Pericallosal Lipomas: A Series of 10 Cases with Clinical and Radiological Features

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ABSTRACT

AIM: A pericallosal lipoma is a fat-containing lesion occurring in the interhemispheric fissure closely related to the corpus callosum, which is often abnormal. This is the most common location for an intracranial lipoma. In this study, we aim to report on the clinical and radiographic aspects of ten patients diagnosed with pericallosal lipomas.

MATERIAL and METHODS: A retrospective analysis of patients who presented to the neurology and neurosurgery outpatient clinics of Kayseri Training and Research Hospital between 2010 and 2014 revealed that 10 patients had the diagnosis of pericallosal lipoma. The clinical and magnetic resonance imaging data were obtained by reviewing their files.

RESULTS: Ten patients with an average age of 35.8 years (11-80 years) were included in the study. The mean follow-up was 17 months (8-31 months). No neurological deficits related to the lesions were found during neurological examination in any of the patients. Four patients had tubulonodular lipomas while the other 6 presented with curvilinear lipomas. Four patients (40%) displayed a coexistent corpus callosum hypoplasia. In contrast to previous reports, 3 of these patients had a curvilinear lipoma while the remaining one had tubulonodular lipoma. Also, one of the patients displayed plaque lesions attributable to multiple sclerosis. During the follow-up period, no growth in the lipomas was recorded in any of the patients. No surgical intervention was performed as none of the patients displayed symptoms caused by the lipoma.

CONCLUSION: In this study, we found a stronger association of corpus callosum hypoplasia with posteriorly situated curvilinear lipomas. Our results are in disagreement with previous studies, which suggested corpus callosum anomalies were more often associated with anteriorly situated tubulonodular lipomas. Pericallosal lipomas are benign, self-limiting or slow-growing lesions that generally remain asymptomatic. These lesions occur in the midline and surround critical neurovascular structures. Therefore, surgical intervention should be avoided in asymptomatic cases.

KEYWORDS: Pericallosal lipoma, Corpus callosum hypoplasia

INTRODUCTION

Intracranial lipomas are rare, fat-containing asymptomatic lesions that are generally considered as congenital malformations (16). The most common location is the close vicinity of the corpus callosum (CC), hence the name pericallosal lipomas. Pericallosal lipomas occur in the interhemispheric fissure closely related to the corpus callosum, which is often abnormal. These lesions make up approximately 0.1- 0.5% of all intracranial lesions (9, 11, 16, 17). They generally occur at midline structures like the corpus callosum and the quadrigeminal cistern (15, 16). Two morphological types have been described;
tubulonodular and curvilinear (10, 15, 16). The tubulonodular lipomas have been suggested to be associated with corpus callosum abnormalities more frequently.

In this study, we report on the radiological and clinical findings of 10 patients diagnosed with pericallosal lipomas at our institution between 2010 and 2014.

**MATERIAL and METHODS**

A retrospective analysis of the patients who presented at the neurology and neurosurgery outpatient clinics of Kayseri Training and Research Hospital between 2010 and 2014 was performed. Ten patients who were diagnosed with pericallosal lipoma were included in this study. The clinical and magnetic resonance imaging (MRI) data were obtained by reviewing their files, medical charts, follow-up records and radiological reports (Figure 1A-D; Table I).

**RESULTS**

Table I shows the clinical and MRI features of the patients diagnosed with pericallosal lipoma. A total of 10 patients (7 females, 3 males) were included in this study and they had an average age of 35.8 years (range 11-80 years). Pericallosal lipomas were detected on MRIs, which were obtained to investigate headache in 6 patients, or headache and vertigo in 2 patients, migraine and depression in 1 patient, and multiple sclerosis in 1 patient. No neurological deficits were found in any of these cases including the patient with multiple sclerosis. Review of the cranial MRIs revealed that 4 patients displayed tubulonodular and 6 patients displayed curvilinear lipomas. Furthermore, 4 (40%) patients also displayed concomitant hypoplasia of the CC. Three out of 4 patients with CC hypoplasia were among the patients with curvilinear pericallosal lipomas while the remaining one patient had a tubulonodular type of lipoma (Figure 1A-D). One case who displayed tubulonodular type of lipoma also had plaque lesions attributable to multiple sclerosis. The patients were followed-up for a mean period of 17 months (range 8-31 months). None of the lipoma lesions showed progression or increased in size during the follow-up. None of the patients were symptomatic and therefore, no surgical treatment was offered.

![Figure 1](image-url)
DISCUSSION

Rokitansky first reported intracerebral lipomas in 1856 (1, 6, 16). They are considered to be benign, congenital malformations (7, 11, 16). Intracerebral lipomas compose approximately 0.1-0.5% of all intracranial tumors (9, 11, 16, 17). Autopsy series have reported prevalence rates between 0.08 and 0.2% (10).

The World Health Organization (WHO) classification of brain tumors places intracranial lipomas among grade I mesenchymal, non-meningothelial neoplasms (10). Lipomas are thought to occur due to abnormal persistence or maldevelopment of primitive meninges, when the subarachnoid spaces develop between the 8th-10th weeks of embryonic development (8-10, 16). Similarly, lipomas around the CC are considered to develop from poorly differentiated meningeal remnants (7, 11). CC agenesis, however, results from a defect during the closure of the neural tube (7).

Intracranial lipomas mostly occur on the midline and most commonly involve midline structures like CC and the quadrigeminal cistern (7, 9, 11, 16). However, they have been reported to occur in the sylvian fissure, pontocerebellar angle, preoptic cistern, Galen vein system, interhemispheric fissure, cervicomedullary junction and cerebral convexity (1, 2, 6, 11, 12, 14-16).

Since lipomas are considered to be congenital malformations, they can occur as part of a complex of malformations including agenesis, hypoplasia or hypertrophy of CC, cortical dysplasia, aneurysms and vascular malformations (7, 11, 15, 16). Accompanying anomalies have been reported at variable rates. CC agenesis or disgenesis has been reported to exist in 50% of the cases (6, 15, 16).

A series of quadrigeminal lipomas reported by the authors showed that 16.6% of the cases had accompanying Chiari malformations while no CC anomalies were present (16). However, in this study, we report a 40% (4 patients) rate of CC hypoplasia accompanying pericallosal lipomas. In contrast to previous reports, most of these lipomas (3 patients) had a curvilinear morphology. Seidl et al. reported an 11.8% rate (2 patients) of CC disgenesis in their 17 patients (11). Gomez-Gosalvez et al. reported a series of 20 pediatric cases with intracranial lipomas and found a 30% rate of CC anomalies along with a total 40% rate of associated anomalies (5).

Most intracerebral lipomas are asymptomatic and come into clinical attention during neuroradiological investigations for other conditions (7, 11). This study and our previous report on quadrigeminal lipomas show that intracranial lipomas are most often detected during investigation for headache (16). Seidl et al. also reported that intracranial lipomas in their 17 patients were mostly detected during investigation for headache (11). They also stressed that none of the complaints of their patients was attributable to their intracranial lipomas (11). However, along with concomitant anomalies, intracerebral lipoma

Table I: Documentation of Pericallosal Lipoma Cases (10 Patients)

<table>
<thead>
<tr>
<th>No</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Clinical Presentation</th>
<th>Neurological Examination</th>
<th>MRI Findings</th>
<th>Associated Malformation</th>
<th>Follow-up period (months)</th>
<th>Clinical and Radiological Course</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>54</td>
<td>F</td>
<td>Headache</td>
<td>Normal</td>
<td>Tubulonodular (Corpus)</td>
<td>None</td>
<td>24</td>
<td>Stable</td>
</tr>
<tr>
<td>2</td>
<td>20</td>
<td>F</td>
<td>Migraine, depression</td>
<td>Normal</td>
<td>Curvilinear (Genu-splenium)</td>
<td>None</td>
<td>31</td>
<td>Stable</td>
</tr>
<tr>
<td>3</td>
<td>31</td>
<td>F</td>
<td>Headache</td>
<td>Normal</td>
<td>Tubulonodular (Splenium)</td>
<td>None</td>
<td>25</td>
<td>Stable</td>
</tr>
<tr>
<td>4</td>
<td>44</td>
<td>F</td>
<td>Headache</td>
<td>Normal</td>
<td>Curvilinear (Genu-splenium)</td>
<td>None</td>
<td>13</td>
<td>Stable</td>
</tr>
<tr>
<td>5</td>
<td>11</td>
<td>M</td>
<td>Headache</td>
<td>Normal</td>
<td>Curvilinear (Genu-splenium)</td>
<td>Corpus callosum hypoplasia</td>
<td>14</td>
<td>Stable</td>
</tr>
<tr>
<td>6</td>
<td>80</td>
<td>M</td>
<td>Headache, vertigo</td>
<td>Normal</td>
<td>Tubulonodular (Splenium)</td>
<td>None</td>
<td>12</td>
<td>Stable</td>
</tr>
<tr>
<td>7</td>
<td>14</td>
<td>M</td>
<td>Headache, vertigo</td>
<td>Normal</td>
<td>Curvilinear (Genu-splenium)</td>
<td>Corpus callosum hypoplasia</td>
<td>9</td>
<td>Stable</td>
</tr>
<tr>
<td>8</td>
<td>14</td>
<td>F</td>
<td>Headache, vertigo</td>
<td>Normal</td>
<td>Curvilinear (Genu-splenium)</td>
<td>None</td>
<td>10</td>
<td>Stable</td>
</tr>
<tr>
<td>9</td>
<td>42</td>
<td>F</td>
<td>Multiple Sclerosis</td>
<td>Normal</td>
<td>Tubulonodular (Splenium)</td>
<td>Corpus callosum hypoplasia</td>
<td>8</td>
<td>Stable</td>
</tr>
<tr>
<td>10</td>
<td>48</td>
<td>F</td>
<td>Headache</td>
<td>Normal</td>
<td>Curvilinear (Genu-splenium)</td>
<td>Corpus callosum hypoplasia</td>
<td>24</td>
<td>Stable</td>
</tr>
</tbody>
</table>
patients may present with a history of epilepsy, psychomotor retardation or cerebral palsy (7). CC agenesis has especially been reported to be associated with epilepsy, psychomotor retardation and cerebral palsy (10, 16). Yildiz et al. reported a series of 24 patients with intracerebral lipomas. Three of their patients presented with epilepsy and surprisingly all 3 of them had sylvian fissure lipomas. Therefore, they reported a statistically significant association between sylvian location of lipomas and epileptic seizures (15).

Symptoms, if present, are related to the location of the lipomas. Pericallosal lesions may present with psychomotor retardation or epilepsy. Interpeduncular lesions may cause periorbital pain, ptosis, and conjunctival injection (3, 9). Quadrigeminal cistern lipomas may present with diplopia, vertigo, quadrant-anopsia, headache or progress to show signs of increased intracranial pressure (ICP) due to hydrocephalus (9). Posterior fossa lesions however, may present with cranial nerve deficits or hemifacial spasm.

Two subtypes, namely tubulonodular and curvilinear, have been designated for pericallosal lipomas, which correspond with their morphology and associated anomalies (10). Tubulonodular lipomas are tubular shaped and situated anteriorly. They may be associated with frontal encephalocele, frontal lobe anomalies and CC disgenesis (8, 10, 16). Curvilinear lipomas are slender shaped and situated posteriorly. On the other hand, curvilinear lipomas have been known to generally present with a normal CC and a low incidence of associated anomalies (10, 13). In contrast to this precept, we found CC hypoplasia in 4 out of 10 patients and 3 of these patients had curvilinear lipomas.

Intracranial lipomas are congenital and may be demonstrable in utero. Thanks to the technical advances in obstetric ultrasonography, many intracranial lipoma cases can now be diagnosed prenatally. The diagnosis can be made at 26 weeks of gestation by demonstrating a hyperechoic midline mass with ultrasonography (10). Postnatally however, a head computed tomography (CT) will show a well-demarcated fat density (-60 to -100 HU) lesion, which may be calcified and sometimes accompanied by other anomalies (10, 11, 15, 16). Interhemispheric lesions will generally show calcifications (4, 11). MRI is the most useful modality for a differential diagnosis and identification of associated congenital malformations. The lipomas create a homogeneously hyperintense impression on T1 weighted images. They do not enhance after intravenous gadolinium injection and they homogenously loose their intensity on fat suppression sequences (15).

For midline lesions, dermoid tumors should be included in the differential diagnosis (11, 15, 16). Pericallosal location is not typical for dermoid cysts and they have a heterogeneous impression on CT and T2 weighted MR images (11). For pontocerebellar lipomas, vestibular schwannoma, meningoia, dermoid and epidermoid cysts should be considered in the differential diagnosis (1). A subacute, methemoglobin stage hemorrhage, which generally differs in its presentation, should also be considered in the differential diagnosis, and appears hyperintense on T1-weighted images and hypointense on T2-weighted images (11). Fat suppression sequences will clarify the confusion in such cases.

Most pericallosal lipoma patients are asymptomatic. These lesions are generally found incidentally and they grow very slowly. However, they may surround critical neural and vascular structures, which may increase the surgical risks. Therefore, surgical treatment is mostly not indicated and should be avoided (1, 7, 10, 11, 15, 16). However, surgical treatments for symptomatic patients have been reported. Subtotal resection has been suggested for posterior fossa lipomas, if symptoms of brainstem or cranial nerve compression are present (1).

■ CONCLUSION

Pericallosal lipomas are benign, self-limiting or slow-growing lesions and they may involve critical neurovascular structures. Therefore, surgical removal is not warranted in most cases. Tubulonodular lipomas are generally accepted to be more frequently associated with CC malformations. In contrast with this precept, in this report, we demonstrate a higher rate of associated CC malformations with the curvilinear lipomas.

■ REFERENCES