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Extensive Cerebrospinal Fluid Enhancement with Gadopentetate Dimeglumine in a Primitive Neuroectodermal Tumor

L. Gill Naul and Michael Finkenstaedt

Summary: We report a case of diffuse enhancement of the cerebrospinal fluid (CSF) after intravenous administration of gadopentetate dimeglumine in an 8-year-old girl with a primitive neuroectodermal tumor. The enhancement of the CSF was caused by leakage of gadolinium chelate complexes into the CSF, which was proved by CSF analysis.

Index terms: Cerebrospinal fluid, magnetic resonance; Magnetic resonance, contrast enhancement; Neuroectodermal tumor

Magnetic resonance (MR) imaging with gadopentetate dimeglumine is useful for the detection of leptomeningeal disease. Several cases mimicking extensive leptomeningeal enhancement on contrast-enhanced MR images thought to be due to hemorrhagic cerebrospinal fluid (CSF) (1) or to enhancement of the CSF itself (2, 3) have been reported. We present a case of diffuse CSF enhancement in a patient with a primitive neuroectodermal tumor, in which analysis of the CSF for gadopentetate dimeglumine was performed for confirmation.

Case Report

An 8-year-old girl had a 3-month history of headaches. Ten days before presentation, she began to experience tonic seizures and abdominal pain, followed by intermittent vomiting. Physical examination revealed a lethargic patient with a decreased level of consciousness. An emergency noncontrast computed tomographic (CT) scan of the brain showed communicating hydrocephalus. Subsequent unenhanced MR imaging revealed abnormal signal intensity in the hypothalamic/sella region consistent with a mass. This mass was hypointense relative to brain and hyperintense relative to CSF on T1-weighted images (Fig 1A). After intravenous administration of 8 mL gadopentetate dimeglumine, vigorous contrast enhancement was seen in the hypothalamic region, which extended into the sella and around the brain stem. In addition, there was extensive and diffuse enhancement of the majority of the

intracranial subarachnoid spaces. The enhancement was evident in the fourth ventricle and basal cisterns, and extended into the cervical spinal canal (Fig 1B). The enhancement of the subarachnoid spaces was less intense than that of the hypothalamic mass. Findings were consistent with an extensive neoplasm centered in the hypothalamic region. The enhancement of the subarachnoid space was initially believed to be due to extensive leptomeningeal neoplasm.

A contrast-enhanced MR image of the spine showed diffuse enhancement of the subarachnoid space throughout the entire spinal canal, which was also initially thought to represent diffuse and extensive neoplastic leptomeningeal seeding (Fig 1C). However, serial CSF samples obtained at the time of ventriculoperitoneal shunt placement and via lumbar puncture revealed no evidence of neoplastic disease. CSF gram, acid-fast bacteria, and fungal stains were negative. CSF cell count revealed one red blood cell and no white blood cells per cubic millimeter, a glucose level of 3.8 mmol/L, and a protein level of 0.61 g/L.

The patient underwent craniotomy for tissue diagnosis, as cytologic studies of the CSF sample were unremarkable. Through a right frontotemporal craniotomy, the tumor was visible along the inferior aspect of the frontal lobe extending into the sylvian fissure. Biopsy specimens obtained from these locations showed an undifferentiated small, round-cell tumor with glial and mesenchymal differentiation, consistent with a primitive neuroectodermal neoplasm.

The possibility that diffuse enhancement of the CSF was caused by the contrast material rather than by extensive leptomeningeal seeding was considered in view of the negative CSF cytology and the atypical pattern of enhancement. CSF samples were obtained before and after intravenous contrast administration during the course of a subsequent MR study. Both samples were imaged with a T1-weighted sequence. The CSF sample obtained after administration of contrast material exhibited higher signal intensity than the CSF sample obtained before contrast administration (Fig 1D). This was confirmed by assaying the CSF samples for the presence of gadolinium chelate complexes. The precontrast CSF sample contained less

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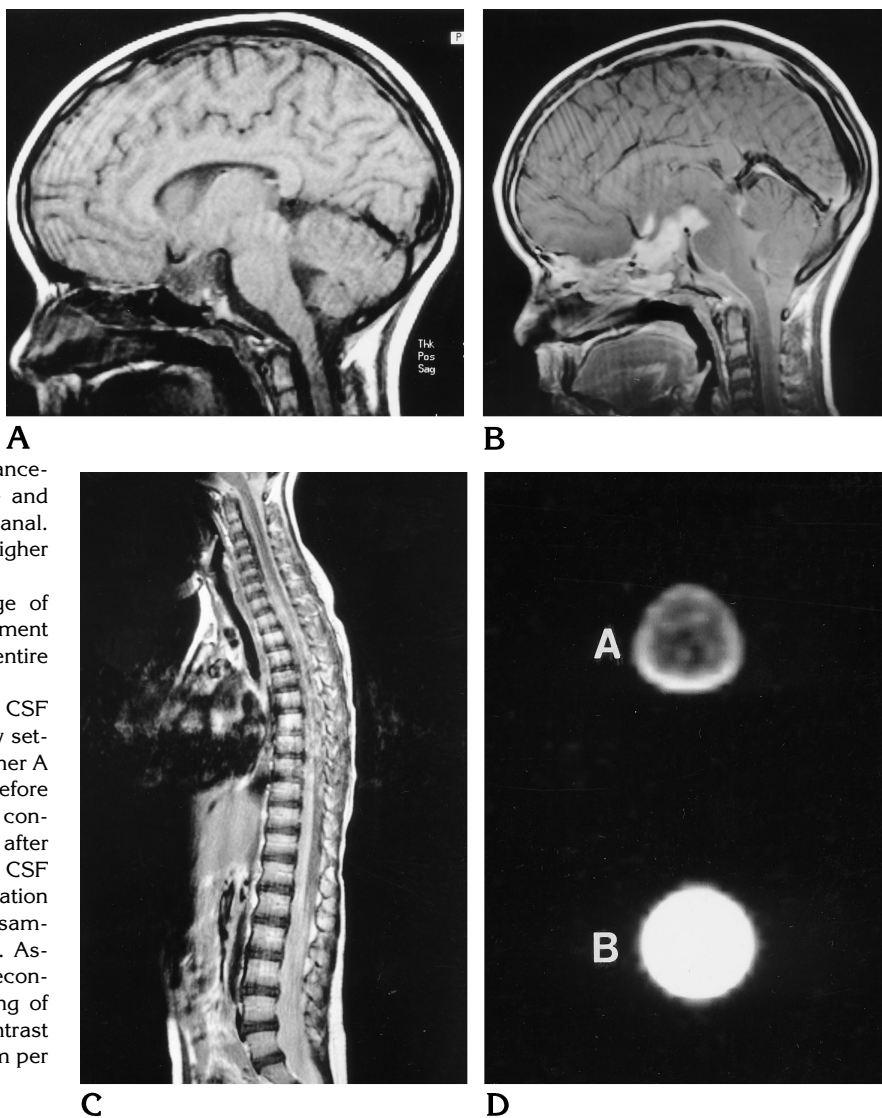
Fig 1. MR findings in an 8-year-old girl with primitive neuroectodermal tumor.

A, Sagittal T1-weighted image (500/15/2 [repetition time/echo time/excitations]) before contrast administration shows added signal intensity in the interpeduncular and suprasellar cistern. The CSF spaces are of normal signal intensity otherwise.

B, Sagittal T1-weighted image (500/15/2) after intravenous administration of gadopentetate dimeglumine shows vigorous contrast enhancement involving the sella turcica, basilar cisterns, meninges around the brain stem, and cerebellum, consistent with leptomeningeal tumor extension. In addition, there is extensive and diffuse enhancement of the CSF. The enhancement is pronounced in the fourth ventricle and basal cisterns, and extends into the cervical canal. The signal intensity of the tumor is slightly higher than the signal intensity of the CSF spaces.

C, Contrast-enhanced T1-weighted image of the spine (600/15/2) shows diffuse enhancement of the subarachnoid space throughout the entire spinal canal.

D, T1-weighted image (520/15/2) of two CSF samples in plastic tubes at a clinical window setting (window level 600; center, 300). Container A holds CSF obtained with a lumbar puncture before intravenous contrast administration, and container B holds CSF obtained immediately after intravenous contrast administration. The CSF sample obtained after contrast administration shows higher signal intensity than the CSF sample obtained before contrast administration. Assaying the CSF samples revealed that the precontrast CSF sample contained less than 10 ng of gadolinium per milliliter, whereas the postcontrast CSF sample contained 24 405 ng gadolinium per milliliter.



than 10 ng gadolinium per milliliter, whereas the postcontrast CSF sample contained 24 405 ng gadolinium per milliliter (results: Mayo Medical Laboratories, Rochester, Minn).

The patient was entered into a chemotherapy protocol for treatment of this tumor with cisplatin and VP-16, as well as cyclophosphamide. Three years 8 months after symptom onset, she remains clinically unchanged, and is currently undergoing further treatment. A large hypothalamic/sella mass persists. Follow-up imaging examinations performed with the same dose of gadopentetate dimeglumine as was used in the original examination showed persistence of the diffuse enhancement of the CSF, although the extent of enhancement had diminished.

Discussion

Primitive neuroectodermal tumors are rare malignant lesions of primitive neuroepithelial

origin. The term is used generically for largely undifferentiated central nervous system tumors with neuroectodermal features in any location, encompassing the entities previously known as medulloblastoma, cerebral neuroblastoma, ependyoblastoma, and pineoblastoma (4). They have a special propensity for subarachnoid seeding (3, 5). For this reason, the entire neuroaxis should be examined with MR imaging after contrast enhancement. Typically, meningeal enhancement in patients with an intracranial primitive neuroectodermal tumor is usually restricted to the basal and perisylvian cisterns, cortical sulci, and tentorium (5).

Our patient experienced a rare consequence of this infiltrative tumor, namely, the presence of contrast material throughout the subarach-

noid space. The hypothalamic/sella neoplasm extended directly to the leptomeninges in multiple locations, as shown by the MR images and demonstrated at surgery. An unusual pronounced enhancement of the CSF in the subarachnoid spaces elsewhere occurred after intravenous administration of contrast agent in this 8-year-old child with histologically proved primitive neuroectodermal tumor. The cause of the CSF enhancement was attributed to a major leakage of contrast agent into the CSF, probably as a consequence of a gross alteration of the blood-tissue barrier over a large area in direct contact with the subarachnoid spaces, which allowed outpouring of gadolinium chelate complexes into the CSF (2, 3).

A tumor that involves the leptomeninges may grow in a linear pattern, creating a thin layer of cells spread diffusely over the brain and meninges. This pattern is often recognized as linear enhancement on T1-weighted images after administration of contrast material. If the tumor is more than a few cells thick, neovascularity occurs. The new vessels are fenestrated capillaries, have no blood-brain barrier, and allow passage of both contrast medium and chemotherapeutic agents into the CSF (6). The neoplasm in this case showed vigorous contrast enhancement, indicating prominent vascularity, which may have contributed to the excessive outpouring of contrast into the CSF.

CSF cytology is diagnostic for leptomeningeal neoplasm in 45% of the cases. Repeated lumbar puncture may increase this yield to 90% (7). The combination of negative CSF cytology and the atypical uniform nature of enhancement, rather than the nodular, masslike, or mar-

ginal enhancement typically seen in metastatic disease, led us to question the presence of diffuse leptomeningeal tumor. The concentration of gadolinium chelate complexes in the CSF was investigated as a result of this uncertainty.

In conclusion, diffuse enhancement of the CSF may prohibit ascertaining whether a diffuse leptomeningeal neoplasm is present. This appearance could mimic diffuse leptomeningeal neoplasm with resultant marked differences in patient treatment. When this phenomenon is suspected, analysis of the CSF for the presence of contrast medium can be of benefit, as in this case.

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