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Nasal Glial Heterotopia in a 2-Year-Old Child: A Case Report and Literature Review

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Abstract **Case Report**

Nasal gliomas or heterotopias are rare, nonhereditary, congenital midline or paramedian tumors composed of heterotopic neuroglial tissue. We report the case of a 2-year-old boy with a right later nasal swelling present since birth, associated at birth with an anterior skull-base meningo-encephalocele with right naso-orbital development for which he underwent neurosurgical repair at the age of 6 months. He was subsequently referred to the maxillofacial surgery department at the age of 2 years for management of the residual nasal mass. Imagery revealed a well-defined ovoid right paranasal soft-tissue process compressing the frontal process of the maxilla without bone lysis and respecting the homolateral orbit, together with a persistent and widened front-nasal suture, and no abnormal cerebral parenchymal enhancement. Complete surgical excision was performed through an external later nasal cutaneous approach. Histopathological examination confirmed the presence of a nasal glial heterotopia without signs of malignancy. The postoperative course was uneventful, with no recurrence. This report reviews the clinical, radiological, and histological features of this rare malformation, together with the main embryopathogenic hypotheses discussed in the literature.

Keywords: Heterotopic tissue, Nasal glioma, Congenital malformation, Pediatrics, Encephalocele.

INTRODUCTION

Nasal glial heterotopia is a rare congenital malformation also referred to as nasal glioma a term that is now considered a misnomer, since the mature neural tissue present in these lesions is not neoplastic in origin but rather represents a developmental anomaly[1][2]. Glial heterotopia is defined as the presence of mature glial cells outside the central nervous system. The nose and nasopharynx are the most commonly involved areas for this anomaly[1]. This congenital lesion is part of a large panel of midline dysraphias and craniofacial anomalies and includes other entities such as dermoid cysts, encephaloceles, and hemangiomas[3]. Nasal glial heterotopias are nonhereditary and usually present in infancy[4][3].

With the refinement of prenatal diagnostic tools, including sonography and fetal MRI, these lesions are increasingly suspected before birth, though postnatal imaging remains mandatory to assess potential intracranial communication [5]. We report here a new case of nasal glial heterotopia in a 2-year-old child, initially associated at birth with an anterior skull base meningoencephalocele [that was treated with neurosurgery during early childhood], and we discuss the clinical management and embryological implications of the nasal glial heterotopia.

CASE REPORT

A 2-year-old boy was referred to our maxillofacial surgery department for the management of a right lateronasal swelling present since birth. Initially the swelling was associated with an anterior skull-base meningo-encephalocele with right naso-orbital development, documented on cranio-facial MRI in infancy, for which he had undergone neurosurgical repair at the age of 6 months. The initial cranio-facial MRI performed [3D T1 and 3D T2 sequences] had demonstrated a bony defect of the anterior skull base, at its junction with the right orbit and in front of the internal canthus of the right eye, measuring 8.5 mm in width and 17 mm in height, through which the right basifrontal cerebral parenchyma herniated together with its meningeal coverings. The herniated parenchyma extended to the right internal canthus, displaced the nasal septum and the ethmoid to the left and the right globe

outward, with no parenchymal abnormalities. The appearance was that of an anterior skull-base meningo-encephalocele with right naso-orbital development, which was repaired neurosurgically at the age of 6 months. The boy was later referred to our maxillofacial department at the age of two. On clinical examination, there was a right lateronasal swelling with healthy overlying skin, measuring approximately 3 cm along its long axis, firm, non-tender, and mobile relative to both the superficial and deep planes, with no vascular thrill. The swelling extended toward the right internal canthus, causing widening of the inter-canthal distance [Figure 1]. There was no limitation of ocular motility.

Figure 1: Preoperative clinical photograph showing the right lateronasal swelling extending to the internal canthus

The radiological workup included cerebral and facial MRI performed using sagittal T1, axial T2, T2 FLAIR, diffusion-weighted, and axial/sagittal gadolinium-enhanced T1 sequences, complemented by CT scan. They demonstrated a well-defined, ovoid expansive process within the subcutaneous fat of the right paranasal region, extending toward the internal canthus, appearing hypointense on T1 and of intermediate signal on T2, with peripheral gadolinium enhancement, measuring 20 × 24 mm in the axial plane with a craniocaudal height of 31 mm. The lesion showed no diffusion restriction. It compressed the frontal process of the maxilla without bone lysis and respected the homolateral orbit. Additionally, they showed a persistent and widened fronto-nasal suture, with a few calcifications along the internal border of the frontal bone, consistent with the sequelae of the previously repaired frontal defect. No abnormal contrast enhancement was seen within the supra- or infratentorial cerebral parenchyma, and no residual intracranial communication was identified. Incidental findings included opacification of the maxillary and ethmoidal cells, consistent with chronic bilateral ethmoido-maxillary sinusitis, together with a right otitis media. Overall, the radiological appearance was consistent with a nasal soft-tissue mass suggestive of nasal glioma [Figure 2].

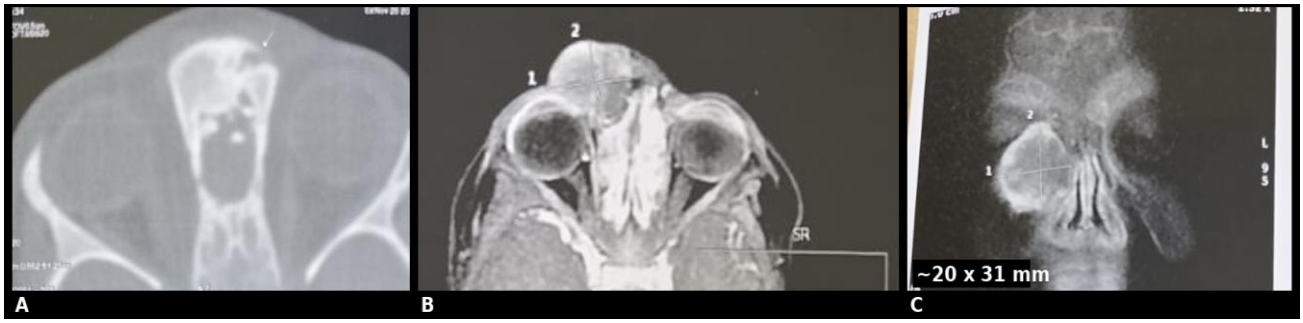

Figure 2: Imaging of the right paranasal mass: [A] bone-window CT showing the paramedian soft-tissue mass and the fronto-nasal bony region; [B] axial gadolinium-enhanced T1-weighted MRI showing the well-defined right medial-canthal mass [calipers] between the orbits; [C] coronal MRI showing the mass with its measurements [approximately 20 and 31 mm]. Patient identifiers have been removed

Given this suggestive radiological appearance, complete surgical excision was performed at the age of 2 years under general anaesthesia. Through a right lateronasal incision, subcutaneous dissection was carried out around the entire mass and in a subperiosteal plane over the nasal vault, allowing en bloc excision of the lesion. Histopathological analysis of the surgical specimen [a 7 g nodular formation measuring 3 × 2.5 × 1.5 cm] demonstrated, within a background of gliosis, the presence of mature astrocytes without neuronal cells, with regular nuclei and no atypia, associated with areas of fibrosis, and without any sign of malignancy. This morphological appearance was consistent with glial heterotopia, confirming the radiological diagnosis. The postoperative course was uneventful, with no local recurrence at the available follow-up.

DISCUSSION

Epidemiological and Clinical Data

First described by Reid in 1852 [1], with the term “glioma” introduced by Schmidt in 1900,[6] nasal gliomas remain rare lesions, with an estimated incidence of 1 per 20,000 to 40,000 live births[7]. The comprehensive systematic review , published by Gallego Compte *et al.,* in 2022, collected 152 cases from 72 original publications: it confirms a male predominance with a sex ratio of 3:2, with 86% of patients are diagnosed and treated before the age of 3 years [66% before 1 year], and only 8% of cases being diagnosed in adulthood[8]. Our patient, treated at age 2, thus falls within the most represented age group. Reported localization frequencies vary across series between the intranasal form, the extra nasal form and combined

forms, earlier series reported a higher prevalence of intranasal lesions [56%]with extra nasal [33%] and combined form less frequent [11%],[2][8]. Our case, with right latero nasal localization and extra nasal form, fits within the typical anatomical distribution described in the manuscript by Saettele and al. in 2012 [9]. Clinically, the most common presentation remains an asymptomatic mass [45–49% of cases] or nasal congestion [38–41%], the isolated extra nasal form typically manifesting as a firm, non-compressible and non-pulsatile mass, not varying in size with the Furstenberg test or crying, unlike encephaloceles[10][11]. Nasal glial heterotopia accounts for approximately 5% of all congenital nasal masses, and beyond the palpable swelling itself, recognized clinical manifestations include nasal obstruction, nasal polyps, chronic sinusitis, and chronic otitis media, as well as an increased inter-canthal distance or hypertelorism when the lesion extends toward the medial canthus[6][12]. In our case, the latero nasal swelling was present since birth and was accompanied by a widened inter-canthal distance and by chronic ethmoido-maxillary sinusitis with a right otitis media, findings concordant with these described associations rather than purely incidental[9].

Role of Imaging

With the improvement in prenatal screening, congenital midline masses are increasingly detected during routine second-trimester ultrasound examinations[9]. When a nasal mass is suspected prenatally, fetal MRI is generally recommended to aid differentiation from dermoid cysts, encephalocele, or vascular anomalies[5]. Gasparella *et al.,* reported two cases of nasal glial heterotopia both suspected during prenatal ultrasound as early as 21 and 29 weeks of gestation. However, postnatal imaging remains mandatory regardless of prenatal findings, as a stalk-like connection to the dura may be missed prenatally and correct diagnosis of intracranial communication is only possible when extracranial CSF flow can be visualized[13]. MRI holds a central role in the preoperative workup, allowing assessment of the tissue nature of the lesion, its local extent, and most importantly the presence or absence of intracranial communication, a key factor in surgical strategy. In the review by Gallego Compte *et al.,*, MRI was the most frequently used modality [39% of cases, alone or combined with CT in an additional 20% of cases]; preoperative diagnostic biopsy remained exceptional [5% of cases], owing to the risk of meningeal breach in cases of undetected intracranial communication [8]. Classically, as illustrated by the index case of Jartti *et al.,* [MRI of a nasal glioma in a 5-week-old infant],[14] the lesion exhibits a gyri-form structure reminiscent of gray matter on T1 and T2, with peripheral gadolinium enhancement limited to the overlying mucosa[14]. In our case, the mass showed T1 hypointensity and intermediate T2 signal with peripheral gadolinium enhancement, and extended toward the internal canthus without crossing the skull base on current imaging a profile compatible with a paranasal glial heterotopia with limited deep extension. The associated persistent and widened fronto-nasal suture with calcifications, together with the anterior skull-base meningo-encephalocele documented on infant MRI, situates this case within the encephalocele–heterotopia spectrum and underscores the value of combined facial and cerebral imaging.[15]

Differential Diagnosis

Patterson *et al.,* emphasized that when a biopsy specimen from a nasal or pharyngeal mass in an infant reveals mature neural tissue, the differential diagnosis is limited to three entities: teratoma, encephalocele, or heterotopic brain tissue[3]. A teratoma, containing tissue from all three germ layers, requires careful microscopic sampling of the entire excised specimen [at least one tissue block per centimeter]; a finding of mature neural tissue alone in a biopsy sample does not exclude this diagnosis. The differential diagnosis should also include retinoblastoma in some contexts, although both are typically excluded by prenatal and postnatal MRI.[16]

The main clinical differential diagnosis remains nasal hemangioma. Macroscopically, nasal glial heterotopias present as hard, incompressible masses with a homogeneous purple surface, whereas congenital hemangiomas [CH] have pale halo borders and a softer consistency[13]. Although CH can macroscopically mimic a nasal glioma, Doppler ultrasound can further assist: hemangiomas show high-velocity arterial flow at end-diastole, whereas glial heterotopia shows low-velocity flow[17]. This distinction is clinically decisive, as hemangiomas undergo spontaneous involution, while glial heterotopia never regresses spontaneously and always warrants surgical excision.

Regarding the distinction from encephalocele: a bony defect of the skull is always present in encephaloceles but can also be found in up to 20% of gliomas[5][18]. When leptomeninges are recognizable histologically, an encephalocele diagnosis is supported; when absent, the distinction from glial heterotopia can only be established after clinicopathological correlation, since glial tissue may be the predominant or exclusive histological component in both entities [14][17].In the systematic review by Gallego Compte *et al.,*, an intracranial fibrous tract was found in 11% of analyzable cases, without direct CSF communication with the subarachnoid spaces. In our case, an anterior skull-base meningo-encephalocele with naso-orbital development was documented on infant MRI and required neurosurgical repair at 6 months; on the subsequent imaging performed before maxillofacial excision, no residual intracranial communication was identified, and the excised nasal lesion corresponded to glial heterotopia. Of note, misdiagnosis of even a tiny intracranial communication could lead to incomplete meningeal closure with subsequent CSF leakage and risk of meningitis.[4]

Histopathological and Immunohistochemical Aspects

Histologically, nasal glial heterotopia is composed of nodules of mature neuroglial tissue, without mitoses, embedded in varying amounts of fibrovascular stroma a consistent pattern across series regardless of lesion localization[2]. Reactive glial changes, including increased cellularity and gemistocytic astrocytes, are commonly observed, as are focal calcifications; these findings are thought to reflect poor vascular supply in the ectopic location. Neurons are identifiable in up to 10% of published cases,[19] and were not observed in our patient's specimen. Ependymal-lined clefts and choroid plexus-like structures are reported in approximately two-thirds of pharyngeal heterotopias but are rarely if ever seen in nasal gliomas, suggesting origin at an earlier point of embryogenesis in the pharyngeal forms[20].The glial nature of the lesion is confirmed by phosphotungstic acid hematoxylin [PTAH] staining, which colors neural tissue red/magenta, and more reliably by immunohistochemistry against glial fibrillary acidic protein [GFAP] and S100 protein [2]. Electron microscopy further characterizes the lesion: closely packed anisomorphic glial processes contain prominent 10-nm intermediate filaments, and a well-defined basal lamina separates cell processes from collagen bundles features ultrastructurally consistent with fibrous astrocytes[21] Masson's trichrome additionally highlights reactive fibrosis. In our case, histopathological examination revealed mature astrocytic gliosis with regular nuclei, no atypia, areas of fibrosis, and no sign of malignancy, fully consistent with published series. A complementary immunohistochemical study [GFAP, S100] was not available in our report but would be recommended in cases of diagnostic uncertainty.

Embryological Aspects

The pathogenesis of nasal glial heterotopia is not definitively established. Three main mechanisms have been proposed [3]: first, a frontal encephalocele losing its cranial connection the most widely accepted theory; second, an isolated totipotential cells giving rise to a glial tumor [monophasic teratoma]; and third, abnormal entrapment or migration of glial cells from the olfactory bulbs. Support for the encephalocele theory comes from two sources: first, numerous reports describe transitional forms between encephaloceles and heterotopias; second, up to 25% of nasal gliomas are associated with an underlying bony defect, with or without dural attachment via a fibrous stalk,[2] and the reported sites of encephaloceles and brain heterotopias closely overlap anatomically.

The prenasal space theory proposed by Grünwald in 1910,[9] a variant of the encephalocele hypothesis, postulates a virtual space between the nasal bones, the cartilaginous nasal capsule, and the dura mater, through which a dural projection transits at the level of the foramen cecum. Gasparella *et al.,* further emphasized premature closure of the metopic suture during the first phase of the embryonic period as the precipitating event, explaining the entrapment of herniated brain tissue and the presence of an intracranial communication in approximately 15–20% of cases.[5]In our observation, the initial documentation of an anterior skull-base meningo-encephalocele with naso-orbital development, repaired neurosurgically at 6 months, followed by the absence of residual intracranial communication on subsequent imaging with a residual nasal component corresponding to glial heterotopia, provides direct clinical support for this encephalocele-derived mechanism. This theory accounts well for the glioma–encephalocele spectrum but is contested for dermoid cysts of the same topography, as it incorrectly assumes a neuroectodermal origin of the dura mater, whereas the dura derives from the ectomesenchyme of the neural crests.[22]

Therapeutic Management

The standard treatment for nasal glial heterotopia is complete surgical excision, with mandatory histological confirmation of total extirpation, as recurrence in cases of microscopic residuals is described in up to 10% of cases.[5] Early resection is advised in order to avoid the development of craniofacial deformities, particularly concerning the nasal architecture, and also to protect from potential visual impairment due to orbital extension.[4] A multidisciplinary approach involving maxillofacial or ENT surgeons, neurosurgeons, plastic surgeons, and neuroradiologists is recommended, particularly when intracranial extension via a fibrous tract toward the foramen cecum is suspected [11% in the most recent systematic review, up to 25% in older series].[2]

Extranasal gliomas are most commonly approached via an external route [external rhinoplasty, midline incision, or coronal approach depending on localization and volume], while the endoscopic or intranasal approach is preferred for intranasal forms. Soft-tissue reconstruction after excision represents a particular challenge, requiring the combination of effective complete tumor removal, closure of any intracranial communication, and an acceptable aesthetic result. Gasparella *et al.,* reported two complementary techniques: a glabellar rotation flap for moderate defects, providing satisfactory cosmesis; and a full-thickness skin graft harvested from the groin for larger skin surface defects, for which a rotational flap would have caused excessive tension[5].

The overall recurrence rate ranges from 4% to 14% depending on series, with recurrences occurring classically between 5 weeks and 11 months after initial surgery[8][17]. In the systematic review by Gallego Compte *et al.,* negative histological margins were associated with no recurrence in all evaluable cases, while a single case with positive margins required reoperation arguing for systematic margin analysis and prolonged surveillance in uncertain cases. In our patient, no recurrence was observed at the available follow-up. A

clinical monitoring of at least 12 months is recommended as virtually all published recurrences occur within this window.

Conclusion

Nasal glial heterotopia is a rare congenital malformation that should be systematically considered in any midline or paramedian mass of the nasal pyramid in infants or young children, particularly in the absence of pulsatility or transillumination. With improving prenatal screening, these lesions may now be suspected as early as the second trimester; however, postnatal MRI remains mandatory to exclude intracranial communication and plan an appropriate interdisciplinary surgical approach[18]. Treatment relies on complete surgical excision with histologically verified margins, the postoperative course of which is generally straightforward, as illustrated by this case. The documented progression in our patient, from an anterior skull-base meningo-encephalocele repaired in infancy to a residual nasal glial heterotopia excised at 2 years, underscores the value of systematic cerebral radiological exploration and of a coordinated neurosurgical and maxillofacial approach in the workup of these lesions.

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