Diagnosing Allergic Rhinitis

Glenis K. Scadding, MD, FRCP[a,*], Guy W. Scadding, MBBS, MRCP[b]

KEYWORDS

• Allergic rhinitis (AR) • Diagnosis • History • Skin prick tests • Specific IgE
• Nasendoscopy • Nasal allergen challenge

KEY POINTS

- Typical symptoms of allergic rhinitis (AR) include nasal blockage, discharge, itching, and sneezing; eye symptoms are common.
- AR may present with comorbidities, including cough, impaired asthma control, chronic otitis media with effusion, and sleep disturbance.
- Diagnosis requires suitable history plus confirmation of allergy by skin or serum IgE testing.
- Nasal allergen challenge may be necessary if local AR is suspected.
- Differential diagnosis is wide, including nonallergic, infective, inflammatory, and structural disease.

INTRODUCTION

The need to diagnose rhinitis accurately and to treat it effectively is undeniable given its prevalence and negative impact on quality of life and productivity.[1] Nevertheless, the condition is too often ignored, underdiagnosed, or misdiagnosed and hence managed inadequately.[2] The starting point is to be aware of the many underlying causes and the many ways in which rhinitis manifests. Beyond establishing the diagnosis of AR, the physician and patient should identify the worst/most troublesome symptoms, their timing, likely exacerbating factors, and effects on quality of life.

DIAGNOSIS OF ALLERGIC RHINITIS

Patients with AR may present with classical symptoms making the diagnosis more straightforward but more challenging when patients present with atypical features,

The authors have nothing to disclose.
[a] Department of Allergy and Rhinology, Royal National Throat, Nose and Ear Hospital, 330 Gray's Inn Road, London WC1X 8DA, UK; [b] Allergy, Royal Brompton Hospital, Sydney Street, London SW3 6NP, UK
* Corresponding author.
E-mail address: g.scadding@ucl.ac.uk

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for example, chronic cough in an atopic child. Rhinitis should also be differentiated from chronic rhinosinusitis (CRS) (**Box 1**).[3]

The next step is accurately making a diagnosis of AR is to determine whether the rhinitis is allergic in origin or due to 1 of myriad alternative, nonallergic causes. In addition, efforts should be made to identify the likely causative allergens. This is generally unproblematic in cases of isolated seasonal allergy (seasonal AR/hay fever) but more complicated in polysensitized individuals with perennial disease. An accurate allergy diagnosis allows selection of appropriate pharmacotherapy, informs the possibility of allergen avoidance, and allows consideration of allergen-specific immunotherapy. Additionally, the importance for patients of understanding triggering factors should be acknowledged because that is likely to improve outcomes, including adherence to therapy.

Diagnosis is made based on patient history, clinical examination, and skin prick tests (SPTs) or serum-specific IgE tests. Additional tests may be required in cases of uncertainty and in consideration of differential, nonallergic diagnoses.[4]

HISTORY

The classic symptoms of rhinitis are nasal running, nasal congestion, sneezing, and itching. Two or more of these symptoms, for more than an hour per day, for more than 2 weeks is diagnostic,[1] but a more detailed history is necessary to identify specific triggers. Causes of rhinitis may be broadly grouped into allergic, infective, structural, or other. There may be overlap between these categories. For example, an individual with AR's condition may be complicated by the presence of nasal septal deviation. To ensure a comprehensive history, patients can be asked to complete a rhinitis questionnaire prior to consultation. They should also be notified ahead of time of the need to avoid use of antihistamines for at least 72 hours prior to the appointment, if possible, to allow for skin testing.

Manifestations of rhinitis, which are particularly suggestive of allergy, include sneezing, itchy nose, itchy palate, and eye involvement. The timing of these – perennial, seasonal, in certain locations, or during certain activities – may provide a clue to the responsible allergen(s). Recurrent seasonal symptoms suggest triggers, such as pollens or mold spores. Symptoms experienced within the home may be due to pets, infestations (cockroaches or mice) or house dust mites. Symptoms predominantly at work might indicate an occupational allergen, for example, bakers sensitized to flour or bread improvers or laboratory animal allergy.[5] Because disease progresses, chronic nasal inflammation produces generalized nasal mucosal hyper-reactivity and more persistent symptoms, potentially masking a clear correlation with allergen exposure. Periods of prolonged absence from allergen, such as holidays, may result in disease remission or attenuation, further suggesting the correlation of an allergic trigger exposure with disease.

Box 1
Symptoms of rhinitis and chronic rhinosinusitis

Rhinitis: nasal running, blocking, sneezing, and itching; eye symptoms, particularly in seasonal allergis rhinitis

CRS: nasal blockage, discharge (anterior and/or posterior), facial pain/pressure, reduced olfaction; diagnosis confirmed by endoscopic findings and/or CT scan

Data from Fokkens W, Lund V, Bachert C, et al. EAACI position paper on rhinosinusitis and nasal polyps executive summary. Allergy 2005;60(5):583–601.

Eye symptoms associated with AR, especially seasonal AR, include itching, redness, and swelling of the conjunctiva with lacrimation. This complex of symptoms is termed *rhinoconjunctivitis*. More severe allergic eye diseases – atopic keratoconjunctivitis and vernal keratoconjunctivitis – may occur in individuals with eczema and warrant a specialist ophthalmologic opinion.[6]

Rhinorrhea (nose running) can be anterior or posterior, manifesting as postnasal drip, and may or may not be due to allergy. Classically, AR causes bilateral clear secretions. Isolated, unilateral clear nasal discharge is uncommon and in this circumstance cerebrospinal fluid leak should be excluded.[7] Cerebrospinal fluid leak most commonly occurs after sinus surgery or trauma, but may be spontaneous. Discolored secretions can be associated with allergy. For example, eosinophils in secretions give a yellow coloration whereas neutrophils yield green secretions, potentially indicating infection, although this may mask underlying AR. Crusting of secretions within the nose is possible in AR but is not usually pronounced. Primary complaints of nasal crusting and nose bleeding may suggest other pathologies, such as CRS, nose picking, Wegener granulomatosis, sarcoidosis, other vasculitides, ozena/atrophic rhinitis (wasting away of the bony ridges and mucous membranes inside the nose), noninvasive ventilation, cocaine abuse, or frequent use of nasal decongestants. Crusting may also occur for a period after nasal or sinus surgery. Intranasal corticosteroids not uncommonly, particularly if applied incorrectly, cause some nasal bleeding and may, rarely, cause crusting.

Nasal obstruction may be accepted as normal by some patients with longstanding rhinitis and also by the parents of some rhinitic children because of the common tendency for them to mouth breathe for some time after the common cold, or because of adenoid hypertrophy. Obstruction may be partial or complete, with severity often correlating with systemic manifestations, for example, sleep problems. AR usually results in bilateral nasal congestion, but other common factors, such as septal deviation, may make it appear unilateral. Alternating nostril obstruction may occur due to changes in blood pooling in capacitance vessels from one side of the nose to the other, contributing to mucosal swelling, which is a normal physiologic phenomenon referred to as the nasal cycle.[8] Other causes of obstruction include nasal polyps, foreign bodies, and rarely tumors. The differential diagnosis is age dependent with consideration of encephaloceles and choanal atresia in young children. Paradoxically, the dry, spacious intranasal appearances seen in atrophic rhinitis or after aggressive inferior turbinectomy surgery (empty nose syndrome) are often associated with a subjective sensation of nasal obstruction.

The diagnosis of AR may be missed when a patient's primary complaint is 1 of its many comorbidities (**Box 2**). Allergic conjunctivitis may be the focus of attention but

Box 2
Comorbidities of allergic rhinitis

Conjunctivitis
Chronic otitis media with effusion; eustachian tube dysfunction
Sleep impairment; obstructive sleep apnea
Rhinosinusitis; hyposmia
Bronchial hyper-reactivity; asthma
Pollen-food cross-reactivity
Laryngeal irritation; globus phenomenon

is virtually always accompanied by rhinitis. Children with chronic otitis media with effusion often have concomitant rhinitis. Most asthmatics have rhinitis or rhinosinusitis of some kind. A diagnosis of rhinosinusitis requires symptoms of nasal obstruction and discharge together, or 1 of these symptoms plus hyposmia, facial pain, or headache, alongside confirmatory findings on endoscopy or CT scan.[3] CRS may be associated with or without nasal polyps. AR seems to be a risk factor for the development of CRS without polyps but less so in CRS with polyps.

AR can be associated with systemic manifestations, such as difficulty sleeping, snoring, fatigue, and impaired concentration, leading to reduced productivity or poor school performance.[9] Repeated sniffing or a nasal intonation of the voice can be caused or exacerbated by nasal obstruction and rhinorrhea from any cause.

Lower respiratory tract symptoms, including cough, wheeze, and exertional dyspnea, may be associated with AR even in the absence of overt asthma. Bronchial hyper-reactivity can be induced by upper airway inflammation, as evidenced by changes in histamine/methacholine bronchial provocation doses after seasonal allergen exposure in hay fever sufferers.[10] Disorders of the upper and lower respiratory tract often coexist: most asthmatics have rhinitis or rhinosinusitis of some kind,[11] whereas a significant minority of individuals with AR have coexistent asthma.[12] Importantly, rhinitis/rhinosinusitis may impair asthma control[13] and should always be considered in the assessment of patients with poorly controlled asthma. Aspirin-sensitive asthma in particular is frequently associated with CRS with polyps. Typically both upper and lower respiratory tract symptoms are more severe than in other forms of rhinitis and asthma.

AR may be associated with food allergy due to cross-reactivity between aeroallergens and allergens within foods, described as pollen-food syndrome (also oral allergy syndome). In Northern Europe, by far the most common presentation is seen in silver birch pollen–allergic patients. The major birch allergen, Bet v 1, shows structural homology with proteins in stone and seed fruits of the Rosaceae family as well as with many tree nuts.[14] Other aeroallergen sensitizations associated with cross-reactivity include grass pollens, weed pollens, and latex. Typically, symptoms on ingestion of cross-reacting foods are limited to the mouth and oropharynx but may occasionally be more generalized.

A diagnosis of AR is more likely when rhinitis is seasonal, in the presence of asthma, or with a family history of atopy. To assess exposure to possible allergens and irritants a full social history is required, including housing conditions (floor level, dampness and mildew odors, dust reservoirs like carpet and bedding, soft toys, carpeting, central forced air heating, or cockroach or rodent infestations), the presence of pets or other contact with animals, and school environment; and, in young children, feeding details should be obtained. Information regarding smoking, exposure to second-hand smoke and other pollutants, hobbies, and alcohol consumption should be considered. Occupational history may be relevant either as a direct cause of AR or because of workplace triggers that exacerbate preexisting rhinitis (work-exacerbated rhinitis).[5] It is important to recognize occupational rhinitis because it usually precedes the development of occupational asthma and, therefore, these patients should be more closely monitored to prevent the development of occupational asthma. Professions most at risk for occupational asthma that may present as occupational rhinitis include bakers, furriers, and animal laboratory workers.[15]

Drug history is important because several medications can cause or aggravate rhinitis symptoms. These include antihypertensive medications, aspirin and other nonsteroidal anti-inflammatory drugs, oral contraceptives, and, in particular, topical sympathomimetics/nasal decongestants, which can provoke a rebound nasal

congestion (rhinitis medicamentosa) if used for extended periods of time without the use of a nasal corticosteroid spray. It is also important to inquire about the efficacy of previous rhinitis treatments and specifically whether they were used preventatively on a daily basis or as needed in response to acute symptoms only.[2]

EXAMINATION

Examination effectively begins during the history taking process – observation of frequent sniffing, mouth breathing, use of tissues, nasal speech, and nose rubbing (allergic salute) may all be seen. Initial inspection of the face may reveal clues to allergy, such as a horizontal nasal crease across the dorsum of nose (**Fig. 1**), the presence of red watery eyes suggestive of allergic conjunctivitis, facial eczema, and dark circles/shadowing beneath the eyes referred to as allergic shiners. Depression of the nasal bridge can be a postsurgical phenomenon or caused by Wegener granulomatosis or cocaine misuse. A widened bridge suggests nasal polyposis. Purple discoloration of the nasal tip can be due to sarcoidosis; prominent telangiectasia suggests hereditary hemorrhagic telangiectasia, which may present with epistaxis.

Chronic mouth breathing suggests complete or near-complete nasal obstruction. Nasal airflow can be simply assessed by observing for misting of a cold metal spatula held beneath the nostrils in patients of any age or by more complex methods (nasal inspiratory peak flow, acoustic rhinometry, and rhinomanometry) in older children and adults.

The nose should then be examined internally, preferably with a nasal endoscope, but if not available using a head-mounted light and nasal speculum (anterior rhinoscopy) or with an otoscope using the largest diameter end piece. Examination by nasal endoscopy is more specific than anterior rhinoscopy and alters the diagnosis in up to a

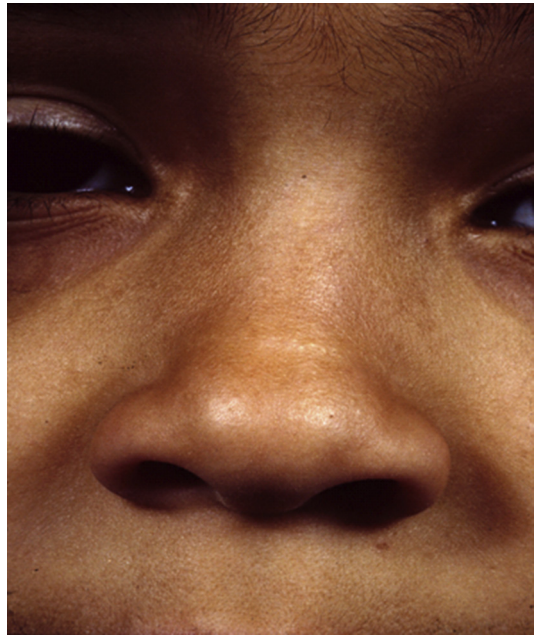

Fig. 1. Transverse nasal crease in a child with AR.

fifth of patients with nasal disease.[16] Appearances may be normal in AR (particularly if examined outside of seasonal allergen exposure) or may classically demonstrate hypertrophic, pale, boggy inferior and/or middle turbinates with clear secretions. Polyps, if visible, can be distinguished from the inferior turbinate by their insensitivity to touch, yellow/gray color, and the ability to get between them and the side wall of the nose. Yellow submucosal nodules with a cobblestone appearance suggest sarcoidosis.[17] Crusting and granulations raise the possibility of vasculitis. Septal perforation may occur after septal surgery, due to chronic vasoconstriction (cocaine or topical decongestants), Wegener granulomatosis, nose picking, or use of nasal-prong oxygen supplementation and rarely secondary to corticosteroid nasal sprays. Although septal deviation is common and rarely the main cause of symptoms, it may contribute to lateralization of symptoms and to difficulty with application of intranasal sprays. The throat, postnasal space, palate, and ears should also be inspected. All patients with persistent rhinitis should also have chest examination, including spirometry or peak flow, to assess for possible asthma.

INVESTIGATIONS

Allergen-specific IgE can be detected with SPTs or by serum immunoassay (**Boxes 3** and **4**). SPTs have the advantage of being immediate, educational for patients, and relatively cheap. SPTs should be carried out routinely in all patients to determine the presence of atopy and possible causative allergens except those patients with dermatographism or chronic eczema or when medications blocking histamine receptors cannot be discontinued (antihistamines, tricyclic antidepressants, and topical, but not oral, corticosteroids). In these circumstances, serum-specific IgE testing should be performed. Skin testing to standardized aeroallergens is extremely safe. Regardless of whether standardized or nonstandardized reagents are used, injectable adrenaline should be available given the theoretic risk of inducing a systemic allergic reaction, even though this complication is extremely rare. Intracutaneous tests are more sensitive and as a result are more likely to yield false-positive results. Furthermore, they are more painful for patients and, therefore, not routinely recommended for inhalant allergens, especially when all SPTs are negative and a patient's clinical

Box 3
Skin prick testing

A basic set of SPT allergens: in the United Kingdom, house dust mite, grass and tree pollens, and cat and dog danders show positivity in up to 95% of AR sufferers. Supplementation with other allergens suggested by the history may further improve diagnosis, for example, other animals, cockroach, rodents, molds, latex, and flour.

In young children, SPTs may be extended to include certain common allergenic foods for example, cow's milk, egg, soy, wheat, fish, peanut, and tree nuts.

A negative control: saline/allergen diluent, plus a positive control; histamine should be used.

Contraindications: widespread eczema, dermographism, recent antihistamines or extensive topical corticosteroid, pregnancy.

Interpret in relation to history: positive tests may be found in symptom-free patients – increased risk of developing allergic symptoms in time – and may persist even after immunotherapy. Negative testing in patients with high clinical suspicion may warrant blood analysis for serum-specific IgE to exclude a false-negative result and, if also negative, possibly nasal challenge to 1 or more specific aeroallergens.

Box 4
Serum-specific IgE testing

Typically by modified sandwich immunoassay

When skin testing not possible (eczema, use antihistamines) or allergen reagent not available

In cases of equivocal/unexpected skin test results

Allergen component diagnostics may aid differentiation between primary allergy and cross-reactivity.[18]

Data from Luengo O, Cardona V. Component resolved diagnosis: when should it be used? Clin Transl Allergy 2014;4:28.

history is less than convincing for AR. In circumstances where a patient gives a clear history of symptoms in response to exposure (ie, cat or dog), it is not unreasonable to place a selective intracutaneous test at a nonirritating dilution (1:1000 weight/volume) to rule out sensitization definitively. This still misses, however, local AR, and nasal provocation testing to 1 or more specific aeroallergens is the most definitive way to exclude an allergic component to chronic rhinitis. If a patient has a history of anaphylaxis or severe allergic symptoms in response to the inciting allergy, SPT administration is inadvisable outside an allergy specialist's office.

Testing total IgE alone is of limited benefit and may lead to erroneous assumption of allergy as the cause of symptoms if it is elevated. It may aid interpretation, however, of specific IgE in certain circumstances, such as severe eczema, where total IgE may be grossly elevated and modest elevations in serum-specific IgE may have a lower positive predictive value. Serum allergen–specific IgE generally correlates with the results of SPTs, showing similar sensitivity for house dust mite, but SPTs are more sensitive for other inhalant allergens, such as cat epithelium, mold, and grass pollen.[19] Regardless of which test is used, a certain degree of expertise is required to read and/or interpret the results to ensure a correct diagnosis of AR is established.

Component-based analysis, in which the actual proteins to which a patient is sensitized are revealed, is probably unnecessary for aeroallergen diagnosis in most settings where there are clearly defined allergen seasons. Confusion can arise, however, due to cross-reacting molecules, such as profilins, present in many pollens and fruits, when there is a long pollen season due to multiple trees and grasses, as in Southern Europe. For example, in this situation, testing for Bet v 1, the major birch allergen, can reveal those genuinely sensitized to birch pollen as opposed to sensitization to a minor profilin allergen only.[18] Such an approach may be advisable before embarking on treatment with allergen-specific immunotherapy.

When SPTs/serum-specific IgE tests and clinical history are concordant, then a diagnosis of AR can be made and treatment instituted. When they are discordant, further investigations may be needed (**Fig. 2**). If AR is strongly suspected from the history but not supported by these initial tests, nasal allergen challenge (**Box 5**) may be considered, but its use is generally limited to specialist centers. Challenges may also be undertaken in cases of occupational rhinitis where a high degree of diagnostic certainty is required and when considering allergen-specific immunotherapy for perennial allergens where causality of rhinitis symptoms is often more difficult to infer compared with seasonal allergens. Nasal allergen challenge typically involves administration of a defined concentration of allergen in aqueous solution by nasal spray. Outcomes include increased clinical symptoms, objective measures of decreased nasal airflow

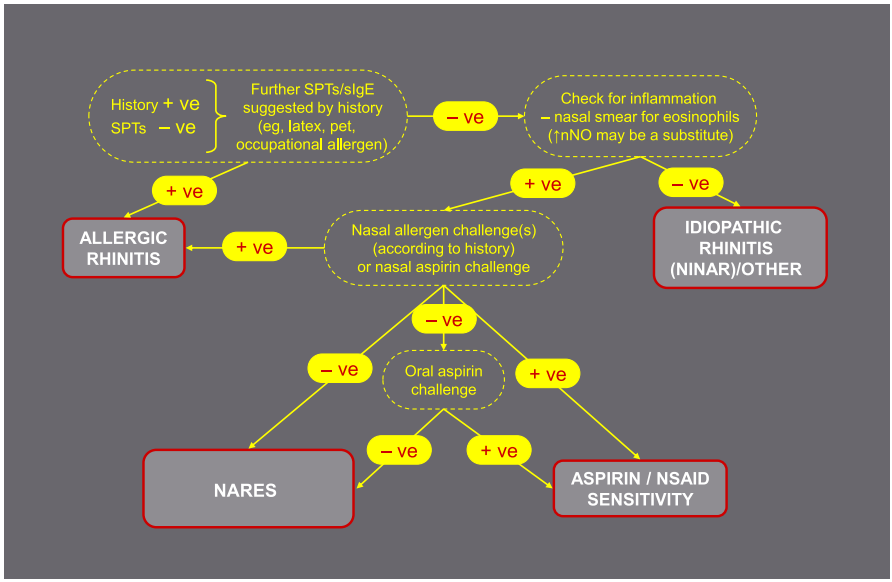

Fig. 2. Further investigations in cases of discordant history and skin/specific IgE tests. NINAR, noninfectious non-AR. NSAID, Non-steroidal anti-inflammatory drug.

or volume, and changes in cytology, local cytokines, tryptase, and other mediators as measured in nasal lavage fluid prechallenge and postchallenge. In individuals with a history of aspirin hypersensitivity, nasal lysine aspirin challenge may confirm hypersensitivity.[20] Nonspecfiic generalized nasal hyperresponsiveness, independent of allergy, can be investigated by provocation with histamine, metacholine or cold, dry air.

Objective measurements of the nasal airway (**Box 6**) are not generally made in routine clinical practice but are important measures of nasal challenge outcomes and may be helpful in assessing the nasal airway objectively if septal surgery or turbinate reduction is contemplated.

Additional laboratory investigations may be considered in cases of diagnostic uncertainty, to address differential diagnoses (**Table 1**), and may provide information to assist with treatment.[4] A complete blood cell count with differential white cell count may reveal peripheral eosinophilia in cases of CRS with nasal polyps or coexistent

Box 5
Uses of nasal allergen challenge

Confirmation of clinical relevance in cases of polysensitization to aeroallergens

Selection of patients for allergen immunotherapy (eg, for house dust mite)

Investigation of symptoms in the absence of evidence of systemic allergen sensitization (local AR)

Proof of symptom causation for novel and occupational allergens

Assessment of pathomechanisms of AR

Assessment of therapeutic interventions, such as antihistamines, corticosteroids, and allergen-specific immunotherapy

Box 6
Objective measures of the nasal airway

Peak nasal inspiratory flow is simple, inexpensive, and reproducible but effort dependent. Results correlate with rhinoscopic evidence of rhinitis but not with symptom scores. Most useful for comparing changes in airway patency within the same subject, although some normative data are now available.[21]

Acoustic rhinometry is the measure of acoustic impedance within the nasal cavity from which the cross-sectional area at different depths within the nasal cavity can be calculated. The method requires standardization and considerable experience to interpret and obtain reproducible results. Guidelines for its use are published.[22]

Rhinomanometry allows an estimation of nasal resistance from pressure-flow relationships and is difficult to perform reproducibly but still regarded by some investigators as the most accurate measure of nasal airway patency. The technique requires expensive equipment and considerable experience in interpretation.[23]

Data from Refs.[21–23]

asthma. C-reactive protein and erythrocyte sedimentation rate may be elevated in inflammatory conditions, such as vasculitis.

Nasal smears/mucus cytologic examination with estimation of eosinophil percentage and/or evidence of eosinophil degranulation (Charcot-Leyden crystals) may provide a diagnosis of non-AR with eosinophilia syndrome (NARES) and may be helpful in

Table 1
Differential diagnosis of allergic rhinitis

Type	Features
NARES	Skin tests negative; nasal smears show eosinophilia. May go on to develop nasal polyposis.
Autonomic rhinitis (vasomotor)	Physical/chemical triggers. More common in middle age with clear rhinorrhea, especially in the morning.
Drug induced	β-Adrenergic blockers, angiotensin-converting enzyme inhibitors. Rhinitis medicamentosa with chronic nasal decongestant use.
Hormonal	Pregnancy, oral contraceptives, thyroid disease.
Food	Gustatory rhinorrhea, for example, with spicy foods, sulphites.
Atrophic	Foul-smelling odor, crusting, hyposmia, nasal blockage.
Cystic fibrosis	Children with polyps must be screened for cystic fibrosis.
Primary ciliary dyskinesia	Rhinosinusitis, bronchiectasis, and reduced fertility.
Systemic/inflammatory	For example, Churg-Strauss syndrome.
Immunodeficiency	Chronic infective sinusitis secondary to antibody deficiency.
Malignancy	Bloody, purulent discharge, pain, and nasal blockage – symptoms may be unilateral.
Granulomatous diseases	Sarcoidosis. Wegener disease.
Structural abnormalities	Unilateral nasal obstruction secondary to nasal septal deviation.
Idiopathic/noninfectious non-AR	Cause unclear; may respond to topical capsaicin.
Local AR	Skin and serum IgE test negative but positive response to nasal allergen challenge.

predicting response to corticosteroids. Nasal swabs, taken from the middle meatus, may provide evidence of relevant infection; however, it should be known that asymptomatic carriage of *Staphylococcus aureus* in particular may be found in many individuals.

In cases of nasal crusting, blood-stained discharge, and/or nasal perforation, consideration should be given to the possibility of cocaine abuse – toxicology testing of urine or hair samples, with patient consent, can confirm recent use. In cases of unilateral, watery discharge, particularly after sinus surgery or head injury, a sample may be sent for assay of beta-2 transferrin, an isoform of transferrin limited to cerebrospinal fluid.

Concerning olfactory tests, the University of Pennsylvania Smell Identification Test is well validated, can identify malingerers,[24] and is accepted for legal cases.

Fractional exhaled nitric oxide (FeNO) measurement can be useful clinically in the diagnosis and monitoring of asthma. Normal levels are less than 20 parts per billion but increase in response to lower respiratory tract inflammation. FeNO levels may be elevated in AR patients, even in the absence of overt, clinical asthma. Nasal levels (nasal nitric oxide [nNO]) are widely variable in AR but in general tend to be elevated; very low FeNO levels can be a guide to the presence of primary ciliary dyskinesia or cystic fibrosis.[25]

Radiology is not routinely recommended for AR patients. Sinus CT scans are abnormal in a third of the adult population and almost half of children, probably because of the common cold and its prolonged after effects, so should only be undertaken when absolutely necessary, as advised by otorhinolaryngologists.

Referral to an otorhinolaryngologist is indicated for patients who have worrying symptoms, such as unilateral symptoms, blood-stained discharge, new-onset nasal polyp(s), and pressure effects on the orbit or orbital cellulitis (urgent referral). Cases of confirmed AR with persistent symptoms despite aggressive medical therapy require referral to an allergy specialist to confirm the diagnosis of AR versus mixed or non-AR and/or for consideration of allergen immunotherapy if appropriate.

SUMMARY

All patients presenting with upper and/or lower respiratory complaints should be questioned for symptoms of AR and asthma. Although AR can be simple to assess, it can also hide amid a variety of comorbidities and complications, thereby obfuscating its diagnosis. It can also form part of a mixed rhinitis alongside nonallergic nasal hyper-reactivity. A detailed history is the most important part of diagnosis, supported by testing for allergen-specific IgE to identify whether allergen sensitization correlates with reported symptoms on exposure. Discordance in test results and history of exposure necessitates further assessment and likely referral to an allergy or otolaryngology specialist.

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