An approach to the patient with a post-nasal drip and rhinosinusitis

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Abstract **SA Fam Pract 2009;51(5):376-380**

Post-nasal drip (PND) can be a bothersome symptom and one of the main reasons for patients visiting a general practitioner (GP), pulmonologist or ear, nose and throat (ENT) surgeon. It can be either a symptom, sometimes not appreciated by the examining practitioner, or an important clinical sign suggesting rhinosinusitis. This article aims to explain the aetiological factors and initial work-up of the patient with a PND and rhinosinusitis and suggest a treatment paradigm for practitioners.

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Introduction

Approximately one litre of mucus is produced by the nasal mucosa and tracheobronchial tree every day. Normal mucociliary function ensures that mucus produced by the paranasal sinuses and nasal cavity is transported into the nasopharynx and is eventually swallowed. For patients to become aware of a post-nasal drip (PND), the mucus characteristics need to be altered. Excess mucus production, a change in mucus viscosity or infected mucus will make the patient aware of the presence of a PND or 'phlegm in the throat'. It is important to have a structured approach to the patient presenting with a PND, to correctly treat the underlying or associated condition and to avoid unnecessary investigations and/or surgery.

Chronic cough and nasal disease

Patients suffering from a chronic cough often first present to a pulmonologist. A chronic cough is defined as one that persists for more than eight weeks. Ninety per cent of patients with chronic cough suffer from either laryngopharyngeal reflux (LPR), bronchial asthma or post-nasal drip syndrome (PNDS), and these conditions need a systematic approach.[1] It is important to first rule out angiotensin-converting enzyme (ACE)-inhibitors and post-infectious causes before requesting unnecessary investigations and prescribing medication. The term PNDS is non-specific and should rather be replaced with rhinosinusitis if a purulent nasal drip is found to be present.[2] Rhinosinusitis is a condition that can be clinically diagnosed by using a group of symptoms. It does not require special investigations such as computed tomography or endoscopy. Medication should be directed at the cause of the PND rather than treating the symptom itself. It has been shown that rhinosinusitis with PND is frequently over-diagnosed as being the underlying cause for a chronic cough. In a recent study it was demonstrated that only a small proportion of patients with purulent rhinosinusitis with no co-morbid lung pathology suffer from chronic cough.[2]

Approach to the patient with a post-nasal drip

When considering causative factors, the following diagnosis should be considered: allergic, infectious and non-allergic non-infective rhinitis. The differential diagnosis should include nasal polyps, mechanical obstruction, tumours, granulomatous conditions and cerebrospinal fluid leaks (Table I).

Table I: Classification of rhinosinusitis

1. Allergic	Intermittent (seasonal) Persistent (perennial)
2. Infectious	Acute Chronic
3. Other	Occupational Nares (Non-allergic rhinitis with eosinophilia syndromes) Hormonal Drug-induced Irritants Food Emotional Atrophic Gastro-oesophageal reflux Idiopathic

1. Does the patient suffer from allergic rhinitis?

It is important to determine whether the patient has any symptoms suggesting rhinitis and, if present, whether this is allergic or non-allergic in nature. Rhinitis is present when the patient has two or more of the following symptoms: blockage, rhinorrhoea, itchiness and sneezing. Allergic rhinitis is diagnosed when these symptoms are due to an immunoglobulin E (IgE)-mediated inflammatory response secondary to allergen exposure. Patients with persistent (perennial) allergic rhinitis can have chronic inflammatory changes that can be misdiagnosed as chronic rhinosinusitis (CRS). It is therefore essential to correctly identify these patients to avoid unnecessary sinus surgery. When allergic rhinitis is strongly suspected based on a positive family history of atopy and the

Table II: Pharmacological treatments and their effects in allergic rhinitis

	Itching/ sneezing	Discharge	Blockage	Impaired smell
Sodium cromoglycate (nasal spray not available in SA)	+	+	+ / -	-
Oral antihistamines	+++	++	+ / -	-
Ipratropium bromide	-	+++	-	-
Topical decongestants	-	-	+++	-
Topical corticosteroids	+++	+++	++	+
Oral corticosteroids	+++	+++	+++	++
Antileukotrienes	-	++	+	+ / -

abovementioned symptom complex, empiric therapy should be instituted and directed at the main symptom (Table II).

The mainstay of treatment for allergic rhinitis remains topical corticosteroids and oral antihistamines. Fluticazone and mometasone have the lowest bioavailability of all the topical corticosteroids, but there is no difference in the efficacy when compared to the other topical corticosteroids.[3] Ipratropium bromide is an atropine-like nasal spray that can be used in patients with watery rhinorrhoea where topical corticosteroids are not effective. Allergen avoidance is important and immunotherapy is an option for selected patients with severe symptoms who fail treatment with the above measures. Immunotherapy is especially effective in patients with seasonal allergies who are allergic to a few allergens only. Those patients not responding to therapy should be further investigated for atopy by either doing skin-prick testing or specific CAP RAST testing. Skin-prick tests are quick and inexpensive, but cannot be performed on patients on antihistamines, or who suffer from severe eczema, dermagraphism or previous anaphylaxis.

2. Does the patient suffer from non-allergic rhinitis?

Non-allergic rhinitis is present if the patient suffers from two or more of the abovementioned symptoms but without an allergic aetiology. Studies show that patients are likely to have nasal disease if they suffer from more than five sneezes or nose blowing episodes a day.[4] A recent study in the United States of America showed that 34% of patients with rhinitis have a combination of allergic and non-allergic rhinitis.[5] Rhinorrhoea can be present with or without the presence of a constant PND. The following aetiologies must be excluded when a patient complains of nasal symptoms:

- **Viral rhinitis or common cold** – this is mostly self-limiting but can occasionally lead to chronic rhinosinusitis, especially if the patient has underlying anatomical abnormalities.
- **Idiopathic or vasomotor rhinitis** – triggered by irritants and changes in atmospheric conditions.
- **Occupational rhinitis** – triggered by irritant or toxic compounds at the workplace (aldehydes, aircraft fuel, solvents).
- **Hormonal rhinitis** – pregnancy rhinitis due to oestrogens causing vascular engorgement in the nasal cavity.
- **Drug-induced rhinitis** – aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs), beta-blockers, ACE-inhibitors, oral contraceptives, oxymetazoline in nasal decongestants (rebound rhinitis or rhinitis medicamentosa).
- **Other** – food-induced, emotional-induced, chemical-induced, smoking.

Treatment is aimed at avoidance of the irritant. Topical corticosteroids are preferred where an inflammatory pathogenesis is suspected. Azelastine nasal spray is an antihistamine and mast cell stabiliser and has been proven to be more effective than a placebo for patients with rhinorrhoea and/or a PND.[6] In a few patients who fail medical therapy and have severe nasal blockage secondary to inferior turbinate hypertrophy, surgery is an option. The inferior turbinates play a very important role in humidification, filtration, heating and respiration, and turbinate surgery can be harmful and cause atrophic rhinitis. Only conservative trimming with preservation of mucosa should therefore be performed to improve the nasal airway and to allow for optimal administration of topical corticosteroids.

3. Does the patient suffer from an infective rhinitis/rhinosinusitis?

The term rhinosinusitis is preferred rather than rhinitis, since the inflammation involves the mucosa of the nasal cavity and the paranasal sinuses. Rhinosinusitis can be classified as follows:[7]

- **Acute** (ARS): seven days to ≤ four weeks
- **Sub-acute:** four to twelve weeks
- **Recurrent acute:** ≥ four episodes of ARS a year
- **Chronic** (CRS): ≥ 12 weeks
- **Acute exacerbation of chronic:** Sudden worsening of CRS with return to baseline thereafter

The diagnosis of acute bacterial rhinosinusitis (ABRS) is based on symptoms/signs and can be made by a general practitioner without the need for nasal endoscopy or imaging studies. For ABRS to be diagnosed, two major factors or one major and two minor factors need to be present (Table III).

Table III: Rhinosinusitis symptoms/signs (two major or one major and two minor factors required for diagnosis)

Major symptoms	Minor symptoms
1. Facial pain/pressure	1. Headache
2. Facial congestion/fullness	2. Fever (non-acute)
3. Nasal obstruction/blockage	3. Halitosis
4. Nasal discharge/purulence/discoloured PND	4. Fatigue
5. Hyposmia/anosmia	5. Dental pain
6. Purulence on nasal examination	6. Cough
7. Fever (ARS only)	7. Ear pain/pressure/fullness

These symptoms are useful in ABRS but become more difficult to apply in CRS because of the variety of associated conditions in CRS. Rhinosinusitis is a multifactorial disease and there are many different causes for sinus ostia obstruction with resultant inflammation of the paranasal sinus mucosa. Patients with recurrent ABRS or CRS not responding to therapy should be referred to a specialist for further investigations. Viral rhinosinusitis is usually self-limiting, but a combination of factors may lead to a chronic situation developing: anatomical variants occluding the natural drainage pathway of the sinuses; permanent damage to the cilia responsible for mucociliary clearance either by medication, pollutants or infections; or the presence of other co-morbidities. ABRS usually follows on an acute viral upper respiratory tract infection. Mucosal swelling leads to obstruction of the sinus ostia (especially in predisposed individuals), resulting in a reduction in oxygen tension in the sinuses with a reduction in mucociliary transport and transudation of fluid into the paranasal sinuses. Mucostasis leads to bacterial colonisation and a

chronic situation can develop if ciliary action is not improved or the ostia remain obstructed.

When patients complain of a PND it is therefore important to diagnose rhinosinusitis correctly and to improve the nasal-sinus environment as quickly as possible. Purulent nasal secretions on anterior rhinoscopy or the presence of a PND in the oropharynx strongly support the diagnosis of rhinosinusitis. The diagnosis of ABRS should be made on clinical grounds and criteria only. Plain radiographs are not cost-effective, have poor specificity and sensitivity and have no role in the acute situation, except possibly in children where maxillary sinus washouts are considered after optimal medical treatment.[8] If the symptom complex suggests the diagnosis, empiric therapy should be commenced according to the most likely organism responsible. *Streptococcus pneumoniae* and *Haemophilus influenzae* are the most common organisms responsible for ABRS. Antibiotic therapy is usually only indicated seven to ten days after the onset of symptoms. Studies have however shown no major difference in cure rates between patients given antibiotics versus a placebo. Antibiotics do however reduce the symptom severity and reduce the recovery time.

CRS is a multifactorial disease and diagnosis and treatment can be challenging. Anterior rhinoscopy is often normal, especially in a patient who has had no previous sinus surgery. Patients often complain of a PND, but this is infrequently seen. If CRS is suspected, nasal endoscopy is useful to determine the presence of anatomical abnormalities such as a concha bullosa (aerated middle turbinate), deviated nasal septum, nasal polyps or lesions/tumours requiring further investigation. Computerised tomography (CT) scans are helpful to determine response to treatment and to decide whether surgical intervention is required after optimal medical treatment, and should only be performed after six to eight weeks after the commencement of such treatment.

The treatment of CRS with polyposis also differs from that of CRS without polyposis. If polyps are visible, empiric therapy consisting of topical and systemic corticosteroids (if no contra-indications exist) with a macrolide antibiotic can be commenced. Macrolide antibiotics have an important immunomodulatory role by down-regulating pro-inflammatory mechanisms and are the preferred antibiotics in patients with CRS.[9] Certain conditions are closely associated with nasal polyposis and patients should be assessed for asthma, cystic fibrosis (especially in children) and aspirin sensitivity. CRS is an inflammatory disease with multiple potential associated or aetiologic factors and a specific organism cannot always be identified. In patients with potential pathogenic bacteria, Staphylococcus species are the most common with Enterobacteriaceae, anaerobes, Gram-negative bacteria and fungi also showing a high prevalence.

Conclusion

A careful history should be obtained in patients complaining of a PND or 'sinusitis'. Allergic rhinitis should first be treated, causes of non-allergic rhinitis looked for and irritants avoided, rhinosinusitis diagnosed and correctly classified and managed. If a PND persists, especially with associated nasal symptoms, patients should be referred to an ear, nose and throat (ENT) surgeon for endoscopy. CT scans are only performed to assess the response to treatment, to exclude the presence of complications in ABRS and to plan endoscopic sinus surgery.

Conflict of interest

No conflict of interest exists.

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