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Papilledema and Diplopia Due to Meningioma Inside the Superior Sagital Sinus: Case Report and Review of the Literature

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Abstract

Small lesions invading or compressing dural sinuses are frequent but secondary intracranial hypertension is not very common, with few examples reported in the literature. This event may be explained in the setting of anatomic variants in pattern of venous sinus circulation. We present the case of a patient who presented with papilledema and loss of visual acuity due to a small meningioma located inside the superior sagittal sinus (SSS). The patient underwent a lumboperitoneal shunt placement with recovery of symptoms. Literature available is also reviewed.

Keywords: Meningioma • Superior sagital sinus • Intracranial hypertension • Papilledema • Lumbo-peritoneal shunt

Introduction

Intracranial hypertension due to small lesions compressing or invading venous sinuses is not a common event, despite the high frequency of meningiomas affecting dural sinuses. They may become symptomatic when there are anatomical variants or significant dominance on one side [1].

Meningiomas located inside venous sinuses are not common; treatment is more challenging in this case and venous sinus opening may lead to significant neurological complications.

We report the case of a patient who presented with papilledema and diplopia due to a small meningioma located in the posterior third of the superior sagittal sinus, treated with a lumbo-peritoneal shunt placement with recovery of symptoms. The role of variants in venous sinus circulation are discussed, as well as treatment modalities [2].

Case Report

A 22-year-old female patient, otherwise healthy, presented with severe headache, visual impairment and vomiting, in addition to diplopia due to paresis of the sixth cranial nerve, all suggestive of intracranial hypertension. A brain CT scan was performed with no apparent intracranial lesions. Ophthalmological examination revealed the presence of papilledema [3].

Given the clinical suspicion of intracranial hypertension, a lumbar puncture was performed with pressure measurement, resulting in 37 mmHg and 30 ml of cerebrospinal fluid were evacuated. After that, the patient presented significant transient clinical improvement.

Magnetic Resonance revealed an small lesion in relation to the posterior third of the superior sagittal sinus, with a size of $12 \times 10 \times 10$ mm, hyperintense in T2 and FLAIR sequences and hypointense in T1. The lesion showed homogeneous enhancement after Gadolinium administration, suggestive of meningioma. Superior sagittal sinus invasion and stenosis by the meningioma

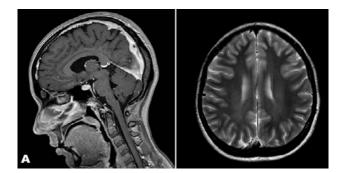
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was observed. On the other hand, no optic nerve sheath or Meckel cavum anomalies were observed, all suggestive of idiopathic or benign intracranial hypertension (Figure 1 a and b) [4].

Given the findings in the MRI, a selective arteriography of both internal carotids and left vertebral arteries was performed (Figure 2). Arterial phase did not show intracranial hypervascularization or other relevant findings. Venous phase confirmed moderate and segmental stenosis of the posterior third of the superior sagittal sinus due to the meningioma, as well as slowed global intracranial cerebral venous circulation time, especially in relation to the



Figures 1A and 1B:Brain MRI in T1 sequence after administration of Gadolinium in sagittal and axial sections, showing a lesion in relation to the posterior third of the superior sagittal sinus, with homogeneous enhancement after Gadolinium administration, suggestive of meningioma.

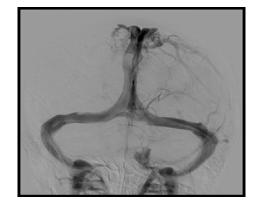


Figure 2. Cerebral angiography of both carotid arteries and left vertebral artery in venous phase and sagittal projection, showing moderate and segmental stenosis of the distal third of the superior sagittal sinus due to the meningioma.



Figures 3A and 3B:Simple abdominal radiography in anteroposterior and lateral projections after placement of lumbo-peritoneal.

cortical veins draining to the superior sagittal sinus [5].

On the other hand, an anatomical variant was observed: the right Labbé vein was hypoplasic, with compensatory hypertrophy of Trolard vein. This finding explained the intensity clinic presented by the patient.

Results

After evacuation of CSF through lumbar punctures, the patient experienced significant clinical improvement. Therefore, she was proposed to undergo surgical intervention by placing a lumbo-peritoneal shunt with programmable valves, Strata Medtronic Iberica SA (E45750), with correct postoperative evolution(Figure 3 a and b) [6]. Intracranial hypertension symptoms remitted. Subsequent ophthalmologic examination showed marked improvement of papilledema and the patient reported subjective visual improvement.

MRI after one month was performed showing stability of the lesion. Available therapeutic options to address meningioma were evaluated, including surgical resection, endovascular techniques through stent placement 4, 6, 8 and radiosurgery 3, 7, the latter being considered the best in this case, given its characteristics (size and risk associated with both resection and stent placement at this location) [7].

Currently, the patient does not present symptoms of intracranial hypertension, she occasionally suffers from paresthesias in lower extremities suggesting seizures and she is under antiepileptic treatment.

The last MRI showed stability of the meningioma with re-expansion of the superior sagittal sinus comparing with the MRI before lumbo-peritoneal shunt placement. At the present time the patient has not received treatment by radiosurgery given the stability and correct evolution she presents [8].

Discussion

Intracranial hypertension secondary to lesions invading or compressing venous sinuses is well known. Different lesions may produce this complication (metastatic tumors, epidermoid cyst, meningioma) and they become symtomatic more frequently if anatomic variants in the pattern of venous circulation are present.

Meningiomas represent the most frequent intracranial tumor. Location

inside dural venous sinuses is not very common. In this situation, the possible occurrence of intracranial hypertension due to small lesions may lead to ophthalmologic symptoms. On the other hand, this location makes tumor resection more challenging with greater associated risks.

The pattern of venous sinus circulation may be very variable and this may explain the exceptional occurrence of intracranial hypertension despite the high frequency of invasion or compression of dural sinuses by different lesions.

In our case, the right Labbé vein was hypoplasic, with compensatory hypertrophy of Trolard vein, explaining the symptoms with the partial stenosis of the superior sagittal sinus.

In our case, given the patient's symptoms we decided to place a lumboperitoneal shunt with programmable valves. However, given the location in the posterior third of the superior sagittal sinus, it was decided not to proceed to the definitive treatment, taking into account the patient clinical stability as well as stability of the meningioma in the imaging controls.

Conclusion

Intracranial hypertension secondary to a meningioma located in the superior sagittal sinus is an uncommon entity. In its management, CSF shunts should be considered for symptomatic management. The definitive treatment of the lesion may be managed by surgical techniques, endovascular techniques or radiosurgery, depending on the location and size. In case of stable and small lesions in an asymptomatic patient, conservative management is a good option.

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