

Intracranial Lipomas; Radiographic and Clinical Characteristics.

A Ramírez- Zamora, J Asconape

Citation

A Ramírez- Zamora, J Asconape. *Intracranial Lipomas; Radiographic and Clinical Characteristics.*. The Internet Journal of Neurology. 2008 Volume 12 Number 1.

Abstract

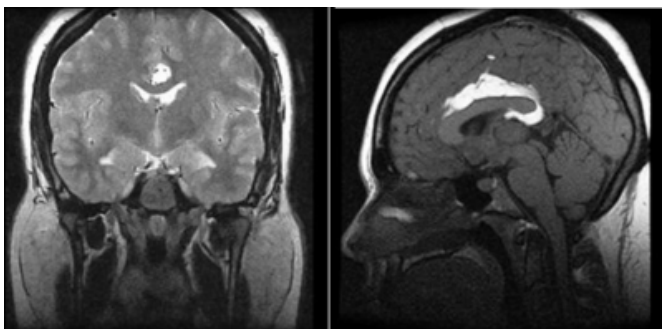
Intracranial lipomas (ICLs) are rare intracranial lesions. They represent a group of congenital malformations of the brain parenchyma, located more frequently in the pericallosal cistern and associated with other parenchymal or brain vascular malformations in up to half of cases.

INTRODUCTION

Intracranial lipomas have a characteristic high signal on T1-weighted images and intermediate/low signal on T2-weighted sequences on Magnetic Resonance Imaging (MRI). Although several reports mention seizures as an associated or presenting feature, this may only represent a higher incidence of intracranial abnormalities compared with the general population. Surgical removal is not recommended because of high complications rate and benign course of these lesions.

Figure 1

Figure 1. Sagittal T1-Weighted and Coronal T2-Weighted MR images. Note the hyperintense lesion in both sequences along the superior aspect of the corpus callosum consistent with an ICL. Note the engulfment of the anterior cerebral arteries within the lipomas.



COMMENTS

Intracranial lipomas (ICLs) are rare lesions that represent congenital malformations resulting from abnormal persistence and mal differentiation of the meninx primitiva during the development of the subarachnoid cisterns¹. ICLs

have a characteristic appearance on unenhanced computed tomography (CT), with low attenuation values ranging between -39 Hounsfield Units (HU) and -80 HU (mean -62 HU). Calcifications are often present in interhemispheric lipomas, most commonly within a fibrous capsule surrounding the lipoma². On MRI, ICL present with a high signal on T1-weighted images and intermediate/low signal on T2-weighted spin-echo sequences³. A relatively small number of uncommon intracranial lesions demonstrate these unusual signal characteristics, including dermoid tumors, epidermoid cysts and paramagnetic substances^{2,3}.

The most common site for ICLs are the midline cerebral structures, involving the pericallosal cistern in over 50% of cases^{4,5}. They represent 5% of all malformations, neoplastic or not in this location. Other locations include the ambient and quadrigeminal cistern (25%), followed by the cerebellopontine angle (9%), superior cerebellar, suprasellar/interpeduncular, and sylvian cisterns (5%) respectively^{5,6}.

Pericallosal lipomas can be subdivided into two subgroups; tubulonodular and curvilinear type. The tubulonodular type is characterized by nodular lesions usually measuring less than two centimeters, and affecting predominantly the anterior corpus callosum². Curvilinear lipomas are usually thin, measuring more than one cm long and located posteriorly. They can be either small or fairly extensive, and they are considered asymptomatic. Hypoplasia of the corpus callosum can be observed in this particular type².

ICLs incidence ranges between 0.08% and 0.2 % in autopsy cases and they represents approximately 0.06 to 0.3 % of all

incidental findings on neuroradiologic studies⁵. They are rarely associated with congenital neurocutaneous disorders, including encephalocraniocutaneous lipomatosis⁷, epidermal nevus syndrome, or congenital infiltrating lipomatosis⁸. Approximately half of the cases are associated with other brain malformations of varying degrees, predominantly dysgenesis of corpus callosum. Associated anomalies are more frequent when their location is anterior to the corpus callosum⁴.

A variety of vascular abnormalities have been described in association with ICLs, including distension, kinking, or narrowing of arteries and veins; engulfment of the cerebral arteries, arteriovenous malformation and aneurysms^{3,4,9}. ICLs have a low proliferation rate and therefore they do not increase in size⁵. Lipoma cells do not multiply and almost never exert a mass effect on adjacent structures². However, there have been reports of hypertrophy of lipomas cells after steroid treatment^{5,10}.

ICL are usually asymptomatic, but they have been associated with seizures (30%) headaches (25%) raised intracranial pressure, dementia and hemiparesis in the past^{5,11}. Although several case reports mention seizures as an associated or presenting symptom, the clinical and electrophysiological characteristics of patients with ICLs remained unclear with only rare EEG correlates¹². Loddenkemper et al¹³ analyzed 3500 consecutive video EEG admissions to a tertiary referral center. Only 5 cases of intracranial lipomas were detected and only in one case, epileptic seizures could be linked to the patient's epilepsy. It appears that the increased incidence of epilepsy in patients with ICLs may be related to higher incidence of associated intracranial abnormalities and malformations compared with general population¹³.

ICLs located near the brainstem may cause ataxia, hydrocephalus, gaze palsies and trochlear nerve paralysis^{5,10}. In the pediatric population, they are associated with non specific neurological complaints including headache, dizziness, seizures or global psychomotor delay affecting language and gross psychomotor skills⁴.

Management of ICLs is usually conservative, as the risks of surgical intervention outweigh the potential benefits in most cases^{5,6}. Surgical removal of these lesions has been

unsuccessful in previous series, leading to significant morbidity and mortality^{4,12,14}. Complete extirpation will almost invariably result in neural or vascular damage due to strong attachment of tumor to surrounding structures. Furthermore, the majority of the tumors are asymptomatic. The only exception and indication for surgical treatment is decompression of near structures (particularly in cases of posterior fossa ICL) requiring placement of ventriculo-peritoneal shunt for treatment of hydrocephalus^{4,14}.

References

1. Truwit CL, Barkovich AJ. Pathogenesis of intracranial lipoma: an MR study in 42 patients. *AJR Am J Roentgenol* 1990;155: 855–865.
2. Harun Yildiz, Bahattin Hakyemez, Mert Koroglu, et al. Intracranial lipomas: importance of localization. *Neuroradiology* (2006) 48: 1–7.
3. Warakaulle, D. R., Anslow, P. Differential Diagnosis of Intracranial Lesions with High Signal on T1 or Low Signal on T2-weighted MRI. *Clinical Radiology* (2003) 58: 922–933.
4. Gómez-Gosálvez, F.A, Menor-Serrano b, F, Téllez de Meneses-Lorenzo et al. Lipomas intracraneales en pediatría: estudio retrospectivo de 20 pacientes. *Rev Neurolo* 2003; 37 (6): 515-521.
5. J. Fandino, J bermudes. Lipoma de la cisterna cuadrigemina y de la cisura calcarina: caso clinico y revision de la literatura. *Neurocirugia* 2005, 16 173-176.
6. Saatci, Isil, Aslan, Cengiz, Renda, Yavuz et al. Parietal Lipoma Associated with Cortical Dysplasia and Abnormal Vasculature: Case Report and Review of the Literature. *AJNR Am J Neuroradiol* (21) October 2000:1718–1721.
7. Moog U, Jones MC, Viskochil DH, et al. Brain anomalies in encephalocraniocutaneous lipomatosis. *Am J Med Genet A*. 2007 Nov 14; 143A (24):2973-80.
8. Canyigit M, Oguz KK. Epidermal nevus syndrome with internal carotid artery occlusion and intracranial and orbital lipomas. *AJNR Am J Neuroradiol*. 2006 Aug; 27(7):1559-61.
9. Tahmouresie A, Kroll G, Shucart W (1979) Lipoma of the corpus callosum. *Surg Neurol* 11:31–34.
10. Haga H,J. Thomanssen, E., Johannesen A., et al. Neural compressive symptoms appearing during steroid treatment in a patient with intracranial lipoma. *Scan J rheumatol* 1999, 28 184-186.
11. Truwit CL, Barkovich AJ. Pathogenesis of intracranial lipoma: an MR study in 42 patients. *AJNR Am J Neuroradiol*, 1989;11:665–674.
12. Gastaut H, Regis H, Gastaut JL, et al. Lipomas of the corpus callosum and epilepsy. *Neurology* (1980) 30:132–138.
13. T. Loddenkemper, H. H. Morris III, B. Diehl. Intracranial lipomas and epilepsy. *J Neurol* (2006) 253 : 590–593.
14. Maiuri F, Cirillo S, Simonetti L, et al. Intracranial lipomas: diagnostic and therapeutic considerations. *J Neurosurg Sci* 1988;32:161–167.

Author Information

A Ramírez- Zamora

Department of Neurology, Loyola University Loyola University Medical Center, Chicago. USA.

J Asconape

Department of Neurology, Loyola University Loyola University Medical Center, Chicago. USA.