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MR Imaging of Choroid Plexus Lipomas

Charles L. Truwit,¹ R. Gayle Williams,² Edward A. Armstrong,³ and Arthur E. Marlin⁴

Pericallosal lipomas, also known as lipomas of the corpus callosum, are well described in the radiologic literature. Formerly thought to be rare congenital tumors, they have been identified with more frequency since the advent of CT and MR imaging.

Pericallosal lipomas are the most common type of intracranial lipoma [1–4]. With lesser frequency, subarachnoid lipomas are noted in other locations, including the quadrigeminal, ambient, chiasmatic, interpeduncular, perimesencephalic, and sylvian cisterns [2, 5]. Occasionally, intracranial lipomas have also been noted within the substance of the choroid plexus of the lateral ventricles in association with pericallosal lipomas [2, 6, 7]. To date, however, MR images of choroid plexus lipomas have not been reported. This paper describes the MR findings of a case of pericallosal lipomas with contiguous choroid plexus lipomas and a right sylvian fissure subarachnoid lipoma.

Case Report

An 8-year-old girl presented with her first seizure in April 1988. She subsequently developed petit mal and temporal lobe seizures, which were controlled with Tegretol. The patient's medical history was significant for a hypotonic paraparesis present during infancy, followed by slow motor development. Pertinent laboratory data included a negative alpha-fetoprotein and beta-human chorionic gonadotropin. Physical examination was unremarkable.

An unenhanced CT scan revealed a large interhemispheric mass of fat density situated between the lateral ventricles as well as additional lesions within the lateral ventricles and sylvian fissure (Fig. 1). Moderate colpocephaly was noted, but the roof of the third ventricle did not appear elevated. No normal corpus callosum or septum pellucidum could be identified.

MR images at 1.5 T revealed similar findings. Sagittal spin-echo 600/25/4 (TR/TE/excitations), as well as axial and coronal spin-echo 2500/25-90/4, images were obtained. Each imaging plane clearly identified all the lipomatous lesions (Figs. 2–4). The contiguous nature of the choroid plexus lipomas via the choroidal fissure was seen only on the coronal views (Fig. 3B). Perilesional calcifications were poorly appreciated on MR, as they were obscured by chemical shift artifact.

The sagittal views demonstrated callosal dysgenesis with a diminutive genu and anterior body (Fig. 2A). The axial views demonstrated frontal white-matter tracts crossing the small genu. The longitudinal bundles of Probst and splayed lateral ventricles characteristic of callosal dysgenesis were identified on coronal views.

Colpocephaly was evident. Moreover, the axial and sagittal images showed the choroid plexus lesions to be potentially obstructing within the lateral ventricles (Figs. 2B and 4). The foramina of Monro were unobstructed. No septum pellucidum could be identified, although the fornices and anterior commissure appeared to be spared.

The patient was operated on for the purpose of excluding a teratoma. The surgery was performed with sonographic localization to remove the right sylvian lesion. The postoperative course was unremarkable. Histological examination revealed a mature lipoma, as predicted radiographically.

Discussion

Lipoma of the corpus callosum was first described by Rokitsansky in 1856. Since that time, numerous reports detailing the clinical, radiographic, and pathologic features have been published. Dysgenesis of the corpus callosum is reportedly associated in 35–50% of cases [7]. Occasionally noted are absence of the septum pellucidum and forniceal dysgenesis. Satellite and/or contiguous lesions of the choroid plexus are said to occur in 20–25% of cases [1, 7].

The term lipoma of the corpus callosum probably is a misnomer, as the most plausible theory of its development suggests the lesion represents a process of maldifferentiation of the subarachnoid tissue, not the corpus callosum itself [2, 6, 7]. Embryologically, the potential subarachnoid cisterns are filled with primitive meningeal tissue, the *meninx primitiva*. Normal embryogenesis results in the resorption of the *meninx*, leaving the subarachnoid cisterns. If, however, the *meninx* fails to completely resorb and, in fact, differentiates into the adipose line, the end result would be a mature lipoma. This embryologic theory has been proposed to explain the subarachnoid nature of most intracranial lipomas [2, 6, 7], and is supported by two key differences between intracranial lipomas and dermoids. First, dermoids may contain cartilage, a

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¹ Department of Radiology, Letterman Army Medical Center, Presidio of San Francisco, CA 94129. Address reprint requests to Medical Editing Section HSHH-CI-ME, Department of Clinical Investigation.

² Department of Radiology, Methodist Hospital, San Antonio, TX 78229.

³ Department of Radiology, Driscoll Foundation Children's Hospital, Corpus Christi, TX 78411.

⁴ Division of Pediatric Neurosurgery, Santa Rosa Children's Hospital, San Antonio, TX 78205.

finding not reported with lipomas. Were the lipomas to be considered an inclusion within the closing neural tube, nonlipomatous elements (other than calcification and occasional ossification) could be expected within the lesion. In addition, dermoids result in vessel displacement, not vascular incorporation, as in lipomas. The concept of subarachnoid maldifferentiation is further supported by the fact that lipomas occur "almost exclusively in the cisterns and choroid plexus" [2]. Thus, the lipoma of the corpus callosum would be better called an interhemispheric or pericallosal lipoma, as has been suggested by both Dean and Yock [3, 7].

In addition to the subarachnoid cisterns, the choroid plexus of the lateral ventricles is a known site for lipomas to occur.

The choroidal lipomas are variable in size and location, and some degree of symmetry is frequently noted. These lesions may be isolated from or attached to the pericallosal lipoma. If attached, a variable callosal defect at the site of contiguity is noted. Embryologically, the choroidal fissure develops as an invagination of the interhemispheric cistern. As this takes place, primitive meninx will be carried into the ventricle via the developing choroidal fissure. The developing choroid plexus will be attached to the medial wall of the lateral ventricle at the choroidal fissure. As Yock [7] pointed out, "lipomatous maldifferentiation might then be expected to involve both regions," referring to the callosal and choroidal lipomas.

With the advent of CT and MR imaging, the diagnosis of these lipomas has become uncomplicated. The fat density of the lesion is readily identified by CT, as is the perilesional calcification typically seen with the larger pericallosal lipomas. While the perilesional calcification appears related to the lesion, pathologically it is known to be within the brain tissue, possibly reflecting pressure necrosis. On the other hand, the early literature on this subject includes a few reports of calcification and ossification within the lesion itself. Although occasionally seen in lipomas that occur elsewhere in the body, intralipomatous calcification would appear to be uncommon intracranially and probably represents "osseous metaplasia following calcification" [2]. The aforementioned incorporation of the anterior cerebral vasculature, particularly the pericallosal artery, within the substance of the lipoma, is also occasionally seen on enhanced CT.

Choroid plexus lipomas can also be identified by CT. Again, fat density will be seen, although at times this is difficult to appreciate because of the low density of the surrounding CSF. Calcification in choroid plexus lipomas may be seen as well, owing to the underlying propensity for the choroid plexus to calcify. Subarachnoid lipomas are usually small lesions, and calcification in adjacent brain is possible.

MR imaging offers additional insights into these lesions. MR readily identifies vessels within the lesions by their flow void characteristic. In our case, anterior and middle cerebral branch vessels were seen within the pericallosal and sylvian lesions, respectively. Choroidal vessels, however, are generally too small to identify by MR, and thus would not be consistently demonstrated in the case of choroidal lipomas.



Fig. 1.—Axial unenhanced CT image reveals lesions of fat density within expected locations of corpus callosum and sylvian fissure, as well as intraventricularly. Perilesional calcifications are noted adjacent to pericallosal lesion.

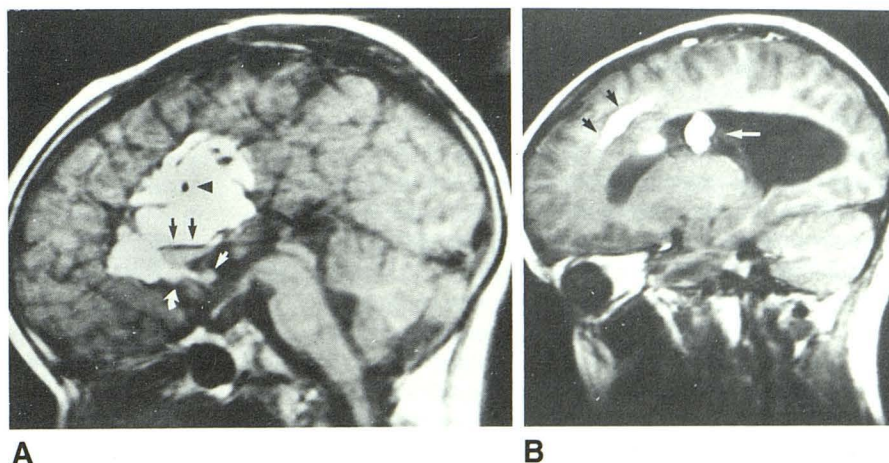


Fig. 2.—A, Sagittal T1-weighted MR (600/25) shows dysgenetic corpus callosum (black arrows) and normal anterior commissure (straight white arrow). Lipoma extends inferiorly into cistern of lamina terminalis (curved white arrow). Flow void within lesion corresponds with anterior cerebral vasculature (black arrowhead).

B, Parasagittal image demonstrates pericallosal lesion within cingulate sulcus on the left (black arrows) and the potentially obstructing choroidal lesion (white arrow).

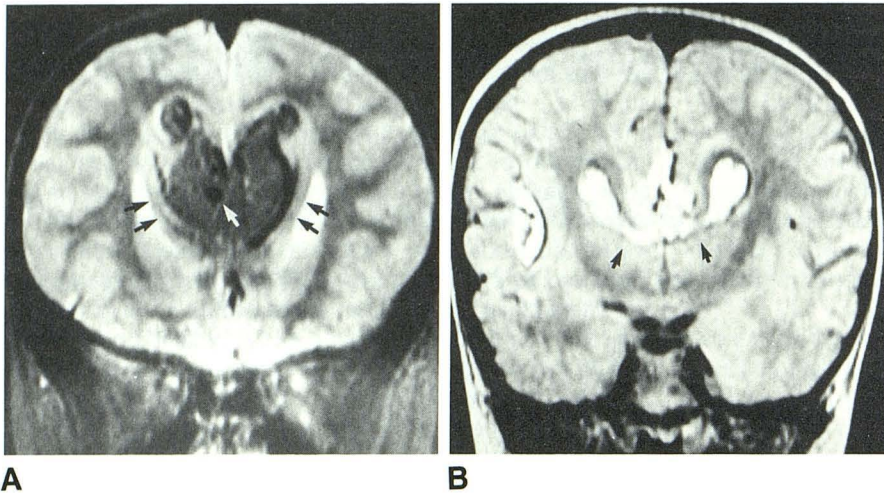


Fig. 3.—A, Coronal T2-weighted MR image (2500/90) clearly demonstrates anterior cerebral vessel within, yet not encased by, pericallosal lesion (white arrow). Lateral ventricles are splayed, typical of callosal dysgenesis. Medial to ventricles are the longitudinal bundles of Probst (black arrows). The chemical shift artifact accounts for the bands of hypointensity along left side of main lesion and cingulate extensions. B, Proton-density image (2500/25) reveals extension of parent lesion via the choroidal fissure (black arrows). Flow void of middle cerebral vessels within right sylvian lesion is noted, as is adjacent chemical shift artifact.

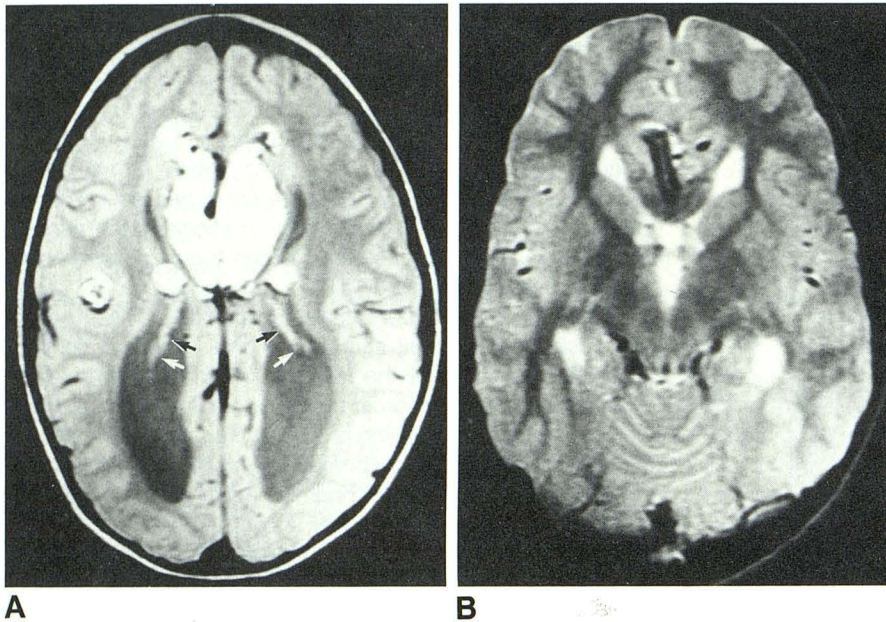


Fig. 4.—A, Axial proton-density image (2500/25) shows normal choroid plexus attached to choroidal lipomas (arrows). Colpocephaly is noted.

B, T2-weighted image (2500/90) reveals patency of foramina of Monro and extension of pericallosal lesion into cistern of lamina terminalis. Hyperintensity in left posterior temporal and posterior parietal regions of both images is artifactual.

The signal characteristics of the lesions themselves are those of fat. The unilateral nature of low signal around the fat, in the frequency-encoding direction, strongly suggests chemical shift artifact rather than signal void caused by calcification. Thus, the actual calcification may not be appreciated.

Lipomas of the choroid plexus are uncommon entities, seen in association with pericallosal lipomas and dysgenesis of the corpus callosum. Although they have been reported to occur in 15–25% of cases of pericallosal lipomas, this figure may increase due to the greater sensitivity of MR imaging. In addition, the contiguity with, or isolation from, the pericallosal lesion is likely to be better appreciated with coronal MR.

To summarize, we report the MR findings of a case of pericallosal lipoma with contiguous bilateral choroid plexus lipomas and an additional lesion within the sylvian fissure. Our case was associated with dysgenesis of the corpus callosum and absence of the septum pellucidum. We conclude that MR

is more useful than CT in demonstrating not only the choroid plexus lesions but also their relation to the parent pericallosal lesion and the presence of associated lesions.

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