Neurovascular Observation
Hydrocephalus due to superior sagittal sinus thrombosis

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Summary
The great variability of clinical appearance is one of the main features of superior sagittal sinus thrombosis. However, hydrocephalus associated with cerebral sinus thrombosis is rare. We report on a patient presented with thunderclap headache, accompanied by nausea, vomiting, and drowsiness. Lumbar puncture ruled out subarachnoid hemorrhage, whereas CT revealed marked hydrocephalus. In addition, magnetic resonance venography then confirmed the diagnosis of cerebral sinus thrombosis. It is a rare occurrence but clinically important, since it entails disastrous sequels if unrecognized, and hydrocephalus is treated in the usual fashion with ventricular drainage.

Keywords: Hydrocephalus; superior sagittal sinus; thrombosis.

Introduction
Hydrocephalus is a condition being frequently treated in neurosurgical practice. The clinical and radiological appearance of chronic hydrocephalus are well-described as the features of subacute communicating hydrocephalus following subarachnoid hemorrhage (SAH) or trauma [2]. Likewise the various forms and etiologies of acute obstructive hydrocephalus are well-known entities. However, review of the literature yielded only one case report of cerebral venous thrombosis (CVT) as a cause of hydrocephalus in man [11]. Though it is of rare occurrence, clinical value is significant since it entails disastrous sequels if unrecognized, and hydrocephalus is treated in the usual fashion.

Case report
A 56-year-old man presented with a two week history of progressive headache with abrupt onset within a few seconds and with nausea, vomiting and varying impairment of consciousness. There was no previous head injury, and the patient’s medical history was completely insignificant, particularly with regard to signs and symptoms of normal pressure hydrocephalus. Specific factors precipitating venous thrombosis were excluded by the initial laboratory and clinical examinations, such as dehydration, diarrhoea, malignancy, inflammatory diseases, blood dyscrasias, and coagulopathies.

On neurological examination the patient was drowsy. Pupillary reflexes, fundoscopy as well as deep tendon reflexes, plantar responses, motor function of the limbs and sensory examination were unremarkable. Seven hours later, the patient had a generalized seizure and remained somnolent thereafter. SAH was ruled out by lumbar puncture. CSF analysis showed a clear fluid, 1 lymphocyte and a protein content of 36 mg%. Cytological examination disclosed neither erythrocytes nor siderocytes. CSF opening pressure was not evaluated. However, CT revealed a marked hydrocephalus (Fig. 1a–c). Owing to ventricular enlargement and apparent clinical signs of raised intracranial pressure, ventricular drainage appeared to be indicated.

A combined device for simultaneous ventricular drainage and monitoring of intracranial pressure (ICP) was implanted in a typical fashion, and the operation was completed without any complications. Though function of the drainage proved to be sufficient, the patient failed to improve postoperatively. He remained somnolent and showed clinical signs of ICP. However, continuous ICP-monitoring did not reveal either plateau waves or B-waves. Two days after surgery, sudden neurological deterioration was noted. The patient became comatose and presented a left-sided fixed and dilated pupil, a right-sided hemiplegia, an inappropriate response to painful stimuli, and a Cheyne Stokes respiratory pattern. Emergency CT revealed a left-sided hemorrhage of the central and parietal region and, moreover, appearance of a typical “empty triangle sign” (Fig. 2). Subsequent magnetic resonance venography (MRV) confirmed the tentative diagnosis of superior sagittal sinus (SSS) thrombosis. Furthermore, the right transverse sinus and some cortical veins proved to be occluded as well (Fig. 3). The patient remained ventilated and was immediately commenced on systemic heparinization. Although a target INR of 2.5 was obtained, follow-up CT three days later showed starting re-absorption of the initial hemorrhage but also development of new hemorrhagic lesions within the left frontal and occipital lobes (Fig. 4). During the following four weeks, the patient improved only gradually. The size of the ventricular system did not show any tendency to decrease and therefore placement of a shunt was considered. Nine weeks after the initial event the patient was
S. Weidauer et al.

**Discussion**

The great variability of clinical appearance is one of the main features of SSS thrombosis [2, 5, 10, 21]. In addition, CVT can present with the typical symptoms for idiopathic intracranial hypertension (IIH) [2, 10, 13]. It may be well tolerated owing to possibly sufficient collateral venous pathways but may also prove to be a life-threatening condition [5, 13]. Moderate headache, seizures, mental disturbances, and focal neurological deficits are well-known symptoms which might culminate in coma and a fatal outcome due to increasing

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**Fig. 1.** Preoperative axial CT showing marked hydrocephalus at the level of the third ventricle (b) and the cella media of the lateral ventricles (c) without a suggestion of a space occupying lesion in the posterior fossa (a). The top slices (c) revealed a partially slight hyperdense sagittal sinus (arrow).

**Fig. 2.** Postoperative contrast-enhanced CT demonstrated a left-sided hemorrhage and appearance of “empty triangle sign”. The small filling defect (arrowhead) and the enlarged collateral channels in the wall of the sinus (arrow) suggests subacute or chronic thrombosis.

**Fig. 3.** MRV (3D time-of-flight (TOF); lateral view) disclosing thrombosis of the SSS without any flow-signals in the anterior part and large filling defects in the distal part (arrows).

**Fig. 4.** Follow-up CT showing emergence of new left-sided frontal hemorrhagic lesions (large arrowhead) and persistence of “empty triangle sign” (arrow and small arrowhead; see also Fig. 2) suitable for further rehabilitative treatment. On discharge, he was lethargic, had a right-sided hemiparesis of motor-grade III and a global aphasia.