Chapter 7
Endoscopic Endonasal Surgery for Uncommon Pathologies of the Sellar and Parasellar Regions

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7.1 The Endoscope and Surgery for Sellar and Parasellar Lesions: A Perfect Match

The microscopic transsphenoidal approach has been the gold standard for pituitary tumor resection since the introduction of the surgical microscope into this type of surgery by Hardy in 1962 [1, 2].

After endoscopes became popularized in paranasal sinus surgery, interest increased in their usage for transsphenoidal pituitary surgery [3]. It was in 1992 when Jankowski et al. reported the first endoscopic endonasal resection of pituitary adenomas in three patients. The procedures were performed via mono- or bi-nostril techniques and included a routine middle turbinectomy [4].

In addition to its important advantage of obviating the need to transgress the critical neurovascular structures when approaching the majority of sellar lesions, further benefits of the endoscopic approach include panoramic visualization, improved illumination, angled views made available by angled lenses, and increased maneuverability of instruments [5]. The improved visualization entailed better identification of the anatomical structures at risk and enabled resection of adenomas with a supra- and parasellar extensions [1, 2].

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From the optical point of view, the superior visualization within the relatively narrow nasal corridor is achieved because the endoscope brings the light and the viewing lens inside the surgical field resulting in a very highly illuminated area of interest. Furthermore, the close proximity of the light source to the structures being viewed eliminates shadows within the field, adding to the extreme clarity of the viewed images. The superiority of the endoscopic view (Fig. 7.1) is also contributed to by the high color fidelity and image definition capabilities of today's state-of-the-art rigid endoscopes. An additional advantage of the endoscopic view is the greater depth of focus in comparison to that of the microscope, which results in lesser need to adjust the focus of the endoscope during the procedure leading to a seamless operative workflow. The use of angled scopes also enables "looking around the corners" and therefore adds further to the efficacy and safety of the procedure specially when it comes to excision of suprasellar and parasellar tumors [6].

As a matter of fact, the demonstration of feasibility of endoscopic skull base surgery was followed by a period of validation and popularization with improved outcomes and newly developed treatment paradigms that incorporated the original and the emerging technologies and procedures [7]. For pituitary adenomas, the endoscopic transsphenoidal approach has subsequently been adopted worldwide as an alternative to the microscopic approach [1], and the extended endoscopic endonasal approaches to various skull base pathologies have exploded in popularity over the last two decades. Presently, the lesions treated can be located anywhere from the frontal sinus to the odontoid in the sagittal plane (Fig. 7.2) and from midline to the middle fossa in the coronal plane [8]. Examples of these pathologies include tuberculum sellae, planum sphenoidale, and olfactory groove meningiomas, craniopharyngiomas, clival tumors like chordomas and chondrosarcomas, petrous apex tumors, and lesions of the infratemporal fossa [9–17].

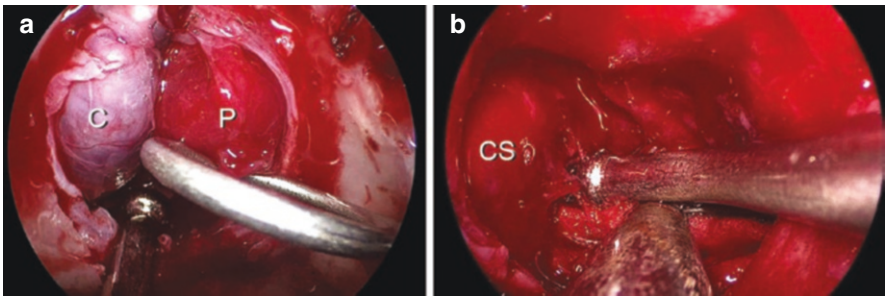


Fig. 7.1 The superiority of endoscopic view. An example from an endoscopic endonasal transsphenoidal approach for a giant pituitary adenoma after complete tumor excision. In (**a**), note the descending cistern, C, and the normal pituitary gland, P. In (**b**), a close-up view of the medial wall of the cavernous sinus; CS is seen

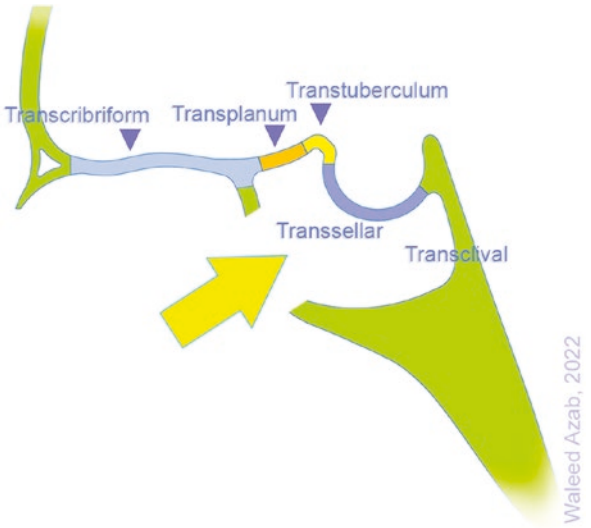


Fig. 7.2 The standard and extended endoscopic endonasal approaches to various skull base pathologies in the sagittal plane

7.2 Surgical Technique of Endoscopic Endonasal Transsphenoidal Approach

In our operating room setup, the operating surgeon stands on the right side of the patient, and the assistant stands on the left side facing the surgeon. The endoscope tower and monitor are positioned at the patient's head with a Mayo stand between the patient's head and the endoscope tower. The navigation monitor is positioned next to the endoscope tower and is facing the operating surgeon. The scrub nurse and instrument tables are on the left side of the operating table next to the assistant surgeon. Under general anesthesia, the patient is positioned supine, and the head is elevated 10–15 degrees above the level of the heart. On a MAYFIELD® Headrest, the head is neutrally positioned and then rotated 10 degrees toward the surgeon. Stereotactic image guidance is not a routine in all cases. We use navigation to identify the carotid arteries based on CTA images, to guide the extent of bone resection when extended approaches are performed, in patients with complex bony anatomy of the paranasal sinuses and in recurrent cases. In cases where neuronavigation is used, the patient's head is fixed by MAYFIELD® skull clamp. Flexion or extension of the patient's head is undertaken in transplanum-transtuberculum sellae and in transclival approaches, respectively. Cottonoids soaked in 0.5% adrenaline solution are packed into the nasal cavities immediately after the patient is anaesthetized and are later removed just before draping. The vasoconstriction induced by epinephrine reduces the thickness of the nasal mucosa and greatly helps developing a sufficiently wide surgical corridor. Furthermore, it decreases mucosal bleeding during the following steps of the procedure.

The procedure is carried out using a purely endoscopic approach primarily with the aid of a 0° 4-mm, 30-cm rigid endoscope (Karl Storz GmbH & Co. KG). The 0°

endoscope is to be held at the 12 o'clock position in the superior aspect of the nasal cavity. In this position, the endoscope shaft is pushed further superiorly against the elastic tissue of the nasal aperture, giving more room for instrument manipulation within the nasal cavity. Suction is usually positioned at 6 o'clock position. At the initial stages of the procedure when the two nasal cavities are still separated, in some patients with ample nasal cavity, another instrument can be inserted between the endoscope and the suction. No endoscopic holders or nasal specula are used.

The surgical technique of the endoscopic endonasal transsphenoidal approach basically consists of three stages, namely, *the nasal*, *the sphenoidal*, and *the sellar* steps.

7.2.1 The Nasal Step

The anatomical landmarks within the nasal cavity are identified and include the nasal floor, the inferior and middle turbinates, and the nasal septum. Adrenaline-soaked cottonoids are then inserted between the nasal septum and the middle turbinate. Lateralization of the middle turbinate is then performed using the shaft of a Freer elevator to develop the surgical corridor (Fig. 7.3a). At this stage, advancing the scope into the nasal cavity enables visualization of the choana, an important landmark based on which the approach proceeds further. Once the choana is reached, the endoscope is directed upward where the sphenoid face, sphenoethmoid recess, and the ostia of the sphenoid sinus are identified.

In cases where an extended endoscopic endonasal approach is performed, a middle turbinectomy is required to obtain sufficient space and angles during tumor resection. To spare the olfactory mucosa, the middle turbinate is cut parallel to the anterior skull base between its upper and lower two thirds using a septum scissors. Proceeding posteriorly, the blades of the scissors should be pushed inferiorly to complete the separation of the turbinate. The disconnected middle turbinate is then grasped using a Blakesley nasal forceps and removed.

7.2.2 The Sphenoidal Step

The sphenoidal step (Figs. 7.3b–f and 7.4) commences by coagulating the mucosa covering the face of sphenoid, the sphenoethmoid recess, and the posterior part of nasal septum using a suction monopolar. When harvesting a nasoseptal flap is planned, the posterior septal artery (supplying the pedicled flap) should be preserved. The nasal septum is then dislocated from its posterior attachment, and a posterior septectomy is performed using a backbiting forceps.

Drilling the whole sphenoid face and posterior vomer is subsequently undertaken using a 3- to 5-mm coarse diamond bit to widely open the sphenoid sinus. The sphenoid opening can be completed using a Kerrison rongeur if needed.

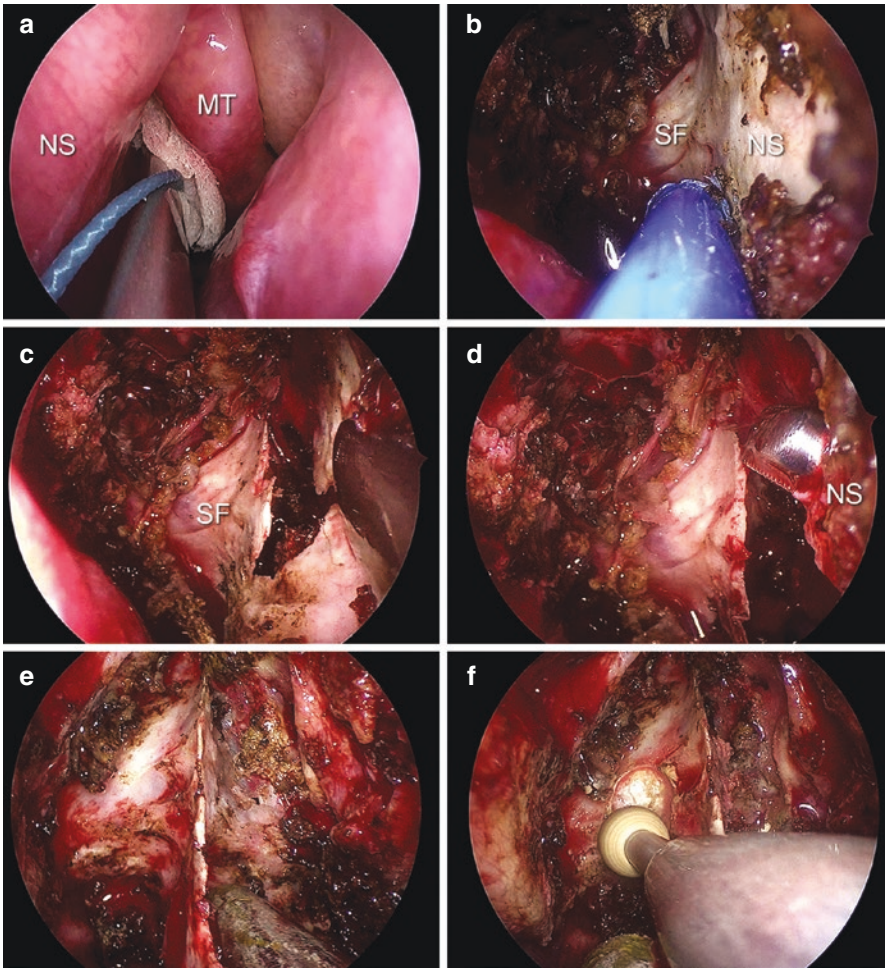


Fig. 7.3 The nasal and sphenoidal steps of the endoscopic endonasal transsphenoidal approach. In the nasal step (**a**), the operative corridor is developed by inserting an adrenaline-soaked cottonoid patty between the middle turbinate (MT) and the nasal septum (NS). The middle turbinate (MT) is then lateralized using a dissector. In the sphenoidal step (**b**–**f**), the mucosa covering the face of sphenoid face (SF) and the sphenoethmoid recess is coagulated and removed (**b**). The mucosa of the posterior nasal septum (NS) is then coagulated and removed bilaterally. The nasal septum (NS) is subsequently dislocated using a dissector (**c**), and a posterior septectomy is performed using a backbiting forceps (**d**). After the sphenoid face is completely exposed (**e**), its drilling is performed using a sharp or sharp diamond drill pit (**f**)

As the sphenoid sinus is entered, key anatomical landmarks within its cavity are visualized. These include the sellar face, optic canal, carotid protuberance, lateral and medial optico-carotid recesses, tuberculum sellae, planum sphenoidale, clival recess, and the paraclival carotids. Because of the variable pneumatization patterns

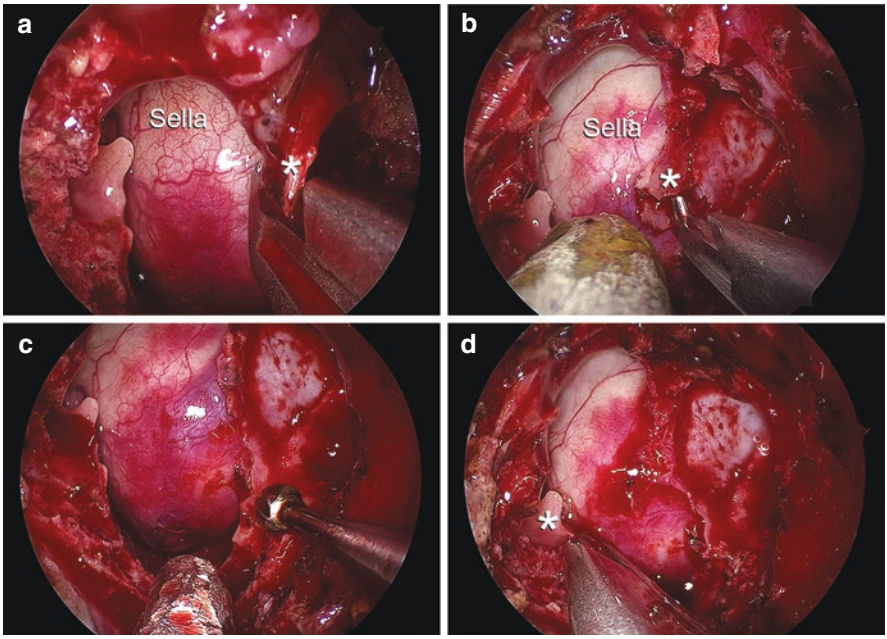


Fig. 7.4 During the sphenoidal step, the bony septa (asterisks) inside the sphenoid sinus should be drilled down to their attachment or carefully removed using a Kerrison rongeur as demonstrated in these serial operative views (**a**–**d**)

of the sphenoid sinus, some of the aforementioned anatomical landmarks may be not very clearly appreciable.

The sphenoid mucosa is then removed, and the bony septations within the sinus are carefully drilled down to their attachment. The bony septa within the sphenoid sinus usually lead to the internal carotid artery. Extreme care should therefore be practiced during their removal to avoid vascular injury.

7.2.3 The Sellar Step

The sellar step (Fig. 7.5) starts by drilling the sellar face down to the clival recess below and up to the tuberculum sellae in the standard transsellar approach. Bilaterally, it extends between the two carotid prominences. Removal of bone of the tuberculum sellae, planum sphenoidale, or the clival recess is undertaken when transtuberculum, transplanum, or transclival approaches are planned, respectively. Bone removal is performed using a 3- or 5-mm diamond burr, and the thin shell of bone remaining after drilling is removed using a fine microdissector. A 1-mm Kerrison rongeur is then used to complete the bone window.

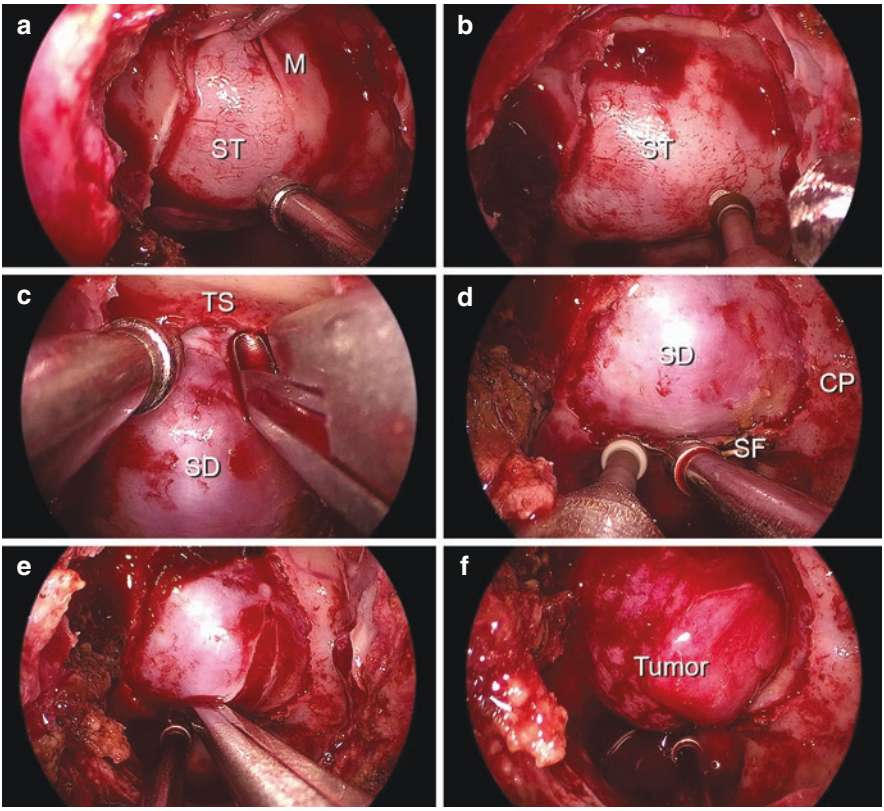


Fig. 7.5 The sphenoidal and sellar steps. (**a**) The sphenoidal step is completed by removing the sphenoid mucosa (M). (**b**) The sellar step then starts by drilling the bony face of the sella turcica (ST). The extent of sellar dura (SD) exposure in the basic transsellar approach extends from the tuberculum sellae (TS) above to the sellar floor (SF) below and between the carotid prominences (CP) bilaterally (**c**, **d**). The dural incision is then performed using a retractable knife and completed with an angled scissors (**e**) to gain access to the tumor (T) as seen in (**f**)

The dural incision is performed using a retractable knife and angled scissors. In cases of small sellar lesions, the incision can be cruciate or U-shaped and extends between the two cavernous sinuses bilaterally and between the superior and inferior intercavernous sinuses craniocaudally. In large pituitary adenomas, resecting a quadrangular dural flap greatly helps with tumor excision. In transtuberculum-transplanum approach, bipolar coagulation of the superior intercavernous sinus is performed before cutting through the sinus to avoid brisk venous bleeding.

For intrasellar pathologies, tissue biopsy is performed using a pituitary rongeur, and then tumor removal proceeds using mainly suction and pituitary ring curettes of variable angles. Piecemeal resection is sometimes needed when tumor is hard in consistency. In cases when the tumor is posterior to the pituitary gland, a vertical incision in the normal gland is made allowing access to the tumor. Thorough

endoscopic inspection of the sellar cavity is performed to make sure that no tumor tissue is left. Full descent of the suprasellar cistern is observed at this stage.

Tumor excision should start first from the inferior, posterior, and lateral parts of the sellar cavity, leaving the superior part of the tumor to the end in order to prevent an early descent of the suprasellar cistern which obstructs the view before tumor removal is completed. Should this occur, a cottonoid patty is used to elevate the bulging cistern and allow visualization of the remaining tumor tissue and its further resection. This maneuver can also be performed using a ring pituitary curette or a second suction tube to retract the redundant cistern and a suction tube to remove the tumor.

In the standard transsellar approach, angled 30° and 45° 4-mm scopes are used for visualization within the cavernous sinuses and the superior and superolateral corners near the end of tumor excision. In extended approaches, angled scopes are also very valuable for visualization of pathoanatomical structures and relationships. Bimanual technique of dissection and resection should be used.

In the transsellar approach, hemostasis and closure (Fig. 7.6) are performed using Floseal® and TISSEEL®, followed by layers of Surgicel®. Autologous fat is used in cases with minor CSF leaks and is reinforced with TISSEEL® and Surgicel®.

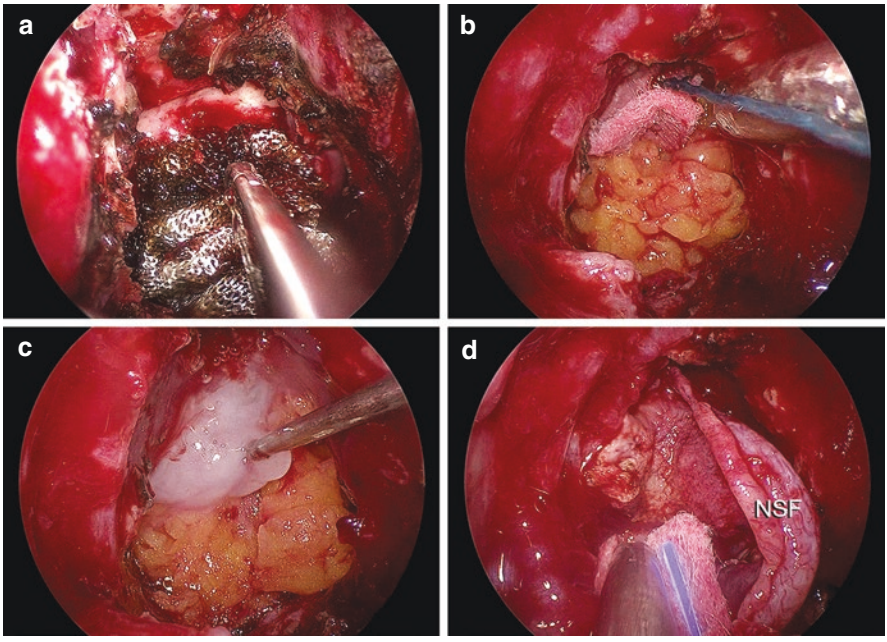


Fig. 7.6 The reconstruction after endoscopic endonasal transsphenoidal approach is performed by applying multiple layers of Surgicel® and applying TISSEEL® in cases without CSF leakage (**a**). In cases with minimal arachnoid breaches and CSF leaks, an autologous fat graft is used and reinforced with TISSEEL® and multiple layers of Surgicel® (**b**, **c**). A nasoseptal pedicled flap (NSF) is harvested and used over the fat graft in cases with large arachnoid defects and vigorous CSF leaks like extended approaches (**d**)

Surgicel® should not be packed in the sellar cavity and is to be left only in the sphenoid sinus. It functions as a scaffold to support the reconstruction and also enhances healing by lowering the local tissue pH. A free mucosal flap harvested from a resected middle turbinate can also be used when CSF leak takes place. In extended approaches, a vascularized nasoseptal flap based on the posterior septal branch of the sphenopalatine artery and autologous fat is used for skull base reconstruction. Before the procedure is concluded, the nasal cavity is inspected to control any bleeding points and to clear blood clots off the nasal passages. The lateralized middle turbinate is then medialized. Nasal packing is not needed in the majority of cases and is only routinely used when a free or nasoseptal flap is used [18].

7.3 The Uncommon Pathologies of the Sella

Apart from the common pathological entities, many other less frequent pathologies are encountered in the sellar and parasellar regions. As a matter of fact, classifying the rare and non-adenomatous lesions of the sellar area has been attempted by many research groups; however, no consensus presently exists in that regard.

The differential diagnosis of nonpituitary sellar masses is very broad [19]. In Table 7.1, many of these lesions are enlisted [20–66]. Differentiating among the potential etiologies may not always be straightforward because many of these rare sellar lesions mimic the clinical, endocrinologic, and radiographic presentations of pituitary adenomas [19, 67, 68]. Owing to the rarity of these lesions, the frequency and types of the encountered pathologies across the published series are quite variable [19, 67–70]. In one study combining cases from two centers, rare sellar pathologies comprised 78 patients out of 346 non-adenomatous sellar pathologies. Arachnoid cysts were the most frequently encountered (15%), followed by metastasis (14%), hypophysitis (10%), and oncocytoma and glioma (8% each). A standard endoscopic endonasal approach was performed in 56% and an extended approach was used in 44% of patients [67].

Extremely rare pathologies have also been reported in the sellar suprasellar area including diffuse large cell B cell lymphoma primary to the sellar/suprasellar region, primary fibrosarcoma of the sella, primary MPNST of the sella, GPA and NS with first presentation in CNS, CLL within a pituitary adenoma, and aberrant nerve fascicles within a pituitary adenoma [71]. An increased awareness of the unusual entities that may involve the sellar region is therefore needed [72]. It is of note that surgical endoscopic experience allows better interpretation of intraoperative features, orienting the diagnosis and subsequent management [67].

An exhaustive review of these pathological entities is beyond the scope of this chapter. In the following paragraphs, however, some of these lesions will be elaborated upon.

Table 7.1 Examples of uncommon sellar pathologies

1. *Neoplastic*	
(a) Uncommon pituitary adenomas (PitNET)	Honegger J, Nasi-Kordhishti I, Giese S. Hypophysenadenome [Pituitary adenomas]. Nervenarzt. 2019 Jun;90(6):568-577. German. doi: 10.1007/s00115-019-0708-4
(b) Posterior pituitary tumors	Kinoshita Y, Yamasaki F, Tominaga A, Usui S, Arita K, Sakoguchi T, Sugiyama K, Kurisu K. Transsphenoidal Posterior Pituitary Lobe Biopsy in Patients with Neurohypophysial Lesions. World Neurosurg. 2017 Mar;99:543-547. doi: 10.1016/j.wneu.2016.12.080. Epub 2016 Dec 27
(c) Pituitary blastoma	Chhuon Y, Weon YC, Park G, Kim M, Park JB, Park SK. Pituitary Blastoma in a 19-Year-Old Woman: A Case Report and Review of Literature. World Neurosurg. 2020 Jul;139:310-313. doi: 10.1016/j.wneu.2020.04.096
(d) Pituitary carcinoma	Sansur CA, Oldfield EH. Pituitary carcinoma. Semin Oncol. 2010 Dec;37(6):591-3. doi: 10.1053/j.seminoncol.2010.10.012
(e) Metastasis to the pituitary	Castle-Kirszbaum M, Goldschlager T, Ho B, Wang YY, King J. Twelve cases of pituitary metastasis: a case series and review of the literature. Pituitary. 2018 Oct;21(5):463-473. doi: 10.1007/s11102-018-0899-x
(f) Others:	
• Glial tumors	
Astrocytoma	Wang J, Liu Z, Du J, Cui Y, Fang J, Xu L, Li G. The clinicopathological features of pituicytoma and the differential diagnosis of sellar glioma. Neuropathology. 2016 Oct;36(5):432-440. doi: 10.1111/neup.12291
Glioblastoma	Mahta A, Buhl R, Huang H, Jansen O, Kesari S, Ulmer S. Sellar and supra-sellar glioblastoma masquerading as a pituitary macroadenoma. Neurol Sci. 2013 Apr;34(4):605-7. doi: 10.1007/s10072-012-1110-1
Pilocytic astrocytoma	Prashant Prasad G, Lang FF, Bruner JM, Ater JL, McCutcheon IE. Transsphenoidal removal of intrasellar pilocytic astrocytoma. J Clin Neurosci. 2014 Jun;21(6):1047-8. doi: 10.1016/j.jocn.2013.10.004
Pleomorphic xanthoastrocytoma	Arita K, Kurisu K, Tominaga A, Sugiyama K, Sumida M, Hirose T. Intrasellar pleomorphic xanthoastrocytoma: case report. Neurosurgery. 2002 Oct;51(4):1079-82; discussion 1082. doi: 10.1097/00006123-200210000-00042
• Glioneuronal and neuronal tumors	
Ganglioglioma	Matyja E, Maksymowicz M, Grajkowska W, Zieliński G, Kunicki J, Bonicki W, Witek P, Naganska E. Ganglion cell tumours in the sella turcica in close morphological connection with pituitary adenomas. Folia Neuropathol. 2015;53(3):203-18. doi: 10.5114/fn.2015.54421
Papillary glioneuronal tumor	Emanuelli E, Zanotti C, Munari S, Baldovin M, Schiavo G, Denaro L. Sellar and parasellar lesions: multidisciplinary management. Acta Otorhinolaryngol Ital. 2021 Apr;41(Suppl. 1):S30-S41. doi: 10.14639/0392-100X-suppl.1-41-2021-03

Table 7.1 (continued)

Gangliocytoma	Quiroga-Padilla PJ, González-Devia D, Andrade R, Escalante P, Jiménez-Hakim E. Sellar Gangliocytoma: Case Report and Review of an Extremely Rare Tumour. Case Rep Neurol. 2021 Jul 19;13(2):475-482. doi: 10.1159/000517368
Neurocytoma	Nery B, Bernardes Filho F, Costa RAF, Pereira LCT, Quaggio E, Queiroz RM, Abud LG, da Cunha Tirapelli DP. Neurocytoma mimicking macroadenoma. Surg Neurol Int. 2019 Jan 21;10:8. doi: 10.4103/sni.sni_387_18 Wang J, Song DL, Deng L, Sun SY, Liu C, Gong DS, Wang Y, Xu QW. Extraventricular neurocytoma of the sellar region: case report and literature review. Springerplus. 2016 Jul 7;5(1):987. doi: 10.1186/s40064-016-2650-2
• Ependymal tumors	
Ependymoma	Wang S, Zong W, Li Y, Wang B, Ke C, Guo D. Pituitary Ependymoma: A Case Report and Review of the Literature. World Neurosurg. 2018 Feb;110:43-54. doi: 10.1016/j.wneu.2017.10.134
• Choroid plexus tumors	
Choroid plexus papilloma	Kuo CH, Yen YS, Tu TH, Wu JC, Huang WC, Cheng H. Primary Choroid Plexus Papilloma over Sellar Region Mimicking with Craniopharyngioma: A Case Report and Literature Review. Cureus. 2018 Jun 20;10(6):e2849. doi: 10.7759/cureus.2849
• Embryonal tumors	
Atypical teratoid/rhabdoid tumor	Liu F, Fan S, Tang X, Fan S, Zhou L. Adult Sellar Region Atypical Teratoid/Rhabdoid Tumor: A Retrospective Study and Literature Review. Front Neurol. 2020 Dec 15;11:604612. doi: 10.3389/fneur.2020.604612
Neuroblastoma	Kalinin PL, Fomichev DV, Abdilatipov AA, Chernov IV, Astafieva LI, Kutin MA, Ryzhova MV, Panina TN, Shishkina LV, Nikitin PV, Kurnosov AB. Pervichnaya sellyarnaya neiroblastoma. Klinicheskoe nablyudenie i obzor literatury [Primary sellar neuroblastoma (clinical case and literature review)]. Zh Vopr Neirokhir Im N N Burdenko. 2020;84(2):83-92. Russian. doi: 10.17116/neiro20208402183
Primitive neuroectodermal tumor	Yakar F, Doğan İ, Meco C, Heper AO, Kahilogullari G. Sellar Embryonal Tumor: A Case Report and Review of the Literature. Asian J Neurosurg. 2018 Oct-Dec;13(4):1197-1201. doi: 10.4103/ajns.AJNS_30_17
• Cranial and paraspinal nerve tumors	

(continued)

Table 7.1 (continued)

Schwannoma	Kong X, Wu H, Ma W, Li Y, Yang Y, Xing B, Wei J, Yao Y, Gao J, Lian W, Xu Z, Dou W, Ren Z, Su C, Wang R. Schwannoma in Sellar Region Mimics Invasive Pituitary Macroadenoma: Literature Review With One Case Report. Medicine (Baltimore). 2016 Mar;95(9):e2931. doi: 10.1097/MD.0000000000002931 Zhang J, Xu S, Liu Q, Li X, Jia D, Li G. Intrasellar and Suprasellar Schwannoma Misdiagnosed as Pituitary Macroadenoma: A Case Report and Review of the Literature. World Neurosurg. 2016 Dec;96:612.e1-612.e7. doi: 10.1016/j.wneu.2016.08.128
Paraganglioma	Lyne SB, Polster SP, Fidai S, Pytel P, Yamini B. Primary Sellar Paraganglioma: Case Report with Literature Review and Immunohistochemistry Resource. World Neurosurg. 2019 May;125:32-36. doi: 10.1016/j.wneu.2019.01.094
• Meningiomas	
Meningioma (pure intrasellar)	Bang M, Suh JH, Park JB, Weon YC. Pure Intrasellar Meningioma Mimicking Pituitary Macroadenoma: Magnetic Resonance Imaging and Review of the Literature. World Neurosurg. 2016 Jul;91:675.e1-4. doi: 10.1016/j.wneu.2016.04.063
• Mesenchymal, non-meningothelial tumors	
Solitary fibrous tumor	Zhong Q, Yuan S. Total resection of a solitary fibrous tumor of the sellar diaphragm: A case report. Oncol Lett. 2013 Jun;5(6):1783-1786. doi: 10.3892/ol.2013.1293
Hemangioma	Singh U, Kalavakonda C, Venkitachalam S, Patil S, Chinnusamy R. Intraosseous Hemangioma of Sella: Case Report and Review of Literature. World Neurosurg X. 2019 Mar 9;3:100030. doi: 10.1016/j.wnsx.2019.100030
Hemangioblastoma	Ajler P, Goldschmidt E, Bendersky D, Hem S, Landriel F, Campero A, Ajler G. Sellar hemangioblastoma mimicking a macroadenoma. Acta Neurol Taiwan. 2012 Dec;21(4):176-9
Sarcoma	Guerrero-Pérez F, Vidal N, López-Vázquez M, Sánchez-Barrera R, Sánchez-Fernández JJ, Torres-Díaz A, Vilarrasa N, Villabona C. Sarcomas of the sellar region: a systematic review. Pituitary. 2021 Feb;24(1):117-129. doi: 10.1007/s11102-020-01073-9
Rhabdomyosarcoma	Arita K, Sugiyama K, Tominaga A, Yamasaki F. Intrasellar rhabdomyosarcoma: case report. Neurosurgery. 2001 Mar;48(3):677-80. doi: 10.1097/00006123-200103000-00048
Ewing sarcoma	Mattogno PP, Nasi D, Iaccarino C, Oretti G, Santoro L, Romano A. First Case of Primary Sellar/Suprasellar-Intraventricular Ewing Sarcoma: Case Report and Review of the Literature. World Neurosurg. 2017 Feb;98:869.e1-869.e5. doi: 10.1016/j.wneu.2016.12.045
Mesenchymal chondrosarcoma	Inenaga C, Morii K, Tamura T, Tanaka R, Takahashi H. Mesenchymal chondrosarcoma of the sellar region. Acta Neurochir (Wien). 2003 Jul;145(7):593-7; discussion 597. doi: 10.1007/s00701-003-0059-5

Table 7.1 (continued)

Chondrosarcoma	Zhang Y, Huang J, Zhang C, Jiang C, Ding C, Lin Y, Wu X, Wang C, Kang D, Lin Z. An Extended Endoscopic Endonasal Approach for Sellar Area Chondrosarcoma: A Case Report and Literature Review. World Neurosurg. 2019 Jul;127:469-477. doi: 10.1016/j.wneu.2019.04.075
• Notochordal tumors	
Sellar chordoma	Kikuchi K, Watanabe K. Huge sellar chordoma: CT demonstration. Comput Med Imaging Graph. 1994 Sep-Oct;18(5):385-90. doi: 10.1016/0895-6111(94)90010-8
• Melanocytic tumors	
Meningeal melanocytoma	Wang F, Ling S. Primary Meningeal Melanocytoma in Sellar Region, Simulating a Nonfunctioning Pituitary Adenoma: Case Report and Literature Review. World Neurosurg. 2018 Apr;112:209-213. doi: 10.1016/j.wneu.2018.01.145
• Hematolymphoid tumors	
– Lymphomas	
Lymphoma	Tarabay A, Cossu G, Berhouma M, Levivier M, Daniel RT, Messerer M. Primary pituitary lymphoma: an update of the literature. J Neurooncol. 2016 Dec;130(3):383-395. doi: 10.1007/s11060-016-2249-z
Primary diffuse large B-cell lymphoma of the CNS	Ravindra VM, Raheja A, Corn H, Driscoll M, Welt C, Simmons DL, Couldwell WT. Primary pituitary diffuse large B-cell lymphoma with somatotroph hyperplasia and acromegaly: case report. J Neurosurg. 2017 May;126(5):1725-1730. doi: 10.3171/2016.5.JNS16828
– Histiocytic tumors	
Erdheim-Chester disease	Oweity T, Scheithauer BW, Ching HS, Lei C, Wong KP. Multiple system Erdheim-Chester disease with massive hypothalamic-sellar involvement and hypopituitarism. J Neurosurg. 2002 Feb;96(2):344-51. doi: 10.3171/jns.2002.96.2.0344
Rosai-Dorfman disease	Zhang Y, Liu J, Zhu J, Zhou X, Zhang K, Wang S, Ma W, Pan H, Wang R, Zhu H, Yao Y. Case Report: Rosai-Dorfman Disease Involving Sellar Region in a Pediatric Patient: A Case Report and Systematic Review of Literature. Front Med (Lausanne). 2020 Nov 30;7:613756. doi: 10.3389/fmed.2020.613756
Langerhans cell histiocytosis	Tan H, Yu K, Yu Y, An Z, Li J. Isolated hypothalamic-pituitary langerhans' cell histiocytosis in female adult: A case report. Medicine (Baltimore). 2019 Jan;98(2):e13853. doi: 10.1097/MD.0000000000013853
• Germ cell tumors	
Teratoma	Saeger W, Ebrahimi A, Beschorner R, Spital H, Honegger J, Wilczak W. Teratoma of the Sellar Region: a Case Report. Endocr Pathol. 2017 Dec;28(4):315-319. doi: 10.1007/s12022-016-9465-0

(continued)

Table 7.1 (continued)

Germinoma	Li CS. Intrasellar germinoma treated with low-dose radiation. Acta Neurochir (Wien). 2006 Jul;148(7):795-9; discussion 799. doi: 10.1007/s00701-006-0776-7
Choriocarcinoma	Musiani P, Mancuso S. Coriocarcinoma primitivo intracranico originato da tumore germinale sellare in una bambina in età prepubere [Intracranial primary choriocarcinoma originating from a sellar germinal tumor in a girl of prepuberal age]. Arch Ital Anat Istol Patol. 1969 Dec;43(1):61-75. Italian. PMID: 5393121
Mixed germ cell tumor	Wildemberg LE, Vieira Neto L, Taboada GF, Moraes AB, Marcondes J, Conceição FL, Chimelli L, Gadelha MR. Sellar and suprasellar mixed germ cell tumor mimicking a pituitary adenoma. Pituitary. 2011 Dec;14(4):345-50. doi: 10.1007/s11102-008-0161-z
• Salivary gland-like lesions	*See text*
2. *Granulomatous, infectious, and inflammatory lesions*	
Xanthogranuloma	Cheng D, Yang F, Li Z, Qv F, Liu W. Juvenile Xanthogranuloma of the Sellar Region with a 5-Year Medical History: Case Report and Literature Review. Pediatr Neurosurg. 2021;56(5):440-447. doi: 10.1159/000515517
Granulomatosis with polyangiitis (GPA) (formerly Wegener granulomatosis)	*See text*
Hypophysitis	*See text*
Pituitary abscess	*See text*
3. *CSF-containing cysts*	
PEIR	*See text*
Intrasellar arachnoid cyst	Mangussi-Gomes J, Gentil AF, Filippi RZ, Momesso RA, Handfas BW, Radvany J, Balsalobre L, Stamm AC. Sellar and suprasellar arachnoid cyst. Einstein (Sao Paulo). 2019 Jan 31;17(1):eAI4269. doi: 10.31744/einstein_journal/2019AI4269
4. *Cell rest lesions*	
Colloid cysts	Guduk M, Sun HI, Sav MA, Berkman Z. Pituitary Colloid Cyst. J Craniofac Surg. 2017 Mar;28(2):e166-e168. doi: 10.1097/SCS.0000000000003142
Dermoid	Pan YB, Sun ZL, Feng DF. Intrasellar dermoid cyst mimicking pituitary apoplexy: A case report and review of the literature. J Clin Neurosci. 2017 Nov;45:125-128. doi: 10.1016/j.jocn.2017.05.023
Epidermoid	Vellutini EAS, Pahl FH, Stamm AEC, Teles Gomes MQ, de Oliveira MF, Martins HO, Ruschel LG. Endoscopic resection of sellar and suprasellar epidermoid cyst: report of two cases and review of literature. Br J Neurosurg. 2021 Feb 1:1-6. doi: 10.1080/02688697.2021.1877610

7.3.1 CSF-Containing Cysts

7.3.1.1 Intrasellar Arachnoid Cysts

Intrasellar arachnoid cysts (Fig. 7.7) are relatively rare, with only few reports available in the literature [73, 74].

Large intrasellar arachnoid cysts often result in compression of the neighboring anatomical structures and therefore produce a clinical picture that is similar to a non-secreting pituitary adenoma. They mainly manifest with visual disturbances and hypopituitarism [75, 76], requiring surgical intervention.

Immunohistochemical evaluation is helpful in differentiating arachnoid cysts from epithelial cysts, a distinction that is sometimes difficult to achieve using routine histopathological examination [74]. Arachnoid cysts are positive for EMA but are negative for cytokeratins, GFAP, S-100 protein, transthyretin, and CEA [77].

The advantages of endoscopic transsphenoidal approach in these lesions include precise identification of the cyst wall and the normal pituitary tissue that is usually adherent and thinned out over the arachnoid cyst wall. This contributes to proper opening of the capsular wall to communicate with the subarachnoid space and to preservation of pituitary function [75].

Communicating the cyst with the subarachnoid space has evidently a downside of higher CSF leakage. Endoscopic endonasal obliteration of the cyst cavity with fat graft has been described with good results [76].

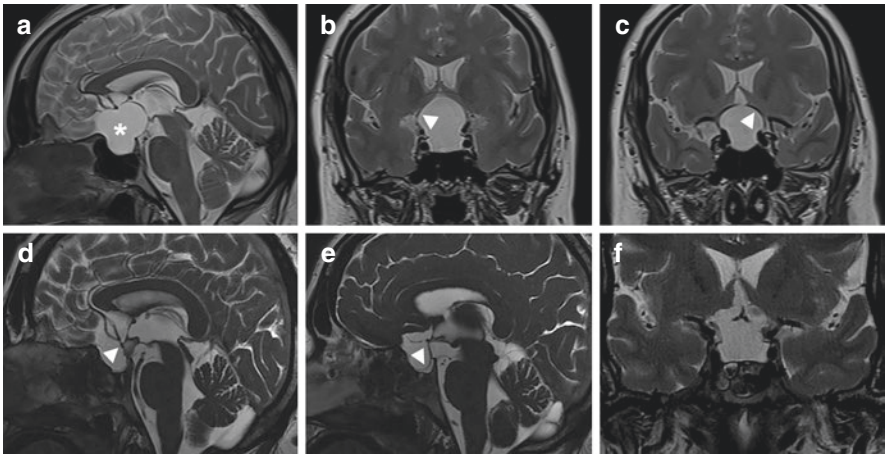


Fig. 7.7 Intrasellar arachnoid cyst. Preoperative T2-weighted MRI (**a**–**c**) revealing an intrasellar arachnoid cyst with suprasellar extension. (**a**) The lesion (asterisk) expands the sella turcica and causes compression of the floor of the third ventricle. (**b**) Coronal image demonstrating the stretched optic chiasm (arrowhead) on the superior aspect of the cyst wall. (**c**) Coronal image demonstrating the stretched A1 segment (arrowhead) on the superior aspect of the cyst wall. Postoperative T2-weighted MRI (**d**–**f**) revealed a full decompression of the cyst with notable descent of the anterior cerebral complex vessels (**d**; arrowhead) and superior cyst wall (**e**; arrowhead and **f**)

McLaughlin et al. described a fully endoscopic technique in which simple cyst fenestration is followed by obliteration of the cyst cavity using an autologous fat graft. A simplified skull base reconstruction is then performed and consists of multilayer repair and intrasellar extradural titanium micromesh reinforcement without pedicled flaps. In this technique, communicating the cyst with the subarachnoid space is avoided, and absence of diaphragmatic defects or arachnoid diverticula is confirmed under close-up endoscopic view [73].

7.3.1.2 Persisting Embryonal Infundibular Recess (PEIR) and TSE

Persisting embryonal infundibular recess (PEIR) is a very rare anomaly of the floor of the third ventricle [78] in which the embryonic morphology of the infundibular recess (IR) persists (Fig. 7.8) [79]. Rather than the normal postnatal configuration in which the IR is seen as a small funnel-shaped extension of the third ventricle within the pituitary stalk, PEIR is characterized by a tubular cavity that outpouches from the third ventricle and continues downward to terminate as dilated inferior end within the sella turcica. The tubular cavity is surrounded by a dilated pituitary stalk and displays the same signal intensity of cerebrospinal fluid (CSF) on magnetic resonance imaging (MRI) (Figs. 7.9 and 7.10) [79–81].

To the best of our knowledge, only 11 cases of PEIR have been reported so far in the English literature [82]. The main findings are generally those of a dilated pituitary stalk within which a tubular cavity that has the same signal intensity of CSF is

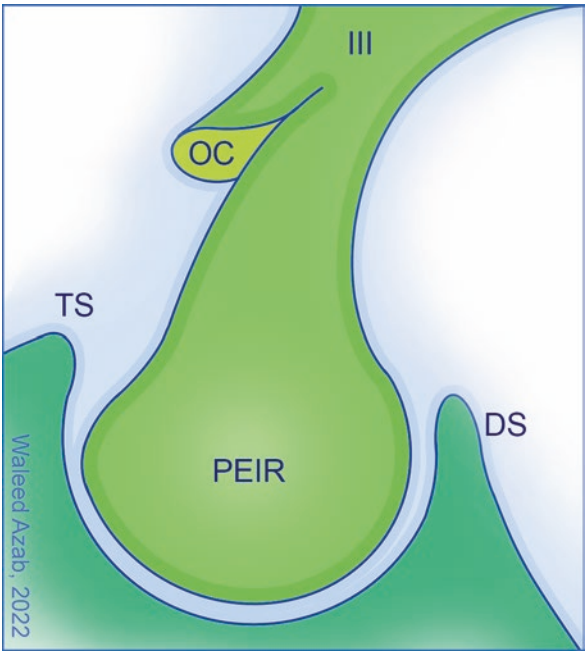


Fig. 7.8 PEIR is characterized by a tubular cavity that outpouches from the third ventricle and continues downward to terminate as dilated inferior end within the sella turcica

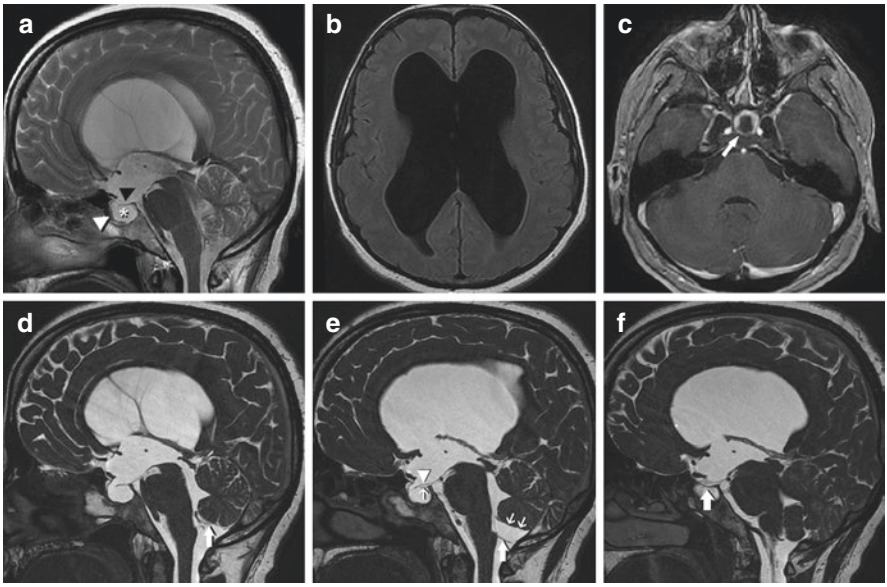


Fig. 7.9 PEIR with tetraventricular hydrocephalus in a patient who underwent ETV. Preoperative sagittal T2-wighted image (**a**) demonstrating an enlarged sella with a picture simulating that of an empty sella (asterisk) but suggestive of PEIR. The pituitary gland is probably seen at the sellar floor (white arrowhead). The diaphragma sellae (black arrowhead) is clearly seen. Note the enlarged third ventricle and patent aqueduct with CSF flow signal. Enlargement of the lateral and fourth ventricles is seen in the axial T2-Flair (**b**) and T1-wighted with contrast (**c**) MR images. Postoperative MRI 3D FIESTA-C (three-dimensional fast imaging employing steady-state acquisition cycled phases) (**d**–**f**) reveal the PEIR as a tubular downward extension of the third ventricular floor into a cystic expansion within the sella. A clear distinction is evident between the diaphragma sellae (**e**, small arrow and, **f**, arrow) and the membranous third ventricular floor with PEIR (**e**, arrowhead)

seen. The tubular cavity is in continuity with the third ventricle superiorly and extends downward into an inferior end within the sella turcica. The intrasellar end is often dilated, with variable morphological descriptions among the reported cases including a round pocket [83], a round cystic formation [78, 81, 84], a multicystic dilatation [85], or a diverticulum of the anterior third ventricle [86].

Recently, Kelsch et al. pointed out that PEIR should perhaps belong to the spectrum of abnormalities of midline encephaloceles [87]. In a recently published work, our group in collaboration with Naples group suggested that PEIR and TSE do actually represent the two extremes of one continuum of malformations [82]. This was based on (1) the very close morphological similarity between the third ventricle's floor and its IR in cases of TSE and those in the published cases of PEIR, (2) the intersecting spectra of associated anomalies in PEIR and TSE, (3) the frequent impossibility to identify the pituitary gland on imaging studies in both PEIR and TSE, (4) the associated pituitary dysfunction in PEIR and TSE, and (5) the possible

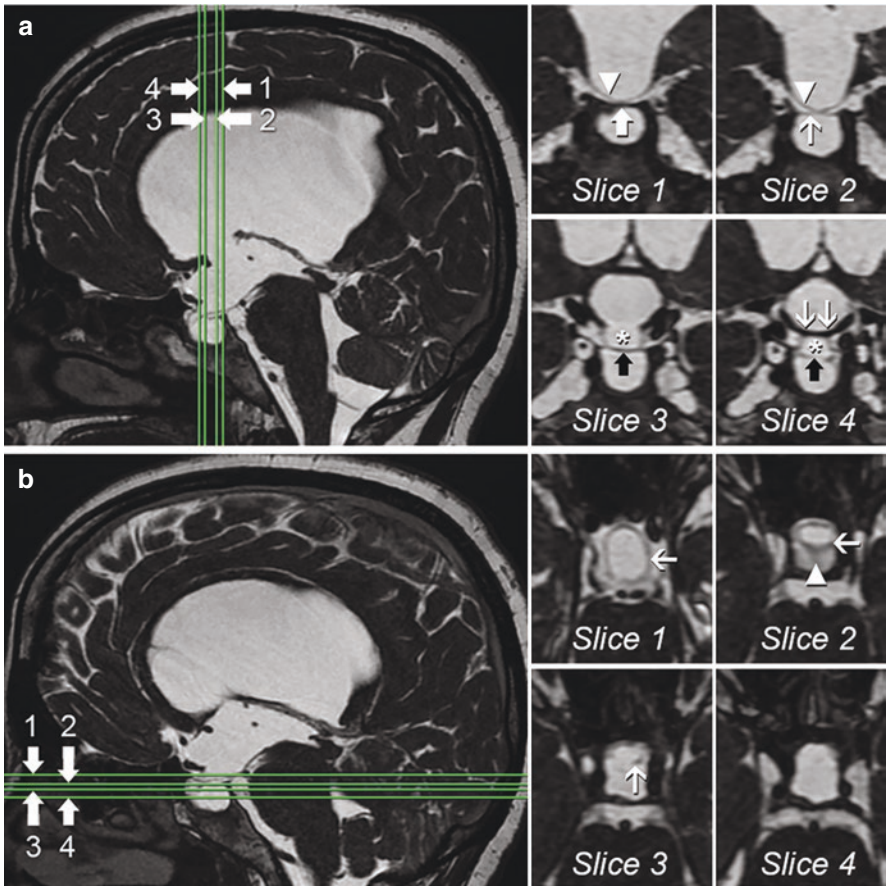


Fig. 7.10 Detailed imaging in PEIR. In (**a**), coronal reformatted images of the 3D FIESTA-C sequence are shown. At the level of the dorsum sellae (Slice 1), the dorsum sellae (arrow) and the third ventricle's floor (arrowhead) are seen. Through the diaphragma sellae (Slice 2), the diaphragma sellae (arrow) and the third ventricle's floor (arrowhead) can be distinguished. Through the plane of PEIR (Slice 3), the posterior wall (arrow) of the tubular-shaped PEIR (asterisk) is seen. Through the plane of PEIR and optic chiasm (Slice 4), the posterior wall (arrow) of the tubular-shaped PEIR (asterisk) is seen, while the anterior wall of the PEIR is attached to the optic chiasm (double arrows). In (**b**), axial reformatted images of the 3D FIESTA-C sequence are shown. At levels above (Slice 1), at (Slice 2), and just below (Slice 3) the level of the diaphragma sellae (arrowhead), the wall of the tubular PEIR (arrows) is seen. The cystic expansion within the sella is seen at a lower level within the sella (Slice 4)

embryogenetic role of the craniopharyngeal canal (CPC) in the pathogenesis of both conditions.

Accurate imaging diagnosis of PEIR and TSE is essential to avoid unnecessary treatment and complications. The condition can be confused with other cystic sellar lesions. While an endoscopic endonasal repair is obviously necessary in case of TSE, attempting remove a PEIR via a transsphenoidal approach is not only

contraindicated but also will result in postoperative CSF leak and risk of meningitis. Surgical treatment should target the associated disease that is per se indicated for surgery [82].

7.3.2 Granulomatous, Infectious, and Inflammatory Lesions

7.3.2.1 Rare Types of Hypophysitis

Hypophysitis is the collective diagnosis for inflammatory disorders of the pituitary gland and infundibulum. Most forms of hypophysitis are mediated by autoimmune reactions. However, some disorders classically considered hypophysitis are infections, e.g., with granulomatous infiltration, or neoplastic, e.g., Langerhans cell histiocytosis [88].

The diagnosis of hypophysitis is challenging and can be made on clinical and laboratory basis of unexplained hypopituitarism in addition to characteristic MRI findings including diffuse pituitary gland enlargement or thickening of the pituitary stalk or both. Pituitary stalk thickening, however, is only present in a subset of patients with hypophysitis [89].

Although scoring systems have been suggested to aid in distinguishing hypophysitis from pituitary adenomas [90], distinguishing hypophysitis from other pituitary lesions based on imaging alone is still at times very difficult [88].

Only in a minority of patients with presumed hypophysitis, a pituitary biopsy may be warranted to confirm the diagnosis if MRI findings are not completely convincing [89].

From the histopathological standpoint, primary hypophysitis falls into five subtypes including lymphocytic (LHP), granulomatous (GHP), xanthomatous (XHP), IgG4-related (IgG4rHP), and necrotizing (NHP) HP [89, 91–94]. On the other hand, secondary hypophysitis is an inflammatory condition of the pituitary gland, related to systemic pathologies, such as sarcoidosis (Fig. 7.11), Wegener granulomatosis, Langerhans cell histiocytosis, tuberculosis (Fig. 7.12), and connective tissue disorders [87].

7.3.2.2 Xanthogranuloma

Xanthomatous lesions of the sellar region have traditionally been divided into two separate categories: xanthomatous hypophysitis (XH) and xanthogranuloma (XG) of the sellar region [95]. Recent evidence suggests that xanthomatous lesions of the sella may represent a continuum and that both XH and XG in most cases can be linked to RCC leakage, rupture, or hemorrhage [95, 96]. Xanthogranulomas (Fig. 7.13) are granulomatous lesions that consist of an accumulation of foamy macrophages, multinucleated foreign body giant cells, cholesterol clefts, hemosiderin deposits, necrotic debris, lymphocytic infiltrates, and fibrous proliferation [97,

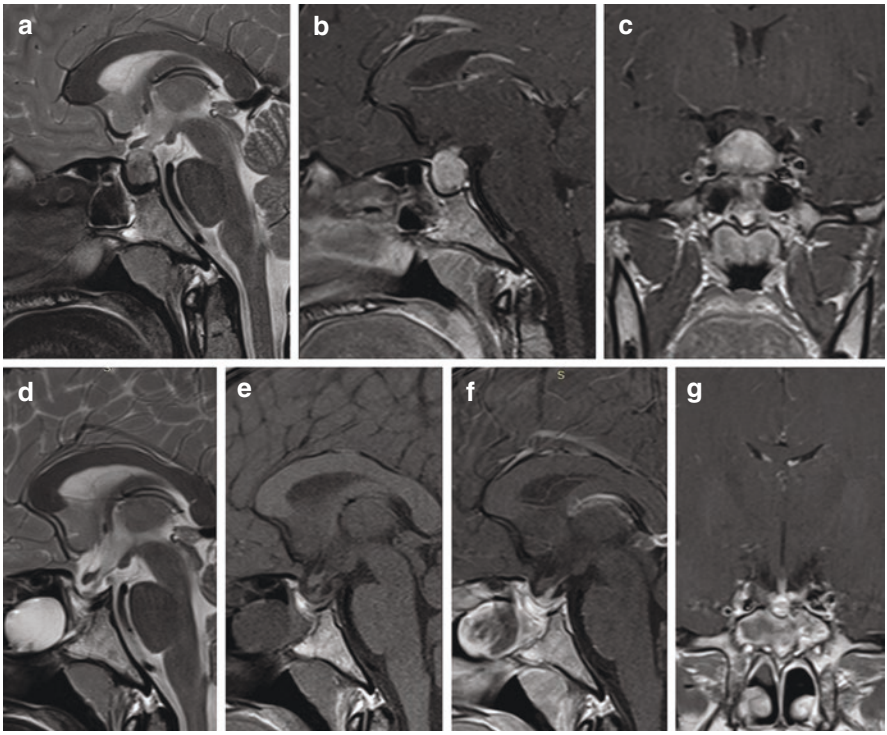


Fig. 7.11 Sarcoidosis of the pituitary gland. Pre- (**a**–**c**) and postoperative (**d**–**g**) MR images after endoscopic endonasal transsphenoidal resection of the lesion

98]. They present clinically with headache, weight loss, anorexia, nausea, fatigue, visual disturbances, and varying degrees of anterior pituitary hormonal deficiencies or panhypopituitarism. They may also lead to diabetes insipidus of central origin and obstructive hydrocephalus [99].

Paulus et al. were the first to suggest these lesions to be a separate entity from adamantinomatous craniopharyngiomas and reported their occurrence in adolescents and young adults, predominant intrasellar location, smaller lesion sizes, more severe endocrinological deficits, longer preoperative history, lower frequency of calcification and visual disturbances, and better resectability [98].

These lesions are difficult to diagnose on the basis of clinical or radiological presentations. There are no typical radiological characteristics or patterns for xanthogranuloma [99]. They present with variable MR signal intensities due to the intralesional inconsistent patterns of hemorrhage and calcification [100, 101]. Surgical resection is the present treatment for sellar xanthogranuloma, and gross total resection is considered as a gold standard treatment for this lesion. The endoscopic endonasal approach is favored over other approaches [102]. The endoscopic endonasal approach (Fig. 7.14) lends itself to the management of these lesions because the exposure and visualization of the supra- and parasellar areas are

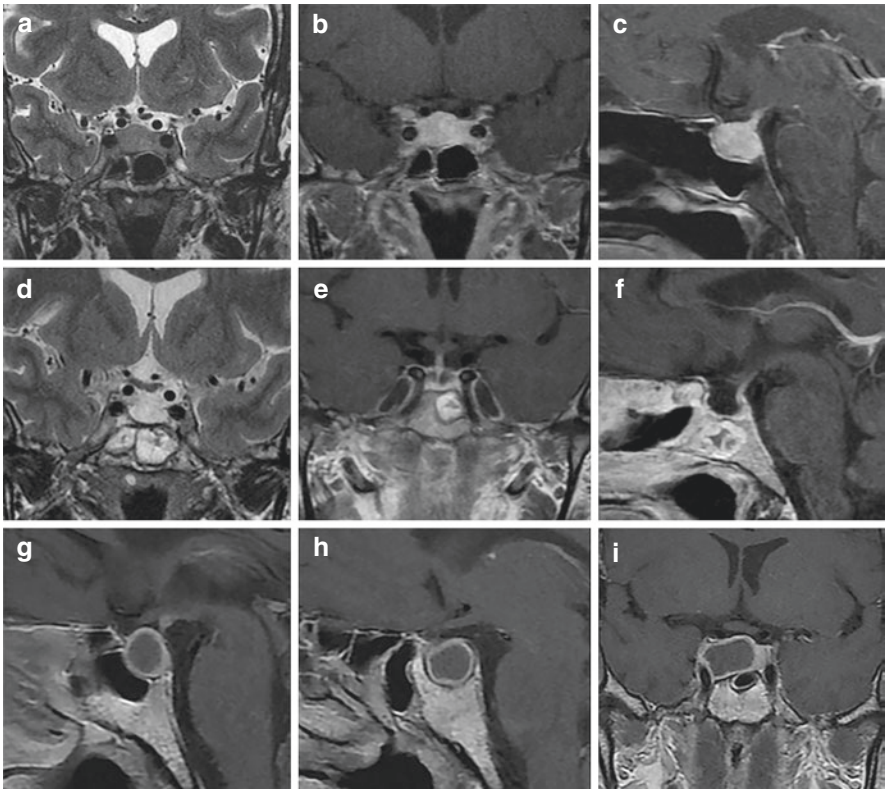


Fig. 7.12 Tuberculous hypophysitis and tuberculous pituitary abscess. Pre- (**a**–**c**) and postoperative (**d**–**f**) MR images in one case of tuberculous hypophysitis that was treated by endoscopic endonasal transsellar approach. (**g**–**i**) Preoperative MR images in another patient with pituitary tuberculous abscess

superior to a microscopic transsphenoidal approach and are less invasive than craniotomy [97].

7.3.2.3 Pituitary Abscess (PA)

Pituitary abscess (PA) is a rare disease that accounts for about 1% of operated sellar lesions [103, 104]. A secondary pituitary abscess may develop after surgery or on top of another pathology or sepsis [103–108]. On the other hand, a primary pituitary abscess occurs in previously healthy normal glands [103]. In these cases, the presentation is similar to other pituitary disorders making the diagnosis of pituitary abscess difficult and is usually made during or after drainage and evacuation of purulent contents of the lesion [104]. On MRI, pituitary abscess is very difficult to distinguish from other sellar cystic lesions and pituitary adenomas. Gadolinium injection is helpful as the absence of central enhancement suggests a fluid or necrotic

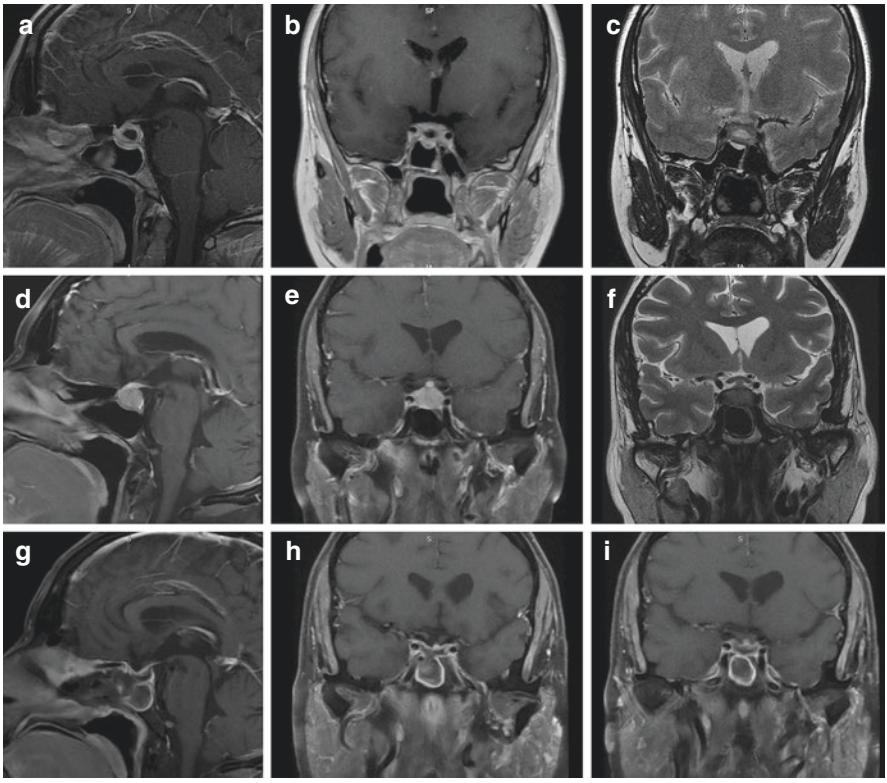


Fig. 7.13 Pituitary xanthogranuloma. First set of MR images (**a**–**c**) with a picture suggestive of hypophysitis. Repeat imaging (**d**–**f**) after medical treatment. (**g**–**i**) Postoperative MR images after endoscopic endonasal transsphenoidal approach

center [104]. Wang et al. described the majority of pituitary abscesses to be cystic or partially cystic, hypointense or isointense on T1-weighted imagine, and hyper- or isointense on T2-weighted imaging, with ring enhancement on post-gadolinium imaging [107]. Central hypo- or iso-intensity with internal heterogeneity and a surrounding hyperintense rim that enhances after contrast administration has also been described [107]. Diffusion restriction has also been demonstrated in pituitary abscesses [109, 110]. The aforementioned MR imaging findings are, however, also seen in sellar tumors undergoing central necrosis. Evidently, surgical evacuation via endoscopic transsphenoidal surgery and subsequent antibiotic therapy should immediately be undertaken.

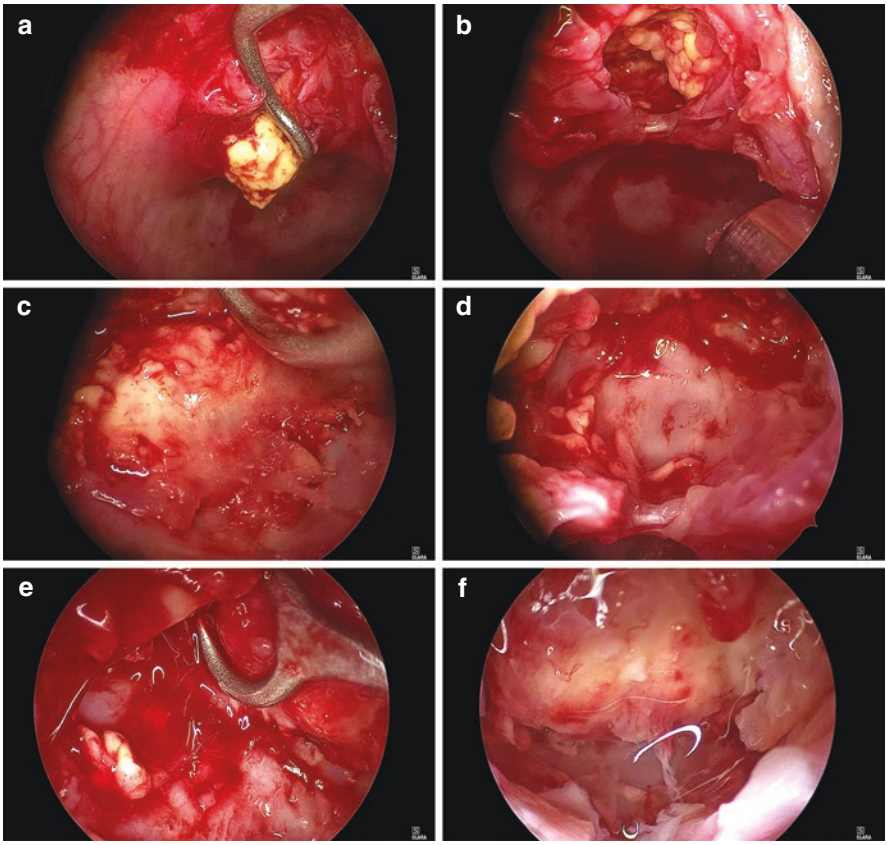


Fig. 7.14 Pituitary xanthogranuloma. Operative views (**a**–**f**) during endoscopic endonasal transsphenoidal approach

7.3.3 Neoplastic Lesions

7.3.3.1 Pituitary Blastoma

Pituitary blastoma is a distinctive and rare type of malignant neoplasms of the anterior hypophysis in infants described first by Scheithauer in 2008 [111, 112]. It appears to be an embryonal tumor originating in utero and is considered to be a pathognomonic feature of germline DICER1 mutation [22].

It presents in infants under 24 months of age and most frequently with Cushing's disease and elevated adrenocorticotropic hormone (ACTH) and occasionally with ophthalmoplegia, signs of increased intracranial pressure, diabetes insipidus, and thyrotropin deficit; Cushing's disease presenting in an infant suggests pituitary blastoma. The levels of other pituitary hormones can be variably affected. Excess serum cortisol causes severe and often lethal Cushing's disease [113–116].

Most cases have varying degrees of ACTH and growth hormone immunoreactivity which is an unusual combination of hormonal secretions in pituitary tumors. Immunohistochemical staining for other pituitary hormones are typically negative. The immunoprofile of pituitary blastoma includes keratins, galectin-3, annexin-1, scant GFAP and S100 expression in folliculostellate cells, and synaptophysin and chromogranin expression in secretory cells [113].

Pituitary blastoma is rare and has no specific imaging findings on MRI [20]. The tumor often appears as a large solid or partially cystic mass arising from the sella with extension into the hypothalamus. They may encompass the optic chiasm and/or cavernous sinuses [115].

Pituitary blastoma should be included in the differential diagnosis when an enhanced sellar and suprasellar mass with peripherally located cysts in the suprasellar portion is found in children; pituitary blastoma size usually ranges from 2 to 4 cm. They rarely calcify [22]. Radiological differential diagnosis includes pituitary adenoma, massive pituitary hyperplasia, hamartoma, and teratoma [111, 115].

The pathologic features include epithelial glands with rosette-like formations resembling immature Rathke's epithelium, small primitive-appearing cells with a blastoma-like appearance, and large secretory epithelial cells resembling adenohypophyseal cells expressing neuroendocrine markers such as ACTH in most cases and rarely GH [114, 115].

Germline *DICER1* mutation is the major and possibly sole predisposing genetic contributor for the development of a pituitary blastoma [111, 116, 117].

The diagnostic criteria of pituitary blastoma include (a) occurrence in infancy, (b) frequent endocrine hyperfunction (ACTH secretion with or without Cushing's disease), and (c) composition of Rathke's epithelium, folliculostellate cells, and secretory cells featuring ACTH and minor GH immunoexpression [112].

Total surgical resection of tumor cannot be achieved; however, gross total or near total resection is associated with better survival, and intensive focal radiotherapy is associated with some complication, particularly vasculopathy. Prognosis is unfavorable with almost half of these children dying shortly after the diagnosis [115, 116].

7.3.3.2 Uncommon Pituitary Adenomas (PitNETs)

Although a pituitary adenoma represents the most common pathology in the sellar region, some adenomas are quite rare. These subtypes include plurihormonal PIT-1-positive adenoma, acidophilic stem cell adenoma (ASCA), Crooke's cell adenoma, and COVID-19-induced pituitary adenoma apoplexy.

Plurihormonal PIT-1-positive adenoma (Fig. 7.15) is newly defined rare entity characterized by a monomorphous population of cells that express variable levels of GH, PRL, β-TSH, and α-SU. These adenomas have been previously called silent adenoma subtype 3 [118, 119]. Most plurihormonal PIT-1-positive adenomas are clinically silent, although some patients may present with acromegaly, hyperprolactinemia, or hyperthyroidism. Their recognition is of significance due to their

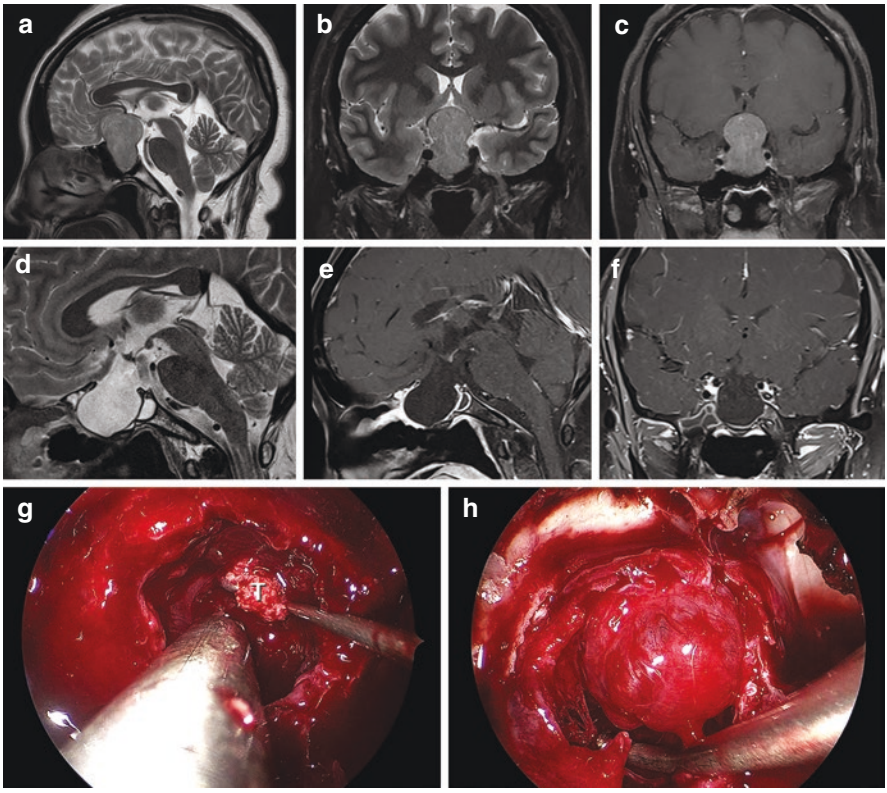


Fig. 7.15 Plurihormonal PIT-1-positive adenoma. Pre- (**a–c**) and postoperative (**d–f**) MR images. Intraoperative views demonstrating the tumor gross appearance (**g**) and its complete resection with full descent of the cistern (**h**)

intrinsic aggressive behavior with high degree of invasiveness, low rates of disease-free survival, and high tendency for recurrence [118].

Acidophilic stem cell adenoma (ASCA) is a rare subtype of lactotroph adenoma accounting for the minority of clinically diagnosed prolactin-secreting adenomas. These tumors are clinically characterized by variable degrees of hyperprolactinemia, with or without elevatation of growth hormone (GH) and insulin growth factor I (IGF-1) levels [118, 120]. The majority are rapidly growing macroadenomas with invasive features and more aggressive behavior than other lactotroph adenomas and a lower surgical cure rate [12]. The tumors are characterized by cells with acidophilic cytoplasm with focal oncocytic changes. Unlike lactotroph adenomas, ASCA display perinuclear, dot-like fibrous bodies confirmed by cytokeratin staining. A characteristic feature of ASCA is accumulation of mitochondria with sometimes dilated giant forms [118].

Crooke's cell adenoma (Fig. 7.16) is a very rare subtype of corticotroph pituitary adenomas that is known to be potentially aggressive [121, 122]. They contain Crooke's hyaline material in the cytoplasm of more than 50% of the tumor cells,

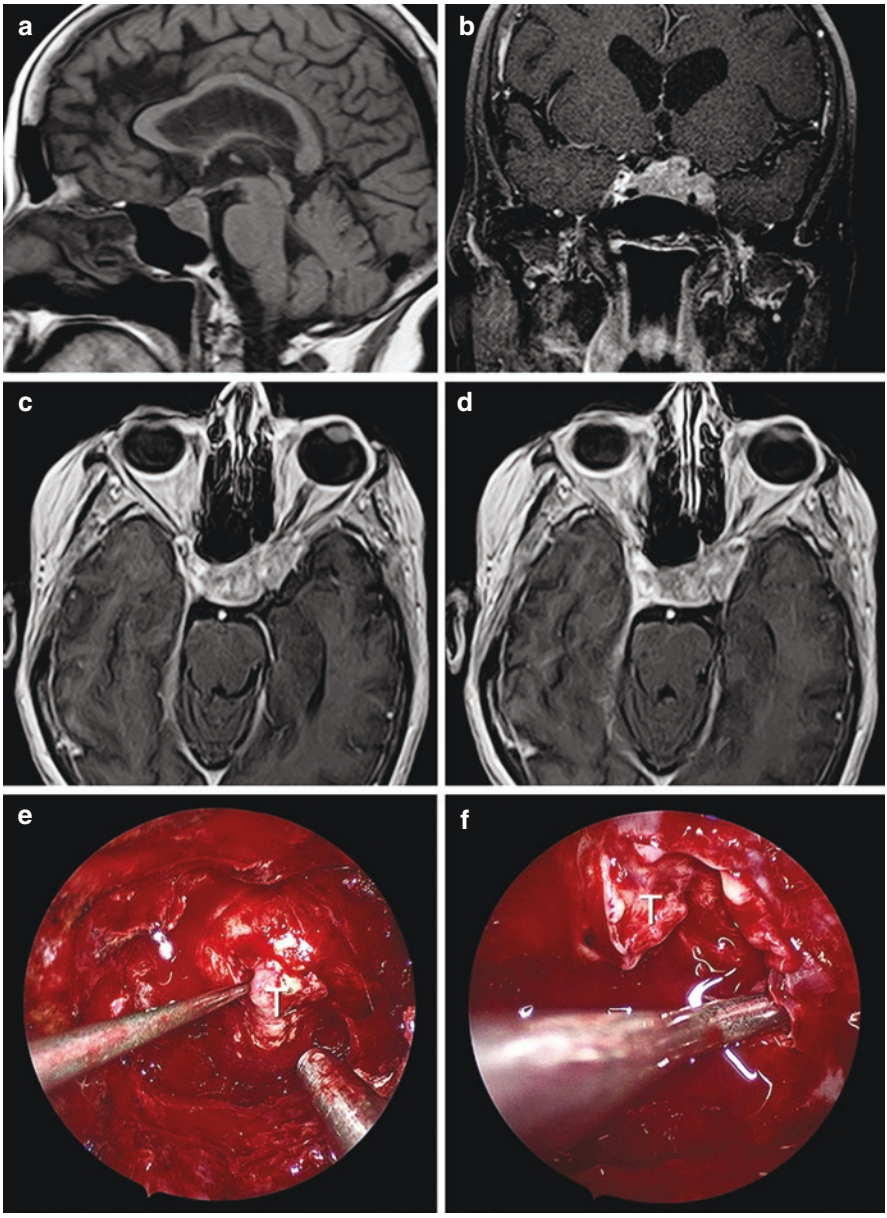


Fig. 7.16 Crooke's cell adenoma. Preoperative MR images (**a**–**d**) and intraoperative views during endoscopic endonasal transsphenoidal resection of the tumor (T), (**e**, **f**)

stain positively for adrenocorticotropic hormone (ACTH), and have variable degrees of clinical expression of Cushing's disease. The majority of Crooke's cell adenomas are macroadenomas with cavernous sinus invasion and suprasellar extension. They

tend to behave aggressively after surgical removal with more than 60% recurrence rate [123]. Endoscopic endonasal transsphenoidal excision is the first line of treatment. Other treatment modalities include fractionated radiation [121], GKRS [122], and temozolomide [124].

COVID-19-induced pituitary adenoma apoplexy is another rare pathology with few cases reported so far. Our group reported the fifth case of this entity (Fig. 7.17) [125]. We postulated that some pathophysiological mechanisms induced by COVID-19 can possibly lead to the development of pituitary apoplexy. In other words, the association between both conditions was not just a mere coincidence. Although the histopathological features of pituitary apoplexy associated with COVID-19 are similar to PA induced by other etiologies, future research may disclose unique pathological fingerprints of COVID-19 virus that explain its capability of inducing pituitary apoplexy [125].

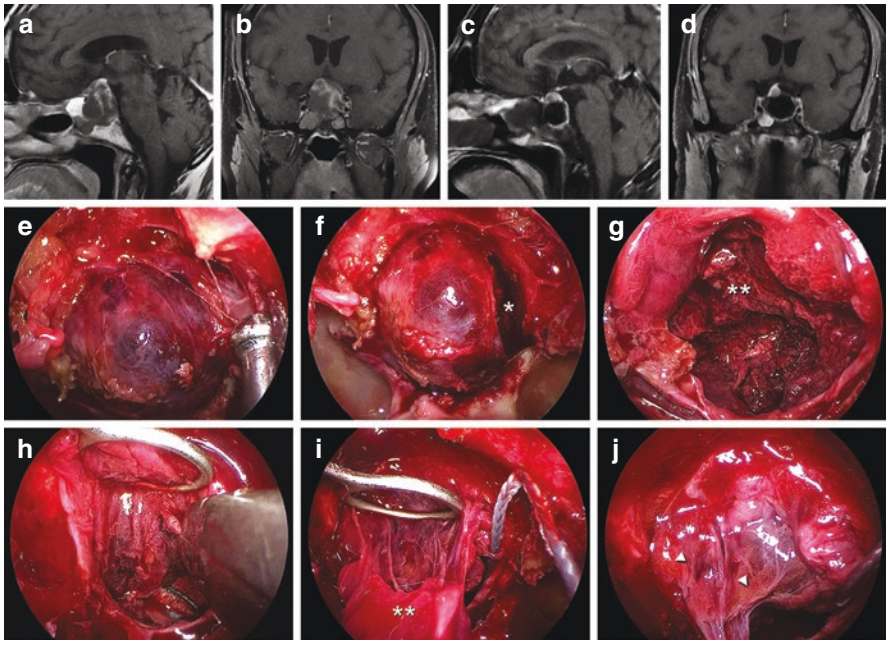


Fig. 7.17 MRI images in a case of COVID-19-associated pituitary apoplexy (**a**–**d**). Preoperative T1-weighted images reveal a large recurrent pituitary macroadenoma with minimal patchy enhancement after gadolinium injection (**a**, **b**). Postoperative T1-weighted images with contrast revealed near total excision of the adenoma (**c**, **d**). Intraoperative views during endoscopic endonasal transsphenoidal tumor excision (**e**–**j**). (**e**) Bluish discoloration of the dura caused by apoplexy of the underlying tumor is evident at the initial exposure. (**f**) Dark blood (asterisk) is seen upon initial dural opening. (**g**) View of the necrotic purple adenoma tissue being resected from within the sella. (**h**) A pituitary ring curette elevates the downward bulging cistern, and a pituitary rongeur is used to excise the superior part of the tumor. (**i**) The uppermost tumor components (double asterisks) have been separated from the arachnoid of the suprasellar cistern. (**j**) Final view after tumor resection. Note the fat from previous surgery (arrowheads) (Images and caption from Kamel et al. 2021 [125], reprinted with permission from Surgical Neurology International©, ScientificScholar)

7.3.3.3 Pituitary Carcinoma

Pituitary carcinomas are rare malignant neoplasms of adenohypophyseal cell origin and account for approximately 0.12% of adenohypophyseal tumors [126]. Pituitary carcinomas are defined by demonstration of craniospinal dissemination and/or systemic metastases [118]. They can be functioning or nonfunctioning. PCs more often are lactotroph or corticotroph tumors, producing prolactin (PRL) or adrenocorticotropic hormone (ACTH), respectively [126, 127]. They metastasize through postoperative drop metastasis, CSF spreading, or blood-borne sellar dura infiltration. They can disseminate to other organs like liver, bone, heart, ovaries, and lymph nodes [126]. The treatment of PCs remains multimodal and includes surgical resection, linear accelerator (LINAC)- and proton-beam-based fractionated radiotherapy, single-dose GKRS, chemotherapy, immunotherapy, and the use of other pharmacological agents targeting hormone production [126].

7.3.3.4 Pituitary Gland Metastasis

The pituitary fossa is an uncommon site for metastatic lesions; they occur in 1% of all pituitary lesions. The most common lesions that metastasize to the sellar region are from the breast and lung. Other primary sites include renal, prostate, and colon. Metastatic lesions to the pituitary fossa involve the neurohypophysis and posterior lobe more commonly than anterior lobe of the pituitary gland. More than 50% of the cases present with diabetes insipidus which rarely occurs with pituitary adenomas. Panhypopituitarism may also be present [128–131].

Metastasis to the pituitary gland should be in the differential diagnosis in a new sellar lesion in patients with known oncological history [132]. Imaging findings may offer some clues to whether a lesion is benign or metastatic, though a significant overlap exists making radiological examination nonspecific [133]. There is no standardized treatment for pituitary metastasis. Generally, surgery plays an important role for improving symptoms caused by the lesion, such as visual abnormalities. Total resection tends to be difficult to achieve due to high tumor vascularization and local invasiveness. EET seems very safe to achieve biopsy and symptomatic relief [134].

7.3.3.5 Ganglioglioma

Gangliogliomas of the sellar region are extremely rare and easily misdiagnosed lesions. Very few cases have been reported to originate from the pituitary [135], neurohypophysis [136, 137], and adenohypophysis [138]. Histologically, ganglioglioma is characterized by a mixture of atypical ganglion cells and neoplastic glial cells. Immunostaining of specific neural marker such as synaptophysin, chromogranin A, NFP, and GFAP can help with the diagnosis; GFAP is critical to diagnose glial component of ganglioglioma [135, 138].

The pathogenesis of ganglioglioma is still unclear. They possibly take origin from pluripotent progenitor cells or as a malformative neuronal lesion with glial component representing a transformed hamartomatous element [138]. In the case reported by Omofoye et al., an endoscopic endonasal transtuberculum sellae approach was used to expose the pituitary stalk. The tumor was 4 mm in diameter, firm, white, and integral to the right side of the enlarged pituitary stalk. The authors concluded that endoscopic endonasal approach for stalk gangliogliomas is a safe surgical approach to establish a tissue diagnosis [135]. In the case reported by Hong et al., the endoscopic endonasal transsphenoidal surgery revealed a dark red mass with firm texture and rich blood supply that was adherent to the diaphragma sellae. Subtotal resection was conducted because of the highly vascular nature of the tumor [138].

7.3.3.6 Gangliocytoma

The pituitary gangliocytoma is extremely rare benign brain tumor representing <1% sellar tumors. Ganglion cell tumors in sellar location are usually associated with functioning or nonfunctioning pituitary adenomas or pituitary cell hyperplasia. They are WHO grade I tumors [139–143]. The most frequent endocrine syndrome associated with gangliocytoma is acromegaly, followed by hyperprolactinemia, and less frequently Cushing's disease; around 85% of gangliocytoma cases are associated with a pituitary adenoma and are more prevalent in female patients [139, 140]. The diagnosis of these tumors is only possible after surgery as radiologically they are indistinguishable of sellar/suprasellar masses; the definitive diagnosis is determined by the histological and/or immunohistochemistry studies. They are well-differentiated slow-growing neuroepithelial tumors. The most common finding is the coexistence of neural tissue and GH-secreting pituitary adenoma [139, 140]. Three main theories have been proposed for the histogenesis of gangliocytoma: (1) primary gangliocytoma inducing an adenoma by paracrine secretion of hypothalamic hormonal stimulation, (2) transdifferentiation of adenomatous cells into neuronal/gangliocytic cells, and (3) a common progenitor/stem cell capable of transformation in the two cellular components [143].

Despite the rarity of mixed adenoma-gangliocytoma, this entity should be considered in the differential diagnosis of sellar masses. Complete tumor resection is considered curative [141, 143].

7.3.3.7 Neurocytoma

Sellar neurocytoma is an extremely rare form of extraventricular neurocytoma that originates from the hypothalamic-pituitary area. Very few cases have been reported in the literature [144].

Magnetic resonance imaging (MRI) remains the imaging modality of choice although no single feature is pathognomonic of extraventricular neurocytoma.

Speckled calcification may also be seen on CT scan. MRI may reveal an expanded sella with erosion of the sellar floor, sphenoid sinus invasion, and cavernous sinus involvement. The tumor may extend into the middle cranial fossa. The main radiological differential diagnosis includes pituitary adenoma, craniopharyngioma, sellar meningioma, and hypothalamic glioma [144].

Central neurocytomas generally have a favorable clinical prognosis, with surgical resection being the mainstay of treatment [144, 145].

Given the sellar and suprasellar location of the tumor, endoscopic transsphenoidal approach is considered safe for tumor resection and biopsy. In case of partial resection of tumor, adjuvant radiotherapy is important and essential because of the possibility of multiple remote disseminations in the spinal cord and drop metastasis in the initial route of surgery [144].

7.3.3.8 Sellar Ependymoma

Ependymomas are glial neoplasms that arise from the ependyma of cerebral ventricles, the spinal cord central canal, or cells of the terminal ventricle in the terminal filum. They may appear in the sellar region due to the presence of ependymal cells in the pituitary infundibulum [146]. It is thought that ependymomas that occur in the sella may be due to neoplastic transformation of either heterotopic ependymal cells or embryological remnants of the ependymal lining in the infundibular process [147].

Clinically, sellar ependymomas present with panhypopituitarism, headaches, and bitemporal hemianopia. Radiologically, both in CT and MRI, they resemble pituitary adenomas, where they have homogeneous enhancement. Parish et al. described the intraoperative findings to consist of yellowish-clear fluid from the suprasellar cyst and a grayish orange neoplasm, which was somewhat firmer than a typical pituitary tumor [147]. Surgery is the mainstay of treatment with the goal being gross total resection when feasible. EET is an excellent choice for ependymomas occurring in the sellar region.

7.3.3.9 Choroid Plexus Papilloma

Rare neuroectodermal tumors that develop from the choroid plexus epithelial cells tend to be in the ventricles. They account for less than 1% of all primary brain tumors. They frequently occur in the ventricular atrium and fourth ventricle. In adults, they generally occur in the fourth ventricle and very rarely do occur in extraventricular sites including the sellar region [148].

Radiologically, they vary from hypointense to hyperintense on both T1 and T2. They enhance after contrast. They resemble pituitary adenomas, and it is difficult to differentiate them based on neuroimaging alone [149]. They may show a cystic component where they appear similar to a craniopharyngioma. Clinically, they show symptoms of compression, with headaches and visual field defects being the most common. EET is an excellent approach for the resection of CPP [35].

7.3.3.10 Atypical Teratoid Rhabdoid Tumor

ATRTs are rare aggressive pediatric malignant tumors that generally occur in the cerebellum (60%). These tumors are extremely rare to occur in adults. When they do occur in adults, they are most common in the cerebral hemisphere followed by the sellar region [150]. Sellar ATRT occurs more frequently in females and presents with visual disturbances and headaches [151]. Radiologically, they are isointense on T1 and enhance with contrast. The diagnosis of AT/RT is based on tumor morphology and immunohistochemical features. They are characterized by rhabdoid cells and loss of INI-1/hSNF5 gene. Treatment requires surgery for which EET is adequate for diagnosis which is followed by chemotherapy and radiotherapy [152].

7.3.3.11 Neuroblastoma

Primary sellar neuroblastoma is an extremely rare tumor. They are easily misdiagnosed as non-secreting pituitary adenomas or other sellar masses. They mostly occur in women in the 4th decade of life [153–155]. They commonly present with visual field defects associated with hyperprolactinemia and gonadotrophin insufficiency [153]. No definite conclusion can be made by neuroimaging studies because of nonspecific CT and MRI characteristics. MRI brain shows a compressive sellar mass lesion with possible suprasellar or parasellar extension, sometimes calcified and tend to be fairly large. On MRI it is usually a differential diagnosis of non-secreting pituitary adenoma, tuberculum sellae, or diaphragma sellae meningiomas [153, 154].

Diagnosis is based on histopathological examination where immunohistochemical examination confirms the neuronal origin. These tumors are positive for neurofilaments, chromogranin, synaptophysin, and S100 protein and are negative for anterior pituitary hormones. Negative staining for EMA, GFAP, or TTF-1 is also present and excludes the possibility of posterior pituitary tumors [153–155].

The first line of treatment is surgical resection of the tumor. After diagnosis of primary sellar neuroblastomas is established, additional intra- and extracranial localizations should be sought, with particular attention to the nasal cavity. Adjuvant treatment to surgical resection might be proposed according to patient conditions and aggressive histopathological characteristics. Close endocrine follow-up and sellar MRI are required to assess for hypothalamo-pituitary dysfunction or tumor recurrence, and whole body imaging ^{123}I-MIBG scintigraphy might be useful as it is 90 to 95% sensitive for neuroblastomas [153–155].

7.3.3.12 Paraganglioma

Paragangliomas are neuroendocrine tumors the are extremely rare to occur in the sellar region. The pathogenesis is poorly understood but has been suggested that they may be derived from intrapituitary embryonic remnants of paraganglion cells

or may result from aberrant migration of the glossopharyngeal nerve to the pituitary [156]. A few cases have been documented in the literature [156–159].

The clinical features are attributed to mass effect and include headaches, visual disturbances, and endocrine abnormalities. They mimic pituitary adenomas and sellar meningiomas. They can be differentiated from adenomas on preoperative MR scan by their vascular flow voids and intense enhancement. SPECT is beneficial in differentiating paraganglioma and pituitary adenoma [158, 159].

Subtotal resections were often reported and followed by radiotherapy. Intraoperatively, these tumors are highly vascularized [41]. Chiasm decompression followed by adjuvant treatment or additional open surgery for further resection was the most common treatment course [160].

7.3.3.13 Purely Intrasellar Meningioma

Purely intrasellar meningiomas that arise from the dura covering the sella turcica are extremely rare [161]. The dura covering the sella turcica has an average surface area of about 6 cm^2 [162]. Preoperatively, they resemble a nonfunctioning pituitary macroadenoma both clinically and radiologically. Very few cases of pure intrasellar meningiomas arising from the floor of the sella turcica have also been published in the literature [163]. Purely intrasellar meningiomas present with symptoms of headaches, visual disturbances, and hormonal abnormalities due to the compression of the optic nerves and pituitary gland [164]. Generally, symptoms develop gradually. On MR scans, meningiomas enhance vividly after the administration of contrast. Hyperostosis of sella floor has been seen; however, it is difficult to identify the dural tail that is seen with meningiomas [165]. They can be approached via endoscopic endonasal transsphenoidal approach [166].

7.3.3.14 Chondrosarcoma

Chondrosarcomas are the most common intracranial malignant cartilage tumor. The occurrence of chondrosarcoma in the pituitary fossa is rare. There have been only a few cases in the literature. They typically present with symptoms attributed to mass effect. CT shows a calcified sella with destruction of the sellar bone. They are heterogeneously enhancing postcontrast administration [50]. The main differential diagnosis of chondrosarcomas is chordomas. They can be differentiated based on histopathology and immunohistochemistry with the prognosis of chondrosarcomas being more favorable than chordomas. Both chondrosarcomas and chordomas are positive for S-100 protein, though chondrosarcomas are negative for cytokeratin markers (CAM 5-2) and epithelial membrane antigens. Chondrosarcomas are divided into three histological grades: grade I (well differentiated), grade II (moderately differentiated), and grade III (poorly differentiated) [167].

Endoscopic endonasal transsphenoidal approach represents a good surgical option. The most important predictor of prognosis is the extent of resection. Since

they are relatively radioresistant, proton beam therapy is preferred over other adjuvant radiation modalities [50].

7.3.3.15 Lymphomas

Primary pituitary lymphoma may be indistinguishable from other sellar lesions as clinical and radiological features are typically nonspecific. At imaging one should consider lymphoma when evaluating an invasive sellar mass that is iso- to hypointense on T2-weighted images. The typical MR findings in primary CNS lymphoma are mass lesions that are iso- to hypointense on T1- and T2-weighted images; typically, contrast enhancement is intense and homogeneous in immunocompetent patients, but it is more likely to be inhomogeneous or ringlike in immunocompromised patients. Pituitary stalk thickening and perineural spread, however, are more distinctive for primary pituitary lymphoma. Pituitary lymphoma should be considered in the differential diagnosis of atypical masses of the sellar region in patients with hormonal dysfunction. Surgical biopsy, however, may prove invaluable in making a diagnosis in challenging cases [54, 168, 169].

7.3.3.16 Salivary Gland-Like Lesions

Microscopic aggregates of a few well-formed salivary acini, lined by low cuboidal epithelium, were first recognized to occur in the normal pituitary gland by Jakob Erdheim in 1940 and were subsequently referred to as "Erdheim rests" [170–172]. Schochet et al. demonstrated the presence of salivary gland tissue fragments in the posterior lobe of the pituitary in approximately 3% of 2300 examined human glands [173]. The presence of salivary gland rests in the sellar region that may probably be explained by the persistence of preexisting seromucous glands from the primitive oral cavity out of which the Rathke's pouch evaginates and migrates to fuse with an extension of the floor of the third ventricle, ultimately giving rise to the pituitary gland. Another explanation may be that during embryogenesis, mesenchymal components accompanying Rathke's pouch into the sella induce primitive pituitary epithelium to differentiate into salivary gland tissue [171]. Typically, these intrasellar ectopic salivary gland rests are localized in the vicinity of the neurohypophysis [174] or in the pars tuberalis and often communicate with the Rathke's cleft [175–177]. In very rare instances, they enlarge and become clinically significant because of mass effect with or without pituitary hormonal dysfunction [178, 179]. Symptomatic enlargement of ectopic SG rests may be in the form of (1) nonneoplastic enlarged ectopic salivary glands (NNESG) or (2) benign or malignant salivary tumors (ST) when neoplastic transformation takes place.

The NNSEGs mimic pituitary adenomas in clinical and radiological manifestations, making them difficult to diagnose before surgery [177]. In a recent systematic review by Feola et al. [174], 15 cases of NNESG have so far been reported. Most patients were younger than 30 years and were females (80%). The most frequent

complaints were headache, bitemporal hemianopia, blurred vision, decreased visual acuity, galactorrhea, and menstrual irregularities. Endocrine dysfunction was found in about 50% of cases and included mild elevation of serum PRL, GH deficiency, panhypopituitarism, and central hypothyroidism. Preoperative diabetes insipidus (DI) was present in 20% of cases [174]. The lesions may be intrasellar, may be typically localized in the posterior pituitary, or may display suprasellar extension [172]. On MR imaging, the lesions are characterized by variable signal intensities on T1- and T2-weighted images with inconstant contrast enhancement patterns and by frequent cystic components [174].

Benign or malignant salivary tumors (ST) mimic other nonfunctioning sellar lesions. One important consideration is that malignant salivary tumors derived from major or minor eutopic salivary glands may reach the sella through local invasion or blood spread; an extra-sellar origin should be excluded before diagnosing a primary malignancy originating from an intrasellar ectopic salivary gland rests [174, 180, 181]. Liu et al. [177] reported on 11 cases operated via a transsphenoidal approach. Intraoperatively, the 11 lesions were all cystic and filled with gelatinous fluid, which can be white, yellow, or gray material. Although intrasellar masses originating from ectopic salivary gland rests are rarely symptomatic, surgery is the preferred method to make a diagnosis and relieve symptoms.

7.3.3.17 Plasmacytoma of Sellar and Parasellar Region

Plasmacytoma or plasma cell tumor of sellar region is a neoplasm arising from monoclonal plasma cells present in the sella or surrounding bone, mucosa within the petrous or sphenoid bone or clivus. The most common plasma cell neoplasia is multiple myeloma; intrasellar plasmacytoma is a rare pituitary tumor, which originates from monoclonal plasma cells. Solitary sellar plasmacytomas are exceedingly rare, and their diagnosis is difficult because clinically and radiologically they mimic benign pituitary tumors [182–185].

The clinical manifestations reported in cases of intrasellar plasmacytoma include headache in 70% of patients. Many present with cranial nerve palsies, mainly involving nerves II–VI with symptoms of diplopia, visual loss, eye pain, ptosis, photophobia, facial numbness, and craniofacial pain. Some of the symptoms result from compression of the cranial nerves in the cavernous sinus (III, IV, V), whereas the anterior pituitary function is mostly intact [182, 183]. Hormonal abnormalities may, however, be present, among which hyperprolactinemia and sex hormone imbalance are the most frequent.

MRI typically reveals sellar mass with expansion into the sphenoid sinuses and suprasellar and parasellar areas, with features similar to other tumors of the region, especially pituitary adenomas and chordomas [185].

On MRI, differential diagnosis of sellar mass lesions such as pituitary adenomas and meningiomas and juxta-sellar mass lesions including craniopharyngioma, chordomas, germ cell tumors, and granulomatous lesions and metastases should be kept in mind [182, 183]. The definitive diagnosis of intrasellar plasmacytoma is made by

histopathological and immunohistochemical analysis of the sellar mass. In patients with diagnosis of intrasellar plasmacytoma, radiotherapy is the treatment of choice [182, 183]. Rapid tumor growth during follow-up and/or compression of the optic chiasm constitutes an indication for surgical decompression and exploration [183].

Endoscopic endonasal transsphenoidal surgical resection combined with postoperative RT might be the optimal initial treatment for the relief of mass effect and control of local recurrence. Long-term follow-up is essential because approximately 50% of cases of plasmacytoma without evidence of multiple myeloma at the diagnosis progress to overt multiple myeloma within 10 years, and 10% of them recur with a plasmacytoma, thereby underlining the importance of a correct and timely diagnosis in the management of these patients. The high-risk period for the development of disseminated disease has been suggested to be the first 3 years [182, 184].

7.4 The Uncommon Pathologies of the Clivus

There is a great variability of diseases involving the clivus; although not as vast as the sellar lesions, the list of these lesions is long. In Table 7.2 [186–210], the majority of these pathologies are enlisted. Such heterogeneity makes management of these lesions at times very challenging [211]. Managing clival lesions using a minimally invasive approach presents numerous therapeutic challenges because of the close proximity of surrounding critical structures, including the basilar artery, internal carotid artery, brain stem structures, and the cranial nerves [212]. Surgical management of clival lesions has evolved considerably over the last 2 decades, and endoscopic endonasal approaches are presently the standard of care for the vast majority of these lesions [212–215].

In the past, options for access included extended middle cranial approaches and transmaxillary, trans-oral, or even transcervical approaches; this was associated with significant morbidity and unnecessary resection of normal tissue. Even when access is achieved, the visibility often remained poor resulting in inadequate resection [216, 217]. The endoscopic endonasal transsphenoidal approach offers a direct route to the clivus. Detailed anatomical knowledge is required to appreciate the exact location and dimensions of surrounding vital structures. It is imperative to identify the internal carotid arteries, the cavernous sinus, and surrounding neurological structures before proceeding to tumor resection. The endoscope gives a clear visualization of these structures and therefore prevents potential complications and ensures optimal surgical results [218]. In the following paragraphs, a short overview of some of these rare lesions is presented.

Table 7.2 Examples of uncommon pathologies of the clivus

Amyloidoma	Schneider JR, Kwan K, Kulason KO, Faltings LJ, Colantonio S, Safir S, Loven T, Li JY, Black KS, Schaeffer BT, Eisenberg MB. Primary solitary retro-clival amyloidoma. Surg Neurol Int. 2018 May 15;9:100. doi: 10.4103/sni.sni_483_17
BNCT	Golden LD, Small JE. Benign notochordal lesions of the posterior clivus: retrospective review of prevalence and imaging characteristics. J Neuroimaging. 2014 May-Jun;24(3):245-9. doi: 10.1111/jon.12013
Brown tumor	Alwani MM, Monaco GN, Harmon SM, Nwosu OI, Vortmeyer AO, Payner TD, Ting J. A Systematic Review of Sellar and Parasellar Brown Tumors: An Analysis of Clinical, Diagnostic, and Management Profiles. World Neurosurg. 2019 Dec;132:e423-e429. doi: 10.1016/j.wneu.2019.08.126
Chondroblastoma	Liu J, Ahmadpour A, Bewley AF, Lechpammer M, Bobinski M, Shahlaie K. Chondroblastoma of the Clivus: Case Report and Review. J Neurol Surg Rep. 2015 Nov;76(2):e258-64. doi: 10.1055/s-0035-1564601
Ecchordosis physaliphora	Park HH, Lee KS, Ahn SJ, Suh SH, Hong CK. Ecchordosis physaliphora: typical and atypical radiologic features. Neurosurg Rev. 2017 Jan;40(1):87-94. doi: 10.1007/s10143-016-0753-4
Ectopic pituitary adenoma	Altafulla JJ, Prickett JT, Dupont G, Tubbs RS, Litvack Z. Ectopic Pituitary Adenoma Presenting as a Clival Mass. Cureus. 2019 Feb 28;11(2):e4158. doi: 10.7759/cureus.4158
Enchondroma	Velagapudi S, Alshammari SM, Velagapudi S. Maffucci Syndrome with Clival Enchondroma in Nasopharynx: A Case Report. Indian J Otolaryngol Head Neck Surg. 2019 Oct;71(Suppl 1):652-656. doi: 10.1007/s12070-018-1463-8
Fibromatosis	Zhang T, Xu L, Gu L, Chen W, Pandey G, Wang J, Wu Y. Calcifying fibrous tumor of the clivus presenting in an adult. Radiol Case Rep. 2019 Apr 10;14(6):771-774. doi: 10.1016/j.radcr.2019.03.028
Fibrous dysplasia	Heman-Ackah SE, Boyer H, Odland R. Clival fibrous dysplasia: Case series and review of the literature. Ear Nose Throat J. 2014 Dec;93(12):E4-9. doi: 10.1177/014556131409301202
Fungal mucocele	Zhang H, Jiang N, Lin X, Wanggou S, Olson JJ, Li X. Invasive sphenoid sinus aspergillosis mimicking sellar tumor: a report of 4 cases and systematic literature review. Chin Neurosurg J. 2020 Apr 9;6:10. doi: 10.1186/s41016-020-00187-0
Giant cell reparative granuloma	Nakamura H, Morisako H, Ohata H, Kuwae Y, Teranishi Y, Goto T. Pediatric giant cell reparative granuloma of the lower clivus: A case report and review of the literature. J Craniovertebr Junction Spine. 2021 Jan-Mar;12(1):86-90. doi: 10.4103/jcvjs.JCVJS_182_20
Giant cell tumor	Patibandla MR, Thotakura AK, Rao MN, Addagada GC, Nukavarapu MC, Panigrahi MK, Uppin S, Challa S, Dandamudi S. Clival giant cell tumor - A rare case report and review of literature with respect to current line of management. Asian J Neurosurg. 2017 Jan-Mar;12(1):78-81. doi: 10.4103/1793-5482.145112

Table 7.2 (continued)

Inflammatory	Tang H, Ding G, Xiong J, Zhu H, Hua L, Xie Q, Gong Y. Clivus Inflammatory Pseudotumor Associated with Immunoglobulin G4-Related Disease. World Neurosurg. 2018 Oct;118:71-74. doi: 10.1016/j.wneu.2018.06.174
Infrasellar craniopharyngioma	Yu X, Liu R, Wang Y, Wang H, Zhao H, Wu Z. Infrasellar craniopharyngioma. Clin Neurol Neurosurg. 2012 Feb;114(2):112-9. doi: 10.1016/j.clineuro.2011.09.010
Lipoma	Caranci F, Cirillo M, Piccolo D, Briganti G, Cicala D, Leone G, Briganti F. A rare case of intraosseous lipoma involving the sphenoclival region. Neuroradiol J. 2012 Dec 20;25(6):680-3. doi: 10.1177/197140091202500607
Lymphoma	Tsai VW, Rybak L, Espinosa J, Kuhn MJ, Kamel OW, Mathews F, Glatz FR. Primary B-cell lymphoma of the clivus: case report. Surg Neurol. 2002 Sep-Oct;58(3-4):246-50. doi: 10.1016/s0090-3019(02)00845-5
Meningioma	Kawaguchi A, Shin M, Hasegawa H, Shinya Y, Shojima M, Kondo K. Endoscopic extended transclival approach for lower clival meningioma. World Neurosurg. 2022 May 2:S1878-8750(22)00568-X. doi: 10.1016/j.wneu.2022.04.115
Metastasis	Mani A, Yadav P, Paliwal VK, Lal H. Isolated clival metastasis: a rare presentation of renal cell carcinoma. BMJ Case Rep. 2017 Aug 11;2017:bcr2017221570. doi: 10.1136/bcr-2017-221570
Osseous hemangioma	Moravan MJ, Petraglia AL, Almast J, Yeaney GA, Miller MC, Edward Vates G. Intraosseous hemangioma of the clivus: a case report and review of the literature. J Neurosurg Sci. 2012 Sep;56(3):255-9
Osteosarcoma	Mathkour M, Garces J, Beard B, Bartholomew A, Sulaiman OA, Ware ML. Primary High-Grade Osteosarcoma of the Clivus: A Case Report and Literature Review. World Neurosurg. 2016 May;89:730.e9-730.e13. doi: 10.1016/j.wneu.2016.01.054
Plasmacytoma	Goyal R, Gupta R, Radotra BD. Plasmacytoma of the clivus: a case report. Indian J Pathol Microbiol. 2006 Oct;49(4):568-70
PNET	Gupta S, Kumar A, Rangari KV, Mehrotra A, Pal L, Kumar R. Intracranial Peripheral Primitive Neuroectodermal Tumor Arising from the Clivus with Intracranial Metastasis in an Elderly Woman: Case Report and Review of the Literature. World Neurosurg. 2018 Nov;119:331-334. doi: 10.1016/j.wneu.2018.08.066
Rhabdomyosarcoma	Seiz M, Radek M, Buslei R, Kreutzer J, Hofmann B, Kottler U, Doerfler A, Nimsky C, Fahlbusch R. Alveolar rhabdomyosarcoma of the clivus with intrasellar expansion: Case report. Zentralbl Neurochir. 2006 Nov;67(4):219-22. doi: 10.1055/s-2006-942118
Sphenoid mucocele	Stavrakas M, Khalil HS, Tsetsos N, Muquit S. Clival Mucocele: A Rare Yet not Forgotten Pathology. Ear Nose Throat J. 2021 Jun 10:1455613211021176. doi: 10.1177/01455613211021176
Xanthoma	González-García L, Asenjo-García B, Bautista-Ojeda MD, Domínguez-Páez M, Romero-Moreno L, Martín-Gallego Á, Arráez-Sánchez MÁ. Endoscopic endonasal resection of clival xanthoma: case report and literature review. Neurosurg Rev. 2015 Oct;38(4):765-9. doi: 10.1007/s10143-015-0630-6

7.4.1 Isolated Sphenoid Fungal Mucocele

Isolated fungal granulomas originating within the sphenoid sinus are extremely rare in immunocompetent patients (Figs. 7.18, 7.19, 7.20, and 7.21) [219]. Its incidence ranges between 1% and 3% among patients with rhinosinusitis [219, 220]. The condition may be underdiagnosed because of its subtle onset, typically presenting with nonspecific facial pain or headaches. They may initially present when complications have already taken place [221, 222]. Complications in isolated fungal sphenoiditis occur because of the anatomical characteristics of the sphenoid sinus where important nearby structures including the pituitary gland, cavernous sinuses, internal carotids, and several cranial nerves are involved [222]. The combined presence of a fungal ball and mucocele together is rarely reported [223]. Once diagnosed, endoscopic endonasal surgery should be undertaken to prevent further compromise of neurological or ophthalmological function.

7.4.2 Giant Cell Tumor of the Clivus

Giant cell tumor of the clivus (Figs. 7.22, 7.23, and 7.24) is a rare entity. In an analysis of the 104 published reports of skull base GCTs, only 12 cases of clival giant cell tumor were encountered [224]. Given its critical location and the aggressive and locally destructive nature of the lesion, clival GCTs often present with significant neurological sequelae. As the disease progresses, more cranial nerves are involved.

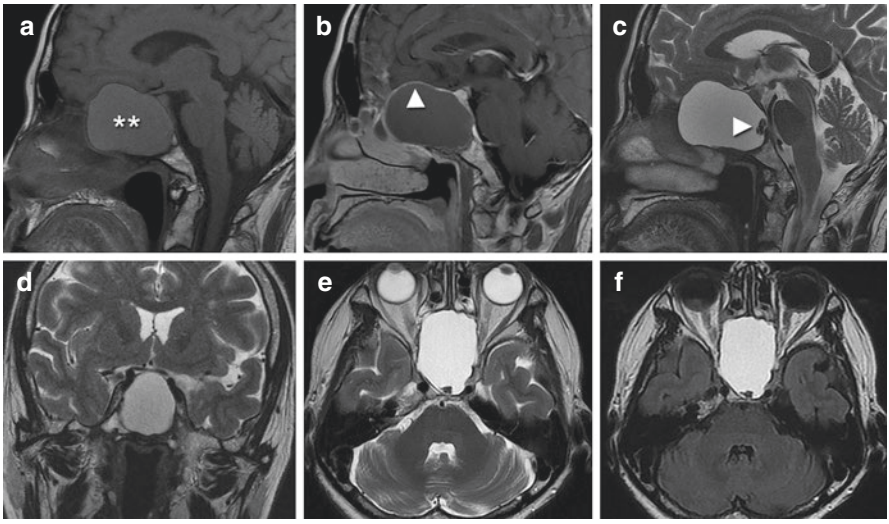


Fig. 7.18 Isolated giant fungal mucocele of the sphenoid sinus. Preoperative MR images (**a**–**f**)

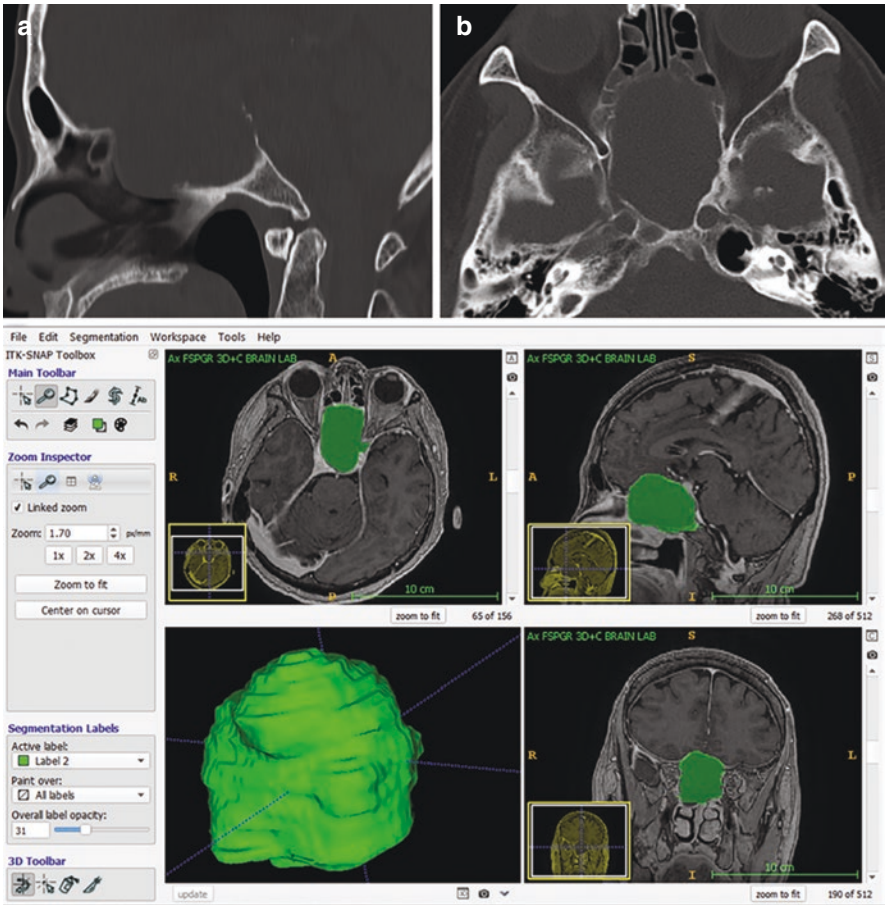


Fig. 7.19 Isolated giant fungal mucocele of the sphenoid sinus. Preoperative CT images (**a**, **b**) and preoperative volumetric analysis using ITK-Snap software

Patients present with headaches, visual field defects, diplopia, ophthalmoplegia, and deafness [225].

Radiologically, these tumors can be difficult to diagnose [226]. CT features usually show destructive and lytic lesions of the bony region involved. The nature of GCTs might vary significantly ranging from expansile to lytic lesions, and the majority show high vascularity. In general, GCTs appear to be isointense on T1-weighted MRI and hypointense on T2- and diffusion-weighted MRI [227].

Histopathological evaluation of GCTs demonstrates osteoclast-like giant cells scattered through the lesion with ovoid or spindle mononuclear stromal cells [228]. The osteolytic activity of the osteoclast-like cells makes the tumor locally destructive to the surrounding bone even though it is classified as a benign tumor [229, 230].

Histone 3.3 mutations of the *H3F3A* gene were recently described in GCT of bone and may prove useful in clarifying diagnosis in challenging cases [231].

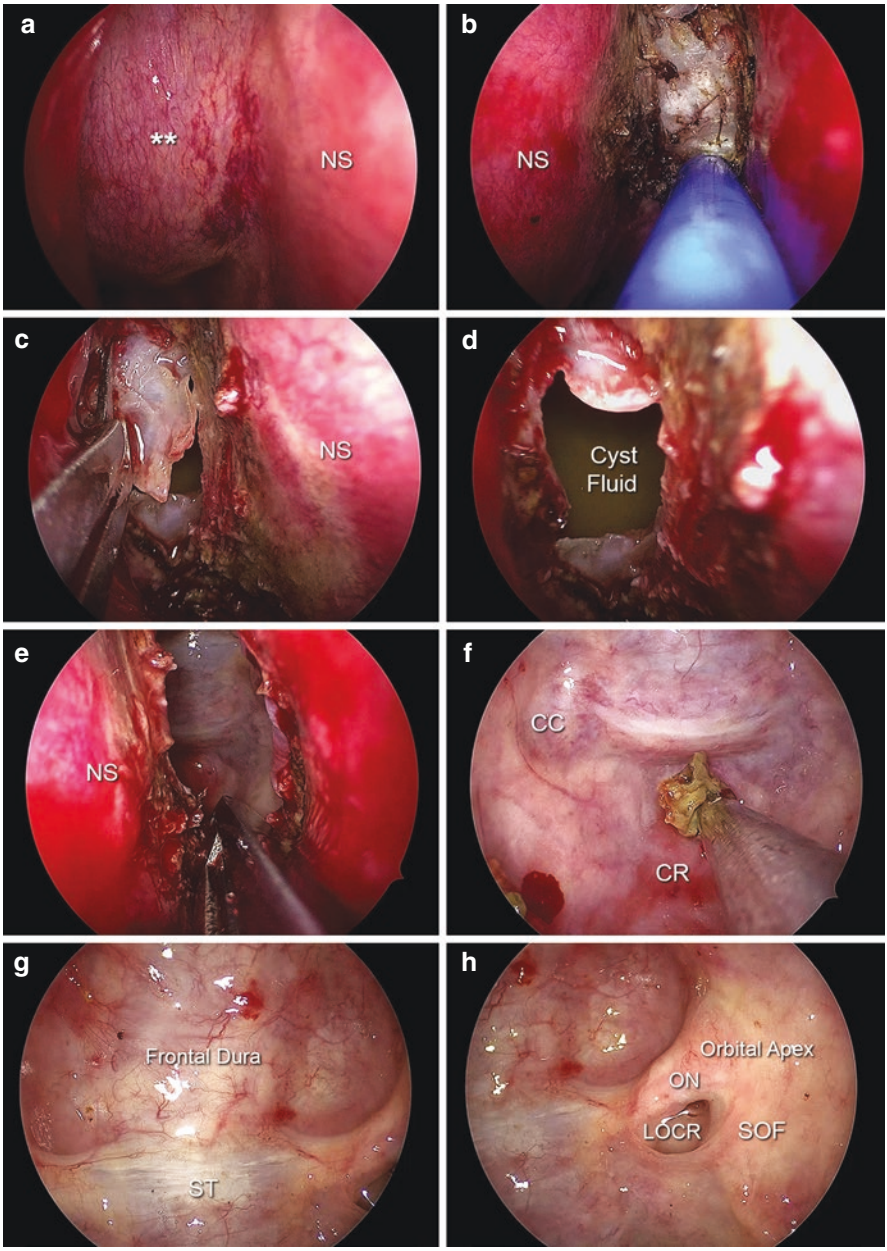


Fig. 7.20 Isolated giant fungal mucocele of the sphenoid sinus. Operative views (**a**–**h**) during cyst drainage via an endoscopic endonasal transsphenoidal approach. Note the fungus ball seen and removed after cyst fluid drainage (**f**). NS, nasal septum; double asterisks, cyst bulge into the nasal cavity; arrowheads, infiltrated clival bone; CC, cavernous carotids; CR, clival recess. ON, optic nerve; SOF, superior orbital fissure; LOCR, lateral optico-carotid recess; ST, sella turcica

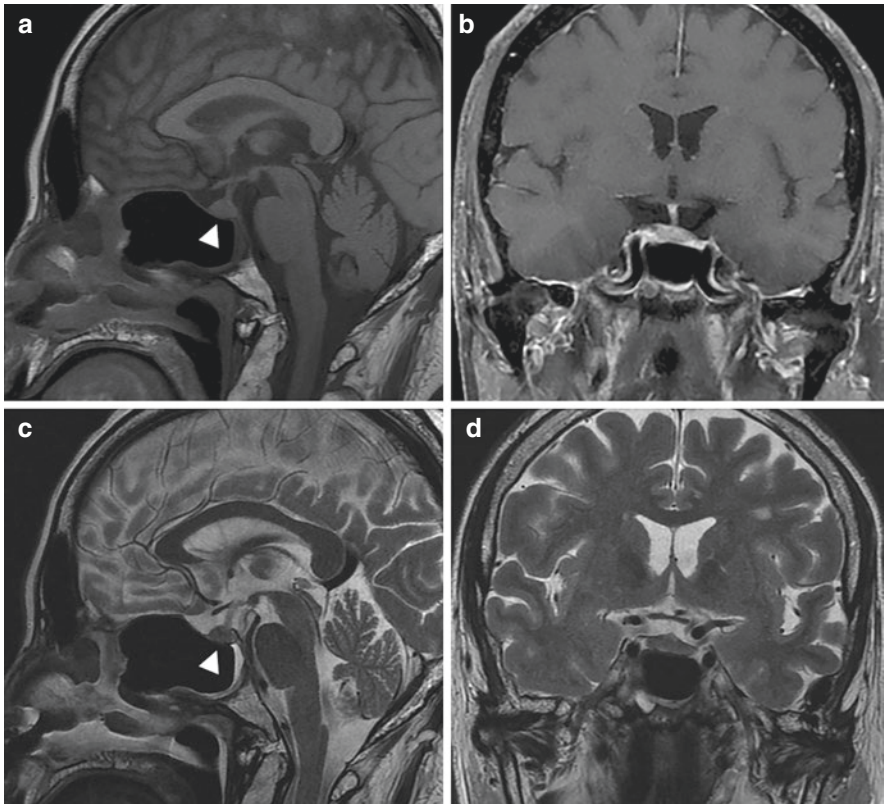


Fig. 7.21 Isolated giant fungal mucocele of the sphenoid sinus. Postoperative MRI images (**a**–**d**). Note the decompressed pituitary gland (**a**, **c**; arrowheads) and the decompressed optic chiasm in (**d**)

Although the giant cells are a significant part of this tumor, the stromal cells constitute the actual neoplastic component [224]. Neoplastic stromal cells of GCT overexpress RANKL (receptor activator of nuclear factor kappa-B ligand) and activate osteoclast-like giant cells [230]. The high expression of RANKL by the stromal cells within clival GCT offers an explanation as of why denosumab, a monoclonal antibody directed against RANKL, is effective in treating these lesions [224, 232, 233].

The aim of treatment of clival GCTs is total resection; unfortunately, this is rarely achieved due to the nature of the disease and its close proximity to vital structures. Endoscopic endonasal transclival approach allows better visualization of the clivus and tumor [234, 235]. Adjuvant radiotherapy is considered as a standard of care in the management of GCTs in most institutions nowadays although the concern that radiation may initiate sarcomatous transformation of the GCTs still remains [234].

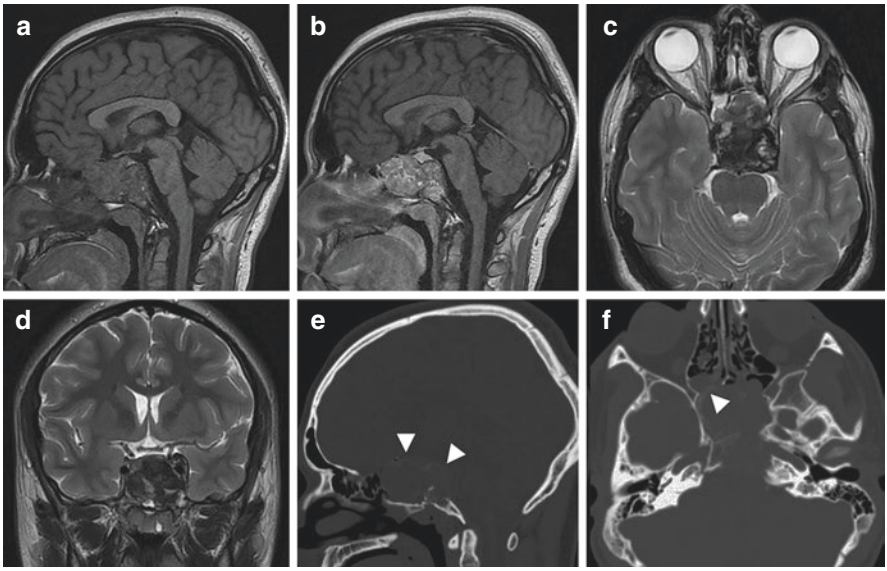


Fig. 7.22 Clival fibrous dysplasia. MR images (**a**–**d**) demonstrating a large clival mass. Note the bony expansion in the CT images (**e**, **f**; arrowheads)

7.4.3 Clival Fibrous Dysplasia

Fibrous dysplasia is a developmental disorder caused by abnormal proliferation of fibroblasts resulting in replacement of normal cancellous bone by structurally weak, immature osseous tissue. It is extremely rare to have fibrous dysplasia in the clivus (Fig. 7.25); only a few cases of fibrous dysplasia have been reported in the literature [236].

Postzygotic activating mutations in *GNAS* are the driving force behind the pathophysiology of fibrous dysplasia. *GNAS* encodes the α-subunit of the G_s stimulatory protein, and two potential mutations can occur resulting in interruption of the intrinsic GTPase activity of $G_s\alpha$. This leads to receptor activation and consequent inappropriate cyclic AMP-mediated signaling. The ultimate result is replacement of the normal cancellous bone and its marrow by proliferating bone marrow stromal cells, which form structurally weak discrete fibro-osseous lesions [237].

Fibrous dysplasia is a benign lesion, which progression usually halts after adolescence. In some instances, it continues to grow into adulthood, with males being more affected than females [238].

Although usually detected incidentally, clinical symptoms and signs may occur due to progressive bone deformation and depend on the location and extension of the abnormality. In the clival region, the symptoms are usually due to cranial nerve deficits (visual, auditory, olfactory, facial) caused by compression of the nerves in the narrow bony canals and skull base foramina. CT and MRI both offer valuable information for clival FD [239].

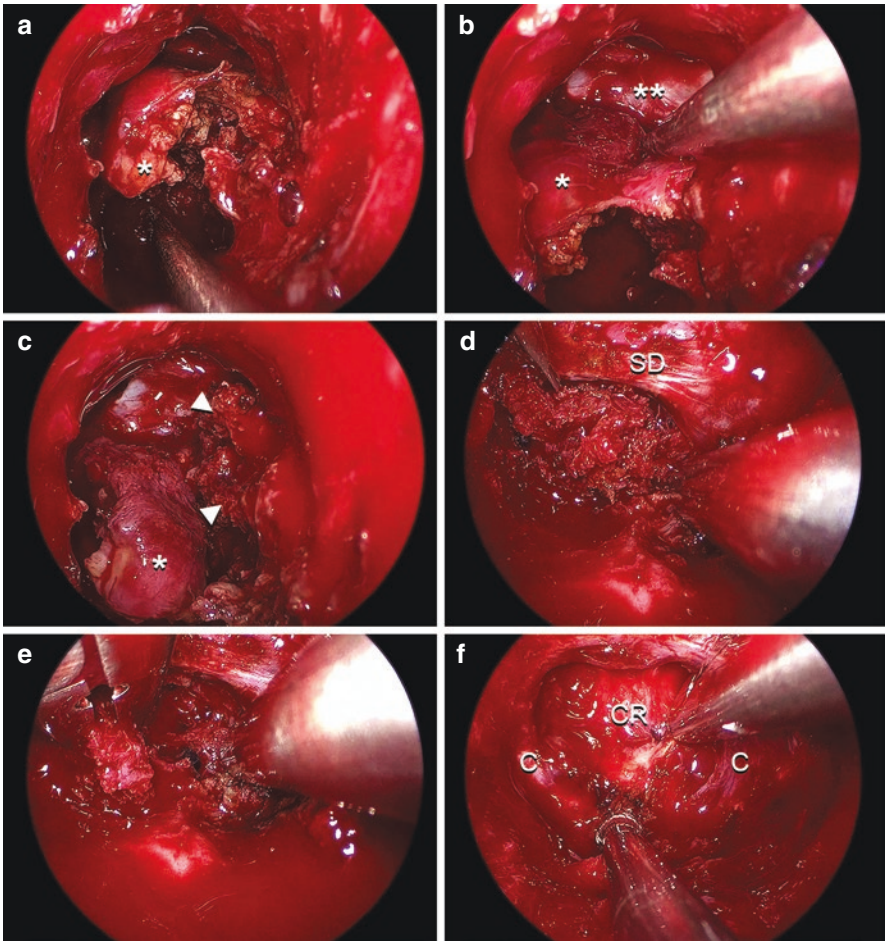


Fig. 7.23 Giant cell tumor of the clivus. Operative views (**a**–**f**) during tumor resection via an endoscopic endonasal transclival approach. SD, sellar dura; double asterisks, posterior fossa dura; arrowheads, infiltrated clival bone; C, paraclival carotids; asterisk, tumor; CR, clival recess

Fibrous dysplasia of clivus should be considered in any isolated clival lesion. Its diagnosis relies on imaging and histopathology. High-resolution CT is of significant importance in the evaluation [240]. A ground glass opacity, ballooning and expansion of the affected bone, and thinning of the cortex are the hallmark of fibrous dysplasia [241]. MR imaging of the clivus tends to show hypointensity in both T1 and T2 [194]. The signal intensity in T1-weighted images is directly related to the ratio of the fibrous tissue versus mineralized matrix, a lower signal intensity in highly mineralized matrix, whereas high fibrous content displays intermediate signal intensity. T2-weighted images show variable intensity depending on the metabolic activity of the lesions [239].

In asymptomatic patients, observation with serial CT scans is performed. In symptomatic patients, a standard endonasal endoscopic approach should be performed.

7.4.4 Metastatic Clival Lesions

Isolated metastatic lesions to the clivus are very rare (Figs. 7.26, 7.27, and 7.28), with less than 60 cases reported in the literature. The most common primary tumors are from prostate and renal cancer [242]. The most common symptom seen in patients with clival metastasis is diplopia due to sixth nerve palsy [243]. Regarding imaging, MR scanning remains the most sensitive in detecting clival lesions. Most metastatic lesions to the clivus are treated via surgery followed by radiotherapy or radiotherapy alone [242].

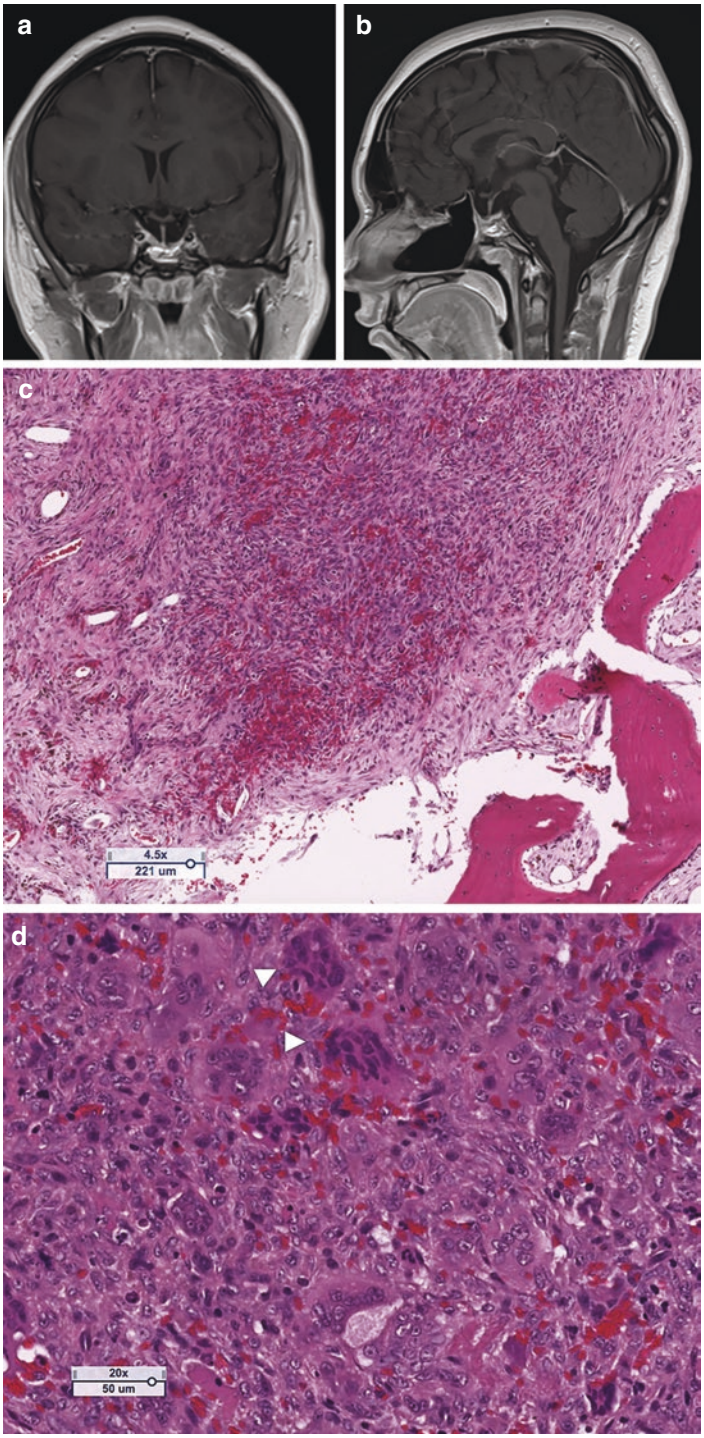
7.4.5 Benign Notochordal Cell Tumors (BNCTs) of the Clivus

Benign notochordal cell tumors (BNCTs) of the clivus are intraosseous benign lesions of notochordal cell origin [244, 245]. They represent notochordal rests that are most frequently found in the retroclival prepontine cistern [243]. A diagnosis of BNCT is favored over chordomas if the lesion has low proliferation index of Mib-1 antibody to Ki-67, less than 2 cm in size, and a gelatinous consistency with no bony invasion [187]. On imaging, BNCTs show no cortical or soft tissue invasion; they do not enhance with contrast and show low intensity on T1, with intermediate to high intensity on T2 [246].

BNCT contains cells with abundant vacuolated clear to eosinophilic cytoplasm, and the immunoprofile is identical to chordoma. BNCT has well-delineated borders and is confined to the bone without cortical permeation. They lack a lobular architecture, necrosis, conspicuous mitoses, and high-grade nuclei. Additionally, BNCT lacks extracellular myxoid matrix [247, 248]. BNCTs were identified in chordomas resected in the sacrococcygeal region, suggesting that they may represent a precursor lesion [249].

Although most cases are asymptomatic and treated with observation, one recent report documented two cases with histological features of BNCT and concomitant chordoma involving the clivus. In both cases, the clival lesions were incidentally

Fig. 7.24 Giant cell tumor of the clivus. Postoperative MRI with contrast in coronal (**a**) and sagittal (**b**) planes revealed near total resection of the tumor. Histopathological examination reveals a relatively well-circumscribed tumor rimmed by a shell of bone (**c**). Higher magnification (**d**) demonstrates a hypercellular tumor composed of giant multinucleated osteoclast-like cells (arrowheads) growing in sheets and mononuclear cells in a richly vascular background. There is no evidence of mitosis or necrosis


a
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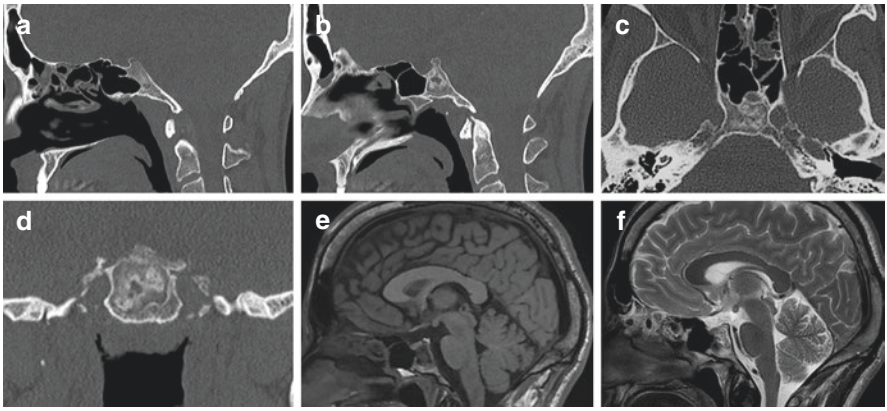

Fig. 7.25 Clival fibrous dysplasia. CT (**a**–**d**) and MR (**e**–**f**)

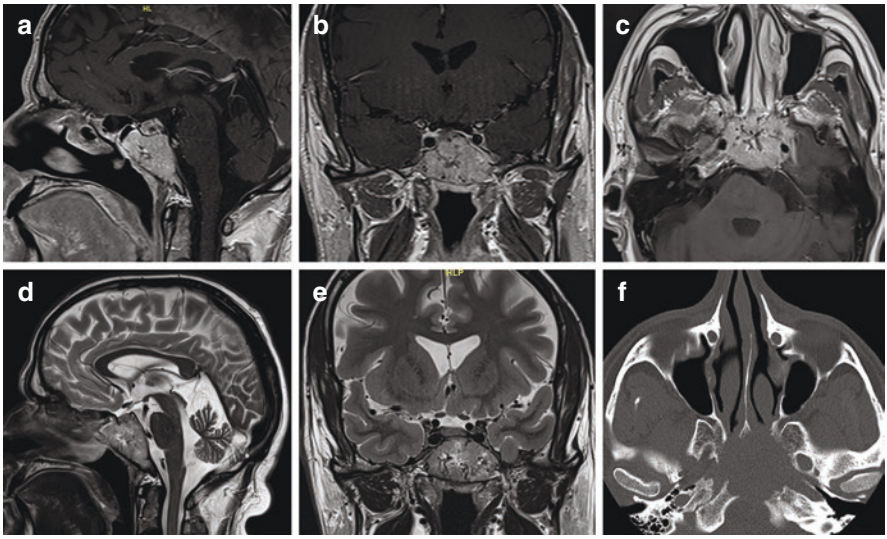

Fig. 7.26 Clival metastasis from a renal clear cell carcinoma. Preoperative T1-weighted MRI with contrast (**a**–**c**) and T2-weighted images (**d**, **e**) reveal a large clival tumor with notable vascular voids. Significant bone erosion is seen on CT (**f**)

discovered. They were both T2 hyperintense and T1 hypointense and non-enhancing in postcontrast imaging. Histologically, the tumor demonstrated areas of classic chordoma and a distinct intraosseous BNCT component. Surgical removal of the

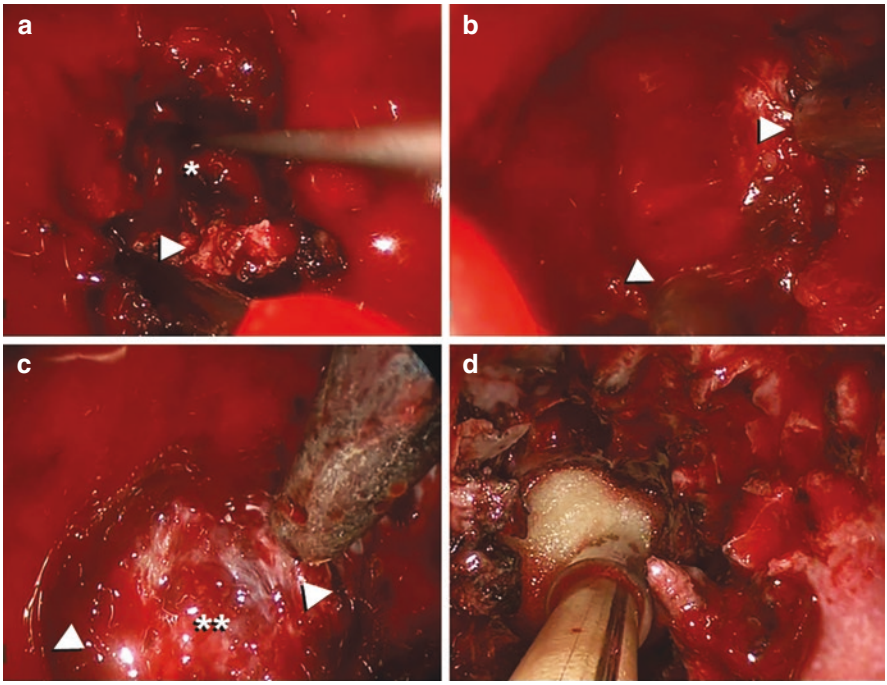

Fig. 7.27 Intraoperative views during endoscopic endonasal transclival approach for resection of a clival metastasis from a renal clear cell carcinoma. (**a**) The tumor is centrally debulked within the clival recess (asterisk), and peripheral parts of the tumor are seen and taken out (arrowhead). (**b**) The high vascularity and significant bleeding required two suction techniques (arrowheads) for visualization of the tumor and normal structures. (**c**) View after tumor resection was completed. Note the cleared posterior fossa dura (double asterisks) seen between the two paraclival carotid arteries (arrowheads). (**d**) Final hemostasis in the tumor bed using Floseal®

lesion was performed through an endoscopic transsphenoidal approach. Histological analysis revealed areas of BNCT with typical features of chordoma. These cases document histologically concomitant BNCT and chordoma involving the clivus, suggesting that the BNCT component may be a precursor of chordoma [250].

Our group had a similar experience in one case that we treated recently (Figs. 7.29, 7.30, and 7.31). It is however still unclear whether these cases are truly different from chordomas with typical features (Fig. 7.32).

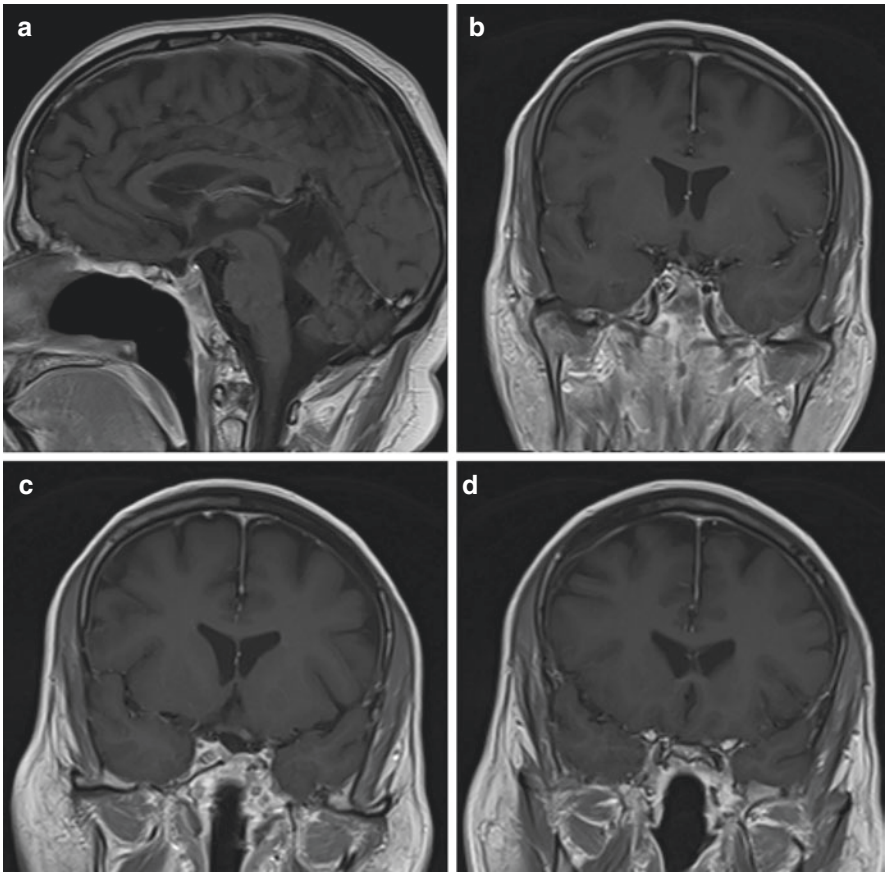

Fig. 7.28 Clival metastasis from a renal clear cell carcinoma. Postoperative T1-weighted MRI with contrast in sagittal (**a**) serial coronal (**b–d**) images demonstrating near total resection of the tumor

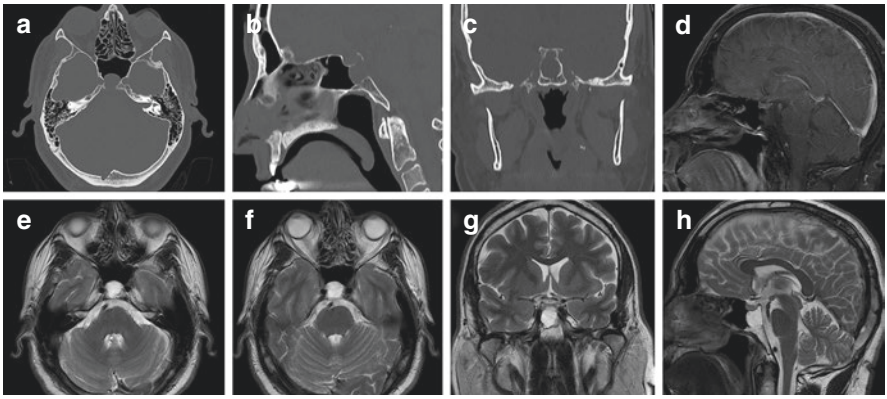

Fig. 7.29 Clival chordoma with BNCT. Preoperative CT images (**a**–**c**) demonstrating the lesion. Preoperative sagittal T1-weighted MRI with contrast revealed no enhancement of the lesion (**d**). Hyperintense signal of the lesion is seen on T2-weighted images (**e**–**h**)

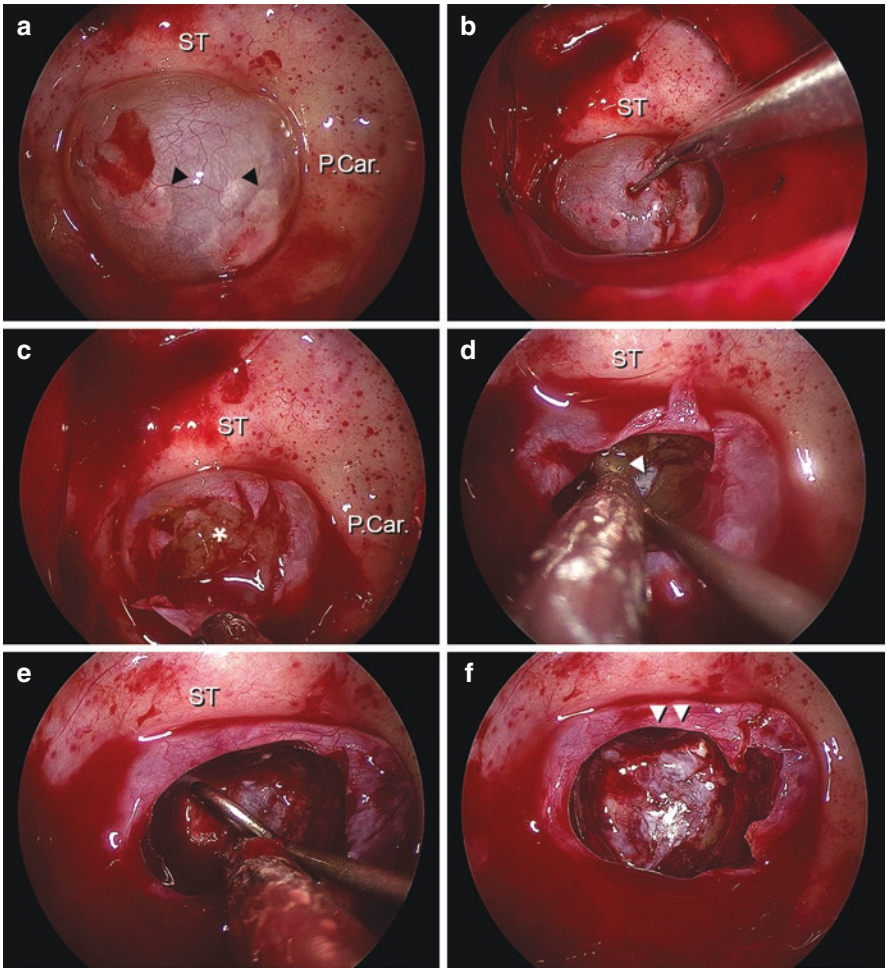

Fig. 7.30 Clival chordoma with BNCT. Operative views (**a**–**f**) during endoscopic endonasal transclival approach. ST, sella turcica; black arrowheads, cortical clival bone; P. Car., paraclival carotids; asterisk, tumor; white arrowhead, posterior fossa dura; double arrowhead, sphenoid sinus mucosa

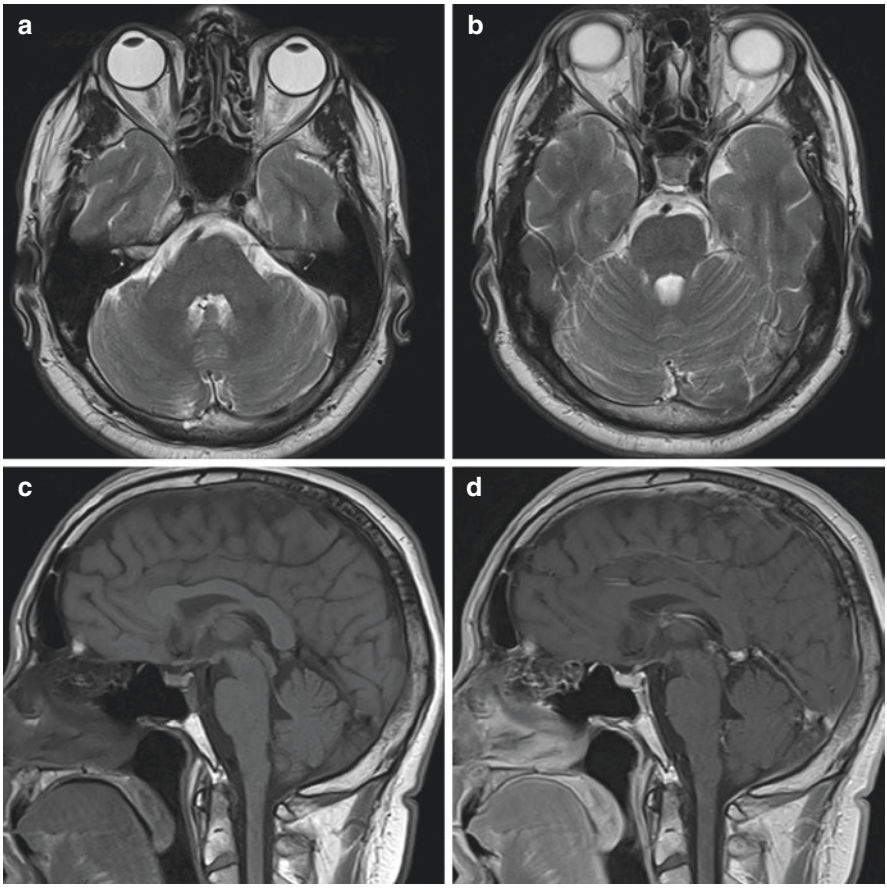

Fig. 7.31 Clival chordoma with BNCT. Postoperative axial T2 weighted (**a**, **b**) and sagittal T1 without (**c**) and with (**d**) contrast

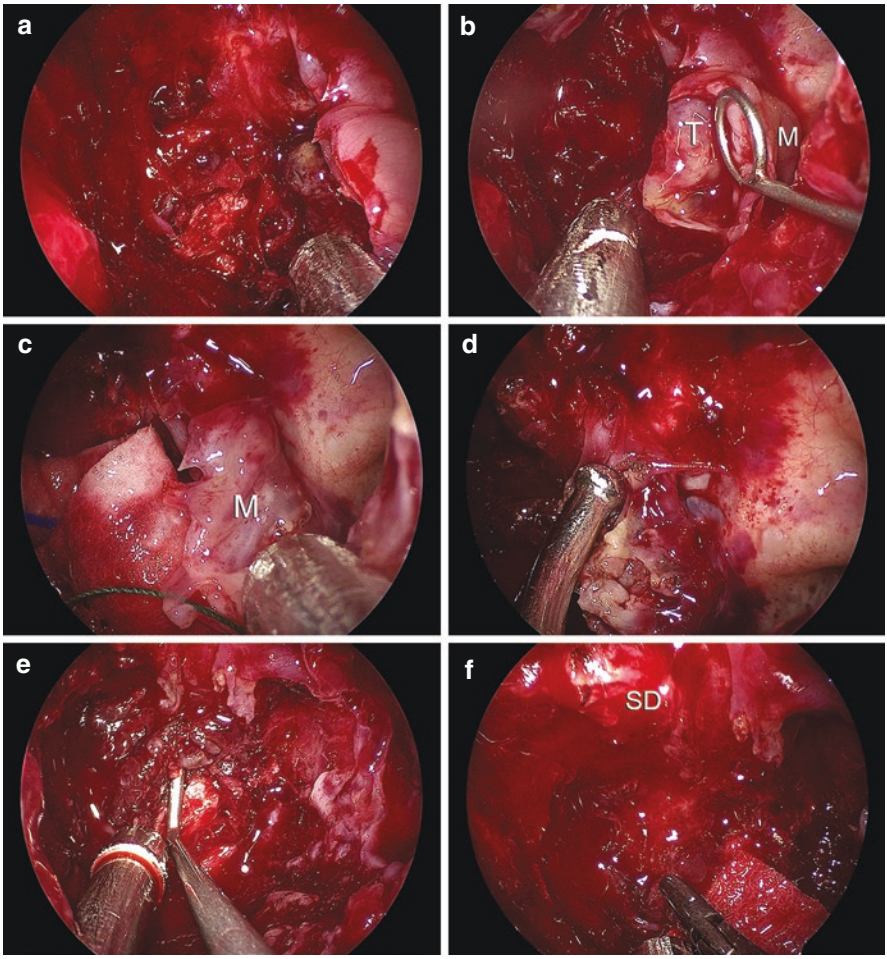

Fig. 7.32 Classic clival chordoma. Operative views (**a**–**f**) during tumor resection via an endoscopic endonasal transclival approach. SD, sellar dura; M, sphenoid sinus mucosa; T, tumor

7.5 The Uncommon Pathologies of the Suprasellar Area and the Cavernous Sinus

Indeed, many of the rare and uncommon pathologies in the suprasellar represent extensions from those originating from the sella or other anatomical structures in its vicinity. These lesions are very well suited to endoscopic endonasal transsphenoidal approach and its variations (Fig. 7.33). A dedicated review of these lesions as well as those purely originating from the cavernous sinuses is beyond the scope of this chapter.

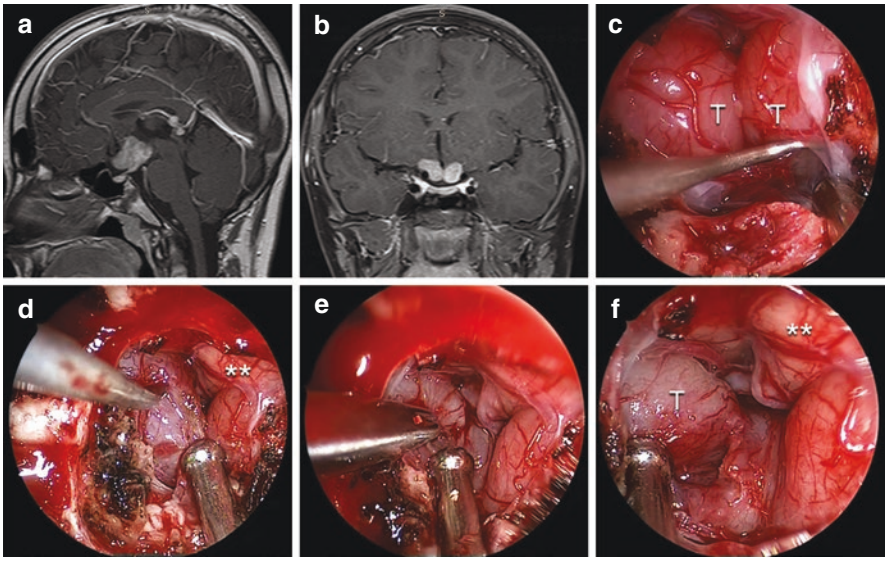

Fig. 7.33 Suprasellar GCT. Preoperative MR images (**a**, **b**). Operative views (**c**–**f**) during tumor biopsy via an endoscopic endonasal transtuberculum sellae approach. T, tumor; double asterisks, optic chiasm

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