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may have resulted from vascular stasis brought on by shearing injury to the anterior commissure.

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CT Findings in Cystic Intramedullary Oligodendroglioma

Various cases of intramedullary tumors with a cystic component, which are basically astrocytomas and ependymomas, have been described [1-6]. However, no description of an intramedullary cystic oligodendroglioma has been reported. We present such a case, which was studied by metrizamide-enhanced CT.

Case Report

A 20-year-old woman had noticed an overall loss of strength in her right upper extremity and resultant thinning over a 2-year period. During the 6 months before admission, she experienced worsening symptoms and thinning of her right lower extremity. Examination showed an overall loss of strength in the right upper extremity, with muscular atrophy from the deltoid muscle to the interosseous muscles. Atrophy of the quadriceps and gemellus muscles was seen in the right lower extremity. Reflexes were absent in the right upper extremity. No alteration in the vibratory or positional sensitivity was observed, although the abdominal cutaneous response was absent. A CT examination was performed 2 hr after an intrathecal injection of metrizamide. Sections 10 mm thick were made consecutively from the cranial base as far as T2. From C3 downward, enlargement of the cord with obliteration of the subarachnoid space as far as C7 was observed. Contrast material filled a cavity with irregular contours and localized peripherally in the interior of the cord from the inferior portion of C4 as far as C7 (Fig. 1).

Laminectomy was carried out from C7 to C4. The neoplasm seemed to occupy the right half of the intradural space. Overall, the tumor could be readily differentiated from the medulla. The lesion was removed completely by microsurgery. Microscopic sections showed areas of nervous tissue, formed by white matter that appeared to be invaded by tumor cells with clearly delimited contours, clear cytoplasm, and hyperchromatic central nucleus corresponding to oligodendrocytes. Reactive astrocytosis was detected throughout. These results established a diagnosis of oligodendroglioma with areas of astrocytic proliferation.

Discussion

As defined by Barnett and Newcastle [7], syringomyelia is characterized by cavitation of the spinal cord and gliosis; one form is marked by a cavity in the spinal cord that is delimited partly by tumor cells. This definition helps clarify the different designations in the literature that refer to a cystic tumor or to a tumor associated with syringomyelia, which are similar clinical entities.

Syringomyelia can be distinguished from cystic intramedullary tumors [6] by three characteristics: (1) It appears at an earlier age (mean 24 vs 34 years); (2) the symptoms last longer (10 vs 5 years);

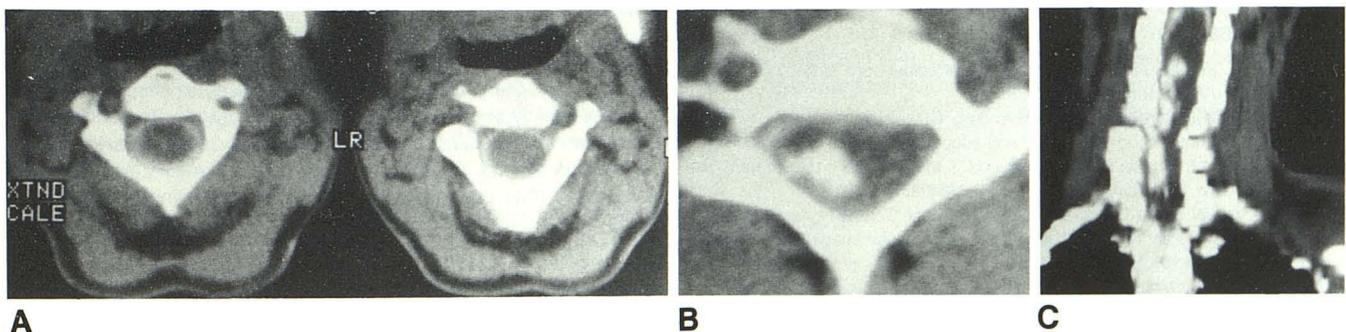


Fig. 1.—Contrast-enhanced CT scans of cystic intramedullary oligodendroglioma.

A, Scan at C3 level shows enlargement of cord with partial obliteration of subarachnoid space. No cyst is seen.

B, Scan at C4 and C5 levels shows metrizamide filling of a cavity with irregular contours and peripheral localization in cord.

C, Reformatted coronal scan at C3-C7 level shows enlargement of spinal cord from C3 to C7 with cavity from C4 to C7.

and (3) paresis, loss of sensation, and low levels of protein in the CSF are more common.

Unenhanced CT examinations can show an intramedullary mass with zones of low density suggestive of a cystic component; if the cord is collapsed, syringomyelia can reliably be assumed to be present. If the cord is not collapsed, which occurs more frequently, it is impossible to differentiate syringomyelia from a cystic intramedullary tumor [3].

Contrast-enhanced CT also can be used to confirm the existence of an intramedullary cyst. After intrathecal injection of metrizamide, opacification of a region of low density confirms the cystic component of the lesion. Sometimes, opacification occurs immediately because the cyst communicates directly with the subarachnoid space. In other cases, opacification occurs after several hours (up to 24 hr) when metrizamide arrives at the cyst by an indirect route (i.e., via the fourth ventricle or by diffusion across the cord).

Whether delayed or undelayed, the occurrence of opacification is not useful in differentiating syringomyelia from cystic tumor or tumor associated with syringomyelia. What does matter is verification of the presence of syringomyelic cavities at each level of cord enlargement because this rules out the existence of a tumor. Evidence of an enlarged cord zone without cavities in its interior strongly suggests formation of a tumor. This was true for our case in which an enlargement of the spinal cord from C3 to C7 was apparent, and the cavity from the inferior part of C4 as far as C7 was filled with contrast material.

When the diagnosis is doubtful despite the criteria mentioned, direct puncture of the cystic cavities and contrast filling (syringogram) may be helpful [8]. An irregular or nodular wall strongly suggests a tumor enclosing the syringomyelic cavity.

Intraoperative echography also can show the cystic nature of these lesions, and the superior and inferior contours are better identified and localized by this method than by CT. Large central cysts with smooth margins are more suggestive of syringomyelia, whereas tumoral cysts are much smaller and marked by eccentric localization and irregular contours [2, 5, 6, 9].

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MR Characteristics of a Primary Melanoma of the Quadrigeminal Plate

Primary CNS melanomas are extremely rare tumors, generally accepted as arising from melanoblasts within the pia-arachnoid [1-3]. Most of these tumors originate from the leptomeninges surrounding the spinal cord and base of the brain. Only four cases of primary melanomas arising in the pineal region have been reported in the literature [1]; all occurred before cross-sectional imaging techniques were available. Although the MR findings of choroidal melanomas [4-6] and of melanoma metastases to the brain [7, 8] have been described, this is the first description of the MR findings of a primary CNS melanoma.

Case Report

A 59-year-old man was admitted complaining of nausea, vomiting, and bilateral temporooccipital headaches of 2 weeks duration. Ophthalmologic examination showed bilateral papilledema.

Initial evaluation included CT examination of the brain, which showed noncommunicating hydrocephalus with obstruction produced by a small, noncalcified, heterogeneously enhancing lesion posterior to the third ventricle (Fig. 1A). The lesion was isodense on noncontrast CT scans (not shown).

MR imaging was performed with a 1.5-T superconducting GE Signa imaging system. Multislice, multiecho, spin-echo sequences provided T1-weighted (TR = 600 msec; TE = 20 msec), T2-weighted (TR = 2500 msec; TE = 80 msec), and balanced (TR = 2500 msec; TE = 20 msec) images in the axial and sagittal planes. A midline lesion, measuring approximately 9 mm in diameter, was shown in the region of the superior colliculus (Figs. 1B and 1C). The small mass caused anterior displacement and narrowing of the proximal aqueduct of Sylvius. The lesion was isointense to adjacent inferior mesencephalic parenchyma, with focal central hypointensity on T1-weighted images. Unlike most primary and secondary parenchymal CNS tumors [9], the lesion remained isointense on balanced and T2-weighted images.

Open biopsy of the lesion was performed, and a black arachnoid membrane was observed overlying a tectal mass. Examination of specimens of the arachnoid matter and tectal mass showed neoplastic cells, mainly in a perivascular distribution, filled with dark melanin and numerous melanosomes. Infiltration of tumor cells into CNS tissue was distinct. A diagnosis of primary CNS melanotic melanoma was made after careful dermatologic and ophthalmologic examinations revealed no evidence of cutaneous or choroidal melanoma.

Discussion

CNS metastases from cutaneous primary melanoma are much more frequent than primary CNS melanoma [3]; therefore, primary CNS melanoma is a diagnosis of exclusion [3].

In humans, melanocytes are found in the skin, mucous membranes, uveal tract, and pia-arachnoid [3]. Precursor melanocytes (melanoblasts), derived from the neural crest, are the cells of origin for both