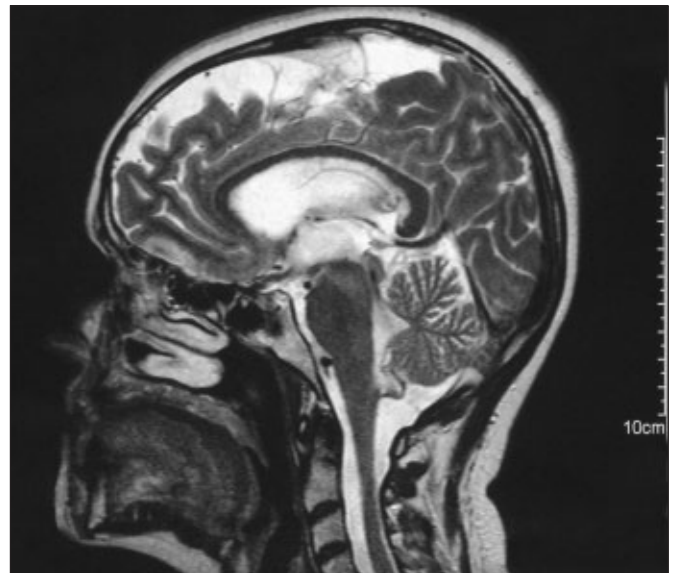


Figure 5: After intraventricular thrombolysis, enhanced sagittal T2-weighted MRI shows an actual flow void in the cerebral aqueduct providing further evidence for complete regression of the clot and permeability.

entirely resolved within 48 hours of the last dose. Control MRI showed partial regression in her hydrocephalus, absence of intraventricular clots and a permeable cerebral aqueduct (Figure 4). Furthermore, there was an actual flow void in the cerebral aqueduct on enhanced sagittal T2-weighted MRI providing further evidence for complete regression of the clot and permeability of the cerebral aqueduct (Figure 5).

A meta-analysis conducted in 2000 suggested that treatment with ventricular drainage combined with fibrinolytics may improve outcome in patients with severe intraventricular hemorrhage caused by extension from subarachnoid hemorrhage.² This study was based on indirect comparisons between observational studies. Since then, safe and efficient use of intraventricular rt-PA after endovascular coiling in patients with ruptured aneurysms and massive intraventricular hemorrhages have been reported, but large randomized trials are still lacking.³⁻⁴ Although there are no guidelines for intraventricular hemorrhage thrombolysis at the moment, our review of the literature suggests that given the appropriate clinical context, a Graeb score ≥ 7 , a clotted extraventricular drain or an intracranial pressure > 15 , and a GCS < 8 are fair indications.⁵ We believe the results from the ongoing Intraventricular Hemorrhage Thrombolysis Clinical Trial will help define the latter indications.⁶ As to our case, we felt that her clinical and radiological presentation prompted an aggressive treatment. In addition to being the first case of a Parinaud's syndrome caused by a cerebral aqueduct clot, to our knowledge this is the first demonstration of a reversible Parinaud following intraventricular thrombolysis.

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