Evaluating and Managing the Patient with Nosebleeds

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KEYWORDS

• Epistaxis • Packing • Cautery • Ligation

Epistaxis is a common clinical problem that is estimated to occur in 60% of people worldwide during their lifetime.[1] It accounts for approximately 1 in 200 emergency department visits in the United States.[2] Although epistaxis can occur at any age, the peak ages of incidence include patients younger than 18 and those older than 50. The episodes that occur in the older population tend to be more severe, whereas those in children are more often minor and self-limited.

NASAL VASCULAR ANATOMY

Epistaxis is described as either anterior or posterior. The origin of epistaxis has important consequences for subsequent treatment; therefore, a complete understanding of the vascular anatomy of the nasal cavity is helpful in defining both origin and treatment of epistaxis. Anteriorly, the terminal branches of the sphenopalatine and anterior ethmoidal arteries, and the superior labial branch of the facial artery, supply an arterial anastomotic triangle known as Kisselbach's plexus, located in Little's area (**Fig. 1**); 90% to 95% of episodes of epistaxis arise from the anterior nasal septum.[3] The external carotid artery supplies the internal maxillary artery, which, after dividing into multiple branches, terminates in the sphenopalatine artery, which enters the nasal cavity just posterior to the maxillary sinus through the lateral nasal wall. The two most common branches of the sphenopalatine artery are the nasopalatine artery, which supplies the posterior nasal septum, and a posterior superior branch, contributing to the middle and inferior turbinates.[4] The remaining 5% to 10% of epistaxis arise from these more posterior vessels. The internal carotid artery supplies the mucosa of the lateral nasal wall above the middle turbinates via the anterior and posterior ethmoidal arteries, which are branches of the ophthalmic artery. Epistaxis caused by anterior or posterior ethmoidal artery bleeding is quite rare, and typically only occurs in trauma with associated skull base fracture. These vessels may also be injured during endoscopic sinus surgery.

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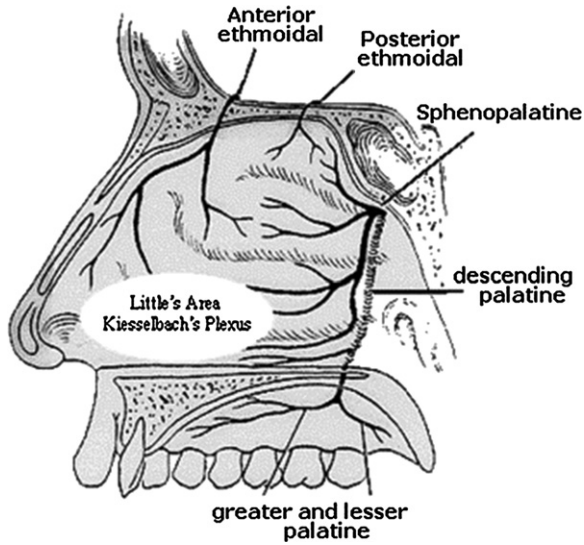

Fig. 1. Nasal septal vasculature. (*From* Viehweg TL, Roberson JB, Hudson JW. Epistaxis: diagnosis and treatment. J Oral Maxillofac Surg 2006;64:511–8; with permission.)

ETIOLOGY

The causes of epistaxis may be divided into local and systemic factors. Digital trauma is common, especially in the younger population. Mucosal dryness can cause episodes of epistaxis. Usually this results from low humidity or turbulent airflow secondary to septal deviations. Emergency room visits for epistaxis are reported to increase in the winter months, likely because of lower humidity during this time.[5] Chemical irritants may also lead to epistaxis. This may include both illicit nasal drug use and prescription topical nasal drugs, such as antihistamines or corticosteroids. Use of these commonly prescribed medications may result in epistaxis in 17% to 23% of patients using these products.[6] Patients should be instructed to direct the spray laterally, away from the septum, to decrease this occurrence. A simple way to achieve this is to have patients use their right hand to spray topical medicine into their left nasal cavity, and their left hand to spray medicine into their right nasal cavity. This decreases the likelihood that medication will be directed onto the nasal septum. Trauma can also lead to epistaxis. This is seen in the context of a nasal bone or septal fracture, and can be profound. Rhinosinusitis leads to increased inflammation and nose blowing, and may be a factor in episodes of epistaxis. One should consider the possibility of neoplasm as the inciting factor, especially if bleeding is persistent and no other cause is identified.

Systemic factors may also play a significant role in epistaxis. Hemophilia and von Willebrand's disease can lead to severe, difficult to control epistaxis. Thrombocytopenia, from either decreased platelet production or increased platelet breakdown, can lead to epistaxis. This includes, but is not limited to, hematologic malignancies, chemotherapy treatment, viral infections, and multiple autoimmune disorders. Chronic alcohol intake or renal failure can affect platelet function despite the presence of normal platelet counts.

Medication use can also contribute to epistaxis. Anticoagulants, such as coumadin, enoxaparin, and heparin all can be related to episodes of epistaxis. Nonsteroidal