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Brief Review

Lipomas of corpus callosum

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ABSTRACT

Intracranial lipomas (iLp) are very rare congenital malformative lesions, being less than 0.1% of intracranial tumors. They originate from abnormal differentiation of mesenchimal tissue of meninx primitiva. Most of cases are asymptomatic pericallosal lesions, often associated with other defects of differentiation of the midline structures. Association with hypogenesis/agenesis of corpus callosum is frequent, being present in 90% of anterior lipomas and in 30% of posterior lipomas. There is no indication to surgical treatment in pure corpus callosum lipoma lesions. Prognosis and symptoms depends on associated malformations. © **Neuroanatomy. 2009; 8: 39–42.**

Key words [lipoma] [intracranial] [corpus callosum]

Introduction

Intracranial lipomas (iLp) are very rare congenital malformative lesions, being less than 0.1% of intracranial tumors [1]. They originate from abnormal differentiation of the mesenchimal tissue of meninx primitiva [2–4]. Intracranial lipomas mainly localize in the pericallosal zone, where they are mostly asymptomatic, being frequently associated to other defects of differentiation of the midline. Symptomatic iLp are very rare, headache is the most common symptom [5].

Intracranial lipomas

Intracranial lipoma was first described in 1818 by Meckel, who found a chiasmatic lipoma; in 1856 Rokitansky first described a pericallosal lipoma associated with corpus callosum agenesis [6].

Incidence in autoptsy series ranges between 0.08% and 0.46% [7,8], which matches with incidental CT diagnosis percentage [9,10].

More than 50% are localized in the pericallosal cistern [11]; other sites are cisterna ambiens and quadrigeminal cistern (20%-25%) [12,13], pontocerebellar cistern (9%) [14,15], cistern of lateral sulcus (5%) [11], and infrequent superficial cerebral hemispheric localizations [16,17].

80% of pontocerebellar lipomas are symptomatic, followed by pericallosal and lipomas of lateral sulcus (50% of cases, respectively); on the other hand, lipomas of cisterna ambiens and quadrigeminal cistern infrequently cause clinical manifestations (20% of cases) [5]. The most common symptoms are: persistent headache, seizures, psychomotor retardation and deficits of cranial nerves [5].

Half of cases are associated with cerebral malformations of various grade, most of all midline anomalies [18]: among these, the most frequent is agenesis/hypogenesis of corpus callosum [1].

In 1990 Truwit and Barkovich summarized the consecution of pathogenetic theories in history supporting the malformative hypothesis [3]. In 1863 Virchow theorized the first pathogenetic hypothesis, that was later reformulated by other authors [19]. He proposed the hypertrophy of a preexistent meningeal fatty tissue, as already described by Chiari.

In 1887 Taubner considered these lesions of this kind as lipomatous gliomas originating from nervous tissue. Pugliese suggested metaplasia of meningeal connective tissue, Bostroem classified iLp among dermoid and epidermoid tumors, and other authors proposed fatty degeneration of the neuroglia [19,20].

However, the hypertrophy proposed by these theories failed to explain the frequent association with congenital encephalic anomalies; moreover, we usually find that the surrounding nervous and vascular structures can be hypoplasic and comprised in lipomas, rather then compressed and displaced by the lesions [3,21].

Theories supporting metaplasic and nervous origins were excluded as well, since iLp are made of univacuolated cells, just like normal fatty tissue.

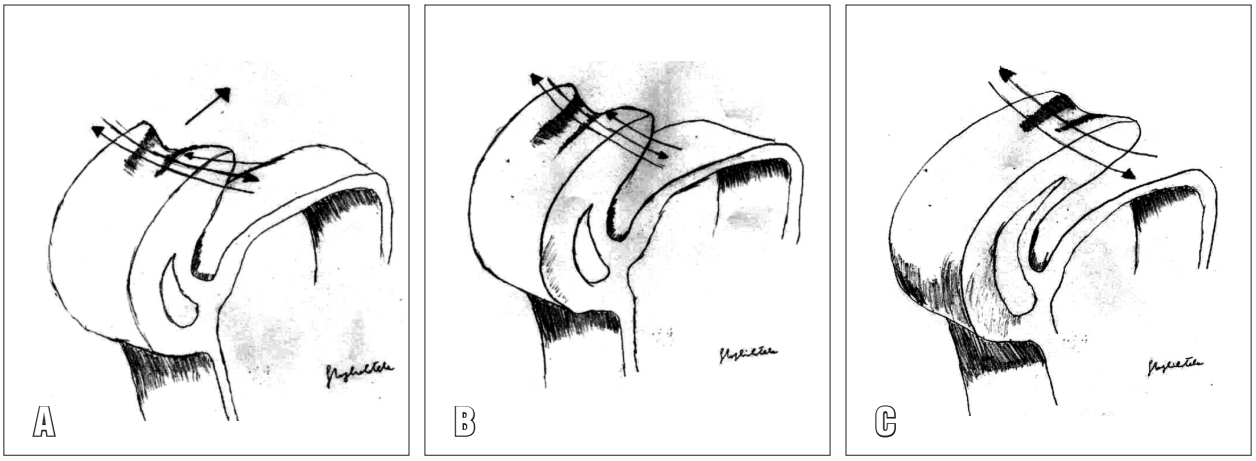

Figure 1. Corpus callosum at an early stage (***A***) anteriorly, commisural plate is thick where transcallosal fibers have already crossed, while posteriorly, ingrowth of fibers is still in early stage, so dorsal callosal groove (sulcus medianus telencephali medii), is deeper. As growth occurs (***B***,***C***), corpus callosum thickens and groove fills, while meninx (not shown) dissolves. *(Modified from reference #3)*

Later, the exclusively disraphic theory was proposed, i.e., the inclusion of mesodermal elements between the borders of the neural tube about to join together [22].

This theory was grounded on the possible presence of subcutaneous lipomas in facial schisis as extensions of a coexisting iLp; in this case, though, we would expect lesions in the inner part of the neural tube, or originating from ventricular cavities; furthermore, disraphic theory cannot explain non-midline lipomas.

As a matter of fact facial bones have mesenchimal origins as well, they cannot generate mesodermal inclusions [3,23], even though facial disraphic anomalies can coexist with interhemispheric lipomas in syndromic contexts [24–26].

The most accredited theory was formulated by Verga in 1929 [4] supporting Salvi's hypothesis of meninx primitiva. He described iLp as malformative disorders of this embryonic tissue, while considering them as neoplastic lesions. Later, Krainer underlined that the spatial relations between iLp and the surrounding vasculo-nervous structures are fairly harmonious, and provided their recurrent cisternal localization, he suggested being a developmental disorder of subarachnoid space [2].

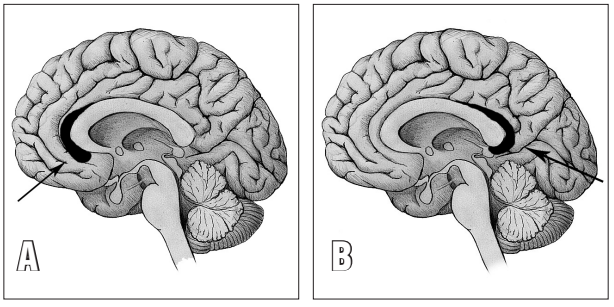

Figure 2. The localization of corpus callosum lipomas. *(**A:** anterior lipoma, tubulonodular shape; **B:** posterior lipoma, curvilinear shape) (Modified from reference #44)*

In 1931, Bailey and Bucy remarked the membranous origin of calcifications and ossifications, which can possibly be associated with iLp [27].

Finally, Ehin and Adson et al., on the basis of Wassermann's work about the fat-forming potential of neural crest, found that meninx primitiva includes a primitive perivascular reticolo-endothelium, which can specialize in fatty cells production [3,28,29].

On these grounds, the assumed mesodermal origin was definitely excluded.

Observing the subarachnoidal cavitation progression, Osaka explained the preferential cisternal localization of iLp [30]. Meninx primitiva cavitation begins from the ventral cisterns of brainstem, and proceeds dorsally and laterally around mesencephalon, followed by cavitation of qudrigeminal cistern. The meninx primitiva located dorsally to lamina terminalis is the last to cavitate, thus enhancing the possibilities of malformation, which is confirmed by evidence that this is the most frequent site of iLp; in addition, since meninx is fused with the borders of lamina reuniens, in case it fails to achieve correct differentiation, it may interfere with the normal development of corpus callosum [20,31] (Figure 1).

Leibrock et al. claim that infratentorial lipomas usually localize at the points of junction between the different segments of the central nervous system, where neural tube bends, hence creating the conditions for the entrapment of the meninx [31].

Truwit and Barkovich affirm that the observations by Osaka et al. and Leibrock et al. also apply to pathogenesis of cortical iLp [3].

Some developmental anomalies can coexist in association with iLp; beyond corpus callosum agenesis/hypogenesis and encephalocele, we can find agenesis/hypogenesis of vermis of cerebellum, spina bifida, and lack of septum pellucidum [20,23]

Because of their malformative origin, iLp usually do not become hyperplasic, whereas it might occur when the patient gains weight, as well as other sites of fat storage [32].

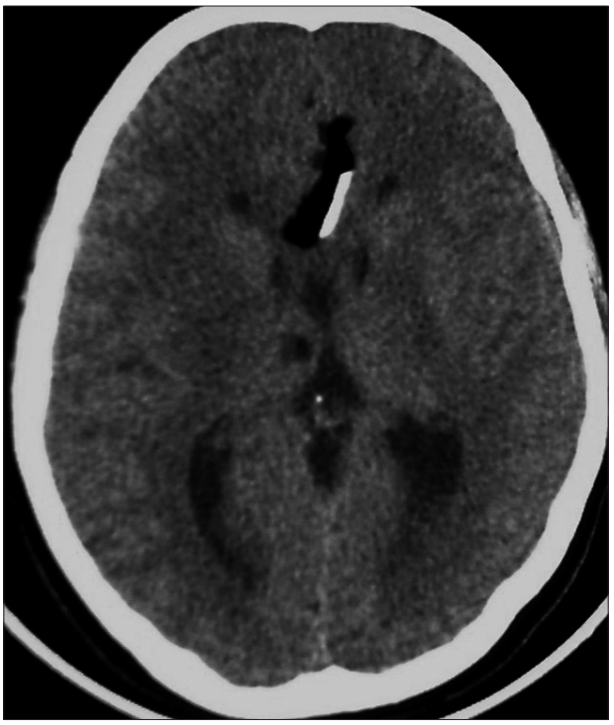

Figure 3. Axial CT scan: interemispheric hypodense area containing calcific component.

Lipomas of corpus callosum

Lipomas of corpus callosum are morphologically classified into two groups: anterior lipomas are indicated as tubulonodular, usually bigger than 2 cm and frequently associated with hypogenesis/agenesis of corpus callosum, frontal lobes anomalies, frontal encephalocele, calcifications, and/or ocular anomalies. Posterior lipomas are indicated as curvilinear, they are thin and usually lay on splenium; they are less frequently associated with corpus callosum anomalies and/ or other encephalic anomalies [33,34] (Figure 2).

Association with hypogenesis/agenesis of corpus callosum is frequent, being present in 90% of anterior lipomas and in 30% of posterior lipomas [35].

A pure (isolated) lesion of corpus callosum probably does not cause symptoms [28]. Clinical manifestations like seizures and mental disorders must be attributed to concomiting nervous tissue anomalies [36]. Epilepsy is one of the most frequent symptom: when present it appears before the age of 15, is often partial and severe [37,38]. Lipomas of corpus callosum can also cause obstructive hydrocephalus.

In hypogenesis/agenesis of corpus callosum, nervous fibers that do not cross the interemispheric fissure, run parallel to it, along the medial walls of both lateral ventricles (Probst's bundles), deforming the III ventricle and the lateral ventricles [39].

It is not yet clearly explained the eventual mechanism that could deform the posterior segments of ventricular cavities. It was hypotized that posterior fibers that don't cross midline, could interfere with correct development of parietal and occipital white matter, being associated with hypogenesis of hippocampus [40].

Diagnosis accidentally occurs during diagnostic procedures in case of an encephalic disorder. Ecographic prenatal diagnosis is possibile at 26 weeks of gestation [41]. On CT scans, lipomas appear as areas of hypodensity, that can contain calcific hyperdensities, so, these aspecific aspects, prospect differential diagnosis with dermoid cysts and teratomas [9] (Figure 3); RM scans do not leave doubts, infact fat signal is characteristically hyper in T1w and T2w sequences, and in FATSAT sequences it is suppressed (Figure 4). Angiography shows displastic cerebral arteries involved in the lesion, but not compressed or displaced.

There's no indication to surgical treatment in isolated (pure) corpus callosum lesions; on the other hand, surgical outcomes are controversial because is difficult a complete debulking that spare the nervovascular structures involved [36,42].

Prognosis and symptoms depends on associated malformations [43]. Risks to be considered, above all in the evolutive age, are hydrocephalus and epilepsy.

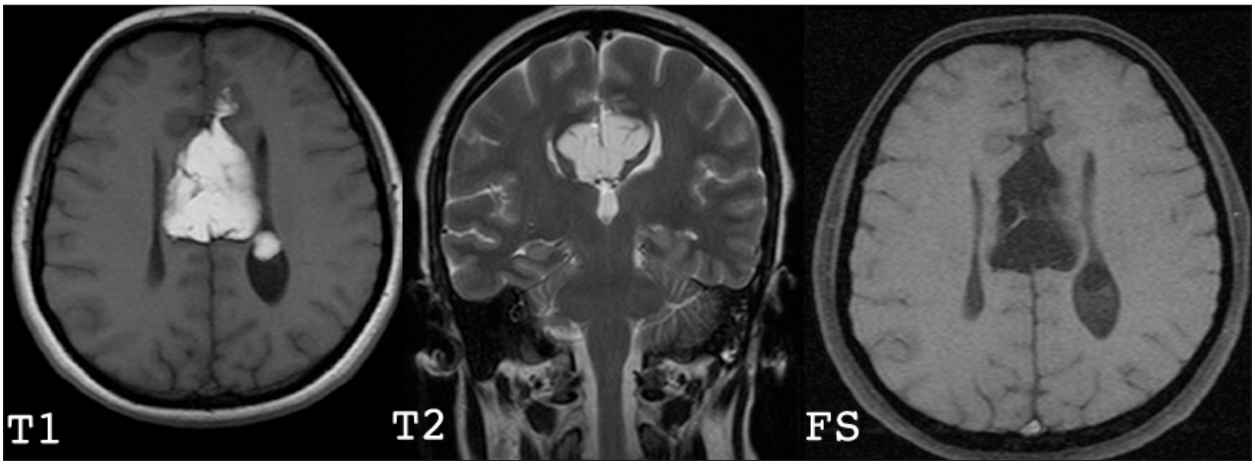

Figure 4. Axial and coronal MR scans: interemispheric area of hyperintensity in T1 and T2, it becomes hypointense in FS (fat saturation) sequences.

References

[1] **Donati F, Vassella F, Kaiser G, Blumberg A.** Intracranial lipomas. **Neuropediatrics. 1992; 23: 32–38.**

[2] **Krainer L.** Die Hirn - und Ruckenmarkslipome. **Virchow Arch [A]. 1935; 295: 107–142.**

[3] **Truwit CL, Barkovich AJ.** Pathogenesis of intracranial lipoma: an MR study in 42 patients. **AJNR. 1990; 11: 665–674.**

[4] **Verga P.** Lipoma ed osteolipomi della pia madre. **Tumori. 1929; 15: 321–357.**

[5] **Maiuri F, Cirillo S, Simonetti L, De Simone MR, Gangemi M.** Intracranial lipomas: diagnostic and therapeutics considerations. **J. Neurosurg. Sci. 1988; 32: 161–167.**

[6] **Rokitansky C.** Lehrbuch der Pathlogischen anatomie. **Vol 2. Vienna, Braumuller. 1856; p. 33.**

[7] **Vonderhae AR, Niemer WT.** Intracranial lipoma: a report of four cases. **J. Neuropathol. Exp. Neurol. 1994; 3: 344–354.**

[8] **Budka H.** Intracranial lipomatous hamartomas (intracranial 'lipomas'): a study of 13 cases includine combinations with medulloblastoma, colloid and epidermoid cysts, angiomatosis and other malformations. **Acta Neuropathol. (Berl). 1974; 28: 205–222.**

[9] **Kazner E, Stochdorph O, Wende S, Gumme T.** Intracranial lipomas: Diagnostic and therapeutic considerations. **J. Neurosurg. 1980; 52: 234–245.**

[10] **Faerber EN, Wolpert SM.** The value of computed tomography in the diagnosis of intracranial lipoma. **J. Comput. Assist. Tomogr. 1978; 2: 297–299.**

[11] **Fandino J, Bermudez J, Aran E.** Lipoma de la cisterna cuadrigemina y cisura calcarina: caso clìnico y revisiòn de la literatura. **Neurocirugia. 2005; 16: 173–176.**

[12] **Halmagyi GM, Evans WA.** Lipoma of the quadrigeminal plate causing progressive hydrocephalus. Case report. **J. Neurosurg. 1978; 49: 453–456.**

[13] **Hori A.** Lipoma of the quadrigeminal region with evidence of congenital origin. **Arch. Pathos. Lab. Med. 1986; 110: 850–851.**

[14] **Roman AM, Olivares G, Katati M, Horcajadas A, Arjona V.** Lipoma del angulo pontocerebeloso: caso clinico. **Neurocirugia. 2002; 1: 38–45.**

[15] **Shah J, Srinivase P, Gale B, Pakar D, Patankar T, Kale H.** Multiple intracranial lipomas, hypogenetic corpus callosum and vestibular schwannoma: an unusual spectrum of RM findings in a patient. **J. Postgrad. Med. 1999; 45: 53–55.**

[16] **Fujii Y, Konishi Y, Kriyama M, Hori C, Sudo M.** Lipoma on surface of centroparietal lobes. **Pediatr. Neurol. 1993; 9: 144–146.**

[17] **Gabrovsky S, Poptodorov G, Aleksiev A, Kruster E, Dimitrov D.** Lipoma of the temporal lobe. **Khirurgiia (Sofia). 1999; 155: 55–59.**

[18] **Macpherson RI, Holgate RC, Gudeman SK.** Midline central nervous system lipomas in children. **J. Can. Assoc. Radiol. 1987; 38: 264–270.**

[19] **Sperling S, Alpers B.** Lipoma and osteolipoma of the brain. **J. Nerv. Ment. Dis. 1938; 83: 13–21.**

[20] **Zettner A, Netsky M.** Lipoma of the corpus callosum. **J. Neuropathol. Exp. Neurol. 1960; 19: 305–319.**

[21] **Saatci I, Aslan C, Renda Y, Besim A.** Parietal lipoma associated with cortical dysplasia and abnormal vasculature: case report and review of the literature. **Am. J. Neuroradiol. 2000; 21: 1718–1721.**

[22] **Nordin W, Tesluk H, Jones R.** Lipoma of the corpus callosum. **Arch. Neurol. Psychiatry. 1955; 74: 300–307.**

[23] **Sadler TW.** Testa e collo. Embriologia medica di Langman. **3rd Ed., Masson Italia Elsevier. 2006; 237–261.**

[24] **Barkovich AJ, Norman D.** Anomalies of the corpus calluosum: correlation with further anomalies of the brain. **AJNR. 1988; 9: 493–501.**

[25] **Osborne A.** Diagnostic Neuro-radiology. **St. Louis, Missouri: Mosby-Year Book Inc. 1994; 826–839.**

[26] **Kodsi SR, Bloom KE, Egbert FE.** Ocular and systemic manifestations of encephalocranio cutaneous lipomatosi. **Am. J. Ophthalmol. 1994; 118: 77–82.**

[27] **Bailey P, Bucy P.** The origin and nature of meningeal tumors. **Am. J. Cancr. 1931; 15: 15–54.**

[28] **List CF, Holt JF, Everett M.** Lipoma of the corpus callosum: a clinicalpathologic study. **Am. J. Roentgenol. Radium Ther. Nucl. Med. 1948; 55: 125–134.**

[29] **Ehni G, Adson A.** Lipoma of the brain. Report of cases. **Arch. Neurol. Psychiatr. (Chic). 1945; 53: 299–304.**

[30] **Osaka K, Handa R, Matsumoto S, Yasuda M.** Development of the cerebrospinal fluid pathway in the normal and abnormal human embryos. **Childs Brain. 1980; 6: 26–28.**

[31] **Leibrock LG, Deans WR, Block S, Shuman RM, Skultety FM.** Cerebellopontine angle lipoma: a review. **Neurosurgery. 1983; 12: 697–699.**

[32] **Barkovich AJ.** Congenital malformation of the brain and skull. Pediatric neuroimaging. **3rd Ed., Philadelphia, Lippincott Williams and Wilkins. 2000; 305–308.**

[33] **Vade A, Horowitz S.** Agenesis of corpus callosum and intraventricular lipomas. **Pediatr. Neurol. 1992; 8: 307–309.**

[34] **Parrish ML, Roessmann U, Levinsohn MW.** Agenesis of the corpus callosum: a study of the frequency of associated malformations. **Ann. Neurol. 1979; 6: 348–354.**

[35] **Tart RP, Quisling RG.** Curvilinear and tubulonodular varieties of lipoma of the corpus callosum: an MR and CT study. **J. Comput. Assist. Tomogr. 1991; 15: 805–810.**

[36] **Patel AN.** Lipoma of the corpus callosum: a nonsurgical entity. **N C Med. J. 1985; 26: 328–335.**

[37] **Gastaut H, Regis H, Gastaut JL.** Lipomas of the corpus callosum and epilepsy. **Neurology. 1980; 30: 132–138.**

[38] **Silva DF, Lima MM, Oliveira CO.** Agenesis and lipoma of the corpus callosum. Case report. **Arq. Neuropsiquiatr. 1995; 53: 667–670.**

[39] **Mulligan G, Meier P.** Lipoma and agenesis of the corpus callosum with associated choroid plexus lipomas: in utero diagnosis. **J. Ultrasound Med. 1989; 8: 583–588.**

[40] **Barkovich AJ, Lyon G, Evrard P.** Formation, maturation and disorders of white matter. **AJNR. 1992; 13: 447–461.**

[41] **Bork MD, Smeltzer JS, Egan JF.** Prenatal diagnosis of intracranial lipoma associated with agenesis of the corpus callosum. **Obstet. Gynecol. 1996; 87: 845–848.**

[42] **Anderson PE.** The radiological diagnosis of lipoma of the corpus callosum. **Radiol. Clin. Biol. 1953; 22: 211–221.**

[43] **Kieslich M, Ehlers S, Bollinger M, Jacobi G.** Midline developmental anomalies with lipomas in the corpus callosum region. **J. Child Neurol. 2000; 15: 85–89.**

[44] **Piovesan EJ, Tatsui CE, Kowacs PA.** Lipoma do corpo calloso associado a ipertrofia do corpo calloso. **Arq. Nuropsiquiatric. 2000; 58: 947–951.**