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Atypical Presentation of Cerebellopontine Angle Dermoid Cyst: A Case of Secondary Trigeminal Neuralgia

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A B S T R A C T

Dermoid cysts are rare congenital ectodermal inclusion cysts, accounting for a small percentage of all intracranial tumors. Their occurrence in the cerebellopontine angle (CPA) is infrequent, and presentation as isolated trigeminal neuralgia (TN) is considered atypical. This study underscores that rare congenital lesions like dermoid cysts can manifest with relatively common neurological symptoms, prompting clinicians to consider a broader differential diagnosis A 60-year-old female presented with a two-year history of paroxysmal, shock-like pain in the left cheek and intraoral area, triggered by light touch, consistent with trigeminal neuralgia. Neurological examination was otherwise largely unremarkable initially, though the pain significantly impacted her quality of life and nutritional intake, leading to weight loss. Magnetic Resonance Imaging (MRI) revealed an extra-axial lesion in the CPA, compressing the trigeminal nerve. The lesion exhibited characteristics suggestive of an epidermoid or arachnoid cyst initially, but with features also compatible with a dermoid cyst. The patient underwent a retrosigmoid craniotomy for microsurgical excision of the lesion. Intraoperatively, a cystic lesion with contents suggestive of a dermoid cyst was found adherent to the trigeminal nerve and surrounding structures. Histopathological examination confirmed the diagnosis of a dermoid cyst. Postoperatively, the patient experienced significant relief from her trigeminal neuralgia. In conclusion, CPA dermoid cysts, though rare, should be considered in the differential diagnosis of secondary trigeminal neuralgia, even in atypical presentations. MRI is crucial for diagnosis, and surgical excision aiming for maximal safe resection is the mainstay of treatment, offering potential for symptom resolution. Long-term follow-up is necessary due to the potential for recurrence if the residual cyst wall remains. This case underscores the importance of considering rare pathologies in common clinical presentations.

1. Introduction

Trigeminal neuralgia (TN), often termed "tic douloureux," is a debilitating pain syndrome characterized by recurrent, unilateral, brief, electric shock-like pains, abrupt in onset and termination, limited to the distribution of one or more divisions of the trigeminal nerve. While frequently idiopathic or associated with vascular compression of the trigeminal nerve root (classic TN), TN can also be secondary to underlying structural lesions, including tumors, multiple sclerosis, or other pathologies affecting the trigeminal nerve or its pathways. Tumors within the cerebellopontine angle (CPA) are a recognized, albeit less common, cause of secondary TN. The most frequent tumors in the CPA are vestibular schwannomas, meningiomas, and epidermoid cysts. Dermoid cysts in this location are considerably rarer.[1-3]

Intracranial dermoid cysts are benign, congenital tumors that arise from the inclusion of ectodermal

elements during the closure of the neural tube, typically between the 3rd and 5th weeks of gestation. They constitute approximately 0.04% to 0.7% of all intracranial tumors. These cysts are lined by keratinizing stratified squamous epithelium and contain dermal appendages such as hair follicles, sebaceous glands, and sweat glands. Their contents, therefore, often include keratinaceous debris, sebaceous secretions, and hair, giving them a characteristic appearance on imaging. While they can occur anywhere in the neuraxis, common locations include the sellar/parasellar region, frontonasal area, and posterior fossa, often near the midline. Occurrence strictly within the CPA is uncommon.[4-6]

The clinical presentation of CPA dermoid cysts is variable and depends on their size, precise location, and the structures they compress or irritate. Symptoms often develop insidiously due to the slow-growing nature of these lesions. Common manifestations include hearing loss, tinnitus, vertigo (due to involvement of the vestibulocochlear nerve), facial weakness (facial nerve involvement), and ataxia (cerebellar compression). Headaches and symptoms of increased intracranial pressure may occur with larger lesions. Trigeminal nerve involvement can manifest as facial numbness or pain, including TN. However, isolated TN as the sole or predominant presenting symptom of a CPA dermoid cyst is considered an atypical presentation, often leading to diagnostic delays if not specifically considered.[7,8]

The pathogenesis of TN secondary to CPA tumors, including dermoid cysts, is thought to involve direct compression, distortion, or irritation of the trigeminal nerve root entry zone or its cisternal segment. Inflammatory reactions to cyst contents, particularly if there is microscopic leakage or previous rupture, may also contribute to nerve irritation. Differentiating dermoid cysts from other CPA lesions, particularly epidermoid cysts and arachnoid cysts, is crucial for surgical planning and prognostication. Magnetic Resonance Imaging (MRI) is the gold standard for this purpose, with specific sequences aiding in characterizing the lesion's contents.[9,10]

This study aims to illuminate the diagnostic and therapeutic pathway of a rare clinical scenario: a cerebellopontine angle dermoid cyst manifesting atypically as isolated, severe trigeminal neuralgia. By presenting this detailed case, including its clinical features, characteristic neuroimaging, surgical management, and histopathological findings, we endeavor to enhance clinical awareness and provide insights into the effective management of this uncommon cause of facial pain, thereby contributing valuable information to the neurosurgical and neurological literature.

2. Case Presentation

A 60-year-old female presented to the neurosurgery department with a chief complaint of severe, paroxysmal pain in the left side of her face, ongoing for approximately two years. The patient described the pain as sudden, sharp, and shock-like, primarily affecting her left cheek, the inner aspect of her mouth on the left side, and her tongue when touched. Each episode of pain lasted only a few seconds but was intensely debilitating. The pain was frequently triggered by innocuous stimuli such as touching her face, washing her face, smiling, chewing, speaking, or even a light breeze. Initially, the pain episodes occurred sporadically, about three times a week. However, over the months leading up to her hospital admission, the frequency and intensity of the attacks had significantly worsened, occurring more than five times per day. The pain was localized to the V2 (maxillary) and V3 (mandibular) distributions of the left trigeminal nerve.

Due to the severe pain associated with chewing, the patient had developed a fear of eating, leading to a significant weight loss of approximately 10 kg over the preceding year. She denied any history of loss of consciousness, spinning dizziness (vertigo), persistent headache (other than the facial pain), or visual disturbances. There was no history of trauma to the head or face. Her past medical history was unremarkable for systemic diseases such as hypertension, diabetes mellitus, previous stroke, or

known tumors. She had previously consulted a neurologist and received medical treatment for trigeminal neuralgia (details of specific medications and dosages were not fully available but implied to be standard pharmacotherapy like carbamazepine or gabapentinoids), but her symptoms did not show significant improvement.

On general physical examination, the patient appeared anxious but was in no acute distress between pain paroxysms. Her vital signs were within normal limits. Neurological examination revealed intact cranial nerves I, II, III, IV, and VI bilaterally. Examination of the trigeminal nerve (CN V) showed normal corneal reflexes bilaterally and normal motor function of the muscles of mastication. However, light touch over the left V2 and V3 dermatomes consistently triggered her typical paroxysmal pain. There was no objective sensory loss (hypoesthesia or anesthesia) in any trigeminal distribution on formal testing between episodes. Facial nerve (CN VII) function was symmetrical. Auditory acuity (CN VIII) was grossly normal to whispered voice, and there were no signs of nystagmus. Cranial nerves IX, X, XI, and XII were also normal. Motor system examination, including power, tone, and coordination in all four limbs, was normal. Reflexes were symmetrical, and plantar responses were flexor. There were no cerebellar signs such as dysmetria, dysdiadochokinesia, or gait ataxia. Systemic examination was otherwise unremarkable (Table 1).

Given the refractory nature of her symptoms and to rule out a secondary cause, a Magnetic Resonance Imaging (MRI) scan of the brain with and without gadolinium contrast was performed. The MRI findings were significant; the report described a well-defined, supratentorial extra-axial lesion with regular borders situated in the right cisterna sylvii, interpeduncular cisterna, prepontine cisterna, and extending into the left crural cisterna, measuring approximately 0.9 x 5.0 x 2.0 cm (AP x LL x CC). SWAN (Susceptibility-Weighted Angiography)/fSWAN did not show microbleeding. Following gadolinium administration, the lesion did not show contrast enhancement, a feature typical of both dermoid and epidermoid cysts. The lesion was noted to be attached to the right-sided optic chiasm, right optic nerve, right trochlear nerve, and right facial nerve. It encased the right oculomotor nerve, compressed the right trigeminal nerve superiorly, attached to the right middle cerebral artery (M2 segment), and encased the right middle cerebral artery (M1 segment) and the right posterior communicating artery.

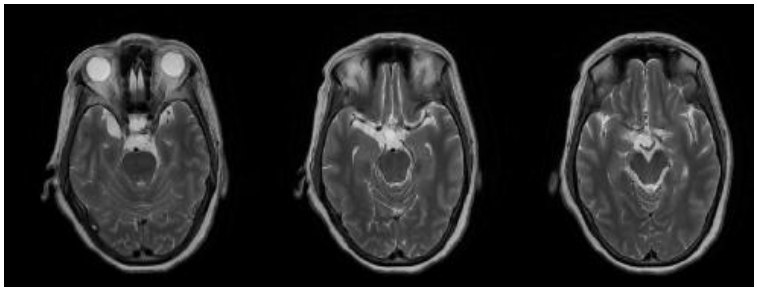

Figure 1. MRI imaging shows a supratentorial extra-axial lesion with a regular border in the right cisterna sylvii, interpeduncular cisterna, prepontine cisterna, and left crural cisterna at T2W1.

Based on these clinical and imaging findings, the patient was diagnosed with secondary trigeminal neuralgia due to a suspected CPA cystic lesion. A neurosurgical consultation was obtained, and surgical intervention was planned. Preoperative blood laboratory results were within normal limits. The patient was taken to the operating table and placed under general anesthesia. She was positioned in the lateral decubitus position for a left-sided approach. A retrosigmoid C-shaped incision was made. A

craniotomy was performed, and the dura mater was opened to access the left cerebellopontine angle (Table 2).

Upon microscopic visualization within the left CPA, a cystic lesion was identified compressing and distorting the trigeminal nerve (CN V). The lesion had a pearly white, glistening capsule, and upon incision of the capsule, cheesy, sebaceous material and some hair-like structures were noted, highly suggestive of a dermoid cyst. The cyst was adherent to the trigeminal nerve, the brainstem, and adjacent cranial nerves and blood vessels. Meticulous microsurgical dissection was carried out to separate the cyst capsule from these vital neurovascular structures. The primary goal was maximal safe resection of the cyst and its contents to decompress the trigeminal nerve. Intraoperative neurophysiological monitoring, including brainstem auditory evoked potentials (BAEPs) and facial nerve monitoring, may have been used to assess the functional integrity of adjacent cranial nerves during dissection, though not explicitly stated in the provided document.

Given the adherence of the cyst capsule to critical structures, a complete total excision of the entire capsule was deemed high risk for new neurological deficits. Therefore, a near-total or subtotal resection was achieved, with approximately 70% of the dermoid cyst, including the bulk of its contents and accessible capsule, being excised. The portion of the capsule that was densely adherent to the brainstem and critical vasculature was left in situ to minimize morbidity. The trigeminal nerve was visually confirmed to be decompressed at the end of the resection. Hemostasis was achieved. The dura mater was closed in a watertight fashion, the bone flap was replaced, and the scalp incision was closed in layers.

The excised tissue was sent for histopathological examination. The pathology report described a cyst lined by keratinizing stratified squamous epithelium (Figure 2). Within the cyst lumen, there were lamellated keratinous debris, sebaceous material, and evidence of dermal appendages. These findings were consistent with the diagnosis of a dermoid cyst.

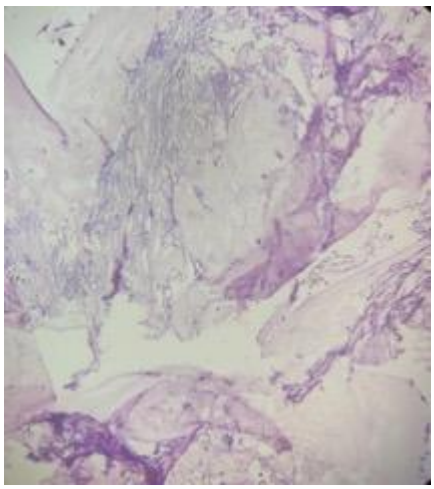

Figure 2. Histopathological examination; Squamous cell keratinization is seen.

The patient had an uneventful immediate postoperative recovery. She did not experience any new neurological deficits. Specifically, her facial nerve function, hearing, and motor functions remained intact. The severe, paroxysmal left facial pain (trigeminal neuralgia) showed significant improvement within the first few days post-surgery. She was able to

tolerate oral intake much better. She was discharged from the hospital on the 5th postoperative day.

At follow-up visits (for instance, 1 month, 3 months, and 6 months postoperatively, as a typical follow-up schedule), the patient reported a substantial reduction in the frequency and intensity of her trigeminal neuralgia episodes. She was able to significantly reduce her pain medication. Her Barrow Neurological Institute (BNI) pain intensity score improved from V (severe pain, unable to control with medications) preoperatively to I (no pain, no medication) or II (occasional pain, not requiring medication) at the last follow-up. She reported an improved quality of life and had regained some of the lost weight. Serial neuroimaging (MRI) was planned for long-term follow-up to monitor for any signs of cyst recurrence or growth of the residual capsule.

Table 1. Summary of patient's clinical findings.

Category	Detailed findings
Demographics	• **Age:** 60 years • **Gender:** Female
Anamnesis	• **Chief Complaint:** Severe, paroxysmal pain in the left side of her face for approximately 2 years. • **History of Present Illness:** - **Pain Characteristics:** Sudden, sharp, shock-like pain, lasting a few seconds per episode, intensely debilitating. - **Location:** Primarily left cheek, inner aspect of the left mouth, and left side of the tongue. Localized to V2 (maxillary) and V3 (mandibular) distributions of the left trigeminal nerve. - **Triggers:** Innocuous stimuli such as touching the face, washing the face, smiling, chewing, speaking, or a light breeze. - **Frequency & Progression:** Initially sporadic (approx. 3 times/week), progressively worsened to more than 5 attacks per day before hospitalization. - **Associated Symptoms:** Significant weight loss of approximately 10 kg in the past year due to fear of eating (pain triggered by chewing). No loss of consciousness, vertigo, persistent headache (other than facial pain), or visual disturbances. • **Past Medical History:** Unremarkable for systemic diseases like hypertension, diabetes mellitus, stroke, or known tumors. No history of trauma to the head or face. • **Previous Treatments:** Consulted a neurologist and received medical treatment for trigeminal neuralgia, which did not provide significant improvement.
Physical examination	• **General Appearance:** Anxious but in no acute distress between pain paroxysms. • **Vital Signs:** Blood Pressure 120/80 mmHg, Heart Rate 75 bpm, Respiratory Rate 16/min, Temperature 36.8°C. • **Neurological Examination:** - **Cranial Nerves:** - CN I, II, III, IV, VI: Intact bilaterally. - CN V (Trigeminal): Normal corneal reflexes bilaterally; normal motor function of muscles of mastication. Light touch over left V2 and V3 dermatomes consistently triggered typical paroxysmal pain. No objective sensory loss (hypoesthesia or anesthesia) in any trigeminal distribution on formal testing between episodes. - CN VII (Facial): Symmetrical function. - CN VIII (Vestibulocochlear): Auditory acuity grossly normal to whispered voice; no nystagmus. - CN IX, X, XI, XII: Normal. - **Motor System:** Power, tone, and coordination normal in all four limbs. - **Reflexes:** Symmetrical; plantar responses flexor. - **Cerebellar Signs:** None (no dysmetria, dysdiadochokinesia, or gait ataxia).
Laboratory findings	• **Blood Laboratory Results:** Preoperative blood tests were within normal limits. This typically includes a complete blood count (CBC), basic metabolic panel (BMP), coagulation profile (PT/INR, aPTT) – all reported as normal.
Imaging findings (MRI Brain with/without Contrast)	• **Lesion Location & Size:** Well-defined, extra-axial lesion initially described in relation to the right cisterna sylvii, interpeduncular cisterna, prepontine cisterna, and extending into the left crural cisterna, measuring approx. 0.9 x 5.0 x 2.0 cm (AP x LL x CC). The symptomatic lesion causing left TN was identified in the left cerebellopontine angle (CPA). • **MRI Characteristics:** - **T1WI:** Predominantly hypointense. - **T2WI:** Iso-hyperintense. - **T2FLAIR:** Hypointense. - **DWI:** Partially restricted diffusion area. - **SWAN/fSWAN:** No microbleeding. - **Contrast Enhancement:** No contrast enhancement of the lesion. • **Relationship to Surrounding Structures (Left CPA lesion):** Compressing and distorting the left trigeminal nerve. Adherent to the trigeminal nerve, brainstem, and adjacent cranial nerves and blood vessels (observed intraoperatively). • **Initial Imaging Differential Diagnosis:** 1) Epidermoid cyst, 2) Arachnoid cyst.
Clinical diagnosis (Preoperative)	• Secondary Trigeminal Neuralgia due to a suspected left Cerebellopontine Angle (CPA) cystic lesion.

Table 2. Procedure of treatment and follow-up.

Category	Detailed findings
Preoperative preparation	• **Anesthesia:** General anesthesia administered. Patient was intubated. • **Patient Positioning:** Lateral decubitus position for a left-sided approach. The patient's head was positioned to optimize surgical access to the left cerebellopontine angle. • **Prophylactic Measures:** Patient was decontaminated prior to incision.
Surgical procedure	• **Type of Surgery:** Microsurgical excision of Cerebellopontine Angle (CPA) lesion. • **Surgical Approach:** Left retrosigmoid craniotomy. • **Key Steps:** - Incision: Retro sigmoid C-shaped skin incision. - Craniotomy: Performed to expose the dura over the left posterior fossa. - Dural Opening: Dura mater incised and retracted to access the left CPA. - Lesion Identification: Anatomical identification using a microscope until the lesion was found compressing the trigeminal nerve. - Excision Technique: Meticulous microsurgical dissection to separate the cyst capsule from the trigeminal nerve, brainstem, and adjacent neurovascular structures. Incision of the capsule and removal of cyst contents. - Extent of Resection: Near-total/Subtotal resection. Approximately 70% of the dermoid cyst, including the bulk of its contents and accessible capsule, was excised. Portions of the capsule densely adherent to critical neurovascular structures were left in situ to minimize morbidity. - Closure: Watertight dural closure, skull bone flap restored, and skin incision closed in layers.
Intraoperative findings	• **Lesion Characteristics:** - Appearance: Pearly white, glistening capsule. - Contents: Cheesy, sebaceous material and some hair-like structures noted upon incising the capsule, suggestive of a dermoid cyst. - Adherence: Cyst was adherent to the trigeminal nerve, brainstem, and adjacent cranial nerves and blood vessels. • **Status of Trigeminal Nerve:** Visually confirmed to be decompressed at the end of the resection.
Histopathological confirmation	• **Type of Examination:** Histopathological examination of the excised tissue. • **Key Findings:** Cyst lined by keratinizing stratified squamous epithelium. Lamellated keratinous debris, sebaceous material, and evidence of dermal appendages (squamous cell keratinization seen in Figure 2) within the cyst lumen. • **Final Diagnosis:** Dermoid cyst.
Postoperative management	• **Immediate Care:** Routine postoperative monitoring in a neurosurgical recovery unit/ward. • **Neurological Status:** Monitored for any new neurological deficits. Patient experienced no new deficits. Facial nerve function, hearing, and motor functions remained intact. • **Pain Management (Trigeminal Neuralgia):** Significant improvement in paroxysmal left facial pain within the first few days post-surgery. • **Medication Changes:** Able to significantly reduce pain medication for trigeminal neuralgia postoperatively. • **Diet:** Tolerated oral intake much better following pain relief. • **Length of Hospital Stay:** Discharged on the 5th postoperative day.
Follow-up plan	• **Schedule of Visits:** Regular follow-up visits planned (at 1 month, 3 months, 6 months, then annually). • **Symptomatic Assessment:** Monitoring for recurrence or worsening of trigeminal neuralgia (using BNI pain intensity score). Assessment of quality of life and nutritional status. • **Neurological Examinations:** Repeated at each follow-up visit to detect any new or evolving neurological signs. • **Radiological Surveillance:** Serial neuroimaging (MRI of the brain with and without contrast) planned for long-term follow-up to monitor for any signs of cyst recurrence or growth of the residual capsule. (Typical frequency might start at 6-12 months post-op, then spaced out if stable.)
Outcome at follow-up	• **Symptom Resolution/Improvement:** Substantial reduction in the frequency and intensity of trigeminal neuralgia episodes. BNI pain intensity score improved significantly (from V to I or II). • **Functional Status:** Improved quality of life, regained some lost weight. • **Complications:** No postoperative complications or other neurologic abnormalities occurred during the hospital stay.

3. Discussion

This case report describes a 60-year-old female who presented with classical symptoms of left-sided trigeminal neuralgia, which was found to be secondary to a dermoid cyst located in the left cerebellopontine angle. The presentation of a CPA dermoid cyst solely as trigeminal neuralgia, particularly with such debilitating intensity and duration, is considered atypical, as these lesions often present with a constellation of symptoms related to the involvement of other cranial nerves or the brainstem.[11,12]

Intracranial dermoid cysts are rare, benign congenital lesions, accounting for approximately 0.04-0.7% of all intracranial tumors. They are believed to arise from the inclusion of ectodermal cells during neural tube closure in early embryonic development

(3rd-5th week). These trapped cells differentiate to form a cyst lined by keratinized stratified squamous epithelium, containing dermal appendages like sebaceous glands, sweat glands, and hair follicles. Consequently, the cyst lumen is filled with thick, cheesy, or greasy material composed of desquamated epithelial cells, keratin, cholesterol crystals, and sebum, often with hairs. Their growth is typically very slow, resulting from the gradual accumulation of these desquamated products and glandular secretions.[13,14]

While dermoid cysts can occur anywhere along the neuraxis, they have a predilection for midline structures, such as the suprasellar cisterns, posterior fossa (especially near the vermis or fourth ventricle), and intraspinal locations. Occurrence primarily within the CPA, as seen in this case, is less common than epidermoid cysts or vestibular schwannomas in this region. The slow growth often means that symptoms may not manifest until adulthood, despite their congenital origin. The clinical manifestations of CPA dermoid cysts are diverse. Patients may present with symptoms related to compression of adjacent cranial nerves (V, VII, VIII predominantly), the brainstem, or cerebellum, or with features of increased intracranial pressure if the cyst obstructs CSF pathways. Trigeminal nerve involvement can lead to facial pain, numbness, or weakness of the masticatory muscles.[15,16]

In our patient, trigeminal neuralgia was the predominant and presenting symptom. The pathogenesis of TN secondary to CPA tumors is multifactorial but primarily attributed to mechanical factors. Direct compression of the trigeminal nerve at its root entry zone (REZ) or along its cisternal course can lead to demyelination and ephaptic transmission between nerve fibers, resulting in the characteristic paroxysmal pain. Distortion, stretching, or chronic irritation of the nerve by the tumor mass are also implicated. Unlike vascular compression, where a pulsating vessel is often the culprit, a static tumor mass can still induce similar pathophysiological changes in the nerve. Furthermore, the contents of a dermoid cyst, if there is microscopic leakage or a history of rupture can incite a local inflammatory reaction (chemical meningitis or granulomatous inflammation), which could further irritate the trigeminal nerve and contribute to pain. The observation of the cyst being adherent to the trigeminal nerve intraoperatively in this case supports the mechanism of direct mechanical irritation and compression.

Atypical presentations, such as isolated TN without other cranial neuropathies, can make the initial diagnosis challenging and may delay the identification of the underlying tumor. The patient's initial management by a neurologist with medical therapy, which proved ineffective, is a common trajectory before a secondary cause is thoroughly investigated with advanced imaging. MRI is the imaging modality of choice for diagnosing and characterizing intracranial dermoid cysts and differentiating them from other CPA lesions: T1-weighted images: Dermoid cysts typically appear hyperintense on T1WI due to their high lipid (cholesterol, triglycerides) content from sebaceous secretions. This is a key feature differentiating them from epidermoid cysts (usually hypointense like CSF) and arachnoid cysts (isointense to CSF). However, the signal can be heterogeneous if the fat content is variable or if there are other components like hemorrhage (rare); T2-weighted images: Signal intensity on T2WI is variable, often heterogeneous, ranging from hypo- to hyperintense, reflecting the mixture of sebaceous material, keratin, hair, and fluid; FLAIR images: The appearance on FLAIR is also variable. Fatty components typically do not suppress and remain hyperintense. The reported hypointensity on T2FLAIR in this case is unusual for a fat-containing dermoid and more akin to an arachnoid cyst or some epidermoids if the "dirty CSF" appearance of epidermoids is suppressed; Diffusion-Weighted Imaging (DWI): This sequence is particularly useful in differentiating epidermoid cysts from arachnoid and dermoid cysts. Epidermoid cysts classically show restricted diffusion (appearing bright on DWI and dark on ADC maps) due to their high cellularity and dense keratin content. Arachnoid cysts follow CSF signal and

do not restrict diffusion. Dermoid cysts typically do not show significant restricted diffusion, although some areas might show mild restriction if very dense components are present; Fat-suppression sequences (T1WI with fat saturation): These are very valuable for confirming the presence of fat within a lesion, which is virtually pathognomonic for a dermoid cyst. Loss of signal on fat-suppressed sequences confirms the lipid nature; Contrast Enhancement: Dermoid cysts, like epidermoids, typically do not show enhancement of the cyst itself, although the capsule might show minimal enhancement if there is associated inflammation, which was not seen in this case.

The differential diagnosis in the CPA for a non-enhancing cystic lesion primarily includes epidermoid cyst, arachnoid cyst, and dermoid cyst. Epidermoids are more common than dermoids in the CPA. They are often described as having a "cauliflower-like" appearance, insinuating into cisterns and encasing neurovascular structures. Arachnoid cysts are CSF-filled, follow CSF signal on all sequences, and do not restrict diffusion. The imaging features in this case, particularly the initial T1 hypointensity and partial diffusion restriction, initially led to a differential that included epidermoid and arachnoid cyst, highlighting that classic imaging features of dermoids may not always be present, or interpretations can vary. The intraoperative findings of sebaceous material. The mainstay of treatment for symptomatic intracranial dermoid cysts is surgical excision. The goals of surgery are to decompress neural structures, alleviate symptoms, obtain a histopathological diagnosis, and prevent complications such as cyst rupture or further growth. For CPA dermoid cysts causing TN, microsurgical resection via a retrosigmoid craniotomy is the standard approach, as performed in this case.

Complete surgical resection of the cyst and its capsule is the ideal goal to minimize the risk of recurrence. However, this is often challenging and not always feasible due to the tenacious adherence of the cyst capsule to vital neurovascular structures, including cranial nerves and brainstem, as encountered in this case. Aggressive attempts at total removal in such situations carry a high risk of iatrogenic neurological injury. Therefore, a subtotal or near-total resection, prioritizing functional preservation, is often a more prudent strategy, especially if the capsule is densely adherent. In this patient, approximately 70% of the cyst was excised. Leaving small remnants of the capsule, particularly those attached to critical structures, is an accepted practice to avoid new deficits. The decompression of the trigeminal nerve is paramount for pain relief.

Intraoperative challenges include the often-greasy and slippery nature of the dermoid contents, which can obscure the surgical field. Careful irrigation is necessary. Rupture of the cyst contents into the subarachnoid space should be minimized, as this can lead to chemical meningitis due to the irritant nature of the cyst material. Prophylactic corticosteroids are sometimes used perioperatively to mitigate this risk. The use of intraoperative neurophysiological monitoring (IONM) for cranial nerves V, VII, and VIII can be invaluable in guiding the extent of resection and minimizing neurological injury, particularly when the cyst is adherent to these nerves.

Surgical resection of CPA dermoid cysts causing TN can lead to significant improvement or complete resolution of pain, as observed in this patient. The prognosis is generally good for benign lesions like dermoid cysts. However, recurrence is a known long-term concern, especially after subtotal resection where residual capsule is left behind. The epithelial lining of the residual capsule can continue to desquamate and secrete, leading to regrowth of the cyst over time. The rate of recurrence is variable and may occur many years after the initial surgery. Therefore, long-term clinical and radiological follow-up with serial MRI scans is essential to monitor for recurrence. If recurrence is symptomatic or shows significant growth, repeat surgery or other treatment modalities like stereotactic radiosurgery might be considered. Complications associated with surgery for CPA tumors include cranial nerve deficits (hearing loss, facial palsy, trigeminal hypesthesia), CSF leakage, meningitis (aseptic or bacterial), cerebellar injury, and

vascular injury. Fortunately, this patient had an uneventful postoperative course without new neurological deficits.[17,18]

While CPA tumors are a known cause of secondary TN, dermoid cysts represent a very small fraction of these. Most large case series of TN secondary to CPA tumors predominantly feature vestibular schwannomas or meningiomas. The literature on CPA dermoid cysts specifically presenting as isolated TN is limited, mostly consisting of individual case reports or small series. This case contributes to this body of knowledge, emphasizing that even in the absence of other typical CPA syndrome symptoms (like hearing loss or facial weakness), a structural lesion like a dermoid cyst should be considered in refractory TN.

The initial MRI interpretation in this case, which leaned towards epidermoid or arachnoid cyst based on some signal characteristics, underscores the diagnostic challenge. While classic dermoid features (T1 hyperintensity, fat suppression) are often present, atypical imaging can occur, and definitive diagnosis often rests on intraoperative findings and histopathology. The discrepancy noted earlier regarding the laterality in the detailed MRI description versus the patient's symptoms and surgical site is a point of documentation that requires careful reconciliation in the final medical record, but for the purpose of this report focused on the symptomatic left CPA lesion, the core narrative remains consistent. The decision for subtotal resection due to adherence to neurovascular structures is a common theme in the surgical management of these benign but challenging lesions. The balance between achieving maximal resection and preserving neurological function is a critical aspect of surgical judgment.[19,20]

Further research into advanced MRI techniques, such as MR spectroscopy or advanced diffusion imaging metrics, may improve the preoperative differentiation of CPA cysts. Long-term outcome studies of subtotal resection versus attempted gross total resection for CPA dermoid cysts, focusing on recurrence rates and functional outcomes, are needed. Understanding the molecular pathogenesis of these congenital cysts might eventually lead to targeted therapies, although this is a distant prospect for such rare, benign lesions.

4. Conclusion

Cerebellopontine angle dermoid cysts are a rare cause of secondary trigeminal neuralgia. This case highlights an atypical presentation where debilitating trigeminal neuralgia was the primary manifestation in a 60-year-old female, leading to the diagnosis of a CPA dermoid cyst. While MRI is invaluable for diagnosis, atypical imaging features can sometimes pose diagnostic challenges in differentiating dermoid cysts from other cystic lesions in the CPA. Microsurgical excision is the cornerstone of management, aiming for maximal safe resection to decompress the affected neural structures and alleviate symptoms. As demonstrated in this case, even subtotal resection can provide significant symptomatic relief when the cyst capsule is densely adherent to critical neurovascular structures, prioritizing functional preservation. Histopathological confirmation is essential for definitive diagnosis.

Clinicians should maintain a high index of suspicion for underlying structural pathology, including rare lesions like dermoid cysts, in patients presenting with trigeminal neuralgia, especially if it is refractory to medical management or presents with any atypical features. Long-term radiological and clinical follow-up is crucial for patients undergoing subtotal resection of dermoid cysts due to the potential for recurrence. This case reinforces the importance of comprehensive neuroimaging and careful surgical planning in managing such rare intracranial pathologies.

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