Influenza, hay fever and sinusitis: Know the differences

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Abstract

Influenza, hay fever and sinusitis are all very different upper respiratory tract syndromes that share very similar symptoms. "Flu" is caused by the influenza virus and usually presents with headaches, myalgia, fever and body aches. Hay fever is an allergic response of the body to a trigger, more often treated by removal of the trigger. There is no place for antibiotic usage in flu or hay fever; there is no clinical evidence to suggest that using antibiotics alters the course of the disease or prevents secondary infection. Treatment is mainly symptomatic and includes many over-the-counter medicines, antivirals and herbal treatments.

On the other hand, sinusitis is a common inflammatory condition defined by persistent symptomatic inflammation of the sinonasal cavities lasting from less than four weeks to longer than three months. Either a virus or bacteria may cause sinusitis. Appropriate use of medical therapies, including antibiotics for sinusitis is necessary to optimise patient quality of life and daily functioning and minimise the risk of acute inflammatory exacerbations. Patients often present at the pharmacy with complaints of "flu". However, in many instances, symptoms may be confused with those of an allergy or sinusitis. It is important to know and understand the difference between influenza and allergies such as hay fever and sinusitis, in order to ensure correct diagnosis and offer appropriate therapeutic approaches. This article will review the clinical manifestations of influenza, allergic rhinitis (hay fever) and sinusitis, and highlight the differences between these three illnesses.

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Introduction

Allergic rhinitis – more commonly known as hay fever – sinusitis and influenza can be difficult to accurately distinguish based on symptoms presented, because they share so many common symptoms. Understanding the differences could assist in making an accurate diagnoses and choosing the best treatment. This will decrease the unnecessary consumption of medications, and increase quality of life. Complications due to acute bacterial sinusitis can be life threatening. It is for this reason critical to make an accurate diagnosis as early as possible to initiate the appropriate antibiotic regimen, in order to prevent these complications.[1]

Allergic diseases are complex diseases caused by a combination of genetic and environmental factors. Allergic diseases are on the increase, affecting approximately 30% to 40% of the world's population. They decrease quality of life and may have an immense influence on personal, social, and economic costs.[1,2,3]

Hay fever, allergies, sinusitis and influenza could all lead to respiratory problems.[3] It is important to accurately treat them to avoid the preventable respiratory tract infections from manifesting. This could decrease the frequency of antibiotic requirement associated with such respiratory tract complications.[4]

ALLERGIC RHINITIS (HAY FEVER)

Hay fever, also known as allergic rhinitis (AR), is an IgE-mediated hypersensitivity response causing inflammation of the nasal passages.[5]

An allergic response is a hypersensitivity reaction mediated by the adaptive immune system. The presence of a trigger, such as an allergen or antigen, induces a humoral immunological response, which in turn initiates a complex immunological reaction. This dysregulation in the immune function elevates the plasma levels of immunoglobulin E (IgE). The release of IgE is followed by binding to the allergen or antigen, which in turn stimulates the mast cells to degranulate and release several pro-inflammatory substances that include histamine, chemokines and numerous cytokines.

There is vast evidence that shows that T-lymphocytes play a major role in allergic diseases. The T-helper cell type 1 (T_h1)/T-helper cell type 2 (T_h2) paradigm has been extensively studied and seems to be the major pathological pathway in allergic diseases. The paradigm explains the relationship between the T_h1 and T_h2 subsets of the T lymphocyte. T_h1 and T_h2 subsets tend to differentiate from CD4+-naïve T lymphocytes. This means that whenever a raised response towards either the T_h1 or the T_h2 subset occurs, the other

will be reduced.[1-3] When there is a reduction in T_h1 production, there are observed decreased levels of interferon gamma (IFN-g), interleukin (IL)-2 and tumour necrosis factor (TNF)-beta. This in turn leads to elevated levels of T_h2 effect, owing to a decrease in IgG production, which inhibits T_h2 formation.[5,6,7]

Allergic rhinitis can be classified either as seasonal, perennial or episodic, according to the frequency of exposure to the stimulus. Table I differentiates between seasonal, perennial or episodic allergic rhinitis. Many different factors come into play when searching for the causative agent of allergy. Environmental influences that occur in pregnancy and early childhood can alter the physiological, immune, structural and behavioural development and thus transform response patterns that influence susceptibility to future diseases. Genetics also play a vital role in the susceptibility of an individual to an allergic disease. The most common allergic conditions around the world include atopic dermatitis, rhinitis, asthma, rhinosinusitis, allergic conjunctivitis and, most recently, allergic oesophagitis.

Allergic rhinitis affects approximately 20% of the world population and is considered to be the most common chronic disease. AR is a type 1 allergic disease which reduces quality of life depending on the severity. Exposure to nasal allergens stimulates an IgE-mediated type 1 hypersensitivity reaction, resulting in symptomatic reactions to the allergen. The early characteristic symptoms of allergic rhinitis are rhinorrhoea, nasal congestion and sneezing. AR can also be associated with various conditions such as bronchial asthma, allergic conjunctivitis, rhinosinusitis and others.[8,9,10]

Diagnosis

A history of nasal symptoms after exposure to allergen and other physical signs related to this condition aid with the diagnosis, however a skin prick test to determine if the reaction is IgE-mediated is preferred for a definitive diagnosis. An alternative, but less sensitive, is in vitro testing (blood). As asthma is an important co-morbidity of allergic rhinitis, a spirometry test can be done to assess pulmonary function.[11]

Pathophysiology

Allergic rhinitis is a condition which occurs due to inflammation of the epithelial lining of the nasal mucosa. This inflammatory process is initiated by the release of histamine owing to the cross-linkage of IgE antibodies with the mast cells. The mast cells then release histamine and other chemokines. The nasal epithelial cells appear to be central in responding directly to exogenous stimuli, such as pollen.[12-17]

The most common antigens for allergic rhinitis are inhaled allergens, of which dust mites, animal dander and pollen are the major ones of concern. When the patient is sensitised, an antigen comes into contact with the nasal mucosa. This leads to a cross-linking of IgE-mediated receptors on the mast cells. In turn, this leads to the degranulation of the mast cells, with a resultant release of histamine and proteases from the preformed granules. In addition, an array of early-phase pro-inflammatory molecules are synthesised and released, especially prostaglandins, leukotrienes, cytokines, tumour necrosis factor-alpha (TNF-α), and IL-4. The release of these molecules causes oedema and fluid secretion, which may result in congestion and other nasal symptoms. The role of leukotrienes as mediators in allergic rhinitis is well-supported in the literature. Cysteinyl leukotrienes are able to facilitate the maturation of eosinophil precursors, and act as eosinophil chemoattractants, promoters of eosinophil adhesion, and inhibitors of eosinophil apoptosis. The leukotrienes and thromboxane A_2 (TXA_2) are arachidonic acid derivatives. It has been shown in animal models that TXA_2 agonists increase nasal airway resistance and vascular permeability. An acute-phase allergic reaction is also characterised by the production of prostaglandin D_2 (PGD_2), a major proinflammatory prostanoid, which results in vasodilation and bronchoconstriction, as well as a number of inflammatory biomarkers, such as N-α-tosyl-L-arginine methyl esterase (TAME)-esterase and eosinophil cationic protein (ECP). PGD_2 is also believed to be associated with hypertrophic inflammation and acts as a recruiter of eosinophils.

The late-phase or chronic inflammatory response involves cellular infiltration, which sustains tissue swelling and oedema, and further exacerbates congestion. The ensuing cytokine release results in the nasal mucosa being infiltrated with inflammatory cells. These inflammatory cells, including eosinophils, neutrophils, basophils, mast cells and lymphocytes, sustain and intensify the nasal mucosal inflammatory reaction. The predominant cell type, namely the eosinophil, characterises the chronic inflammatory process which is present during the late-phase allergic response. These eosinophils release a broad range of proinflammatory mediators, including the cysteinyl leukotrienes, ECP, eosinophil peroxidase and major basic protein. These cells are also known to serve as a major source of IL-3, IL-5, granulocyte colony-stimulating factor and IL-13. The number of circulating eosinophils is increased in patients with allergic disorders, and the infiltration at the site of aggravation has generally been attributed to the influx of mature cells. In some studies, eosinophil infiltration has been shown to have a significantly negative correlation with nasal airflow in patients with allergic rhinitis.[12-22]

Table I. Classification of allergic rhinitis (Clinical Practice Guideline: Allergic Rhinitis, 2015)[11]

Seasonal	Perennial	Episodic
IgE-mediated response that occurs only during specific seasons. Example: Pollen during pollen season.	IgE-mediated inflammatory response is caused by contact to a stimulus that is not normally in the individual's environment. Example: A cat at a friend's house.	IgE-mediated inflammatory response caused by allergens that are present throughout the year. Example: Mites and moulds.

In addition to the eosinophils, other proinflammatory cells are also known to accumulate within the nasal epithelium during the late-stage response. These include basophils, mast cells, and T cells. The key inflammatory mediator of the late-phase response is TNF-α. TNF-α levels increase dramatically approximately an hour after an allergen challenge. This cytokine has been confirmed to activate T cells, endothelial cells, fibroblasts and macrophages. TNF-α is also responsible for an increase in the expression of cell adhesion molecules. Patients with allergic rhinitis also have elevated proinflammatory interleukins (IL-1β, IL-6 and IL-8). All of these events, including IgE synthesis and eosinophil or basophil priming, contribute to the venous engorgement, inflammation, nasal and ocular hyperreactivity, and the symptoms of allergic rhinoconjunctivitis.[12-22]

Management of allergic diseases

Allergic diseases can be strategically managed both non-pharmacologically and pharmacologically. The use of pharmacological preparations is usually preferred for use when non-pharmacological methods prove ineffective or insufficient in alleviating the allergic symptoms. Different pharmaceutical preparations (systemic, intranasal, topical etc.) are used depending on the symptoms and type of allergic disease.[23-25]

Local decongestants

Local decongestants are mainly sympathomimetic drugs that stimulate α_1-adrenergic receptors producing vasoconstriction. This in turn decreases mucosal oedema and local vasodilation.[26-30] Examples of the most commonly used drugs include xylometazoline, phenylephrine and oxymetazoline. Local decongestants are usually indicated to reduce acute symptoms as prolonged use can produce undesirable effects to the user. After continued use (usually more than five days) rebound rhinitis and conjunctivitis medicamentosa start to appear. Oxymetazoline and xylometazoline have a long-acting effect on the α_1-receptor, whereas phenylephrine has a shorter duration of action, lasting up to approximately four hours.[23-25]

Table II. The differences between first- and second-generation histamine$_1$-antihistamines[23-25]

	Older, first-generation H_1-antihistamines	Newer, second-generation H_1-antihistamines
Drug examples	Promethazine Chlorpheniramine Dexchlorpheniramine Hydroxyzine Cyclizine	Cetirizine and levocetirizine Loratadine Ebastine Fexofenadine Mizolastine Rupatadine
Frequency	Usually administered in 3–4 daily dosages.	Usually administered once or twice a day.
Mechanism of action	Potent blockers of H_1, α_1 and muscarinic receptors.	Selective H_1-receptor antagonists.
Blood-brain barrier	Cross the blood-brain barrier (lipophilicity, low molecular weight and lack of recognition by the p-glycoprotein efflux pump).	Generally, do not cross the blood-brain barrier at recommended dosages (lipophobicity, high molecular weight and recognition by the p-glycoprotein efflux pump).
Indications	The options for sedation include hydroxyzine, promethazine and diphenhydramine. However, more suitable agents may be used in the management of insomnia.	Fexofenadine has the shortest half-life of the systemic agents. Furthermore, it also does not display any H_1-receptor occupancy inside the central nervous system at therapeutic dosages.
	As an antiemetic agent, choose from cyclizine (*syn.* meclizine), diphenhydramine, hydroxyzine or promethazine, for example. First-generation H_1-antihistamines may be very useful in the management of postoperative nausea and vomiting, as well as vertigo.	Cetirizine has the greatest likelihood of displaying some degree of H_1-receptor occupancy inside the central nervous system, which may result in some level of sedation, albeit at higher-than-recommended dosages.
	Chlorpheniramine displays lower levels of sedation than many of the other examples in this group, and may therefore be better suited to the management of allergic reactions.	Rupatadine fumarate is approved for the treatment of allergic rhinitis and chronic urticaria for adults and children aged 12 years and older.
Side-effects	Potentially cause side-effects, such as: Sedation Drowsiness and dizziness Hyperactivity (meta-reaction) Insomnia Convulsions Impaired driving performance Fatigue and lassitude (well documented)	Do not cause relevant side-effects (sedation, fatigue, hyperactivity and convulsions) in the absence of drug interactions.
	Anticholinergic side-effects, including a dry mouth, urinary retention, gastrointestinal upset and appetite stimulation.	Minor side-effects include: Nausea Light headedness Drowsiness Headaches Agitation and a dry mouth
Toxicity	Case reports of toxicity are regularly published.	There have been no reports of serious toxicity.
Overdose	A lethal dosage has been identified in infants and young children.	Do not cause fatality in overdose.

Systemic decongestants

These agents stimulate α_1-receptors producing vasoconstriction, reducing oedema, redness and itching. Their preparations usually contain an antihistamine. It is important to note that combination therapy of a systemic decongestant and an older-type H_1-antihistamine can produce drowsiness and a lack of motor coordination. Systemic decongestants available in South Africa include pseudoephedrine, phenylpropanolamine and phenylephrine. The use of phenylpropanolamine has produced sub-arachnoid bleeding with a haemorrhagic stroke in women using it as an appetite suppressant. The total daily dosage of phenylpropanolamine should not exceed a 100 mg.[23-25]

Corticosteroids

Glucocorticosteroids can be used for various allergic conditions such as asthma, allergic rhinitis and minimal use in allergic conjunctivitis. They exert their pharmacological action by modifying protein synthesis through regulating transcription, and indirectly by modifying the activity or half-life of transcription factors and mRNA. The currently available intranasal corticosteroids include: beclomethasone, budesonide, fluticasone, mometasone, triamcinolone and ciclesonide. The newer agents, namely mometasone, fluticasone, and ciclesonide, are also administered intranasally and result in minimal systemic effects. The most common local side-effects experienced with the intranasal corticosteroids include dryness, stinging, burning, and epistaxis. Chronic use of topical corticosteroids may lead to atrophy of the nasal mucosa. It is therefore advisable to use these agents for the shortest time possible to prevent unpleasant adverse effects associated with long-term use. Systemic corticosteroids such as hydrocortisone and prednisone can be used in chronic dermatitis to reduce frequency of allergic flares.[26]

The H_1-antihistamines

H_1-antihistamines based on pharmacological classification, are grouped into different generations. This system of classification is based on their target receptors as well as side-effect profile. The H_1-antihistamines are classified into first generation (older, sedating multi-potent blockers) and second generation (non-sedating, newer) antihistamines. First-generation antihistamines include promethazine, chlorpheniramine, dexchlorpheniramine and cyclizine whilst the second-generation antihistamines include cetirizine (and levocetirizine), loratadine, ebastine, fexofenadine and mizolastine. The most significant difference between the two classes is that first-generation H_1 antihistamines have the ability to cross the blood-brain barrier and the second-generation non-sedating H_1 antihistamines have very limited ability, if none at all, to cross the blood-brain barrier. It is also important to note that two generations of systemic (oral and/or parenteral) agents, topical (including intranasal and ophthalmic) H_1 antihistamines are available as well.[23-25] Table II summarises the differences between first- and second-generation histamine$_1$-antihistamines.

First-generation H_1-antihistamines

These older H_1-receptor blockers have shown to have sedative and multi-potent receptor-blocking abilities. Their ability to cross the blood-brain barrier distinguishes them from the newer generation H_1-antihistamines. The chemical structure of the first-generation antihistamines permits them to have a certain degree of non-selectivity, exerting antagonistic effects of an antimuscarinic or anticholinergic, antihistaminergic, α_1-adrenergic blocking, anti-serotonergic and local anaesthetic nature. Because of their wide range of receptor blocking, the first-generation H_1-antihistamines have a variety of indications and uses, which range from allergies and rhinoconjunctivitis, to nausea and vomiting, motion sickness and insomnia. Some of their effects on multiple receptors, on the other hand, are undesirable and they are not recommended to be used in patients who suffer from glaucoma, benign prostatic hyperplasia and in cardiac patients (i.e. ischaemic heart disease, myocardial infarction and congestive heart failure).[23-25] Table III, summarises the adverse effects of the first-generation histamine$_1$-antihistamines, as reflected by their receptor activity.

Table III. The adverse effects of first-generation histamine$_1$-antihistamines, as reflected by receptor activity[23-25]

Receptor antagonistic interaction	Side-effects
Histamine$_1$-receptor	A reduction in central nervous system neurotransmission, sedation, reduced cognitive and neuro-psychomotor performance, and an increased appetite
Muscarinic receptor	Xerostomia, urinary retention and sinusoidal tachycardia
α-adrenergic receptor	QTc-interval prolongation and ventricular arrhythmias
Serotonergic receptor	An increased appetite
IKr and other cardiac channel receptors	QTc-interval prolongation and ventricular arrhythmias

The following drugs in this group of first-generation histamine$_1$-antihistamines are of note:

- The options include hydroxyzine, promethazine and diphenhydramine. These drugs are used in the management of insomnia but there are more suitable agents that may be used.
- Cyclizine (*syn.* meclizine), diphenhydramine, hydroxyzine or promethazine, are examples of antiemetic agents. First-generation H_1-antihistamines may be very useful in the management of postoperative nausea and vomiting, as well as vertigo.
- Chlorpheniramine is better suited for use in allergic reactions due to its relatively lower sedation levels than the other first-generation antihistamines.

It should be noted that these "older" drugs have never been optimally investigated and profiled from a clinical pharmacology perspective.[23-25]

Second-generation H_1-antihistamines

Second-generation H_1-antihistamines are relatively newer antihistamines that do not possess the ability to cross the blood-brain barrier. They also have no antiemetic, anticholinergic and central nervous system effects, unlike the first-generation antihistamines. Drugs like fexofenadine are actively transported into the lumen of the gut, kidney and brain by p-glycoproteins, which restrict their ability to accumulate and cause unwanted side-effects. However, agents such as rifampicin, which induce p-glycoprotein, may increase the clearance of fexofenadine and reduce its efficacy.[30-35] Second-generation H_1-antihistamines are mostly dosed once daily with minimal risk of developing tolerance. The long-term safety of the second-generation H_1-antihistamines, cetirizine, desloratadine, fexofenadine, levocetirizine, and loratadine, has been documented in randomised controlled trials lasting 6–18 months in adults, and in children as young as 1–2 years old.[23-25]

Ophthalmic (eyedrop) preparations include levocabastine, epinastine, olopatadine and ketotifen (the latter also acts as a mast cell stabiliser). Levocabastine, in addition to azelastine, is also available as a nasal spray for use in patients who suffer from allergic rhinitis.[23-25]

Rupatadine fumarate is a newly launched, second-generation, long-acting histamine antagonist (H_1-receptor antagonist) and platelet-activating factor receptor inhibitor. Rupatadine fumarate is approved for the treatment of allergic rhinitis and chronic urticaria in adults and children aged 12 years and older. It inhibits the degranulation of mast cells and the subsequent release of cytokines, more specifically of tissue necrotising factor which is available in mast cells and monocytes.[23-25]

The leukotriene-receptor antagonists

Examples of leukotriene receptor antagonists include zafirlukast and montelukast. They are competitive antagonists of the cysteinyl leukotriene receptor-1 (*cys*LT-1). They have the advantage of oral administration. Montelukast is also available as a sprinkle and in a chewable tablet form for convenient use in paediatrics. Montelukast presents an additional option in the management of seasonal allergic rhinitis in children with asthma.[23-25]

SINUSITIS

Sinusitis is characterised by the inflammation of paranasal sinuses[27]; the inflammation and fluid build-up in one or more of the sinuses make it hard to breathe through the nose.[28] Sinusitis is also referred to as rhinosinusitis because the inflammation involves the paranasal sinuses and the nasal mucosa.[28]

Sinisitis is classified either as acute or chronic sinusitis, based on the pathological findings and duration of infection.[28] Table IV differentiates between bacterial and viral sinusitis, based on the causative strain, duration, type of nasal secretions and treatment.

Classification

Based upon symptom duration:

- Acute sinusitis – Symptoms < four weeks
- Subacute sinusitis – Symptoms for 4–12 weeks
- Chronic sinusitis – Symptoms continue > 12 weeks
- Recurrent acute sinusitis – Four or more episodes per year[28]

Diagnosis of sinusitis

- Based on physical examination: signs and symptoms
- Computer tomography (CT) or magnetic resonance imaging (MRI) for chronic sinusitis[28]

Table IV. Difference between diagnosis of bacterial and viral sinusitis[28]

Bacterial versus viral sinusitis		
	Bacterial sinusitis	**Viral sinusitis**
Cause	• *Streptococcus pneumoniae* • *Haemophilus influenzae* • *Moraxella catarrhalis* • *Staphylococcus aureus*	• Rhinovirus • Influenza virus • Parainfluenza virus
Duration	7–10 days	10–14 days
Nasal secretions	Thick purulent	Thick purulent
Treatment	Antibiotics where needed	Symptomatic treatment

Pathogenesis of sinusitis

The nasal cavity is divided by the nasal septum, and is composed of bone more proximally and cartilage more distally.

The inferior, middle, and superior turbinates, which are lined with mucosa, are made up of pseudostratified columnar ciliated epithelium that overlies the basement membrane and submucosa that consist of seromucous nasal glands, nerves and a large number of blood vessels. The nasal epithelium is covered by a thin layer of mucus that moves by ciliary action towards the nasopharynx. Viral upper respiratory tract infections commonly cause mucosal swelling, loss of ciliary activity and an increase in mucous secretions; mucociliary function might also be impaired by cigarette smoke and environmental pollutants.[29]

Sinusitis can either be classified as acute, subacute or chronic depending on the symptom duration, while the causes vary from viruses, bacteria, fungi, and noninfectious infections and can be associated with asthma, allergic rhinitis, smoking, or viral upper respiratory tract infections.[27]

Complications of acute sinusitis

Complications occur most frequently in children and patients with depressed immune functions. Involvement of the particular sinus determines the type of complication. *Haemophilus influenzae*, *Streptococcus pneumoniae*, and *Staphylococcus* are the most frequently cultured bacteria.

These complications may be categorised as extracranial, intracranial, and those involving the bone of the sinus wall (osteitis). Extracranial manifestations may be divided into preseptal or postseptal infections. When misdiagnosed, orbital complications may result in permanent visual loss, or the infection may spread to the intracranial structures giving rise to life-threatening conditions like epidural empyema, subdural empyema, meningitis, brain abscess, and rarely, cavernous sinus thrombosis.[30]

INFLUENZA

Influenza, commonly referred to as flu, is a viral illness that is caused by the influenza virus and has a high mortality and hospitalisation rate.[30-36] Influenza can occur all year round but is seen more from May through winter.[31] Due to the constant evolution of the influenza strains there is a high fatality rate associated with the virus.[31]

The estimated death rate from flu is between 6 000–11 000 in South Africa every year.[32] About half of those deaths are in elderly patients, and 30% in HIV-infected individuals.[32] During the flu season in South Africa about 8–10% of patients hospitalised for pneumonia and 25% of patients with flu-like illness (fever and cough) will test positive for influenza.[32]

Pathophysiology

The influenza virus is transmitted via air droplets when a person comes into close contact with an infected person or via self-infection when a person comes into direct contact with an infected person or object.[33]

A rapid onset of fever, headaches, myalgia, body aches and pains, sore throat and rhinitis (runny nose) are associated with the flu.[31]

These symptoms generally last for 4–5 days and then disappear, however a person may experience coughing and malaise for more than 14 days.[31] Influenza-like illness (ILI) is an acute respiratory infection that presents with a fever greater than 38 ˚C, coughing or pharyngitis.[31]

Diagnosis

The diagnosis of influenza-like illness is rarely based on the patient's clinical picture. Laboratory diagnosis usually includes:

- Virus isolation in cell culture
- A polymerase chain reaction (PCR) test
- Antigen detection[34]

Table V summarises the main differences in symptoms between influenza, allergic rhinitis and sinusitis.

Table V. Difference in symptoms between influenza, allergic rhinitis and sinusitis[35]

Factors	Influenza	Allergic rhinitis (hay fever)	Sinusitus
Symptoms	• Sudden fever, chills • Aching muscles and joints • Headache • Severe malaise • Dry cough and lack of appetite • Blocked and/or runny nose • Your "whole body" feels sick	• No fever • Congestion • Runny or stuffy nose (clear, white thin mucus) • Sneezing • Itchy nose, throat and eyes • Cough is rare	• Thick yellowish/green nasal discharge • Facial tenderness • Headache • Congestion • Fatigue • Fever
Causes	• Viruses spread by: • Sneezed or coughed droplets into the air from an infected person • Cold and dry weather, as people spend more time close together indoors	• Exposure to irritants/triggers: • Dust mites • Animal dander • Pollen • Mould spores	• Caused by bacterial infection in most cases • Usually occurs as a late complication of a common cold • Those with asthma/allergies may also be predisposed to the development of sinusitis
Onset and duration of symptoms	• Sudden onset • Lasts about a week	• Weeks, months or all year • Symptoms last as long as you are exposed to the allergen	• Can last weeks, months or even years
Prevention	• Highly recommended to administer a flu vaccine to prevent the flu, especially if history of asthma, recurrent ear infections, and sinusitis • Best time for flu vaccine is March–June	• Avoid allergens for example: • Remove carpeting to reduce dust mites and mould • Air conditioning may help reduce mould • Use mattress and pillow covers to reduce dust mites	• Sinus drainage medications (e.g. decongestants) during times of increased susceptibility, such as flu and cold season
Treatment	• Most people recover without treatment	• Antihistamines • Intranasal steroids • Immunotherapy may help	• Inhaled nasal corticosteroids • Decongestants • Antibiotics to control a bacterial infection, if present • Pain relievers to reduce any pain or fever • Steam inhalations and/or nasal saline washes

Conclusion

Some similarities in symptoms between flu, allergic rhinitis and sinusitis can cause confusion and lead to incorrect diagnosis and treatment. An incorrect treatment approach to allergic rhinitis may lead to complications, such as sinusitis and/or ear infections. Whereas an incorrect treatment approach to acute sinusitis can lead to life-threatening conditions such as permanent visual loss, epidural empyema, subdural empyema, meningitis, brain abscess, and cavernous sinus thrombosis. Flu can give rise to sensorineural hearing loss, Guillain-Barre syndrome, pneumonia and secondary infections with methicillin-resistant *Staphylococcus aureus*. Understanding the differences between these illnesses and asking the right questions can assist in making the correct diagnosis and choosing an appropriate treatment approach. Patients understandably confuse an allergy with flu and or sinusitis. The approach to treatment for these three illnesses is different. Therefore, it is important for the pharmacist to correctly evaluate the patient in order to advise the patient appropriately.

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