Chapter

REAHs and REAH-Like Lesions: Underdiagnosed lesions Often Misconfused with Nasal Polyps

Ph. Eloy, C. Fervaille and M.C. Nollevaux

Abstract

REAH is the eponym for respiratory epithelial adenomatoid hamartoma. The disease is under diagnosed. It is clearly a disease in the olfactory cleft. It is characterized by a polypoid process located in the olfactory cleft which does not evolve in inverted papilloma or malignancy set at 10–15 cm. The lesion can be isolated in one or both olfactory cleft. It can be asymptomatic or can cause nasal obstruction and impairment of smell. More commonly the lesions, often multiple, are associated to the recurrence of the nasal polyposis. They can contribute to the development of loss of smell, nasal obstruction or even the blockage of the frontal recesses. The definitive diagnostic is based upon the histologic examination. Surgery is the treatment. In case of isolated lesion, complete excision without complete ethmoidectomy is the option. In case of lesions embedded in a recurrent massive polyposis, a complete exenteration of the olfactory clefts associated to a revision of full house ethmoidectomy and even a Draf III must be considered.

Keywords: REAHs, REAH-like, nasal polyposis, olfactory cleft

1. Introduction

REAH is the eponysm for Respiratory Epithelial Adenomatoid Hamartoma. It is a relatively new diagnosis, only added to the World Health Organization classification of tumors in 2005 [1].

Wenig and Heffner were the first to describe it in 1995 in a series of 31 cases [2]. They described it as "a proliferation of glands lined by multi-layered ciliated respiratory epithelium, often with admixed mucocytes, arising in direct continuity with the surface epithelium, which invaginated downward into the submucosa."

The lesion is usually diagnosed in middle-aged patients. Clinically it may manifest as a solitary lesion or in association with sinonasal polyposis. The former is far less common than the latter. The lesion takes its origin either in the olfactory cleft, the posterior septum, or the rhinopharynx [1–5] Because the incidence, clinical signs, imaging, modality of treatment, outcomes, and pathogenesis seem to be quiet different between these two clinical patterns, we call the first ones "REAHs" and the second ones "REAH-like." This terminology is proposed by Jankowski [3, 5] and Hawley [4] as well.

The purpose of this paper is to remind the histopathological characteristics and differential diagnosis of REAHs/REAH-like lesions and to report two different

cohorts of patients (one with REAHs and the other with REAH like lesions), treated in the ENT department of the CHU UCL Namur.

2. Histopathological characteristics of REAHs and REAH-like lesions

Grossly, REAH looks like a "polypoid fleshy to firm mass with areas of induration." It is yellow or white [6]. It may have varying sizes (**Figures 1–3**).

The histologic picture is dominated by the presence of a glandular proliferation with a polypoid appearance. The proliferation starts from the surface epithelium and tends to be submucosal.

The glands are lined by ciliated respiratory epithelium originating from the surface respiratory epithelium. The glands are typically round to oval in shape and were small to medium in size with prominent dilation. Stromal tissue separates the glands. The epithelium may be cuboidal or flat, and mucinous gland metaplasia is often seen. Occasionally the gland lumina are filled with mucinous or eosinophilic amorphous material. It often demonstrates periglandular stromal hyalinization, and there is often a mixed inflammatory infiltrate within the stroma.

In the literature we can find another type of REAH called **COREAH**. It is characterized by a chondro-osseous differentiation. Flavin [7] and Roffman [8] were the two first authors to publish this entity, respectively, in 2005 and 2006. Since then 11 cases have been reported [9, 10]. It can occur in children or adults.

The histological features are almost exactly the same as REAH, but COREAH has islands of immature hyaline cartilage interspersed throughout the lesion (**Figures 4** and **5**).

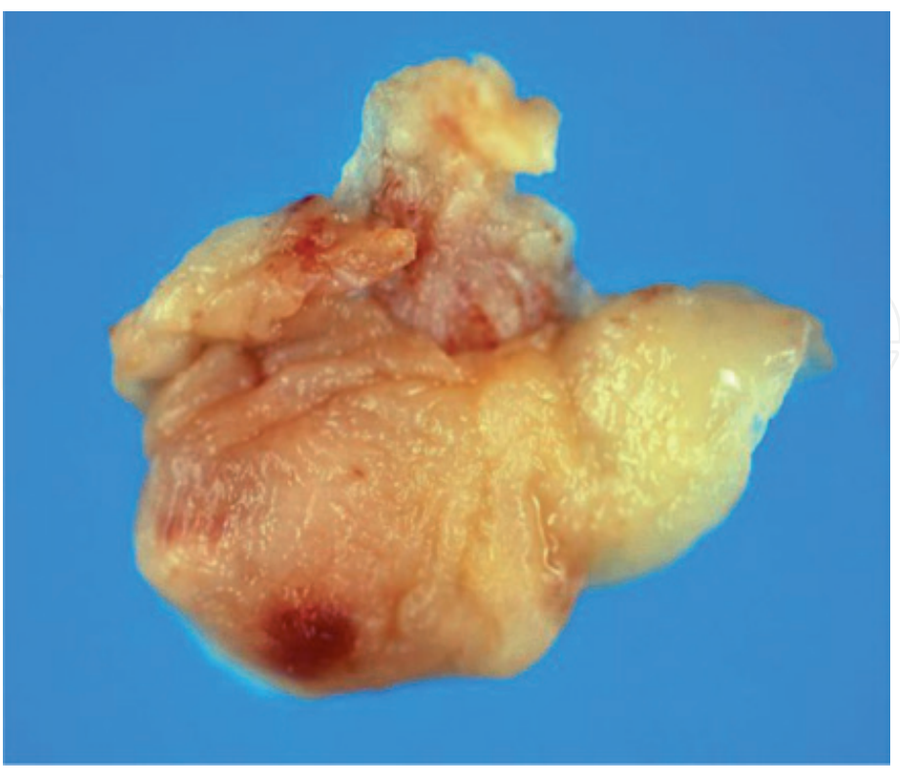


Figure 1.
Gross appearance of a REAH.

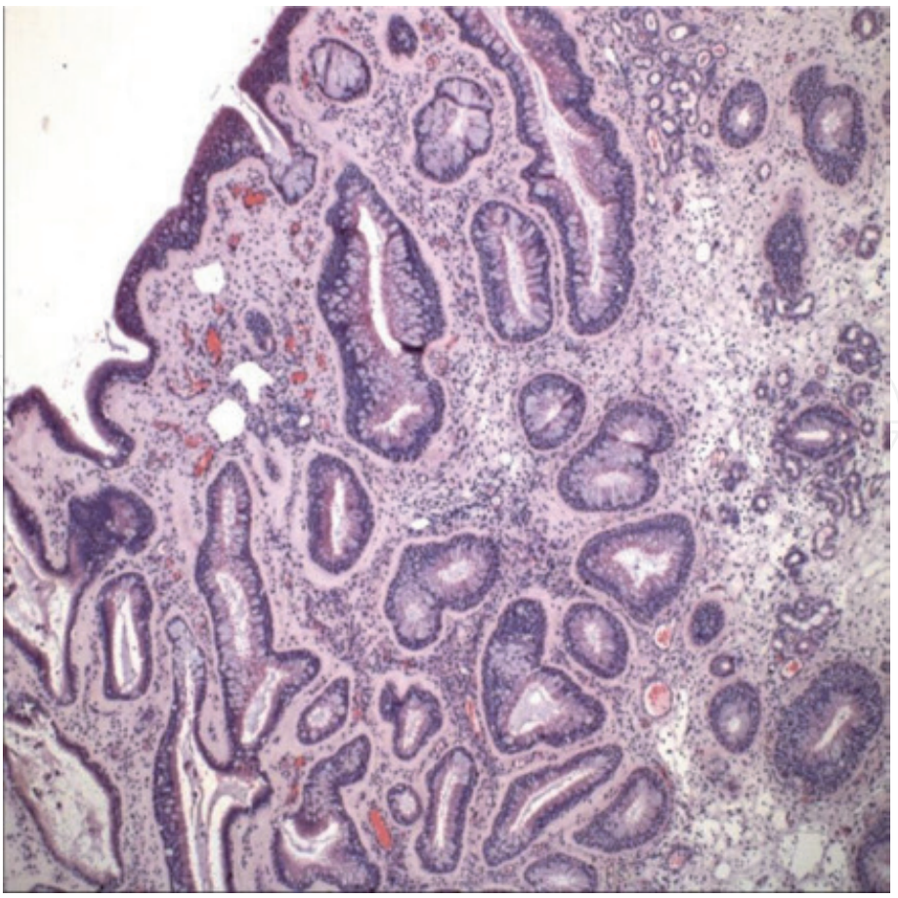


Figure 2.
REAHs: The glandular proliferation arises in direct continuity with the surface epithelium with invagination downward into the submucosa. Clusters of seromucinous glands are seen.

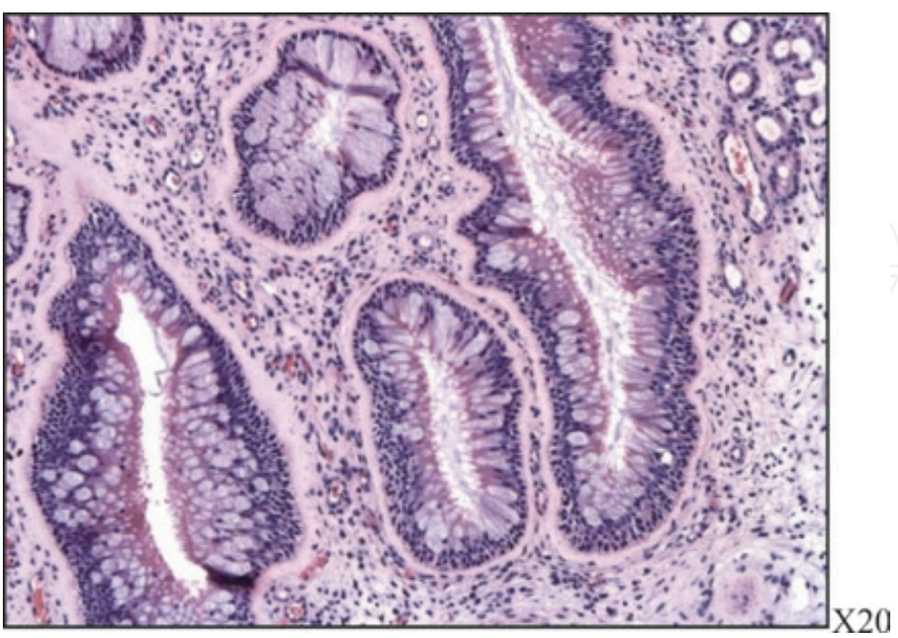


Figure 3.
REAHs: Occasionally the gland lumina are filled with mucinous or eosinophilic amorphous material. It often demonstrates periglandular stromal hyalinization, and there is often a mixed inflammatory infiltrate within the stroma.

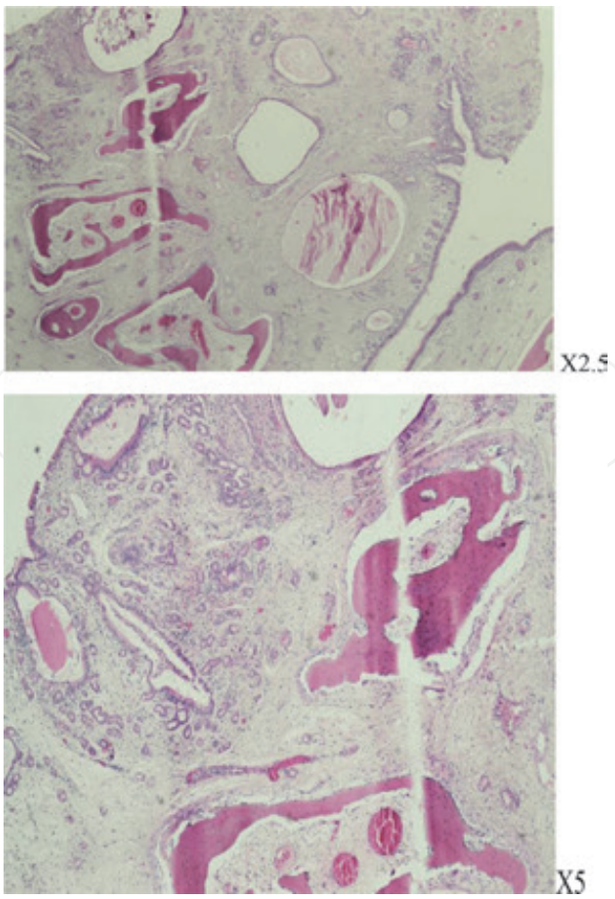


Figure 4.
COREAH: Nasal chondromesenchymal hamartoma. Multiple tumor fragments with a mucosal surface and nodules of cartilage (in red).

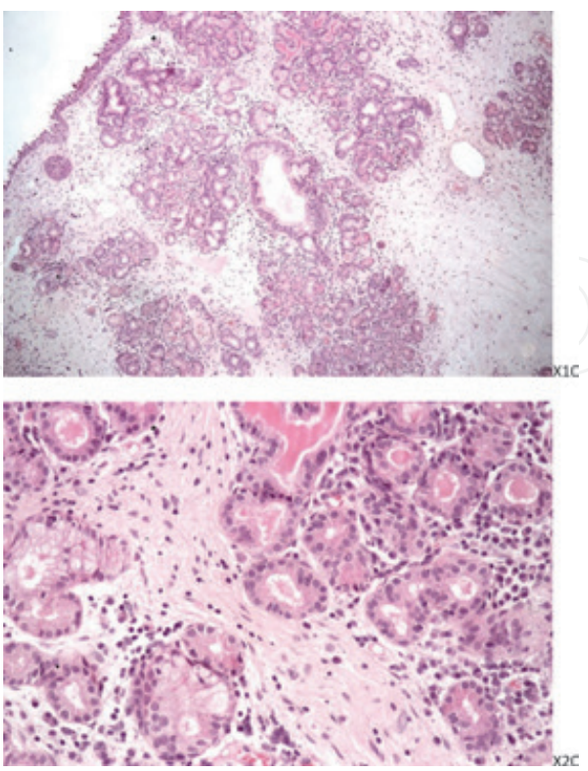
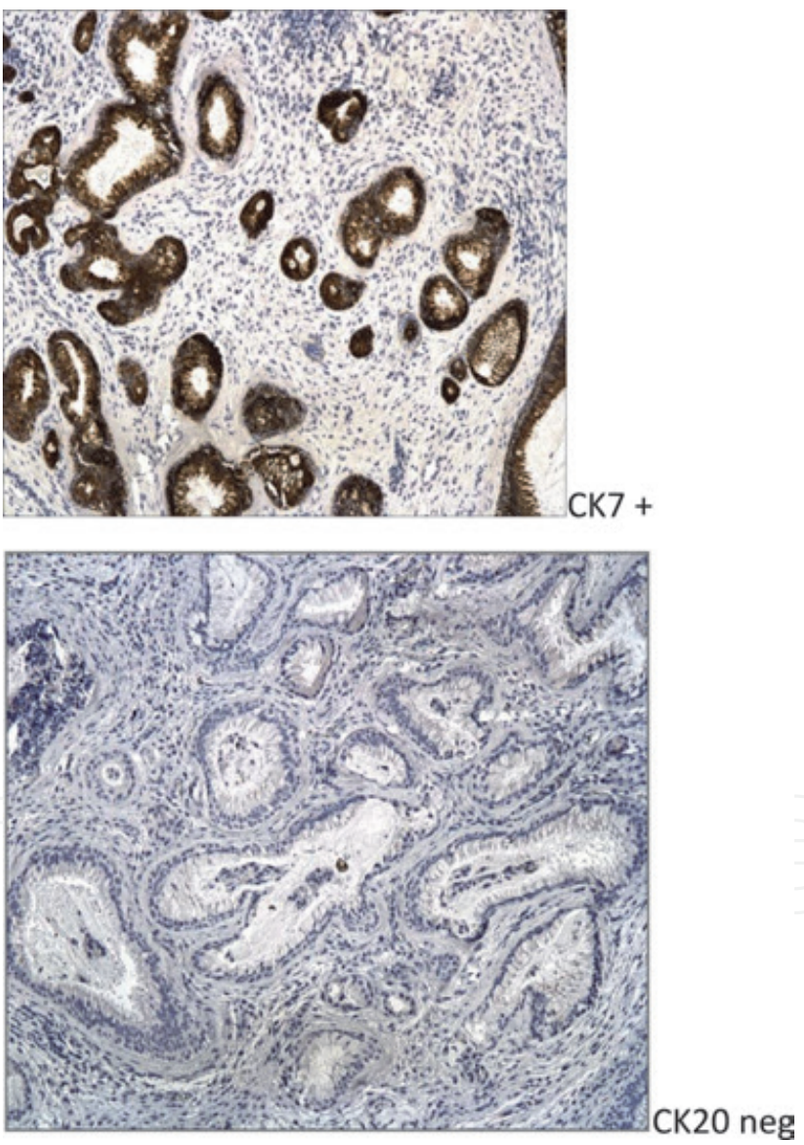


Figure 5.
Seromucinous hamartoma: The mass is covered by respiratory epithelium and is comprised of lobular or haphazard proliferations of small to large glands and ducts which are lined by a single layer of cuboidal or flattened epithelial cells.

3. Immunohistochemistry of REAHs

Immunohistochemistry has not been used to an extensive degree in the diagnosis of REAH, and it is not absolutely necessary to use it to make the definitive diagnosis of it.

However, Ozolek et al. did an immunohistochemical study in which they examined the profile of REAH [11]. REAHs are positive for CK7, negative for CK20 and CDX-2, and positive for MIB1 and Ki67. p63 staining was seen in the basal cells of REAH, which had a low proliferation index. The use of Mindbomb 1 (MIB-1) is useful in distinguishing REAH from other neoplasms, since neoplasms tend to have a high proliferation index.



REAHs: Immunohistochemistry: Positivity for CK7 and negativity for CK20.

4. Differential diagnosis of REAHs

The differential diagnostic of REAHs concerns the inflammatory polyps, Schneiderian papillomas, seromucinous hamartoma, and low-grade non-intestinal adenocarcinoma.

4.1 Inflammatory polyps

Inflammatory polyps are certainly the most common lesions that are confused with REAHs.

The most notable clinical differences between REAHs and inflammatory polyps are the location and their gross appearance.

Inflammatory polyps are typically the clinical manifestation of a sinonasal polyposis. Nasal polyps are rarely isolated. They are multiple and bilateral and usually extrude from the middle and superior meati. They are rarely attached to the posterior septum. REAHs originate specifically from the olfactory cleft.

Nasal polyps are usually edematous and not indurated. On microscopy, both lesions can show fibroblastic and vascular proliferation, stromal edema, a mixed inflammatory cell infiltrate, and seromucinous gland proliferation. However, inflammatory polyps do not have florid adenomatoid proliferation and stromal hyalinization which, when present, favor REAHs (**Figures 6–9**).

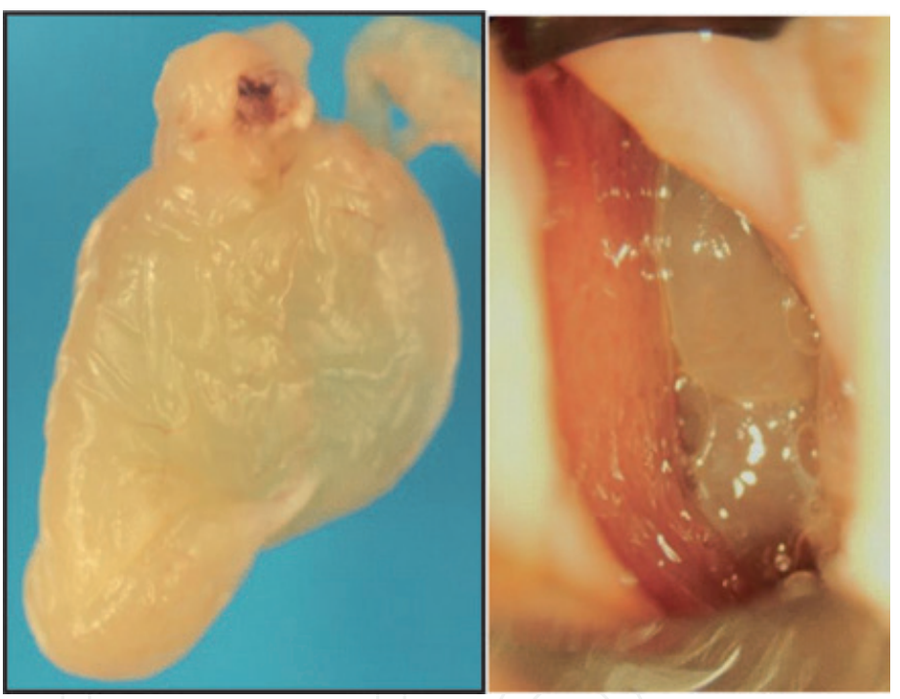


Figure 6.
Macroscopic and endoscopic view of a nasal polyp.

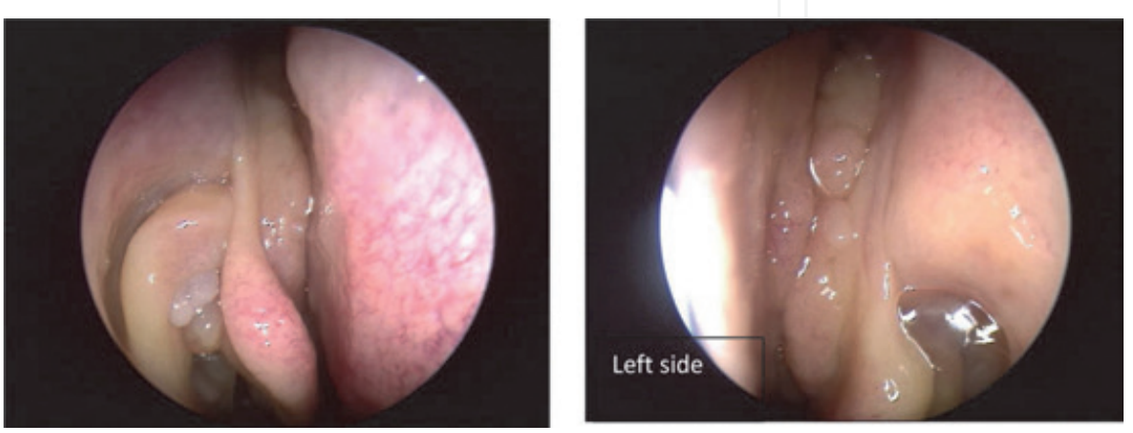


Figure 7.
Endoscopic view: right and left nasal cavity: presence of nasal polyps in the middle and superior meati.

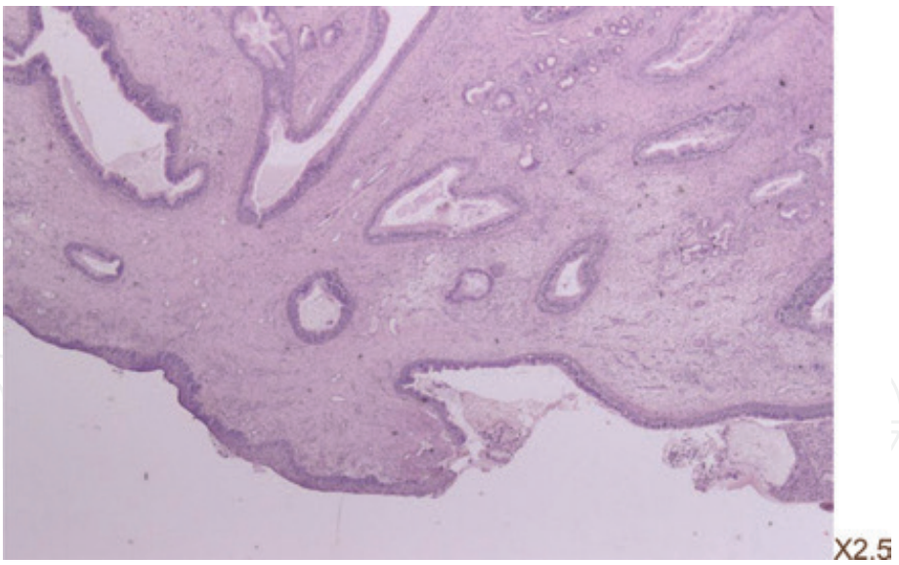


Figure 8.
Inflammatory polyp with edematous inflammatory stroma and single-layered glands.

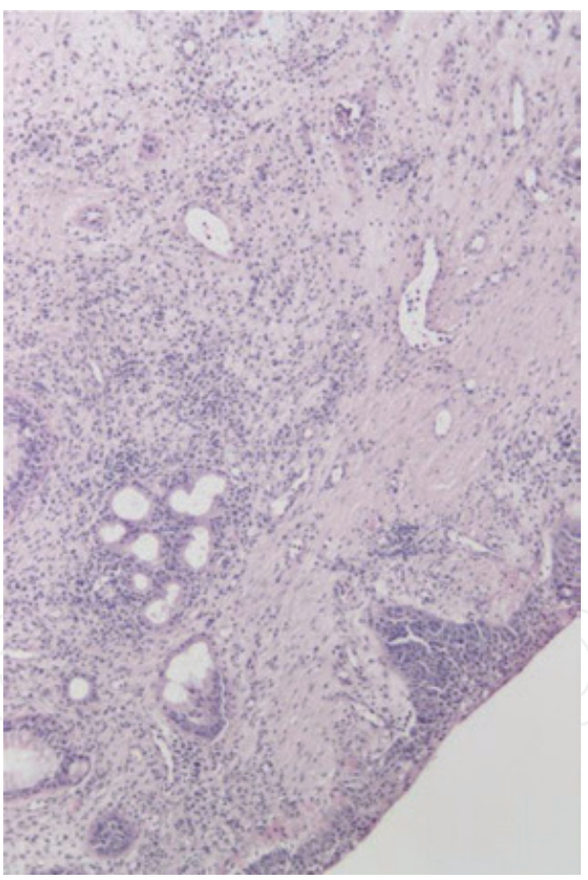


Figure 9.
Inflammatory polyp showing inflammatory stroma without hyalinization.

4.2 Schneiderian papillomas

This is the second important differential diagnosis of REAHs.

Schneiderian papillomas are benign epithelial neoplasms of the sinonasal tract. Their annual incidence ranges between 0.2 and 1.5/100,000 people per year.

They are classified in three types: exophytic/fungiform papilloma, endophytic/inverted papilloma, and oncocytic/cylindrical cell papilloma. The inverted type is

the most common, accounting for nearly two thirds of the cases. We limit the description to this type.

It is mostly unilateral. It occurs mainly in adults during the fifth or sixth decade. There is a predilection for men.

Unlike inflammatory polyps and REAHs, inverted papillomas are considered true neoplasms. While REAHs tend to be located medial to the turbinate lamella, inverted papillomas have a predilection for the lateral wall of the nasal cavity or the paranasal cavities. Maxillary and ethmoid sinuses are the most common origins followed by the sphenoid and frontal sinuses. Even if inverted papillomas are benign histologic lesions, clinically they may be aggressive with a relatively strong potential for local destruction, high rate of recurrence (more or less 50%), and a risk of carcinomatous evolution. This transformation in squamous cell carcinoma can be synchrone or metachrone and more likely in case of recurrence. This malignant transformation has never been observed in the case of REAHs.

Human papilloma virus seems to be implicated in the pathogenesis of inverted papillomas. Chronic inflammation seems to be a favorizing factor in REAHs.

The treatment of inverted papilloma requires a more extensive and radical excision with a subperiosteal dissection and a drilling of the base of implantation. Endonasal medial maxillectomy is the golden standard for maxillary sinus origin. Recurrence is more likely in frontal sinus papillomatosis due to the localization and the difficulty to completely eradicate the lesion. The surgical treatment for REAHs is a complete excision without ethmoidectomy.

Grossly, inverted papilloma looks like a reddish-gray lobulated tumor, more firm than an inflammatory polyp, with a fairly characteristic "raspberry" aspect (**Figure 10**).

Histologically inverted papillomas have an endophytic growth pattern. There is an invagination of stratified squamous epithelium with an admixture of mucin containing cells and microcysts. The epithelium may be of squamous, transitional,

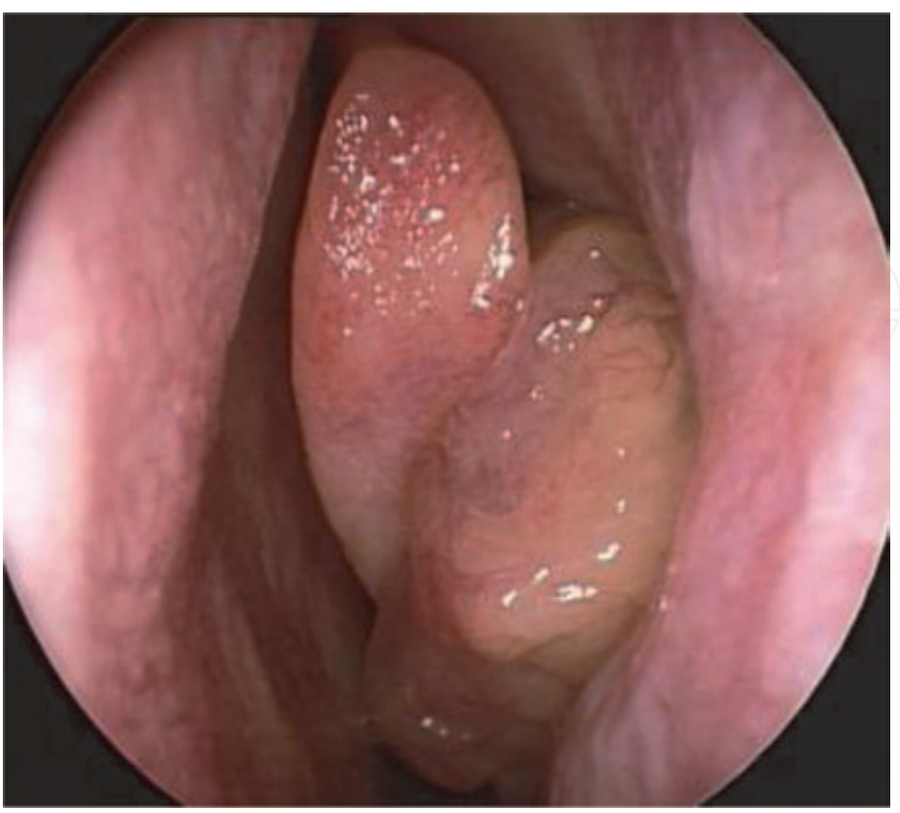


Figure 10.
Endoscopic view of an inverted papilloma originating from the left maxillary sinus.

or respiratory type. The endophytic growth of squamous epithelium is not seen in REAH. Transmigrating neutrophils and neutrophilic microabscesses may be seen. Occasional mitoses may be seen in the basal layer. Mild to moderate atypia may be seen. Edema or chronic inflammatory infiltrate is present in the stroma (**Figures 11–13**).

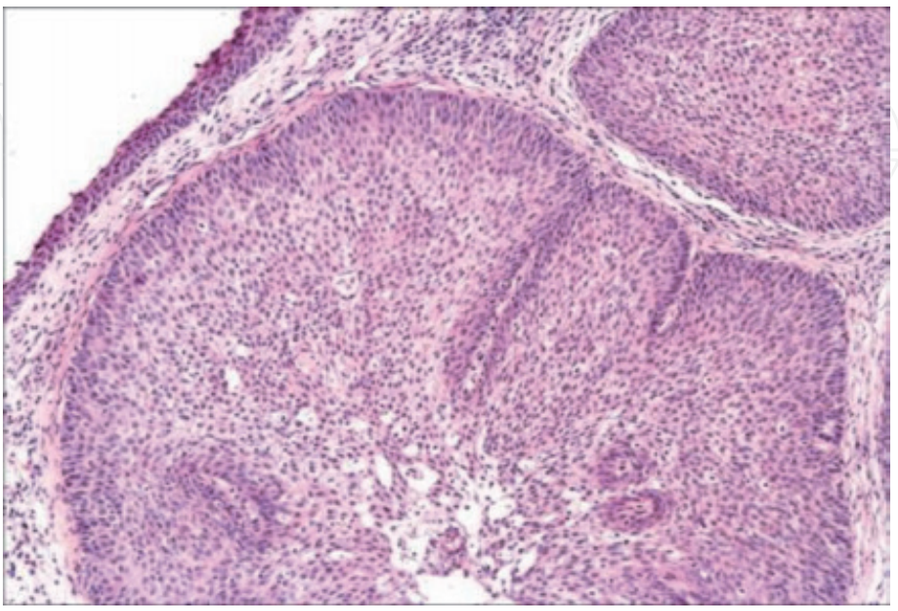


Figure 11.
Low magnification: typical view of an IP: it shows an endophytic growth pattern consisting of markedly thickened squamous epithelial proliferation growing downward into the underlying connective tissue stroma to form large clefts, ribbons, and islands. Note the absence of mucoserous glands. Delicate basement membrane.

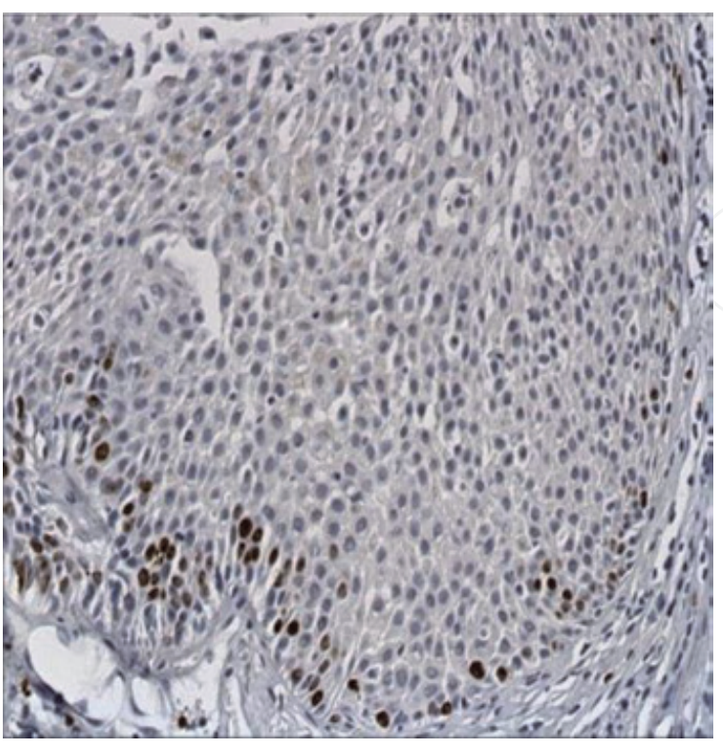


Figure 12.
Immunohistochemistry: high power shows the epithelium to be composed of pseudostratified columnar cells/ positivity of MIB1 in the basal cells.

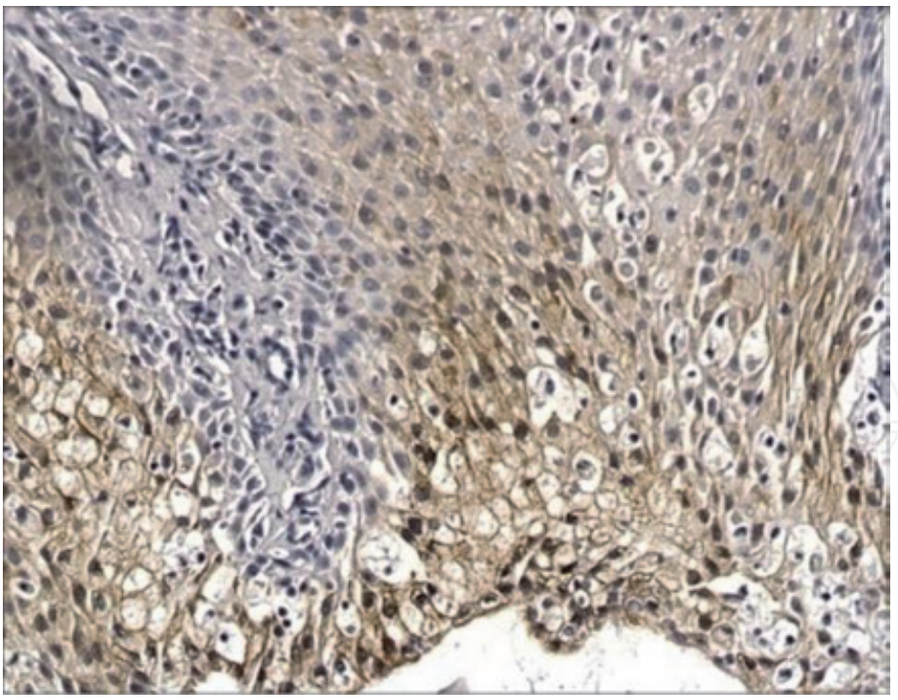


Figure 13.
Immunohistochemistry: high power/positivity of CK7.

4.3 Seromucinous hamartoma

Seromucinous hamartoma is another subtype of hamartoma, recently described. It is a benign lesion of the sinonasal tract as well, located in the posterior nasal cavity or rhinopharynx frequently associated to REAHs. Since its description in 1974 [12], only a small number of additional cases have been reported.

The lesion occurs equally in men and women. Patients are middle-aged to elderly (mean age 50–60) and have complaints with nasal obstruction or nosebleeds. Surgical excision is the treatment of choice. It is included in the differential diagnosis of low grade epithelial proliferations of the sinonasal tract. The differentiation with low-grade non-intestinal adenocarcinoma can be tricky.

Histologically, the mass looks like a benign lesion. Lobular or haphazard proliferations of small to large glands and ducts lined by a single layer of cuboidal or flattened epithelial cells are visualized. Eosinophilic secretions are often present within tubules. The surrounding stroma often contains a lymphoplasmocytic inflammatory infiltrate. Periglandular hyalinization can be observed. There are no features suggesting a malignancy. There are no nuclear atypia.

Seromucinous hamartomas are positive for CK7 and CK19 and negative for CK14 and CK20. The serous glands are usually S100 positive and negative for p63 and muscle-specific antigen.

4.4 Low-grade sinonasal adenocarcinoma (LGSNAC)

Sinonasal adenocarcinoma is the third differential diagnosis for REAH. It accounts for approximately 20% of all sinonasal malignancies and is classified into intestinal and non-intestinal salivary and non-salivary types. Intestinal adenocarcinomas (also called ITAC) take their origin in the olfactory cleftl and then extend into the ethmoid sinus, the orbit, or the anterior cranial fossa. It is an occupational disease; they typically occur in woodworkers. They look like an irregular exophytic pink necrotic and friable mass bulging into the nasal cavity located between the nasal septum and the turbinate lamella.

On microscopy, papillary and colonic types are the most common architectures. Differentiating ITAC from REAH is usually not difficult as the cell types, high-grade features, and increased mitotic index are characteristics for ITAC. ITAC is positive for CK20 and MIB1 and negative for CK7 (**Figure 14**).

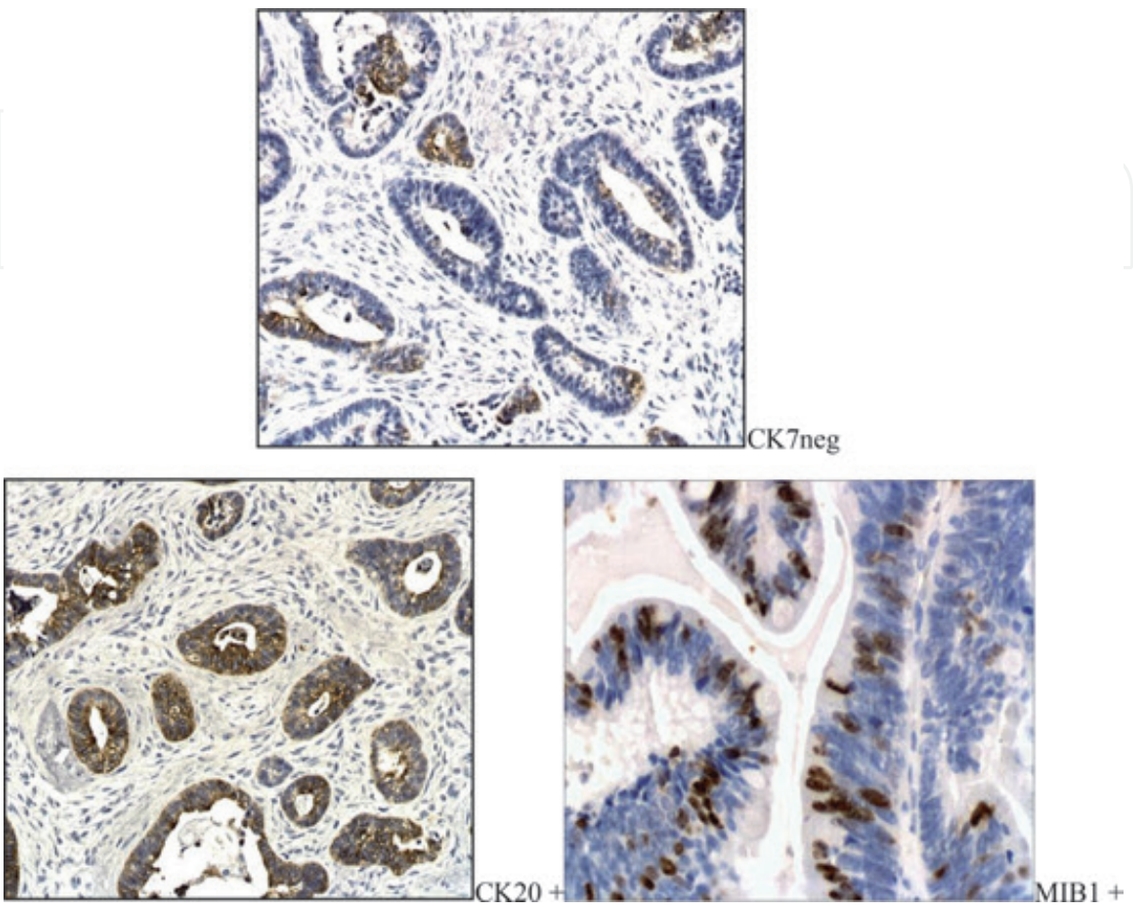


Figure 14.
Intestinal-type adenocarcinoma: Immunohistochemistry—Positivity for CK20, CK7, and MIB1.

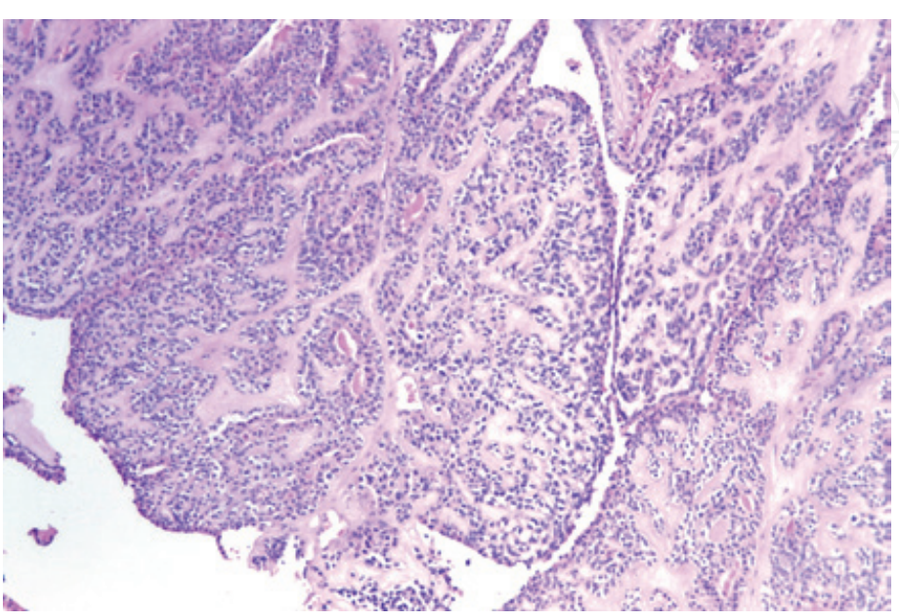


Figure 15.
LGSNAC: Glandular proliferations lined by cuboidal to columnar cells which are usually monomorphic and cytologically bland.

On the other hand, low-grade non-intestinal adenocarcinomas (LGSNAC) are less common and less invasive. There is no sex or racial predilection. There is no association with wood dust exposure. They have no tendency to give systemic metastasis. However, they have a potential for local invasion and destruction of tissue. Extensive surgery is recommended to be associated with radiotherapy in some cases.

Histologically, the mass originates from the surface epithelium and the seromucinous glands of the submucosa. It consists of glandular proliferations lined by cuboidal to columnar cells which are usually monomorphic and cytologically bland. It forms a diverse group of bland tubular and/or papillary tumors. Mitoses are rare. Necrosis, perineural invasion, and lymphovascular invasion are absent. The stroma contains an inflammatory infiltrate as in REAHs.

Immunohistochemistry shows the positivity for CK7 and S100 and negativity for CK20 and CDX2.

The main differential diagnosis is between LGSNAC and seromucinous hamartoma (**Figure 15**).

5. Our experience

5.1 REAHs as a solitary lesion

This clinical presentation of REAHs is actually the less frequent.

Table 1 reports a cohort of eight cases diagnosed and treated in the ENT department of the CHU UCL Namur since 2008.

There were seven women. The mean age was 65 years old. Ranges are 27 and 81.

There was one man: age 53 years old.

The lesions were unilateral in six patients (three left sided; three right sided) and bilateral in two.

Two patients were asymptomatic. REAH was diagnosed by nasal endoscopy and a sinus CT scanner performed for an assessment of epiphora, a case of nasal dysfunction and another one to rule out sinus disease associated to his allergic rhinitis.

The other patients complained with nasal obstruction and rhinorrhea.

Patient	Gender	Side	Symptoms
Br, L.	Female	R/olfactory cleft	Asymptomatic dacryoscan; 2009
Sch, M.	Female	L/septal implantation	Unilateral nasal obstruction 2008
Van rent. M.	Female	X2/olfactory cleft	Bilateral nasal obstruction 2012
W. Jes.	Female Seromucinous hamartoma	R/sphenochoanal recess	Unilateral nasal obstruction 2012
B. Pat.	Female/COREAH	L/sphenochoanal recess	Unilateral nasal obstruction/nasal collapse 2019
H. C.	Female	L/olfactory cleft	Unilateral nasal obstruction 2019
Tr. M.	Female	R/olfactory cleft	Asymptomatic Ct finding 2019
N, A.	Male	X2 R > L	Allergic rhinitis; paucisymptomatic/nasal endoscopy/CT scan 2019

Table 1.
Reports our experience of REAHs.

REAHS originated from the olfactory cleft in six patients and from the anterior wall of the sphenoid sinus in two cases.

On nasal endoscopy the lesion looked fleshy, with no vascular component and no necrosis.

On imaging the lesion was solitary in the olfactory cleft. No chronic rhinosinusitis was present (**Figure 16**).

The MRI performed in three cases was not so helpful. There were no pathognomonic features whatever was the localization. In the literature, REAHs appear as a homogeneous mass with post-contrast enhancement on T1-weighted sequences as well as hyperintensity on T2-weighted images (**Figure 17**) [13].

The diagnosis of REAH was confirmed in all the cases by the pathologist.

In one case it was a COREAH, and in another case it was a seromucinous hamartoma. These two hamartomas were located in the posterior nasal fossa.

A biopsy was performed under local anesthesia in asymptomatic cases to make a formal diagnosis. For the other the diagnosis was made on the surgical specimen.

There was no clear etiologic factor that could have played a role in the development of REAH except in one patient suffering from allergic rhinitis. There was no concomitant chronic sinusitis, asthma, or aspirin intolerance.

Concerning the management, in two patients a wait and see attitude was proposed as the patient was not symptomatic. For the others an endoscopic resection of the lesion was performed under general anesthesia. The dissection was done in a subperiosteal plane. We have never drilled out the site of implantation. There was no need to do a full house ethmoidectomy.

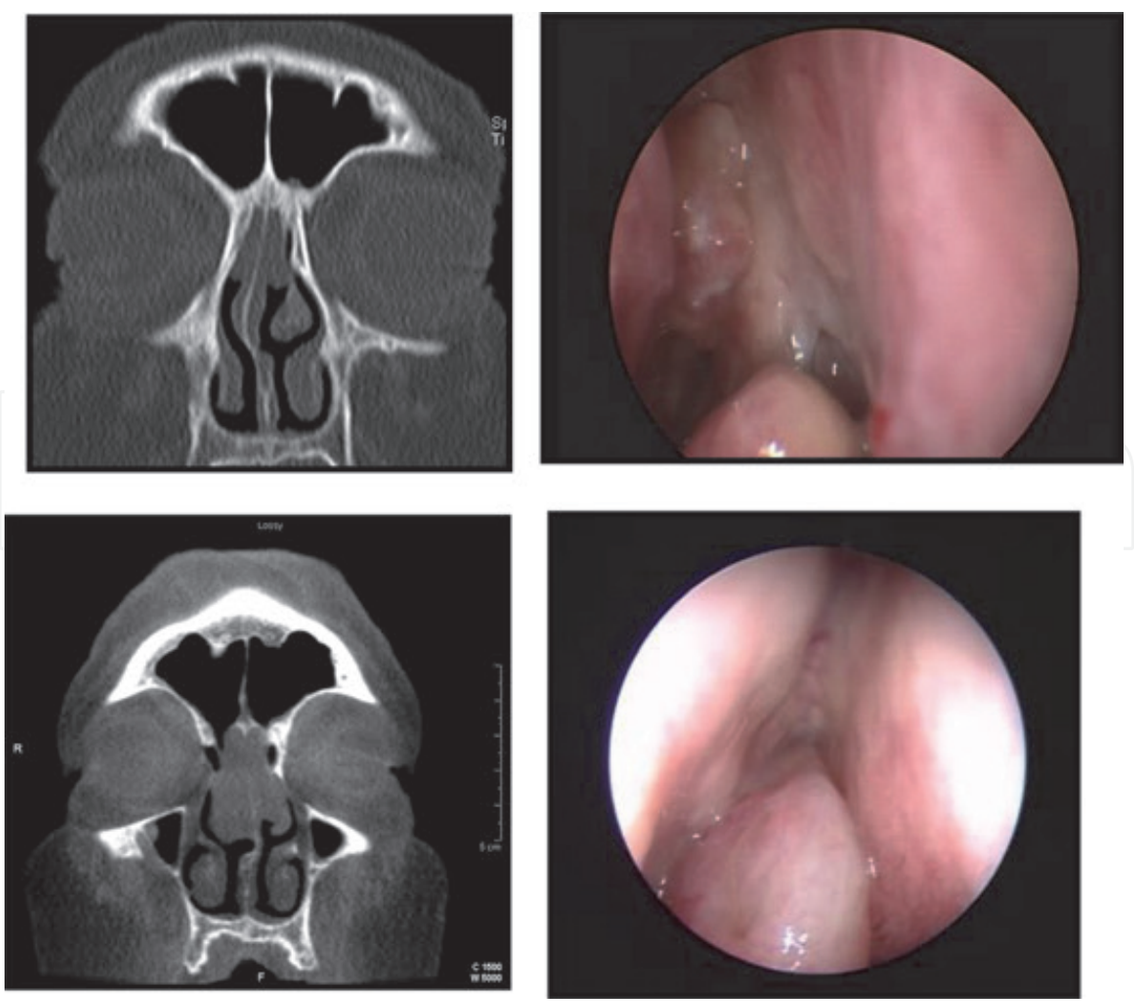


Figure 16.
Comparison between CT scan and nasal endoscopy.

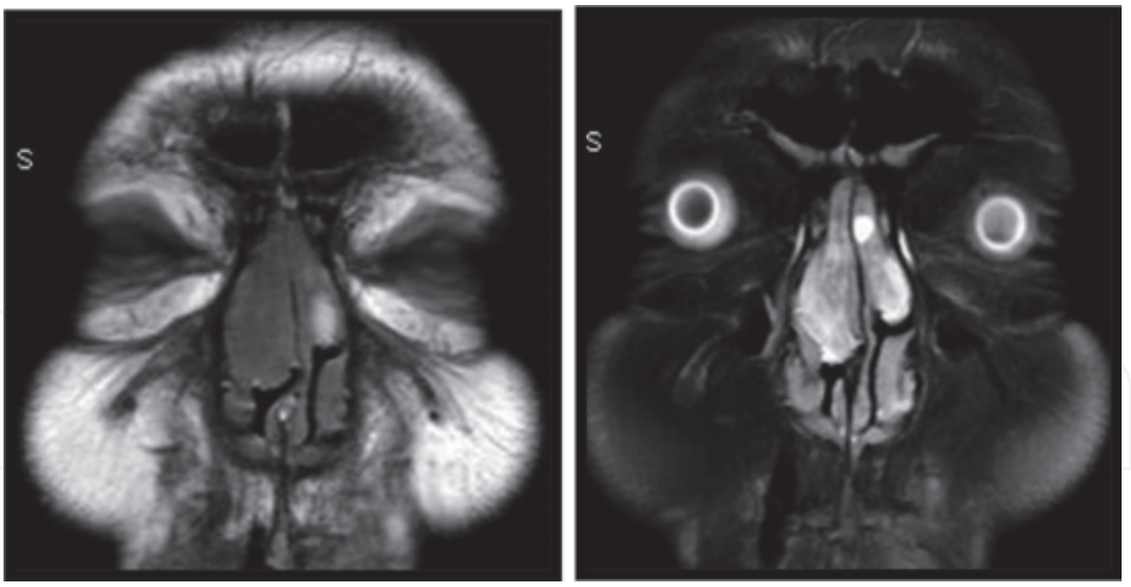


Figure 17.
MRI of a patient with bilateral REAHs: T1- and T2-weighted sequences.

Until now we have had no recurrence (**Figure 18**).

5.2 REAHs associated to a nasal polyposis often previously operated also called REAH-like lesions

This is the second clinical pattern of REAHs, and certainly this is the most common type.

Table 2 reports a cohort of 16 patients diagnosed with such a pattern during the past 18 months.

5.2.1 Epidemiology

The series includes 13 men and 2 women. The mean average is about 63 years old.

The majority of the patients are in the fifth and sixth decades.

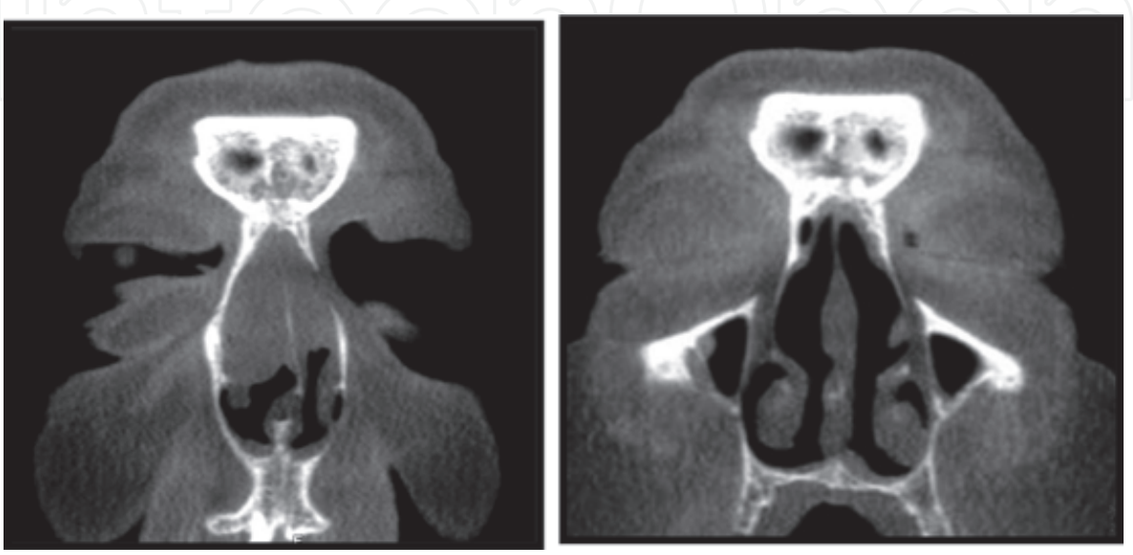


Figure 18.
Illustration of a case with bilateral REAHs: pre- and postop imaging.

Patient	Sex	Disease/surgery
S. Jac.	M	Asthma/rev surgery: REAH-like X2/draf III
Fris. JL	M	Polypose XZ/revision ethmoidectomy/REAH: chronic otitis media
Ros. G	M	Recurrent NP/asthma: REAHs/rev ethmoidectomy
Hub. S.	M	Sever NP/complete ethmoidectomy
L.; Th.	M	Asthma: seromucous otitis media/revision surgery/REAH
Br. Cl.	M	Aspirin intolerance; asthma/recurrence
De Ras.Ge.	M	Nasal polyposis and asthma /previous surgery
Bran. Ar	M	Revison surgery for massive polyposis
De pl. Ph	M	Nasal polyposis operated 3 times/no asthma
Hou. Adr.	M	Recurrentpolyposis/rev ethmoidectomy
Aig; Ph.	M	Recurrentpolyposis/allergic rhinitis/aspirin intolerance/:metabisulfite intolerance
Mas. Cl	F	Revision surgery/asthma/aspirin intolerance
Corl. W.	M	Primary severe nasal polyposis: no asthma: operated in 2013/REAHs
Tri. Fr.	M	Ethmoidectomy 10 y ago/nasal polyposis/ REAHs
V. Mo	F	Nasal polyposis /asthma/previous FESS/REAHs X2

Table 2.
Cohort of patients with REAH-like lesions.

All the patients suffer from a nasal polyposis. In two cases it was a massive primary polyposis. The other patients have a nasal polyposis operated in the past. The REAHs were diagnosed at the revision surgery.

Eight patients have concomitant asthma. Two patients have aspirin intolerance.

Two patients have allergic rhinitis.

Chronic inflammation plays a role in the development of REAHs in this clinical pattern.

5.2.2 Nasal endoscopy

REAHs are located in the olfactory cleft. Their macroscopic aspect is different than usual nasal polyps extruding from the ethmoid sinus. They are more fleshy and firm. There is no necrosis.

As the following pictures show, it is extremely difficult to differentiate with the fibroscopy REAHs and inflammatory polyps in case of recurrent nasal polyposis. The histologic examination of the surgical specimens is mandatory for this differentiation (**Figure 19**).

5.2.3 Imaging

CT imaging findings are described in only a limited number of studies [1, 4, 5, 14]. Lima et al. [5], Hawley et al. [4], and Lee et al. (51 cases) [14] conclude that REAHs cause widening of the olfactory cleft more than 10 mm but generally do not cause bone erosion.

All the paranasal sinus cavities can be opaque as illustrated by the following pictures (**Figures 20–22**):

Some patients have a long-standing disease; REAHs develop after the surgery with time. Some of them are attached to the anterior and superior portion of the

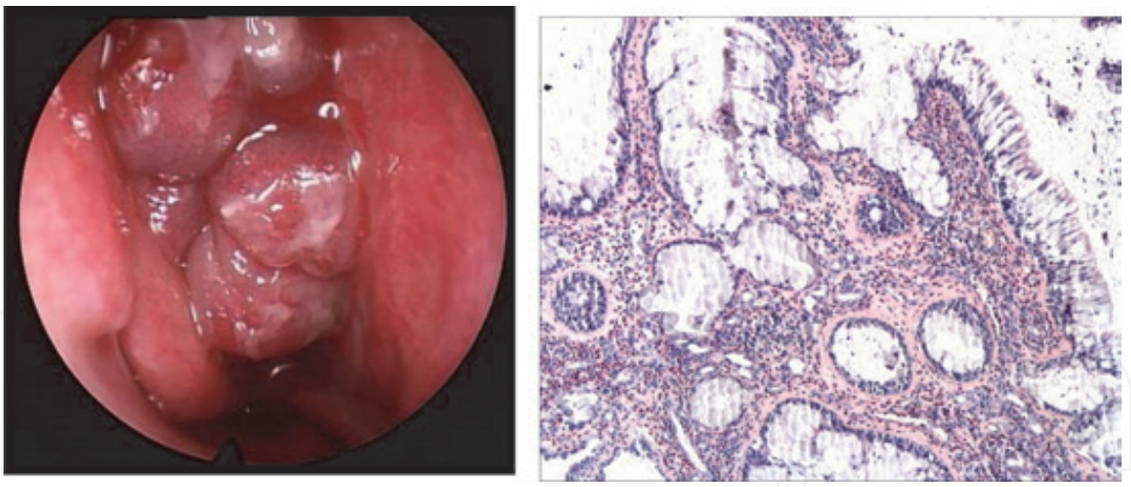


Figure 19.
Comparison of the nasal endoscopy and the histological pattern. The inflammation in the stroma is much more important than in pure REAHs.

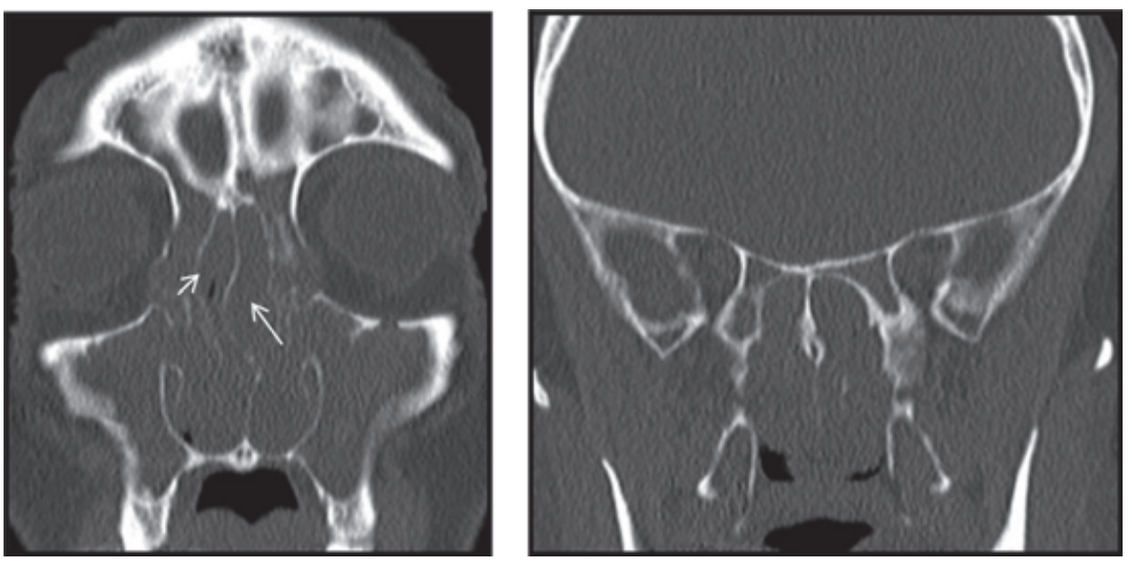


Figure 20.
CT showing a severe nasal polyposis; widening of the olfactory cleft raises suspicion of REAHs.

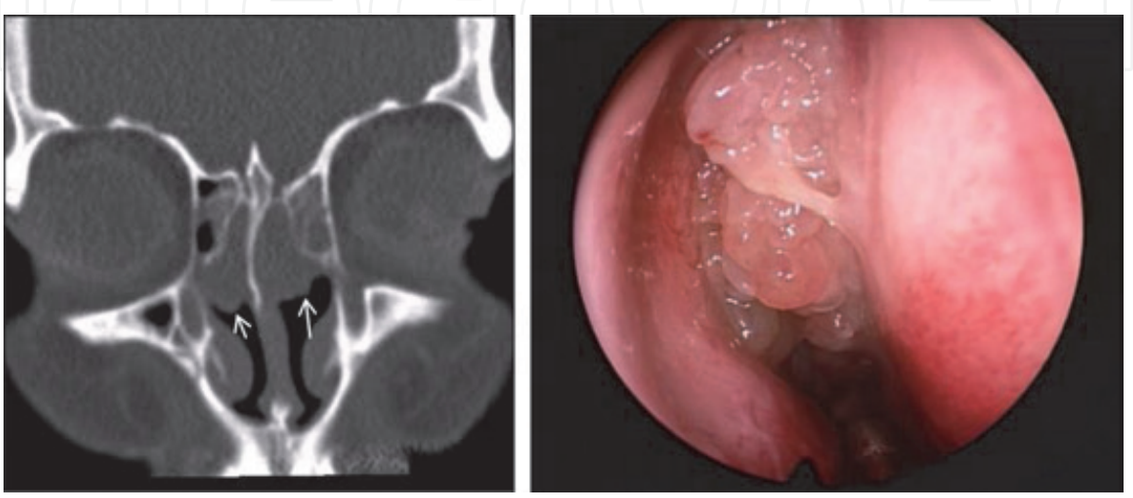


Figure 21.
Patient with REAHs in the olfactory cleft. She had a standard ethmoidectomy for nasal polyposis. We observe REAHs in the olfactory cleft. Correlation between CT scan and nasal endoscopy.

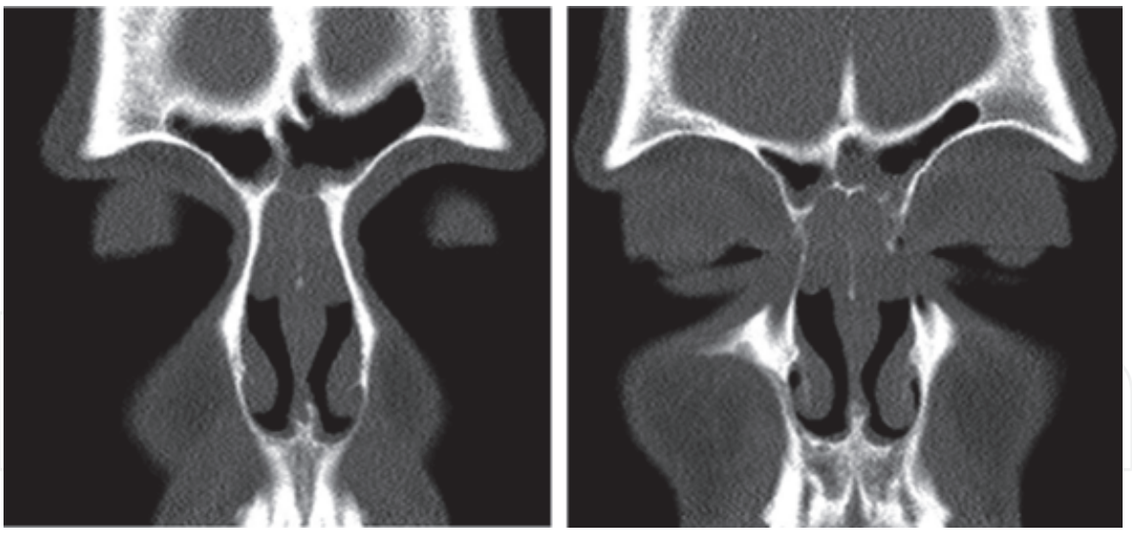


Figure 22.
Typical CT scan showing the opacity of both olfactory clefts caused by REAHs.

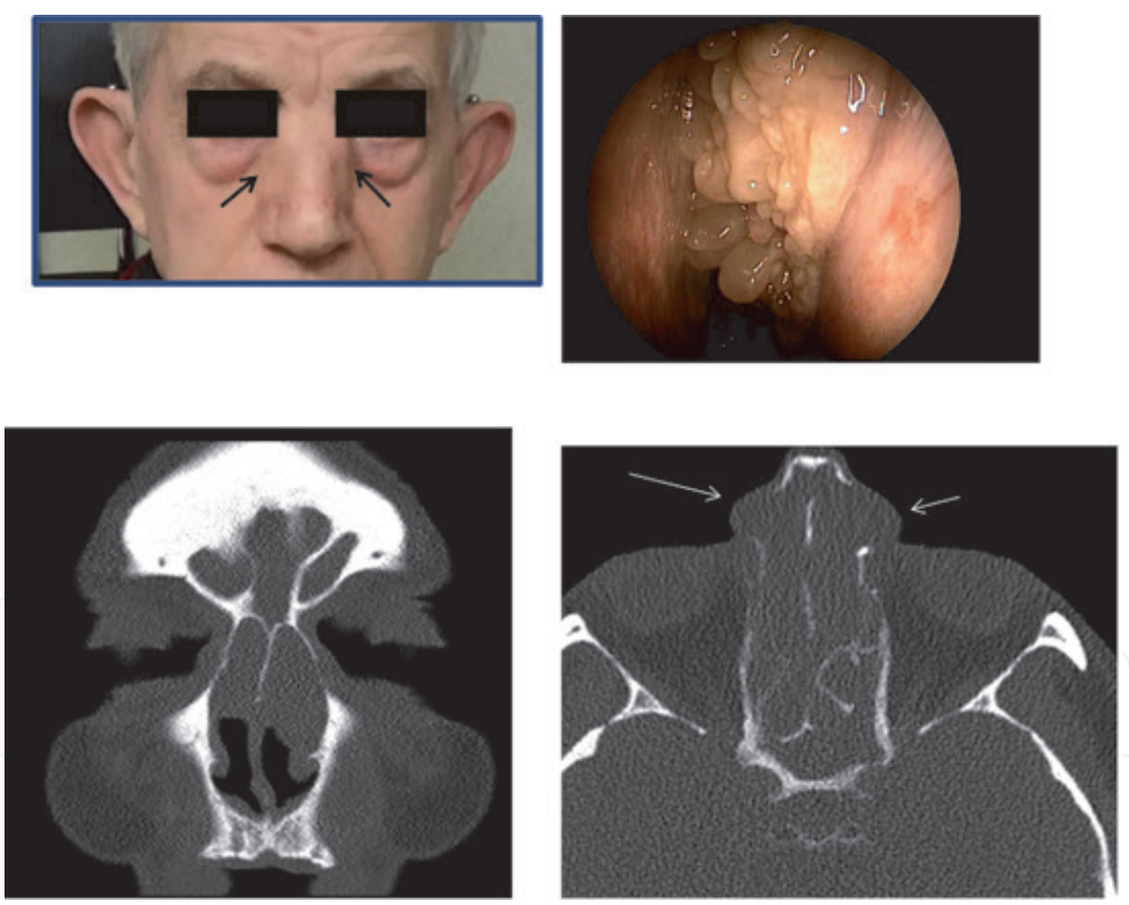


Figure 23.
Same patient with thinning and erosion of the nasal bones (arrows) and opacity of both frontal sinuses.

nasal septum and cause blockage of the frontal sinus pathway or even thinning and erosion of the nasal bones.

Figure 23 show such an exceptional evolution.

MRI can be of some help to rule out other lesions such as encephalocele, olfactory neuroblastoma, or glioma.

The management of REAHs associated with nasal polyposis must be discussed case by case.

A complete sphenoethmoidectomy is usually necessary to manage the recurrent nasal polyposis.

For the REAHs, debulking or better exenteration of the olfactory cleft must be considered. But we know that it can be tricky and risky for the skull base with a risk of CSF leak if the surgery is too aggressive. Resection of the REAHs is usually more bloody than during a polypectomy.

In the case of frontal opacity caused by REAHs attached to the anterior and superior septa, a Draf III procedure must be considered.

After surgery medical treatment of the nasal polyposis and asthma remains absolutely necessary to prevent or delay as much as possible recurrences.

6. Conclusion

REAHs and REAH-like lesions are relatively new clinical entities. Despite numerous publications they are still underdiagnosed. These lesions are located in the olfactory clefts. They can be isolated or in association with nasal polyposis typically in the case of recurrence after FESS.

The clinicians and pathologists must know these lesions. They are usually benign, but in some cases they are associated to frontal sinus blockage and widening of the nasal vault; loss of smell is common. The differential diagnosis includes diseases with more severe morbidities such as inverted papilloma, seromucinous hamartomas, and low-grade non-intestinal adenocarcinoma.

Histological examination of all the surgical specimens is necessary.

The treatment is dictated by the disease.

The extent of the surgery depends on the type and size of the REAHs and the associated disease.

It consists of a limited polypectomy or a complete exenteration of the olfactory cleft associated or not to a full house ethmoidectomy and even a Draf III procedure.

Author details

Ph. Eloy[1]*, C. Fervaille[2] and M.C. Nollevaux[2]

1 ENT Department, CHU UCL Namur, Site of Godinne, Yvoir, Belgium

2 Department of Pathology, CHU UCL Namur, Site of Godinne, Yvoir, Belgium

*Address all correspondence to: philippe.eloy@uclouvain.be

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References

[1] VandenBossche S, De Vos G, Lemmerling MA. Typical but underdiagnosed nasal cavity Mass. Journal of the Belgian Society of Radiology. 2018;**102**(1):35

[2] Wenig BM, Heffner DK. Respiratory epithelial adenomatoid hamartomas of the sinonasal tract and nasopharynx: A clinicopathologic study of 31 cases. Annals of Otology, Rhinology, and Laryngology. 1995;**104**(8):639-645

[3] Nguyen DT, Gauchotte G, Arous F, Vignaud J-M, Jankowski R. Respiratory epithelial adenomatoid hamartoma of the nose: An updated review. American Journal of Rhinology & Allergy. 2014; **28**(5):187-192

[4] Hawley KA, Ahmed M, Sindwani R. CT findings of sinonasal respiratory epithelial adenomatoid hamartoma: A closer look at the olfactory clefts. American Journal of Neuroradiology. 2013;**34**(5):1086-1090

[5] Lima NB, Jankowski R, Georgel T, Grignon B, Guillemin F, Vignaud J-M. Respiratory adenomatoid hamartoma must be suspected on CT-scan enlargement of the olfactory clefts. Rhinology. 2006;**44**(4):264-269

[6] Bullock MJ. Low-grade epithelial proliferations of the sinonasal tract. Head and Neck Pathology. 2016;**10**(1): 47-59

[7] Flavin R, Russell J, Phelan E, McDermott MB. Chondroosseous respiratory epithelial adenomatoid hamartoma of the nasal cavity: A case report. International Journal of Pediatric Otorhinolaryngology. 2005;**69**:87-91

[8] Roffman E, Baredes S, Mirani N. Respiratory epithelial adenomatoid hamartomas and chondro-osseous respiratory epithelial adenomatoid hamartomas of the sinonasal tract: A case series and literature review. American Journal of Rhinology. 2006; **20**:596-590

[9] Daniel A, Wong E, Ho J, Singh N. Chondro-osseous respiratory epithelial adenomatoid hamartoma (COREAH): Case report and literature review. Case Reports in Otolaryngology. 2019;**2019**. Article ID 5247091. 4p

[10] Ozolek JA, Barnes EL, Hunt JL. Basal/myoepithelial cells in chronic sinusitis, respiratory epithelial adenomatoid hamartoma, inverted papilloma, and intestinal-type and nonintestinal-type sinonasal adenocarcinoma: An immunohistochemical study. Archives of Pathology & Laboratory Medicine. 2007;**131**:530-537

[11] Ozolek JA, Barnes EL, Hunt JL. Basal/myoepithelial cells in chronic sinusitis, respiratory epithelial adenomatoidhamartoma, inverted papilloma, and intestinal-type and nonintestinal-type sinonasal adenocarcinoma: An immunohistochemical study. Archives of Pathology & Laboratory Medicine. 2007;**131**:530-537

[12] Baillie EE, Batsakis JG. Glandular (seromucinous) hamartoma of the nasopharynx. Oral Surgery, Oral Medicine, and Oral Pathology. 1974;**38**: 760-762

[13] Braun J-J, Riehm S, Averous G, Billing A, Veillon F. MRI in respiratory epithelial adenomatoidhamartoma of nasal cavities. Journal of Neuroradiology. 2013;**40**(3):216-219

[14] Lee JT, Garg R, Brunworth J, Keschner DB, Thompson LDR. Sinonasal respiratory epithelial adenomatoid hamartomas: Series of 51 cases and literature review. American Journal of Rhinology & Allergy. 2013; **27**(4):322-328