When Surgery, Antibiotics, and Steroids Fail to Resolve Chronic Rhinosinusitis

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KEYWORDS

• Omalizumab • Zileuton • Immunodeficiency • Adenoiditis
• Aspirin • Desensitization • Montelukast • Odontogenic
• Sinusitis

Chronic rhinosinusitis (CRS) is a clinical syndrome encompassing a heterogeneous group of diseases characterized by sinonasal mucosal inflammation for at least 12 consecutive weeks.[1] Culture-directed antibiotic therapy and systemic and intranasal corticosteroid (INCS) therapy are considered the mainstay therapies for CRS. Consensus documents from the Rhinosinusitis Task Force and European Position Paper on Rhinosinusitis and Nasal Polyps codify diagnostic criteria and treatment strategies to guide and streamline CRS management.[1] This article discusses recalcitrant CRS after standard medical therapy and primary endoscopic sinus surgery (ESS). Optimal management is centered on a rational approach designed to identify and treat underlying etiologic and exacerbating factors, to maximize antiinflammatory therapy, and to ensure that confounding causes of sinus symptoms are addressed.

CONFOUNDING LOCAL AND REGIONAL FACTORS THAT MIMIC PURULENT CRS

Endoscopy and sinus computed tomography (CT) scan are essential to the diagnosis of CRS, and both techniques are useful in evaluating local and regional processes that mimic purulent CRS (**Box 1**). With endoscopy, the presence of a foreign body or tumor should be readily apparent.

Adenoiditis is less obvious although recognized as the primary source of nasal purulence in children. In adults, persistent mucopurulent postnasal drainage may be secondary to an infected Thornwaldt diverticulum or bursa. A Thornwaldt bursa represents the embryonic remnant of adhesion of the pharyngeal ectoderm to the cranial end of the notochord. If this becomes closed at the orifice, a cyst develops. Remnants

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Box 1
Differential diagnosis of chronic rhinosinusitis in patients with mucopurulence

- Sinonasal
  - Rhinitis (acute, chronic)
  - Foreign body
  - Neoplasia
- Locoregional
  - Adenoiditis
  - Thornwaldt cyst
  - Dental abscess
  - Neoplasia

that fail to close, and instead form a diverticulum, may be a source of repeated episodes of purulent postnasal drainage in the absence of sinus pathology. Treatment is endoscopic marsupialization or removal.

Underlying dental pathology should be considered in any patient with recalcitrant purulent CRS, particularly those with unilateral disease. Identification of dental disease not only helps with appropriate antibiotic selection but also facilitates improved long-term outcomes.[2,3] Sinus CT is helpful in assessing periodontal disease or periapical abscess as a cause of sinusitis and is more sensitive than plain dental films or a panorex.[4] A recent study showed that more than 80% of CT scans with maxillary sinus fluid levels greater than two-thirds the height of the sinus and with mucosal thickening had dental pathology compared with 10% incidence of dental pathology in normal maxillary sinuses.[4] Not all patients have specific dental symptoms or signs. However, once the diagnosis is established, appropriate treatment of the offending tooth by dental surgeons frequently obviates the need for sinus procedures. Directed endoscopic sinus surgery is reserved for situations in which persistent edema prevents maxillary sinus drainage.

LOCOREGIONAL AND SYSTEMIC FACTORS ASSOCIATED WITH PERSISTENT DISEASE

In persistent CRS (after medical therapy and primary ESS), locoregional and systemic factors known to be predictive of failed ESS include smoking, allergies,[5] and asthma and aspirin intolerance.[6,7] Additional factors that can play a role in actual or simulated persistent sinus symptoms include gastroesophageal reflux disease (GERD) and allergies.

Gastroesophageal Reflux Disease

GERD is associated with CRS in several studies; however, there is no direct evidence of causality.[8] In a prospective trial using pH probe monitoring, acid reflux into the nasopharynx was significantly greater in patients with refractory CRS after surgery than in patients in whom ESS successfully relieved symptoms. DelGaudio[9] suggested that there was an association between GERD and recalcitrant CRS. An alternative explanation for failure could be that CRS was not causing the patient's symptoms; rather the inadequately treated GERD caused the symptoms, as the sensation of post-nasal discharge can be simulated by pharyngeal and glottic irritation from GERD. Proton pump inhibitors can reduce the frequency of postnasal drainage symptoms in patients with extraesophageal manifestations of GERD.[10] An open-label study