

## CASE REPORT

# Papilloedema in association with spinal lipoma and bilateral chronic subdural bleeding

F Al-Zain, A Gräwe and U Meier

Department of Neurosurgery, Unfallkrankenhaus Berlin, Berlin, Germany

**Study design:** A case report describing a patient presenting with papilloedema, headache and saddle hypoesthesia caused by a lumbo-sacral intraspinal extradural lipoma in the presence of a bilateral chronic subdural haematoma (cSDH).

**Objective:** The aim of this report is to discuss the pathophysiology of papilloedema in spinal tumours and the effect of the cSDH on the development of papilloedema. A search of the Medline database yielded no case reports describing papilloedema arising from spinal extradural lipoma in the presence of intracranial cSDH.

**Setting:** Department of Neurosurgery, Unfallkrankenhaus Berlin, Berlin, Germany.

**Case report:** We report a clinical case of cauda equina compression due to an extradural lipoma presenting with papilloedema. Cranial computer tomography (CT-scan) additionally revealed a thin, bilateral cSDH. The patient underwent surgical excision of the lipoma subsequent to an L5–S3 laminectomy. On duratomy, a membranous thrombus formation was discovered between the nerve filaments. The patient experienced clinical improvement with regression of his neurological symptoms. Histological findings confirmed the diagnosis of lipoma and intradural thrombus.

**Conclusion:** Spinal tumours may cause complex cerebrospinal fluid (CSF) dynamic and resorptive changes. These changes are mechanical, physiological or combined in their effect. Patients with papilloedema or increased intracranial pressure should be carefully examined by clinical and neuro-radiological means for cranial and spinal pathologies. The treatment of the primary cause might save the patient a series of unnecessary procedures.

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**Keywords:** papilloedema; spinal tumours; chronic subdural haematoma

### Case report

A 27-year-old male patient, with low back pain radiating to the occiput and bilateral retro-bulbar pain with blurred vision and diplopia, was admitted to our neurosurgical department. Except for a saddle hypoesthesia, no pareses or sensory disturbances were identified on clinical examination. Meningeal signs were positive. The patient reported a recent lifting injury and an ice-skating accident a number of weeks ago. His medical history was unremarkable. The ophthalmological examination revealed a diplopia on upwards gaze and fundoscopy showed bilateral papilloedema. Cranial computer tomography showed bilateral thin (8 mm) chronic subdural haematoma (cSDH) (Figure 1). The spinal magnetic resonance imaging revealed a ventral intraspinal extradural lipoma extending from L4 to S3 and occupying almost the entire spinal canal with dorsal displacement of the dural sac (Figure 2). CT-angiography

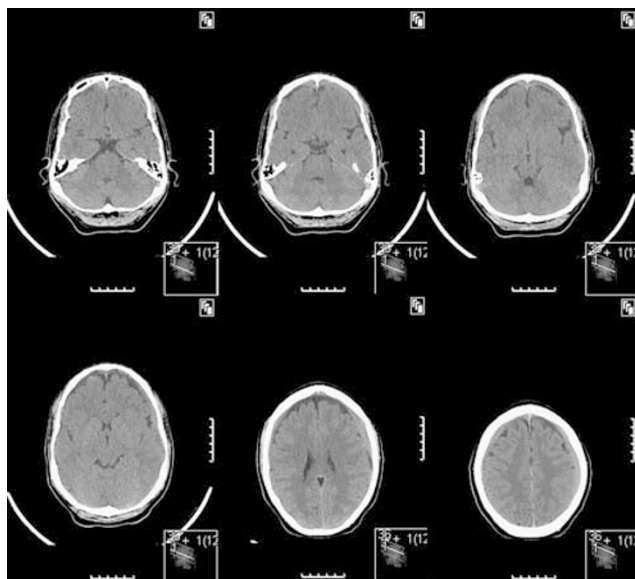
excluded a venous sinus thrombosis (Figure 3). The extradural tumour was excised surgically using a CUSA-device following L5–S3 laminectomy. On opening the dura, we found a membranous-like thrombus with haemosiderin depositions on the cauda filaments. The operative and postoperative course was unremarkable. Histopathological examination confirmed the suspected diagnosis of extradural benign lipoma. The suspected membranous thrombus was also confirmed histopathologically as such. There was no evidence of intradural tumour material. Postoperatively, the radiating pain diminished, the saddle hypoesthesia decreased in intensity and fundoscopy showed a rapid regression of the papilloedema. At follow-up 4 months after operation, the patient remained satisfied with the outcome. He had no neurological deficits, fundoscopy showed complete remission of the papilloedema and cranial CT showed only a thin residual left frontal subdural hygroma.

Correspondence: Dr F Al-Zain, Department of Neurosurgery, Unfallkrankenhaus Berlin, Warener Strasse 7, Berlin D-12683, Germany.  
E-mail: ferass@al-zain.de

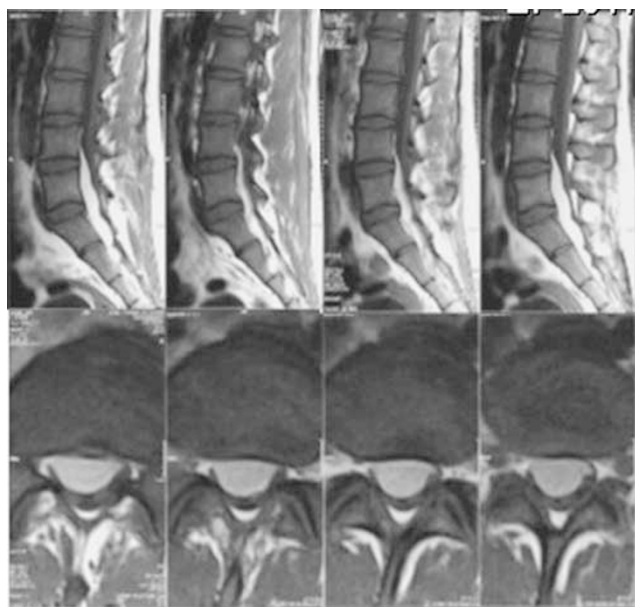
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### Discussion

The major cause of exogenic papilloedema is interference of the axon plasma flow in nerve fibres and consequent



**Figure 1** Cranial computer tomography (axial sections) showing bilateral chronic subdural haematoma with minimal flattening of the surface of the gyri as an indicator for local compression of the subdural space on the cortex.



**Figure 2** Magnetic resonance imaging of the thoraco-lumbar spine (sagittal and axial T1-weighted MR) showing a hypointense, ventral intraspinal extradural space occupying lesion from the 4th lumbar vertebra to the mid-sacrum (S3) occupying almost the entire spinal canal with a severe dorsal displacement of the dural sac.

swelling of the axon, initiating vascular stasis in the optic canal leading to disc oedema. The central retinal vein may be exposed to a higher resistance passing through the subarachnoid space when, for example, the intracranial pressure is pathologically raised. Venous stasis results, aggravating disc swelling.

Two theories attempt to explain the pathogenesis of papilloedema in benign spinal tumours:

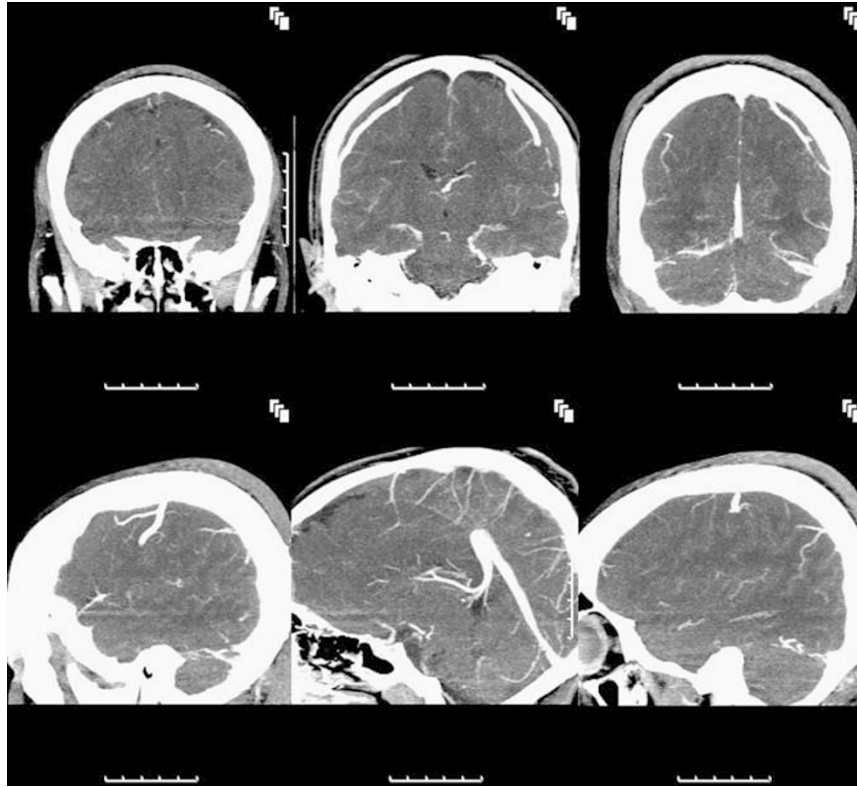
The first hypothesizes that excessive protein secretion as well as mechanical obstruction of the cerebrospinal fluid (CSF) flow by the spinal tumour can increase CSF viscosity. This may influence axoplasmal flow directly, causing disc oedema or indirectly through raised intracranial pressure caused by a reduction in CSF resorption secondary to adhesions in the subarachnoid space.<sup>1,2</sup> Aresni<sup>3</sup> hypothesizes that spinal cord tumour secretions may stimulate a basal aseptic reactionary leptomeningitis, further compromising CSF absorption.

In the case reported here, since lumbar puncture could exacerbate the subdural haematoma, CSF protein levels were not examined. However, raised protein content was suspected not only because of the CSF obstruction but also because of the protein content of blood found intraoperatively around the cauda filaments. This blood might have additionally been initiating aseptic leptomeningeal reaction causing increased levels of CSF protein as well as obstructing CSF absorption in the subarachnoid space. The clinical meningeal signs in our patient were clearly related to the subdural bleeding rather than the slowly growing spinal tumour due to its chronological association with the former.

The second theory concerns CSF dynamics. The lumbosacral spinal CSF compartment is considered the main CSF reservoir, functioning along with the intracranial vascular pool to maintain constant intracranial pressure.<sup>4</sup> Any changes of this balanced system will lead to increased intracranial pressure and papilloedema even without ventriculomegaly in a pseudotumour-like condition.<sup>5</sup> Spinal tumours may in fact reduce the capacity of this reservoir and cause elevated intracranial pressure.<sup>6</sup>

In this case, the mass effect of the cSDH was an additional factor, further compromising the CSF space and dynamic system and may have been the cause of sudden deterioration of symptoms. In other words, the increasing amount of space occupied by the incompressible subdural haematoma resulted in a breakdown of the delicately balanced system, which up to this point compensated for the slow-growing lipoma in the extradural space. This decompensation manifested itself with the clinical signs and symptoms of raised intracranial pressure. Removing the tumour restored the large CSF reservoir with the positive effect of improving the patient's symptoms. Our decision to go for the lipoma and not the cSDH was due to the rather small mass effect of the cSDH on the brain and because of its relatively tiny effect on the CSF compartment in comparison to the spinal lipoma. In this case, the cSDH precipitated decompensation of a system already destabilized by the spinal tumour. It is unlikely that the presence of the cSDH alone would cause this clinical picture.

Our experience with this exceptional case of a spinal tumour superimposed by a cSDH leads us to the conclusion that the subdural haematoma not only acted as a general CSF-compartment mass occupying effect. Instead, it also had the minor local effect of compromising the subdural and



**Figure 3** Cranial computer tomography angiography (coronal sections and sagittal sections in the venous phase) showing the cortical veins, bridging veins and part of the venous sinuses, excluding the presence of sinus venous thrombosis.

subarachnoidal space and decreasing the CSF resorptive surface area, especially in the area of the superior sagittal sinus and the cortical veins where most CSF resorption takes place.<sup>7</sup>

In summary, the cause of the papilloedema in the case presented here is multi-factorial and could not be fully explained by either of the extant theories. The question remains as to whether the subdural haematoma was coincidental due to the trauma detailed in the patient's history or arose as a result of the primary spinal pathology. We noticed on CT that the subdural space in areas not involved in the haematoma is asymptotically enlarged in our young, healthy patient. One of the possibilities is that bilateral hygromas were present before the trauma as a result of spontaneous cerebrospinal fluid leak syndrome. The rupture of tense bridging veins then easily explains the secondary bleeding. One possible indicator is the presence of blood between the cauda filaments, suggesting a tear of the arachnoidae and the possibility of CSF flow in the subdural space.

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