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Enhancing Meningeal Blood Vessels Masquerading as Leptomeningeal Spread of Tumor in Obstructive Hydrocephalus

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Summary: MR showed an enhancing mass in the pineal region and hydrocephalus and leptomeningeal enhancement, thought to indicate pinealoblastoma with leptomeningeal spread. During resection there was no evidence of spread, and repeat MR showed no residual tumor or meningeal enhancement, so the patient was not treated for metastasis. Because there were no signs of leptomeningeal tumor 4 months after surgery, the meningeal enhancement is thought to have been related to venous stasis secondary to obstructive hydrocephalus.

Index terms: Magnetic resonance, contrast enhancement; Meninges, magnetic resonance; Pineal gland, neoplasms

Leptomeningeal enhancement is a nonspecific radiographic finding that may be found in a variety of conditions, including leptomeningeal carcinomatosis (1), infectious meningitis (bacterial, viral, or granulomatous), subarachnoid hemorrhage (2), spontaneous intracranial hypotension (3), and changes after surgery (4), radiation therapy (1), or intrathecal chemotherapy (5). In this report, we describe a patient with pinealoblastoma in whom we believe the leptomeningeal enhancement was caused by obstructive hydrocephalus rather than tumor seeding.

Case Report

This 8-year-old girl presented to the emergency department with a 4-day history of severe headaches and a 1-day history of diplopia. She denied seizures, nausea, vomiting, or weight loss. The only significant finding on physical examination was a right sixth nerve palsy. Magnetic resonance (1.0 T) demonstrated a $2.5 \times 1.5 \times 2$ -cm inhomogeneously enhancing mass in the pineal region. T1-weighted images obtained after the administration of gadopentate dimeglumine (0.1 mmol/kg), revealed hydrocephalus and diffuse curvilinear leptomeningeal enhancement especially over the convexities (Fig 1A and B).

The findings were interpreted as a pinealoblastoma with possible leptomeningeal spread of the tumor.

The patient underwent complete resection of the pinealoblastoma. There was no gross evidence of leptomeningeal spread of the tumor at surgery. Cerebrospinal fluid (CSF) was negative for malignant cells twice. Repeat magnetic resonance 3 weeks after surgery (Fig 1C and D) (performed with the same protocol as before surgery) demonstrated no evidence of residual tumor, a decrease in the hydrocephalus, and complete resolution of the meningeal enhancement. Thereafter, a decision was made to treat the patient for only the primary brain tumor and not leptomeningeal metastasis. The patient was given systemic chemotherapy and prophylactic doses of craniospinal radiation. She is doing well with no signs of recurrence 4 months after surgery.

Discussion

In this patient, it is clear that the enhancement was unrelated to leptomeningeal spread of the tumor. Several factors support a benign cause of the leptomeningeal enhancement, similar to findings in a case report (6) of leptomeningeal enhancement caused by hydrocephalus in a patient with an intraparenchymal brain tumor. Although pinealoblastomas may demonstrate leptomeningeal seeding at the time of initial diagnosis, this patient had two CSF taps that were negative for malignant cells and no evidence of seeding at surgery. According to the literature, leptomeningeal spread of a tumor correlates with positive cytologic results in CSF in up to 80% of cases (7). Moreover, the spontaneous resolution of the meningeal enhancement 3 weeks after surgery implies that malignant involvement was unlikely. The radiographic appearance of serpiginous, curvilinear enhancing structures resembles vessels rather

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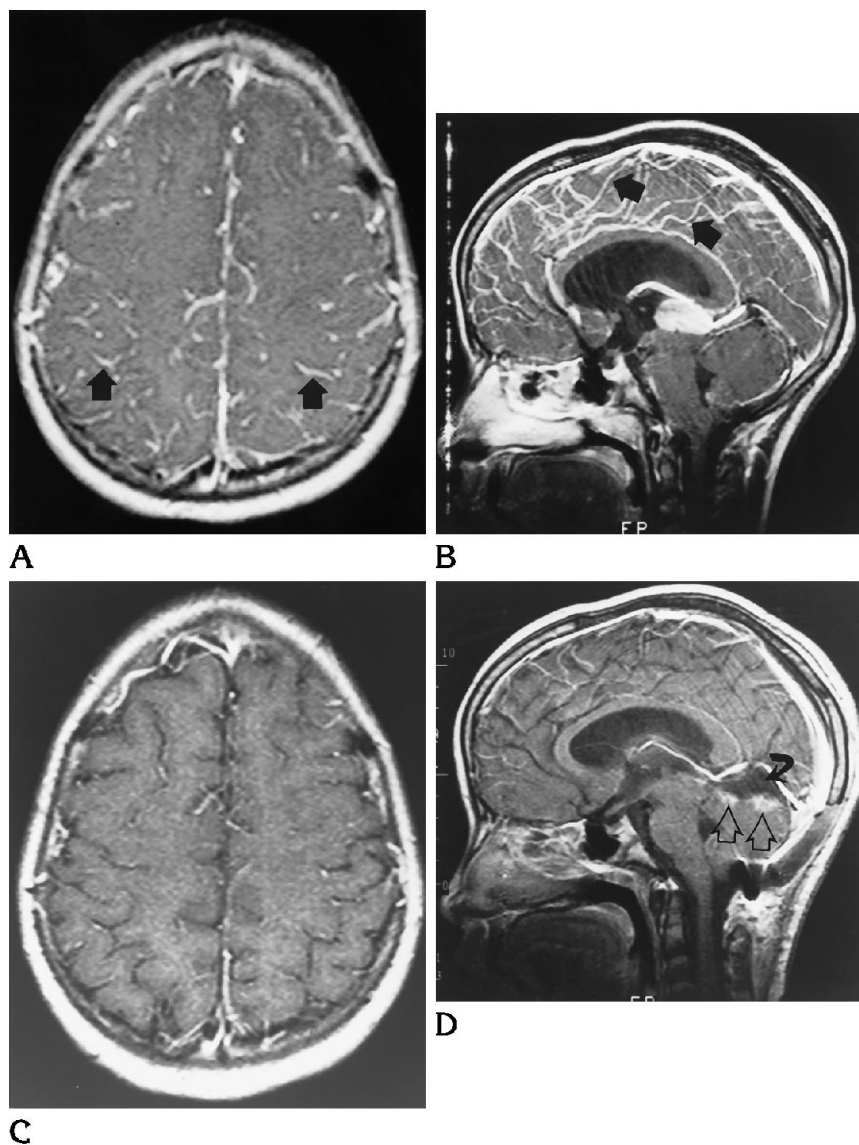


Fig 1. A and B, Preoperative T1-weighted spin-echo (600/30/1 [repetition time/echo time/excitations]) postgadolinium axial (A) and midline sagittal (B) magnetic resonance images. A large enhancing pineal mass is compressing the tectum and aqueduct, resulting in obstructive hydrocephalus. There is marked curvilinear enhancement (arrows) within the leptomeningeal space.

C and D, Three-week postoperative examination with the same imaging sequences at the corresponding levels and section positions. After tumor removal, there is improvement in the ventricular dilatation and resolution of meningeal enhancement. There are postoperative changes, including enhancement along the surgical tract (open arrows) and a small fluid collection on the surface of the cerebellum (curved arrow).

than the more typical nodular and patchy malignant meningeal seeding.

Although the clinical history and CSF analysis did not provide a plausible reason for the meningeal enhancement, additional radiographic evaluation proved helpful. Obstructive hydrocephalus may produce slow flow in pial vessels, and several investigators have explored the physiology of vascular stasis with intracranial hypertension (8–10). Radiographically, the T1 shortening effects of slow flow and gadolinium manifest as enhancement. Noting similar physiologic mechanisms, other authors have reported enhancement in arteries distal to an occlusion during cerebral infarction (11) and arterial ectasia (12) with stagnant flow in focal dilated segments.

The infrequent visibility of prominent pial vessels with hydrocephalus is probably multifactorial. Greitz (13) demonstrated that CSF pressures can be normal in the setting of hydrocephalus. Also, the critical point at which increased pressures cause vascular stasis may vary from patient to patient. Imaging parameters including scan sequences and dose timing of contrast administration can influence the appearance of various structures, including the meninges. Finally, Farn et al (14) reported that some degree of meningeal enhancement can be a normal finding. More subtle abnormal leptomeningeal enhancement may be interpreted as normal.

This case emphasizes the importance of interpreting meningeal enhancement cautiously,

especially in a patient with a known primary intracranial neoplasm and obstructive hydrocephalus. In situations such as the case described here and by Schumacher et al (6), lack of a definitive diagnosis should suggest vascular stasis secondary to hydrocephalus as a plausible cause. Unnecessary therapy such as intrathecal chemotherapy and therapeutic doses of craniospinal radiation may be avoided.

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