

oedema, perivascular cuffing (at basal ganglia, cortex, cerebellum and medulla) and cellular degeneration (at medulla and midbrain) were present in all cases with or without glial nodules [4]. In the other reported case, autopsy findings showed petechial haemorrhage over the white matter [5].

The predominant cerebellar involvement on MRI in our patient was of a similar location as described in the previous reported autopsical findings. Cerebellitis has also been reported in other viral encephalitis including VZV, HSV, EBV and enterovirus infection; whilst cingulate gyrus and insular involvement is typically reported in HSV [6,7]. The MRI findings showing cerebellar vasogenic oedema and gyriiform cortical hyperintensity also correlate well with reported autopsical histological findings of cerebral oedema and glial degeneration [4].

In the reported 7 cases of diphtheria encephalitis including our case, CSF findings were only reported in 2 cases (including ours). Both of the CSF findings were essentially normal with no organism isolated from CSF culture, and in one case a slightly elevated white cell count was present [5]. Thus in patients suspected of diphtheric encephalitis, our case highlights the importance of brain neuroimaging in making the diagnosis as the CSF findings may be unremarkable.

4. Conclusion

This is the first reported MRI findings of diphtheric encephalitis. Our case report reiterates the importance of clinicians being vigi-

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lant of possible paediatric diphtheria infection particularly in unvaccinated children. Our case also highlights the importance of neuroimaging in diagnosing diphtheric encephalitis especially under circumstances whereby CSF findings can be negative.

Conflict of interest/disclosures

The authors declare that they have no financial or other conflict of interest in relation to this research and its publication.

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Corpus callosal lipoma extending as nasal encephalocoel/cranial lipomeningocele



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ABSTRACT

A hitherto unreported case is presented wherein a 2 year old child had a 'cranial lipomeningocele' or a 'nasal lipo-encephalocoel'. The child presented with a growing mass in the base of the nose. Investigations revealed that the nasal mass was a lipoma that was an extension of intracranial lipoma. The intracranial component extended up to corpus callosum. Resection of the extracranial extension and basal reconstruction resulted in cosmetic recovery.

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1. Introduction

Corpus callosal lipomas have been frequently identified in the brain. A case is reported wherein intracranial lipoma extended extracranially in the base of the nose through a defect in the anterior cranial fossa floor. It is unclear if such a swelling should be labeled as a 'cranial lipomeningocele' or a 'nasal lipo-encephalocoel'. We could not locate any similar report in the literature.

2. Case report

A 2-year-old female child presented with a swelling at the base of the nose that was present since birth (Fig. 1). The parents reported that the swelling progressively grew in size. For last few months, the child had difficulty in breathing that resulted in disturbed sleep in the night and frequent crying spells. The child was otherwise normal, had no neurological deficits. Her milestones were normal for her age. A computed tomography of the brain showed a hypodense swelling suggestive of a lipoma that extended from the genu of the corpus callosum and extended extracranially through a paramedian defect in the anterior cranial fossa floor on the right side (Fig. 2). Due to cosmetic reasons and due to her

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Fig. 1. Picture of the patient with swelling at the root of the nose.

symptoms of breathlessness it was decided to treat the patient surgically. A bifrontal approach was planned and a bicoronal incision was taken behind the hairline. A vascularized pericranial flap was harvested at the time of raising the skin flap. Additional scalp flaps were obtained that were based on the temporalis muscle and its fascial layers [2–4]. After securing the vascularized flaps, a bifrontal craniotomy was performed. Extradural dissection was performed, bilateral frontal lobes were retracted and the defect in the midline of the anterior cranial fossa was circumferentially identified. It measured approximately 3 cm in diameter. The intracranial dura extended extracranially and covered the nasal dome of the lipoma. The extracranial lipoma and its meningeal cover was disconnected from cranial cavity and excised. The lipoma that had impacted itself into the base of the nasal cavity was also dissected and excised. The anterior cranial floor defect was repaired with the vascularized pericranial graft (Fig. 3). The lipoma extended intracranially up to the genu of the corpus callosum and it was straddled on the right side by a large pericallosal artery. The intracranial part of the lipoma was not handled during surgery. The child was well following the surgery. She was able to sleep comfortably. There was a depression at the nasal bridge

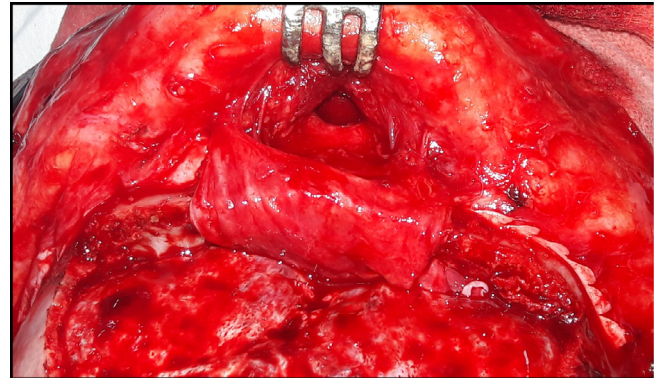


Fig. 3. Intraoperative picture showing the bifrontal craniotomy and the harvesting of the large pericranial flap that is rotated to cover the anterior cranial fossa floor defect.

where the swelling had been previously. A nasal reconstruction is now planned at a later date when the child grows up.

3. Discussion

Corpus callosal lipoma is a relatively rare but a well-defined clinical entity [7]. More often such lipomas are identified as incidental observations [5,9]. Less commonly, they are identified during investigations for epilepsy or for altered or abnormal behavior pattern. Lipomas are generally recognized to be a benign tumor growth that is a result of developmental aberration. Corpus callosal agenesis is a frequent association of corpus callosal lipoma [1]. Absence of growth in size and innocuous long-term presence are hallmarks of intracranial lipomas. Growth in the size of lipomas and malignant transformation has been recorded, but such events are identified as rare case reports.

The extension of an intracranial lipoma extracranially in the form of nasal encephalocele has never been recorded earlier in the literature. Such extension of lipoma from the brain extracranially mimicked a lipomeningocele wherein the lipoma in the spinal canal extends into the subcutaneous tissue. In general lipomas are known for their slow growth pattern. However, in the presented case the lipoma progressively grew in size. As cranial

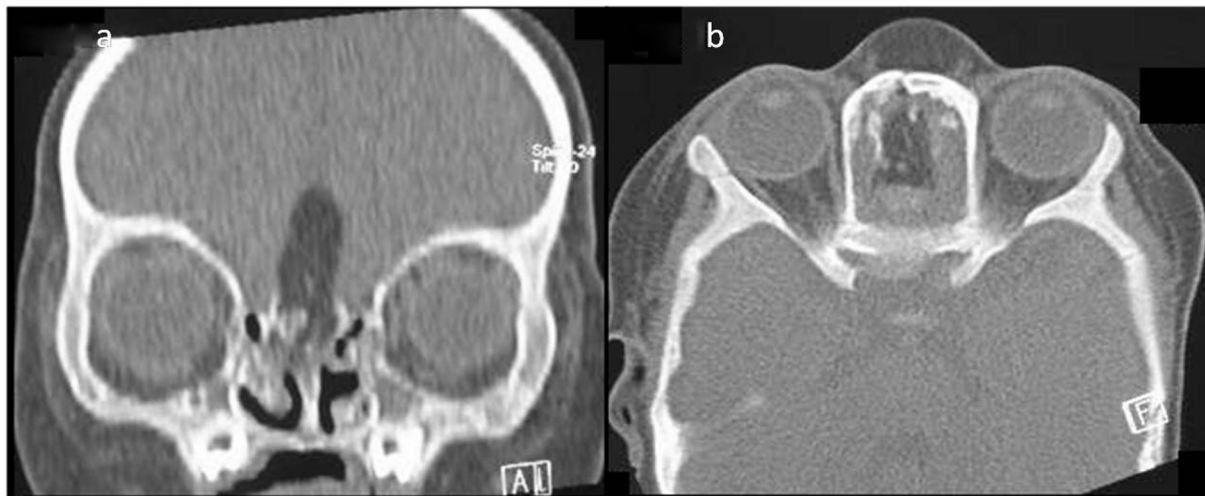


Fig. 2. CT scan of the patient. a. Coronal CT scan showing the lipoma communicating with the intracranial compartment. b. Axial CT scan showing the anterior cranial fossa floor defect and communication of the lipoma into the subcutaneous compartment.

investigations were not sequentially done, it is unclear if the cranial component of the lipoma also grew in size during the period. The child in the presented case had no other neural symptoms, and cosmetic deformation of the base of the nose was the major presenting complaint. Surgery involved disconnecting the nasal and cranial components of the lipoma, identification of the skull and dural defect and multilayered reconstruction of the anterior cranial fossa floor. A multilayered reconstruction of the anterior cranial fossa floor was done as discussed in our earlier reports [2–4]. The fat content of the extracranial extension into the nose was resected.

Our group had reported earlier a case of intracranial corpus callosal lipoma that extended superiorly into the cerebral parenchyma and then extended into the subcutaneous paramedian posterior frontal scalp [6,8,10]. The treatment in this case was disconnection of the intracranial part of the lipoma from extracranial lipoma and reconstruction of the convexity dural and bone defect.

The location and extension of fat in our case presents an opportunity to further evaluate the embryogenesis of the brain. Presence of fat within the confines of cranial cavity and its extension into the subcutaneous tissues raises questions about the genesis of such communication and its relationship with brain development.

Conflict of interest

The authors declare that they have no conflict of interest.

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Informed patient consent

The patient and his next of kin have consented to submission of this case report to the journal.

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Pathological crying induced by deep brain stimulation of the subthalamic nucleus in Parkinson's disease



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ABSTRACT

We report on a patient with deep brain stimulation (DBS) of the subthalamic nucleus (STN) for Parkinson's disease (PD) who developed pathologic crying (PC) immediately postoperatively. A spread of DBS current to adjacent cortico-ponto-cerebellar pathways might be responsible. In contrast to the few cases published previously, there was no pre-existing additional lesion that would have favoured occurrence of PC. Treating physicians should be aware of DBS-induced PC.

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1. Introduction

Deep brain stimulation (DBS) of the subthalamic nucleus (STN) has become an accepted treatment for patients with Parkinson's disease (PD) [1]. STN-DBS is generally well tolerated, when contraindications are considered [2].

Pathological crying (PC) is a disorder of emotional expression [3]. Affected subjects present with episodic crying inappropriate to their own emotional perception, independent of any underlying

mood or personality disorder. In a milder form crying and feelings may not be completely incongruent, but of considerably higher intensity or frequency compared to previously [4].

2. Case report

We report on a 66-year-old man, diagnosed with idiopathic PD at age 56. At age 64 he complained about motor fluctuations under medication with Ropinirole 24 mg/d and Selegiline 2.5 mg/d. The UPDRS (III) motor score varied between 37 (On) and 49 (Off). After comprehensive evaluation with unremarkable psychiatric assessment and only discrete executive deficits in neuropsychological evaluation, he underwent STN-DBS operation at age 65. Preopera-

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