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CASE REPORT

Spinal Subdural Enhancement Mimicking Metastases Following Suboccipital Craniotomy

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ABSTRACT

A 14-month-old boy underwent suboccipital craniotomy for cerebellar tumour. Magnetic resonance screening of the spine a day after the craniotomy revealed striking spinal subdural enhancement in the thoraco-lumbar region. The differential diagnosis included subacute subdural haematoma (in view of the recent operation) versus spinal metastases (in a view of the marked enhancement). The enhancement resolved spontaneously within 1 month of the operation. Thus, striking enhancement can occur in patients with subdural haematoma and should not be misinterpreted as a disastrous advanced metastatic disease.

Key Words: *Craniotomy; Ganglioneuroma; Hematoma, subdural; Magnetic resonance imaging*

中文摘要

枕骨下開顱術後擬似癌轉移的椎管硬膜下造影強化

DD Rasalkar、朱昭穎、蕭容媛、BK Paunipagar、黃海東、朱獻倫

一名14個月大的男孩因小腦腫瘤而接受枕骨下開顱術。術後一天的磁共振成像掃描顯示胸腰段椎管硬膜下有明顯造影強化。鑒別診斷包括亞急性硬膜下血腫（由於病人剛接受完開顱術）及椎管癌轉移（由於有明顯的造影強化）。術後一個月內強化自行緩解。由此可見，硬膜下血腫患者有可能出現明顯造影強化，但不應把它誤作嚴重的癌轉移病。

CASE REPORT

A 14-month-old full-term boy born to a mother with an uneventful antenatal history, presented with sudden onset of generalised tonic convulsion in June 2008. Physical examination showed a bulging anterior fontanelle, sun-set eyes, and dilated superficial scalp veins. Urgent computed tomography of the brain showed a 2.1 x 4.2 cm lesion, consistent with a mixed solid and cystic mass in the posterior cranial fossa that was compressing and displacing the 4th ventricle to the left. There was evidence of obstructive hydrocephalus with retrograde dilatation of both the lateral and third ventricles. Preoperative cranial magnetic resonance (MR) showed a complex solid and cystic mass, epicentred in the cerebellum extending into the 4th ventricle, the cerebellar vermis and both cerebellar hemispheres. The lesion was T1-isointense to grey matter and heterogeneously hyperintense on T2-weighted sequencing (Figures 1a and b). A well-defined cystic component was seen at its superior aspect extending adjacent to the 3rd ventricle. Multiple nodular foci of avid enhancement were evident after gadolinium injection (Figure 1c).

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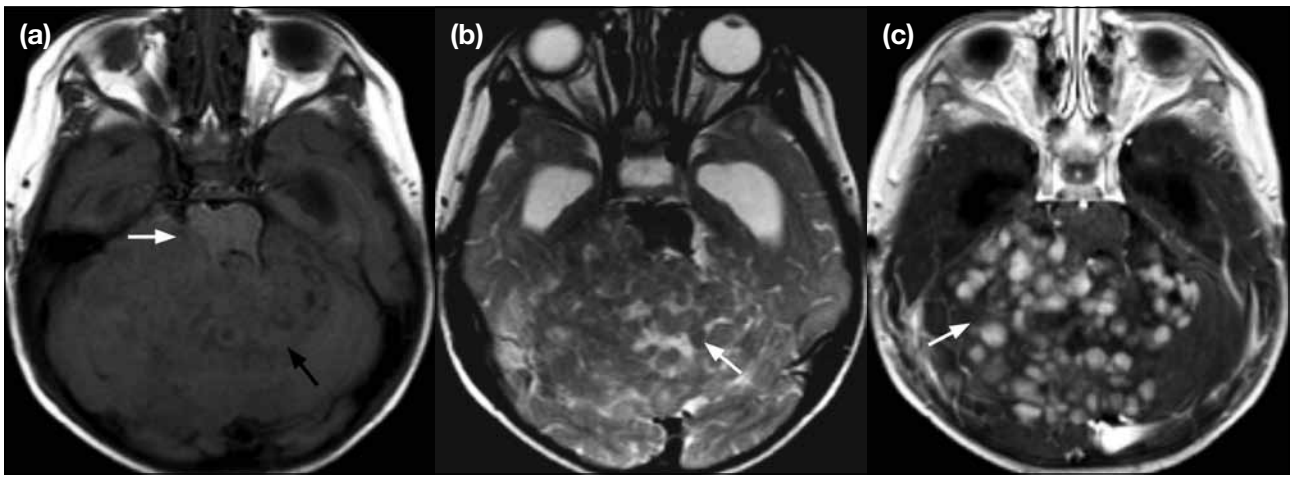

Figure 1. A 14-month-old boy presented with a generalised convulsion and underwent magnetic resonance imaging of the brain. (a) Axial T1-weighted image shows an ill-defined multi-nodular mass, isointense to grey matter (black arrow) causing a mass effect onto the brainstem (white arrow). (b) Axial T2-weighted image shows the nodular appearance of this large heterogeneous intensity lesion (white arrow) in the cerebellum. (c) Contrast-enhanced T1 axial image shows avid nodular enhancement (white arrow) of the lesion.

MR spectroscopy showed elevated choline and reduced N-acetyl aspartate within the lesion, suggestive of neoplastic growth. Emergency suboccipital craniectomy and intraventricular shunt placement were performed on the same day as the MR imaging. In view of reduced power and reflexes in both lower limbs 1 day after the operation, an MR imaging examination of the brain and spine was carried out. This revealed a small amount of blood at the operative bed and the intracranial subdural space, whereas the primary cerebellar tumour was noted to be largely excised. Unexpectedly however, there was a thick rind of T1-isointense and T2-hyperintense tissue along the subdural space in the thoraco-lumbar region, causing a reduction in canal area of more than 70% (Figures 2a and b). This thickened subdural tissue showed intense post-gadolinium enhancement (Figure 2c). The above findings were interpreted as a spinal subdural haematoma (SSH) or extensive spinal metastases. Subsequent histopathology confirmed the cerebellar lesion to be gangliocytoma, which is a benign hamartomatous lesion. No further adjuvant treatment was given. The child regained the lower limb power gradually. A follow-up study of the spine 1 month later showed complete resolution of the abnormal spinal subdural enhancement (Figure 3).

DISCUSSION

In this case, the primary brain tumour was relatively benign, hence rebutting the differential diagnosis of spinal metastasis. There was a strong temporal association between the craniectomy and abnormal spinal subdural enhancement, which subsequently resolved spontaneously, thus supporting the diagnosis of SSH.

Weiner et al[1] described similar features on contrast-enhanced MR spine examinations in 3 patients who had increased signal intensity within the spinal canal 3 days after resection of posterior fossa tumours. The authors ascribed the abnormal diffuse cerebrospinal fluid hyperintensity to the presence of occult subarachnoid blood and / or diffuse leptomeningeal enhancement as a result of meningeal irritation due to subarachnoid blood. In that series, non-contrast images were not available to determine whether the T1 hyperintensity represented contrast enhancement or T1 shortening (presence of blood). In our case, there was no T1 hyperintensity within the spinal thecal sac with non-contrast imaging, but definite enhancement of the subdural tissue in post-contrast images. Meningeal enhancement detected on MR imaging brain is commonly associated with cranial subdural collections, including postoperative subdural collections and especially in children.[2] Neovascularisation, which is seen histologically, may explain for the enhancement of these collections[3,4] that may be provoked by inflammatory components of the fluid containing blood products. The capillaries in this neovascularisation bed have numerous fenestrations, with open endothelial junctions, which are absent in normal capillaries. Exudation from these vessels is one mechanism to account for such chronic subdural collections. Contrast leaking from this neovasculature could accumulate subdurally and very likely explains for the subdural enhancement we encountered. As proven for pineal lesions that also lack a blood-brain barrier,

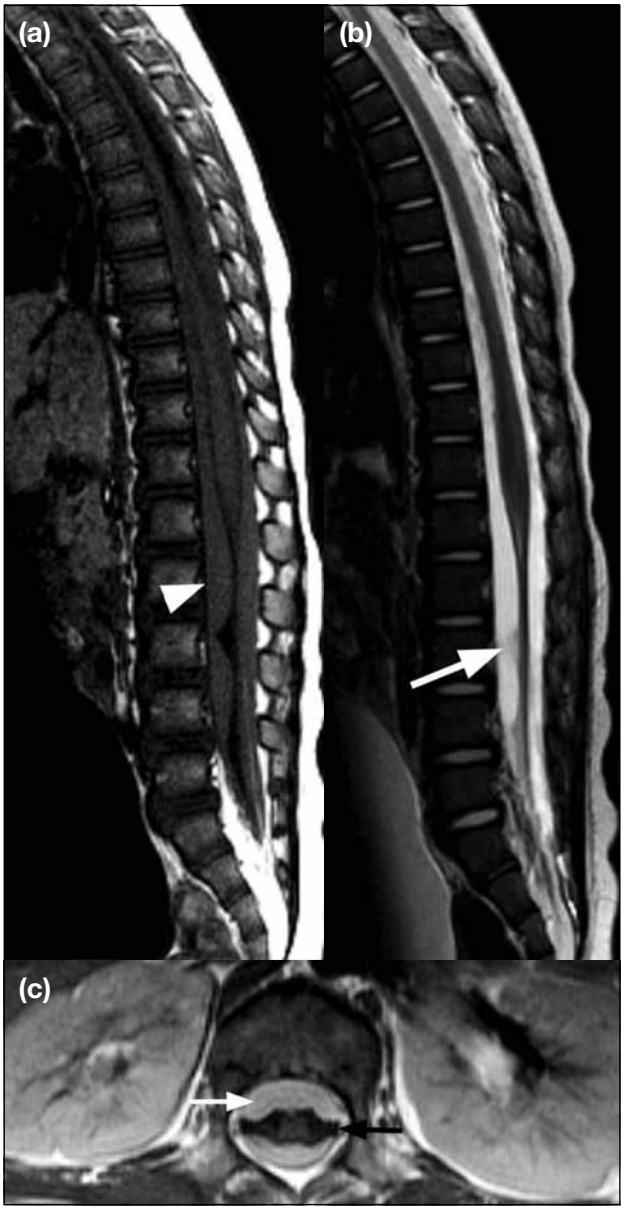

Figure 2. Magnetic resonance imaging of the spine of the same child within 24 hours of the suboccipital craniectomy and intraventricular shunt placement. (a) Sagittal T1-weighted image shows a thick rind of T1-isointense tissue (arrowhead) at the anterior and posterior subdural space extending from T8 down to the lumbar region. (b) Sagittal T2-weighted image shows the subdural tissue is hyperintense with compression of the cauda equina (white arrow). (c) Contrast-enhanced T1 axial image shows more intense enhancement of the subdural lesion (white arrow). Note the mass effect on the spinal cord (black arrow).

delayed accumulation of contrast agent is probably related to passive diffusion of the contrast material.[5]

SSH is a rare condition. In the few cases reported following cranial surgery, there were co-existent intracranial subdural haematomas complicating ventriculoperitoneal shunting. In our case, we postulate that

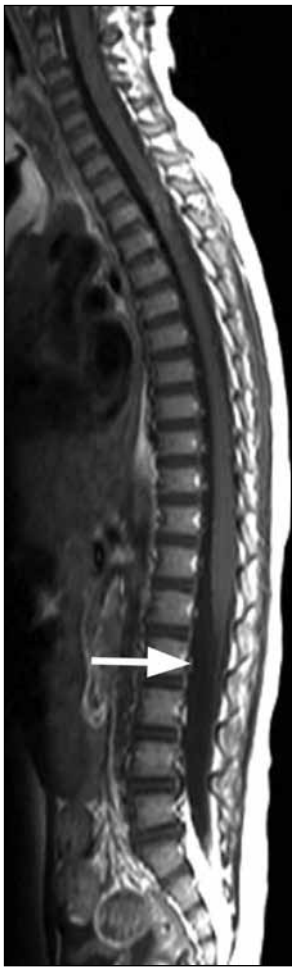

Figure 3. Magnetic resonance imaging of the spine taken 1 month later. A sagittal post-gadolinium T1-weighted image shows complete resolution of the enhanced subdural lesion. The previous compression onto the cauda equine (level indicated by white arrow) has also resolved.

following craniotomy and ventriculoperitoneal shunting, there was acute migration of blood from the cranial subdural space into the most dependent spinal subdural space (at the thoracolumbar level) under the influence of gravity.[6] This presumably gave rise to the T1-isointense and T2-hyperintense signals in the subdural space on the pre-contrast scan. Subsequently, contrast materials exudated into the subdural collection via neovascularisation, resulting in subdural enhancement that became very intense. Thus, enhancement of the postoperative spinal subdural collections can sometimes be striking and should not be confused with metastases. Neovascularisation in the early postoperative period and contrast leaking from fenestrated neovasculature very likely explain the subdural enhancement. Similar mechanism may also explain the postoperative meningeal enhancement and thickening, which has been well reported predominantly over the cerebral convexities.[7]

In conclusion, post-craniotomy meningeal enhancement very likely results from an inflammatory or neovascularisation process caused by bleeding into the

subarachnoid space at the time of surgery, and does not necessarily indicate leptomeningeal tumour spread. Thus, it is crucial to perform MR staging of the spine preoperatively, to avoid possible misdiagnosis of spinal metastasis, and thereby prevent the overstaging of malignant posterior fossa tumours.

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