

EDITORIAL

Endoscopic appearance of frequently encountered anatomical abnormalities in nasal endoscopic examination

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The nasal endoscopic examination, when performed rigorously for diagnostic purposes, can provide otorhinolaryngologist with valuable information in his attempt to establish an accurate etiological diagnosis and appropriately address the respective pathological entity therapeutically. I will briefly present the most suggestive anatomical abnormalities identified and their pathological significance.

Deviation of the nasal septum

Significant nasal septal deviations not only cause nasal obstruction but can also lead to secondary involvement of the paranasal sinuses¹⁻³. This is particularly true when a septal deviation forcibly displaces the middle turbinate laterally, narrowing the entrance to the middle meatus. Septal spurs can be a cause of severe headaches, especially when they are in close contact with the turbinates or other areas of the lateral wall of the nasal cavities².

However, there is also the opposite situation when large septal deviations and spurs are asymptomatic and do not result in any sinus pathology.

Anatomical variants of the middle meatus

Anatomical variants of the middle turbinate

1. Concha bullosa

The pneumatization of the middle turbinate is known as concha bullosa (Figure 1). It usually occurs bilaterally, although the degree of pneumatization varies from patient to patient and even within the same individual. Pneumatization

can be minimal, observable only on a cranio-facial CT scan, or, on the contrary, it can be so significant that it occupies the middle meatus and even partially the nasal cavities^{2,4,5}.

In most cases, a concha bullosa contains a single air cell, but sometimes there may be two or even three cells. The interior of a concha bullosa can be affected by any pathological process that also affects the paranasal sinuses. There have been cases when, upon opening a concha bullosa, we discovered mucosal edema, polyps, retention of mucous secretion, mucocele, and in two cases, even a pyocele⁶. A solitary concha bullosa, as the only modification of the middle meatus, is not necessarily pathological.

However, small pneumatizations associated with other abnormalities of the middle meatus (e.g., uncinata process, agger nasi cell, ethmoidal bulla) can cause a significant narrowing of the ostial transitional areas, triggering secondary etiopathogenic mechanisms^{1,2}. Endoscopically, a concha bullosa appears as hypertrophy or “widening” of the anterior part or body of the middle turbinate, which may come into contact with the nasal septum or even the lateral wall, partially or totally obliterating the middle meatus. It should be emphasized that the examination with angled optics (30°, 70°) can be misleading, as the large angles of the optics tend to render objects smaller than they really are.

2. Hypertrophied and anteriorly expanded middle turbinate

The hypertrophy of the anterior third of the

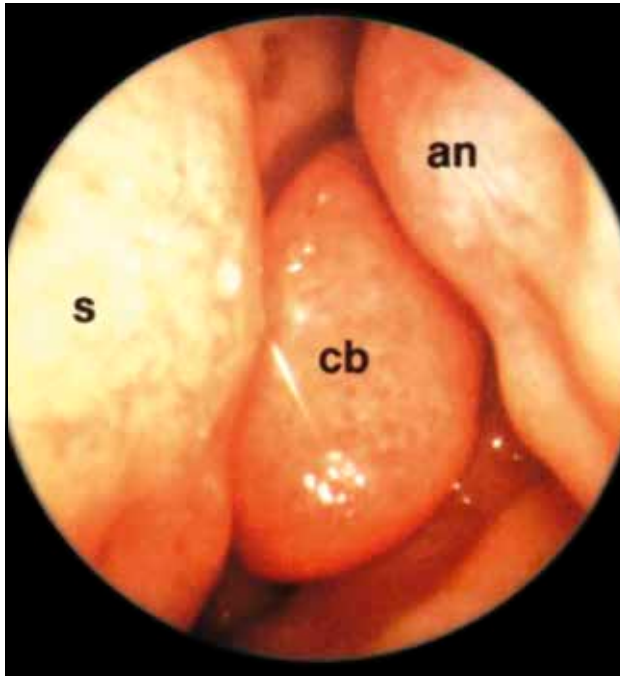


Figure 1. Concha bullosa (cb) associated with excessive pneumatization of the agger nasi cell (an); s - nasal septum.

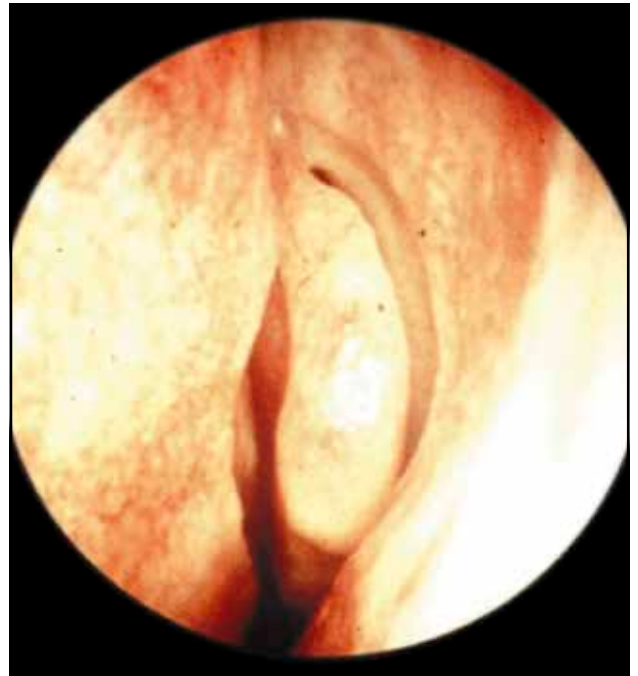


Figure 2. Paradoxically curved middle turbinate.

middle turbinate is determined both by mucosal hyperplasia and by abnormalities of the turbinate's bony lamellae. The vertical lamella of the middle turbinate can have various curvatures⁷. The anterior third of the middle turbinate may be medially convex, while its posterior two-thirds may be paradoxically curved.

It should be remembered that during surgery, the shape and contour of the middle turbinate anterior third are not important topographic landmarks.

3. Paradoxically curved middle turbinate

This situation occurs when the middle turbinate is curved (Figure 2) laterally, with the concavity facing the nasal septum and the convexity towards the lateral wall of the nasal fossa. It usually occurs bilaterally. As with other middle turbinate anomalies, if it is unique, this anomaly is not an etiological factor for the development of sinusitis. However, if the curvature is extreme and blocks the middle meatus or is associated with other middle meatus anomalies, it becomes an important etiopathogenic factor¹⁻³. Severely curved turbinates pose significant challenges during the surgical approach to the ethmoid cells.

Uncinate process anomalies

1. Medially curved uncinat process

The most important pathological variant of the uncinat process is its medial curvature (Figure 3). This may involve the entire turbinate or only certain portions of it. The curvature can be

so pronounced that the free edge of the uncinat process comes into contact with the lateral surface of the middle turbinate. Sometimes, it is also pushed anteriorly, creating the initial impression of a duplicated middle turbinate.

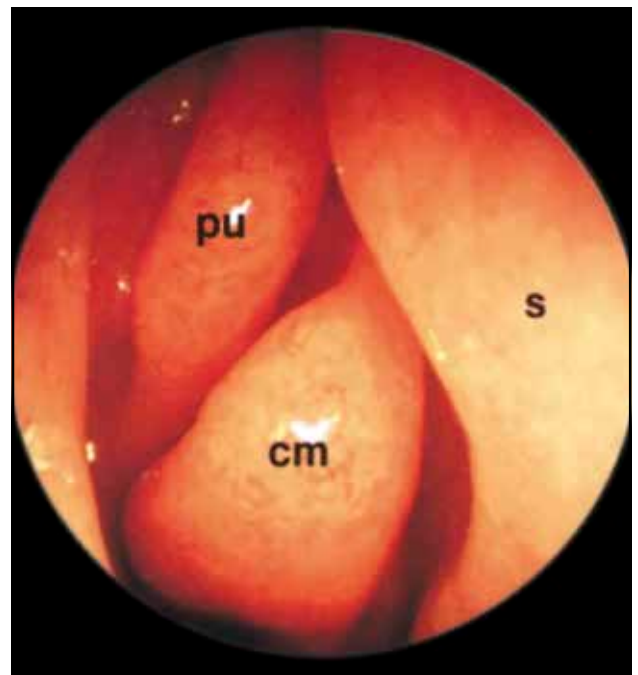


Figure 3. Medially curved uncinat process (pu), pneumatized middle turbinate (cm), s - nasal septum.

A marked medial curvature of the uncinate process associated with an extensive contact area with the middle turbinate represents one of the most common pathogenic features in patients with recurrent rhinosinusitis¹⁻³.

2. Laterally curved uncinate process

The uncinate process may be curved laterally along its entire length or only partially. This lateral displacement of the uncinate process can narrow the infundibulum, especially when the uncinate process inserts laterally, away from the middle turbinate.

3. Elongated and hypertrophied uncinate process

The uncinate process can extend posteriorly, thus pushing its posterior free edge onto the ethmoidal bulla. Thus, the semilunar hiatus is significantly narrowed. The same result is achieved by hypertrophy of the uncinate process, covering the bulla posteromedially.

When combined with other middle meatus abnormalities (e.g., concha bullosa, excessively pneumatized bulla), quasi-total blockage of the ostial transitional areas occurs^{1-3,6}.

4. Secondary changes of the uncinate process

Since the uncinate process forms the medial bony wall of the ethmoidal infundibulum, any pathological process affecting its mucosa or bony structure is an important indicator of an inflammatory process in the infundibulum.

Changes in the uncinate process mucosa suggest alterations in the infundibulum, frontal recess, or adjacent areas of the anterior ethmoidal cells. Sometimes, these changes consist of mucosal edema, polyps, and in some cases, the entire medial wall of the infundibulum bulges medially⁶.

5. Pneumatization of the uncinate process

In rare cases, the uncinate process can be pneumatized. When pneumatized, it occupies the free areas of the infundibulum and creates mucosal contrast zones.

Accessory ostium of the maxillary sinus

The natural ostium of the maxillary sinus cannot be directly visualized from the middle meatus, as it is “hidden” behind the uncinate process, in the posterior 1/3 depth of the ethmoidal infundibulum.

In more than 25% of cases, the endoscopic examination reveals accessory ostia located anteriorly or posteriorly to the natural one^{1,8,9}. In some cases, secretions may be seen entering the maxillary sinus through the accessory ostium. These are then drained through the natural ostium into the ethmoidal infundibulum, from where they can once again enter the accessory ostium, thus creating a mucus recircu-

lation phenomenon.

Anomalies of the ethmoidal bulla

1. Hypertrophy of the ethmoidal bulla

The pneumatization of the ethmoidal bulla is variable, ranging from its absence to massive pneumatization that brings the bulla anteriorly below the middle turbinate. Excessive pneumatization brings the bulla into close contact with the posterior edge of the unciform, thus blocking the semilunar hiatus. When excessive pneumatization occurs, the bulla may obliterate the middle meatus, making the initial endoscopic diagnosis difficult. In other cases, the pneumatization of the bulla creates a large contact surface between the medial surface of the bulla and the lateral surface of the middle turbinate. In his experience, Stammberger observed that this is the site where inflammatory polyposis originates¹⁰.

Due to its central position and multiple relationships with key anatomical areas, the ethmoidal bulla is frequently involved in the etiopathogenesis of ethmoidal cells diseases. An interesting observation noted by Hawke is that a large and “diseased” bulla may be responsible for chronic posterior rhinorrhea¹⁰.

Ethmoidal infundibulum involvement

There are four important endoscopic signs that suggest ethmoid involvement during nasal endoscopic examination¹⁻³ (Figure 4), namely:



Figure 4. Obstruction of the infundibulum with purulent secretions and edema.

1. Evident pathological changes in the infundibulum or semilunar hiatus.
2. Pathological secretions draining from the infundibulum, via the semilunar hiatus.
3. Identification of anatomical variants capable of causing infundibular obstruction.
4. Observation of mucosal changes on the medial surface of the uncinate process.

Several pathological aspects can be noted, including mucosal edema in the infundibulum, isolated or clustered polyps, changes in the mucosa of the peri-infundibular areas, granulomatous-type changes, secretions, and others.

Frontal recess involvement

The frontal recess is frequently affected by pathological processes originating in the areas adjacent to the ostiomeatal complex.

Since the frontal recess serves as an ethmoidal “antechamber” to the frontal sinus, infectious processes propagate from the nasal cavity through this antechamber. Consequently, frontal sinus pathology cannot occur without involvement of the frontal recess and, therefore, the ethmoid cells. The endoscopic aspects of a frontal sinus pathology are unsuggestive; sometimes, only pathological secretions in the middle meatus serve as a clue.

Occasionally, small polyps can be identified in the area of the anterior insertion of the middle turbinate. Bulging of the superior portion of the uncinate process and mucosal edema in the anterior part of the area of the middle turbinate insertion are signs of frontal sinus involvement¹⁻³.

Involvement of the agger nasi cell

A pneumatized agger nasi cell is a variant that appears as a bulging of the lateral nasal wall just anterior to the insertion of the middle turbinate.

When pneumatization is excessive, the insertion of the middle turbinate is pushed superomedially. Any pathological process affecting the agger nasi cell can extend to the lacrimal sac (via the lamina papyracea).

Significant for the inflammatory condition of the agger nasi cell is the endoscopic appearance of a marked bulging of the lateral nasal wall, anterior to and sometimes superior to the insertion of the middle turbinate^{2,11}.

Involvement of the Haller cells

The Haller cells are another anatomical variant that plays an important role in maxillary sinusitis. These are the cells that develop in the floor of the orbit (the roof of the maxillary

sinus), adjacent to the natural ostium of the maxillary sinus. When excessively pneumatized, they can narrow the posterior part of the infundibulum and the ostium of the maxillary sinus from the upper part.

Secondary changes, such as a slight bulging of the lateral wall, inferior to the ethmoidal bulla, and narrowing of the posterior part of the semilunar hiatus suggest the presence of the Haller cell¹². Confirmation is made through cranio-facial CT examination.

Haller cells cannot be visualized through nasal endoscopy but only during sinusoscopy of the ipsilateral maxillary sinus.

Posterior ethmoidal cells involvement

Isolated involvement of the posterior ethmoidal cells is rare. Typically, it is part of an anterior-posterior ethmoid pathology.

On endoscopic examination, the presence of pathological secretions from the superior meatus or from the sphenoidal recess may be the only sign of posterior ethmoid involvement^{2,3}. Additionally, inflamed or polypoid mucosa protruding from the superior meatus may be observed.

In some cases of severe nasosinus polyposis, edematous polyps are noted appearing between the middle turbinate and the nasal septum.

Sphenoid sinus involvement

The sphenoid sinus is involved in 16% of patients with chronic sinusitis^{2,3}. Isolated involvement of the sphenoid is undoubtedly more common than that of the posterior ethmoid.

The major endoscopic sign is the presence of pathological secretions from the sphenoidal recess towards the nasopharynx.

The examination of the sphenoidal recess is performed using a 2.7 mm endoscope with 30° or 70° optics. Even when the sphenoid ostium cannot be visualized, the presence of secretions in the recess area, along with hyperemia and edema of its mucosa, are endoscopic signs suggesting sphenoiditis.

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