Nasal polyposis in children

Dr T Balasubramanian

Abstract:

This article discusses various causative factors of nasal polyposis in children. It is a review of literature on this subject, supplemented by the author's personal experience. Even though nasal polyposis is rather uncommon in children, when present they should be thoroughly investigated to rule out other sinister lesions. Imaging has a vital role to play in diagnosis of these patients. Antrochoanal polyp is currently the commonest nasal polyp seen in children.

Introduction:

Studies reveal that eventhough nasal obstruction and discharge are common in children, nasal polyposis is rather uncommon. Majority of nasal polypi in children are caused by infection and inflammation of nasal / sinus mucosal lining. Among the types of nasal polyposis seen in children about 1/3 of these patients have antrochoanal polyp.

Causes of nasal polyposis in children:

1. Antrochoanal polyp
2. Inflammatory polyp
3. Polyp due to cystic fibrosis

Antrochoanal polyp:

Synonyms: Antrochoanal polyp, Killian's polyp, Nasal polyp.

Palfyn described the first case of antrochoanal polyp in 1753. Since he found the polyp filling the nasopharynx and extending below uvula he thought that it could have arisen from the choana. Killian in 1906 demonstrated that this polyp arose from maxillary sinus antrum. According to Stammberger 70% of antrochoanal polyp exited out of the maxillary sinus antrum via the accessory ostium.

Definition:

Antrochoanal polyp is a benign solitary polypoidal lesion arising from the maxillary sinus antrum causing opacification and enlargement of antrum radiologically without any evidence of bone destruction. It exits the antrum through the accessory ostium reaches the nasal cavity, expands posteriorly to exit through the choana into the post nasal space.

Incidence:
It commonly affects young children and adolescents.

Etiopathogenesis:

Antrochoanal polyp is said to originate in the maxillary antrum due to inflammation. This
condition has been commonly documented only in non atopic persons. Its etiology is still unknown. Various theories have been proposed to account for the pathogenesis of this disorder:

**Proetz theory:**
Proetz suggested that this disease could be due to faulty development of the maxillary sinus ostium, since it was always been found to be large in these patients. Hypertrophic mucosa of maxillary antrum sprouts out through this enlarged maxillary sinus ostium to get into the nasal cavity. The growth of the polyp is due to impediment to the venous return from the polyp. This impediment occur at the level of the maxillary sinus ostium. This venous stasis increases the oedema of the polypoid mucosa thereby increasing its size.

**Bernoulli's phenomenon:** Pressure drop next to a constriction causes a suction effect pulling the sinus mucosa into the nose. According to this theory there is a pressure drop at the level of infundibular area causing a relative negative pressure. This negative pressure is sufficient to cause prolapse of maxillary antral mucosa into the nasal cavity. This prolapsed mucosal lining begins to enlarge in size due to oedematous reaction causing formation of polypoidal tissue.

**Mucopolysaccharide changes:** Jakson postulated that changes in mucopolysaccharides of the ground substance could cause nasal polyp. These changes lead to water retention within the submucosal compartment could lead to polyp formation.

**Mill's theory:**
Mills postulated that antrochoanal polyp could be maxillary mucoceles which could be caused due to obstruction of mucinous glands.

**Ewing's theory:** Ewings suggested that an anomaly which could occur during maxillary sinus development could leave a mucosal fold close to the ostium. This fold could later be aspirated into the sinus cavity due to the effects of inspired air causing the development of antrochoanal polyp.

**Vasomotor imbalance:** This theory attributes polyp formation due to autonomic imbalance.

**Infections:** Recurrent nasal infections have also been postulated as the cause for nasal polyp. This theory suggests that acinous mucous glands within the maxillary sinus cavity gets blocked due to infection / inflammation involving the mucous lining of the sinus cavity. This leads to the formation of a cystic lesion within the maxillary sinus cavity. This cyst gradually enlarges to occupy the whole of the maxillary sinus cavity. It exits the sinus cavity by enlarging the accessory ostium and enters the nasal cavity. Usually these cysts arise from the antero inferior / medial wall of maxillary antrum. Macroscopically the portion of A/C polyp within the maxillary antrum is cystic in nature, while the component that has prolapsed via the accessory ostium is solid in nature.

Possible reasons for posterior migration of antrochoanal polyp:

Classically antrochoanal polyp presents posteriorly. The polyp could be clearly seen occluding the post nasal space. Possible reasons for this posterior presentation include:

1. The accessory ostium through which the polyp gets out of the maxillary antrum is present posteriorly.
2. The inspiratory air current is more powerful than the expiratory air current thereby pushes the polyp posteriorly.
3. The natural slope of the nasal cavity is directed posteriorly, hence the polyp
always slips posteriorly.
4. The cilia of the ciliated columnar epithelial cells lining the nasal cavity always beats anteroposteriorly pushing the polyp behind.

Histology:
Shows respiratory epithelium over normal basement membrane. The interstitial layer is grossly oedematous, with no eosinophils. The interstitial layer contains other inflammatory cells.

Clinical features:
This disorder is commonly unilateral. Bilateral antrochoanal polyp is very rare condition. Only a few handful of such cases have been reported in literature so far.

1. Unilateral nasal obstruction
2. Unilateral nasal discharge
3. Headache (mostly unilateral)
4. Epistaxis
5. Sleep apnoea
6. Rhinolalia clausa due to presence of polyp in the post nasal space
7. Difficulty in swallowing if the polyp extends into the oropharynx

Figure showing antrochoanal polyp exiting maxillary antrum via the accessory ostium
Anterior rhinoscopy may show the polyp as glistening polypoidal structures. They will be insensitive to touch. This feature helps to differentiate it from a hypertrophied nasal turbinate. Postnasal examination will show the polyp if extending posteriorly at the level of choana. If it fills up the nasopharynx it will be visible there.

X-ray paranasal sinuses will show a hazy mazillary antrum.

CT scan of paranasal sinuses is diagnostic. It will show the polyp filling the maxillary antrum and exiting out through the accessory ostium into the nasal cavity.

CT scan nose and paranasal sinuses axial cut showing antrochoanal polyp

The antrochoanal polyp is dumb bell shaped with three components i.e. antral, nasal and nasopharyngeal.

Treatment:
This is a surgical problem. Formerly it was treated by avulsion of the polyp transnasally. This method led to recurrences. A caldwell luc approach was preferred in patients with recurrences. In caldwell luc procedure in addition to the polypectomy, the maxillary antrum is entered via the canine fossa and the antral component is completely excised.
Endoscopic approach: With the advent of nasal endoscope this approach is the preferred one. Using an endoscope it is always easy to completely remove the polypoid tissue. The uncinate process must also be completely excised. Endoscopic approach has the advantage of a complete surgical excision with negligible recurrence rates.

Picture showing the choanal component of antrochoanal polyp

Differences between antrochoanal polyp / Ethmoidal polyp

<table>
<thead>
<tr>
<th>Antrochoanal polyp</th>
<th>Ethmoidal polyp</th>
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<tbody>
<tr>
<td><strong>Solitary</strong></td>
<td><strong>Multiple</strong></td>
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<tr>
<td>Arises from maxillary antrum</td>
<td>Arises from ethmoidal air cells</td>
</tr>
<tr>
<td>Has three components</td>
<td>Has only one component</td>
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<tr>
<td>Infection plays a role in its pathogenesis</td>
<td>Allergy is supposed to play a role</td>
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<tr>
<td>Common in adolescents</td>
<td>Common in adults / elderly</td>
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Recent advances:
Current research involving Nitric oxide has thrown light into the possible etiopathogenic factors involved in the genesis of antrochonal polyp. Nitric oxide have been shown to play a major role in nonspecific immune reactions and inflammation in a variety of tissues. Endogenous nitric oxide is synthesized from L-arginine by the effect of nitric oxide synthase. This all important nitric oxide synthase exists in three forms:

1. Endothelial nitric oxide synthase
2. Neuronal nitric oxide synthase
3. Inducible nitric oxide synthase

Out of these three types the Inducible nitric oxide synthase has been detected not only in epithelium but also in macrophages, fibroblasts, neutrophils, endothelium and vascular smooth muscle. Studies have revealed that antrochoanal polyp tissue contained more nitric oxide than normal tissues. Increased nitric oxide production could be from epithelial / inflammatory cells. Among inflammatory cells eosinophils play an important role in production of nitric oxide. Studies have also revealed that Inducible nitric oxide synthase play an important role in the pathogenesis of antrochonal polyp.

Sphenochoanal polyp is another rare unilateral nasal polyp that presents posteriorly occluding the choana. In fact this condition should be differentiated from antrochoanal polyp.

Cystic fibrosis:

This is another condition that can cause nasal polyposis in children. These children present with:

1. Pneumonia
2. Pancreatic insufficiency
3. Meconium ileus
4. Rectal prolapse
5. Biliary cirrhosis & portal hypertension

This genetic disorder is known to affect approximately 1 in 2500 live births. These patients have abnormal chloride transport, which is actually caused by defective chloride channel conductance which is actually regulated by cyclic AMP. This disorder is caused by mutation involving chromosome 7 which codes for chloride channel protein.

Attempts to seek evidence in adult patients with nasal polyposis for the presence of cystic fibrosis has not been fruitful.

The incidence of nasal polyposis in patients with cystic fibrosis ranges between 15 – 40%. This is a high variation considering the frequency of nasal polyposis in children. Patients with cystic fibrosis invariably develop nasal polyposis after their 5th year or before they reach 20 years of age.

Studies performed by Toss et al have not demonstrated any morphological / histological differences between nasal polypoidal tissue between cystic fibrosis and non cystic fibrosis groups. This actually points towards the common underlying common pathogenesis.
Role of sweat test in the diagnosis of cystic fibrosis:

This is actually the gold standard test in the diagnosis of cystic fibrosis.

Sweat test is usually done in the forearm. It can also be done on the thighs.

Stimulation of sweat production:

This is actually the first step in sweat test. Electrodes containing pilocarpine is placed over the skin. Small current is passed through the electrodes so that pilocarpine will enter skin and stimulate secretion of sweat. This current is actually not painful but causes a tingling sensation. After about 10 minutes the electrodes are removed and a filter paper patch known as sweat patch is used to collect sweat. Chloride levels in sweat of patients with cystic fibrosis is supposed to be very high.

Seat chloride ranges:

Less than 30 = normal
30-59 = Borderline
60 and above is indicative of cystic fibrosis

Screening for the presence of AF508 gene could serve as a pointer for diagnosing cystic fibrosis. Serum levels of immunoreactive trypsinogen has been found to be elevated in infants with cystic fibrosis.

Characteristic feature of sinusitis in these patients is the range of microbes that have been isolated by culturing the secretions. These organisms include: Psuedomonas aeruginosa, and staphylococcus aureus.

Major nasal symptoms seen in these patients include:

1. Nasal block
2. Mucopurulent secretions
3. Head ache

Medical management has a very limited role to play in the managment of chronic sinusitis in patients with cystic fibrosis. Only role played by antibiotics in these children is to limit the damage due to repeated lower airway infections. Colonization of lower respiratory tract by pseudomonas is commonly seen in these patients. Nasal douching if preformed repeatedly will help in minimizing these colonies being formed in the lower airway.

Encephaloceles / Meningoceles:

These are congenital neural tube defects presenting as polypoidal masses inside the nasal cavity. It is imperative to differentiate these lesions from nasal polypi. These lesions can be identified by the presence of cough reflex. These masses change in size according to the phases of respiration.

High resolution CT scan images and MRI images helps in the diagnosis of this condition. After excision of these masses the defect in the skull base should be closed using a three layer graft. This will help in avoiding troublesome CSF leaks which are common in these patients following surgery.
Nasal polyposis associated with Primary ciliary dyskinesia:

This condition when associated with bronchiectasis and situs inversus totalis is known as kartagener's syndrome. These patients have unrelenting nasal discharge without any symptom free interval. Since nasal mucosal ciliary beat is suboptimal in these patients saccharin clearance test will help in diagnosing this condition.

Saccharin test:
This test is performed by placing a 1 mm diameter saccharine tablet just behind the anterior end of inferior tubinate / corresponding area of nasal septum. Patient is asked to sit quietly leaning forward. Patient is instructed not to sniff or attempt to clear the nose. The time taken for perception of saccharine taste after placement in the nasal cavity is recorded. Saccharine is dissolved in the mucous layer and is transported posteriorly to the nasopharynx by the nasal mucosal ciliary clearance mechanism. Average saccharine clearance time is 7 – 15 minutes. In patients with primary ciliary dyskinesia the clearance time could well be in excess of 1 hour.

FESS is useless in these patients, because ciliary mechanism is not going to become normal following surgery.

Allergic fungal sinusitis:

This is a non invasive disorder. Commonly caused by aspergillus infection. It is seen in immunocompetent individuals. These patients present with unilateral nasal polyposis with presence of greenish white crusts.

References:

5. Stammberger and Hawke 1993 Essentials of endoscopic sinus surgery Mosby year book