Low-Grade Astrocytoma Causing Dural and Calvarial Destruction

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Abstract

Most of the literature on intra-axial lesions causing calvarial and dural destruction comes from case reports for glioblastoma, lymphoma, metastasis, and aggressive meningioma. Destruction of dura and calvaria by low-grade gliomas is extremely uncommon; cases reported so far have been mostly oligodendrogliomas. This article describes the unusual case of a 23-year-old male patient with a left-sided intra and extracranial tumor involving the frontal lobe, destroying the overlying dura and calvaria, who underwent maximal safe resection. Histopathology showed the tumor to be a low-grade astrocytoma. The calvarial thinning or remodeling caused by low-grade gliomas is thought to result from their chronic mass effect, by displacing the overlying layer of cerebrospinal fluid and transmitting brain pulsations directly to the inner table of the skull. Pressure thinning of the inner table of the skull may be caused by Pacchionian granulations close to the midline. Although this is extremely uncommon, magnetic resonance imaging may include low-grade astrocytoma in the differential diagnosis in such cases.

Keywords
► low-grade astrocytoma
► dural destruction
► calvarial destruction
► magnetic resonance imaging
► surgical excision

Introduction

Most of the literature on intra-axial lesions causing calvarial and dural destruction comes from case reports for glioblastoma, lymphoma, metastasis, and aggressive meningioma,1 extremely uncommon with low-grade gliomas2 cases reported so far have been mostly oligodendrogliomas.3 To our knowledge, only one case of calvarial and dural erosion with low-grade astrocytoma has been well documented. This article describes the unusual case of a 23-year-old male patient with a left-sided intra- and extracranial frontal low-grade astrocytoma spontaneously destroying the overlying dura and calvaria, who underwent maximal safe resection.

Case Presentation

A 23-year-old man, previously healthy, presented to us in the emergency room with generalized convulsions. This was his second episode; there was a history of headaches and occasional vomiting 1 month before admission. On examination, he was fully conscious, orientated, and without neurological deficits. A soft tissue through bony...
round defect (2 cm in diameter) was palpable in the left frontal area.

A computed tomographic (CT) brain scan revealed a mass of low density with isodensity component in the left frontal region as well as an area of destruction of the ipsilateral frontal bone; a bone window revealed an overlying destruction of the calvaria ( Fig. 1A–C). Brain magnetic resonance imaging (MRI) showed a 3.5-cm intra-axial lesion in the left frontal area that was hypointense on T1 and hyperintense on T2 with enhancement of superficial component on gadolinium causing moth-eaten, permeative destruction of calvaria. It also showed inward displacement of the hypointense dura, suggesting extra-axial extension ( Fig. 1D–F).

Left hemicoronal skin incision over the tumor was made. After the scalp was opened, the erosions of the calvaria were immediately visible. The tumor mass was seen and had not infiltrated the soft tissue of the overlying scalp. Craniectomy was performed by using wide margins around the destroyed calvaria. After the bone flap was removed, a tumor mass was found in the epidural space; the dura under this mass has a ring-shaped perforation. Both the epidural and subdural part of the tumor mass was easily separate off the dura mater. The dura was then open in a star-shape manner and attention was focused on the intraparenchymal part of this lesion. The bulk of the tumor was gray and soft without clearly identifiable margins between tumor and surrounding normal brain tissue in the depth. Near-total resection was achieved, the dura was repaired with dural substitute, the bone flap was then inspected, the rest tumor resected, and acrylique cement was used to repair the defect ( Fig. 2A–C).

Postoperatively, the patient’s clinical symptoms were improved. The histopathologic specimens showed a fibrillar background of a glial tumor proliferation of low cell density composed of cells are with nucleus showing mild cytonuclear atypia without mitosis, necrosis, or endovascular proliferation, tumor cells are positive for glial fibrillary acidic protein, negative for isocitrate dehydrogenase 1 and low proliferative index (Ki67) ( Fig. 3A–D). No pathological difference existed.

![Fig. 1](A–C) Computed tomography scan showing hypodense lesion with isodensity component in the left frontal region with overlying calvarial destruction, slightly enhanced after contrast. (D–F) Brain magnetic resonance imaging showing a 3.5-cm intra-axial lesion in the left frontal area that was hypointense on T1, with enhancement of superficial component on gadolinium causing moth-eaten, permeative destruction of calvaria. It also showed inward displacement of the hypointense dura (arrow), suggesting extra-axial extension of the lesion, well seen in fluid-attenuated inversion recovery sequence.
between intra and outer dura part; the diagnosis of low-grade astrocytoma without bone or dural infiltration was confirmed. A 6 months’ follow-up brain MRI post-surgery showed post-operative changes without evidence of recurrence. The patient was kept on anticonvulsants for 12 months and has also had no seizures since discontinuation.

**Discussion**

Intracranial masses that may produce calvarial erosion include meningioma, lymphoma, intracranial cysts, dural metastases, chronic juvenile subdural hematomas, and anomalous development of the cisterna magna.³ Destruction of dura and calvaria by low-grade gliomas is extremely uncommon² cases reported so far have been mostly oligodendrogliomas.³ These superficially located gliomas usually present with seizures. Brain CT will show a hypodense lesion located superficially and without enhancement on contrast. It will have irregular margins. Calcification is absent in astrocytomas, and bone windows will show the erosion above. The erosion is presumed to be a pressure erosion caused by the peripheral location and slow-growing nature of this lesion.⁴ Brain MRI will show a hypodense lesion on T₁ and hyperdense lesion on T₂; the lesion does not show any enhancement on contrast injection. Our case is unusual, which showed an enhancement area in and under the bone destruction.

To the best of our knowledge, four case reports about low-grade astrocytoma with bone erosion have been made, of which three are representative and well documented.²,⁵–⁷ The clinical characteristics (demographics, clinical presentation, calvarial erosion and tumor localization, surgical findings and outcomes) of five cases including our case were gathered on a summarized table (►Table 1). Handzhiev et al described a case of parietal low-grade glioma with destruction of the dura and calvaria in the absence of prior surgery or radiation; in his case the tumor mass was found beneath the intact external table in the diploe and the dura under this mass had a ring-shaped perforation.⁵ Khan and Hashmi reported a case of a low-grade astrocytoma causing calvarial scalloping in the right anterior parietal region with intact dura.² In the context of the reviewed cases, there are some peculiarities in our patient; our case is analogous to the case of Handzhiev et al, however, with involved both tables of the skull that not have been described before in low-grade astrocytoma.

The calvarial thinning or remodeling caused by low-grade gliomas is thought to result from their chronic mass effect, by displacing the overlying layer of cerebrospinal fluid and transmitting brain pulsations directly to the inner table of the skull. Pressure thinning of the inner table of the skull may be caused by Pacchionian granulations close to the midline.⁴

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**Fig. 2** Tumor eroding through the bone before the bone flap was created (arrow) (A). The bone flap with the rest tumor involved both tables and the diploe of the skull (B). Epidural tumor leaving the dura that has a ring-shaped perforation; dotted circle indicates the tumor (C).

**Fig. 3** Microscopic image showing on a fibrillar background a glial tumor proliferation of low cell density composed of cells with nucleus demonstrating mild cytonuclear atypia without mitosis, necrosis, or endovascular proliferation (A: ×10) hematoxylin and eosin staining. Photomicrograph showing tumor cells positive for glial fibrillary acidic protein (B: ×100), negative for isocitrate dehydrogenase1(C: ×100) and low proliferative index (D: Ki67 × 100).
<table>
<thead>
<tr>
<th>First author, year of publication</th>
<th>Journal</th>
<th>Age year/sex</th>
<th>Clinical presentation</th>
<th>Location and type of overlying erosion bone (CT)</th>
<th>Radiological findings of the mass (CT/ MRI)</th>
<th>Surgical finding</th>
<th>Histology</th>
<th>Outcome/ follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osborn and Ley (1986)³</td>
<td>American Journal of Neuroradiology</td>
<td>26/F</td>
<td>Tinnitus, visual field defects, headaches, seizure</td>
<td>Left frontal bone, 5 cm bony erosion of the inner table and thinning of the diploe</td>
<td>Large left frontal mass with edema and contralateral shift</td>
<td>Total resection</td>
<td>Low-grade astrocytoma</td>
<td>Good</td>
</tr>
<tr>
<td>Khan and Hashmi (2007)²</td>
<td>Pediatric Neurosurgery</td>
<td>15/M</td>
<td>Generalized convulsions, palpatation of the bony swelling in the right parietal area</td>
<td>Right anterior parietal region, remodeling and scalloping of the internal table of the skull</td>
<td>2-cm lesion in the right parietal area, hypointense on T1, hyperintense on T2, cystic component superficially located</td>
<td>Total resection</td>
<td>Low-grade astrocytoma</td>
<td>Good without recurrence or seizures / 4 months</td>
</tr>
<tr>
<td>Handzhiev et al (2017)⁶</td>
<td>Clinical Neuroscience</td>
<td>64/M</td>
<td>Headaches, right hemianopsia, right hemiparesis, palpation of the firm tissue mass with bony consistence over the left parietal area</td>
<td>Left parietal region, erosion of the inner table and the diploe with the intact external table</td>
<td>Heterogeneous left parietal mass</td>
<td>Total resection</td>
<td>Low-grade astrocytoma</td>
<td>Good without recurrence or seizures / 4 months</td>
</tr>
<tr>
<td>Lee et al (2017)⁷</td>
<td>Brain Tumor Research and Treatment</td>
<td>28/F</td>
<td>Symptoms due to increased intracranial pressure</td>
<td>Bony thinning on the right frontal bone</td>
<td>Cystic and enhancing mass on the right frontal lobe, dural enhancement suspected to be the dural tail sign</td>
<td>Total resection</td>
<td>Pilocytic astrocytoma</td>
<td>Recurrence at 9 months follow-up, second surgery (anaplastic astrocytoma). Postoperative radiation therapy, no tumor recurrence/ 3 years</td>
</tr>
<tr>
<td>Our case</td>
<td></td>
<td>23/M</td>
<td>Generalized convulsions, headaches, vomiting, palpation of the soft tissue through bony round defect in the left frontal area</td>
<td>Left frontal bone, destruction of both tables and diploe of the skull</td>
<td>3.5-cm intra-axial lesion in the left frontal area, hypointense on T1, hyperintense on T2, enhancement of superficial component, inward displacement of the hypointense dura suggesting extra-axial extension</td>
<td>Total resection</td>
<td>Low-grade astrocytoma</td>
<td>Good without recurrence or seizures / 6 months</td>
</tr>
</tbody>
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Abbreviations: CT, computed tomography; MRI, magnetic resonance imaging.
Conclusion

Superficially located low-grade astrocytoma can exceptionally cause calvarial and dural destruction. Although this is extremely uncommon, MRI may include low-grade astrocytoma in the differential diagnosis in such cases.

Authors’ Contributions
HB contributed to the conception, drafting, reporting of the cases and acquired the clinical data. AA contributed to the revision of the manuscript. All authors have read and approved the final manuscript.

Data Availability
All data generated and analyzed in this study are included in this article.

Informed Consent
The patient provided written informed consent for publication of patient clinical details and clinical images.

Conflict of Interest
None declared.

References