Inflammatory and Infectious Lesions of the Sinonasal Tract

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KEYWORDS

• Sinonasal • Rhinosinusitis • Vasculitis • Midline destructive disease

Key points

- The sinonasal tract is affected by a variety of nonneoplastic inflammatory diseases, which may be infectious, inflammatory, or autoimmune in origin.
- The differential diagnosis of granulomatous disease of the sinonasal tract includes infection (bacterial, mycobacterial, fungal, parasitic), vasculitic disease, sarcoidosis, cocaine-induced injury, and idiopathic disease.
- Sinonasal fungal disease comprises a spectrum of diseases in which fungi are noninvasive or invasive. Allergic fungal rhinosinusitis is not a fungal infection but an inflammatory reaction to fungi in the sinonasal tract.
- The end result of many sinonasal inflammatory processes is destruction of the midline, resulting in facial deformity.

ABSTRACT

The sinonasal tract is frequently affected by nonneoplastic inflammatory diseases. Inflammatory lesions of the sinonasal tract can be divided into 3 main categories: chronic rhinosinusitis, which encompasses a heterogeneous group of entities, all of which result in mucosal inflammation with or without polyps-eosinophils; infectious diseases; and autoimmune diseases and vasculitides, which can result in midline necrosis and facial deformities. This article reviews the common inflammatory lesions of the sinonasal tract with emphasis on infectious diseases, vasculitis, iatrogenic, and diseases of unknown cause. Many of these lesions can result in midline destruction and result in facial deformity.

OVERVIEW

The sinonasal tract is frequently affected by a variety of nonneoplastic inflammatory diseases. Inflammatory lesions of the sinonasal tract can be divided into 3 main categories:

1. Chronic rhinosinusitis (CRS), which encompasses a heterogeneous group of entities, all of which result in mucosal inflammation with or without polyps-eosinophils
2. Infectious diseases
3. Autoimmune diseases and vasculitides, which can result in midline necrosis and facial deformities

This article focuses on a variety of nonneoplastic inflammatory diseases of the sinonasal tract (**Box 1**).

Disclosure Statement: The authors have nothing to disclose.
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Surgical Pathology ■ (2016) ■–■
http://dx.doi.org/10.1016/j.path.2016.11.002
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Box 1
Inflammatory lesions of the sinonasal tract

- Rhinosinusitis
  - Acute rhinosinusitis
  - CRS
- Sinonasal inflammatory polyps
- Infectious diseases
  - Bacteria
    - *Klebsiella rhinoscleromatis*; other
  - Fungal
    - Allergic fungal rhinosinusitis
    - Fungal ball
    - Acute invasive fungal rhinosinusitis
    - Chronic invasive fungal rhinosinusitis
    - Other
  - Parasitic
    - Leishmaniasis
    - Rhinosporidiosis
  - Viral
- Extranodal Rosai-Dorfman disease
- Small vessel vasculitides
  - Granulomatosis with polyangiitis
  - Eosinophilic granulomatosis with polyangiitis
- Cocaine-induced midline destructive lesion
- Other/unknown cause
  - Sarcoidosis
  - Immunoglobulin G4 disease

CHRONIC RHINOSINUSITIS

INTRODUCTION

Rhinosinusitis can be classified as acute (symptoms for <1 month), subacute (symptoms between 1 and 3 months), and chronic (symptoms >3 months).

Acute rhinosinusitis is usually the result of an infection or allergic process and often does not generate a pathologic specimen. Persistent acute rhinosinusitis develops into a chronic process. CRS comprises a mixture of diseases all of which lead to obstruction and mucosal inflammation.[1,2] The pathogenesis of CRS is multifactorial, with infectious, genetic, and environmental exposures all thought to be important factors.[1–3]

GROSS DESCRIPTION

The mucosa in CRS may appear thickened and edematous. Polypoid changes may be noted. There is often a lack of correlation between the clinical symptoms and the gross (as well as microscopic) pathology.

MICROSCOPIC DESCRIPTION

On histology, CRS is characterized by submucosal edema and a mixed inflammatory infiltrate consisting of lymphocytes, plasma cells, eosinophils, histiocytes, and rare neutrophils (**Fig. 1**). Recent studies have identified Rosai-Dorfman–histiocytes in some patients with CRS.[4] Fibrosis is often not seen with the exception of those undergoing repeat surgery. The surface mucosa may show squamous metaplasia and, in long-standing cases, there may be goblet cell hyperplasia, mucosal thickening, and papillary hyperplasia of the mucosa. As mentioned earlier, the histologic features do not always correlate with the severity of the clinical symptoms.

DIFFERENTIAL DIAGNOSIS

Most cases of CRS are nonspecific but histologic examination may confirm a definitive cause like specific infectious agents or autoimmune diseases. Other entities in the differential diagnosis of CRS include sinonasal inflammatory polyps (which are often found in patients with CRS) and epithelial neoplasms (particularly in the presence of mucosal hyperplasia).

DIAGNOSIS

The diagnosis of CRS is based predominantly on clinical findings. Patients present with symptoms of nasal stuffiness/blockage, headache, and rhinorrhea of more than 3 months' duration. Pathologic examination is not needed for clinical diagnosis but cases that do not respond to conservative therapy often require functional endoscopic sinus surgery (FESS). Tissue removed during these procedures should be examined in its entirety to rule out other causes of CRS symptoms, such as specific infections, vasculitides, and neoplasms. Although the cause of CRS is not completely understood, it is not surprising that infection, including bacterial and fungal, has been implicated in the pathogenesis.[1–3,5–8] A genetic basis for CRS is supported by familial tendencies as well as the association of CRS with genetic syndromes such as cystic fibrosis (CF).[1–3,9,10]

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