Lateral Sphenoidal Encephalocele as an Uncommon Cause of Trigeminal Neuralgia: A Case Report

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Dr. Yaser Al Msalmeh & Dr. Said Huwaijah: Writing – review & editing. Supervision.

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Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

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ABSTRACT

Introduction: Meningoencephaloceles are rare structural defects characterized by the herniation of brain tissue, meninges, and cerebrospinal fluid through bony defects in the skull base. While typically congenital, they may also result from trauma or chronically elevated intracranial pressure. Lateral sphenoidal encephaloceles, a rare subtype of basal encephaloceles, can present with neurological symptoms depending on their location. We describe a rare case of trigeminal neuralgia (TN) secondary to a lateral sphenoidal encephalocele, highlighting an underrecognized structural etiology of this condition.

Case Presentation: A 35-year-old woman with a body mass index (BMI) of 37.4 presented with recurrent, paroxysmal electric shock-like pain localized to the proper trigeminal nerve distribution, consistent with classical TN. Initial brain MRI suggested a sphenoid sinus mucocele. However, a contrast-enhanced MRI performed one year later demonstrated herniation of gliotic temporal lobe tissue and enhancing meninges into the sphenoid sinus, consistent with a lateral sphenoidal encephalocele. CT imaging confirmed a bony defect in the lateral wall of the sphenoid sinus. The clinical and radiological findings supported a diagnosis of secondary TN due to the encephalocele. The patient was managed conservatively with oxcarbazepine, with partial symptom improvement.

Discussion: Lateral sphenoidal encephaloceles are rare but essential structural causes of trigeminal neuralgia, particularly due to their proximity to the trigeminal nerve. While classical TN is most often due to neurovascular compression, secondary causes such as encephaloceles should be considered in atypical cases. This case highlights the importance of contrast-enhanced imaging of the skull base in patients with unexplained facial pain, underscoring encephaloceles as a potentially underrecognized etiology.

CASE REPORT

BACKGROUND

Lateral sphenoidal encephaloceles are exceedingly rare and often misdiagnosed structural anomalies of the skull base. While trigeminal neuralgia (TN) is most commonly caused by neurovascular compression, secondary structural causes remain an important differential in atypical or refractory cases. To date, only a handful of cases have linked encephaloceles, particularly those involving the lateral sphenoid sinus, to trigeminal symptoms. This case report contributes to the limited literature by emphasizing the diagnostic value of high-resolution contrast-enhanced MRI and CT in identifying subtle skull base defects. Moreover, it reinforces the need to consider structural lesions in patients with persistent or atypical TN, especially in those with risk factors such as obesity or suspected elevated intracranial pressure.

INTRODUCTION

Meningoencephaloceles refer to the herniation of brain parenchyma, meninges, and cerebrospinal fluid through a defect in the skull or skull base. While predominantly congenital, they can also arise secondary to trauma, neoplasms, or chronically elevated intracranial pressure [1]. The global birth prevalence of encephaloceles is estimated to be 1–5 per 10,000 live births, with classification based on anatomical location, commonly occurring in the occipital, frontal (sincipital), and parietal regions [2,3]. A less frequent variant, basal encephalocele, constitutes approximately 1.5% of cases and involves herniation through the skull base [4]. This subtype is further divided into transethmoidal, spheno-orbital, sphenomaxillary, and transsphenoidal types, the latter representing approximately 5% of basal encephaloceles, with a prevalence of 1 in 70,000 live births [5]. Clinical presentation ranges from asymptomatic cases to those with seizures, visual disturbances, or signs of elevated intracranial pressure [2].

Trigeminal neuralgia (TN), a common cause of facial pain, has an annual incidence of 4 to 13 per 100,000 individuals [6]. The classic form, accounting for approximately 75% of cases, results from neurovascular compression [7]. Secondary TN, comprising 15% of cases, is attributed to structural abnormalities such as demyelinating lesions or compressive masses, while idiopathic TN (10%) lacks identifiable pathology on imaging [8,9]. Although most secondary causes are well-characterized, rare structural lesions، including encephaloceles, should be considered, particularly those involving the skull base [10].

We report a unique case of a 35-year-old woman presenting with trigeminal neuralgia due to a lateral sphenoidal encephalocele. This report underscores an exceedingly rare etiology of TN and highlights the importance of considering atypical structural lesions in the diagnostic evaluation of facial pain.

CASE PRESENTATION

A previously healthy 35-year-old woman reported to the outpatient department with recurrent, electric shock-like pains localized to the right temporal region for many months. Each episode lasted approximately 5 seconds and occurred up to five times per day. The pain was confined to the distribution of the right trigeminal nerve, without associated facial numbness, tingling, or weakness. The episodes were exacerbated by stress and alleviated by rest and sleep, occasionally interfering with her ability to focus during the day. There were no red flag symptoms such as visual disturbances, hearing changes, or constitutional signs. She denied any history of previous trauma. Her medical, surgical, and social history was otherwise unremarkable, aside from smoking approximately 10 cigarettes per day.

On examination, she was hemodynamically stable with a BMI of 37.4. General and neurological examinations, including cranial nerve assessment, were unremarkable. No facial asymmetry, motor deficits, or signs of elevated intracranial pressure were noted.

Based on her clinical presentation, a diagnosis of trigeminal neuralgia (TN) was made. She was started on carbamazepine, which provided partial relief but was discontinued due to adverse effects. She was subsequently switched to oxcarbazepine.

Brain MRI with intravenous contrast was performed to evaluate for a structural cause. It revealed a defect in the lateral wall of the right sphenoid sinus, with herniation of temporal lobe tissue, meninges, and cerebrospinal fluid into the sinus cavity. The herniated brain parenchyma demonstrated gliosis, and the enhancing meningeal tissue was consistent with a lateral sphenoidal encephalocele.

A complementary brain CT scan with and without contrast confirmed herniation of the right temporal lobe into the sphenoid sinus, measuring 3.5 × 2.5 × 2.0 cm, through a bony defect in the lateral sphenoid sinus wall measuring 8 × 7 × 6 mm.

The anatomical proximity of the encephalocele to the trigeminal nerve correlated with the patient's clinical symptoms, supporting a diagnosis of secondary TN caused by a lateral sphenoidal encephalocele. The patient was referred to neurosurgery; however, because her symptoms remained stable and based on her personal preference, conservative management was continued. At the most recent follow-up, she reported a reduction in symptom frequency while continuing oxcarbazepine therapy (Figures 1-8).

DISCUSSION

Lateral sphenoidal encephaloceles involve herniation of brain tissue into the sphenoid sinus through bony defects in the greater wing of the sphenoid, often caused by congenital thinning of the lateral recess [1]. This congenital vulnerability can be exacerbated by chronically increased intracranial pressure, leading to skull base dehiscence and the persistence of embryonic structures, such as the Sternberg canal [11]. Other causes include trauma, previous surgery, neoplasms, and chronic infections that weaken the skull base [12].

In adults, clinical signs vary greatly. The most common symptoms include spontaneous cerebrospinal fluid (CSF) rhinorrhea and recurrent meningitis. One case described a 53-year-old woman presenting with strabismus and hypertelorism, features suggestive of a congenital cause [13]. Other reported symptoms include visual and endocrine problems, such as bitemporal hemianopsia, diabetes insipidus, and nystagmus, due to involvement of the optic pathway [13,14].

Although rare, sphenoidal encephaloceles can present as trigeminal neuralgia (TN) due to their anatomical connection with the trigeminal nerve. The trigeminal ganglion is located within Meckel's cave near the petrous apex, and its maxillary (V2) and mandibular (V3) branches exit through the foramen rotundum and foramen ovale, respectively—both positioned in the greater wing of the sphenoid [15]. A lateral encephalocele, especially of the transalar subtype, may exert mass effect on these structures, leading to nerve compression. Similar mechanisms have been reported in cases involving meningoceles of Meckel's cave [16]. Additionally, chronic pulsatile CSF flow within the herniated sac may cause demyelination, mimicking the pathophysiology of classic neurovascular compression. Direct irritation from prolapsed nerve rootlets into the encephalocele sac has also been suggested as a cause of pain [10].

Our case aligns with the demographic and radiologic features described in the most extensive systematic review to date, which identified nine cases of encephalocele-associated TN, all in female patients with a median age of 43.5 years [10]. About one-third of these presented with classic TN, and several showed radiological signs of intracranial hypertension, such as an empty sella, although CSF rhinorrhea was not observed. Notably, elevated body mass index (BMI) has been linked to spontaneous skull base defects, especially in cases of idiopathic intracranial hypertension—a plausible contributing factor even without papilledema or CSF leak [17]. This could be relevant in our patient, a 35-year-old woman with a high BMI, who experienced classic paroxysmal facial pain without sensory deficits or signs of increased intracranial pressure.

Surgical repair is typically indicated in the presence of CSF leaks, recurrent meningitis, or progressive neurological deficits [18]. Endoscopic endonasal approaches, particularly transpterygoid techniques, have become the preferred modality due to their minimally invasive nature and high success rates, often exceeding 90% when combined with multilayer closure and vascularized flaps [18,19]. Transcranial approaches, such as a middle cranial fossa craniotomy, are reserved for cases not amenable to endonasal access or when resection of gliotic brain tissue is necessary [20]. Conversely, conservative management may be appropriate in asymptomatic or minimally symptomatic patients, especially in the absence of CSF leak or infection. While anterior cranial fossa encephaloceles are typically repaired prophylactically due to their higher risk of complications, those arising from the middle cranial fossa have been shown to remain stable with observation [21].

In our patient, the absence of CSF rhinorrhea, stable neurological condition, and patient preference supported a non-surgical approach with ongoing medical therapy. The exact cause of the encephalocele remains unclear, although congenital factors likely worsened by elevated intracranial pressure are probably involved. This case adds to the limited but increasing literature describing sphenoidal encephaloceles as an uncommon cause of secondary TN. It highlights the importance of considering structural lesions in the differential diagnosis of unusual or resistant facial pain. It emphasizes the diagnostic value of high-resolution, contrast-enhanced skull base imaging in such cases.

CONCLUSION

This case highlights that even in patients with a classic clinical presentation of trigeminal neuralgia, rare structural causes such as lateral sphenoidal encephaloceles may underlie the symptoms. Although the majority of trigeminal neuralgia cases are attributed to neurovascular compression, secondary causes should be considered when the clinical course is atypical. This reinforces the value of neuroimaging in select cases, as it can reveal unexpected etiologies that may alter the diagnostic and therapeutic approach. Our report contributes to the limited but growing literature on encephalocele-related trigeminal neuralgia, underscoring the importance of individualized evaluation and treatment.

TEACHING POINT

Contrast-enhanced MRI and CT of the skull base are essential in evaluating patients with atypical trigeminal neuralgia, as rare structural lesions such as lateral sphenoidal encephaloceles may be the underlying etiology—highlighting the need for thorough radiological assessment beyond neurovascular conflict.

QUESTIONS

Q1: What is the most common cause of classical trigeminal neuralgia?

(a) Skull base encephalocele
(b) Demyelinating lesions
(c) Vascular compression of the trigeminal nerve root (□)
(d) Sphenoid sinusitis
(e) Trigeminal schwannoma

Q2: Which imaging modality is most useful in identifying encephaloceles involving soft tissue and meningeal herniation?

(a) Non-contrast CT
(b) Contrast-enhanced MRI (□)
(c) Skull X-ray
(d) MR spectroscopy
(e) Plain sinus radiograph

Q3: What anatomical feature predisposes the lateral sphenoid sinus to encephalocele formation?

(a) Cribriform plate erosion
(b) Thin lateral recess and incomplete fusion at the skull base (□)
(c) Mastoid air cell pneumatization
(d) Hypoplastic temporal bone
(e) Prominent jugular bulb

Q4: In which of the following clinical scenarios should a secondary cause of trigeminal neuralgia be strongly considered?
(a) A patient over 60 with no neurological deficits
(b) TN that responds well to carbamazepine
(c) Bilateral TN symptoms or young age of onset (□)
(d) TN associated with migraines only
(e) Short, rare episodes of cheek pain

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FIGURES

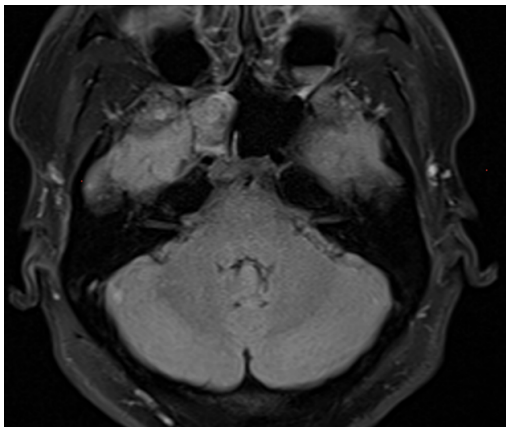

Figure 1: Axial FLAIR shows the herniated brain parenchyma at the right recess of the sphenoid sinus

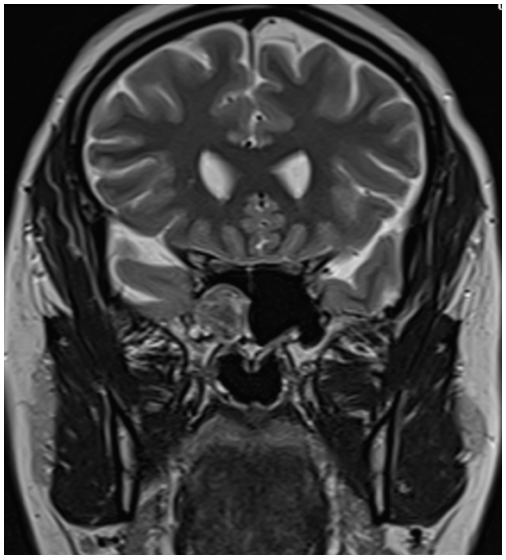

Figure 2: Coronal T2-weighted MRI demonstrating high-resolution visualization of the ventricles and temporal lobes, with evidence of brain herniation into the right recess of the sphenoid sinus.

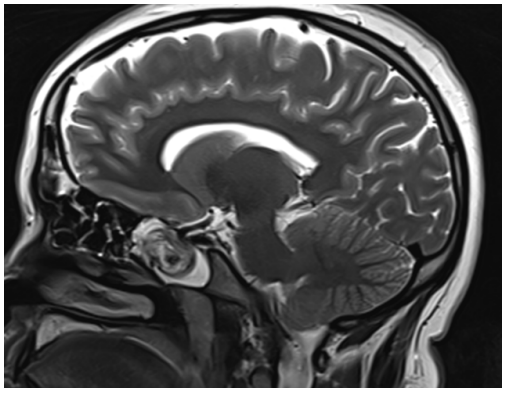

Figure 3: Sagittal T2-weighted MRI showing clear visualization of midline structures, including the corpus callosum and brainstem, with evidence of brain herniation into the right recess of the sphenoid sinus.

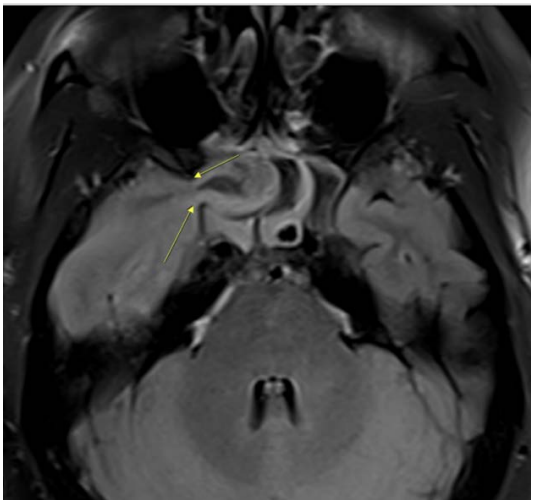

Figure 4: Axial FLAIR with arrows indicating the defect at the lateral wall of the right sphenoid sinus.

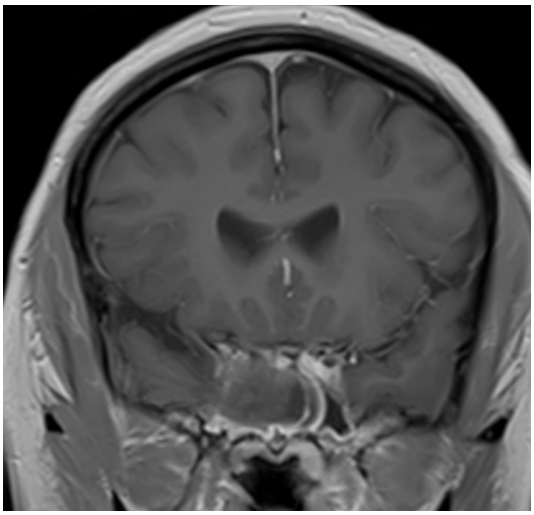

Figure 5: Coronal T1-weighted post-contrast MRI showing herniated brain tissue into the sphenoid sinus. Enhancing structures suggest involvement of the cavernous sinus and adjacent vascular structures.

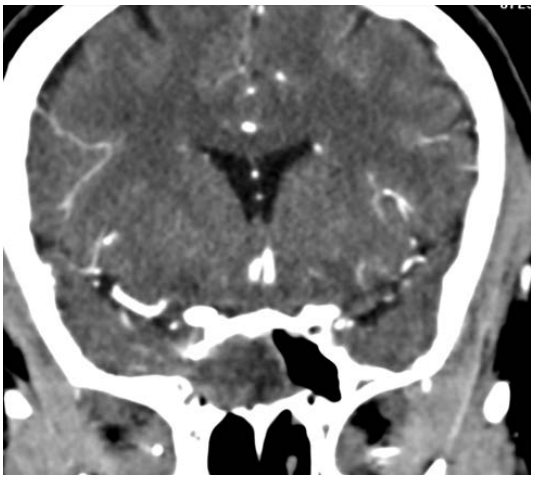

Figure 6: Coronal contrast-enhanced CT scan shows herniation of right temporal lobe tissue into the sphenoid sinus through a bony defect in the lateral wall of the sphenoid sinus.

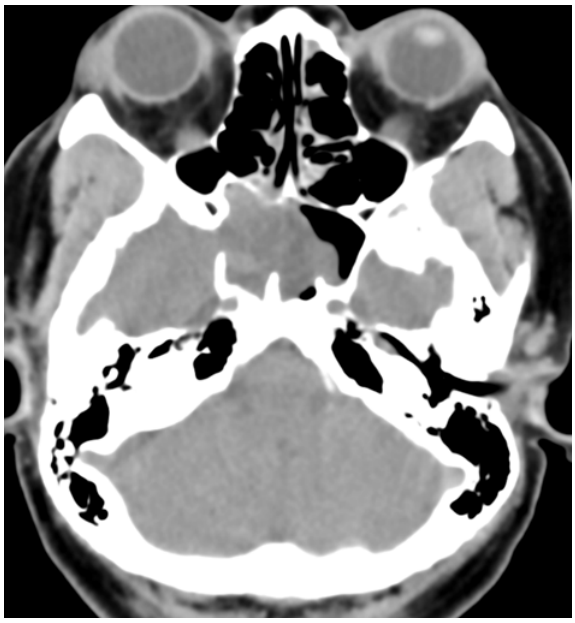

Figure 7: Axial non-contrast CT demonstrates the herniated brain parenchyma in the right part of the sphenoid

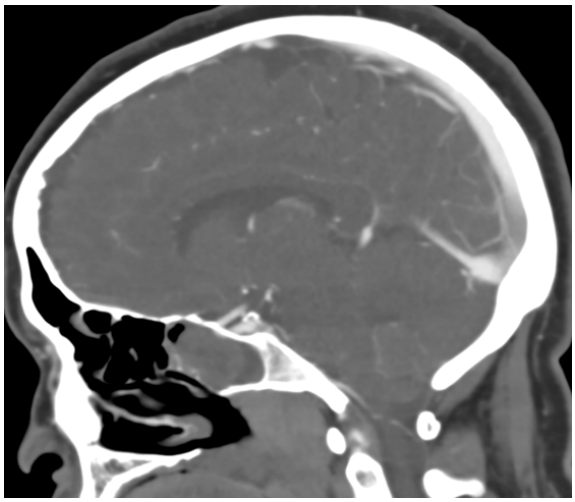

Figure 8: *Sagittal contrast-enhanced CT confirming the encephalocele sac extending into the sphenoid sinus, without evidence of associated CSF leak or mass effect on adjacent structures.*

KEYWORDS

Lateral sphenoidal encephalocele, Trigeminal neuralgia, Basal encephalocele, Idiopathic intracranial hypertension, Skull base imaging, Secondary trigeminal neuralgia

ABBREVIATIONS

BMI = Body Mass Index
CSF = Cerebrospinal Fluid
CT = Computed Tomography
MRI = Magnetic Resonance Imaging
TN = Trigeminal Neuralgia

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