



Get Clarity On Generics

Cost-Effective CT & MRI Contrast Agents

**FRESENIUS
KABI**

[WATCH VIDEO](#)

AJNR

MR imaging of pericallosal lipoma.

B Dean, B P Drayer, D C Beresini and C R Bird

AJNR Am J Neuroradiol 1988, 9 (5) 929-931

<http://www.ajnr.org/content/9/5/929>

This information is current as
of August 29, 2025.

MR Imaging of Pericallosal Lipoma

Bruce Dean¹
 Burton P. Drayer
 Don C. Beresini
 C. Roger Bird

Early pathologic reports of corpus callosal lipoma described a consistent relationship between the lipoma and the dorsal surface of the corpus callosum, particularly when the lipoma is not associated with corpus callosal agenesis. MR imaging, especially T1-weighted sagittal acquisitions, exquisitely demonstrated this anatomic relationship in three relatively asymptomatic patients. Therefore, in most cases, a lipoma of the corpus callosum is more accurately described as a pericallosal lipoma. In one individual, common associated findings (partial agenesis of the corpus callosum and choroid plexus lipoma) were also noted. Surgical therapy is usually not indicated because symptoms are generally not related and the anterior cerebral artery is often encased by the lipoma.

Rokitansky [1] first described a corpus callosal lipoma on the posterior part of the corpus callosum in 1856. The early reports described a consistent relationship between the lipoma and the dorsal surface of the corpus callosum [1,2], particularly as an isolated finding without agenesis of the corpus callosum. When a large lipoma distorts the corpus callosum or is found simultaneously with agenesis it is described as a lipoma in the region of the corpus callosum or an interhemispheric lipoma. MR imaging exquisitely and routinely demonstrates the anatomy of the corpus callosum on sagittal, axial, and coronal projections. The sagittal T1-weighted images clearly define the pericallosal or paracallosal location of the lipoma and the absence of direct callosal involvement. The purpose of this study is to illustrate this relationship between the lipoma and the corpus callosum by MR.

Subjects and Methods

The study involved three individuals (two men, ages 36 and 39, respectively, and a 43-year-old woman) evaluated for tension vascular headaches without focal neurologic deficit (cases A and B) and depression (case C). MR studies were performed on a 1.5-T GE Signa scanner (case A), a 0.35-T Dasonics system (case B), and a 0.5-T Technicare system (case C). T1-, intermediate-, and T2-weighted images were routinely obtained in patients in the axial plane after a T1-weighted sagittal sequence. A multislice, multiecho gradient echo (GRASS) pulse sequence (500/10, 40/flip angle 20°) was also acquired on the 1.5-T study. Nonenhanced and IV enhanced CT was performed in two individuals.

Results

A characteristic appearance was noted in all cases (see Fig. 1, case A).

T1-Weighted MR. An approximately 1-cm thick curvilinear strip of prominent signal hyperintensity (intensity similar to orbital and subcutaneous adipose tissue) was noted bordering the dorsal aspect of the genu, body, and splenium of the corpus callosum. This posteriorly tapering band of hyperintensity was seen best on the sagittal images and caused no mass effect. In case B, the hyperintensity (caused by adipose tissue) was not seen in the genu region. The corpus callosum

Received September 28, 1987; accepted after revision February 10, 1988.

¹ All authors: Division of Neuroradiology and the Department of Radiology, Barrow Neurological Institute of St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Address reprint requests to B. P. Drayer.

AJNR 9:929-931, September/October 1988

0195-6108/88/0905-0929

© American Society of Neuroradiology

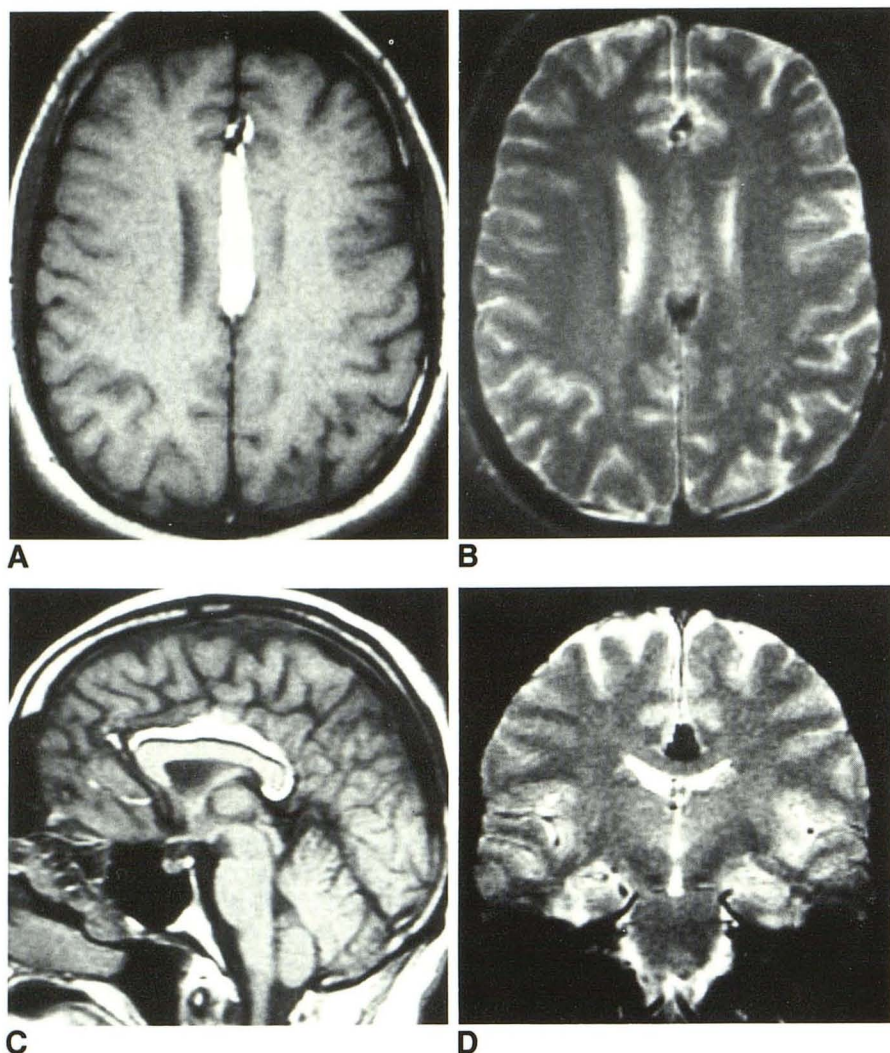


Fig. 1.—Pericallosal lipoma.

A and B, Axial T1-weighted (600/20) and T2-weighted (2500/80) spin-echo images show decreasing signal intensity with increased T2-weighted images compatible with pericallosal adipose tissue.

C, Sagittal T1-weighted (600/20) spin-echo image better illustrates the absence of agenesis and the pericallosal localization of the lipoma that extends from genu to splenium of corpus callosum. Linear decreased intensity in anterior portion of lipoma represents pericallosal artery. Chemical shift artifact is noted along ventral surface of lipoma.

D, Coronal T2-weighted (500/40) 20° flip angle gradient-echo image defines hypointensity of lipoma above corpus callosum.

was clearly delineated in all cases and there was no evidence for infiltration by lipoma. In case C, the lipoma extended from the pericallosal region to the glomus of the right choroid plexus. There was partial agenesis of the splenium of the corpus callosum with lipoma extending into this region.

T2-Weighted MR. The lipoma was of lower signal intensity than on the T1-weighted images, paralleling changes in the orbital and subcutaneous fat. A linear focus of signal hypointensity in the anterior portion of the lipoma coincided with either linear calcification or the pericallosal branch of the anterior cerebral artery. In case A, the calcification was masked on the GRASS images, as both fat and calcium were prominently hypointense. There was no evidence of involvement or compression of the corpus callosum. In case C, as described above, there was partial agenesis of the splenium of the corpus callosum, and this area was replaced by lipoma.

CT. Prominent hypodensity with linear calcification, concurring with findings on MR, was noted in cases A and B. However, a clear distinction of lipoma from corpus callosum could not be made. There was no abnormal enhancement.

Discussion

Intracranial lipomas are unusual, with approximately 30–50% of them located in the region of the corpus callosum [3]. Lipomas occur almost exclusively in leptomeningeal cisterns and choroid plexi and generally spare the adjacent meninges and neural structures. Their biologic behavior resembles a malformation [4]. They are discretely separate from adjacent brain structures except when neighboring structures are incidentally included. Histologically, cerebral lipomas are similar to mature adipose cells elsewhere [5].

A lipoma that is adjacent to the corpus callosum may vary in size from less than a centimeter to a large mass [6]. It may form an ovoid mass, thin streak, or two longitudinal columns with a central groove [7]. Larger lipomas may be bordered by a thick fibrous capsule adherent to and involving surrounding neural structures [8]. The anterior cerebral artery and its branches may be incorporated into the lipoma [9] and there may be areas of calcification in the fibrous capsule or in the adjacent brain substance [10, 11]. There may also be enlarge-

ment of the anterior cerebral branches or of an azygos anterior cerebral artery [8].

When a lipoma occurs as an isolated finding, it is usually pericallosal and closely contoured to the dorsal surface of the corpus callosum [7]. A larger lipoma may displace and alter the shape of the corpus callosum. With agenesis of the corpus callosum, the lipoma may replace or fill in the area normally occupied by the corpus callosum but does not infiltrate it except by incidental contiguous positioning. Although a lipoma most often involves the region adjacent to the dorsal callosal surface, it may extend to the region of the lamina terminalis, fornix, and choroid plexus. Zettner and Netsky [12] describe the tumor as replacing the corpus callosum or lying on its dorsal surface—actually “outside” the brain. A lipoma extending into the choroid plexus merely follows a mesenchymal infolding.

The most commonly associated anomaly is partial or complete agenesis of the corpus callosum, and this occurs in 48% of cases [12]. Other associated anomalies include additional lipomas at other sites, hypoplastic fornix, absent septum pellucidum, spina bifida, myelomeningocele, frontal bone defects, encephaloceles, heterotopic gray matter, agenesis of the vermis, and cleft lip [13–16]. Various theories have been offered to explain the association of corpus callosal agenesis (usually partial) and lipomas. One hypothesis is that the lipoma occurs at a very early stage of physiologic development and that it interferes with the formation of the interhemispheric commissural system, which develops at approximately 3–4 months. Another possible explanation for the frequent and simultaneous occurrence of two lesions may be that of pleiotropy (more than one effect produced by the same gene) or two genes in close association on the same chromosome [12, 17]. The intracranial lipomatous malformation may originate from the primitive meninx [7, 12, 18] or from a proliferation of fat cells normally present in leptomeninx [12].

The reported occurrence of pericallosal or paracallosal lipomas varies from 0.004–0.08% in CT and autopsy series [19, 20]. The most frequent clinical presentation is seizure [21] or as an incidental finding in an evaluation of headache, dizziness, or head trauma. MR, particularly T1-weighted sagittal acquisitions, exquisitely demonstrates the anatomic relationship between the lipoma and the adjacent dorsal surface of the corpus callosum. A lipoma of the corpus callosum is

thus a misnomer since it is most commonly pericallosal in location. This dorsal pericallosal localization is especially typical when the lipoma is not associated with agenesis of the corpus callosum.

REFERENCES

1. Rokitsky C. *Lehrbuch der pathologischen anatomie*. Vienna: Braumüller, 1856;2:468
2. Coats J. A peculiar growth on the upper surface of the corpus callosum. *Br Med J* 1874;2:75
3. Schmid A. Lipoma of the cerebellum. *Acta Neuropathol* 1973;26:75–80
4. Tatsunori S, Shun-ichi N, Natsuko K, Kenji N. Lipoma of the corpus callosum: report of a case and review of the literature. *Child's Brain* 1979;5:476–483
5. Graham D. Lipoma of the corpus callosum. In: Wilkins R, Rengachary S, eds. *Neurosurgery*. New York: McGraw-Hill, 1985:1036–1038
6. Nordin W, Tesluk H, Jones R. Lipoma of the corpus callosum. *Arch Neurol Psychiatry* 1955;74:300–306
7. List C, Holt J, Everett M. Lipoma of the corpus callosum: a clinicopathologic study. *AJR* 1946;55:125–134
8. Gerber S, Plotkin R. Lipoma of the corpus callosum. *J Neurosurg* 1982;57:281–285
9. Probst F. Congenital defects of the corpus callosum. *Acta Radiol [Suppl] (Stockh)* 1973;331:1–152
10. Kinal M, Rasmussen G, Hamby W. Lipoma of the corpus callosum. *J Neuropathol Clin Neurol* 1951;1:168–178
11. Wallace D. Lipoma of the corpus callosum. *J Neurol Neurosurg Psychiatry* 1976;39:1179–1185
12. Zettner A, Netsky M. Lipoma of the corpus callosum. *J Neuropathol Exp Neurol* 1960;19:305–319
13. Curnes J, Laster O, Koubek T, Moody D, Ball M, Witcofski R. MRI of corpus callosal syndromes. *AJNR* 1986;7:617–622
14. Fujii T, Takao T, Ito M, Konishi Y, Okuno T, Suzuki J. Lipoma of the corpus callosum: a case report with a review. *Comput Radiol* 1982;6:301–304
15. Nabawi P, Dobben G, Mafee M, Espinosa G. Diagnosis of lipoma of the corpus callosum by CT in five cases. *Neuroradiology* 1981;21:159–162
16. Tahmouresie A, Kroll G, Shucart W. Lipoma of the corpus callosum. *Surg Neurol* 1979;11:31–34
17. Cooper W, Von Hagen K. Lipoma of the corpus callosum. Report of case. *Bull LA Neurol Soc* 1962;27:39–44
18. Kudoh H, Sakamoto K, Kobayashi N. Lipomas in the corpus callosum and the forehead, associated with a frontal bone defect. *Surg Neurol* 1984;22:503–508
19. Patel A. Lipoma of the corpus callosum. A non-surgical entity. *N Carolina Med J* 1965;26:328–338
20. Vonderahe A, Niemer W. Intracranial lipoma. A report of four cases. *J Neuropathol Exp Neurol* 1944;3:344–354
21. Gastaut H, Regis H, Gastaut J, Yermenos E, Low M. Lipomas of the corpus callosum and epilepsy. *Neurology* 1980;30:132–138