10 Expansile Lesions Arising from Structures and Spaces Adjacent to the Paranasal Sinuses

Luca Oscar Redaelli de Zinis, Pietro Mortini, Davide Farina, and Francesca Mossi

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L. O. Redaelli de Zinis, MD
Department of Otorhinolaryngology, University of Brescia, Piazzale Spedali Civili 1, Brescia, BS, 25123, Italy
P. Mortini, Professor, MD
Skull Base and Endocrinoneurosurgery Center, Department of Neurosurgery, University Vita e Salute, Via Olgettina 60, Milan, MI, 20132, Italy
D. Farina, MD; F. Mossi, MD
Department of Radiology, University of Brescia, Piazzale Spedali Civili 1, Brescia, BS, 25123, Italy

10.1 Introduction

Nose and paranasal sinuses can be involved by a variety of different neoplasms arising from adjacent spaces/structures and secondarily invading the bony framework composing the peripheral border of the sinonasal tract.

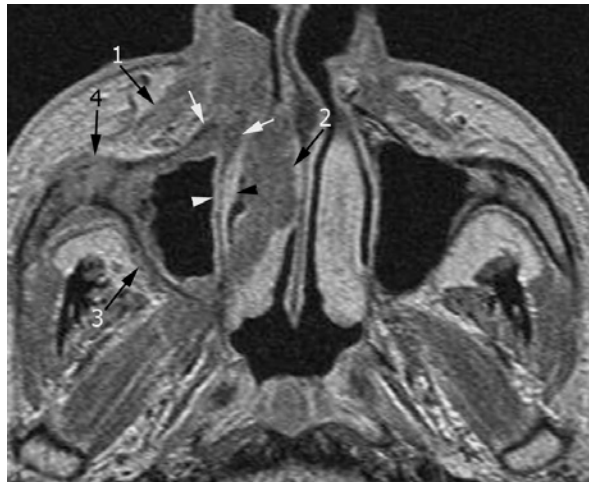
Upwards, the skull base separates the frontal sinus, the ethmoid and the sphenoid sinus from the anterior and middle cranial fossae. In exceedingly rare cases, meningioma or meningosarcoma can breach the cribriform plate, the fovea ethmoidalis and/or the planum sphenoidale (Rubinstein and Arbit 1985). Moving posteriorly, the list of lesions encompasses neoplasms arising from embryonic remnants (chordoma, craniopharyngioma), from bone and cartilage (chondroma, chondrosarcoma, osteosarcoma), and from the pituitary gland (adenoma) (Chakrabarty et al. 1998; DeMonte et al. 2000; Brown et al. 1994; Johnsen et al. 1991).

No real anatomic boundary separates nasal cavities from the nasopharynx. Thereby, nasopharyngeal tumors may have an unimpeded access to the nasal

fossae through the choanae. Invasion of the paranasal sinuses is less common, probably because nasopharyngeal cancer typically takes origin from the vault or the lateral recess. As these areas are closer to the skull base, the preferential pathway of spread is towards the anterior foramen lacerum and the clivus (Ng et al. 1997).

The posterolateral wall of the maxillary sinus is the boundary between the sinonasal tract and the masticator space. Neoplasms originating within this space are basically sarcomas or lymphomas, less frequently neurogenic tumors (Yu et al. 1998).

The inferior boundary of the sinonasal tract (i.e., nasal cavity floor, alveolar recess of the maxillary sinus) may be involved by expansile lesions arising from the mucosa or submucosa lining the oral cavity. These lesions include epithelial neoplasms (basically squamous cell carcinoma) and tumors arising from minor salivary glands (Ginsberg and DeMonte 1998). Several benign and malignant lesions arise from the alveolar process of maxillary bone and extend toward the sinonasal tract, mostly into the maxillary sinus and hard palate. Peculiar imaging findings may be observed in cysts and tumors arising from cells and tissues involved in odontogenesis. Finally, basal or squamous cell carcinoma of the face can invade adjacent sinonasal tract structures and further extend submucosally into bones or access the skull base via perineural spread (Williams et al. 2001) (Fig. 10.1–2).



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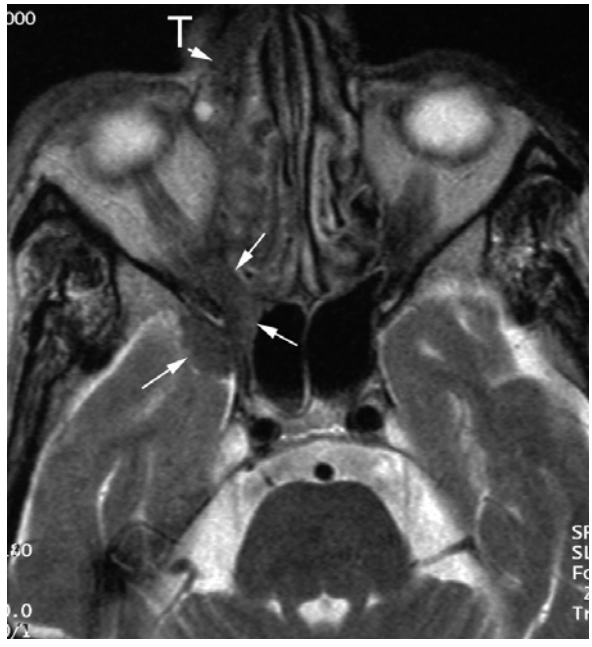

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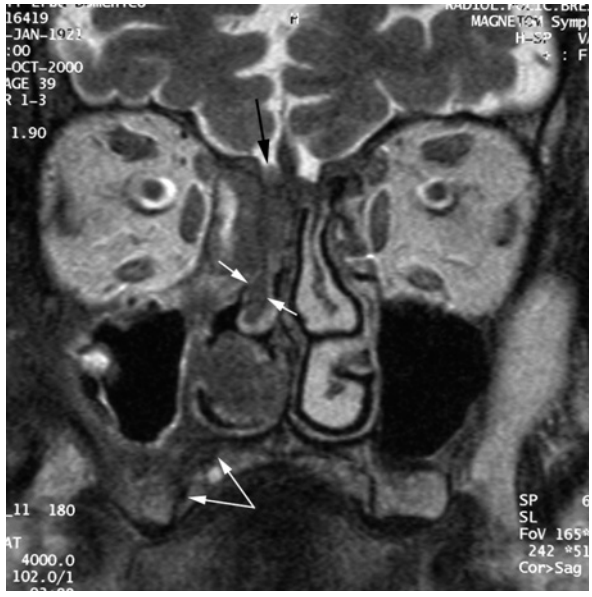

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Fig. 10.1a–c. Basal cell carcinoma of the face. **a** Enhanced T1-weighted axial plane. Tumor spreads from the nasal skin into the muscles of the lip (*1*), and grows mainly along the bony framework of sinonasal structures into the inferior turbinate (*2*) (replacing the hyperintense signal of the vascularized tissue), anterior (*arrows*) and postero-lateral maxillary wall from which the hypointense tumor tissue penetrates the masticator space (*3*) and pterygopalatine fossa. Bone marrow invasion and cortical erosion of the zygomatic bone (*4*) is also present. At this level, the medial maxillary sinus wall is partially spared from the subperiosteal invasion (*arrowheads*). **b** The coronal plane (T2-weighted image) shows the hypointense tumor invading the hard palate (*long white arrows*), inferior and middle (*opposite white arrows*) turbinate. Spread through the cribriform plate appears with effacement of CSF signal on right side (*black arrow*). **c** A T2-weighted axial image demonstrates intracranial invasion through the superior orbital fissure (*white arrows*) with involvement of the foramen rotundum structures (*arrows*). Tumor (*T*)

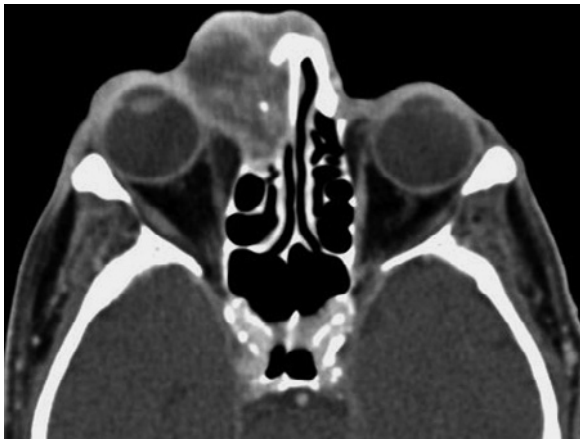


Fig. 10.2. Keratoacanthoma of the nasal skin with invasion of nasal bones, orbit, and anterior ethmoid

10.2 Skull Base Tumors Extending into the Sinonasal Tract

10.2.1 Chordoma

10.2.1.1 Definition, Epidemiology, Pattern of Growth

Chordoma, a tumor taking origin from embryonic remnants of the primitive notochord (Batsakis 1979) accounts for less than 1% of central nervous system tumors (Weber et al. 1995). The most common locations are the sacrococcygeal region (45%–49%), the base of the skull (36%–39%), and the spinal axis (8%–15%) (Harrison and Lund 1993). In the cranio-cervical region, seven points of origin have been identified: dorsum sellae, Blumenbach's clivus, retropharyngeal notochord vestiges, remnants in the apical ligament of the dens, nuclei pulposi of the cervical vertebrae, vestiges in the squama occipitalis, and ectopic localizations (Binkhorst et al. 1957). Primitive chordomas of the sinonasal tract, which have been rarely reported (Loughran et al. 2000), are interpreted not as real ectopic localizations but more properly as lesions arising from embryonic remnants of the notochord (Shugar et al. 1980).

Male-to-female ratio is generally reported to be 2:1 (Perzin and Pushparaj 1986; Weber et al. 1995). The lesion can be observed at any age, with a predominance for the third and fourth decades in intracranial localizations, while spinal chordomas are generally diagnosed at an older age because of a late occurrence of signs and symptoms (Perzin and Pushparaj 1986; Weber et al. 1995). No association with irradiation or any other environmental factors has been observed. A small percentage of cases have a familial pattern of inheritance (Dalprà et al. 1999).

Chordomas develop from the bone, so they initially grow extradurally with bone destruction and secondary extension into the adjacent soft tissues (Fig. 10.3a) (Oikawa et al. 2001). They present some of the typical features of a malignant tumor, such as local invasiveness, tendency to recur, and a potential for developing distant metastases, which have been reported in up to 43% of patients (Higinbotham et al. 1967). This event is typical for sacrococcygeal localizations. The low rate of systemic spread of skull base chordomas, ranging from 0 to 10%, is considered related to the fact that patients die for local progression before developing metastases (Gay et al. 1995; Hug et al. 1999).

Chordoma generally presents as a whitish, soft, multilobulated mass with a fibrous pseudocapsule, sometimes filled by a mucoid substance (secondary to previous hemorrhage), sometimes with hemorrhages, necrosis and/or calcifications and fragments of bone (Batsakis 1979). Microscopically, it is characterized by vacuolated physaliphorous cells, which are translucent cells of different sizes, rich in mucin and glycogen (Batsakis 1979).

At histology, the differential diagnosis includes primary bone tumors, cartilaginous neoplasms such as chondromas or chondrosarcomas, epithelial neoplasms such as mucinous-forming adenocarcinoma or salivary neoplasms, metastases, schwannoma, neurofibroma, meningioma, neuroblastoma, hemangioma and lymphoma. Cytokeratin antibodies and epithelial membrane antigen positivity differentiates chordoma from cartilaginous neoplasms, while S-100 positivity, not a constant feature, may help the differentiation from epithelial neoplasms (Bottles and Beckstead 1984; Walker et al. 1991). Some chordomas stain positive for vimentin, which reflects a mesenchymal differentiation (Bouropoulou et al. 1989).

10.2.1.2 Clinical Findings

Headache and diplopia, more frequently due to abducens nerve involvement, are the most common presenting symptoms (Volpe et al. 1993). The fifth cranial nerve is also frequently involved, due to the

progressive neoplastic lateral growth with invasion of adjacent structures such as the cavernous sinus. Signs and symptoms also include visual loss and limitation of the visual field, and extraocular complaints such as dysphagia, dyspnea, dysphonia, facial pain, facial paresis, hearing loss, tinnitus, dizziness and ataxia, due to brain stem compression. Anterior extension of the lesion into the nasopharynx can explain pharyngolaryngeal and otological symptoms, whereas extension toward the sinonasal tract can cause nasal obstruction, hypo-anosmia, hyponasal speech, mucopurulent discharge, and, rarely, epistaxis (Perzin and Pushparaj 1986; Harrison and Lund 1993).

10.2.1.3 Treatment Guidelines and Prognosis

The treatment of choice for chordoma is surgery and a wide spectrum of approaches (transoral, anterior transfacial, subfrontal transcranial, frontotemporal transcavernous, lateral transpetrosal, subtemporal transpetrous-transcavernous, subtemporal infratemporal, and extreme lateral retrocondylar-transcondylar) (Strugar and Sekhar 2000) have been described. Selection of surgical access mainly depends on tumor location, which is grouped in upper-, mid-, and lower-clival. Postoperative radiotherapy, both by linear accelerator, or proton beam, can provide a better control of the disease (Rosenberg et al. 1999; Hug et al. 1999; Crockard et al. 2001).

Prognosis of chordoma is related to the extent of surgical removal: 5-year survival of 35% is reported after incomplete resection combined with conventional radiation therapy (Zorlu et al. 2000). Better results are obtained with aggressive surgical treatment and proton-beam postoperative radiotherapy, with 5-year and 10-year disease-free survival rates ranging from 50% (Colli and Al-Mefty 2001) to 77% (Crockard et al. 2001) and from 45% to 69%, respectively (Rosenberg et al. 1999; Crockard et al. 2001). No effective chemotherapeutic agents are available for the treatment of this disease.

The prognosis of chordoma is affected by a variety of clinical and pathologic characteristics. Important features include tumor location, size and resectability, as well as the age and the gender of the patient. Larger tumors, female gender, and age greater than 40 years are associated with a poorer outcome (Forsith et al. 1993; O'Connell et al. 1994; Gay et al. 1995; Hug et al. 1999).

10.2.1.4 Key Information to Be Provided by Imaging

- Involvement of the sella turcica, clivus, sphenoid sinus, pyramid apex.
- Displacement or encasement of internal carotid artery, involvement of the cavernous sinus, displacement of the basilar artery.
- Compression of central nervous system structures, involvement of cranial nerves.
- Involvement of the ventricular system, presence of hydrocephalus.
- Extent toward other skull base areas (foramen lacerum, pterygopalatine fossa) or into the nasopharynx, the atlanto-occipital joint and proximal cervical spine.
- Presence of imaging features highly suggestive for this specific lesion.

10.2.1.5 Imaging Findings

The site of origin of most skull base chordomas is the basiocciput-basisphenoid where the terminal portion of the notochord ends reaching the sphenoid bone just inferior to the sella turcica and dorsum sellae. Nasopharyngeal and intracranial locations are rarely observed, their origin being explained by the extra-osseous path of the notochord, which may have short segments running outside the bone, either

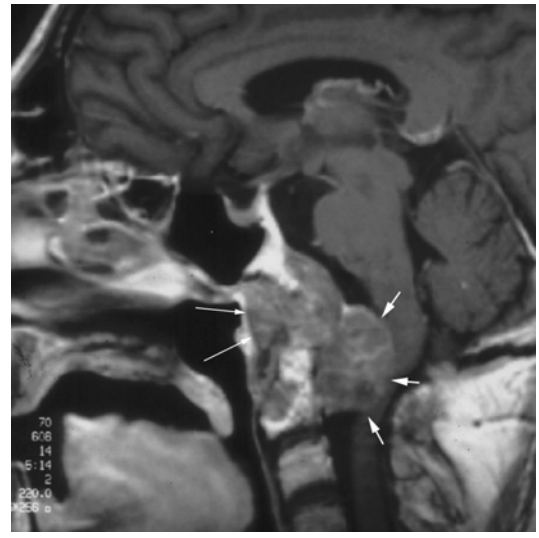


Fig. 10.3. Chordoma. On sagittal enhanced T1 sequence the lesion has a cervical location with invasion of the lower aspect of the atlanto-occipital joint; posteriorly the foramen magnum is invaded (*short arrows*). Anteriorly, the neoplasm extends into the nasopharynx (*long arrows*)

within nasopharyngeal soft tissues or within the posterior cranial fossa (Fig. 10.3).

On CT, chordoma appears as a midline clival soft tissue mass with bone destruction (Oot et al. 1988). Invasion of the body of the sphenoid usually occurs, accounting for the presence of coarse high densities within the mass, assumed to be remaining fragments of the eroded bone, rather than calcifications or new matrix formation (Fig. 10.4). No sclerotic changes are detected at the boundary with the invaded bone. Cystic components are frequently observed. Enhancement is present in at least some parts of the soft tissue component. When chordoma arises in nasopharynx or posterior fossa, the bone may or may not be eroded.

Intracranial extent frequently leads to posterior displacement of the basilar artery and mass effect on the brain stem. Lateral clival chordoma may present as a cerebello-pontine angle mass.

The MR appearance varies in relation to the composite histologic pattern of the tumor. In up to 80% of lesions, MR shows heterogeneous hyperintensity on T2 - moderate to extreme (Meyers et al. 1992) with possible dark areas, reflecting the presence of mucoid or old hemorrhagic areas, respectively. Soft tissue components show iso to hypointensity on T1, and variable degrees of contrast enhancement (Sze et al. 1988) (Fig. 10.5). Cystic areas may present bright signal on unenhanced T1.

Sagittal plain T1 sequences are particularly useful, as the hypointense chordoma replacing the hyperintensity of the clival bone marrow can be clearly seen (Sze et al. 1988). On all sequences, hypointense areas standing for large intratumoral calcifications can be identified. On T2, low intensity strands – composed of fibroconnective tissue – form hypointense septations enclosing lobulated hyperintense areas.

Though imaging findings of chordoma are rather nonspecific, the site of origin and patient's age may help in the differential diagnosis.

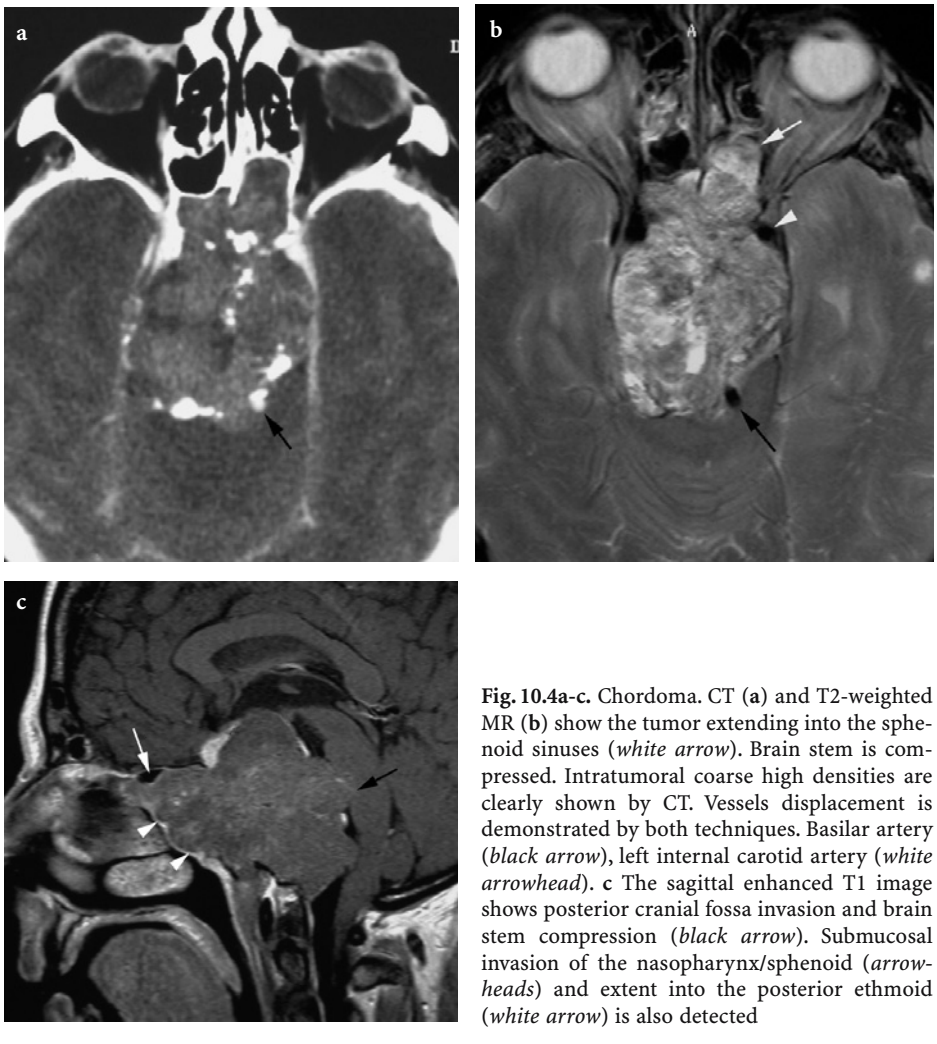
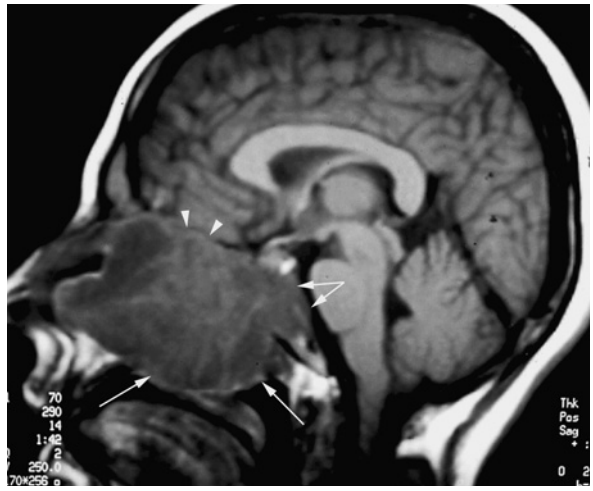


Fig. 10.4a-c. Chordoma. CT (**a**) and T2-weighted MR (**b**) show the tumor extending into the sphenoid sinuses (*white arrow*). Brain stem is compressed. Intratumoral coarse high densities are clearly shown by CT. Vessels displacement is demonstrated by both techniques. Basilar artery (*black arrow*), left internal carotid artery (*white arrowhead*). **c** The sagittal enhanced T1 image shows posterior cranial fossa invasion and brain stem compression (*black arrow*). Submucosal invasion of the nasopharynx/sphenoid (*arrowheads*) and extent into the posterior ethmoid (*white arrow*) is also detected



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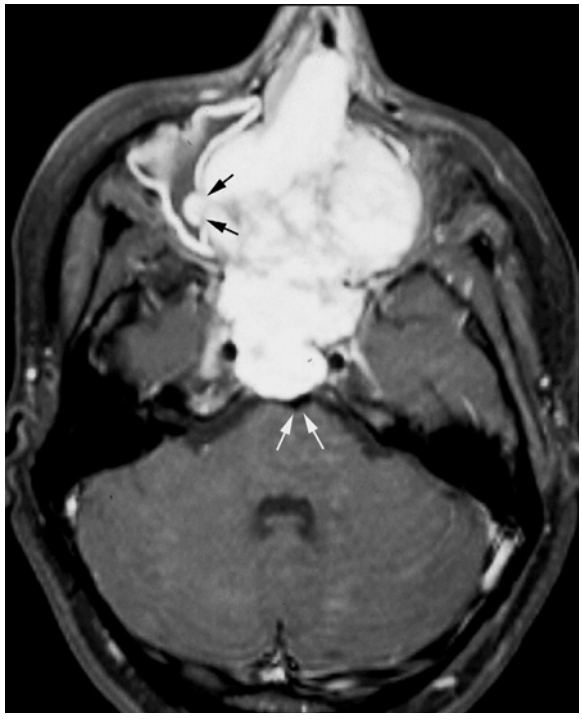

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Fig.10.5 a,b. Chordoma. **a** Unenhanced T1-weighted sequence in the sagittal plane. A large extradural chordoma with invasion of the sphenoid sinus and ethmoid is shown. Replacement of bone marrow signal within the sphenoid bone (*double arrows*) with erosion of the posterior bony rim is detected. Intranasal extent reaches the planum sphenoidale from below (*arrowheads*). The mass projects into the nasopharynx (*white arrow*). **b** The chordoma shows relevant and heterogeneous enhancement. Extradural intracranial extent with basilar artery displacement (*white arrows*). Initial extent through the medial maxillary wall is present

10.2.2 Cartilaginous Tumors (Chondroma, Chondrosarcoma)

10.2.2.1 Chondroma (or Osteochondroma)

This benign neoplasm is composed of mature hyaline cartilage. It occurs as either solitary or multiple lesions. When the tumor arises within the medullary osseous cavity, it is termed enchondroma. As the tumor grows outward from the cortex, it develops on the external bone surface (osteochondroma) as a painless, slow-growing lesion producing symptoms and signs depending on the location. Surgical treatment has to be considered only for symptomatic tumors.

10.2.2.2 Chondrosarcoma

10.2.2.2.1 Definition, Epidemiology, Pattern of Growth

Chondrosarcoma of the skull base is a rare primary intraosseous neoplasm, which is frequently included in the differential diagnosis of chordoma. It can be isolated or multiple, being part of one of the enchondromatosis syndromes (Ollier disease, Maffucci syndrome, metachondromatosis) (Weber et al. 1995).

Prevalence of chondrosarcoma is less than 0.2% among intracranial tumors and 6% among skull base tumors (Cianfriglia et al. 1978; Kveton et al. 1986). The peak incidence is in the second and third decades (Hassounah et al. 1985), and the male-to-female ratio is 1:1 (Gay et al. 1995). Chondrosarcoma may arise from primitive mesenchymal cells or from embryonal rests of the cranium's cartilaginous matrix (Gay et al. 1995). Conventional chondrosarcomas are grouped in 3 grades, according to the degree of their cellularity, cytologic atypia, and mitotic activity. Grade 1 is the least aggressive neoplasm with features of a benign tumor, while grade 3 is the most aggressive type. Other histological types are clear cell, myxoid, mesenchymal, and dedifferentiated chondrosarcoma (Dorfman and Czerniak 1998). Conventional chondrosarcoma is composed of hyaline, myxoid, or an mixture of hyaline and myxoid cartilage. Typically it grows with an infiltrative pattern, replacing the normal marrow elements, encasing preexisting cancellous bone, and permeating

haversian channels. It is by this mechanism that the tumor frequently transgresses the cortex and forms a soft tissue mass.

The clivus, the sphenoid bone - particularly in the parasellar area and the petrous apex (Korten et al. 1998; Rosenberg et al. 1999) - are the most frequent sites of localization of skull base chondrosarcomas, which are considered to arise from residual endochondral cartilage (Neff et al. 2002). Chondrosarcomas have more commonly a paramedian location and they involve the sphenoethmoidal area in 33% of cases (Sekhar and Oliveira 1999).

10.2.2.2.2 Clinical Findings

Since chondrosarcoma is a slow growing tumor, signs and symptoms occur late and depend on the site of origin and pattern of growth. Usually, the clinical findings are undistinguishable from those of chordoma, the latter being more frequently asymptomatic, while chondrosarcoma is more commonly associated with visual loss, facial numbness, and multiple cranial nerves impairment (Volpe et al. 1993).

10.2.2.2.3 Treatment Guidelines and Prognosis

Surgical treatment is the first line therapy, generally followed by proton-beam postoperative radiotherapy. In a group of 200 patients who received proton beam irradiation after biopsy or surgical treatment, the 5- and 10-year local control rates were 99% and 98%, respectively, and 5- and 10-year disease-specific survival rates were both 99% (Rosenberg et al. 1999). As the outcome is significantly better than that of chordoma, this finding emphasizes the importance of an accurate distinction between the two neoplasms.

10.2.2.2.4 Key Information to Be Provided by Imaging (see Section 10.2.1.4)

10.2.2.2.5 Imaging Findings

Most frequent sites of origin of skull base chondrosarcoma include the spheno-occipital, spheno-petrosal and petro-occipital synchondroses, and a large part of the petrous bone where the tumor is hypothesized to develop from residual enchondral cartilage (Lee and van Tassel 1989; Watters and Brookes 1995; Neff et al. 2002)

The quantity of chondroid matrix within the lesion influences the appearance on CT. The soft tissue component appears hypodense on CT and shows variable degrees of enhancement.

Calcifications largely vary, ranging from small and scattered, often arranged as a peripheral rim, to large, dense, and diffuse. They tend to have an interrupted ring-like shape (Fig. 10.6). Nonetheless, intratumoral calcifications may be absent (Brown et al. 1994). Bone destruction is a rather constant finding (Lee and van Tassel 1989).

MR shows moderately high to very high hyperintense T2 signal within the nonmineralized portion of the lesion (Fig. 10.7). The signal is non-homogeneous in about 60% of cases, due to intratumoral areas of hypointensity corresponding to coarse chondroid mineralization or fibrocartilaginous areas on CT. Otherwise to CT, small calcifications may be undetected on MR. Chondrosarcoma appears iso to hypointense on T1, and may show marked non-homogeneous contrast enhancement (immediately after contrast agent administration), partly related to the presence of calcifications and chondroid matrix (Meyers et al. 1992) (Fig. 10.8).

Based on pathologic findings, it is often difficult to discriminate low-grade chondrosarcoma from its benign counterpart. Imaging may play a role providing information about cortical bone destruction.

The imaging based differential diagnosis in the skull base includes a variety of lesions, the most challenging of which is represented by chordoma. Some

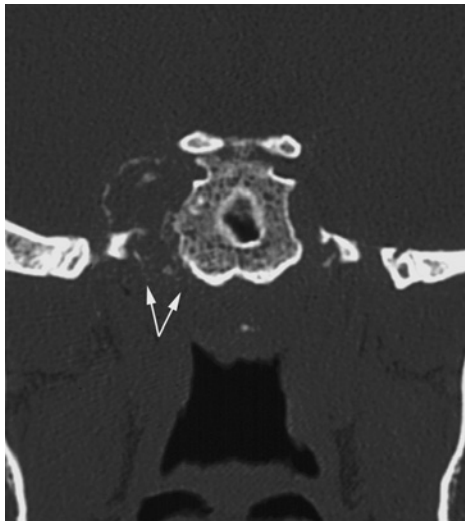


Fig. 10.6. Chondrosarcoma of the right petro-clival suture. Unenhanced CT in the coronal plane shows small, thin calcifications 1s lining the boundary of the mass that extends submucosal through the foramen lacerum into the nasopharynx (*arrows*)

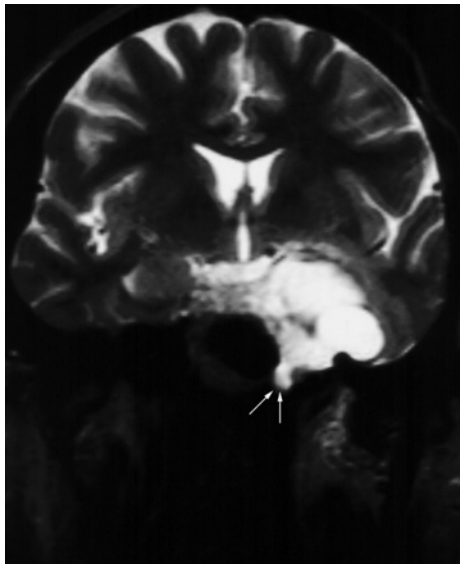


Fig. 10.7. Chondrosarcoma of the left petro-clival suture, highly hyperintense on the coronal T2-weighted image. Left cavernous sinus and internal carotid artery are involved. Downward extent toward the nasopharynx is shown (*arrows*)

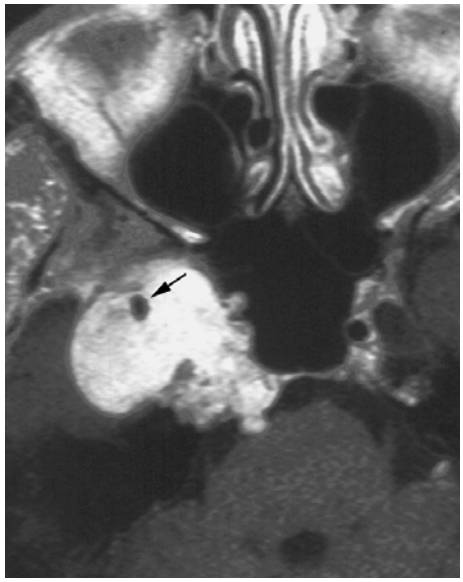


Fig. 10.8. Chondrosarcoma of the right petrous apex with internal carotid artery encasement (*black arrow*), cavernous sinus and Meckel cave involvement. Part of the lesion extends into left pre-pontine cistern

clues are offered by calcifications, which are larger (probably representing residual bone) in chordoma, whereas they appear smaller (denoting organic matrix production) in chondrosarcoma. Nonetheless, the site of origin (midline for chordoma, paramedian for chondrosarcoma) is probably the most reliable criterion (Bourgouin et al. 1992).

10.2.3 Pituitary Tumors

10.2.3.1 Definition, Epidemiology, Pattern of Growth

Pituitary tumors are frequent neoplasms representing about 8%-10% of all intracranial tumors (Kaye 1997). They are incidentally found in 10% of autopsies (Nammour et al. 1997; Fajfr et al. 2002). Most pituitary tumors occur in young adults, with a predilection for females, and the highest incidence in the third and the sixth decades. The pathogenesis of pituitary tumors can be sometimes related to oncogenes anomalies (G-protein, ras gene, p53) and to the syndrome of multiple endocrine neoplasia (Corbetta et al. 1997; Spada et al. 1998; Suhardja et al. 1999). Most pituitary tumors are benign, but some of them may show a high growth rate and aggressiveness. Central nervous system and distant metastases are exceptionally described (Pichard et al. 2002). Hardy's radiological classification distinguishes tumors on the basis of the size and gross features (bone changes, extent) (Hardy 1969). According to the size, tumors are divided in microadenomas (less than 10 mm) and macroadenomas (greater than 10 mm). On the basis of imaging features, 5 classes of tumors are identified. Microadenomas are designated as being either grade 0 or grade I, depending on whether the sella appears normal, or minor focal sellar changes are present. Macroadenomas causing diffuse sellar enlargement, focal sellar erosion or extensive sellar and skull base destruction are classified as grade II, grade III, and grade IV, respectively. Macroadenomas are further subclassified by the degree of suprasellar extension (Hardy 1969). Pituitary adenomas have also been classified by their staining in chromophobic and chromophilic, but this classification is disused because of the overwhelming importance of immunohistochemistry and electron microscopy, that sometimes show the hormone production and also multiple hormonal production in the same tumor.

Differential diagnosis includes many other intracranial neoplasms such as craniopharyngioma, meningioma, germinoma, and secondary tumors. Granulomatous and infectious disorders involving the intrasellar region should also be considered. In case of sphenoid invasion by a pituitary adenoma or ectopic sphenoid localization, the differential diagnosis includes mucocele and fungus ball.

10.2.3.2 Clinical Findings

Clinical findings depend on the local extent of the tumor and on the possible endocrine disorders due to hormonal overproduction and/or hypopituitarism. In about 70% of cases, the clinical picture is dominated by features of anterior pituitary hypersecretion resulting in a characteristic hypersecretory syndrome. (Zervas and Martin 1980). Acromegaly, Cushing's disease, amenorrhea-galactorrhea syndrome and, rarely, secondary hyperthyroidism represent the classical paradigms of GH, ACTH, PRL and TSH hypersecretion, respectively. It is important to recognize that hyperprolactinemia is not always a feature specific for PRL-producing adenomas. Moderate hyperprolactinemia (<120 ng/ml) can occur in a variety of lesions involving the sellar region. This phenomenon, frequently referred to as the "stalk section effect", is the result of compressive or destructive lesions involving the hypothalamus or the pituitary stalk. Pituitary tumors can also present with symptoms suggesting partial or total hypopituitarism (fatigue, weakness, hypogonadism, regression of sexual secondary characteristics, hypothyroidism). This often occurs insidiously in association with pituitary macroadenomas which compress and impair the secretory capability of the adjacent nontumorous pituitary gland. Pituitary insufficiency can also acutely occur in the context of pituitary apoplexy. A progressively enlarging pituitary mass can also generate a constellation of neurologic signs and symptoms, depending on its growth path.

Headache, due to stretching of the enveloping dura or of the diaphragma sellae, may be an early finding. However, the single most common neurologic sign is visual loss, which is related to suprasellar extension of the tumor, with compression of the optic nerves and chiasm. The classic and most common pattern of visual loss is that of a bitemporal hemianopic field deficit, often in association with decreased visual acuity. Large pituitary adenomas can encroach upon the hypothalamus, causing alteration of sleep, alertness, behavior, eating and emotion. These tumors can extend into the region of the third ventricle, where obstruction to effluent CSF flow can result in obstructive hydrocephalus. Quite commonly, the tumor extends laterally in the region of the cavernous sinus. With progressive cavernous sinus invasion, cranial nerves (oculomotor, trochlear, trigeminal, abducens) can occasionally be affected. Finally, some pituitary adenomas can reach a giant size, extending into the anterior, middle and posterior cranial fossae. Neuro-ophthalmologic examination together with hormones testing are indicated prior to imaging (Lissett and Shalet 2000).

10.2.3.3 Treatment Guidelines and Prognosis

Therapy for pituitary tumors should be directed at the following goals: reversal of endocrinopathy and restoration of the normal pituitary function; removal of tumor mass and restoration of normal neurologic function. Nowadays, these goals can be more frequently achieved due to the evolution of microsurgical techniques, the development of receptors-mediate pharmacotherapy, and refinements in the delivery of radiation therapy. Each of these treatment modalities has specific advantages and limitations, therefore treatment selection should be thoughtfully individualized to each patient (Wilson 1984).

Medical management can sometimes control endocrine manifestations, particularly in case of prolactinomas and growth hormone secreting adenomas. Two classes of pharmacologic agents have emerged as primary or adjuvant therapies for pituitary tumors: dopamine agonists and somatostatin analogues. Surgical treatment is advised to control local expansion signs and symptoms and to release the patient from chronic drug treatment. Transsphenoidal access (microscopic or endoscopic) is the approach of choice (Cappabianca et al. 2002; Cho and Liau 2002; Zada et al. 2003). A transcranial approach can be added in larger lesions (Landolt 2001). Transsphenoidal surgery is associated with low morbidity (Ciric et al. 1983). Currently, more than 95% of pituitary tumors are approached transsphenoidally, with conventional transcranial approaches reserved for the remaining few cases in which anatomic features of the sella or unusual intracranial tumor extension (dumb-bell shape) limit the transsphenoidal accessibility (Mac Carty et al. 1973; Mortini and Giovanelli 2002). Prognosis of pituitary tumors is generally good (Rees et al. 2002). Whenever residual neoplasm is left, medical treatment adequately controls the disease. Radiosurgery has also been proposed for small residual lesions or for the cavernous sinus invasion (Sheehan et al. 2002; Losa et al. 2004).

10.2.3.4 Key Information to Be Provided by Imaging (see Section 10.2.1.4)

10.2.3.5 Imaging Findings

Pituitary adenoma may have an extrasellar extension, laterally into the cavernous sinus, inferiorly into the sphenoid sinus. The latter may occasionally occur in

the absence of a suprasellar growth. In this case, the floor may be the only bony boundary of the sella to be disrupted. Such a pattern of growth may hinder the discrimination between pituitary adenoma, chordoma, chondrosarcoma, and superior extension of a nasopharyngeal carcinoma.

More than CT and MR appearance, correlation of site of the lesion with laboratory tests and clinical history may help to properly address the diagnosis (Cottier et al. 2000).

MR is the technique of choice in the diagnosis of pituitary adenomas, as its high contrast resolution allows to detect even small microadenomas, which are not identified on CT (Thuomas 1999). In addition, in the preoperative work-up of macroadenomas, MR allows to demonstrate lateral extension toward the cavernous sinus, internal carotid artery, middle and anterior cerebral arteries as well as vertical extension towards the suprasellar cistern, optic chiasm and nerves (upwards) and sphenoid sinus (downwards) (Fig. 10.9) (Davis et al. 1987).

Pituitary adenomas share some common MR features regardless of their size (micro- or macroadenomas). T1 sequence is the most appropriate tool to identify these lesions – generally hypointense – and to discriminate them from the adjacent normal parenchyma – more hypointense – unless totally compressed. In this sequence, spontaneous hyperintensity is the hallmark of the presence of blood (Ostrov et al. 1989). On T2 sequence, hyperintensity is more commonly observed in macroadenomas; according to Iuchi et al. (1998) high T2 signal indicates a softer tumor and, therefore, better predicts resectability.

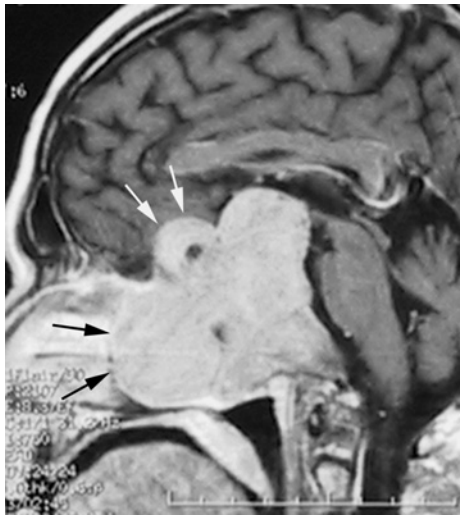


Fig. 10.9. Large pituitary non-functioning adenoma in a 48 years old male who experienced decreased visual acuity on both eyes with bitemporal hemianopsia. The tumor inferiorly extends through the sphenoid sinus into the nasopharynx and posterior nasal cavity (*black arrows*). Anteriorly, the pituitary adenoma extends into the anterior cranial fossa (*white arrows*)

Cystic degeneration of pituitary adenomas is heralded by the presence of T1 hypo-and T2 hyperintense areas, which are infrequently associated with fluid on fluid levels.

Sphenoid sinus invasion can be demonstrated when the iso- to hypointense signal of the adenoma protrudes into the air-containing sinus. Expectedly, subtle abnormalities of cortical bone of the sella may be complex to identify on MR. As a result, the interpretation of MR findings may be sometimes difficult, particularly when the sphenoid sinus shows inflammatory changes (mucosal thickening, retained secretions with variable degrees of dehydration, fungal superinfection). Narrow display windows are recommended to identify subtle sinus septa, as these may represent valuable landmarks during transsphenoidal surgery. The intra-sphenoidal portion of the pituitary adenoma has been reported to result less intense on both T1 and T2 images and on contrast-enhanced images when compared with the sellar/suprasellar solid portion of the adenoma, which show marked contrast enhancement. Histological examination of adenoma specimens demonstrate some degree of fibrosis within the portion extending into the sphenoid sinus (Ishii et al. 1996). Rarely, isolated ectopic pituitary adenomas arising within sphenoid sinus have been reported, showing nonspecific MR findings such as soft tissue mass, isointense with gray matter on T1-weighted images with heterogeneous enhancement (Slonim et al. 1993).

10.2.4 Craniopharyngioma

10.2.4.1 Definition, Epidemiology, Pattern of Growth

Erdheim in 1904 contributed the first adequate histological description of the lesion (Erdheim 1904). However, the term craniopharyngioma was introduced by Cushing in 1932 (Cushing 1932). Craniopharyngioma is thought to arise from small ectodermal cellular clusters, which are usually found in the transition area of the pituitary stalk with the pars distalis of the adenohypophysis, but may also be detected in the pars tuberalis along the upper part of the stalk. Two main hypotheses about the origin of

the tumor exist in the literature. According to the first one, craniopharyngioma develops from the ectodermal clusters of primitive craniopharyngeal duct and adenohypophysis (embryologic theory). The second one hypothesizes an origin from metaplasia of the residual squamous epithelium found in the adenohypophysis and anterior infundibulum (metaplastic theory). Although the origin of craniopharyngioma is still controversial, some authors believe that it may have a dual origin (Giangaspero et al. 1984). They attribute the so-called childhood (adamantinous) craniopharyngioma to embryonic remnants, and the adulthood (squamous papillary) craniopharyngioma to metaplastic foci of adenohypophysis cells.

Craniopharyngioma accounts for 1%–4% of intracranial tumors (5–10% in children) and has a bimodal age distribution with a peak in childhood (5–10 years) and in the middle-aged patients (50–60 years). There is no difference in distribution between males and females or among races (Einhaus and Sanford 2000). Primitive or secondary sinonasal involvement has been exceptionally observed (Akimura et al. 1989; Bret and Beziat 1993; Chakrabarty et al. 1998; Jiang et al. 1998; Falavigna and Kraemer 2001).

Craniopharyngioma typically arises in the infundibulo-hypophyseal axis in the sella and suprasellar area, frequently occupying the suprasellar cisterns, but it may grow in any direction. A better topographic classification of craniopharyngioma was established by MR, recognizing four major types: intrasellar, infundibulum-tuberian, intraventricular, and dumbbell-shaped craniopharyngioma (Raybaud et al. 1991). Important surgical classifications have been proposed by Samii and Bin, (1991) and Yasargil et al. (1990). They are based on the vertical extension of the tumor and its relationship with the third ventricle.

Craniopharyngioma is an epithelial neoplasm with solid and cystic components with frequent calcifications, which has a tendency to infiltrate and to produce a glial reaction causing strong adherences (Einhaus and Sanford 2000). Child and adult types have a different growth pattern, with a higher tendency for brain infiltration in the adamantinous type. Differential diagnosis encompasses other tumors, infectious or inflammatory processes and other congenital anomalies (Shin et al. 1999).

10.2.4.2 Clinical Findings

Since craniopharyngioma is a slow-growing tumor, it may reach even a large size before causing symptoms. In the majority of cases, the time interval between the onset of symptoms and the diagnosis ranges from 1 to 2 years.

Most frequently, craniopharyngiomas have an intrasellar and suprasellar localization. Their growth usually is associated with involvement of surrounding structures in all directions, eventually resulting in intracranial hypertension, endocrine dysfunction and neurologic signs and symptoms (Rohrer et al. 2002). Increased intracranial pressure results from an enlarging intracranial mass or obstructive hydrocephalus. Visual deficits may be related to direct compression of the optic pathways or may be secondary to intracranial hypertension. Endocrine abnormalities, which appear in men as decreased sexual drive (88%) and in women as amenorrhea (82%) (Carmel 1990), are caused by compression of the hypothalamic-hypophyseal axis. In case of large tumors of the adult, psychiatric, cognitive, and complex neurologic symptoms have been described (Donnet et al. 1999; Fitzgerald and Morgenstern 2000).

Children frequently present with symptoms of increased intracranial pressure (65–75%), such as headache and vomiting. About 20% of them have papilloedema. Obstructive hydrocephalus is present in about one third of the cases. Visual deficits are commonly well tolerated by children. Pang (1993) described the typical child with a craniopharyngioma as being short, obese, dull, half-blind, and with a poor school record.

10.2.4.3 Treatment Guidelines and Prognosis

There is general agreement that surgery plays a major role in the treatment of craniopharyngioma. A transsphenoidal approach is elected when the lesion is mostly intrasellar, while a pterional or a subfrontal approach is appropriate for suprasellar lesions, and a transcallosal approach is used for third ventricle floor lesions (infundibulum and tuber cinereum) (Norris et al. 1998; Fahlbusch et al. 1999; Chen 2002; van Effenterre and Boch 2002; Wang et al. 2002). Radiotherapy is added when an incomplete tumor excision is obtained (Kalapurakal et al. 2003). Limited surgery with postoperative radiotherapy have been proposed with the aim of limiting the sequelae of a more extended surgery (Isaac et al. 2001; Merchant et al. 2002). In addition, stereotactic radiosurgery has been used as exclusive treatment (Schulz-Ertner et al. 2002; Ulfarsson et al. 2002).

Perioperative morbidity is mainly related to intracranial complications (Kalapurakal et al. 2003). Endocrine deficits are frequently expected (Honegger et al. 1999).

Five-year survival rates ranging from 92% to 100% have been reported (Fahlbusch et al. 1999; van Effenterre and Boch 2002; Kalapurakal et al. 2003). Owing to the young age at diagnosis and the potential clinical impact of treatment sequelae, a longer survival estimation shows the 10-year and 15-year survival rates to decrease down to 68% and 59%, respectively (Bulow et al. 1998). Tumor recurrence, which accounts for about 28% (Choux et al. 1991), often occurs along the operative track. Factors associated with an increased risk of recurrence are preoperative visual symptoms, tumor adhesiveness at surgery, and subtotal resection (Duff et al. 2000).

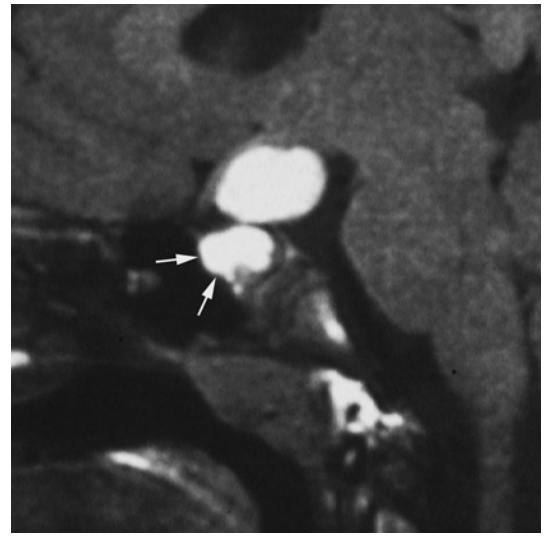


Fig. 10.10. Sellar and suprasellar craniopharyngioma showing a double cystic component which has a bright signal on the T1-weighted sagittal plane (*arrows*)

10.2.4.4 Key Information to Be Provided by Imaging (see Section 10.2.1.4)

10.2.4.5 Imaging Findings

Because it arises from the pituitary stalk axis, craniopharyngioma is usually located on the midline, most frequently within an area extending from the third ventricle to the sphenoid body. Moreover, due to its origin from remnants of the craniopharyngeal duct, it shares a common embryologic origin from oral ectoderm. This feature accounts for the histological and imaging similarity with ameloblastoma and with keratinizing and calcifying odontogenic cyst, which is characterized by proliferating ameloblastic epithelium, ghost keratin, calcification, and cyst formation. The term itself *adamantinous* craniopharyngioma derives from this histological similarity (Bernstein and Buchino 1983).

Adamantinous craniopharyngioma, which is the most common form, typically presents as a mass with both cystic and solid components. On CT, a cyst with nodular or rim calcifications (90% of cases) and some solid enhancing components is the most typical feature (Gupta et al. 1999). Though variable, the density of the cystic part is generally superior to cerebrospinal fluid. Site of the lesion is both suprasellar and sellar in 60% of cases, entirely intrasellar or suprasellar in 10% and 20% of cases, respectively.

The most typical MR finding is a heterogeneous suprasellar-sellar signal mass containing a cystic component, well defined, with internal uniform signal, hyperintense on both T1 and T2 sequences (Fig. 10.10). Nonetheless, a variety of different MR patterns may be present, including solid, calcified, CSF-like, hematin-like, and protein-like signals. A solid component is also invariably present, often partially calcified. In up to 92% of cases, more than one pattern coexists in the same lesion, reflecting the histopathologic complexity of the lesion (Mollà et al. 2002; Warakaulle et al. 2003). It is important to note that the adamantinous craniopharyngioma often elicits a relevant inflammatory reaction from adjacent tissues. At the interface with brain, this results in a dense gliosis that may be very difficult to separate from the neoplasm. Infrasellar extension is rare (Hillman et al. 1988; Bret and Beziat 1993; Chakrabarty et al. 1998; Fujimoto et al. 2002; Cheddadi et al. 1996). The solid component of the infrasellar lesion may extend into the sinonasal tract or into the nasopharynx. Cystic signals are present both within and at the boundaries of the mass. Sclerotic changes within the sphenoid bone are also observed (Bret and Beziat 1993).

Papillary craniopharyngioma, typically found in adulthood, is more frequently located within the floor of third ventricle, in up to 41% of cases. It appears more solid, calcifications are usually absent, cystic components are less relevant than in the adamantinous type, and do not show hyperintensity on T1. Post contrast studies show a partially cystic mass that enhances peripherally, mural nodules are detected in about 70% of lesions (Crotty et al. 1995). The mass

is usually encapsulated and readily separable from adjacent structures.

Imaging features useful for differentiating the two tumor types are the encasement of vessels, the lobulated shape, and the presence of hyperintense cysts in adamantinous type, and the more round shape, the presence of hypointense cysts, and the predominantly solid appearance in squamous-papillary tumors (Sartoretti-Schefer et al. 1997).

Differential diagnosis includes the *Rathke's pouch or cleft cyst* which shares the same common embryologic origin of craniopharyngioma, but is lined by a single layer of epithelial cells (cuboidal or columnar), filled by mucoid or – less commonly serous – fluid, or cellular debris. Mucoid contents accounts for the hyperintensity on both T1 and T2 sequences, making the distinction with craniopharyngioma difficult. Serous content results in CSF-like findings on CT and MR. Those cysts containing cellular debris are more dense on CT and show heterogeneous signal on MR: focal components with low signal on T2 that become iso- to slightly hyperintense on T1 (Kucharczyk et al. 1987). Unlike craniopharyngioma, Rathke's cleft cyst does not enhance except for a thin, peripheral enhancement of its wall. Only large lesions are symptomatic. Most are intrasellar, though they can be confined to the sphenoid bone, making the differential diagnosis with mucocele very difficult.

10.2.5 Meningioma

10.2.5.1 Definition, Epidemiology, Pattern of Growth

Meningioma is thought to take origin from arachnoidal cap cells (Greenberg 2001). It accounts for more than 20% of all intracranial neoplasms (Longstreth et al. 1993; D'Alessandro et al. 1995; Surawicz et al. 1999), with an annual incidence rate ranging from 2 to 13 new cases per 100,000 inhabitants (Bondy and Ligon 1996; Helseth 1997; Cordera et al. 2002). There is a female predominance of about 2:1 (Helseth 1997; Surawicz et al. 1999; Cordera et al. 2002), with a predominance in African people (Bondy and Ligon 1996). Every age can be involved, with a peak incidence in the seventh and eighth decades (Bondy and Ligon 1996).

There are many hypotheses for the etiology of meningioma. Previous radiation therapy – latency period inversely proportional to the dose of radiations (Sadetzki et al. 2002) –, viruses (SV-40, adenovirus, and papovavirus) (Bondy and Ligon 1996), chromosomal abnormalities (long arm chromosome 22, but also other chromosomes have been considered) (Bondy and Ligon 1996; Arslantas et al. 2003), hormonal alterations (estrogen, progesterone, androgens, epidermal growth factor, platelet derived growth factor, fibroblast growth factor) (Detta et al. 1993; Black et al. 1994; Figarella-Branger et al. 1994; Lambe et al. 1997), and various occupational exposures (Longstreth et al. 1993; Hu et al. 1999) have been suggested to play a role in the pathogenesis of meningioma. Also head traumas have been considered (Phillips et al. 2002), but many controversies exist about this hypothesis (Longstreth et al. 1993; Bondy and Ligon 1996).

Meningioma can arise from many different intracranial sites. Among these, tuberculum sellae (12.8% of intracranial locations) (Greenberg 2001), olfactory groove (9.8% of intracranial meningiomas) (Greenberg 2001), and petroclival meningiomas (2% of intracranial meningiomas) (Pieper and Al-Mefty 1999) can potentially extend toward the sinonasal tract and the nasopharynx (Morris et al. 1990; Maiuri et al. 1998), an event observed in 3% of cases (Farr et al. 1973).

Primary sinonasal meningioma has also been observed (Perzin and Pushparaj 1984; Thompson and Gyure 2000; Hatfield et al. 2001; Swain et al. 2001), accounting in a large series for 0.17% of sinonasal and nasopharyngeal neoplasms (Thompson and Gyure 2000).

The World Health Organization histological classification includes three types of meningiomas: benign, atypical and malignant (Kleihues et al. 1993). The vast majority of cases belong to the benign type. Histologic subtypes of meningiomas are the meningothelial (syncytial), fibroblastic and transitional (Greenberg 2001). The latter type, which encompasses features of the other types, is frequently characterized by the typical psammoma bodies. Papillary growth pattern is one of the features of malignancy, which is associated with brain infiltration and distant metastases (lung, liver, lymph nodes, heart) (Greenberg 2001). Hyperostosis can be due to bony reaction to the tumor or more frequently to direct invasion (Pieper et al. 1999). However, one should keep in mind that even primary intraosseous meningioma may be associated with either hyperostotic or osteolytic changes (Arana et al. 1996; Devi et al. 2001). In the "en-plaque meningioma", which is more commonly found in the sphenoid wings, the amount of hyperostotic bone is disproportionate to the volume

of the intradural tumor, thereby appearing as a thin layer of tissue investing the inner table of the skull (Derome 1991).

10.2.5.2 Clinical Findings

Primary intracranial meningioma can silently, but relentlessly grow and eventually results in a variety of signs and symptoms. Apart from general complaints induced by cortical irritation (i.e. seizures) or hydrocephalus (i.e., headache), specific signs (cranial nerves paralysis, hormonal deficiency, specific brain stem compression syndromes, head and face deformity secondary to hyperostosis, exophthalmos) related to tumor location may be the clinical hallmarks.

Clinical findings of sinonasal tract meningioma, either primary or secondary, are similar to those of other benign and malignant neoplasms (Perzin and Pushparaj 1984; Thompson and Gyure 2000). Specific locations may be associated with a typical clinical presentation. Meningioma of the olfactory groove may present with anosmia and Foster-Kennedy syndrome (optic atrophy, scotoma in the ipsilateral eye, papilloedema in the contralateral); tuberculum sellae meningioma with early visual loss; cavernous sinus meningioma with proptosis and diplopia; foramen magnum meningioma with nuchal and suboccipital pain, stepwise appendicular sensory and motor deficits.

10.2.5.3 Treatment Guidelines and Prognosis

The mainstay of treatment for meningioma is still surgical resection. Critical parameters affecting resectability include tumor location, size, consistency, vascular and neural involvement, and, in recurrent lesions, prior surgery and/or radiotherapy. New and innovative approaches have been devised to reach and widely expose meningiomas in any location. Furthermore, a better knowledge of risk factors for tumor recurrence changed the philosophy of surgical management. In order to decrease the incidence of recurrences, resection of a meningioma should include not only the gross lesion, but also the involved dura, soft tissue, and bone. Postoperative radiotherapy is advisable for atypical and malignant meningiomas and in selected cases of incomplete resection. Instead of a standard fractionated radiation therapy, stereotactic radiosurgery is indicated for residual tumor (Kondziolka et al. 1991), particularly when cavernous sinus invasion is present (Nicolato et al. 2002). However, the size of the lesion should not exceed 35–40 mm. The morbidity of meningioma treatment is strongly related to its location and extension. Overall, 5-year survival for benign and malignant meningiomas is about 70%; and 55%, respectively (McCarthy et al. 1998). A more favorable prognosis with a 5-year and 10-year disease-free survival of 82% and 79% has been reported for sinonasal meningiomas (Thompson and Gyure 2000).

10.2.5.4 Key Information to Be Provided by Imaging (see Section 10.2.1.4)

10.2.5.5 Imaging Findings

Approximately 33% of benign intracranial meningiomas arise from the skull base, mostly at the level of sphenoid wings. Extracranial spread more frequently occurs along skull base foramina or through direct destruction of middle cranial fossa floor.

CT findings consist of a plaque-like enhancing mass along with hyperostosis of the adjacent bone, particularly at the level of sphenoid wings, being due more frequently to neoplastic invasion rather than reactive sclerosis (Ginsberg 1992).

Relative to cerebral white matter, meningioma is generally hyperintense on T2 and hypointense on T1 images. In comparison with the cortex, it is isointense to hyperintense on T2, more often isointense than hypointense on T1 (Spagnoli et al. 1986). T2 signal is the least predictable, as 50% of lesions are described to be isointense to brain, 40% hyperintense and the remaining 10% hypointense. A certain degree of signal heterogeneity is reported, basically related to tumor vascularity, calcifications, or the presence of cystic areas. Since on unenhanced MR images meningioma may have a signal intensity similar to the brain, its detection is improved by indirect signs such as mass effect, white matter edema, dural thickening, bone hyperostosis (Bradac et al. 1987; Yeakley et al. 1988; Castillo et al. 1989).

Relevant enhancement is invariably obtained after contrast agent administration, even when the lesion is densely calcified. The enhancement is related to the absence of blood barrier in meningioma capillaries. Frequently, thickening and enhancement of the adjacent dura may be observed (*dural tail sign*), a finding that can be due either to direct dural infiltration with accompanying dural congestion or to nonspecific inflammatory changes (Tokumaru et al. 1990; Kawahara et al. 2001).

Meningioma may involve the paranasal sinuses either through skull base encroachment or as ectopic lesions. When the lesion arises from the planum sphenoidale or from the sphenoid sinus roof the bone may be sclerotic or show a characteristic *blistering* consisting of upward expansion; an associated pneumosinus dilatans has been described (Fig. 10.11) (Miller et al. 1996). Primary intranasal meningioma has been reported to have slow growth and to exhibit high degree of enhancement (Spindler et al. 1989; Wolters and Kleinsasser 1985).

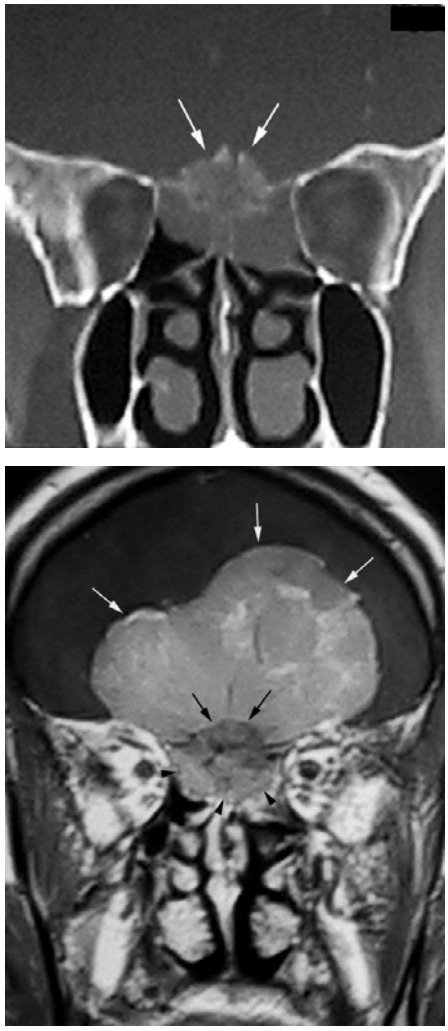


Fig. 10.11a,b. Meningioma of the planum sphenoidale in a 60 years old male who complained anosmia, visual acuity loss and mental changes. **a** Coronal CT shows a large olfactory groove meningioma with *blistering* of the bone toward the inner cranium (*white arrow* on **a**, *black arrow* on **b**) and invasion of the ethmoid. **b** On postcontrast coronal T1-weighted image, both the intracranial (*white arrows*) and extra-cranial (*black arrowheads*) components remarkably enhance. Bone blistering at the olfactory groove is hypointense than the surrounding soft component

10.3 Masticator Space Tumors Extending into the Sinonasal Tract

10.3.1 Definition, Epidemiology, Pattern of Growth

From an anatomical and surgical perspective, an ideal line drawn from the medial pterygoid plate to the glenoid fossa subdivides the extracranial surface of the middle cranial fossa into two compartments, infratemporal and petrotemporal (Kumar et al. 1986). The infratemporal compartment is anterior-lateral located and corresponds to the roof of masticator space, whereas petrotemporal compartment is posterior-medial located and corresponds to the roof of parapharyngeal space.

The masticator space is an irregularly shaped pyramidal space including the temporal and the infratemporal fossae. Two leaflets originating from the superficial layer – which invests the mandible – of the deep cervical fascia and extending upwards to envelope masticatory muscles are considered to limit the masticator space. The roof of the masticator space is made up of the extracranial surface of sphenoid wing and a part of the temporal bone. The anterior-medial limit is the posterior wall of the maxillary sinus. The posterior-medial limit is the medial pterygoid fascia, which separates the masticator space from the prestyloid compartment of the parapharyngeal space. The superficial layer of the temporalis muscle, the zygomatic arch and the superficial layer of the masseter muscle can be considered the lateral limit of the masticator space.

Masticator space tumors have been classified by Conley (1964) according to their site of origin in primary, secondarily extending into the space from adjacent site, and metastases.

Primary tumors account for about 30%, lesions spreading from adjacent sites for about 70%, while metastases are exceedingly rare (Johnson and Maran 1982; Shaheen 1982; Kaplan and Duckert 1984; Cohen and Rosenheck 1998; Hsu et al. 2002).

Each anatomic structure contained within the masticator space may give origin to a tumor so that a great variety of different pathological entities have been observed in this area. Most of them are soft tissue tumors (Lorigan et al. 1989; Toriumi et al. 1989; Abdel-Fattah et al. 1990; Knox et al. 1990; Colmenero et al. 1991; Dohar et al. 1991; Grundfast et al. 1991; Ogren et al. 1991; Papageorge et al. 1992; Rubin and Sadoff 1992; Ummat and Nasser 1992; Shinohara et al. 1993; Woolford et al. 1994;

Kornfehl et al. 1996; Simsir et al. 1996; Goto et al. 1998; Krishnamurthy et al. 1998; Lee et al. 1998; Herman et al. 1999; Lopes et al. 1999; Vajramani et al. 1999; Sarac et al. 2000; Kanazawa et al. 2001; Rangheard et al. 2001; Bianchi et al. 2002; Hicks and Flaitz 2002); but bone, cartilaginous, epithelial, neuroendocrine, neurogenic, odontogenic, and lymphoreticular neoplasms have also been described (Mendelsohn et al. 1983; Cantrell et al. 1984; Tashiro et al. 1988; Akimura et al. 1989; Schreiber et al. 1991; Eavey et al. 1992; Koo et al. 1992; Currie et al. 1993; Weissman et al. 1993; Zhao et al. 1993; Hochberg et al. 1994; Saleh et al. 1994; Sichel et al. 1994; Gormley et al. 1995; McCluggage et al. 1995; Hirabayashi et al. 1997; Galant et al. 1998; Kawai et al. 1998; Jung et al. 1999; Pieper and Al-Mefty 1999; Minoda et al. 2001; Vogl et al. 2001). About 20% of primitive tumors are benign (Shaheen 1982). Differential diagnosis should also include non-neoplastic expansile lesions, which have been rarely observed in the masticator space (O'Ryan et al. 1987; Weisman and Osguthorpe 1988; Patel et al. 1998; Flacke et al. 1999; Reiter et al. 2000; Acarturk and Stofman 2001; Uppal et al. 2002).

Maxillary sinus, orbit, oral cavity, pharynx and parapharyngeal space, parotid gland, middle ear and middle cranial fossa are the sites of origin of other tumors secondarily involving the masticator space (Conley 1964; Shaheen 1982), which are mostly epithelial in origin (Bouaziz et al. 1991).

10.3.2 Clinical Findings

During the early phase of growth, masticator space tumors are asymptomatic or they may be associated with subtle, nonspecific signs and symptoms such as headache, paresthesia or facial pain, unnoticeable paresis of masticator muscles or taste modifications (Shapshay et al. 1976). Trigeminal sensory dysfunction can be determined by testing corneal sensation with blink reflex, but it is easily underestimated. Eustachian tube dysfunction due to compression can be present for a long time before the lesion is identified. Only when the neoplasm considerably increases in size, a cheek swelling can develop. Lateral deviation of the jaw upon opening may be due to paralysis or tumor invasion of the pterygoid muscles or to dysfunction of the temporomandibular joint. Similarly, trismus may be due to several causes: mechanical effect of the tumor, muscle adhesions due to scarring, neoplastic ankylosis of the temporomandibular joint, or pain.

As a general rule, benign tumors tend to respect anatomic boundaries and they expand along soft-tissue planes, or preexisting pathways (i.e., foramen ovale, foramen spinosum, pterygomaxillary fissure, inferior orbital fissure) (Som et al. 1997). Conversely, malignant tumors tend to infiltrate surrounding structures. Symptoms clearly reflect the dominant pattern of spread (Som et al. 1997).

10.3.3 Treatment Guidelines and Prognosis

Due to the heterogeneity of tumors involving the masticator space, therapeutic planning greatly depends on the histology of the lesion. Instead of an open biopsy, which may be indicated only when there is skin involvement, fine needle aspiration cytology or needle biopsy have to be preferred.

Surgery is indicated for most masticator space tumors. Contraindications include patients with lymphoreticular tumors, which are best treated by radiation and/or chemotherapy, patients who are poor surgical candidates due to pulmonary, cardiac, renal, or other significant comorbidities, and patients with disseminated disease.

A major surgical procedure is usually required even for small tumors, in order to obtain a wide exposure, which is essential to perform an en bloc resection of the lesion and to preserve at the same time uninvolved vascular and neural structures, thus optimizing functional and aesthetic results. As a consequence of the location and complexity of the masticator space, several surgical approaches have been developed. They can be summarized as follows: anterior transfacial, lateral preauricular, lateral postauricular, and combined antero-lateral approach (Fisch 1978; Conley and Price 1979; Friedman et al. 1981; Krespi and Sisson 1984; Wetmore et al. 1986; Gates 1988; Krespi 1989; Lawson et al. 1990; Smith et al. 1990; Howard and Lund 1992; Catalano and Biller 1993; Bigelow et al. 1999; Shahinian et al. 1999). Each technique can be associated to a frontal or temporal craniotomy and to a transorbital approach (Sekhar et al. 1987; Seifert and Dietz 1992; Leonetti et al. 1993) to have a better control of critical extensions of the tumor into the cranial fossa/e and into the orbit, respectively.

Anterior transfacial approaches are indicated for sinonasal tumors invading the masticator space, while indications for lateral preauricular approaches

include tumors taking origin from the masticator space and involving the anterior aspect of the temporal bone or the greater wing of the sphenoid bone. Postauricular approaches are designed for lesions extended to the posterior aspect of the temporal bone.

The reconstructing part of the surgical procedure is meant to correct functional and cosmetic deficits and to minimize complications. A water-tight dural closure is required when resection of the dura has been performed. The surgical defect is usually filled by rotating the temporalis muscle or, less frequently, by using a free flap (Sekhar et al. 1987; Bigelow et al. 1999).

The most important sequelae of these procedures can be facial paralysis, face and corneal anesthesia, loss of motor and sensitive functions of the mandibular nerve with asymmetry of jaw opening; further impairment of mastication may derive from resection of the temporo-mandibular join or mandibular ramus (Guinto et al. 1999; Leonetti et al. 2001). Infections and wound necrosis of the reconstructive flap, neurovascular endocranial complications or CSF leak are uncommon events (Sekhar et al. 1987; Bigelow et al. 1999; Guinto et al. 1999).

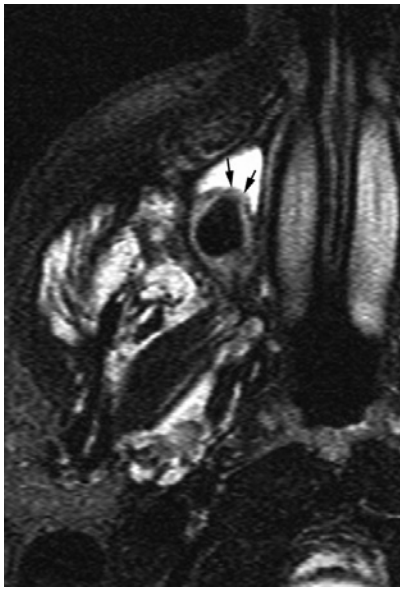
Only a few data regarding the prognosis of patients with tumors of the masticator space, mostly coming from single case reports or from very small series, are available in the literature (Sekhar et al. 1987; Bigelow et al. 1999; Guinto et al. 1999; Shahinian et al. 1999). Generally, benign tumors have an excellent prognosis with a low recurrence rate (Leonetti et al. 2001). Conversely, prognosis of malignant tumors is poor and strongly influenced by histology and by the radicality of the resection (Sekhar et al. 1987; Bigelow et al. 1999; Guinto et al. 1999; Shahinian et al. 1999). Nevertheless, even aggressive malignant neoplasms with a low chance of cure may be candidate to surgery with the intent to palliate symptoms such as pain or trismus (Rosenblum et al. 1990).

10.3.4 Key Information to Be Provided by Imaging

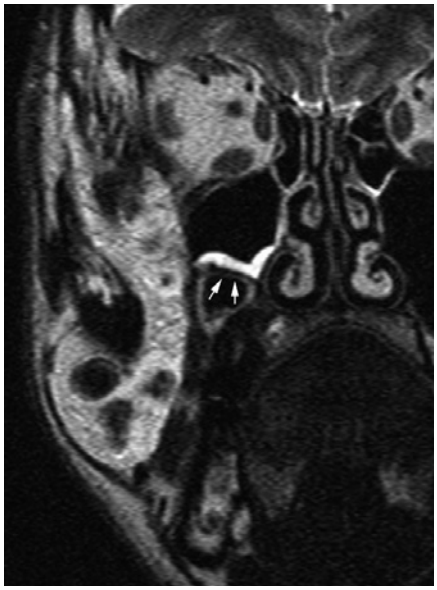
- Differentiation between primary masticator space masses/metastases and neoplasms from adjacent sites secondarily invading the space
- Identification of imaging features suggesting the nature (benign or malignant) or content (solid, cystic, adipose, vascular) of the lesion.
- Assessment of tumor extent and relationship with adjacent critical anatomical structures (pterygopalatine fossa, skull base foramina and fissures, orbit, middle cranial fossa, internal carotid artery)
- Guidance for a needle biopsy
- Identification of regional and distant metastases in aggressive malignant tumors.

10.3.5 Imaging Findings

Some important hallmarks on CT and MR studies help to indicate the masticator space as the site of origin of a lesion. The center of the mass is expected to be within masticator muscles or in the mandible,



a



b

Fig. 10.12a,b. Masticator space hemangioma appears hyperintense on fat-saturated axial T2-weighted sequences, extending around masticator muscles. **a** A double posterior sinus wall with a sharp boundary (*arrows*) is apparently subdividing the maxillary sinus into an anterior hyperintense and posterior hypointense compartments. **b** The *double posterior sinusal wall* sign is related to upward displacement of the sinusal floor by a radicular cyst

anterior to the prestyloid compartment of parapharyngeal space (Fig. 10.12) (Aspestrand and Boysen 1992). As a consequence, the latter is generally posteriorly displaced (Chong and Fan 1996). Secondary invasion by lesions arising from adjacent structures is suggested by the identification of the most probable epicenter of the mass (Fig. 10.13).

As the medial pterygoid muscle fascia attaches to the skull base medial to foramen ovale, the mandibular nerve and its inferior alveolar branch run within the masticator space. This accounts for the need to thoroughly scrutinize this nerve, as it may represent the route for perineural spread into the middle cranial fossa, towards the cavernous sinus (Laine et al. 1990). Perineural spread may also occur when the lesion extends medially into the pterygopalatine fossa, a crossroads between intra and extracranial course of multiple nerve structures.

Destruction of posterolateral maxillary sinus wall is the most common pathway of tumor invasion into the sinonasal tract.

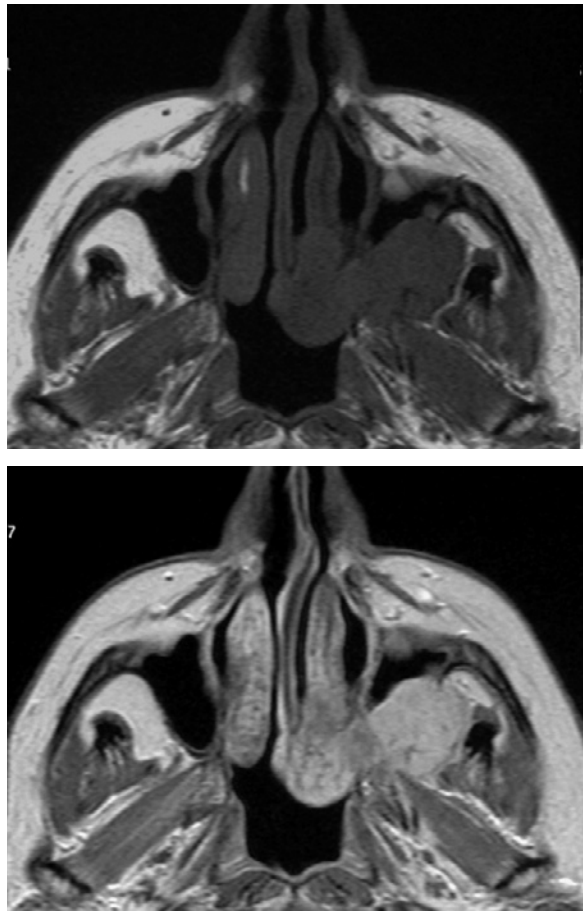


Fig.10.13a,b. Maxillary sinus squamous cell carcinoma invading the left masticator space and pterygopalatine fossa

10.4 Hard Palate Expansile Lesions

10.4.1 Definition, Epidemiology, Pattern of Growth

The palate is an anatomical region which is included into the oral cavity (hard palate) and the oropharynx (soft palate). The hard palate represents the inferior boundary of the nasal cavities and the maxillary sinuses. Malignant neoplasms of the hard palate have been included in this chapter because they can extend into the nose and paranasal sinuses. Malignant neoplasms of the hard palate are rare, accounting for about 2%–5% of oral cavity cancers, they represent about 50% of hard palate neoplasms (Krolls and Hoffman 1976; Pogrel 1994; Inagi et al. 2002). Most tumors have an epithelial origin with squamous cell carcinoma accounting for about half to two thirds of cases (Chung et al. 1980; Petruzzelli and Myers 1994; Truitt et al. 1999; Yorozu et al. 2001). Salivary gland tumors are also frequently observed, being the hard palate the most common localization of minor salivary gland neoplasms (Chung et al. 1980; Tran et al. 1987; Lopes et al. 1998; Truitt et al. 1999; Hyam et al. 2004). Among minor salivary gland tumors of the hard palate, adenoid cystic carcinoma and mucoepidermoid carcinoma are the most common histotypes (Tran et al. 1987; Lopes et al. 1998; Truitt et al. 1999; Yorozu et al. 2001; Jansisyanont et al. 2002; Hyam et al. 2004). Non-epithelial or secondary neoplasms have been rarely reported in the hard palate (Chung et al. 1980; Florio and Hurd 1995; Hefer et al. 1998; Lahoz Zamarro et al. 2001; Moriya et al. 2001; Pritchyk et al. 2002; Chang et al. 2003). The peak incidence is in the second half of the sixth decade for squamous cell carcinoma and in the first half of the same decade for salivary tumors. There is a male predominance in squamous cell carcinoma of about 2:1, whereas salivary tumors are equally distributed in males and females or there is a slight female predominance (Lopes et al. 1998; Truitt et al. 1999; Inagi et al. 2002).

Extension beyond the hard palate is a frequent finding, in particular for squamous cell carcinoma, which is more rapidly growing than salivary tumors, which have a more subtle long-standing clinical history (Kornblut 1987; Pogrel 1994; Beckhardt et al. 1995). Posterior mucosal extension to the soft palate and the nasopharynx is more typical for squamous cell carcinoma, whereas posterosuperior submucosal perineural spread to the sphenopalatine foramen, then to the pterygopalatine fossa, the infratemporal

fossa and through the foramen rotundum, foramen ovale, or inferior orbital fissure to the skull base is a feature of adenoid cystic carcinoma (Beckhardt et al. 1995). Invasion of the floor of the nasal cavity or of the maxillary sinus by direct bone infiltration or through dental sockets is another frequent route of spread for squamous cell cancer (Kornblut 1987). Lymph node metastases are infrequent (Truitt et al. 1999; Yorozu et al. 2001) and the first echelon of nodal drainage are the submandibular and upper deep jugular nodes (I and II levels) (Rouviere 1932).

10.4.2 Clinical Findings

Squamous cell carcinoma usually presents as an infiltrative ulcerated lesion, while salivary tumors are mostly submucosal with a smooth, normal mucosal swelling, sometimes with a bluish appearance (Pogrel 1994; Manganaro et al. 1997). In both cases, the patient can be asymptomatic in the early stage. Squamous cell carcinoma is typically associated with poor oral hygiene in heavy smokers and drinkers. Bleeding, foul odor, dental numbness, ill-fitting dentures, malocclusion, or loose teeth are other manifestation of neoplastic growth. Signs and symptoms of local advanced growth are velopharyngeal insufficiency and hypernasal speech, palatal hypoesthesia, middle ear effusion, hypoesthesia along the mandible or wasting of the temporalis or masseter muscles, trismus, and absence of corneal reflex.

10.4.3 Treatment Guidelines and Prognosis

Surgery is the mainstay for treatment of hard palate neoplasms. Even though radiotherapy can be considered effective for squamous cell carcinoma, the high risk of osteoradionecrosis justifies why it is rarely recommended as a primary treatment. Its use as a complementary post-operative treatment is indicated in advanced diseases.

The extent of the resection is dictated by the local spread of tumor, particularly by perineural extent, which can be suggested by imaging findings but has to be looked for and checked especially in adenoid cystic carcinoma. To this purpose, multiple frozen sections are strongly recommended. Tumors of limited extent not invading the bone may be removed by a simple transoral excision; on the other site, major procedures with a skull base and/or a cervical access and temporary tracheotomy may be required to manage advanced tumors (Futran and Haller 1999; Truitt et al. 1999). When a maxillectomy (inferior, subtotal or total) is required, the oroantral defect can be occluded by a prosthetic obturator, or it can be definitively closed by a temporalis flap or by more sophisticated reconstructive techniques using free flaps (see Chapter 5) (Bernhart et al. 2003). Owing to the low rate of neck metastases, a neck dissection is performed only when there is clinical evidence of lymph node involvement (Shah and Lydiatt 1999).

Conventional postoperative radiation therapy is performed for involved or uncertain surgical margins, perineural invasion, for multiple lymph node metastases or extracapsular spread of at least one metastatic lymph node (Beckhardt et al. 1995; Truitt et al. 1999). A controversy still exists about the efficacy of radiotherapy for adenoid cystic carcinoma: promising results have been obtained with fast neutrons radiotherapy, which is expected to be more effective in this specific tumor than the conventional treatment (Douglas et al. 1996; Prott et al. 2000; Huber et al. 2001).

Survival of patients with malignant tumors of the hard palate is strictly dependent on the local extent and the presence of nodal metastases (Chung et al. 1980; Tran et al. 1987; Truitt et al. 1999; Yorozu et al. 2001). Usually, 5-year survival is higher for adenoid cystic carcinoma and minor salivary gland malignancies. However, when a longer follow-up is considered, a progressive decrease of disease-specific survival for adenoid cystic carcinoma is observed: 5-year survival generally ranges from 87% to 93% and 10-year survival from 75% to 80% (Tran et al. 1987; Beckhardt et al. 1995; Truitt et al. 1999). In squamous cell carcinoma, a better survival is reported when surgery is used as a primary modality treatment, with a 5-year survival up to 76% (Truitt et al. 1999) compared to 48% for radiotherapy (Yorozu et al. 2001).

10.4.4 Key Information to Be Provided by Imaging

- Identification and assessment of bony invasion as well as of submucosal, subperiosteal and perineural spread in malignant tumors
- Identification of regional and distant metastases in aggressive malignant tumors.

10.4.5 Imaging Findings

Salivary gland carcinomas and squamous cell carcinoma which arise from the palate can spread along the greater and lesser palatine nerves to reach the skull base and eventually extend intracranially. MR imaging is superior to CT either in primary tumor detection and in the assessment of perineural extent (Ginsberg and DeMonte 1998; Caldemeyer et al. 1998; Maroldi et al. 1999; Tomura et al. 1999; Blandino et al. 2000). Proper selection of the imaging technique and of the MR parameters is essential for detecting both small hard or soft palate tumors and, particularly, for the identification of the subtle changes associated with perineural spread (Ginsberg 1999). Unenhanced T1 coronal images increase the detectability of even small palatal lesions because the hypointense tumor stands upon the high signal intensity of the hard palate, its fat tissue component acting as a natural contrast (Fig. 10.14). Similarly, on contrast-enhanced MR sequences fat saturation improves the identification of both the primary tumor and of the involved nerves. Thin slices are necessary in all cases. Sagittal MR planes are useful in case of soft palate carcinoma.

Particular attention has to be paid to the inferior opening of the greater and lesser palatine canals, where tumor can gain access to the palatine nerves. Axial and coronal unenhanced T1 and fat-saturated post-contrast images should be carefully scrutinized to detect the effacement of fat surrounding the opening of the canal by the hypointense tumor, or the enhancing nerve or the neoplastic tissue replacing the fat within the pterygopalatine fossa (Fig. 10.15). Sub-millimetric VIBE sequences are well suited at identifying subtle nerve abnormalities at this level.

While MR findings are based on direct signs of nerve involvement, CT has to rely on indirect findings, such as effacement of fat tissue within fissures, canals and foramina or the occurrence of bone erosion, which is usually late (Curtin 1998). Once the palatine nerves have been involved, perineural spread can progress toward the sphenopalatine ganglion, and from this step can further extend either toward the skull base foramina or canals (*centripetal* direction), or perineural tumor can grow along the opposite direction (*centrifugal*). Detailed description of the patterns of growth, intracranial extent and imaging findings is reported in Chapter 9, section 9.2.5.1.

10.5 Odontogenic and Nonodontogenic Cysts and Tumors

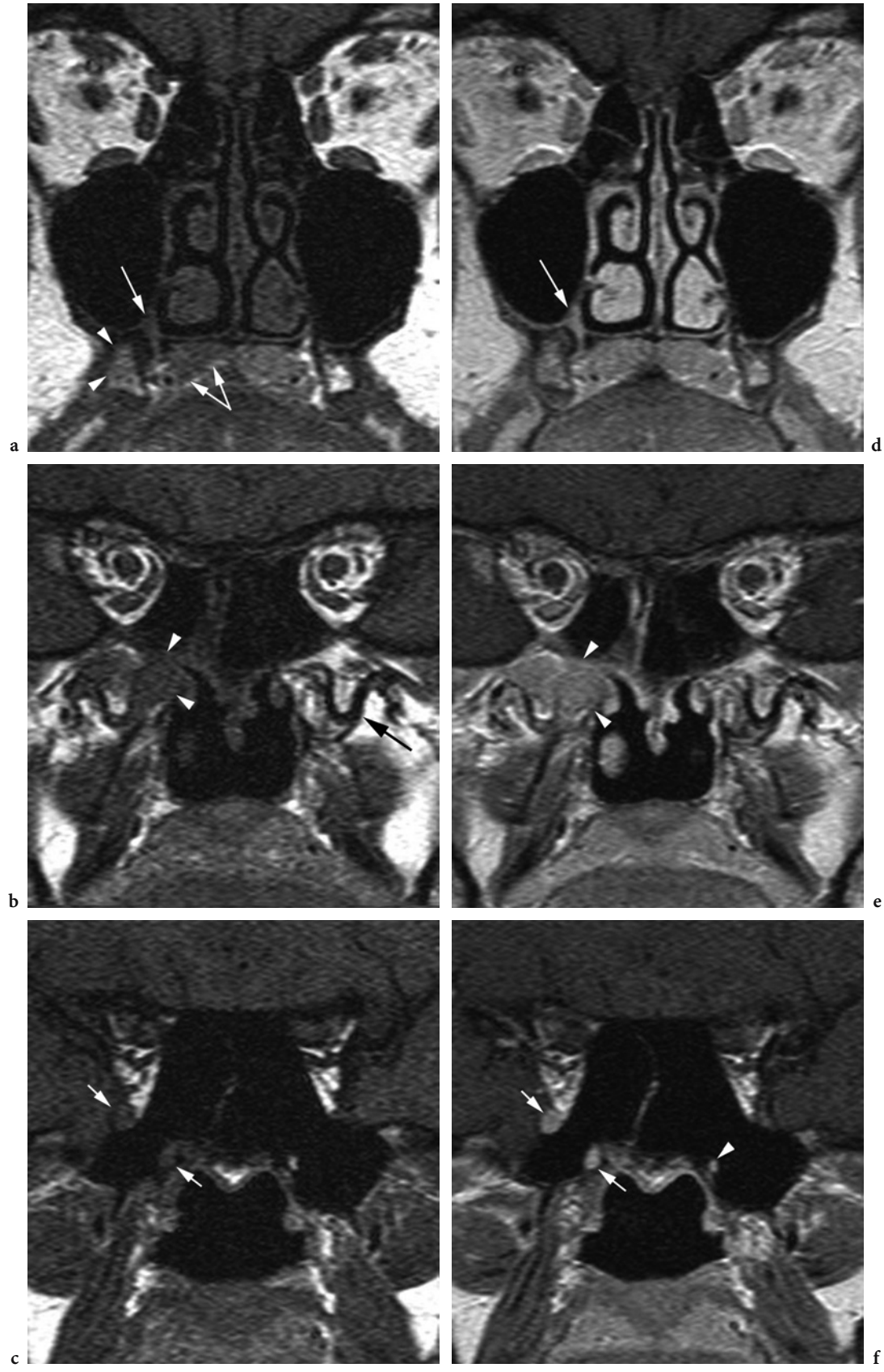
10.5.1 Introduction and Definition

A wide range of cystic lesions and tumors of odontogenic origin involving the mandible and the maxilla have been described in the literature.

This chapter will not provide a detailed description of any of them, but instead an overview of the common clinical aspects together with some peculiarities of those lesions which more often involve the adjacent structures of the sinonasal tract.

According to pathogenesis, epithelial cysts of the jaws are classified in developmental and inflammatory. Developmental cysts are subdivided in odontogenic and non odontogenic (Kramer et al. 1992). Since their epithelial lining is composed by squamous cells, the developmental odontogenic cysts are further divided by their topographic relation to specific structures, rather than by histologic features.

Fig. 10.14a–f. Adenoid cystic carcinoma with perineural spread in a 32 years old female who complained hypoesthesia of right cheek for 8 months. Unenhanced coronal T1-weighted images show a small hypointense neoplasm of the hard palate (**a**) (*double arrows*) associated with fat signal effacement at the opening of the greater palatine canal (*arrow*) and hypointense signal in the adjacent alveolar process (*arrowheads*). **b** At the level of the upper pterygopalatine fossa, a soft tissue mass (*arrowheads*) replaces the fat content, effacing the pterygopalatine artery, clearly demonstrated on the contralateral side (*black arrow*). **c** Thickening of right maxillary and vidian nerves is present (*arrows*). **d** Postcontrast images show enhancement of tumor tissue, which becomes less detectable in the hard palate (when compared to unenhanced image), stands out at the greater palatine canal opening (*arrow*) and give rise to *mild* bone marrow enhancement in the adjacent alveolar process. Enhancement of pterygopalatine neoplastic component (*arrowheads* in **e**) and both maxillary and vidian nerve (*arrows* in **f**) is clearly demonstrated. Contralateral vidian nerve (*arrowhead*). Note that the pterygopalatine mass is larger than the primitive tumor in the hard palate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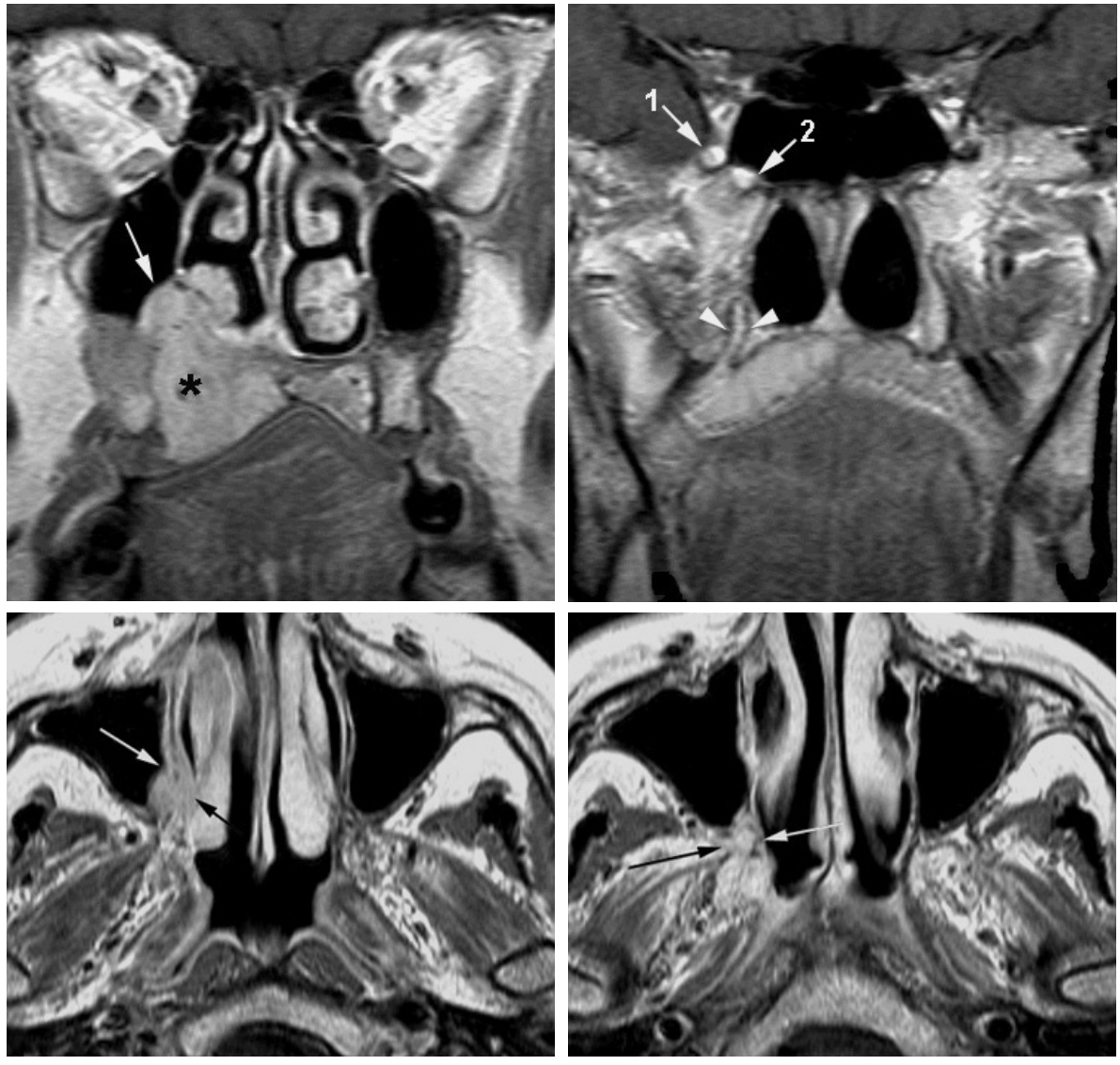


Fig. 10.15a–d. Adenoid cystic carcinoma with perineural spread Contrast enhanced T1-weighted coronal (**a–b**) and axial (**c–d**) planes. **a** The neoplasm (*asterisk*) arise from the right hard palate, abutting the alveolar recess of maxillary sinus (*arrow*). **b** Perineural spread is detected along palatine nerves (*arrowheads*) and more centrifugally along both the maxillary nerve (*1*) and vidian nerve (*2*). **c** Submucosal tumor growth is shown along both surfaces of medial antral wall (*arrows*) reaching posteriorly the pterygopalatine fossa. **d** Solid and enhancing tissue fills the widened pterygopalatine fossa (*arrows*)

Odontogenic tumors are very rare lesions arising from the tissues of developing teeth. They are believed to result from an interruption of odontogenesis or from the reactivation of special tissues participating into odontogenesis. Many histologic variants with different presentation and behavior have been recognized. Their classification is based on histogenetic, organogenetic, and embryologic aspects of tooth development.

10.5.2 Epidemiology, Clinical Findings and Treatment Guidelines

The most frequent jaw cyst is the apical radicular cyst, followed by dentigerous cyst and odontogenic keratocyst (Mosqueda-Taylor et al. 1997).

Radicular cyst results from pulp necrosis due to inflammation. The Malassez epithelial rests in the periodontal ligament are stimulated to produce a periapi-

cal granuloma, which subsequently evolves in a cyst (apical radicular cyst). Rarely, are these cysts found laterally to the root (lateral radicular cyst). Radicular cysts are most frequently seen between 10 and 40 years, with a 60% male preponderance. Most of them develop in the anterior maxillary region (37%–82% are found in the maxilla) (McDaniel 1999). They are often asymptomatic, while pain and swelling are present if the cyst becomes inflamed. Radicular cysts are lined by stratified nonkeratinizing squamous epithelium, with a dense infiltrate of chronic inflammatory cells. Most lesions are treated by endodontic therapy of the involved tooth. Surgical removal can be added in cases of failure.

Dentigerous (follicular) cyst is the most common developmental odontogenic cyst (Mosqueda-Taylor et al. 1997). Its location surrounding the crown of an impacted or partially unerupted tooth is quite typical. The cyst is commonly observed in the second, third and fourth decades with a male predominance. The most frequent sites are the region of impacted teeth (mandibular and maxillary third molars, maxillary canines, and mandibular premolars, in decreasing order of frequency). Most of them are small and asymptomatic although they can grow to reach even remarkable size. Owing to their origin from the follicular epithelium, which has a great potential for degeneration, dentigerous cysts are sometimes associated with benign or malignant neoplasms. In view of this peculiarity, removal of impacted teeth with a large pericoronal radiolucency is recommended.

Odontogenic keratocyst is an aggressive lesion characterized by the production of keratin, without a specific topographic location. It is the second most common developmental odontogenic cyst (Mosqueda-Taylor et al. 1997; Regezi 2002). Multiple odontogenic keratocysts can be part of the basal cell nevus syndrome, also known as Gorlin syndrome (5% of cases of parakeratinized type) (Oda et al. 2000; Regezi 2002). There is an age predominance from the second to the fourth decade, and males are most frequently affected than women (McDaniel 1999). The posterior region of the mandible is the preferred site of origin, followed by the posterior region of the maxilla (McDaniel 1999). Swelling and drainage are the most frequent findings (50% of patients are symptomatic) followed by pain, paresthesia and trismus (Brannon 1976). Cysts producing orthokeratin have a lower tendency to recur compared to those producing parakeratin (2.2% versus 42.6%) (Crowley et al. 1992). Surgical excision with free margins is the treatment of choice (Bataineh and al Qudah 1998; Zhao et al. 2002).

Nasopalatine duct cyst (incisive canal cyst) taking origin from embryonic epithelial remnants of the nasopalatine duct or incisive canal, is the most frequent developmental non-odontogenic cyst (Daley et al. 1994). It is generally observed in the middle age (fourth to sixth decade) with a slight predilection for males (Vasconcelos et al. 1999a). Palatal swelling is the most common complaint, rarely associated with root resorption of incisor teeth; most cases are asymptomatic (Vasconcelos et al. 1999a). The cyst is lined by stratified squamous epithelium and/or respiratory epithelium. Differential diagnosis includes odontogenic keratocyst and periapical cyst, when located in the midline (Neville et al. 1997). Enucleation is generally sufficient to avoid recurrences, which are reported in less than 10% of cases (Spinelli et al. 1994).

Nasolabial (nasoalveolar) cyst is a developmental non-odontogenic cyst originating from the epithelial remnants of the nasolacrimal duct and developing in soft tissues (Vasconcelos et al. 1999b). Most cases are observed in the middle-aged females (Vasconcelos et al. 1999b). The most common clinical finding is an asymptomatic soft tissue swelling involving the canine fossa/nasal alar base region. Microscopically, this cystic structure is composed of a fibrous capsule with an unremarkable layer of pseudostratified columnar epithelium (Vasconcelos et al. 1999b). The treatment of choice is surgical excision by sublabial approach rarely followed by recurrence (Vasconcelos et al. 1999b; Choi et al. 2002). Also transnasal endoscopic marsupialization has been described (Su et al. 1999).

Benign odontogenic tumors are rare. The most frequent is *odontoma*, which accounts for about 50% of all odontogenic tumors. It is considered quite similar to a malformative lesion, since all dental tissues can be observed within it. Odontomas are subdivided in compound and complex in relation to the distribution of dental tissues (more or less ordered). The second most common benign odontogenic tumor is *ameloblastoma*, which is the most aggressive. Adenomatoid odontogenic tumor, which preferably arises in the maxilla, is the third most frequent lesion.

Most benign odontogenic tumors are observed in the middle age, and there is a various distribution among males and females. Compound odontoma is more frequently observed in the anterior part of the maxilla. A painless swelling is the only complaint in almost all type of tumors with the exception of odontoma and cementoblastoma. If tumors reach a relevant size, pain, loss of teeth, malocclusion, ulceration, rhinorrhea and nasal obstruction (in upper jaw locations), can be observed.

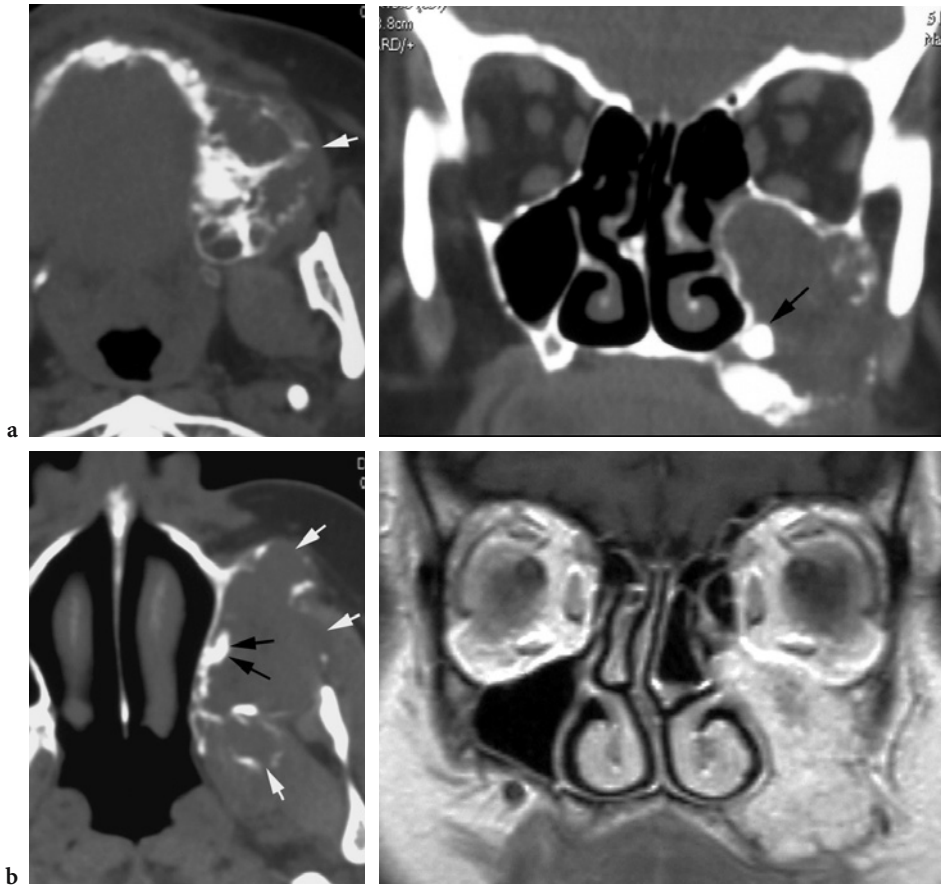


Fig. 10.16a-d. Calcifying odontogenic cyst (Pindborg tumor). **a** Axial CT scans show the irregular destruction of the alveolar process with spread into the buccal fat pad (*arrow*). **b** The lesion extends into the masticator space (*arrows*), irregular intratumoral densities are shown *(black arrows* in **b** and **c**). Mottled enhancing appearance of the lesion on post-contrast T1 coronal plane (**d**).

Enucleation or limited surgical excision is a proper treatment for most benign odontogenic tumors. On the other site, ameloblastoma, ameloblastic fibroma, odontoameloblastoma, calcifying epithelial odontogenic tumor (Fig. 10.16) and myxoma require wide free margins to avoid recurrences.

Malignant odontogenic tumors are divided in carcinomas and sarcomas (Kramer et al. 1992). The most frequent odontogenic malignant neoplasm is the *primary intraosseous carcinoma* (McDaniel 1999). The diagnosis requires that metastatic disease had been excluded (Eversole 1999). It is observed in elderly patients, with a predilection for the mandibular body (Suei et al. 1994). Pain and paresthesia can derive from compression of the alveolar nerve. Regional and distant metastases are frequent and a 30% to 40% 5-year survival is reported (To et al. 1991).

Malignant ameloblastic tumors include *malignant ameloblastoma*, which shows the same histologic features of benign ameloblastoma, but it is usually associated with late distant metastases to the lung (less frequently to the brain, viscera, skin, and bone), and ameloblastic carcinoma, in which clear histologic signs of malignancy are evident (Eversole 1999). The latter has no gender predilection, is more common in the mandible, and generally displays a local aggressiveness rather than a tendency to metastasize (Eversole 1999).

Odontogenic sarcoma is the malignant counterpart of the ameloblastic fibroma (ameloblastic fibrosarcoma), being characterized by benign epithelium with a malignant fibrous stroma. When it exhibits dysplastic dentin, it is called ameloblastic fibrodentinosarcoma, and when focal deposits of dysplastic enamel proteins are present, it is called ameloblastic fibro-odontosarcoma (Bregni et al. 2001). Finally, when a jaw tumor with an ameloblastic fibroma-like pattern displays both a carcinomatous and a malignant spindle cell component it is defined as odontogenic carcinosarcoma (Slater 1999; Slama et al. 2002).

10.5.3 Key Information to be Provided by Imaging

- Differentiation between cystic and solid lesions
- Presence of imaging features highly suggestive for a specific lesion
- Relationship with adjacent anatomical structures (hard palate, masticator space structures, pterygopalatine fossa)
- Assessment of distant metastases for malignant tumors

10.5.4 Imaging Findings

Maxillary sinus and hard palate can be encroached by a variety of lesions of odontogenic origin; the most commonly observed are radicular cyst, dentigerous cyst, odontogenic keratocyst, ameloblastoma, and non-odontogenic (developmental cysts), such as nasopalatine and nasolabial cyst .

All the odontogenic lesions share some common imaging features, such as radiolucency on plain films and hypodensity on CT. Ameloblastoma and, less frequently, odontogenic keratocyst may have a multilocular appearance being crossed by coarse and curved internal septa. Dystrophic calcifications may be detected, especially in radicular cysts. At their periphery, all these lesions are usually bordered by a thin corticated rim. Nonetheless, this outer boundary may be effaced or may appear sclerotic (particularly after secondary infections in cystic lesions), thus making identification of the site of origin of the lesion – inside or outside the maxillary sinus – extremely difficult. On coronal native or reconstructed MPR coronal images, the identification of such a thin bony plate between the lesion and maxillary sinus cavity is the hallmark to distinguishing extra-antral odontogenic lesions from antral lesions (Han et al. 1995).

Evaluation of the site of origin of the lesion and assessment of its relationship with adjacent teeth is useful in distinguishing among some of the more frequent odontogenic lesions involving the adjacent sinonasal tract structures.

Radicular cyst arises from epithelial cells rests after inflammatory stimulation. It is located close to the apex of a non-vital tooth. In the maxilla, it is more frequently located around incisors and canines roots. CT findings and signal intensities on MR are nonspecific, they mainly consist of a unilocular, purely cystic pattern, with homogeneous fluid (Fig. 10.17) (Minami et al. 1996).

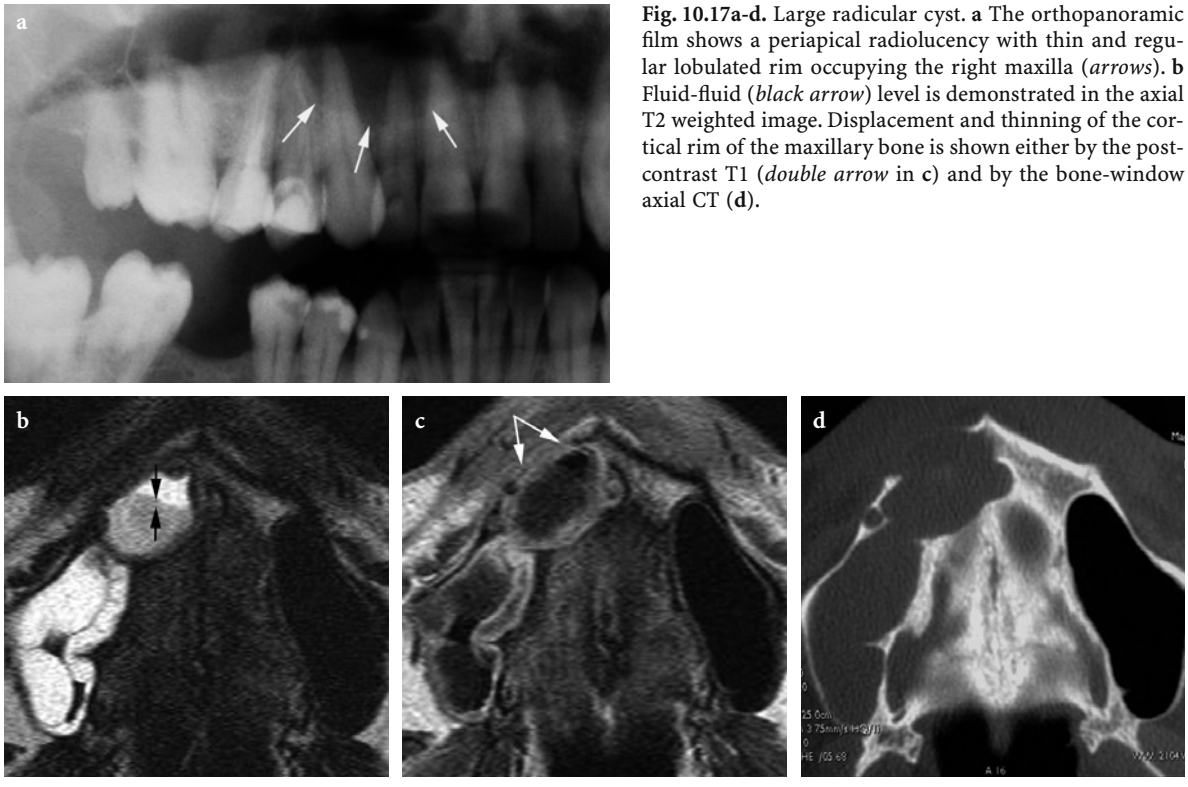
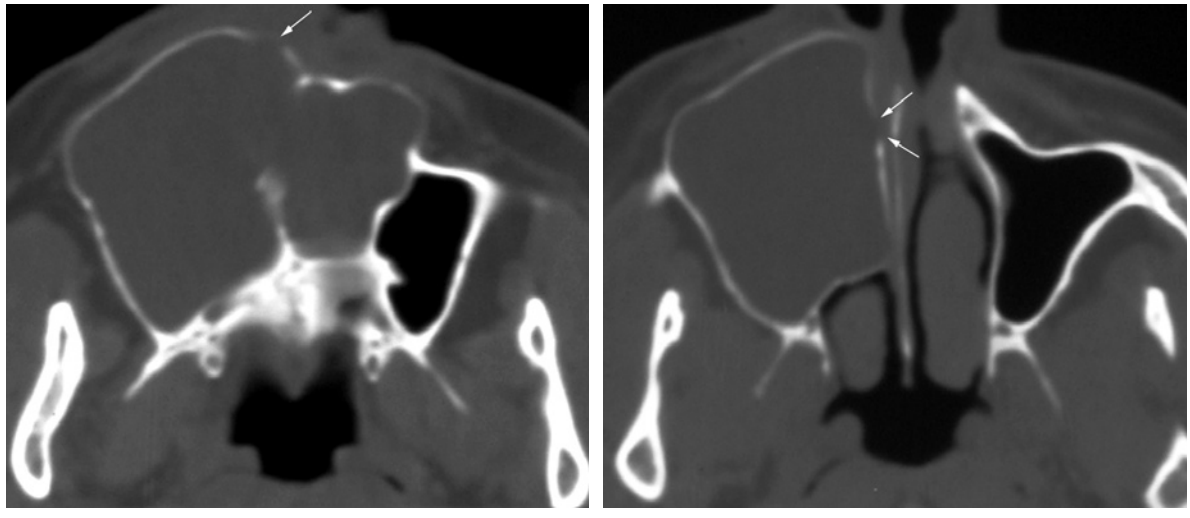


Fig. 10.17a-d. Large radicular cyst. **a** The orthopanoramic film shows a periapical radiolucency with thin and regular lobulated rim occupying the right maxilla (*arrows*). **b** Fluid-fluid (*black arrow*) level is demonstrated in the axial T2 weighted image. Displacement and thinning of the cortical rim of the maxillary bone is shown either by the post-contrast T1 (*double arrow* in **c**) and by the bone-window axial CT (**d**).

Otherwise than radicular cyst, *dentigerous cyst* develops around – eventually "containing" – the crown of an unerupted tooth, which is more frequently a canine in the maxilla, or supernumerary tooth (Som et al. 1992). It has to be distinguished from a normal dental follicle. If more than 20 mm in size, a dental follicle is probably developing a dentigerous cyst. When the dentigerous cyst grows upwards, the maxillary sinus floor is remodeled; its bone content can be reabsorbed. Typically, the floor is posteriorly and superiorly displaced, accounting for a *double posterior sinus wall* on axial CT images, where the posterior one is the true sinusal wall and the anterior one is actually the displaced floor. Sagittal or coronal sections improve this distinction. The displaced tooth is clearly demonstrated within the cyst (Okita et al. 1991). Except for the tooth, which appears hypointense, or totally signal void, on MR sequences, the cyst has a homogeneous signal content, hyperintense on T2 and hypo to intermediate on T1 (Hisatomi et al. 2003).

Odontogenic keratocyst is far most common in the posterior body or ramus of the mandible than in the maxilla. This lesion, which arises from dental lamina, may have a pericoronal position, thus being difficult to distinguish from dentigerous cyst on radiographic films. On CT a unilocular or multilocular cystic lesion is seen, invading the adjacent "scalloped" bone and presenting thin, reactive and smooth bony walls (Fig. 10.18). MR images of odontogenic keratocyst show a unilocular or multilocular lesion, the appearance of cyst walls can greatly vary: they can be absent or thin or even thick. The cystic contents usually is heterogeneous, the signal can be predominantly intermediate to low on T2, and intermediate to high on T1 (Hisatomi et al. 2003; Minami et al. 1996). Contrast enhanced MR is superior to CT in detecting features that enable to distinguish the odontogenic keratocyst from other cysts, such as focal rim enhancement and iso-intense intraluminal soft-tissue components which correlate with the histological findings of focal inflammatory ulceration of the cyst lining, orthokeratosis and cell debris (Janse van Rensburg et al. 1997). MR findings are particularly useful in the diagnosis of nevoid basal cell carcinoma syndrome, characterized by multiple odontogenic keratocysts, by the early development of multiple basal cell carcinomas and skeletal development abnormalities (Palacios et al. 2004).

Nasopalatine and nasolabial are the most frequent non-odontogenic developmental cysts of the maxilla (Holtmann et al. 1985). The nasopalatine cyst develops from the nasopalatine-incisive canal area to present as a well-defined radiolucency above or between the root apices of the central incisors, exceeding 0.6 mm in size (Verbin and Barnes 2001). On CT the cyst has a midline location, shows smooth expansion with sclerotic margins and displacement of teeth apices. Radicular cysts differ in that the teeth apices are within the cyst rather than being displaced (Pevsner et al. 2000). On MR, its keratin and viscous fluid contents account for high signal intensity on T1 (Hisatomi et al. 2001).



a **b**

Fig. 10.18a,b. a A large odontogenic keratocyst arises from right maxilla and grows towards the contralateral side. **b** The cyst fills the right maxillary sinus. Extensive remodeling and thinning of bone is observed. Focal bony dehiscences are demonstrated (*arrows* in **a** and **b**)

Nasolabial cyst, thought to origin fro the nasolacrimal duct apparatus, is generally located in the nasal vestibule or ala or in the upper lip (Som and Norton 1991). It is an extra-osseous soft tissue mass that may remodel the adjacent bone (Cure et al. 1996).

Ameloblastoma is a locally invasive but almost invariably slow growing benign tumor arising from remnants of odontogenic epithelium (dental lamina, dental organ). In the maxilla, ameloblastoma more frequently develops in the third molar region, it behaves more aggressively than in its mandibular counterpart (Jackson et al. 1996). Radiographically, the most typical finding includes a multiloculated lytic lesion, in which the number and arrangement of internal "septa" may give the lesion a honeycomb-like appearance. On CT, maxillary ameloblastoma appears as a multilocular solid and cystic lesion, non-enhancing, with well-defined thin cystic borders (Hertzanu et al. 1984) MR findings encompass: mixed solid and cystic pattern, irregularly thick walls, papillary projections, and strong enhancement of solid components including papillary projections, walls, and septa (Minami et al. 1996). Unilocular lesions are more frequent in the mandible (Philipsen and Reichart 1998).

References

Abdel-Fattah HM, Adams GL, Wick MR. Hemangiopericytoma of the maxillary sinus and skull base. Head Neck 1990;12:77-83.

Acarturk TO, Stofman GM (2001) Posttraumatic epidermal inclusion cyst of the deep infratemporal fossa. Ann Plast Surg 46:68-71

Akimura T, Kameda H, Abiko S et al (1989) Infrasellar craniopharyngioma. Neuroradiology 31:180-183

Arana E, Diaz C, Latorre FF et al (1996) Primary intraosseous meningiomas. Acta Radiol 37:937-942

Arslantas A, Artan S, Oner U et al (2003) Detection of chromosomal imbalances in spinal meningiomas by comparative genomic hybridization. Neurol Med Chir (Tokyo) 43:12-18; discussion 19

Aspestrand F, Boysen M (1992) CT and MR imaging of primary tumors of the masticator space. Acta Radiol 33:518-522

Bataineh AB, al Qudah M (1998) Treatment of mandibular odontogenic keratocysts. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 86:42-47

Batsakis JG (1979) Tumors of the head and neck : clinical and pathological considerations. 2d edition Baltimore: Williams & Wilkins

Beckhardt RN, Weber RS, Zane R et al (1995) Minor salivary gland tumors of the palate: clinical and pathologic correlates of outcome. Laryngoscope 105:1155-1160

Bernhart BJ, Huryn JM, Disa J et al (2003) Hard palate resection, microvascular reconstruction, and prosthetic restoration: a 14-year retrospective analysis. Head Neck 25:671-680

Bernstein ML, Buchino JJ (1983) The histologic similarity between craniopharyngioma and odontogenic lesions: a reappraisal. Oral Surg Oral Med Oral Pathol 56:502-511

Bianchi B, Poli T, Bertolini F et al (2002) Malignant hemangiopericytoma of the infratemporal fossa: report of a case. J Oral Maxillofac Surg 60:309-312

Bigelow DC, Smith PG, Leonetti JP et al (1999) Treatment of malignant neoplasms of the lateral cranial base with the combined frontotemporal-anterolateral approach: five-year follow-up. Otolaryngol Head Neck Surg 120:17-24

Binkhorst CD, Schierbeek P, Petten GJW (1957) Neoplasms of the notochord. Acta Otolaryngol 47:10-20

Black PM, Carroll R, Glowacka D et al (1994) Platelet-derived growth factor expression and stimulation in human meningiomas. J Neurosurg 81:388-393

Blandino A, Gaeta M, Minutoli F et al (2000) CT and MR findings in neoplastic perineural spread along the vidian nerve. Eur Radiol 10:521-526

Bondy M, Ligon BL (1996) Epidemiology and etiology of intracranial meningiomas: a review. J Neurooncol 29:197-205

Bottles K, Beckstead JH (1984) Enzyme histochemical characterization of chordomas. Am J Surg Pathol 8:443-447

Bouaziz A, Chabardes E, Laccourreye O et al (1991) Extension to the infratemporal fossa of malignant tumors of the face. Ann Otolaryngol Chir Cervicofac 108:113-118

Bourgouin PM, Tampieri D, Robitaille Y et al (1992) Low-grade myxoid chondrosarcoma of the base of the skull: CT, MR, and histopathology. J Comput Assist Tomogr 16:268-273

Bouropoulou V, Bosse A, Roessner A et al (1989) Immunoistochemical investigation of chordomas: Histogenetic and differential diagnostic aspects. Current Topics in Pathology 80:183-203

Bradac GB, Riva A, Schorner W et al (1987) Cavernous sinus meningiomas: an MRI study. Neuroradiology 29:578-581

Brannon RB (1976) The odontogenic keratocyst. A clinicopathologic study of 312 cases. Part I. Clinical features. Oral Surg Oral Med Oral Pathol 42:54-72

Bregni RC, Taylor AM, Garcia AM (2001) Ameloblastic fibrosarcoma of the mandible: report of two cases and review of the literature. J Oral Pathol Med 30:316-320

Bret P, Beziat JL (1993) Craniopharyngiome sphenoido-nasopharynge. Un cas avec exerese radicale par maxillotomie de Le Fort I. Neurochirurgie 39:235-240

Brown E, Hug EB, Weber AL (1994) Chondrosarcoma of the skull base. Neuroimaging Clin N Am 4:529-541

Bulow B, Attewell R, Hagmar L et al (1998) Postoperative prognosis in craniopharyngioma with respect to cardiovascular mortality, survival, and tumor recurrence. J Clin Endocrinol Metab 83:3897-3904

Caldemeyer KS, Mathews VP, Righi PD et al (1998) Imaging features and clinical significance of perineural spread or extension of head and neck tumors. Radiographics 18:97-110; quiz 147

Cantrell RW, Kaplan MJ, Atuk NO et al (1984) Catecholamine-secreting infratemporal fossa paraganglioma. Ann Otol Rhinol Laryngol 93:583-588

Cappabianca P, Cavallo LM, Colao A et al (2002) Endoscopic endonasal transphenoidal approach: outcome analysis of 100 consecutive procedures. Minim Invasive Neurosurg 45 193-200

Carmel PW (1990) Brain tumors of disordered embryogenesis.

In: Youmans JR (ed) Neurological surgery : a comprehensive reference guide to the diagnosis and management of neurosurgical problems. Saunders, Philadelphia, pp 3223-3249

Castillo M, Davis PC, Ross WK et al (1989) Meningioma of the chiasm and optic nerves: CT and MR findings. J Comput Assist Tomogr 13:679-681

Catalano PJ, Biller HF (1993) Extended osteoplastic maxillotomy. A versatile new procedure for wide access to the central skull base and infratemporal fossa. Arch Otolaryngol Head Neck Surg 119:394-400

Chakrabarty A, Mitchell P, Bridges LR (1998) Craniopharyngioma invading the nasal and paranasal spaces, and presenting as nasal obstruction. Br J Neurosurg 12:361-363

Chang CC, Rowe JJ, Hawkins P et al (2003) Mantle cell lymphoma of the hard palate: a case report and review of the differential diagnosis based on the histomorphology and immunophenotyping pattern. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 96:316-320

Cheddadi D, Triki S, Gallet S et al (1996) Obstruction rhinopharyngee neonatale par craniopharyngiome. Arch Pediatr 3:348-351

Chen HJ (2002) The neurological abnormalities and operative findings in the transcallosal approach for large juxtasellar-ventricular craniopharyngiomas. J Clin Neurosci 9:159-163

Cho DY, Liau WR (2002) Comparison of endonasal endoscopic surgery and sublabial microsurgery for prolactinomas. Surg Neurol 58:371-6

Choi JH, Cho JH, Kang HJ et al (2002) Nasolabial cyst: a retrospective analysis of 18 cases. Ear Nose Throat J 81:94-96

Chong VF, Fan YF (1996) Pictorial review: radiology of the masticator space. Clin Radiol 51:457-465

Choux M, Lena G, Genitori L. Le craniopharyngiome de l'enfant. Neurochirurgie (Paris) 1991;37(suppl1):12-165.

Chung CK, Johns ME, Cantrell RW et al (1980) Radiotherapy in the management of primary malignancies of the hard palate. Laryngoscope 90:576-584

Cianfriglia F, Pompili A, Occhipinti E (1978) Intracranial malignant cartilaginous tumours. Report of two cases and review of literature. Acta Neurochir (Wien) 45:163-175

Ciric I, Mikhael M, Stafford T et al (1983) Transsphenoidal microsurgery of pituitary macroadenomas with long-term follow-up results. J Neurosurg 59:395-401

Cohen HV, Rosenheck AH (1998) Metastatic cancer presenting as TMD. A case report. J N J Dent Assoc 69:17-19

Colli B, Al-Mefty O (2001) Chordomas of the craniocervical junction: follow-up review and prognostic factors. J Neurosurg 95:933-943

Colmenero C, Rivers T, Patron M et al (1991) Maxillofacial malignant peripheral nerve sheath tumours. J Craniomaxillofac Surg 19:40-46

Conley J, Price JC (1979) Sublabial approach to the nasal and nasopharyngeal cavities. Am J Surg 138:615-618

Conley JJ (1964) Tumors of the Infratemporal Fossa. Arch Otolaryngol 79:498-504

Corbetta S, Pizzocaro A, Peracchi M et al (1997) Multiple endocrine neoplasia type 1 in patients with recognized pituitary tumours of different types. Clin Endocrinol (Oxf) 47:507-512

Cordera S, Bottacchi E, D'Alessandro G et al (2002) Epidemiology of primary intracranial tumours in NW Italy, a population based study: stable incidence in the last two decades. J Neurol 249:281-284

Cottier JP, Destrieux C, Brunereau L et al (2000) Cavernous sinus invasion by pituitary adenoma: MR imaging. Radiology 215:463-469

Crockard HA, Steel T, Plowman N et al (2001) A multidisciplinary team approach to skull base chordomas. J Neurosurg 95:175-183

Crotty TB, Scheithauer BW, Young WF, Jr. et al (1995) Papillary craniopharyngioma: a clinicopathological study of 48 cases. J Neurosurg 83:206-214

Crowley TE, Kaugars GE, Gunsolley JC (1992) Odontogenic keratocysts: a clinical and histologic comparison of the parakeratin and orthokeratin variants. J Oral Maxillofac Surg 50:22-26.

Cure JK, Osguthorpe JD, Van Tassel P (1996) MR of nasolabial cysts. AJNR Am J Neuroradiol 17:585-588

Currie WJ, Thompson WD, Stassen LF (1993) Adenocarcinoma: the use of computed tomography and magnetic resonance imaging for investigation of a tumour in the infratemporal fossa. Dentomaxillofac Radiol 22:155-158

Curtin HD (1998) Detection of perineural spread: fat is a friend. AJNR Am J Neuroradiol 19:1385-1386

Cushing H (1932) The craniopharyngiomas. In: Cushing H (ed) Intracranial tumours; notes upon a series of two thousand verified cases with surgical-mortality percentages pertaining thereto. C. C. Thomas, Springfield, 93-98

D'Alessandro G, Di Giovanni M, Iannizzi L et al (1995) Epidemiology of primary intracranial tumors in the Valle d'Aosta (Italy) during the 6-year period 1986-1991. Neuroepidemiology 14:139-146

Daley TD, Wysocki GP, Pringle GA (1994) Relative incidence of odontogenic tumors and oral and jaw cysts in a Canadian population. Oral Surg Oral Med Oral Pathol 77:276-280

Dalprà L, Malgara R, Miozzo M et al (1999) First cytogenetic study of a recurrent familial chordoma of the clivus. Int J Cancer 81:24-30

Davis PC, Hoffman JC, Jr., Spencer T et al (1987) MR imaging of pituitary adenoma: CT, clinical, and surgical correlation. AJR Am J Roentgenol 148:797-802

DeMonte F, Ginsberg LE, Clayman GL (2000) Primary malignant tumors of the sphenoidal sinus. Neurosurgery 46:1084-1091

Derome PJ (1991) Bony reaction and invasion in meningiomas. In: Al-Mefty O (ed) Meningiomas. Raven Press, New York, pp 169-174

Detta A, Kenny BG, Smith C et al (1993) Correlation of proto-oncogene expression and proliferation and meningiomas. Neurosurgery 33:1065-1074

Devi B, Bhat D, Madhusudhan H et al (2001) Primary intraosseous meningioma of orbit and anterior cranial fossa: a case report and literature review. Australas Radiol 45:211-214

Dohar JE, Marentette LJ, Adams GL (1991) Rhabdomyosarcoma of the infratemporal fossa: diagnostic dilemmas and surgical management. Am J Otolaryngol 12:146-149

Donnet A, Schmitt A, Dufour H et al (1999) Neuropsychological follow-up of twenty two adult patients after surgery for craniopharyngioma. Acta Neurochir (Wien) 141:1049-1054

Dorfman HD, Czerniak B (1998) Bone tumors. Mosby , St. Louis:

Douglas JG, Laramore GE, Austin-Seymour M et al (1996) Neutron radiotherapy for adenoid cystic carcinoma of minor salivary glands. Int J Radiat Oncol Biol Phys 36:87-93

Duff JM, Meyer FB, Ilstrup DM. (2000) Long-term outcomes for surgically resected craniopharyngiomas. Neurosurgery; 46:291-305.

Eavey RD, Janfaza P, Chapman PH et al (1992) Skull base dumbbell tumor: surgical experience with two adolescents. Ann Otol Rhinol Laryngol 101:939-945

Einhaus SL, Sanford RA (2000) Craniopharyngiomas. In: Robertson JT, Coakham HB, Robertson JH (eds) Cranial base surgery. Churchill Livingstone, London, pp 417-429

Erdheim J (1904) Uber Hypophysenganggeschwülste und Hirncholesteatome. Sitzungsbericht der Kaiserlichen Akademie der Wissenshaften. Mathematisch-naturwissenschaftliche Classe (Wien) 113:537-726

Eversole LR (1999) Malignant epithelial odontogenic tumors. Semin Diagn Pathol 16:317-324

Fahlbusch R, Honegger J, Paulus W et al (1999) Surgical treatment of craniopharyngiomas: experience with 168 patients. J Neurosurg 90:237-250

Fajfr R, Muller B, Diem P (2002) Hypophysares Inzidentalom bei Patientin mit autosomal dominanter polyzystischer Nierenerkrankung. Schweiz Rundsch Med Prax 91:1123-1126

Falavigna A, Kraemer JL (2001) Infrasellar craniopharyngioma: case report. Arq Neuropsiquiatr 59:424-430

Farr HW, Gray GF, Jr., Vrana M et al (1973) Extracranial meningioma. J Surg Oncol 5:411-420

Figarella-Branger D, Vagner-Capodano AM, Bouillot P et al (1994) Platelet-derived growth factor (PDGF) and receptor (PDGFR) expression in human meningiomas: correlations with clinicopathological features and cytogenetic analysis. Neuropathol Appl Neurobiol 20:439-447

Fisch U (1978) Infratemporal fossa approach to tumours of the temporal bone and base of the skull. J Laryngol Otol 92:949-967

Fitzgerald SG, Morgenstern E (2000) Global improvement in intellectual and neuropsychological functioning after removal of a suprasellar cystic craniopharyngioma. Mil Med 165:496-499

Flacke S, Pauleit D, Keller E et al (1999) Infantile fibromatosis of the neck with intracranial involvement: MR and CT findings. AJNR Am J Neuroradiol 20:923-925

Florio SJ, Hurd TC (1995) Gastric carcinoma metastatic to the mucosa of the hard palate. J Oral Maxillofac Surg 53:1097-1098

Forsith PA, Cascino TL, Shaw EG et al (1993) Intracranial chordomas: a clinicopathological and prognostic study of 51 cases. J Neurosurg 78:741-747

Friedman WH, Katsantonis GP, Cooper MH et al (1981) Stylohamular dissection: a new method for en bloc reaction of malignancies of the infratemporal fossa. Laryngoscope 91:1869-1879

Fujimoto Y, Matsushita H, Velasco O et al (2002) Craniopharyngioma involving the infrasellar region: a case report and review of the literature. Pediatr Neurosurg 37:210-216

Futran ND, Haller JR (1999) Considerations for free-flap reconstruction of the hard palate. Arch Otolaryngol Head Neck Surg 125:665-669

Galant C, Rombaux P, Hamoir M et al (1998) Meningioma of the infratemporal fossa: report of a case. Acta Clin Belg 53:282-284

Gates GA (1988) The lateral facial approach to the nasopharynx and infratemporal fossa. Otolaryngol Head Neck Surg 99:321-325

Gates GA. The lateral facial approach to the nasopharynx and infratemporal fossa. Otolaryngol Head Neck Surg 1988;99:321-5.

Gay E, Sekhar LN, Rubinstein E et al (1995) Chordomas and chondrosarcomas of the cranial base: results and follow-up of 60 patients. Neurosurgery 36:887-897

Giangaspero F, Burger PC, Osborne DR et al (1984) Suprasellar papillary squamous epithelioma („papillary craniopharyngioma"). Am J Surg Pathol 8:57-64

Ginsberg LE (1992) Neoplastic diseases affecting the central skull base: CT and MR imaging. AJR Am J Roentgenol 159:581-589

Ginsberg LE (1999) Imaging of perineural tumor spread in head and neck cancer. Semin Ultrasound CT MR 20:175-186

Ginsberg LE, DeMonte F (1998) Imaging of perineural tumor spread from palatal carcinoma. AJNR Am J Neuroradiol 19:1417-1422

Gormley WB, Beckman ME, Ho KL et al (1995) Primary craniofacial chordoma: case report. Neurosurgery 36:1196-1199

Goto TK, Yoshiura K, Tanaka T et al (1998) A follow-up of rhabdomyosarcoma of the infratemporal fossa region in adults based on the magnetic resonance imaging findings: case reports. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 86:616-625

Greenberg MS (2001) Handbook of Neurosurgery. 5th Edition, Thieme, New York:.

Grundfast K, Healy G, Richardson M (1991) Fibrosarcoma of the infratemporal fossa in an 8-year-old girl. Head Neck 13:156-159

Guinto G, Abello J, Molina A et al (1999) Zygomatic-transmandibular approach for giant tumors of the infratemporal fossa and parapharyngeal space. Neurosurgery 45:1385-1398

Gupta K, Kuhn MJ, Shevlin DW et al (1999) Metastatic craniopharyngioma. AJNR Am J Neuroradiol 20:1059-1060

Han MH, Chang KH, Lee CH et al (1995) Cystic expansile masses of the maxilla: differential diagnosis with CT and MR. AJNR Am J Neuroradiol 16:333-338

Hardy J (1969) Transsphenoidal microsurgery of the normal and pathological pituitary. Clinical Neurosurgery 16:185-217

Harrison DFN, Lund VJ (1993) Tumours of the upper jaw Edinburgh; New York: Churchill Livingstone

Hassounah M, Al-Mefty O, Athar M et al (1985) Primary cranial and intracranial chondrosarcoma, a survey. Acta Neurochir 78:123-132

Hatfield DL, White M, Araoz C (2001) Nasal meningioma: report of one case and review. J Ark Med Soc 97:416-417

Hefer T, Manor R, Zvi Joachims H et al (1998) Metastatic follicular thyroid carcinoma to the maxilla. J Laryngol Otol 112:69-72

Helseth A (1997) Incidence and survival of intracranial meningioma patients in Norway 1963-1992. Neuroepidemiology 16:53-59

Herman P, Lot G, Chapot R et al (1999) Long-term follow-up of juvenile nasopharyngeal angiofibromas: analysis of recurrences. Laryngoscope 109:140-147

Hertzanu Y, Mendelsohn DB, Cohen M (1984) Computed tomography of mandibular ameloblastoma. J Comput Assist Tomogr 8:220-223

Hicks J, Flaitz C (2002) Rhabdomyosarcoma of the head and neck in children. Oral Oncol 38:450-459

Higinbotham NL, Phillips RF, Farr HW et al (1967) Chordoma: thirty-five year study at the Memorial Hospital. Cancer 20:1841-1850

Hillman TH, Peyster RG, Hoover ED et al (1988) Infrasellar craniopharyngioma: CT and MR studies. J Comput Assist Tomogr 12:702-704

Hirabayashi S, Yanai A, Muraishi Y (1997) Huge pleomorphic adenoma of the upper retromolar area. Ann Plast Surg 38:184-186

Hisatomi M, Asaumi J, Konouchi H et al (2001) MR imaging of nasopalatine duct cysts. Eur J Radiol 39:73-76

Hisatomi M, Asaumi J, Konouchi H et al (2003) MR imaging of epithelial cysts of the oral and maxillofacial region. Eur J Radiol 48:178-182

Hochberg MG, Currie WR, Rafetto LK (1994) Osteotomy technique for accessing a meningioma in the infratemporal fossa: report of a case. J Oral Maxillofac Surg 52:499-502

Holtmann S, Martin F, Permanetter W (1985) Die dysontogenetischen, medianen Oberkieferzysten. Laryngol Rhinol Otol (Stuttg) 64:331-334

Honegger J, Buchfelder M, Fahlbusch R (1999) Surgical treatment of craniopharyngiomas: endocrinological results. J Neurosurg 90:251-257

Howard DJ, Lund VJ (1992) The midfacial degloving approach to sinonasal disease. J Laryngol Otol 106:1059-1062

Hsu HC, Huang EY, Eng HL (2002) Cheek mass as a presentation of metastatic rectal cancer. Chang Gung Med J 25:345-348

Hu J, Little J, Xu T et al (1999) Risk factors for meningioma in adults: a case-control study in northeast China. Int J Cancer 83:299-304

Huber PE, Debus J, Latz D et al (2001) Radiotherapy for advanced adenoid cystic carcinoma: neutrons, photons or mixed beam? Radiother Oncol 59:161-167

Hug EB, Loredo LN, Slater JD et al (1999) Proton radiation therapy for chordomas and chondrosarcomas of the skull base. J Neurosurg 91:432-439

Hyam DM, Veness MJ, Morgan GJ (2004) Minor salivary gland carcinoma involving the oral cavity or oropharynx. Aust Dent J 49:16-19

Inagi K, Takahashi H, Okamoto M et al (2002) Treatment effects in patients with squamous cell carcinoma of the oral cavity. Acta Otolaryngol Suppl (547):25-29

Isaac MA, Hahn SS, Kim JA et al (2001) Management of craniopharyngioma. Cancer J 7:516-520

Ishii K, Ikeda H, Takahashi S et al (1996) MR imaging of pituitary adenomas with sphenoid sinus invasion: characteristic MR findings indicating fibrosis. Radiat Med 14:173-178

Iuchi T, Saeki N, Tanaka M et al (1998) MRI prediction of fibrous pituitary adenomas. Acta Neurochir (Wien) 140:779-786

Jackson IT, Callan PP, Forte RA (1996) An anatomical classification of maxillary ameloblastoma as an aid to surgical treatment. J Craniomaxillofac Surg 24:230-236

Janse van Rensburg L, Nortje CJ, Thompson I (1997) Correlating imaging and histopathology of an odontogenic keratocyst in the nevoid basal cell carcinoma syndrome. Dentomaxillofac Radiol 26:195-199

Jansisyanont P, Blanchaert RH, Jr., Ord RA (2002) Intraoral minor salivary gland neoplasm: a single institution experience of 80 cases. Int J Oral Maxillofac Surg 31:257-261

Jiang RS, Wu CY, Jan YJ et al (1998) Primary ethmoid sinus craniopharyngioma: a case report. J Laryngol Otol 112:403-405

Johnsen DE, Woodruff WW, Allen IS et al (1991) MR imaging of the sellar and juxtasellar regions. Radiographics 11:727-758

Johnson AT, Maran AG (1982) Extra-cranial tumours of the infratemporal fossa. J Laryngol Otol 96:1017-1026

Jung SL, Choi KH, Park YH et al (1999) Cemento-ossifying fibroma presenting as a mass of the parapharyngeal and masticator space. AJNR Am J Neuroradiol 20:1744-1746

Kalapurakal JA, Goldman S, Hsieh YC et al (2003) Clinical outcome in children with craniopharyngioma treated with primary surgery and radiotherapy deferred until relapse. Med Pediatr Oncol 40:214-218

Kanazawa T, Nishino H, Miyata M et al (2001) Haemangiopericytoma of infratemporal fossa. J Laryngol Otol 115:77-79

Kaplan JN, Duckert LG (1984) Lesions metastatic to the infratemporal fossa: report of a case and review of the literature. Otolaryngol Head Neck Surg 92:241-246

Kawahara Y, Niiro M, Yokoyama S et al (2001) Dural congestion accompanying meningioma invasion into vessels: the dural tail sign. Neuroradiology 43:462-465

Kawai T, Murakami S, Kishino M et al (1998) Diagnostic imaging in two cases of recurrent maxillary ameloblastoma: comparative evaluation of plain radiographs, CT and MR images. Br J Oral Maxillofac Surg 36:304-310

Kaye AH (1997) Essential neurosurgery. 2nd edition. Churchill Livingstone, New York

Kleihues P, Burger PC, Scheithauer BW et al (1993) Histological typing of tumours of the central nervous system. 2nd edition. Springer-Verlag, Berlin

Knox RD, Pratt MF, Garen PD et al (1990) Intramuscular hemangioma of the infratemporal fossa. Otolaryngol Head Neck Surg 103:637-641

Kondziolka D, Lunsford LD, Coffey RJ et al (1991) Stereotactic radiosurgery of meningiomas. J Neurosurg 74:552-559

Koo JY, Kadonaga JN, Wintroub BV et al (1992) The development of B-cell lymphoma in a patient with psoriasis treated with cyclosporine. J Am Acad Dermatol 26:836-840

Kornblut AD (1987) Clinical evaluation of tumors of the oral cavity In: Thawley SE, Panjie WR, Batsakis JG, Lindberg RD (eds) Comprehensive Management of Head and Neck Tumors. Saunders, Philadelphia, pp 460-479.

Kornfehl J, Gstottner W, Kontrus M et al (1996) Transpalatine excision of a cavernous hemangioma of the infratemporal fossa. Eur Arch Otorhinolaryngol 253:172-175

Korten AG, Berg HJ, Spincemaille GH et al (1998) Intracranial chondrosarcoma: review of the literature and report of 15 cases. J Neurol Neurosurg Psychiatry 65:88-92

Kramer IRH, Pindborg JJ, Shear M (1992) Histological typing of odontogenic tumours. 2nd edition. Springer-Verlag, Berlin

Krespi YP (1989) Lateral skull base surgery for cancer. Laryngoscope 99:514-524

Krespi YP, Sisson GA (1984) Transmandibular exposure of the skull base. Am J Surg 148:534-538

Krishnamurthy S, Holmes B, Powers SK (1998) Schwannomas limited to the infratemporal fossa: report of two cases. J Neurooncol 36:269-277

Krolls SO, Hoffman S (1976) Squamous cell carcinoma of the oral soft tissues: a statistical analysis of 14,253 cases by age, sex, and race of patients. J Am Dent Assoc 92:571-574

Krolls SO, Hoffman S. Squamous cell carcinoma of the oral soft tissues: a statistical analysis of 14,253 cases by age, sex, and race of patients. J AM Dent Assoc 1976;92:571-4.

Kucharczyk W, Peck WW, Kelly WM et al (1987) Rathke cleft cysts: CT, MR imaging, and pathologic features. Radiology 165:491-495

Kumar A, Valvassori G, Jafar J et al (1986) Skull base lesions: a classification and surgical approaches. Laryngoscope 96:252-263

Kveton JF, Brackmann DE, Glasscock ME 3rd et al (1986) Chondrosarcoma of the skull base. Otolaryngol Head Neck Surg 94:23-32

Lahoz Zamarro MT, Martinez Subias J, Muniesa Soriano JA et al (2001) Melanoma de paladar duro. Acta Otorrinolaringol Esp 52:422-425

Laine FJ, Braun IF, Jensen ME et al (1990) Perineural tumor extension through the foramen ovale: evaluation with MR imaging. Radiology 174:65-71

Lambe M, Coogan P, Baron J (1997) Reproductive factors and the risk of brain tumors: a population-based study in Sweden. Int J Cancer 72:389-393

Landolt AM (2001) History of pituitary surgery from the technical aspect. Neurosurg Clin North Am 12:37-44

Lawson W, Naidu RK, Le Benger J et al (1990) Combined median mandibulotomy and Weber-Fergusson maxillectomy. Arch Otolaryngol Head Neck Surg 116:596-599

Lee RJ, Smith SH, Hicks WL, Jr. et al (1998) Management of extraosseous ewing sarcoma of the infratemporal fossa. Med Pediatr Oncol 31:31-35

Lee YY, Van Tassel P (1989) Craniofacial chondrosarcomas: imaging findings in 15 untreated cases. AJNR Am J Neuroradiol 10:165-170

Leonetti JP, al-Mefty O, Eisenbeis JF et al (1993) Orbitocranial exposure in the management of infratemporal fossa tumors. Otolaryngol Head Neck Surg 109:769-772

Leonetti JP, Wachter B, Marzo SJ, Petruzzelli G (2001) Extracranial lower cranial nerve sheath tumors. Otolaryngol Head Neck Surg 125:640-644.

Lissett CA, Shalet SM (2000) Management of pituitary tumours: strategy for investigation and follow-up. Horm Res 53 Suppl 3:65-70

Longstreth WT, Jr., Dennis LK, McGuire VM et al (1993) Epidemiology of intracranial meningioma. Cancer 72:639-648

Lopes M, Duffau H, Fleuridas G (1999) Primary spheno-orbital angiosarcoma: case report and review of the literature. Neurosurgery 44:405-407; discussion 407-408

Lopes MA, Santos GC, Kowalski LP (1998) Multivariate survival analysis of 128 cases of oral cavity minor salivary gland carcinomas. Head Neck 20:699-706

Lorigan JG, O'Keeffe FN, Evans HL et al (1989) The radiologic manifestations of alveolar soft-part sarcoma. AJR Am J Roentgenol 153:335-339

Losa M, Valle M, Mortini P et al. (2004) Gamma Knife surgery for treatment of residual nonfunctioning pituitary adenomas after surgical debulking. J Neurosurg 100:438-444

Loughran S, Badia L, Lund V (2000) Primary chordoma of the ethmoid sinus. J Laryngol Otol 114:627-629

Mac Carty CS, Hanson EJ Jr, Randall RV et al (1973) Indications for and results of surgical treatment of pituitary tumors by the transfrontal approach. International Congress Series 303. Diagnosis and teratment of pituitary tumors. Excerpta Medica, Amsterdam

Maiuri F, Salzano FA, Motta S et al (1998) Olfactory groove meningioma with paranasal sinus and nasal cavity extension: removal by combined subfrontal and nasal approach. J Craniomaxillofac Surg 26:314-317

Manganaro AM, Will MJ, Ragno JR, Jr. et al (1997) Red-blue lesion of the hard palate. J Oral Maxillofac Surg 55:159-165

Maroldi R, Battaglia G, Farina D et al (1999) Tumours of the oropharynx and oral cavity: perineural spread and bone invasion. Jbr-Btr 82:294-300

McCarthy BJ, Davis FG, Freels S et al (1998) Factors associated with survival in patients with meningioma. J Neurosurg 88:831-839

McCluggage WG, McBride GB, Primrose WJ et al (1995) Giant cell tumour of the temporal bone presenting as vertigo. J Laryngol Otol 109:538-541

McDaniel RK (1999) Odontogenic cyst and tumors: clinical evaluation and pathology,. In: Thawley SE, Panje WR, Batsakis JG, Lindberg RD (eds) Comprehensive Management of Head and Neck Tumors. 2nd edn. Saunders, Philadelphia, pp 1566-1610.

Mendelsohn DB, Hertzanu Y, Glass RB (1983) Computed tomographic findings in primary mandibular osteosarcoma. Clin Radiol 34:153-155

Merchant TE, Kiehna EN, Sanford RA et al (2002) Craniopharyngioma: the St. Jude Children's Research Hospital experience 1984-2001. Int J Radiat Oncol Biol Phys 53:533-542.

Meyers SP, Hirsch WL, Jr., Curtin HD et al (1992) Chordomas of the skull base: MR features. AJNR Am J Neuroradiol 13:1627-1636

Miller NR, Golnik KC, Zeidman SM et al (1996) Pneumosinus dilatans: a sign of intracranial meningioma. Surg Neurol 46:471-474

Minami M, Kaneda T, Ozawa K et al (1996) Cystic lesions of the maxillomandibular region: MR imaging distinction of odontogenic keratocysts and ameloblastomas from other cysts. AJR Am J Roentgenol 166:943-949

Minoda R, Masako M, Masuyama K et al (2001) Malignant myoepithelioma arising within the masticator space ectopically. Otolaryngol Head Neck Surg 124:342-343

Molla E, Marti-Bonmati L, Revert A et al (2002) Craniopharyngiomas: identification of different semiological patterns with MRI. Eur Radiol 12:1829-1836

Moriya S, Tei K, Notani K et al (2001) Malignant hemangiopericytoma of the head and neck: a report of 3 cases. J Oral Maxillofac Surg 59:340-345

Morris KM, Campbell D, Stell PM et al (1990) Meningiomas presenting with paranasal sinus involvement. Br J Neurosurg 4:511-515

Mortini P, Giovanelli M (2002) Transcranial Approaches to Pituitary Tumors. Operative Techniques in Neurosurgery 3:1-14

Mosqueda-Taylor A, Ledesma-Montes C, Caballero-Sandoval S et al (1997) Odontogenic tumors in Mexico: a collaborative retrospective study of 349 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 84:672-675

Nammour GM, Ybarra J, Naheedy MH et al (1997) Incidental pituitary macroadenoma: a population-based study. Am J Med Sci 314:287-291

Neff B, Thayer Sataloff R, Storey L et al (2002) Chondrosarcoma of the skull base. Laryngoscope 112: 134-139

Neville BW, Damm DD, Brock T (1997) Odontogenic keratocysts of the midline maxillary region. J Oral Maxillofac Surg 55:340-344

Ng SH, Chang TC, Ko SF et al (1997) Nasopharyngeal carcinoma: MRI and CT assessment. Neuroradiology 39:741-746

Nicolato A, Foroni R, Alessandrini F et al (2002) Radiosurgical treatment of cavernous sinus meningiomas: experience with 122 treated patients. Neurosurgery 51:1153-1159; discussion 1159-1161

Norris JS, Pavaresh M, Afshar F (1998) Primary transsphenoidal microsurgery in the treatment of craniopharyngiomas. Br J Neurosurg 12:305-312

O'Connell JX, Renard LG, Liebsch NJ (1994) Base of skull chordoma: a correlative study of hystologic and clinical features of 62 cases. Cancer 74:2261-2267

Oda D, Rivera V, Ghanee N et al (2000) Odontogenic keratocyst: the northwestern USA experience. J Contemp Dent Pract 1:60-74

Ogren FP, Wisecarver JL, Lydiatt DD et al (1991) Ancient neurilemmoma of the infratemporal fossa: a case report. Head Neck 13:243-246

Oikawa S, Kyoshima K, Goto T et al (2001) Histological study on local invasiveness of clival chordoma. Case report of autopsy. Acta Neurochir (Wien) 143:1065-1069

Okita W, Ichimura K, Iinuma T (1991) Dentigerous cyst of the maxilla and its image diagnosis. Rhinology 29:307-314

Oot RF, Melville GE, New PF et al (1988) The role of MR and CT in evaluating clival chordomas and chondrosarcomas. AJR Am J Roentgenol 151:567-575

O'Ryan F, Eversole LR, Alikpala A (1987) Juvenile fibromatosis of the infratemporal fossa. Oral Surg Oral Med Oral Pathol 64:603-608

Ostrov SG, Quencer RM, Hoffman JC et al (1989) Hemorrhage within pituitary adenomas: how often associated with pituitary apoplexy syndrome? AJR Am J Roentgenol 153:153-160

Palacios E, Serou M, Restrepo S et al (2004) Odontogenic keratocysts in nevoid basal cell carcinoma (Gorlin's) syndrome: CT and MRI evaluation. Ear Nose Throat J 83:40-42

Pang D. Surgical management of craniopharyngioma. (1993) In: Sekhar LN, Janecka IP (eds) Surgery of cranial base tumors. Raven Press, New York, pp 787-807

Papageorge MB, Doku HC, Lis R (1992) Solitary neurofibroma of the mandible and infratemporal fossa in a young child. Report of a case. Oral Surg Oral Med Oral Pathol 73:407-411

Patel PC, Pellitteri PK, Vrabec DP et al (1998) Tumefactive fibroinflammatory lesion of the head and neck originating in the infratemporal fossa. Am J Otolaryngol 19:216-219

Perzin KH, Pushparaj N (1984) Nonepithelial tumors of the nasal cavity, paranasal sinuses, and nasopharynx. A clinicopathologic study. XIII: Meningiomas. Cancer 54:1860-1869

Perzin KH, Pushparaj N (1986) Nonepithelial tumors of the nasal cavity, paranasal sinuses, and nasopharynx. A clinicopathologic study. XIV: Chordomas. Cancer 57:784-796

Petruzzelli GJ, Myers EN (1994) Malignant neoplasms of the hard palate and upper alveolar ridge. Oncology (Huntingt) 8:43-48; discussion 50, 53

Pevsner PH, Bast WG, Lumerman H et al (2000) CT analysis of a complicated nasopalatine duct cyst. N Y State Dent J 66:18-20

Phillips LE, Koepsell TD, van Belle G et al (2002) History of head trauma and risk of intracranial meningioma: population-based case-control study. Neurology 58:1849-1852

Pichard C, Gerber S, Laloi M et al (2002) Pituitary carcinoma: report of an exceptional case and review of the literature. J Endocrinol Invest 25:65-72

Pieper DR, Al-Mefty O (1999) Management of intracranial meningiomas secondarily involving the infratemporal fossa: radiographic characteristics, pattern of tumor invasion, and surgical implications. Neurosurgery 45:231-237; discussion 237-238

Pieper DR, Al-Mefty O, Hanada Y et al (1999) Hyperostosis associated with meningioma of the cranial base: secondary changes or tumor invasion. Neurosurgery 44:742-746; discussion 746-747

Pogrel MA (1994) The management of salivary gland tumors of the palate. J Oral Maxillofac Surg 52:454-459

Pritchyk KM, Schiff BA, Newkirk KA et al (2002) Metastatic renal cell carcinoma to the head and neck. Laryngoscope 112:1598-1602

Prott FJ, Micke O, Haverkamp U et al (2000) Results of fast neutron therapy of adenoid cystic carcinoma of the salivary glands. Anticancer Res 20:3743-3749

Rangheard AS, Vanel D, Viala J et al (2001) Synovial sarcomas of the head and neck: CT and MR imaging findings of eight patients. AJNR Am J Neuroradiol 22:851-857

Raybaud C, Rabehanta P, Girard N (1991) Aspects radiologiques des craniopharyngiomes. In: Choux M, Lena G, Genitori L (eds) Le craniopharyngiome de l'enfant. Neurochirurgie (Paris) 37(suppl1):12-165.

Rees DA, Hanna FW, Davies JS et al (2002) Long-term follow-up results of transsphenoidal surgery for Cushing's disease in a single centre using strict criteria for remission. Clin Endocrinol (Oxf) 56:541-551

Regezi JA (2002) Odontogenic cysts, odontogenic tumors, fibroosseous, and giant cell lesions of the jaws. Mod Pathol 15:331-341

Reiter ER, Varvares MA, August M et al (2000) Mucocele of the infratemporal fossa as an unusual complication of midfacial fracture. Ann Otol Rhinol Laryngol 109:522-525

Rohrer T, Gassmann K, Buchfelder M et al (2002) Klinische Symptome und Befunde zum Zeitpunkt der Diagnosestellung bei Kindern und Jugendlichen mit Kraniopharyngeom. Klin Padiatr 214:285-290

Rosenberg AE, Nielsen GP, Keel SB et al (1999) Chondrosarcoma of the base of the skull: a clinicopathologic study of 200 cases with emphasis on its distinction from chordoma. Am J Surg Pathol 23:1370-1378

Rosenblum BN, Katsantonis GP, Cooper MH et al (1990) Infratemporal fossa and lateral skull base dissection: long-term results. Otolaryngol Head Neck Surg 102:106-110

Rouviáere H (1932) Anatomie des lymphatiques de l'homme. Masson, Paris

Rubin MM, Sadoff RS (1992) Rhabdomyosarcoma of the infratemporal fossa. N Y State Dent J 58:32-35

Rubinstein AB, Arbit E (1985) Intracranial meningiomas presenting with epistaxis--case report and literature review. J Otolaryngol 14:248-250

Sadetzki S, Flint-Richter P, Ben-Tal T et al (2002) Radiation-induced meningioma: a descriptive study of 253 cases. J Neurosurg 97:1078-1082

Saleh EA, Taibah AK, Naguib M et al (1994) Giant cell tumor of the lateral skull base: a case report. Otolaryngol Head Neck Surg 111:314-318.

Samii M, Bini W (1991) Surgical treatment of craniopharyngiomas. Zentralbl Neurochir 52:17-23

Sarac S, Koybasi S, Kaya S (2000) Transmaxillary excision of a rare cavernous hemangioma of the infratemporal fossa. Ear Nose Throat J 79:448-449, 452

Sartoretti-Schefer S, Wichmann W, Aguzzi A et al (1997) MR

differentiation of adamantinous and squamous-papillary craniopharyngiomas. AJNR Am J Neuroradiol 18:77-87
Schreiber A, Kinney LA, Salman R (1991) Large-cell lymphoma of the infratemporal fossa presenting as myofacial pain. J Craniomandib Disord 5:286-289
Schulz-Ertner D, Frank C, Herfarth KK et al (2002) Fractionated stereotactic radiotherapy for craniopharyngiomas. Int J Radiat Oncol Biol Phys 54:1114-1120
Seifert V, Dietz H (1992) Combined orbito-frontal, sub- and infratemporal fossa approach to skull base neoplasms. Surgical technique and clinical application. Acta Neurochir (Wien) 114:139-144
Sekhar LN, Oliveira ED (1999) Cranial microsurgery: approaches and techniques. Thieme, New York
Sekhar LN, Schramm VL, Jr., Jones NF (1987) Subtemporal-preauricular infratemporal fossa approach to large lateral and posterior cranial base neoplasms. J Neurosurg 67:488-499
Shah JP, Lydiatt WM Buccal mucosa, alveolus, retromolar trigone, floor of mouth, hard palate, and tongue tumors (1999) In: Thawley SE, Panjie WR, Batsakis JG, Lindberg RD (eds) Comprehensive Management of Head and Neck Tumors. 2nd edn. Saunders, Philadelphia, pp 686-694
Shaheen OH (1982) Swellings of the infratemporal fossa. J Laryngol Otol 96:817-836
Shahinian HK, Suh RH, Jarrahy R (1999) Combined infratemporal fossa and transfacial approach to excising massive tumors. Ear Nose Throat J 78:350, 353-356.
Shapshay SM, Elber E, Strong MS (1976) Occult tumors of the infratemporal fossa: report of seven cases appearing as preauricular facial pain. Arch Otolaryngol 102:535-538
Sheehan JP, Kondziolka D, Flickinger J et al. (2002) Radiosurgery for residual or recurrent nonfunctioning pituitary adenoma. J Neurosurg 97 (Suppl 5):408-414
Shin JL, Asa SL, Woodhouse LJ et al (1999) Cystic lesions of the pituitary: clinicopathological features distinguishing craniopharyngioma, Rathke's cleft cyst, and arachnoid cyst. J Clin Endocrinol Metab 84:3972-3982
Shinohara Y, Uchida A, Hiromatsu T et al (1993) A case of neurilemmoma in the infratemporal fossa showing the antral bowing sign. Dentomaxillofac Radiol 22:214-215
Shugar JM, Som PM, Krespi YP, Arnold LM, Som ML (1980) Primary chordoma of the maxillary sinus. Laryngoscope 90:1825-1830
Sichel JY, Monteil JP, Elidan J (1994) Skull base chondroma of extracranial origin. Head Neck 16:578-581
Simsir A, Osborne BM, Greenebaum E (1996) Malignant granular cell tumor: a case report and review of the recent literature. Hum Pathol 27:853-858
Slama A, Yacoubi T, Khochtali H et al (2002) Carcinosarcome odontogene mandibulaire. Rev Stomatol Chir Maxillofac 103:124-127
Slater LJ (1999) Odontogenic sarcoma and carcinosarcoma. Semin Diagn Pathol 16:325-332
Slonim SM, Haykal HA, Cushing GW et al (1993) MRI appearances of an ectopic pituitary adenoma: case report and review of the literature. Neuroradiology 35:546-548
Smith PG, Grubb RL, Kletzker GR et al (1990) Combined pterional-anterolateral approaches to cranial base tumors. Otolaryngol Head Neck Surg 103:357-363
Som PM, Curtin HD, Silvers AR (1997) A re-evaluation of imaging criteria to assess aggressive masticator space tumors. Head Neck 19:335-341
Som PM, Norton KI (1991) Lesions that manifest as medial cheek and nasolabial fold masses. Radiology 178:831-835
Som PM, Shangold LM, Biller HF (1992) A palatal dentigerous cyst arising from a mesiodente. AJNR Am J Neuroradiol 13:212-214
Spada A, Lania A, Ballare E (1998) G protein abnormalities in pituitary adenomas. Mol Cell Endocrinol 142:1-14
Spagnoli MV, Goldberg HI, Grossman RI et al (1986) Intracranial meningiomas: high-field MR imaging. Radiology 161:369-375
Spindler MB, Philipp A, Laszig R (1989) Das extrakranielle Meningeom: eine seltene Differentialdiagnose eines intranasalen Tumors. Hno 37:162-164
Spinelli HM, Isenberg JS, O'Brien M (1994) Nasopalatine duct cysts and the role of magnetic resonance imaging. J Craniofac Surg 5:57-60
Strugar J, Sekhar LN (2000) Chordomas and chondrosarcomas. In: Robertson JT, Coakham HB, Robertson JH (eds) Cranial base surgery. Churchill Livingstone, London, pp 397-415
Su CY, Chien CY, Hwang CF (1999) A new transnasal approach to endoscopic marsupialization of the nasolabial cyst. Laryngoscope 109:1116-1118
Suei Y, Tanimoto K, Taguchi A et al (1994) Primary intraosseous carcinoma: review of the literature and diagnostic criteria. J Oral Maxillofac Surg 52:580-583
Suhardja AS, Kovacs KT, Rutka JT (1999) Molecular pathogenesis of pituitary adenomas: a review. Acta Neurochir (Wien) 141:729-736
Surawicz TS, McCarthy BJ, Kupelian V et al (1999) Descriptive epidemiology of primary brain and CNS tumors: results from the Central Brain Tumor Registry of the United States 1990-1994. Neuro-oncol 1:14-25
Swain RE, Jr., Kingdom TT, DelGaudio JM et al (2001) Meningiomas of the paranasal sinuses. Am J Rhinol 15:27-30
Sze G, Uichanco LS 3rd, Brant-Zawadzki MN et al (1988) Chordomas: MR imaging. Radiology 166:187-191
Tashiro M, Nagase M, Nakajima T et al (1988) Malignant paraganglioma. Report of a case and review of the Japanese literature. J Craniomaxillofac Surg 16:324-329
Thompson LD, Gyure KA (2000) Extracranial sinonasal tract meningiomas: a clinicopathologic study of 30 cases with a review of the literature. Am J Surg Pathol 24:640-650
Thuomas KA (1999) Pituitary microadenoma. MR appearance and correlation with CT. Acta Radiol 40:663
To EH, Brown JS, Avery BS et al (1991) Primary intraosseous carcinoma of the jaws. Three new cases and a review of the literature. Br J Oral Maxillofac Surg 29:19-25
Tokumaru A, O'Uchi T, Eguchi T et al (1990) Prominent meningeal enhancement adjacent to meningioma on Gd-DTPA-enhanced MR images: histopathologic correlation. Radiology 175:431-433
Tomura N, Hirano H, Kato K et al (1999) Comparison of MR imaging with CT in depiction of tumour extension into the pterygopalatine fossa. Clin Radiol 54:361-366
Toriumi DM, Shermetaro CB, Pecaro BC (1989) Cavernous hemangioma of the infratemporal fossa. Ear Nose Throat J 68:252 258-259
Tran L, Sadeghi A, Hanson D et al (1987). Salivary gland tumors of the palate: the UCLA experience. Laryngoscope 97:1343-1345
Truitt TO, Gleich LL, Huntress GP et al (1999) Surgical management of hard palate malignancies. Otolaryngol Head Neck Surg 121:548-552

Ulfarsson E, Lindquist C, Roberts M et al (2002) Gamma knife radiosurgery for craniopharyngiomas: long-term results in the first Swedish patients. J Neurosurg 97:613-622

Ummat S, Nasser JG (1992) Fibrosarcoma of the infratemporal fossa in childhood: a challenging problem. J Otolaryngol 21:441-446

Uppal HS, D'Souza AR, De R et al (2002) Dermoid cyst of the infratemporal fossa. J Laryngol Otol 116:150-152

Vajramani G, Devi I, Santosh V et al (1999) Benign triton tumor of the trigeminal nerve. Childs Nerv Syst 15:140-144

Van Effenterre R, Boch AL (2002) Craniopharyngioma in adults and children: a study of 122 surgical cases. J Neurosurg 97:3-11

Vasconcelos R, de Aguiar MF, Castro W et al (1999a) Retrospective analysis of 31 cases of nasopalatine duct cyst. Oral Dis 5:325-328

Vasconcelos RF, Souza PE, Mesquita RA (1999b) Retrospective analysis of 15 cases of nasolabial cyst. Quintessence Int 30:629-632

Verbin R., Barnes L. (2001) Cyst and cystic like lesions of the oral cavity, jaws, and neck. In: Barnes L (ed) Surgical pathology of the head and neck. M. Dekker, New York, pp 1437-1555

Vogl TJ, Mack MG, Straub R et al (2001) MR-guided laser-induced thermotherapy of the infratemporal fossa and orbit in malignant chondrosarcoma via a modified technique. Cardiovasc Intervent Radiol 24:432-435

Volpe NJ, Liebsch NJ, Munzenrider JE et al (1993) Neuro-ophthalmologic findings in chordoma and chondrosarcoma of the skull base. Am J Ophthalmol 115:97-104

Walker WP, Landas SK, Bromley CM et al (1991) Immunohistochemical distinction of classic and chondroid chordomas. Mod Pathol 4:661-666

Wang KC, Kim SK, Choe G et al (2002) Growth patterns of craniopharyngioma in children: role of the diaphragm sellae and its surgical implication. Surg Neurol 57:25-33

Warakaulle DR, Anslow P (2003) Differential diagnosis of intracranial lesions with high signal on T1 or low signal on T2-weighted MRI. Clin Radiol 58:922-933

Watters GW, Brookes GB (1995) Chondrosarcoma of the temporal bone. Clin Otolaryngol 20:53-58

Weber AL, Brown EW, Hug EB et al (1995) Cartilaginous tumors and chordomas of the cranial base. Otolaryngol Clin North Am 28:453-471

Weisman RA, Osguthorpe JD (1988) Pseudotumor of the head and neck masquerading as neoplasia. Laryngoscope 98:610-614

Weissman JL, Snyderman CH, Yousem SA et al (1993) Ameloblastoma of the maxilla: CT and MR appearance. AJNR Am J Neuroradiol 14:223-226

Wetmore SJ, Suen JY, Snyderman NL (1986) Preauricular approach to infratemporal fossa. Head Neck Surg 9:93-103

Wetmore SJ, Suen JY, Snyderman NL. Preauricular approach to infratemporal fossa. Head Neck Surg 1986;9:93-103.

Williams LS, Mancuso AA, Mendenhall WM (2001) Perineural spread of cutaneous squamous and basal cell carcinoma: CT and MR detection and its impact on patient management and prognosis. Int J Radiat Oncol Biol Phys 49:1061-1069

Wilson CB (1984) A decade of pituitary microsurgery. The Herbert Olivecrona Lecture. J Neurosurg 61:814-833

Wolters B, Kleinsasser O (1985) Intranasale extradurale meningeome. Fallbericht und Literaturzusammenstellung. Laryngol Rhinol Otol (Stuttg) 64:198-201

Woolford TJ, Birzgalis AR, Ramsden RT (1994) An extensive vestibular schwannoma with both intracranial spread and lateral extension to the external auditory canal. J Laryngol Otol 108:149-151

Yasargil MG, Curcic M, Kis M et al (1990) Total removal of craniopharyngiomas. Approaches and long-term results in 144 patients. J Neurosurg 73:3-11

Yeakley JW, Kulkarni MV, McArdle CB et al (1988) High-resolution MR imaging of juxtasellar meningiomas with CT and angiographic correlation. AJNR Am J Neuroradiol 9:279-285

Yorozu A, Sykes AJ, Slevin NJ (2001) Carcinoma of the hard palate treated with radiotherapy: a retrospective review of 31 cases. Oral Oncol 37:493-497

Yu Q, Wang P, Shi H et al (1998) The lesions of the pterygopalatine and infratemporal spaces: computed tomography evaluation. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 85:742-751

Zada G, Kelly DF, Cohan P et al (2003) Endonasal transsphenoidal approach for pituitary adenomas and other sellar lesions: an assessment of efficacy, safety, and patient impressions. J Neurosurg 98:350-358

Zervas NT, Martin JB (1980) Current concepts in cancer: management of hormone-secreting pituitary adenomas. N Engl J Med 302:210-214

Zhao K, Liu N, Qi DY et al (1993) Primary meningioma of the infratemporal fossa: a clinicopathologic and immunohistochemical study. J Oral Maxillofac Surg 51:597-601

Zhao YF, Wei JX, Wang SP (2002) Treatment of odontogenic keratocysts: a follow-up of 255 Chinese patients. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 94:151-156

Zorlu F, Gurkaynak M, Yildiz F et al (2000) Conventional external radiotherapy in the management of clivus chordomas with overt residual disease. Neurol Sci 21:203-207