

## PSEUDO-SUBARACHNOID HEMORRHAGE SIGN ASSOCIATED WITH SEVERE CEREBRAL EDEMA

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**Key-word:** Brain, edema

**Background:** A 34-year-old caucasian male underwent laparoscopic gastric bypass. The procedure was complicated by leakage and secondary peritonitis with subsequent shock and diffuse intravascular coagulation. He underwent re-intervention with splenectomy, subtotal colectomy and resection of the distal ileum. Moreover, he developed acute kidney failure necessitating hemodialysis. A few hours after the dialysis, the patient developed bilateral non light-responding mydriasis.

A CT scan of the brain was performed.

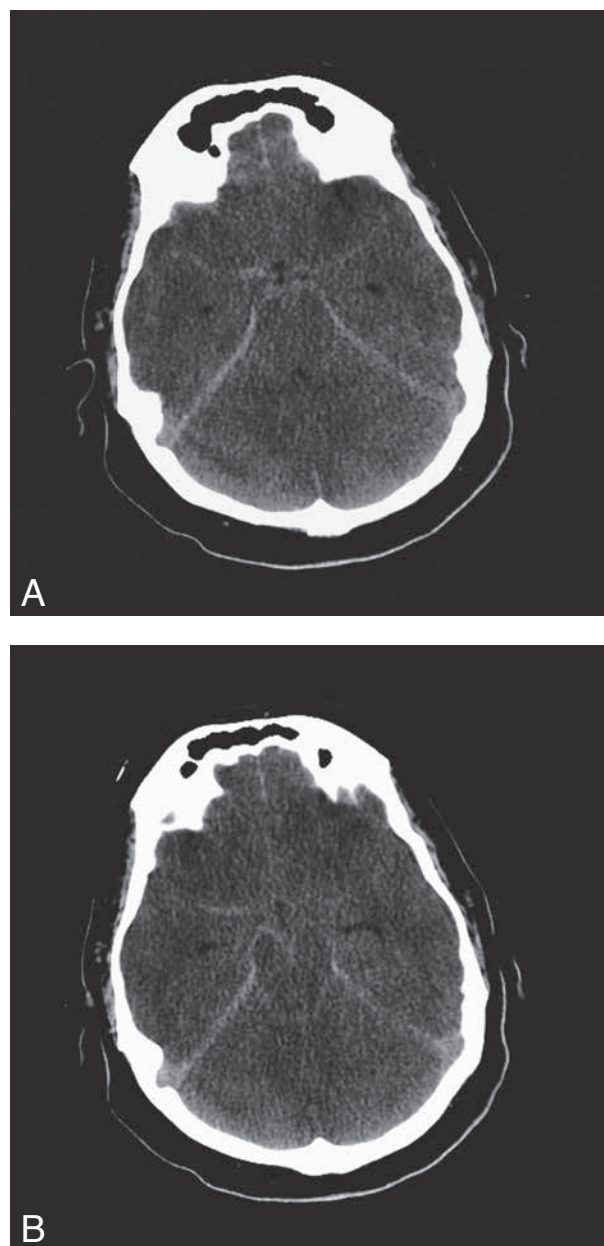


Fig.  $\frac{1A}{1B}$

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## Work-up

Unenhanced CT scan of the brain (Fig. 1) shows on A a hypodense aspect of the brain parenchyma, effacement of the cortical sulci, narrowed ventricles, hypodense aspect of the cerebellum and blurring of the basal cisternae. The tentorium appears relatively hyperdense, compared to the hypodense cerebellar and cerebral parenchyma, and the middle and anterior cerebral artery show increased attenuation, resembling subarachnoid hemorrhage as can be seen secondary to aneurysm rupture.

On B, the middle and posterior cerebral artery show increased attenuation, resembling subarachnoid hemorrhage, hence known as the 'pseudo-subarachnoid hemorrhage sign'.

## Radiological diagnosis

Based on the unenhanced CT findings, including 'the pseudo-subarachnoid hemorrhage sign', the diagnosis of *severe cerebral edema* could be made.

Misled by this imaging sign, a CT angiography of the circle of Willis was performed, only showing the proximal part of the great vessels. Repeat contrast-enhanced scan of the brain parenchyma showed prolonged arterial contrast enhancement, suggestive of underlying venous congestion.

Administration of contrast medium in this case should have been avoided.

In this particular case, dialysis disequilibrium syndrome was considered to be the underlying etiology.

## Discussion

Dialysis disequilibrium syndrome (DDS), defined initially by Kennedy et al, is an acute neurological complication of hemodialysis, characterized by signs and symptoms of cerebral edema. Ultimately, it can lead to death. The pathogenesis is based on a reversed osmotic gradient of ureum in the brain, promoting water movement into the brain and inducing development of secondary cerebral edema.

The 'pseudo-subarachnoid hemorrhage' sign (SAH) in brain edema was firstly described by Spiegel et al in 1986.

Increased attenuation within the basal cisterns and subarachnoid spaces on CT scans is a characteristic

finding of acute subarachnoid hemorrhage. The pseudo-SAH appearance may be seen in patients with acute neurologic deficits in whom other evidence of diffuse cerebral edema is present at CT examination.

With development of cerebral edema, the increase in intracranial pressure and swelling of the brain narrow the subarachnoid spaces and displace the cerebrospinal fluid (CSF). The increased intracranial pressure causes congestion and dilatation of the superficial venous structures. The resultant subarachnoid spaces lose the hypoattenuated CSF and fill with a larger fraction of meninges and blood vessels than in the normal state, potentially increasing their CT attenuation.

In case of brain swelling due to acute hypoxic encephalopathy, the increased average tissue density immediately deep to the dura and superficially within the cerebral sulci results in hyperdensity on unenhanced CT, simulating the appearance of SAH.

Radiologists should be aware of this potential mimic of SAH when evaluating patients with cerebral edema.

## Bibliography

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