Epistaxis

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KEYWORDS

• Epistaxis • Nosebleed • Nasal mucosa • Nasal packing • Hemostasis
• Emergencies

KEY POINTS

- Most epistaxis cases (80%–90%) are anterior and can be managed conservatively with topical therapies and/or proper compression techniques.
- Kiesselbach's plexus, located in the anteroinferior nasal septum, is a common area of epistaxis and is often the treatment target for anterior epistaxis.
- Local nasal trauma, mucosal dryness, systemic comorbidities, and certain medications are associated with an increased risk of epistaxis.
- Preventative management is crucial to reduce the frequency and severity of epistaxis.
- Patients should be referred to an otolaryngologist if they present with recurrent, severe, or posterior epistaxis that does not respond to initial management efforts.

INTRODUCTION

Epistaxis is a condition characterized by hemorrhage from the nostril, nasal cavity, or nasopharynx. Regardless of the underlying etiology, epistaxis results from a disruption in the integrity of the nasal mucosa and its associated vascular structures. The nasal cavity is anatomically predisposed to hemorrhage due to its robust vascular supply, which forms a dense capillary network essential for air filtration, humidification, temperature regulation, olfaction, and immune defense.[1] Epistaxis has been recognized for millennia, with references to its management dating back to Aradnana, a surgeon to the Assyrian court over 2500 years ago.[2] Originally known as "haemorrhagia of Hypocrates," it was not until the eighteenth century that the term epistaxis came into use.[3,4] It was also during this era that interest began to focus on the exact origin of the bleeding, with the Italian anatomist Antonio Valsalva attributing it to the anterior septum "*about a finger's breadth…from the bottom of the nostrils*" and advising direct compression with a finger to manage it.[5]

From an epidemiologic standpoint, up to 60% of the general population will experience epistaxis, with 10% seeking medical attention and 0.16% requiring hospitalization.[6] Epistaxis accounts for 0.5% of all emergency department (ED) visits

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Abbreviations	
CHF	congestive heart failure
CI	confidence interval
CKD	chronic kidney disease
CPAP	continuous positive airway pressure
DOACs	direct oral anticoagulants
ED	emergency department
HHT	hereditary hemorrhagic telangiectasia
INR	international normalized ratios
JNA	juvenile nasopharyngeal angiofibroma
LMWH	low-molecular-weight heparin
PT/INR	prothrombin time/international normalized ratio
RR	relative risk
SSRIs	selective serotonin reuptake inhibitors
TXA	tranexamic acid
vWD	von Willebrand disease

and up to 33% of otolaryngology-related ED encounters.[7,8] Its prevalence follows a bimodal age distribution. A 10 year review of the National Hospital Ambulatory Medical Care Survey found that the frequency of ED visits for epistaxis peaks in children (4 visits per 1000 population) and in adults aged over 70 years (12 visits per 1000 population).[8] Seasonal variation is also well-documented, with a notable increase during the winter months across geographic regions.[9–11] Additionally, a population-based cohort analysis of 4000 Medicare beneficiaries revealed an inverse correlation between ED visits for epistaxis and both ambient temperature and annual humidity.[12]

Epistaxis is associated with significant health care costs, from approximately US $1000 for an initial ED visit to almost US$60,000 for admissions requiring arterial embolization.[13,14] Delayed treatment can increase overall health care costs and lead to life-threatening complications.[14] Given that patients with epistaxis may present to various clinical settings, it is crucial for primary care providers to understand the fundamental principles of its management. This review serves as a practical primer on the diagnosis and treatment of epistaxis for the nonotolaryngologist.

DISCUSSION

Review of Anatomy

Unlike many head and neck regions, the nasal cavity receives a rich arterial blood supply from both internal and external carotid systems.[15] **Fig. 1** illustrates this vascular anatomy. Clinically, this complex network of anastomosing vessels is often divided into anterior and posterior circulations. Roughly 90% of epistaxis cases originate from the anterior circulation, with the remaining 10% arising from posterior sources.[16] While there is no strict anatomic boundary between anterior and posterior epistaxis,[17] Kiesselbach's plexus—an anastomotic network of up to 5 arteries located in the anteroinferior nasal septum, also known as Little's area (see **Fig. 1**)—is the most common site of anterior nosebleeds.[15,18] In contrast, posterior epistaxis often originates from arterial branches of the sphenopalatine and ascending pharyngeal arteries that supply the posterior septum or from venous structures such as Woodruff's plexus in the lateral nasal wall.[19,20]

Although both the septum and lateral nasal wall are highly vascularized, most epistaxis cases originate from the septum due to its thin mucosa and dense network of superficial vessels.[7,18] Recognizing these anatomic sources of hemorrhage can help clinicians promptly localize and treat the bleeding site.

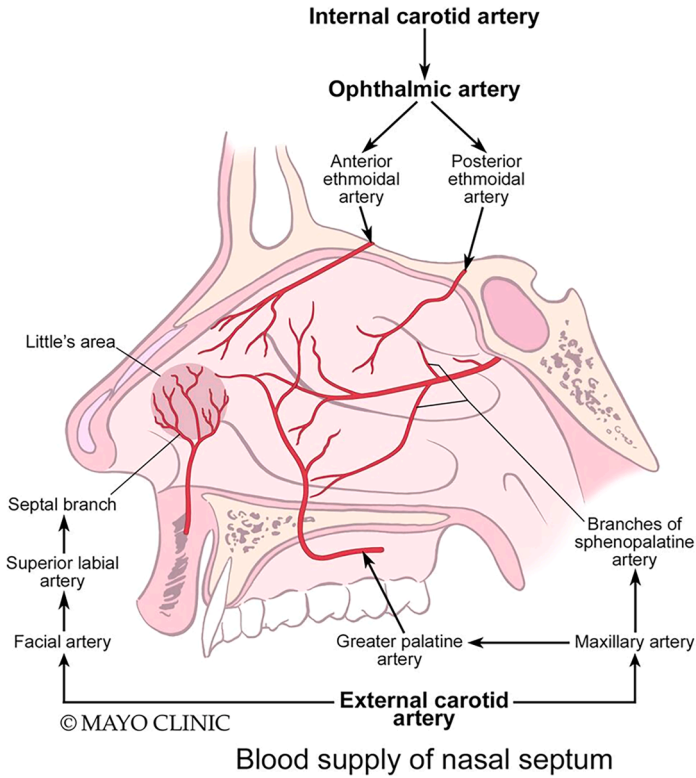

Fig. 1. Vascular anatomy of the nasal cavity. (*From*: Farnsworth PJ, Campeau NG, Diehn FE, Yu L, Leng S, Zhou Z, Fletcher JG & McCullough CH. High Resolution CTA of the Orbit using a Photon Counting CT Scanner. Interv Neuroradiol. 2023 Jul 3:15910199231175198; used with permission of Mayo Foundation for Medical Education and Research, all rights reserved.)

Risk Factors

While epistaxis can occur spontaneously, several local and systemic risk factors are well documented. Local factors include traumatic, inflammatory, neoplastic, structural, and iatrogenic conditions (**Table 1**). Inflammatory conditions like allergic rhinitis and chronic rhinosinusitis increase the risk of epistaxis, likely due to mucosal vascular changes associated with chronic inflammation.[9] Environmental factors such as lower humidity and colder temperatures—which can dry out the nasal mucosa—may also play an important role in the development of epistaxis.[9–11]

Systemic comorbidities affecting coagulation can also increase the risk and severity of epistaxis (see **Table 1**). In one retrospective study, 45.4% of patients admitted for epistaxis had a systemic condition, such as genetic, hepatic, renal, hematologic, or cardiovascular disease, which could potentially account for the episode.[21] Although uncontrolled hypertension has not been definitively established as a cause for nosebleeds, elevated blood pressure readings are often observed in patients presenting with epistaxis.[7,22] A study using the Korean National Health Insurance Service-National Sample Cohort found patients with hypertension had higher epistaxis incidence and were more likely to require ED visits and posterior nasal packing.[23] Similarly, a recent systematic review with a total of more than 9000 patients found an association between hypertension and epistaxis (odds ratio, 1.532; 95%

Table 1
Common risk factors for epistaxis

Category	Examples
Local causes	• Digital trauma • Facial trauma • Inflammatory conditions (eg, allergic rhinitis and sinusitis) • Anatomic irregularities (eg, deviated nasal septum and septal perforation) • Neoplastic conditions (eg, JNA and sinonasal malignancy) • Iatrogenic: nasal surgery, CPAP use, and nasal oxygen therapy
Environmental causes	• Cold weather • Low ambient humidity (dry air)
Systemic causes	• Hypertension • HHT • Inherited bleeding disorders (eg, hemophilia and vWD) • Hematologic malignancies • Chronic liver disease • CKD • CHF • Immune thrombocytopenia
Medication-related causes	• Vitamin K antagonists (eg, warfarin) • Heparin and LMWH • Antiplatelet agents (eg, aspirin and clopidogrel) • DOACs: dabigatran, rivaroxaban, and apixaban • Topical nasal steroids or antihistamines

Abbreviations: CHF, congestive heart failure; CKD, chronic kidney disease; CPAP, continuous positive airway pressure; DOACs, direct oral anticoagulants; HHT, hereditary hemorrhagic telangiectasia; JNA, juvenile nasopharyngeal angiofibroma; LMWH, low-molecular-weight heparin; vWD, von Willebrand disease.

confidence interval [CI], 1.181–1.986).[24] Interestingly, the association was present in case–control studies but not in cohort studies, supporting the conclusion that further prospective studies are needed to determine causation.

Finally, medications are another important contributor to epistaxis (**Table 2**). Mucosal trauma from intranasal corticosteroid sprays may result in epistaxis in approximately 20% of patients using these products.[25,26] However, the risk of epistaxis depends on the specific formulation used. A meta-analysis examining the safety and adverse events of intranasal corticosteroid therapy found that the relative risk (RR) of epistaxis was significantly higher for drops (RR 4.23, 95% CI: 1.81–9.85) compared to spray formulations (RR 1.40, 95% CI: 1.01–1.95).[27] Anticoagulant and antiplatelet medications are also well-documented risk factors for epistaxis. The use of warfarin, aspirin, and clopidogrel has been shown to increase the risk of recurrent nosebleeds,[28,29] while novel oral anticoagulants, such as apixaban and rivaroxaban, are associated with lower rates of epistaxis compared to warfarin.[30,31] Other medications, such as selective serotonin reuptake inhibitors (SSRIs), have been associated with an increased risk of epistaxis likely due to their effect on platelet function.[29]

Workup

It is often forgotten that in a minority of patients, epistaxis can result in potentially life-threatening airway obstruction or hemodynamic instability. The first step in evaluation should always be assessment of airway patency and hemodynamic status, especially

Table 2
Medications associated with epistaxis

Medication Class	Examples	Mechanism	Clinical Considerations
Anticoagulants	Warfarin, heparin, DOACs (dabigatran, rivaroxaban, and apixaban)	Impaired coagulation cascade leading to increased bleeding risk	Consider PT/INR monitoring; evaluate for dose adjustment or reversal agents if bleeding is severe
Antiplatelet agents	Aspirin and clopidogrel	Inhibited platelet aggregation delays hemostasis	Weigh bleeding risk against thrombotic risk; consider holding if appropriate
Intranasal corticosteroids	Fluticasone propionate or furoate, mometasone furoate, and triamcinolone acetonide	Mucosal thinning and local irritation	Instruct patients to aim spray away from septum; consider switching formulations
SSRIs	Sertraline, fluoxetine, and citalopram	Impaired platelet aggregation due to decreased serotonin uptake	Encourage preventive measures

Abbreviations: DOACs, direct oral anticoagulants; PT/INR, prothrombin time/international normalized ratio; SSRIs, selective serotonin reuptake inhibitors.

in cases of active bleeding.[7] Any concern for compromise should be immediately referred for emergency evaluation. Once life-threatening conditions are ruled out, a focused history and physical examination can take place. Having adequate suction available during the evaluation is essential. For patients with active bleeding, an often effective, noninvasive, and low-cost intervention is nasal compression.[7] The correct technique is illustrated in **Fig. 2**A–C. The history should include information on duration, laterality, frequency, and severity of bleeding, along with any prior episodes and treatments. Comorbidities, medications, triggers, and symptoms that may indicate acute hypovolemia (ie, syncope, tachycardia, and orthostatic hypotension) should also be documented.[7]

Anterior rhinoscopy is a procedure that allows visualization of the anterior one-third of the nasal cavity using equipment readily available in most outpatient offices and EDs, such as a headlamp or otoscope and a nasal speculum.[32] This procedure can help determine the bleeding site, laterality, and whether the source is anterior or posterior.[7,33] Anterior rhinoscopy also allows the application of directed therapy (ie, topical vasoconstrictors or cautery) to the suspected bleeding site (**Fig. 3**A, B).[34,35] Transoral inspection of the back of the throat is important to identify posterior drainage of blood, which can suggest a posterior bleed. Some patients benefit from additional visualization of posterior nasal structures with nasal endoscopy. This includes those with bleeding refractory to packing or cautery, persistent postnasal bleeding seen along the back of the throat, recurrent unilateral epistaxis, or symptoms suggestive of unrecognized nasal pathology, such as unilateral nasal obstruction.[7,36–38] Studies have found that persistent or recurrent epistaxis is more common in patients with unidentified or posterior bleeding sites, which cannot be reliably detected with anterior rhinoscopy.[39–41] Nasal endoscopy, typically performed by an otolaryngologist, can localize the bleeding site in approximately 90% of cases and allows for targeted intervention.[42] Importantly, any clinical scenario that warrants endoscopic evaluation should prompt referral to otolaryngology.

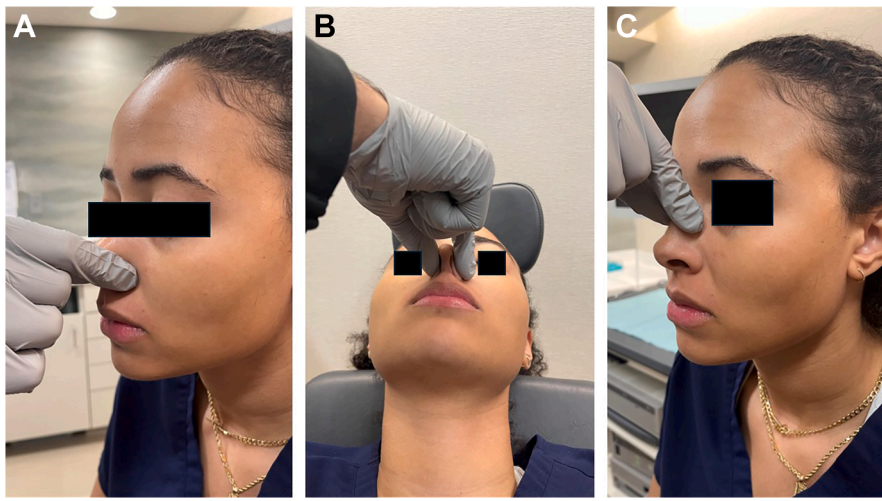

Fig. 2. Correct and incorrect techniques for nasal compression. (*A*) Shows proper compression of the soft, lower third of the nose. (*B*) Provides an inferior view, illustrating correct compression of the nasal ala against the septum. (*C*) Demonstrates incorrect technique, with pressure applied too high on the nasal bones

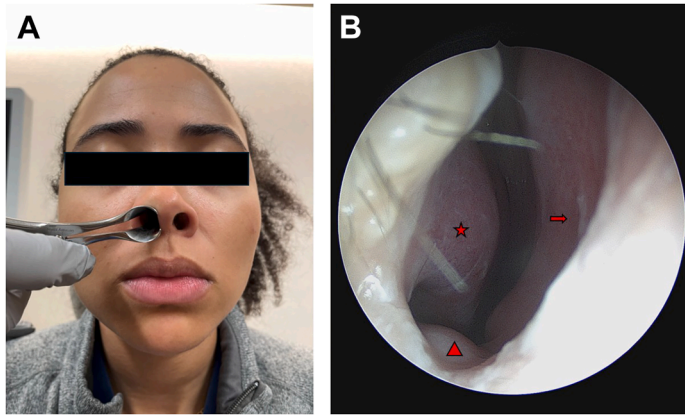

Fig. 3. (*A*) Shows the proper technique for phalange placement during anterior rhinoscopy. (*B*) Displays intranasal endoscopy highlighting nasal structures visible during anterior rhinoscopy. Endoscopic images of the right nasal cavity are shown for illustrative purposes. *Star* indicates the inferior turbinate; *arrow*, the nasal septum; and *triangle*, the nasal floor.

Laboratory testing should be reserved for specific indications and not performed routinely. Routine coagulation screening has limited value and increases treatment costs and ED occupancy times.[21] However, testing should be considered in patients on anticoagulants and those with bleeding disorders or chronic liver conditions.[21,40] Finally, imaging should be reserved for suspected sinonasal tumors, foreign bodies, or other anatomic abnormalities that cannot be identified on physical examination.[7,40,43]

Diagnosis

At the time of initial consult, the first goal of the clinician should be to distinguish patients who require prompt management and stabilization (eg, active and profuse bleeding) from those who do not (eg, minor or nonactive bleeding).[7] Similarly, because hemostasis is more difficult to achieve in posterior epistaxis, early differentiation between anterior and posterior bleeding is essential and should guide treatment strategy.[39,44]

Providers should also recognize characteristic clinical presentations that may indicate a more serious underlying pathology and require further workup. The first is an adolescent male with persistent or recurrent unilateral epistaxis that may or may not be accompanied by nasal obstruction. This presentation should raise suspicion for juvenile nasopharyngeal angiofibroma (JNA), a benign but locally invasive vascular tumor.[45] The second scenario is a patient with progressive and persistent epistaxis, accompanied by unilateral nasal obstruction, weight loss, and systemic symptoms. This scenario should raise suspicion for malignancy and prompt imaging.[46] Finally, in a patient with a personal or family history of spontaneous, recurrent nosebleeds, clinicians should assess for nasal or oral mucosal telangiectasias, which may be indicate hereditary hemorrhagic telangiectasia (HHT).[7,47]

Treatment

First-line management (outpatient)

Epistaxis management should follow a stepwise approach, escalating from least to most invasive interventions. **Table 3** summarizes first-line management options and key clinical pearls. For patients with active bleeding, they should be positioned

Table 3
First-line management options and clinical pearls

Intervention	Details	Clinical Pearl
Nasal compression	5–15 min using a clock; lower third of the nose	Premature release is a common cause of treatment failure
Topical vasoconstrictors	Oxymetazoline, phenylephrine, and epinephrine	Use with caution in patients with cardiac, hypertensive, or stroke history
Nasal cautery	Silver nitrate to visible bleeding site	Use local anesthetic; avoid bilateral cauterization
Nasal packing	Resorbable or nonresorbable based on risk of rebleeding and patient tolerance; follow manufacturer instructions	Insert along nasal floor; educate on complications
TXA	Topical or oral antifibrinolytic agent; used adjunctively with usual care	Shown to reduce risk of rebleeding

in the "sniffing position" with the head slightly flexed and a slight lean forward. The clinician should then instruct the patient to apply sustained compression to the lower third of the nose, below the nasal bones, by pinching the nasal alae against the septum using the thumb and index finger (see **Fig. 2**).[36] Currently, there is no consensus on the optimal duration of nasal compression. The American Academy of Family Physicians recommends continuous pressure for 10 to 15 minutes,[44] while the French Otorhinolaryngology-Head and Neck Surgery Society advises a minimum duration of 10 minutes.[36] In contrast, the American Academy of Otolaryngology-Head and Neck Surgery recommends a minimum of 5 minutes.[7] Regardless of the specific recommendation followed, compression time should be measured with a clock rather than relying on estimation, as discomfort and anxiety often lead patients or providers to terminate this simple yet highly effective therapy prematurely.

While one randomized controlled trial suggested alar clips may be more effective than manual compression,[48] the overall quality of evidence is low and insufficient to recommend this therapy over digital compression.[7,49] Additionally, use of these devices should be weighed against potential complications, including pain, discomfort, and pressure-related complications (eg, alar necrosis and septal perforation).

Adjunct therapies such as topical vasoconstrictors (eg, oxymetazoline, phenylephrine, and epinephrine), emollients, and nasal cautery may be used at this time, especially when a bleeding site is visible on anterior rhinoscopy.[7,36] Although the impact of alpha-1-adrenergic-agonists on blood pressure and cardiovascular risk is not well documented in patients with epistaxis, caution should be exercised when using these therapies on patients with pre-existing hypertensive, cardiac, or cerebrovascular conditions.[7]

Chemical cautery with silver nitrate is simple, effective, and should be preceded by topical anesthetic (eg, lidocaine or tetracaine).[50] Electrocautery is also effective but requires specialized equipment and technical expertise. While the evidence supporting an increased risk of septal perforation following bilateral cauterization remains limited,[36,51] various guidelines recommend using direct visualization and limiting application to identified bleeding sites.[7,36]

If bleeding persists despite compression, or no bleeding site is visible on physical examination, nasal packing should be considered.[7,36] There are various types of

packing options available, with limited evidence to support the use of one specific material.[7] Packing should be inserted along the nasal floor in a horizontal trajectory and following manufacturer instructions.[16] The choice between resorbable and nonresorbable packing should be based on availability, patient/clinician preference, and patient factors that may complicate packing removal by increasing the risk of rebleeding (eg, bleeding disorders, anticoagulation, or increased patient discomfort).[7] Primary care providers with adequate resources (ie, headlight, nasal speculum, packing material, and lubricant) may perform anterior nasal packing in the outpatient office or ED, while posterior packing requires otolaryngology consultation.[7] Regardless of the chosen packing type, clinicians should educate patients on complications, warning signs of infections, and provide follow-up instructions for postprocedural care and/or removal.[7] Potential complications include discomfort, mucosal injury, infection, pressure-related injuries (eg, septal perforation), and airway obstruction.[7,52] Packing is typically left in place for 48 to 72 hours, and no longer than 5 days, to mitigate complication risk.[7,44] Packing should be avoided in patients with facial or skull base fractures.[53] Routine use of systemic antibiotics to prevent infectious complications is not supported by current literature, and their use should be individualized based on patient risk factors and overall health.[7,54,55]

Tranexamic acid (TXA) is an antifibrinolytic agent that has gained popularity in recent years to control epistaxis. A Cochrane review found moderate-quality evidence that both oral and topical TXA in addition to usual care reduce the risk of rebleeding in adult patients with epistaxis compared to placebo with usual care.[56] Additionally, recent meta-analyses have demonstrated that topical TXA significantly reduces rebleeding rates compared to standard treatment and traditional nasal packing.[57,58] Although additional studies are needed to understand its efficacy and indications, current evidence supports TXA use as a valuable adjunct in epistaxis management.

Second-line management (inpatient)

While the vast majority of epistaxis cases resolves with first-line treatment, approximately 6% of patients require more advanced interventions for persistent or recurrent episodes.[7] Historically, prolonged posterior nasal packing for 2 to 7 days, was recommended as second-line treatment of intractable epistaxis. However, this approach is associated with significant discomfort, prolonged hospitalization, high rebleeding rates, and major cardiopulmonary complications.[59–61] Excellent success rates and reduced morbidity have allowed surgical and endovascular techniques to become commonplace in the management of intractable epistaxis.[62] Currently, those who fail first-line measures should be referred to otolaryngology for further evaluation.[7] Second-line options include endoscopic sphenopalatine artery ligation and endovascular embolization, among others. However, a discussion of surgical and procedural techniques to address persistent and severe epistaxis is outside of the scope of this study.[62]

Preventive Management

Preventative management is crucial to reduce the frequency and severity of epistaxis, ultimately leading to improved patient outcomes and quality of life.[63] Clinicians should counsel patients to avoid nasal trauma, including nose picking and vigorous nose blowing, which are common triggers. In patients with additional risk factors, such as those using nasal oxygen or continuous positive airway pressure (CPAP), the use of moisturizers, lubricants, and humidifiers can help maintain mucosal hydration and

prevent bleeding.[7] Finally, optimizing control of underlying conditions, including hypertension and supratherapeutic international normalized ratios (INR) in patients on anticoagulants, is essential to minimizing bleeding risk.[64,65]

When to Refer to Otolaryngology?

Patients should be referred to otolaryngology if they experience recurrent or severe epistaxis that does not respond to initial measures such as nasal compression, packing, cautery, or topical vasoconstrictors. While there is no standard definition for "severe epistaxis," evidence or suspicion of prolonged or profuse bleeding, bilateral bleeding, bleeding from the mouth/posterior site, signs of acute hypovolemia, suspicion of underlying nasal pathology, and presence of comorbidities such as anemia, bleeding disorders, hypertension, cardiopulmonary disease, liver or kidney disease, should prompt referral to otolaryngology.[7,44,66] Additionally, other patient-related factors such as multiple recent bleeding episodes or the need for hospitalization or blood transfusion should also be considered for otolaryngology referral.[7,40]

SUMMARY

Epistaxis is a common yet potentially life-threatening condition that has been recognized and treated for millennia. Most episodes originate from the richly vascularized anterior nasal septum and respond well to conservative measures such as nasal compression, cautery, and topical vasoconstrictors. A focused assessment—including evaluation of airway and hemodynamic status, identification of the bleeding source, and recognition of contributing risk factors—is essential to guide management. Persistent, recurrent, or posterior bleeding, as well as cases associated with significant systemic comorbidities, may require specialist referral and second-line interventions. A stepwise, evidence-based approach can help clinicians to manage this condition confidently across both outpatient and inpatient settings.

CLINICS CARE POINTS

- The majority of epistaxis cases originates from the anteroinferior nasal septum and responds well to conservative, first-line management.
- First-line management includes sustained compression of the lower third of the nose—the soft, cartilaginous area below the nasal bones—using the thumb and index fingers.
- Premature interruption of nasal compression is a common cause of treatment failure. Compression should be sustained for 5 to 15 minutes, using a clock rather than estimation to ensure accurate timing.
- In cases of active bleeding, the assessment of airway compromise and hemodynamic stability should take precedence over all other interventions. Having suction available is essential.
- Inability to identify a bleeding source on anterior rhinoscopy, the presence of bilateral bleeding, bleeding into the throat, or failure to respond to conservative measures suggests posterior epistaxis and warrants referral to an otolaryngologist (ear, nose, and throat specialist).
- Routine coagulation studies should be avoided, except in patients receiving anticoagulant therapy, those with known bleeding disorders, or individuals with chronic liver disease.
- There is currently limited evidence supporting the use of one packing material over another. Packing should be inserted along the nasal floor and following the manufacturer's instructions.

- Regardless of the type of packing used, clinicians should educate patients about potential complications, warning signs of infection, and provide clear follow-up instructions for postprocedural care and removal.
- Prolonged posterior packing is no longer the preferred second-line treatment. Patients with persistent or recurrent epistaxis unresponsive to first-line management should be referred for surgical ligation or endovascular embolization.

DISCLOSURE

The authors have nothing to disclose.

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