

## Clinical Notes

### Spontaneous bilateral subacute subdural hematoma revealing intracranial hypotension

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Bilateral subdural hematoma is one of the major complications of spontaneous intracranial hypotension. It is a rare, but serious condition resulting from tearing of bridging veins or rupture of the dilated thin-walled blood vessels in the subdural space.<sup>1</sup> Most of these subdural hematomas can be managed with treatment directed to the underlying spinal CSF leak.<sup>2</sup> However, surgical drainage by craniotomy may be required depending on the clinical status of the patient. In this report, we aim to describe a case of spontaneous subacute bilateral subdural hematoma revealing intracranial hypotension in a young patient, and to discuss the pathogenesis as well as the treatment modalities of this disease.

Our patient is a 20-year-old man that presented to the emergency department with a history of progressive diffuse headache occurring 2 weeks earlier, associated with nausea, diplopia, visual fog, and bilateral papilledema. There was no history of connective tissue disorders, trauma, or drug intake, especially anticoagulant therapy. On admission, he was lethargic with stable vital signs. His Glasgow coma scale was 13/15. The neurological exam discovered a tetra pyramidal syndrome associated to left sixth nerve paralysis with convergent squint. The general examination was normal. The cerebral CT scan showed a bilateral sided hyperdense subdural collection over the convexity of the hemisphere. The collection was considerably larger on the left side causing a slight left-right shift of the interventricular septum (Figure 1). Biochemical investigation revealed no abnormality, and cerebral angiography was normal. He underwent an evacuation of the bilateral subdural hematoma as these were considered the cause of obtunding. At the postoperative stage, he became perfectly conscious, but the headaches did not disappear completely. A CT scan of control showed a good evacuation of the subdural hematoma with no other abnormalities. The next day, the headaches increase in intensity justifying a lumbar puncture CSF. This was carried out in the sitting position at the L4-L5 level, and showed a decreased intracranial opening pressure at 50 mm H<sub>2</sub>O with increased white blood cells and red blood cells. Biochemical examination of the CSF showed lymphocytic pleocytosis and elevated protein content (1200 mg/dL, normal: 300 mg/dL). Spinal and myelo-MRI failed to demonstrate a CSF leak. He then underwent a lumbar autologous epidural blood patch (EBP) in the Trendelenburg position with injection of 30 ml of blood mixed with

one ml of gadolinium. The injection was stopped when he reported an increased pressure sensation in the neck. Subsequently, he reported a significant improvement of the headaches and was discharged 4 days later on bed rest and limited activity. After 3 years of follow-up, he is in good health, with no recurrence of the headache.

Acute subdural hematoma is commonly associated with a brain contusion or tearing of the bridging veins in the subdural space that usually occurs after a severe cranial trauma. Rarely, it can occur spontaneously. Then, multiple etiologies are possible such as hypertension, arterial bleeding from the dural fistula, cerebral venous thrombosis, arterio-venous malformation, tumor, amyloid angiopathy, inflammatory disease, coagulation disorders, or dehydration.<sup>1</sup> In our patient, the CT scan has excluded an obvious tumor. The biochemical investigation revealed no inflammatory or hematological disorder, and amyloid angiopathy does not occur in young adults. Elsewhere, there was no sign of dehydration. Subdural fluid collections occur in approximately 50% of patients with spontaneous intracranial hypotension (SIH).<sup>2</sup> Most of these collections represent hygromas and are thin, bilateral, located over the cerebral convexities, and do not cause significant mass effect. Subdural hematomas are also usual in SIH and are around half as frequent as subdural hygromas.<sup>1</sup> The MRI has revolutionized the understanding and diagnosis of SIH.<sup>3</sup> The 5 characteristic imaging features of SIH on MRI are 1) subdural fluid collections, 2) enhancement of the pachymeninges, 3) engorgement of venous structures, 4) pituitary hyperemia, and 5) sagging



**Figure 1** - Axial cerebral CT scan demonstrating a spontaneous bilateral hyperdense subdural hematoma noticeably larger on the left side (arrows).

of the brain. Most of these features can be explained as compensatory changes related to the loss of CSF volume. Indeed, several options are available to treat patients with symptomatic SIH. A purely conservative approach consists of bed rest, oral hydration, administration of steroids, and use of an abdominal binder. However, symptoms may be debilitating, and more timely results may be desired. The mainstay of treatment is the injection of autologous blood into the spinal epidural space, the so-called EBP.<sup>4</sup> The relief of symptoms is often instantaneous, thereby also serving a diagnostic purpose. If the EBP is unsuccessful it can be repeated, and consideration should be given to a large-volume of injected blood (20-100 ml). Surgical repair of CSF leak is reserved for those patients in whom these non-surgical measures have failed. It consists of direct suture or percutaneous placement of fibrin sealant.<sup>5</sup> Outcome studies have shown that patients with abnormal brain MRI findings and a focal spinal CSF leak have an excellent prognosis.<sup>3</sup> Improvement of abnormalities on MRI can be expected within days to weeks of successful treatment of the CSF leak. Clinical improvement generally precedes that demonstrated on MRI.

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