Allergic Rhinitis and Sinusitis

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RHINITIS

GENERAL PRINCIPLES

• One of the most common chronic diseases, allergic rhinitis (AR) is characterized by rhinorrhea, nasal congestion, postnasal drainage, nasopharyngeal itching, and sneezing.
• AR symptoms are caused by environmental allergens.
• The prevalence of AR is increasing.
• Rhinitis includes AR, nonallergic rhinitis (NAR), and nonallergic rhinitis with eosinophilia syndrome (NARES).

Definition

• AR is allergen-driven mucosal inflammation.
• AR must contain one or more of the following symptoms:1
  ○ Nasal congestion
  ○ Sneezing
  ○ Itching
  ○ Rhinorrhea
  ○ Postnasal drip
• For rhinitis to be classified as allergic, the patient must have evidence of immunoglobulin E (IgE) sensitization to an allergen by skin testing or radioallergosorbent test (RAST).
• Other associated symptoms include palatal pruritus, pruritus of the ear canals, ocular pruritus and watering, and some patients have anosmia or reduced sense of smell.
• Nonallergic rhinitis (NAR) is not mediated by IgE.
  ○ There is nasal mucosal inflammation.
  ○ Symptoms are similar to AR usually without itching.
  ○ No sensitization to allergens is demonstrated.
• NARES is NAR with eosinophilia syndrome.
  ○ Symptoms are very similar or identical to AR.
  ○ There is no IgE sensitization to allergen.
  ○ Large numbers of eosinophils are present on nasal smear (may be >20%).
  ○ Patients tend to be middle-aged and often have paroxysmal exacerbations.
  ○ Patients are at increased risk for obstructive sleep apnea.

Classification

• Allergic rhinitis can be classified into seasonal, perennial, and episodic.
  ○ Seasonal AR: Patients have signs and symptoms of AR occurring in only one or more seasons, but not year round. They are sensitized to seasonal allergens such as trees, grasses, or weeds.
○ **Perennial AR:** Patients have signs and symptoms of AR throughout the year, though they may also have seasonal exacerbations if they are sensitized to seasonal allergens.
  - Allergens typically include dust mites, molds, pet dander, or insects.
  - Symptoms must be present >2 hours/day, >9 months out of the year.
○ **Episodic AR:** Patients have signs and symptoms of AR to allergens they are sensitized to, but which are not present regularly in their environment. An example would be a patient who has symptoms when visiting a friend who has a cat, but the patient does not come into daily contact with the cat. \(^1\)

- **Mixed rhinitis:** Patients have a combination of AR and NAR.

**Epidemiology**
- AR affects between 10 and 30% of all adults. \(^1\)
- Mixed rhinitis affects 44–87% of patients with rhinitis. \(^1,2\)
- In 2002, the financial burden in the US (direct and indirect costs) was estimated at $11.58 billion. \(^1\)
- Prevalence ranges from 3 to 19%.
- 80% of AR develops before age 20.
- Equal male and female distribution among adults.
- Adults have a higher prevalence of perennial AR and children have a higher prevalence of seasonal AR.

**Etiology**
- **Allergens:** Sensitization to aeroallergens may occur even in the first 2 years of life. \(^1\)
- **Anatomic causes** of rhinitis include septal deviation, foreign bodies, adenoid hypertrophy, choanal atresia, and tumors.

**Pathophysiology**
- AR is caused by specific IgE-mediated reaction to environmental allergens.
- Mast cells and basophils located on the superficial mucosa of the respiratory tract have specific IgE bound to its cell membrane. When allergens bind and cross-link the IgE, cellular degranulation occurs.
  ○ Mast cells degranulate and cause release of pre-formed mediators and newly synthesized mediators that cause the allergic reaction. \(^2\)
  ○ Preformed mediators include histamine, tryptase, chymase, kininogenase, heparin, and other enzymes.
  ○ Newly formed mediators include prostaglandins, leukotriene (LT) C4, LTD4, and LTE4.
- Nasal congestion is typically a late-phase response.
- Eosinophils release mediators causing tissue damage in the late phase response. \(^1\)
- **Priming** occurs with prolonged allergen exposure resulting in repeated late-phase responses even with very small exposures—inflammatory mediators continue to be released and symptom resolution may lag behind the decrease in pollen. \(^1\)
- NAR causes include hormonal, vasomotor, and medication-induced.

**Risk Factors**
- Family history of atopy.
- Serum IgE >100 IU/mL before age 6.
- Higher socioeconomic status.
- Presence of a positive skin prick test. \(^1\)
• First-born children are more likely to have AR.
• Environmental risk factors include smoke exposure and allergen exposure in infancy.

**DIAGNOSIS**

**Clinical Presentation**
• Patients typically present with sneezing, rhinorrhea, postnasal drip, nasal itching, and congestion.¹
• Other symptoms include itching of palate, conjunctiva, throat, Eustachian tubes, and middle ear.
• Ear fullness and popping, as well as pressure over cheeks and forehead may be reported.
• Occasionally, chronic cough may be the presenting symptom.
• Often patients can associate the onset of symptoms to a particular trigger.
• Common comorbidities include:¹
  ○ Asthma.
  ○ Obstructive sleep apnea.
  ○ Nasal obstruction from severe nasal septal deviation.
  ○ Inferior turbinate hypertrophy.
  ○ Adenoidal hypertrophy.
  ○ Refractory sinusitis.
  ○ Allergic conjunctivitis.

**History**
• History is the most important step in diagnosis.
• Important elements of the history:
  ○ Frequency of symptoms.
  ○ Severity of symptoms (both past and present).
  ○ Relationship to past symptoms.
  ○ Length of time symptoms appear after triggers.
• Triggers may be multiple.
• Assess whether the symptoms occur at home and/or work on vacation.
• Assessment of home environmental conditions should include:
  ○ Water damage or mold.
  ○ Pets.
  ○ Carpet.
  ○ Age of pillow and mattress, type of fillers.
  ○ What other irritants are nearby (e.g., farms, woods, and vacant lots)?
  ○ Are heat and air conditioning central?
  ○ Use of fireplace or humidifiers.
  ○ Are symptoms exacerbated by dusting or vacuuming the house?
• Medications specifically inquire about:
  ○ Does the patient take aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs), oral contraceptives, angiotensin-converting enzyme (ACE)-inhibitors, or β-blockers?
  ○ What are current and past medications used to treat AR?
• Family history of atopic disease should be determined.
• Quality of life should be assessed.
  ○ Ask about fatigue, learning and attention problems, and sleep disturbance.
  ○ Ask about time missed from work or school.
  ○ Effect on quality of life is often under-recognized and inadequately treated.¹
• Rhinorrhea should be described as predominately clear. Persistent, colored rhinorrhea may indicate sinus disease.
• Time frame of exacerbations should be established.
  ○ Are symptoms always worse on awakening?
  ○ Are there particular seasons that are worse?
  ○ Are symptoms completely gone during portions of the year?

Physical Examination
• A thorough examination of the head, eyes, ears, nose, and throat should be performed.
• Note if findings are unilateral or bilateral.
• Common findings in AR include:
  ○ Allergic salute is a crease across the bridge of the nose and is a result of rubbing the nose.
  ○ Dennie’s lines are infraorbital creases.
  ○ Conjunctivitis may be present in those with ocular symptoms.
  ○ Allergic shiners are infraorbital hyperpigmentation secondary to nasal congestion.
  ○ Turbinates are often edematous and pale. They may sometimes appear blue.
  ○ Cobblestoning in the posterior oropharynx indicates post-nasal drainage.
  ○ Ears should be evaluated for otitis or Eustachian tube dysfunction.
  ○ It should be noted whether septal deviation or nasal polyps are present.
  ○ Care should be taken to ensure there is no sinusitis (see below).
  ○ Heart and lung examination should be performed. Note whether wheezing is heard on lung examination.
  ○ The skin should be examined for signs of atopic dermatitis.
• If septal perforation is present, differential diagnosis includes:
  ○ Inappropriate use of nasal medications.
  ○ Adverse effects of other nasal medications.
  ○ Intranasal narcotic abuse.
  ○ Previous surgery.
  ○ Systemic granulomatous disease.

Differential Diagnosis
• The differential diagnosis of rhinitis is presented in Table 3-1.

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<tr>
<th>TABLE 3-1</th>
<th>DIFFERENTIAL DIAGNOSIS FOR RHINITIS</th>
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<td>Nonallergic rhinitis (NAR)</td>
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<td>Nonallergic rhinitis with eosinophilia syndrome (NARES)</td>
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<td><strong>Sinusitis</strong></td>
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<td>Rhinitis medicamentosa</td>
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<td>Cystic fibrosis</td>
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<td>Ciliary dysfunction</td>
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<td>Cerebrospinal fluid rhinorrhea</td>
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<td>Anatomical abnormalities</td>
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• Vasomotor rhinitis:
  ○ This is a type of NAR in which excessive vasomotor activity leads to chronic nasal congestion.
  ○ The mechanism is not completely known.
  ○ Etiologies include odors, alcohol, spicy foods, emotions, temperature change, and bright lights.
• Drug-induced rhinitis:
  ○ Common offenders include ACE-inhibitors, β-blockers, ASA, NSAIDs, oral contraceptives, phosphodiesterase-5-selective inhibitors, α-receptor antagonists, and cocaine.
• Hormonal rhinitis:
  ○ A type of NAR in which hormone altering events induce nasal obstruction and hypersecretion.
  ○ Events include hypothyroidism, oral contraceptive use, and pregnancy.
  ○ For pregnant women, symptoms usually appear during the second trimester but disappear after delivery.
• Rhinitis medicamentosa:
  ○ Occurs from prolonged use of intranasal decongestants.
  ○ Rebound congestion occurs and later nasal hypertrophy. This appears as beefy, red mucosa.
  ○ Rhinitis medicamentosa will resolve on discontinuation of the agent.
• Nasal polyps are outgrowths from the nasal passages.
  ○ Polyps typically start at the lateral wall and appear smooth, round, pale, and gelatinous.
  ○ Growth likely occurs from eosinophil-associated growth factors found in the eosinophils and immunoglobulins they contain.
  ○ The possibility of cystic fibrosis should be entertained if nasal polyps are found in children.
• Anatomic abnormalities should be considered, particularly in difficult-to-treat rhinitis.
• If cerebrospinal fluid (CSF) rhinorrhea is suspected, evaluate with β-transferrin in nasal secretions.1

Diagnostic Testing
• Skin testing and RAST testing are used to determine allergen sensitization. These are discussed in detail in Chapter 8.
• Purposes of serum IgE testing are three-fold: To provide evidence of allergic basis, to confirm suspected allergens, and to determine suspected sensitivity for avoidance measures and/or immunotherapy.1
• Epicutaneous skin testing is preferred.
• Testing to local trees, weeds, and grasses is usually performed. Testing is also usually performed to molds and perennial allergens.

Laboratories
• RAST testing is usually used only if skin testing is unable to be performed.
• The average sensitivity of serum-specific IgE assays is only 70–75%.1
• Reasons for skin testing might be contraindicated include use of antihistamines, extensive skin disease, and uncooperative patients who are not able to sit still for 15–20 minutes.
• Serum IgE and IgG subclasses are not used as diagnostic tools for AR.
Imaging
If anatomic abnormality or chronic sinusitis is suspected, a CT scan may be helpful.

Diagnostic Procedures
• Rhinoscopy may be used to:
  ○ Assess nasal passage structure.
  ○ Evaluate for nasal polyps and sinusitis.
  ○ Evaluate vocal chords.
• Nasal provocation testing is rarely performed and is used primarily for research purposes to confirm sensitivity to allergen.¹

Treatment
• When treatment with one class fails despite compliance, substitution of another class should be considered.
• If AR is mild, single-agent therapy or combination therapy may be used in addition to avoidance measures.
• For all intranasal preparations, patients should be instructed to spray medication away from the septum to avoid irritation and perforation.

Medications
First Line
• Intranasal steroids are the mainstay of therapy.
  ○ They are considered superior to all other medication choices for AR because they help prevent both early and late-phase response.
  ○ Steroids for intranasal use include beclomethasone, budesonide, flunisolide, fluticasone furoate, fluticasone propionate, mometasone, triamcinolone, and ciclesonide.
  ○ Typical adult dose is two sprays in each nostril daily.
  ○ Patients with seasonal AR should start intranasal steroids at least 1 week before the onset of pollen season.
  ○ May be used on an as needed basis but this is not as effective as daily use.
  ○ Systemic side effects are minimal.
  ○ Patients should be instructed on their proper use to prevent nasal septum trauma.
  ○ Nasal examination in patients on nasal steroids should evaluate for complications such as septal ulceration and perforation (rare).
  ○ Epistaxis may occur, and if frequent, discontinuation of the nasal steroid should be considered.
• Oral antihistamines are also commonly used.³
  ○ They reduce symptoms of rhinorrhea, nasal pruritus, sneezing, ocular pruritus, and tearing.
  ○ They are less effective at reducing nasal congestion.
  ○ Nonsedating second-generation antihistamines include loratadine, desloratadine, fexofenadine, cetirizine, and levocetirizine.
  ○ First-generation antihistamines are not generally used for AR secondary to their sedating properties. These include chlorpheniramine, diphenhydramine, doxepin, and hydroxyzine.
• Nasal antihistamines:
  ○ These may be as effective or superior to oral second-generation antihistamines.¹
  ○ They are generally less effective than intranasal steroids.
  ○ Examples include azelastine and olopatadine.
Second Line

- **Montelukast** is approved for seasonal and perennial AR.
- **Intranasal cromolyn**:\(^1,3\)
  - Inhibits mast cell degranulation.
  - Onset of action is 4–7 days.
  - Effective for episodic AR.
  - Must be used four times daily for maximal effect.
  - Has a good safety profile.
  - Is not as efficacious as nasal steroids or nasal antihistamines.
- **Intranasal anticholinergics (ipratropium)**:\(^1,3\)
  - Reduces rhinorrhea.
  - Not useful for nasal congestion.
  - Side effects include epistaxis and nasal dryness.
  - Use with caution in patients with glaucoma or prostate hypertrophy.
- **Nasal decongestants**:
  - Cause vasoconstriction and improve nasal edema, but no actual effect on the antigen-provoked nasal response.\(^1\)
  - Should not be used as single agent.\(^3\)
  - Continuous use should be limited to <5 days otherwise may lead to rhinitis medicamentosa.\(^3\)
  - Examples are oxymetazoline and phenylephrine.
- **Oral decongestants**:
  - Are occasionally useful in selected patients.
  - Most products generally contain phenylephrine.
  - Chronic use of these agents is not recommended.\(^3\)
- **Oral steroids**:\(^\)
  - Are rarely indicated in AR secondary to systemic side effects.
  - For severe, intractable nasal symptoms or if nasal polyps are present, a 5- to 7-day course may be considered.
- **Immunotherapy** (see Chapter 21):
  - Immunotherapy may be used for treatment of perennial and seasonal rhinitis when a specific allergen has been identified.
  - Successful approximately 80% of the time.
  - Considered unsuccessful if the patient has no relief from symptoms after 1 year of maintenance therapy.
  - Current recommendations suggest 3–5 years of therapy.

**Other Nonpharmacologic Therapies**

- **Environmental modification**:\(^\)
  - Dust mite avoidance:
    - Dust mite proof covers for mattresses and pillows are designed to help decrease the amount of dust mites and other allergens.
    - Vacuum with a HEPA filter.\(^1\)
    - Wash linens in hot water.
    - Indoor humidity should be kept <50% to avoid growth of fungi and dust mites.\(^1\)
    - Hard surface flooring is preferable
  - Avoid contact with pets. For cat allergen, the cat may be confined to a HEPA-filtered room.\(^1\)
Pollen counts are highest on sunny, windy days with lower humidity.
- Close windows and doors during pollen season.
- Performing outdoor activity in the evening when pollen counts are lower.

Mold exposure should be avoided at home.
- To eliminate fungi, sources of moisture should be eliminated initially.
- Porous surfaces should be replaced.

Eliminate cockroaches (much easier said than done).

Wear a HEPA and pollen-proof face mask (e.g., N95) when allergens cannot be avoided.

Nasal saline irrigations may help symptoms of chronic rhinitis.1,4

SPECIAL CONSIDERATIONS

Pregnancy
- Symptoms of AR increase in one-third of pregnant patients.1
- Both first- and second-generation antihistamines may be used. Cetirizine is a pregnancy class B medication.
- Oral decongestants should be avoided, particularly in the first trimester.
- Other medications that may be used include intranasal steroids (budesonide, beclomethasone, fluticasone propionate, class B), montelukast (class B), and sodium cromolyn (class B).
- Immunotherapy may be continued without dose escalation during pregnancy, but immunotherapy should not be initiated during pregnancy.

Elderly Patients
- Age-related changes such as cholinergic hyperactivity, anatomic changes, or concomitant medication use may affect rhinitis.
- Allergy is not a common cause of new onset rhinitis in persons >65 years.2
- Intranasal steroids and ipratropium may be used safely.
- If antihistamines are used, nonsedating agents should be chosen.2

COMPLICATIONS

- Medical complications of improperly or untreated AR include rhinosinusitis, otitis media, and rhinitis medicamentosa.1
- Psychological impact can include depression, anxiety, low self-esteem, and shyness.
- Septal irritation or perforation may occur as a complication of incorrect nasal steroid use.

REFERRAL

- There are multiple indications for referral to an allergist-immunologist (Table 3-2).
- Consultation with an allergist-immunologist has been demonstrated to improve outcomes such as compliance, quality of life, and patient satisfaction.1
- Patients that should be referred to an otolaryngologist for surgical management include:1
  - Nasal obstruction from severe nasal septal deviation (septoplasty is preferred over submucosal resection and has a high reported success rate).
  - Inferior turbinate hypertrophy requiring reduction in patients who have failed medical therapy.
TABLE 3-2  WHEN TO REFER TO AN ALLERGIST

Rhinitis with prolonged, severe disease and comorbid conditions such as:
- Asthma
- Recurrent sinusitis
- Nasal polyps

Complications occur
Patient has required systemic steroids for treatment
Symptoms interfere with quality of life or ability to function
Current medications are:
- Ineffective
- Associated with adverse reactions
- Multiple or costly over a prolonged period

Rhinitis medicamentosa has been diagnosed
Specific allergic triggers need identification
Increased level of education is desired
Allergen immunotherapy is considered
More education is needed
Patient requests consultation

- Adenoid hypertrophy (adenoidectomy).
- Nasal polyps which require removal (polypectomy).
- Patients with complications from refractory rhinosinusitis (functional endoscopic sinus surgery).

MONITORING/FOLLOW-UP

- Clinical improvement is a better measure for appropriate environmental control than the amount of allergen concentration.\(^1\)
- Patients should be assessed 2–4 weeks after initiation of therapy.\(^5\)
  - Single-agent therapy with intranasal steroids or combination therapy with intranasal steroids and oral antihistamines is usually a good starting point.
  - Oral antihistamines should be tried before leukotriene inhibitors.\(^3\)
  - Intranasal antihistamines and leukotriene inhibitors are more appropriate for those with seasonal AR.\(^3\)
  - If one medication regimen does not seem to be effective, addition of an agent or change to a different class may be warranted.

SINUSITIS

GENERAL PRINCIPLES

- Normal sinus function requires:
  - All sinus ostia must be patent.
  - Normal mucociliary function.
  - Normal local and systemic immune function.\(^2\)
- Rhinitis and sinusitis often coexist and rhinitis often precedes sinusitis.\(^2,6\)

Definition
Sinusitis is simply defined as inflammation of one or more of the paranasal sinuses.
Chapter 3 Allergic Rhinitis and Sinusitis

Classification

- Sinusitis is classified as acute, chronic, or recurrent.
- No consensus standards exist for defining chronic rhinosinusitis (CRS) versus acute rhinosinusitis (ARS).
- ARS is generally defined as symptoms for $\leq 1$ month.
  - Nasal drainage must be purulent.$^2$
  - Of note, acute sinusitis may last up to 12 weeks per episode.$^6$
- CRS consists of inflammation of the nasal passages lasting 12 weeks at a minimum despite medical management.$^2$
- Recurrent sinusitis is characterized by $>4$ episodes of acute sinusitis per year.$^6$
  Patients with recurrent sinusitis may need evaluation for immunodeficiency.

Epidemiology

- Ninety to ninety-eight percent of episodes of sinusitis are preceded by an acute viral upper respiratory infection.$^1$
- About 31 million people in the US have rhinosinusitis annually.$^2$
- Prevalence is estimated at 10–30% in Europe and 15% in America.$^6$
- Viral upper respiratory infections become bacterial rhinosinusitis only in 0.5–2% of the population.$^2$
- Chronic sinusitis is associated with AR in 60% of adults.$^2$
- Increasing resistance to first-line therapies is well known and includes $\beta$-lactamase production (gram-negative organisms) and alterations in penicillin-binding proteins (gram-positive organisms).
- More than one-third of Haemophilus influenza strains and virtually all Moraxella catarrhalis strains are penicillin resistant.

Etiology

- ARS is usually infectious, viral, bacterial, or fungal.$^2$
  - Viruses are the most common cause of acute sinusitis.
  - The most common bacterial etiologies in acute sinusitis are the following:
    - Streptococcus pneumoniae
    - H. influenza
    - M. catarrhalis
- Staphylococcus aureus, coagulase-negative Staphylococcus, and anaerobic bacteria are more common in CRS, but CRS is more often inflammatory.$^3$
- S. aureus is increasing in prevalence in sinusitis patients with nasal polyps.$^2$
- Pseudomonas aeruginosa frequently occurs in patients with cystic fibrosis.
- Sinusitis is less commonly a manifestation of systemic illness.

Pathophysiology

- Acute sinusitis often develops when the sinus ostia are obstructed leading to infection.
- Conditions that disrupt mucociliary clearance of secretions and promote ostial obstruction predispose patients to sinusitis including:
  - Rhinitis:
    - Ostial narrowing secondary to mucosal inflammation due to a viral infection.
    - Chronic mucosal changes from allergic disease.
  - Nasal polyps.
  - Anatomic abnormalities.
○ Foreign bodies.
○ Problems with mucociliary transport:
  ■ Cystic fibrosis.
  ■ Primary ciliary dyskinesia.
  ■ Viral infections and other causes of inflammation may also result in ciliary dysfunction.

### DIAGNOSIS

#### Clinical Presentation

- The diagnosis of rhinosinusitis is usually entirely clinical and the differentiation between viral and bacterial infections can be difficult.
- Multiple studies regarding the utility of symptoms and signs for diagnosing acute sinusitis have sometimes reached differing conclusions. A few have used the true gold standard (i.e., sinus puncture and culture), but more have used a surrogate standard (e.g., sinus plain films and CT). Radiography cannot differentiate viral from bacterial sinusitis.
- ARS symptoms within the first 7–10 days of illness typically indicate a viral rhinosinusitis.²
- Regarding ARS, The American Academy of Allergy, Asthma, and Immunology (AAAAI) states the following (strength of recommendation level C):⁷
  ○ Acute bacterial rhinosinusitis may be suspected when an upper respiratory tract infection last longer than 10–14 days.
  ○ Bacterial infection is more likely with a history of persistent purulent rhinorrhea, postnasal drainage, and facial pain.
  ○ The prominent symptoms of acute bacterial rhinosinusitis are nasal congestion, purulent rhinorrhea, facial-dental pain, postnasal drainage, headache, and cough.
  ○ Signs of ARS include sinus tenderness, purulent nasal discharge, erythematous mucosa, pharyngeal secretions, and periorbital edema.
- The American Academy of Otolaryngology-Head and Neck Surgery guideline indicates that ARS may be diagnosed by the presence of one or both of the following:⁸
  ○ Up to 4 weeks of purulent nasal discharge accompanied by nasal obstruction.
  ○ Facial pain-pressure-fullness.
  ○ Acute bacterial (rather than viral) rhinosinusitis is diagnosed when:
    ■ Symptoms or signs persist ≥10 days after the onset of upper respiratory symptoms or
    ■ Symptoms or signs worsen within 10 days after an initial improvement.
- The American College of Physicians’ clinical practice guideline advises the clinical diagnosis of acute bacterial rhinosinusitis should be made in those who:⁹
  ○ Have symptoms lasting ≥7 days and
  ○ Have maxillary pain or tenderness in the face or teeth and
  ○ Purulent nasal secretions.
- Two or more of the following symptoms when present are helpful when making the diagnosis of CRS in the setting of documented mucosal inflammation:¹
  ○ Mucopurulent nasal drainage (anterior and/or posterior).
  ○ Nasal obstruction or blockage.
  ○ Facial pain, pressure, and/or fullness.
  ○ Decreased sense of smell.
Differential Diagnosis
• The differential diagnosis for rhinosinusitis is presented in Table 3-3.
• Allergic fungal sinusitis:
  ○ Rare cause of chronic sinusitis.
  ○ The hallmark feature is the presence of sinus opacification due to accumulation of “allergic mucin” that is thick, inspissated secretions heavily laden with eosinophils, Charcot–Leyden crystals, and fungal hyphae.
  ○ Diagnosis usually requires surgery to establish.2
• Infectious fungal sinusitis:
  ○ More likely to be seen in immunocompromised patients.
  ○ Aspergillus fumigatus is the most common cause of fungal sinusitis.

Diagnostic Testing

Laboratories
If immunodeficiency is suspected, obtain a complete blood count (CBC) with differential and quantitative immunoglobulin levels. Otherwise blood tests are of little use.

Imaging
• Imaging to confirm the diagnosis of acute uncomplicated rhinosinusitis is usually not necessary.2–9
• Radiography cannot readily differentiate viral from bacterial acute sinusitis.
• The limited sinus CT has become the most widely used radiographic study for the diagnosis of sinusitis.
  ○ It is obtained in the coronal projection with cuts through the frontal sinus, anterior ethmoid/maxillary sinuses, and posterior ethmoid and sphenoid sinuses.
  ○ Allows for assessment of patency of the osteomeatal unit, the critical confluence of drainage from the maxillary and anterior ethmoid sinuses.
○ Imaging studies with CT usually reveal mucosal thickening and ostial plugging if the sinusitis is chronic.
○ CT is the test of choice for chronic sinusitis.²
• MRI is useful for evaluating allergic or infectious fungal sinusitis to rule out soft tissue extension.
○ It is not good for evaluating mucosal thickening or bony deformities.
○ Should not be used as an initial imaging method.

Diagnostic Procedures
• Skin prick testing evaluate for underlying AR may be performed.²
• Endoscopically directed middle meatus cultures may be helpful in adults.¹
• Rhinoscopy with the flexible fiber optic rhinoscope can help determine nasal and sinus anatomy.
• Biopsy is indicated if there is suspicion of a tumor or vasculitis. A biopsy may also be helpful to confirm the presence of invasive fungal infection.
• Ciliary function testing is indicated in the setting of recurrent otitis, sinusitis, and pneumonia with bronchiectasis (primary ciliary dyskinesia or Kartagener’s syndrome).
○ It is possible to do ciliary visual assessments, but a practical approach in the office setting is the saccharine test.
○ Electron microscopy of nasal mucosal biopsy is the only way to document abnormal cilia structure.
• Nasal cytology:
○ Presence of eosinophils may indicate AR, NARES, aspirin sensitivity, or nasal polyps.
○ Neutrophils are more indicative of infection.

TREATMENT
• Most cases of ARS are caused by viruses and are expected to significantly improve without antibiotic treatment within 10–14 days. Treatment should, therefore, be symptomatic for those without clinical signs suggestive of a bacterial infection.⁷
• Trials of the efficacy of antibiotics in ARS have been of variable quality and differing outcome measures. Most of the randomized trials did not definitively enroll only subjects with bacterial infections. Nonetheless, taken together, there does appear to be a modest benefit from antibiotic treatment. Of significant note, many patients in the control/placebo groups did spontaneously improve (on average 80% improved vs. 90% with antibiotics).¹⁰
• Uncomplicated acute bacterial rhinosinusitis may be treated with or without antibiotics.²,⁸–¹⁰
○ Patients without severe or prolonged symptoms may be managed initially with symptomatic treatment alone and followed for resolution. Worsening of symptoms during this time should prompt a reconsideration of antibiotic therapy.
○ Those with initially severe symptoms or those with symptoms for >7 days after diagnosis are typically treated with antibiotics.
○ Individual clinical judgment should be exercised when making the decision to forgo or prescribe antibiotic therapy.
• Appropriate first-line antibiotics for uncomplicated acute bacterial rhinosinusitis include amoxicillin, sulfamethoxazole-trimethoprim, and azithromycin for 10–14
Significant differences between groups of antibiotics, including newer more expensive ones, have not been demonstrated.\textsuperscript{8,10–12} The optimal duration of antibiotic therapy is unclear.\textsuperscript{7} Alternative antibiotic therapy should be considered in patients who worsen or do not improve during the initial 7 days of therapy.\textsuperscript{2,7,8,11} Reasonable second-line choices include high-dose amoxicillin–clavulanate, and oral fluoroquinolones and second- or third-generation cephalosporins.

While evidence is somewhat limited, the addition of intranasal steroids may have modest positive benefit in the treatment of ARS.\textsuperscript{2,7,13} There are no controlled trials of systemic steroids, and these are not routinely recommended. Analgesics should be prescribed to those with significant pain.\textsuperscript{8} Data to support the use of decongestants, antihistamines, mucolytics/expectorants, and sinus irrigation are lacking, but they are at least theoretically beneficial and often recommended.\textsuperscript{2,7} The treatment of CRS (i.e., ≥12 weeks), which is an inflammatory condition often accompanied by infection, is a more complicated matter. It may be associated with nasal polyposis or allergic fungal rhinosinusitis, or neither. Multicomponent treatment is necessary.

- Intranasal steroids are generally recommended for all patients.\textsuperscript{2,14} Oral steroids are sometimes used for severe symptoms.
- Data regarding the use of antimicrobials in CRS are sparse; however, the potential for an infectious contribution to chronic inflammation seems clear. When there is an evidence of purulence, most clinician will treat with an antibiotic. On the basis of microbiologic studies, amoxicillin–clavulanate, clindamycin, or an oral fluoroquinolone are reasonable choices.\textsuperscript{2,7,15} Duration of treatment is unclear, but 3 weeks is often recommended.
- Nasal irrigation is frequently recommended.\textsuperscript{16} Patients with AR should be maximally treated as needed.
- Surgical polypectomy may be indicated for severe polyposis.
- Functional endoscopic sinus surgery is frequently done for refractory CRS.

**Complications**

- Rare but dangerous complications may occur when sinus disease extends outside of the sinus cavity. These include orbital cellulitis, cavernous vein thrombosis, brain abscess, meningitis, osteomyelitis, oral-antral fistula, and mucocele.\textsuperscript{2,6}
- It should be remembered that *Clostridium difficile* colitis and candidiasis can be a complication of prolonged antibiotic therapy.

**Referral**

Indications for referral to an ENT surgeon are as follows:\textsuperscript{1,2,6}

- Evidence of anatomic defects by CT or physical examination, including foreign bodies, and tumors.
- Nasal polyps that obstruct sinus drainage despite medical treatment.
- Persistent sinusitis despite aggressive medical management.
- Sinus condition requiring biopsy.
- Sinusitis complicated by extension into local structures.
MONITORING/FOLLOW-UP

- Symptoms are expected to resolve between episodes of ARS.
- If symptoms continue after one course of antibiotics, an alternative antibiotic may be considered.
- If symptoms have not resolved after multiple courses of antibiotics, CT scan should be performed, and further workup for predisposing conditions should be performed.

REFERENCES