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Case Report

A rare case of giant infrasellar craniopharyngioma with extensive invasion of the pterygopalatine fossa: A case report and literature review

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ABSTRACT

Background: Craniopharyngiomas are benign epithelial tumors that arise along the craniopharyngeal duct, commonly located in the sellar or suprasellar region. Infrasellar extension is a rare variant and may involve the nasopharynx, sphenoid sinus, clivus, and pterygopalatine fossa.

Case Description: A 66-year-old male patient is presented to the otorhinolaryngology service due to a complaint of left ear obstruction for the past 4 months. After no response to clinical treatment, investigation with computed tomography and magnetic resonance imaging showed a heterogeneous lesion with areas of calcification and bone destruction located in the sphenoid sinus region, which projected inferiorly and laterally invading the clivus in its entirety, the petrous apex, middle fossa, pterygopalatine, and infratemporal fossae with no involvement of the sellar/suprasellar region. The patient was referred to a multidisciplinary skull base surgery group that performed an extended transpterygoid endoscopic endonasal approach with gross total resection. The anatomopathological study was consistent with adamantinomatous craniopharyngioma.

Conclusion: We present a rare case of a giant infrasellar craniopharyngioma with extensive invasion of the skull base without involvement of the sella or the pituitary gland.

Keywords: Case report, Craniopharyngioma, Literature review, Skull base neoplasms, Sphenoid sinus

INTRODUCTION

Craniopharyngiomas are benign epithelial tumors that arise along the craniopharyngeal duct.[20,25,33] Despite being classified as benign, their local aggressiveness can cause significant morbidity.[15,33] Craniopharyngiomas have a very low incidence rate, approximately 0.13/100,000 people/year.[20] Despite their rarity, they account for 2–5% of intracranial neoplasms in adults, and children, their incidence rises to 5.6–15%, making them the most common neoplasm in the hypothalamic-pituitary region in childhood.[15,20,22,25] This tumor presents a bimodal age distribution, with the first peak occurring from 5 to 14 years old and the second from 50 to 74 years old.[20,22,25]

Craniopharyngiomas usually grow along the vertical axis from the sella to the third ventricle.[27] From these areas, they may expand into the anterior (2–5%), middle (2%), or posterior (1–4%) cranial fossa. Infrasellar extension is a rare variant found in only about 5% of cases, which may involve the nasopharynx, sphenoid sinus, clivus, and ethmoids.[15,25,32] In addition, medical literature records cases of craniopharyngiomas arising in locations such as the third ventricle, pineal gland, Sylvian fissure, and cerebellopontine angle.[15] The most common location of infrasellar craniopharyngiomas is the sphenoid sinus, either alone or combined with other sites such as the nasopharynx, sella turcica, suprasellar, ethmoid sinuses, or maxillary sinus.[33]

Isolated infrasellar craniopharyngiomas without sellar and pituitary involvement are exceedingly rare. In this case report and literature review, we will scrutinize an infrasellar craniopharyngioma, an uncommon incidence, discussing its clinical and diagnostic characteristics, as well as the treatment approaches.

CASE REPORT

A 66-year-old male patient with a medical history of diabetes mellitus using oral hypoglycemic agents, without other comorbidities or prior surgeries, sought the otorhinolaryngology (ORL) service due to the sensation of aural fullness on the left for approximately 4 months. In the initial investigation with the ORL team, during clinical examination, secretion was found in the left middle ear. After the lack of response to clinical treatment with oral antibiotics, ventilation tube implantation was performed, followed by an investigation with computed tomography (CT) of the skull and mastoid region.

The brain CT not only revealed signs of chronic involvement of the left mastoid but also depicted a heterogeneous lesion with areas of calcification and bone destruction located in the sphenoid sinus region. This lesion projected inferiorly and laterally, invading the clivus throughout its extent, the petrous apex on the left side, the middle fossa on the same side, and extending to the pterygopalatine and infratemporal fossae on the left [Figure 1]. Following the brain CT, the ORL team requested brain magnetic resonance imaging (MRI). The MRI demonstrated an increased volume of the lesion, which was already obstructing the region of the left auditory tube and bilateral nasopharynx; however, it did not reveal the involvement of the sellar/suprasellar region. The lesion exhibited isosignal on T1, hyposignal on T2, and on contrast-enhanced MRI, it presented with heterogeneous contrast enhancement [Figure 2]. During the month in which preparatory complementary examinations for surgery were conducted, the patient progressed to complete bilateral nasal obstruction.

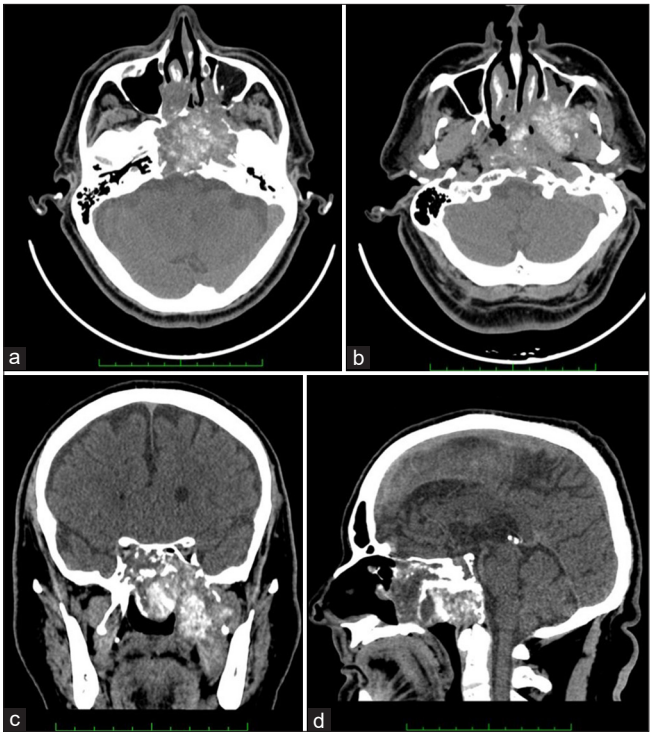

Figure 1: Preoperative computed brain tomography showing a heterogeneous lesion in the sphenoid region with signs of calcification. In (a), an axial image showing the invasion of the clivus, petrous apex in the temporal bone. In (b), another inferior image shows the extension to the pterygopalatine and infratemporal fossae on the left. In (c) (coronal) and (d) (sagittal) images, we can see the integrity of the sellar floor and all anterior skull bases.

After MRI, the patient was referred to our multidisciplinary skull base surgery group. Following clarification of risks and benefits, surgical treatment was indicated through an extended endoscopic endonasal approach, as this provided optimal access to the tumor epicenter without jeopardizing major vessels and nerves. Meticulous planning was undertaken to ensure safe tumor removal in proximity to the internal carotid artery, particularly at its paraclival segment, considering tumor extension through the middle cranial fossa superiorly and the infratemporal fossa inferiorly. Under general anesthesia, the patient was placed in a horizontal supine position, with the head secured using Mayfield three points, and neurophysiological monitoring of cranial nerves was performed. Extended endoscopic endonasal access was performed with a transmaxillary and transpterygoid approach, followed by harvesting of a vascularized nasoseptal flap from the right side. At this point, a sizable, bleeding lesion with coarse calcifications occupying the nasal fossa was observed. We performed a central debulking of the lesion, followed by an attempt to reach all its boundaries. Subsequently, the entire anterior portion of the sellar floor was exposed to completely resect the lesion. The petrous apex

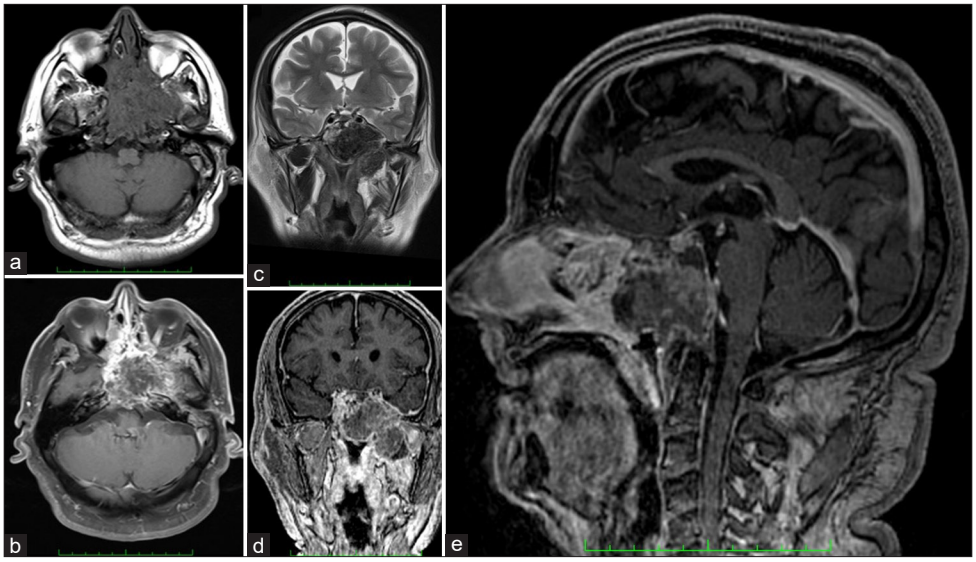

Figure 2: Preoperative magnetic resonance image showing in (a and b) axial T1 without and T1 with contrasted showing a heterogenous enhancement of contrast. In (c), a coronal T2 shows a hypo signal lesion. In (d) (coronal) and (e) (sagittal) contrasted T1, showing the extension of the lesion to pterygopalatine and infratemporal fossae without invasion of the sellar floor and anterior skull base.

was exposed without a contralateral transmaxillary corridor, allowing visualization of the genu of the petrous segment of the internal carotid artery and removal of the extradural lesion in the middle cranial fossa. Later, we addressed the pterygoid fossa region, achieving apparent complete resection. At the end of the procedure, the vascularized nasoseptal flap was positioned over the exposed left carotid, with no signs of cerebrospinal fluid leakage at any point.

The surgical pathology report revealed that it was an epithelioid neoplasm with calcifications, immunohistochemistry showed p63, cytokeratin (AE1/AE3) and Ki-67 (<1%), and grade 1 adamantinomatous craniopharyngioma as the final diagnosis. The postoperative MRI revealed gross total resection [Figure 3]. The patient showed favorable progress, with no significant clinical complications, remaining in the intensive care unit for only 1 day and being discharged from the hospital on the 5th day after the surgical procedure. Thirty days after surgery, the patient underwent intensity modulated radiotherapy (IMRT) and volumetric modulated arc therapy (VMAT) radiotherapy with a total dose of 5040 cGy, divided into 28 sessions. During the 12 months of outpatient follow-up, the patient progressed well with no signs of recurrence of the lesion.

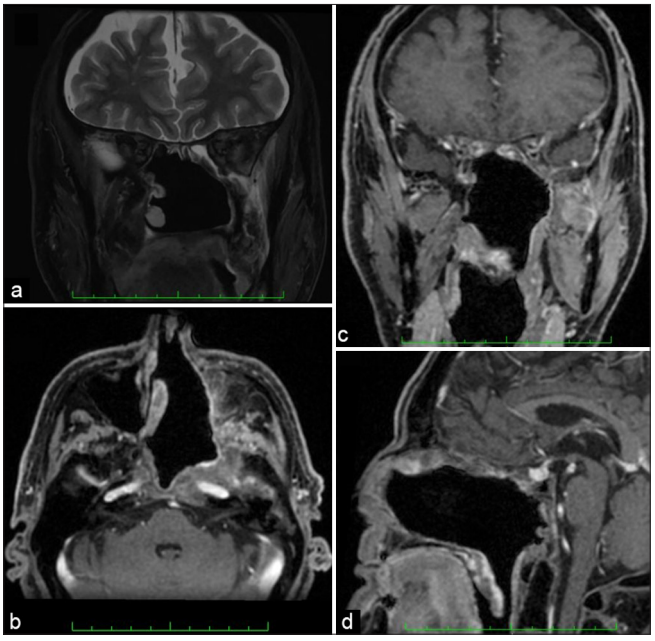

Figure 3: Postoperative MRI showing gross total resection with preservation of the anterior skull base in (a) T2-weightened image coronal view, (b) T1 axial, (c) coronal and (d) sagittal view.

DISCUSSION

Craniopharyngiomas are benign neoplasms characterized by the presence of cystic, solid, or mixed components, exhibiting irregular morphology and adherence to surrounding structures.[31] They can be classified into two main types: adamantinomatous and papillary, with the latter accounting for 10–30% of cases.[28] Adamantinomatous craniopharyngiomas can manifest at any age, although they are more prevalent in children and adolescents. In contrast, papillary craniopharyngiomas are predominantly observed in adults.[20,28,31] Approximately 80% of papillary craniopharyngiomas originate in the superior infundibular

portion of the pituitary stalk and the tuber cinereum, expanding into the third ventricle. On the other hand, adamantinomatous craniopharyngiomas most commonly arise from the solid portion of the infundibulum. Infrasellar craniopharyngiomas are predominantly of the adamantinomatous type.[28]

The pathogenesis of infrasellar craniopharyngiomas is primarily attributed to the persistence of remnants of the craniopharyngeal duct.[20,25,27,28,33] This transient structure, which connects the stomodeal ectoderm to Rathke's pouch, normally involutes during fetal development. However, incomplete regression may lead to neoplastic transformation of the remaining cells.[20,28] During embryogenesis, Rathke's pouch, formed from the primitive oral ectoderm, grows toward the diencephalon to form the adenohypophysis. However, cellular remnants may persist along the craniopharyngeal canal, which extends from the sella turcica to the vomer, giving rise to ectopic craniopharyngiomas along the midline.[25,27] In addition, epithelial cells displaced from the embryonic oral cavity or Rathke's pouch, deposited beneath the sella before the involution of the craniopharyngeal duct, may contribute to the formation of these tumors.[25] Erdheim (1904) suggested that craniopharyngiomas could arise at any point along this pathway, including the vomer, the roof of the nasopharynx, and the sphenoid bone, supporting the hypothesis of aberrant migration or embryonic cell entrapment.[14,28,33]

The clinical findings of these lesions are closely related to their size and location, as well as the degree of compression of surrounding structures.[33] The symptomatology of suprasellar tumors is often characterized by defects in the visual field, pituitary deficits, and/or features of increased intracranial pressure.[31,33] On the other hand, infrasellar craniopharyngiomas clinically present with symptoms such as frontal headache, nasal obstruction, epistaxis, cavernous sinus syndrome, and nasopharyngeal and/or nasal fossa masses.[15,21,31,33] It is important to emphasize that signs of pituitary dysfunction are not commonly observed in infrasellar craniopharyngiomas.[15] The present case manifested with progressive obstruction of the left auditory tube and progressed to complete bilateral nasal obstruction without pituitary insufficiency, suggesting that the tumor originated in the sphenoid sinus, with subsequent extensive invasion of the skull base.

Infrasellar craniopharyngiomas share radiological characteristics with intracranial craniopharyngiomas.[13] The diagnosis of these neoplasms can be established through imaging studies such as radiography, CT, magnetic resonance imaging, and cerebral angiography. According to Pusey *et al*., CT is particularly indicated for assessing bone anatomy and detecting calcifications, providing specificity in diagnosing craniopharyngiomas. On the other hand, MRI, especially when employed with contrast, is valuable for analyzing tumor structure, demonstrating greater sensitivity than CT in identifying the extent of the tumor, and is therefore preferable in presurgical and radiotherapeutic planning.[3,29] It is noteworthy that the specific determination of infrasellar craniopharyngiomas requires endoscopic biopsies.[13] The precise identification of lesions in the infrasellar region is a diagnostic challenge that demands a comprehensive and specialized approach involving different professionals and tools to differentiate between a variety of intracranial and extracranial conditions, with the crucial role of otolaryngologists in distinguishing among these conditions. The differential diagnosis includes encephaloceles, mucoceles, chordomas, squamous cell carcinomas, sarcomas, rhabdomyosarcoma, juvenile angiofibroma, and inverted papilloma.[13,16,25]

Craniopharyngiomas represent a formidable surgical challenge, even with advancements in contemporary neurosurgical techniques.[20] The fundamental approach to these tumors involves either complete or partial surgical resection, a principle particularly applicable to extracranial variants that lack close association with the optic apparatus or hypothalamus.[1,33] The choice of surgical approach should ensure wide exposure of all parts of the tumor while minimizing damage to surrounding vital structures. This decision is nuanced by factors such as the tumor's location, consistency, degree of calcification, shape, and size, along with the surgeon's preferences and expertise.[20] In the strategic planning of the surgery, critical considerations include evaluating the tumor's extension to the optic chiasm, pituitary, stalk, hypothalamus, carotid artery, and cerebral artery, skull base bone invasion, and relationship with cranial nerves.[5] At present, radiotherapy stands as the most prevalent adjuvant treatment, playing a pivotal role in forestalling recurrence and enhancing overall survival.[33] Even after total surgical removal, long-term recurrence of craniopharyngioma is a concern, with the extent of resection being a key factor in recurrence rates. Following complete removal, the 5-year recurrence rate is 13%, escalating to as high as 50% with incomplete excision.[25,26,33] In addition, lesions exceeding 5 cm in size, as well as the adamantinomatous subtype, portend a more unfavorable prognosis. Recurrence-free survival rates exhibit a range between 60% and 93% at the 10-year mark, while overall survival ranges from 64% to 96%.[22]

The endoscopic approach stands out for the significant advantage of providing extensive visualization of the tumor's origin site and the areas affected by the tumor. In comparison to the transcranial microscopic approach, the endoscopic approach holds the promise of a higher rate of total resection.[5] The use of angled endoscopes offers an expanded field of view, while the "four hands" technique allows the employment of additional instruments through the nasal cavities.

Table 1: List of reported cases of infrasellar craniopharyngioma.

Author	Year	Sex/Age	Symptoms	Histological type	Tumor location	Surgical approach	Degree of resection	Radiotherapy	Recurrence
Illum *et al.*[18]	1977	Female/14	Headache, visual impairment, diplopia	NA	NPX, SS, ST	Transpalatal	NA	Yes	Yes
Mukada *et al.*[24]	1984	Male/13	Visual impairment	NA	SS, NPX, C, ST, S, PC	Sublabial rhinoseptal transsphenoidal	Subtotal	NA	NA
Benitez *et al.*[6]	1988	Male/29	Nasal obstruction	NA	NPX, SS, ES, MS, C, CS, MCF, FR	Lateral rhinotomy and ethmoidectomy	Subtotal	Yes	NA
Akimura *et al.*[3]	1989	Female/12	Visual impairment	NA	SS, ES, CS, PPF, ITF	Transnasal	Subtotal	NA	NA
Byrne and Sessões.[8]	1990	Male/29	Nasal obstruction	Adamantinomatous	NF, NPX, SS, MS, ES, C	Lateral rhinotomy	Subtotal	Yes	No
Cheddadi *et al.*[10]	1996	Female/0	Nasal obstruction	NA	NPX	Endoscopic endonasal	Total	NA	No
Deutsch *et al.*[13]	2001	Male/8	Headache, visual impairment	NA	SS, ES	Subfrontal transbasal	Subtotal	Yes	No
Falavigna and Kraemer[15]	2001	Female/34	Headache, diplopia	Adamantinomatous	SS, ES, ST	Sublabial rhinoseptal transsphenoidal	Subtotal	Yes	No
Chen[11]	2001	Male/8	Visual impairment	Adamantinomatous	SS, S	Right frontotemporal craniotomy	Total	NA	No
Buhl *et al.*[7]	2001	Female/4	Visual impairment	NA	NPX, SS, ES, CS, S	Subfrontal	Subtotal	No	No
Kachhara *et al.*[19]	2002	Male/28	Headache, diplopia	Adamantinomatous	NPX, SS, ES, C, ST	Extended frontobasal	Total	NA	NA
Fujimoto *et al.*[16]	2002	Male/8	Headache, nasal obstruction, visual impairment, exophthalmos	Adamantinomatous	SS, ES, MS, ST, S	Endonasal transsphenoidal	Subtotal	Yes	Yes
Ahsan *et al.*[2]	2004	Male/59	Epistaxis	NA	NF, ES, MS	Denker's medial maxillectomy	Total	No	No
Rakheja *et al.*[30]	2005	Female/12	Headache, visual impairment	Adamantinomatous	SS, ES, ST, C	NA	NA	NA	NA
Koral *et al.*[21]	2006	Female/12	Headache, visual impairment	NA	SS	NA	NA	Yes	No
Shuman *et al.*[32]	2007	Male/8	Nasal obstruction, snoring	Adamantinomatous	SS, NPX, C, NF	Le Fort I osteotomy	Total	NA	NA
Arndt *et al.*[4]	2007	Female/16	Headache, nasal obstruction	Adamantinomatous	SS, ES, NF, C, ST, PP	Midfacial degloving	Total	NA	NA

(Contd...)

Table 1: *(Continued).*

Author	Year	Sex/Age	Symptoms	Histological type	Tumor location	Surgical approach	Degree of resection	Radiotherapy	Recurrence
Hwang *et al.*[17]	2009	Male/53	Nasal obstruction	Adamantinomatous	PPF, MS	Denker's medial maxillectomy Endoscopic	Subtotal	NA	No
Nourbakhsh *et al.*[25]	2010	Female/25	Headache	NA	SS, ES	Endoscopic transsphenoidal	Total	NA	NA
Magill *et al.*[23]	2011	Female/46	Headache, nasal obstruction, epistaxis	Adamantinomatous	NPX, SS, C	Midfacial degloving	Total	Yes	No
Chiun *et al.*[12]	2012	Male/6	Nasal obstruction	Adamantinomatous	NF, NS	NA	NA	NA	No
Kundu *et al.*[22]	2014	Male/55	Headache, nasal obstruction, epistaxis, hyposmia	Adamantinomatous	NPX, SS, NF	Midfacial degloving	Total	Yes	No
Senthilvel *et al.*[31]	2014	Male/68	Headache, epistaxis	Papillary	SS, NF, ES, C, ST	Endoscopic transnasal	Total	NA	NA
Zhang *et al.*[33]	2015	Female/16	Headache, nasal obstruction	Adamantinomatous	SS, C	Endoscopic endonasal transsphenoidal	Total	NA	No
Abou-Al-Shaar *et al.*[1]	2016	Male/22	Headache	Adamantinomatous	NPX, SS, C, NS	Expanded endoscopic endonasal	Total	No	No
Preti *et al.*[26]	2017	Male/17	Epistaxis	Adamantinomatous	ES, NF	Endoscopic endonasal	Total	NA	No
Caklili *et al.*[9]	2023	NA	NA	NA	NA	NA	NA	NA	NA

NPX: Nasopharynx, SS: Sphenoid sinus, ST: Sella turcica, S: Suprasellar, ES: Ethmoid sinuses, MS: Maxillary sinus, NF: Nasal fossa, NS: Nasal septum, PPF: Pterygopalatine fossa, C: Clivus, PP: Pterygoid process, CS: Cavernous sinus, PC: Prepontine cistern, ITF: Infratemporal fossa, MCF: Middle cranial fossa, FR: Foramen rotundum, NA: Not available

Furthermore, recent technological advancements, such as neuronavigation and intranasal Doppler, serve as precise tools for safely addressing the skull base. The most common postoperative complication observed following the extended endoscopic endonasal approach for craniopharyngiomas is cerebrospinal fluid leakage. Other complications associated with this technique include meningitis and hydrocephalus.[5] Thus, the endoscopic endonasal technique proves to be safe and less traumatic compared to external approaches, avoiding aesthetic deformities and enabling shorter hospitalization periods.[26]

In published studies, 27 cases of infrasellar craniopharyngiomas have been documented [Table 1]. Among these, 23 cases reported tumors without sellar or suprasellar invasion. One cohort was identified; Caklili *et al.*[9] evaluated the surgical outcomes and follow-up of 44 pediatric craniopharyngioma cases. Of these, only one case corresponded to an infrasellar craniopharyngioma, which was included in the present literature review [Table 1]. The most frequently observed symptom was headache, followed by nasal obstruction and visual impairment. Auditory obstruction was not mentioned in any of the analyzed cases. Regarding tumor location, most cases involved the sphenoid sinus region, followed by the ethmoid sinus, nasopharynx, and clivus. The pterygopalatine fossa was affected in only two cases: concerning the histological type, adamantinomatous tumors predominated, reinforcing their prevalence in this anatomical region. A wide variety of surgical approaches were observed, with emphasis on endoscopic techniques, such as transnasal and transsphenoidal approaches. Total resection was accomplished in most cases, especially in lesions with well-defined borders. Radiotherapy was mainly indicated in cases of subtotal resection. Tumor recurrence was recorded in only two cases.

CONCLUSION

We present a rare case of a giant infrasellar craniopharyngioma with extensive invasion of the skull base without involvement of sella turcica or the pituitary gland. Neuroimaging and surgical findings indicate the tumor's origin in the sphenoidal region with subsequent invasion of the clivus, petrous apex, middle fossa, pterygopalatine, and infratemporal fossae. Although classified as benign tumors, craniopharyngiomas represent significant challenges due to their local aggressiveness, resulting in considerable morbidity. Concerns about recurrence persist even after a good degree of resection, making long-term follow-up indispensable. The extended endoscopic endonasal approach stands out for providing excellent visualization of the involved anatomy, allowing for a safe and effective approach.

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