

thrombotic tendencies or arterial atherosclerosis. Fresh frozen plasma infusion can be used for acute attacks in a setting that lacks access to newer modalities. Published studies are conflicting on the efficacy of bradykinin 2 receptor antagonists and C1INH protein in the treatment of ACE inhibitor–induced angioedema. Treatment of the underlying autoimmune disease or malignancy is indicated for acquired C1INH deficiency.

ALLERGIC RHINITIS

■ DEFINITION

Rhinitis is characterized by sneezing; rhinorrhea; obstruction of the nasal passages; conjunctival, nasal, and pharyngeal itching; and lacrimation and can be classified as allergic or nonallergic. A clinical history of rhinitis symptoms occurring in a temporal relationship to allergen exposure and documentation of sensitization to an environmental allergen are required for a diagnosis of allergic rhinitis. Although commonly seasonal due to elicitation by airborne pollens, it can be perennial in an environment of chronic exposure to house dust mites, animal danders, or insect (cockroach) products. The overall prevalence in North America has increased in the past 20 years and is 10–30%, with the peak prevalence of >30% occurring in the fifth decade.

■ PREDISPOSING FACTORS AND ETIOLOGY

Allergic rhinitis generally occurs in atopic individuals, often in association with atopic dermatitis, food allergy, urticaria, and/or asthma ([Chap. 287](#)). Up to 50% of patients with allergic rhinitis manifest asthma, whereas 70–80% of individuals with asthma and 80% of individuals with chronic bilateral sinusitis experience allergic rhinitis. Female sex, particulate air pollution exposure, and maternal tobacco smoking increase the risk of developing allergic rhinitis.

Wind-pollinated trees, grasses, and weeds produce sufficient quantities of pollen suitable for wide distribution by air currents to elicit seasonal allergic rhinitis. The dates of pollination of these species historically varied little from year to year in a particular locale