Chapter 2
Nasal Cavity and Paranasal Sinuses
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2.1 Introduction

2.1.1 Embryology

The midface, or area between the upper lip and forehead, develops at between 4 and 8 weeks’ gestation [219]. The frontal prominence forms during the 4th postovulatory week and gives rise to the superior and middle portions of the face. The maxillary and nasal swellings form beneath the frontal prominence. At the end of the 4th week surface thickening of the nasal swellings forms the nasal placodes, which are of ectodermal origin and give rise to the epithelial lining of the nasal cavity and paranasal sinuses. The placodes invaginate, producing the nasal pits that become the anterior choanae (nostrils) and, less superficially, the primitive posterior choanae. The lateral nasal and maxillary processes fuse to form the philtrum and columella. The cartilaginous nasal capsule forms deep to the nasal and frontal bones from the chondrocranium (skull base) during the 7th and 8th postovulatory weeks. The paranasal sinuses develop from the lateral nasal walls at the 6th foetal week, and their growth continues after birth, throughout childhood and adolescence.

2.1.2 Anatomy

The nasal cavities are separated by the nasal septum, and limited by a roof, which is formed by the cribriform plate of the ethmoid, and a floor, which is formed by the hard palate [261]. The lateral walls have three turbinates or conchae, and three horizontal spaces, or meatus, on each side. The nasolacrimal duct opens in the inferior meatus, whereas the middle meatus receives drainage from the frontal, anterior ethmoid and maxillary sinuses. Below the superior turbinate is the sphenoethmoid recess, with the openings of the sphenoid and posterior ethmoid sinuses. Each nasal cavity communicates posteriorly with the nasopharynx through the choanae. The paranasal sinuses develop from the lateral nasal walls at the 6th foetal week, and their growth continues after birth, throughout childhood and adolescence.

2.1.3 Histology

The nasal vestibule and skin share a similar histology. At the level of the limen nasi, the keratinising squamous epithelium gradually changes first to cuboidal or columnar epithelium, and then to ciliated respiratory-type epithelium, which lines most of the nasal cavity and all the paranasal sinuses, with the exception of the roof [261]. Numerous goblet cells are interspersed in the respiratory-type epithelium. The lamina propria contains several seromucous glands, lymphocytes, monocytes, and a well-developed vascular network, particularly evident in the inferior and middle turbinate. The olfactory epithelium is predominantly made of columnar non-ciliated sustentacular cells, with scattered bipolar sensory neurons and basal cells.

2.2 Acute and Chronic Rhinosinusitis

2.2.1 Viral Infections (Common Cold)

Infectious rhinitis is typically viral and is often referred to as the “common cold”. It is more common in children than in adults, and the most frequently identified agents are rhinovirus, myxovirus, coronavirus and adenovirus [67, 271]. Swelling of the mucosa may cause obstruction of a sinus ostium, with subsequent secondary bacterial infection (acute bacterial sinusitis). The histologic findings include marked oedema and a non-specific mixed inflammatory infiltrate of the lamina propria.

2.2.2 Bacterial Infections

Bacterial rhinosinusitis usually follows a viral infection or allergic rhinitis, and the most commonly involved agents are Streptococcus pneumoniae, Haemophilus influenzae and Moraxella catarrhalis [11, 34]. A dense inflammatory infiltrate mainly made of neutrophils occupies the lamina propria. Acute bacterial rhinosinusitis usually resolves with antibiotic therapy. Complications are rare and include contiguous infectious involvement of the orbit or central nervous system.

2.2.3 Allergic Rhinitis

Allergic rhinitis (hay fever) is part of an inherited syndrome, which may also manifest as atopic eczema and asthma. In allergic rhinitis, airborne particles, such as grass pollens, moulds and animal allergens, are deposited on the nasal mucosa giving rise to acute and chronic reactions. Allergens combine with the IgE antibodies produced by the plasma cells of the nasal mucosa, which are avidly bound to the Fc-epsilon receptors on mast cells. This triggers degranulation of mast cells and releases the inflammatory mediators of the type I hypersensitivity reaction, causing rhinorrhea and nasal obstruction. Microscopically, the nasal mucosa shows numerous eosinophils, abundant plasma and in some cases an increased number of mast cells. There is goblet cell...