Learning objectives

Nasal cavity contains different kinds of tissues such as the epithelial (squamous, neuroendocrine, olfactory) and the mezenchimal (bone, cartilage, muscle, vascular) ones which all of those carry the risk for the variety of tumoral differentiation. The literature mentions tumoral growth from all these tissue types; despite the wide range of diversity, the incidence of the nasal tumors are as low as 1/100,000 (Table I) (1) Primary nasal malignancies consist of 0.2-0.8% of all the malignant tumors and 3.6% of the malignant upper airway tumors (1). Our primary goal was to mention the common features of the benign and malignant nasal tumors that usually give symptoms such as nasal congestion, blockage, rhinorrhea, headache, proptosis, trismus, cranial neuropathy; whereas the radiologic examination of the cross-sectional images of various types of nasal tumors that mostly block the airway. We also stated the main clinic and surgical approaches to those cases and gave a brief discussion of the literature.

Table I. A detailed practical classification scheme for nasal cavity masses.

| PRIMARY | BENIGN |
|---------|--------|
| Osteoma |
| Inverted papilloma |
| Minor salivary gland tumors |
| Angiofibroma (locally aggressive) |
| Polyps (antrochonal, sphenochonal) |
| Hemangioma, dermoids and miscellaneous |
| MALIGNANT |
| Squamous cell carcinoma |
| Adenocarcinoma |
| Adenoid cystic carcinoma |
| Esthesioneuroblastoma |
| Lymphoma |
Sarcomas
Malignant melanoma

Miscellaneous
  • INFECTIOUS-INFLAMATORY

Inflamatory polyps
Mucocele, atypical retension cysts
Rhinolithiasis
Granulomatous destruction (wegener's disease)
Fungal sinusitis

Miscellaneous
  • CONGENITAL

Fronthoethmoid encephalocele
Glioma
Teratoma

Dermoid-Epidermoid cyst
  • SECONDARY
    • Metastasis (renal cell carcinoma, lung)
    • Invasion-infiltration from surrounded area (meningioma, chordoma, lymphoma, paranasal sinus tumors).
  • IDIOPATHIC
    • Fibrouz dysplasia
    • Midline granuloma
Background

Sinonasal tumors are rare. Both the benign and the malignant cases usually give the same symptoms and mimic the inflammatory sinonasal pathologies leading to a delay for the accurate diagnosis. Drug resistant sinusitis must be thoroughly examined against malignancy. Usually repeat biopsies are needed to differ the malignant sinonasal pathologies (4, 5). On the other hand mostly the malignant cases alters the nasal air passage but the clinical and symptomatologic algoritm may not help for differential diagnosis, so cases with unilateral nasal obstruction, diplopia, proptosis, cranial nerve paralysis must be scanned urgently.

Clinical evaluation may not help to detect the invasion. Therefore during the cross-sectional scanning the infraorbital cavity, intracranial fossa, pterygomaxillary fossa, pterygopalatine fossa and the infratemporal fossa must be clearly identified and examined. Also staging and therapeutical options are changed according to the invasion of these anatomical regions and new lesions may be encountered (3, 4).

Benign lesions are usually asymptomatic and coincidentally diagnosed during the radiologic evaluations. Nevertheless, the become symptomatic once the airway passage is narrowed or the ostiums of the sinuses are blocked (1). For example; one of our cases was a dermoid cyst localised laterally in the nasal wall with no extension or invasion. It was differentiated by its hypodense (-123 HU-Hounsfield Unit) nature. Especially small lipomateous lesions must be examined by the multiplanar thin section views against intracranial extensions (1).

Generally the malignant tumors widening in the nasal cavity have low local recurrence rate and curability but highly aggressive with often metastasis. The local invasion is due to the perineural and perivascular invasion or bony lysis. Local recurrence is due to unsatisfactory resection or perineural spreading. Skeletal and pulmonary metastasis is common. Therefore it is always a rule to scan for metastasis in advanced sinonasal tumors. Those sinonasal tumors can extend intracranially through the cribriform plates, fovea ethmoidalis, planum sphenoidale, posterior frontal sinus wall, or medial orbital roof. Malignant tumors of the nasal cavity are highly cellular tumors with little free water, and they may contain focal areas of hemorrhage or necrosis. This is reflected in their heterogenous MR imaging appearances and a low-to-intermediate signal intensity on both T1 and T2-weighted images contrary to benign ones (5-7).

Today in the sinunasal pathologies, after the clinical exammination, if further examination is needed, excluding the malignancies and detecting distant metastasis, contrast enhanced CT or MRI or PET/CT is preferred technique. Soft tissue is better visualised by the MRI where on the bony areas CT has more advantagious. Thus, both methods can be used tandemly. Especially invasion of the orbital roof, cribriform plate, fovea ethmoidalis, posterior maxillary sinüs wall, pterygopalatine fossa, erosion of the sphenoid sinüs wall represent the locally aggressive nature and extranasal invasion of the tumor.
The artefact free scans could be better enhanced by the multidetector CT rather than the conventional spiral ones (8). On the other hand, usually the ostiums of the sinuses are blocked in the sinunasal tumors, so superimposing sinusitis, inflammatory soft tissue deposits and the retention cysts may not provide evident differentiation in the density in CT, they may only be detected by the contrast enhanced multiplanar MRI. In this context, MRI can also differentiate the tumoral tissue from the surrounding edema, fluid or inflammation. Generally the sinunasal tumors have intermediate intensity in the T1w and T2w (w: weighted) sequences, thus even without contrast, T2w hyperintensity of the edema and inflammation could be identified. Intracranial extensions, especially the dural ones, can better viewed by the intravenous contrast enhancement type (8)
Imaging findings OR Procedure details

Sixty-nine cases (male: 37, female: 32, mean age: 43 years) complaining of nasal obstruction and those previously underwent maxillofacial imaging with at least one of the following cross-sectional images (computerized tomography, CT (n=46); magnetic resonance imaging, MRI (n=21); positron emission tomography, PET/CT (n=2)) between January 2005 and May 2010 were recruited. In 7 CT examinations and in all MRI examinations intravenous contrast administration were achieved. CT scans used spiral, 16 or 64 detector multislice systems and axial sections with the coronal, sagittal reformat views were built by the workstations and all cross-sectional images were examined thoroughly. MRI scans used 1.5 T field power and both the multiplanar conventional sequency and contrast coronal and axial fat-saturated sequences were examined thoroughly. All cases that were included in the study had pathologic lesions arising from the nasal septum, nasal passage walls, concha or the paranasal sinuses, which invaded or narrowed the nasal passage. Commons variations such as simple concha bullosa, paranasal sinus derived simple retention cysts, allergic rhinitis, nonfungal rhinosinusitis or nasal polyposis were excluded.

Benign cases (n=48) consisted of nasal alar hemangioma (n=1), nasal angiofibroma (n=1), columellar dermoid cyst (n=1), fibrous dysplasia of the maxillary sinus (n=5), pleomorphic adenoma (n=1), antrochoanal polyp (n=4), sphenoido-anal polyp (n=1), inverted papilloma (n=6), complicated concha bullosa (n=6), rhinolith (n=4), mucocele (n=6), atypical retention cyst (n=4), fungal destructive sinusitis (n=3), nasoethmoid encephalocele (n=4), nasal paraganglioma (n=1). Malignant cases (n=18) consisted of lethal midline granuloma (n=3), sinonasal lymphoma (n=5), chondrosarcoma (n=2), squamous cell carcinoma (n=4), melanoma (n=1), adenoid cystic carcinoma (n=1), esthesioneuroblastoma (n=1), sinonasal Ewing sarcoma (n=1).
Conclusion

The mainstay of the radiologic examination in the tumoral cases of the sinunasal cavity is far more than to make a differential histopathologic diagnosis but to explore the origin, dimensions, orientation of the mass to the airway passage and nasal walls, contours and the contrast enhancement of the tumor. Therefore the surgeon may be briefed preoperatively about the nature of the mass and the need for biopsy (also including the guidance) is questioned.

Concludingly; the cross-sectional hints might help assess the tumoral specifications, extensions, possible differential diagnosis and surgical approaches (Scheme I). The benign and malignant cases may be accompanied by the inflammatory changes but the typical radiologic views may classify and diagnose them.

**Scheme I.** Hints for diagnosis of a mass which presented by the obliteration of the nasal cavity.

**Step by Step to the Lesion differentiation involving Nasal Cavity with MRI and CT**

**Hypointense appearence on MRI or suspicious bony changes, continue with MDCT**

- Benign: generally nonenhanced lesions

Rhinolith (mildly dense), fibrous dysplasia (ground-glass density) or osteoma (very dense) (Figure 1), retention cyst (nonscalloped osseous borders) or mucocele (scalloped-enhancing rim) with insiccipated secretions (Figure 2)

- Benign aggressive: With associated concomitant diseases like diabetes, immune suppression

Fungal infections (mucormycosis).

- Malign: Densely scattered or arch like calcific islands throughout the tumoral stroma on CT images

Soft tissue sarcomas including calcification (osteosarcoma, chondrosarcoma) (Figure 3)

**Existence of the lipomateous content**

- Dermoid (Figure 4), epidermoid lesions, postoperative flap or repositioned graft (history!)

*In the nonspecisific appearence on both the T1w-T2w MRI sequences, examination must be continued after contrast administration.*
• Mural enhancement

Sinusitis, mucocele (Figure 2), retention cyst

• Internal septal contrast enhancement

Polipozis (diffuse), polyp (pedunculated with smooth contours), inverting papilloma (pedunculated with lobulated contours, usually originated from osteomeatal unit) (Figure 5)

• Erosion, nekrosis or destruction with heterogenous enhancement

Malignant lesions (Squamous cell carcinoma, adenocarcinoma, metastasis), Lymphoma, granulomatous diseases (Figure 6)

• Homogenous and dense contrast enhancement

Tubulary hypodense or hypointense vasculary traces (hemangioma), Angiofibroma (infiltrative and expansile) (Figure 7)

_Pronounced midline destruction (can be diagnosed only with histopathological examination)_

• Benign

Granulomatous disease (Wegener)

• Malignant

Lymphoma (Figure 8), other primary malignant tumors or metastases

_Masses with smooth borders but enhanced differently from mucoceles or retention cysts_

Nerve tissue (Schwannoma, Paraganglioma) (Figure 9), minor salivary gland (pleomorphic adenoma) (Figure 10)

_Cranial Vault Associated (Can be diagnosed by typical appearences)_

• Benign

Fronthoethmoid encephalocele (a reces which contains basla frontal gyri)

• Malignant

Estesioneuroblastoma, Meningioma, Chordoma (typical location and appearences)
Fig. 0: a) Coronal CT section a case with rhinolith which shows an oval lesion with mild density with relatively lucent central area seated near to right middle meatus, b) after extirpation, it is seen as fragmented nature. c) A densely sclerosed lesion originated from inferior ethmoid cells and obliterates the left osteomeatal unite on the left side d) Diffusely thickened and acalloped borders of right maxillary sinus medial wall also narrows the right nasal passage.

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Fig. 0: MRI examination of an ethmoid mucocele. a) hyperintense on coronal T2w image (arrow), b) hypointense on T1w precontrast image (arrow), c, d) On these contrast enhanced coronal fatsaturated images, extention (arrows in c and d) and origination (arrow in d) are seen. Lesion enhanced only murally and showed no internal signal increase on enhanced images. Endoscopic image shows the lesion an-phase that emanating from the ethmoid recess.

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Fig. 0: Nasal (left: total, right: partially) cavity aeration is obliterated by the soft tissue mass that containing scattered calcific islands (chondrosarcoma).
Fig. 0: Nasal dermoid lesion. a) Three-dimensional CT appearance of a columellar lesion. b) Axial CT image shows the hypodense, lipomaceous lesion (arrow).
Fig. 0: Certain polypoid nasal cavity masses. a) Antrocoanal polyp, originating from left maxillary sinus antrum (arrow), b) And extending to the left coanal passage (arrow). c) A sphenocoanal polyp which originating from the left sphenoid sinus ostium (arrow) with its vertical orientation is diagnostic (arrow) d) Although having smooth (atypically) borders, erosion of the right inferior conca and chondrovelomeral junction (arrow) direct the diagnosis to inverting papilloma.

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Fig. 0: Squamous cell carcinoma. a) Obliteration of the right nasal passage with a mass that has not obvious borders that seated between right ethmoid cells and nasal cavity (between arrows). b) Unfortunately, erosion of the right cribriform plate and enhancement with frontobasal focal dural thickening (arrow) shows the cranial involvement. c) Also, obliteration of the right frontonasal recess causes right frontal sinusitis (arrow). permits transfer to the dural surface of anterior cranial fossa (b, arrow).

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**Fig. 0:** a) Left nasal alar area located lesion causes partially narrowed aeration and contains some hypodense tubulolineary focuses (arrow), hemangiom. b) At the posterosuperior pharyngeal location, an expansile mass lesion is seen with scalloped anterior borders of the anterior wall of the sphenoid sinus (arrow), angiofibroma.

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**Fig. 0:** Lymphoma. a) An expansile nasomaxillary mass causes facial asymmetry at 3D-CT volume rendered image. b) When the window settings are adjusted to the bone level, it is possible to see the destruction with free teeth. c) Saggitally reconstructed CT image shows the obliteration of the nasal passage inferiorly (arrow).

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**Fig. 0:** Paraganglioma. a) A small right nasal mass located in middle meatus, causes accumulation of T1w hyperintense secretion in the right maxillary sinus due to obliteration of the right osteomeatal unit (arrow). b) On postcontrast axial T1w sequence, lesion enhanced densely and heterogenously. c) At the cytopathologic examination, it can be seen that tumor is composed of cells which have pale eosinophilic cytoplasm and round nucleus. There is prominent vascular network separating the tumor nests (H&E x 100).

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**Fig. 0:** Involvement of the palatum durum with an expansile lesion (paraganglioma) on coronal CT image (a, arrow). And on coronal T1w MRI image (b, arrow). Postcontrast coronal fatsaturated T1w image shows the heterogenously enhancement of the lesion (c, arrow). The epithelial-myoepithelial islands of tumor in myxoid stroma (d) (H&E x 200).
Fig. 0: Frontoethmoid encephalocele. a) T2w coronal sequence and b) T1w postcontrast saggital image show the continuity of left fronthobasal neural parenchyma thorough the left upper and middle nasal meati. This typical appearance must be taken into consideration to prevent any dangerous intervention.
References

1. Cody DT, Lawrence WD: Neoplasms of the nasal cavity. In: Cummings CW, Fredrickson JM, Harker LA, et al, eds. Otolaryngology-Head and Neck Surgery. Mosby; 1999.

2. Carrau RL, Myers EN, Johnson JT. Paranasal sinus carcinoma diagnosis, treatment, and prognosis. Oncology 1992;6:43-50.14. Kraus DH, Roberts JK, Medendorp SV, et al. Non squamous cell malignancies of the paranasal sinuses. Ann Otorhinolaryngol. 1990;99:5-11.

3. Spiro JD, Soo KC, Spiro RH. Non squamous cell malignant neoplasms of the nasal cavities and paranasal sinuses. Head Neck l995;17:114-118.

4. Grossman RI, Yousem DM. Sinonasal disease. In: Neuroradiology the Requisites. Philadelphia, PA: Mosby, 2003. p.611-38.

5. Mark D. Murphey, MD, Eric A. Walker, MD, Anthony J. Wilson, MB, ChB, Mark J. Kransdorf, MD, H. Thomas Temple, MD and Francis H. Gannon. Imaging of Primary Chondrosarcoma: Radiologic-Pathologic Correlation. Radiographics. 2003;23:1245-1278.

6. Carrau RL, Myers EN, Johnson JT. Paranasal sinus carcinoma diagnosis, treatment, and prognosis. Oncology 1992;6:43-50.14. Kraus DH, Roberts JK, Medendorp SV, et al. Non squamous cell malignancies of the paranasal sinuses. Ann Otorhinolaryngol. 1990;99:5-11.

7. Spiro JD, Soo KC, Spiro RH. Non squamous cell malignant neoplasms of the nasal cavities and paranasal sinuses. Head Neck l995;17:114-118.

8. Allbery SM, Chaljub G, Cho NL, Rassekh CH, John SD, Guinto FC. MR imaging of nasal masses. Radiographics. 1995 Nov;15(6):1311-27.