



## Clinical Study

## Intracranial hypertension secondary to a skull lesion without mass effect

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## ABSTRACT

We report and discuss five patients with intracranial hypertension due to a skull lesion reducing cerebral sinus patency with a compressive, non-thrombotic mechanism. We illustrate the importance of a high level of suspicion for this condition in patients presenting with headache, papilledema and increased intracranial pressure in the absence of focal signs or radiological evidence of mass effect.

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## 1. Introduction

Two major mechanisms of elevated intracranial pressure (ICP) are attributed to brain mass effect or impaired venous drainage [1]. Intracranial hypertension secondary to cerebral dural sinus thrombosis is well-defined in the literature [2]. In contrast, raised ICP subsequent to a compressive, non-thrombotic occlusion of the cerebral sinuses is uncommonly reported and limited data are available on its presentation and clinical course.

## 2. Case reports

## 2.1. Patient 1

A previously healthy, 10-year-old girl presented to the Pediatric Department with intermittent blurred vision and headaches. The physical examination was normal with neither motor nor sensory deficits. The child was admitted for further evaluation following a finding of bilateral papilledema and a normal CT scan. A lumbar puncture (LP) measured cerebrospinal fluid (CSF) opening pressure of 340 mmH<sub>2</sub>O and the diagnosis of idiopathic intracranial hypertension was suggested according to the modified Dandy criteria [3]. Treatment with acetazolamide was initiated, but in the absence of clinical improvement, gadolinium-enhanced MRI was performed on day 4. The latter revealed a small, solitary lesion

adjacent to the superior sagittal sinus (SSS) with scalp involvement, causing SSS obstruction (Fig. 1). The patient underwent neurosurgical resection that resulted in ICP reduction. Pathological examination of the specimen revealed a metastatic neuroblastoma and further work-up revealed a primary tumor in the adrenal gland and an additional tibial lesion. Surgical and systemic treatment according to the routine clinical protocol resulted in a positive post-operative course and complete recovery.

## 2.2. Patient 2

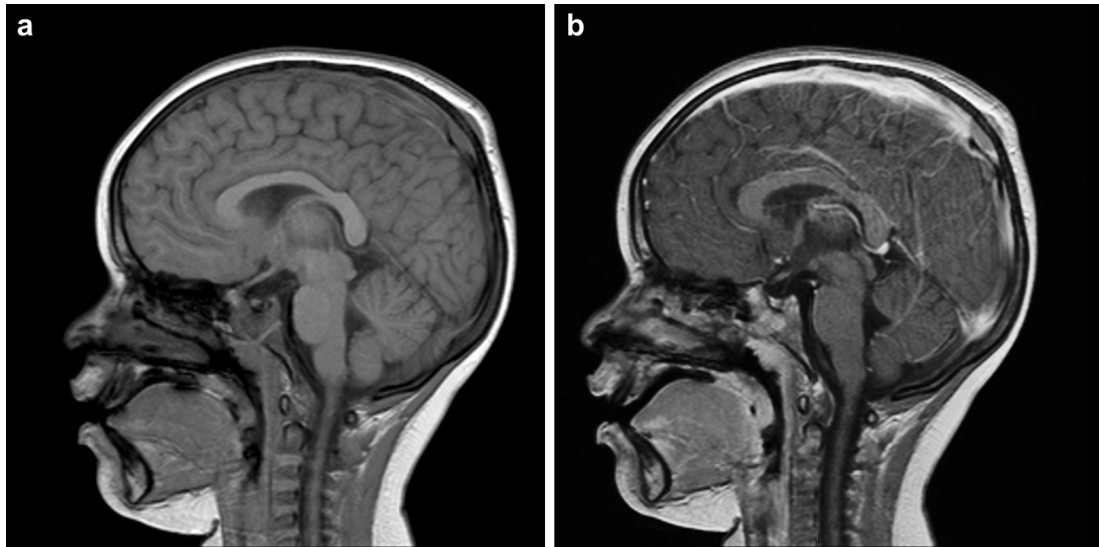
A 55-year-old man with no significant past medical history presented to the Emergency Department with a 1-month history of progressive blurred vision. General physical and neurological examinations were normal except for bilateral papilledema. The initial CT scan showed a homogenous soft tissue lesion infiltrating the left temporal bone, without mass effect (Fig. 2a). Obstruction of the left sigmoid sinus was demonstrated with maximum-intensity projection (MIP) reconstruction of post-contrast computed tomographic venography (CTV) along the left occipital region (Fig. 2b). CSF opening pressure of 370 mmH<sub>2</sub>O was measured and a lesional biopsy was consistent with a neoplastic infiltrate of clonal plasma cells (plasmacytoma). The patient was treated with chemoradiation with a consequent reduction in ICP.

## 2.3. Patient 3

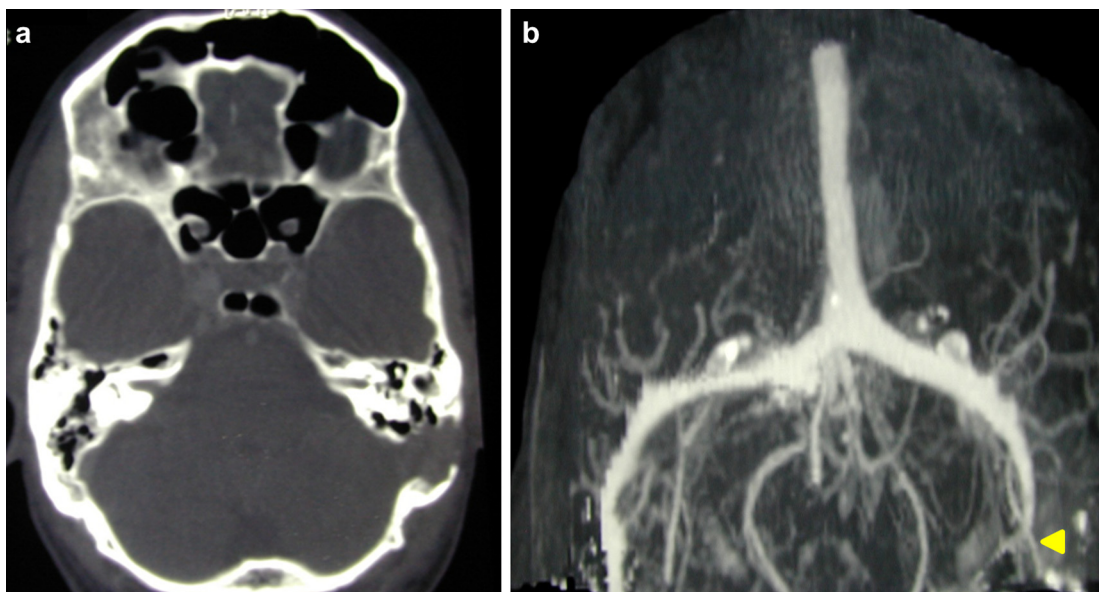
A 62-year-old woman with a history of basal cell carcinoma (BCC) of the scalp developed progressive headaches and blurred

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**Fig. 1.** Patient 1. Sagittal T1-weighted images (a) before and (b) after contrast administration revealed a small skull lesion adjacent to the superior sagittal sinus (SSS) with scalp and soft tissue involvement, causing an obstruction of the SSS.



**Fig. 2.** Patient 2. (a) Axial CT scan in a bone window demonstrated a lytic lesion in the left temporal bone without brain mass effect, and (b) a tumor-related obstruction of the sigmoid sinus, seen in maximum intensity projection reconstruction of post-contrast CT venography (arrowhead).

vision over a period of 2 months. At presentation she was noted to have bilateral papilledema. Brain imaging with three-dimensional volume rendering CT reconstruction demonstrated extensive infiltration of the skull with nearly complete destruction of the bone (Fig. 3a). Post-contrast CTV-MIP reconstruction showed obliteration of the SSS (Fig. 3b). CSF opening pressure at presentation was 360 mmH<sub>2</sub>O. This did not resolve despite a craniotomy and surgical resection of the lesion, which was performed due to incomplete expansion of the SSS. Further clinical details were unavailable as the patient was lost to follow-up.

#### 2.4. Patient 4

A 24-year-old man fell and struck the back of his head. He presented with intact consciousness and complained of a mild headache. The physical and neurological examinations were unremarkable and initial non-contrast CT scan was normal except

for a small depressed occipital fracture without mass effect. The patient was discharged from the Neurosurgery Service after 24 hours of observation. At day 7 after the injury he was readmitted with severe headaches. The physical examination was normal but fundus examination revealed bilateral papilledema. A subsequent CTV reconstruction demonstrated a fracture along the medial aspect of the occipital bone with a small extra-axial hematoma compromising the SSS patency (Fig. 4a), as well as bilateral widening of the optic nerve (Fig. 4b). A craniotomy for evacuation of the hematoma and ICP reduction was performed successfully and the patient was discharged after 48 hours, upon clinical improvement.

#### 2.5. Case 5

A 71-year-old man with a history of metastatic colon cancer and a known cranial lesion overlying the sagittal suture, presented

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