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Review Article

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Epistaxis: An Updated Review

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Abstract

Epistaxis is a common clinical condition in the general population. While it is often benign and resolves spontaneously, it can sometimes be severe due to its frequency, recurrence, etiology, or the patient's underlying comorbidities.

Management must be cautious, tailored to each individual case, and gradual following the principle of therapeutic escalation,and may sometimes require a multidisciplinary approach.

General practitioners are often on the front lines, playing a crucial role in diagnosis, assessing severity, and identifying cases that require specialist referral. Recent data on epistaxis have revealed the emergence of new etiologies (such as toxic and inflammatory diseases) and, most importantly, a shift in management strategies toward atraumatic treatments using modern hemostasis techniques, including bioresorbable agents and procoagulant balloons. Surgical ligation of the sphenopalatine artery and embolization remain valuable, complementary treatments for severe, refractory, or patient-specific epistaxis.

Keywords: Epistaxis, Embolization, Kisselbach Capillaritis, Arteriography, Nasopharyngeal Carcinoma, Resorbable Packing.

I. Introduction

Epistaxis is defined as the externalization of blood from the nose, originating within the nasal cavities, paranasal sinuses, or nasopharynx. The global prevalence of epistaxis is 21.7%, with a formal hospitalization rate of 11.8% observed among pediatric and geriatric populations. Furthermore, the lifetime risk of developing at least one episode of epistaxis is estimated at 60%. Although the vast majority of cases resolve spontaneously, only 6% of individuals experiencing an episode seek medical attention [1].

While epistaxis represents a frequent clinical occurrence in daily medical practice and is predominantly benign, it can rapidly escalate into a critical medical-surgical emergency. This severity is typically driven by profuse hemorrhage, high recurrence rates, or underlying systemic patient fragility.

Primary or idiopathic epistaxis constitutes the most frequent clinical presentation. Consequently, the initial clinical approach must focus primarily on ruling out severe underlying pathologies, such as malignant neoplasms, vascular disorders, or hematological diseases.

The comprehensive management framework relies on three consecutive pillars: assessing the clinical severity of the hemorrhage, achieving definitive hemostasis through a stepped care escalation protocol (ranging from the least to the most invasive interventions), and conducting a thorough etiological investigation.

Current international guidelines favor non-invasive, targeted therapeutic methods. Topical tranexamic acid, bioresorbable hemostatic fluids, absorbable packing materials, procoagulant balloons, and endoscopic sphenopalatine artery ligation form the cornerstones of this modern approach.

Finally, structured patient health education, particularly targeted toward pediatric cohorts and their families, is essential to mitigate unnecessary emergency department visits and prevent long-term recurrences.

Standardized clinical practice guidelines are highly desirable to harmonize and update epistaxis management strategies across multidisciplinary networks, including emergency physicians, otolaryngologists, and family practitioners, in strict accordance with modern diagnostic and therapeutic advances.

2. Vascularization of the Nasal Cavities

While classic anatomical data remain unchanged, establishing that the nasal cavities depend on a rich anastomotic network supplied by both the external carotid (sphenopalatine artery) and internal carotid (ethmoidal arteries) systems, recent research has refined our understanding of this vascular bed.

Microscopic studies have identified unique, large-diameter "venous sinusoids" characterized by Prox1 protein expression. These specialized vessels fill with blood to regulate the nasal cycle, and their walls feature specific cell junctions adapted to rapid, massive blood flow designed to instantly warm inhaled air [2].

Chronic inflammation or infection, such as allergic rhinitis, damages these Prox1(+) venous sinusoids, leading to a loss of airflow regulation. Furthermore, aging causes a loss of muscular support within the vessel walls, inducing abnormal dilation and chronic nasal congestion [3].

3. General Epidemiology of Epistaxis

Literature regarding the exact incidence of epistaxis in general practice, emergency departments, and the general population remains scarce, particularly concerning demographic variables like age, sex, and environmental factors.

incidence of epistaxis requiring hospitalization to achieve hemostasis is estimated at 42 cases per 100,000 in men and 28 cases per 100,000 in women.

In Thuringia, this rate reaches a significant peak of 222 cases per 100,000 in men over 85 years of age (3).

Overall, only 6% to 10% of individuals experiencing epistaxis seek medical attention. Age distribution typically displays a bimodal pattern, peaking in children under 10 years and adults between 50 and 70 years, with a male predominance observed in approximately 68% of cases [4].

Regarding ethnicity, some publications report a higher prevalence among Caucasians compared to African American populations [5]. Additionally, a predominance of patients from rural origins has been noted.

In a landmark 10-year nationwide analysis of emergency department (ED) data in the United States, Pallin et al. (2005) identified 4,503,000 ED visits for epistaxis, accounting for 0.46% of all consultations (6). This study confirmed the bimodal age distribution, with peaks under 10 years and between 70 and 79 years. Non-traumatic etiologies were predominant (83%), presenting an older average age (49 years) compared to traumatic causes (31 years).

Furthermore, seasonal variations were evident: non-traumatic epistaxis occurred in 0.50% of winter consultations (December to February) compared to 0.34% during non-winter months. Within this cohort, 15% of patients required ambulance transport, and 6% necessitated hospitalization [6].

4. Clinical Diagnosis of Epistaxis

The investigation of epistaxis in the acute phase (hemorrhage) and especially later (after hemostasis) relies on three key elements: nasal endoscopy, facial imaging, and laboratory testing.

4.1 Nasal Endoscopy: This allows for a comprehensive examination of the nasal cavities, pinpoints the location of the bleeding and its nature (flooding) or localized, and sometimes contributes to the diagnosis (angiomas, tumors of the nasal cavities) or even to the therapeutic procedure (coagulation, cauterization, vascular ligation under endoscopic guidance).

Endoscopy is sometimes impossible to perform in cases of significant bilateral bleeding. In these cases, it is postponed until after hemostasis (2 to 3 days). Endoscopy requires simple preparation: aspiration of clots and application of a local vasoconstrictor (5% lidocaine naphthazoline). It can be done indifferently with a rigid optic (0 or 30°) or a flexible fiberscope.

Clinical diagnosis of epistaxis relies on three key pillars, nasal endoscopy, imaging, and laboratory tests, performed during or after the acute hemorrhage. Nasal endoscopy allows for precise localization, etiological diagnosis, and therapeutic intervention (cauterization/ligation), though it may be postponed for 2 to 3 days in cases of severe, bilateral bleeding.

4.2. Imaging: The role of imaging is primarily etiological and is typically performed during the interictal phase. However, severe epistaxis occurring under specific circumstances, such as a suspected internal carotid artery aneurysm or a carotid-cavernous fistula, necessitates immediate, first-line radiological investigation.

Diagnostic angiography visualizes the specific arterial pedicles supplying the hemorrhage and enables concurrent endovascular embolization. Depending on the clinical presentation and suspected etiology, imaging modalities include computed tomography (CT) with CT angiography, magnetic resonance imaging (MRI) with magnetic resonance angiography (MRA), or conventional digital subtraction angiography [7].

4.3. Laboratory Tests: Routine laboratory evaluations are frequently performed to assess the systemic impact of the hemorrhage and to screen for primary hemostasis or coagulation disorders.

This initial panel generally includes blood typing, hemoglobin and hematocrit levels, platelet count, and a standard coagulation profile (Prothrombin Time/International Normalized Ratio and Activated

Partial Thromboplastin Time). If these baseline tests reveal abnormalities, a specialized hematological workup involving more complex, targeted assays is indicated.

5. Clinical Varieties of Epistaxis

Depending on the severity of the hemorrhage, epistaxis is conventionally classified into two distinct clinical presentations: benign and severe.

5.1. Benign Epistaxis

This is the most common clinical scenario, typically presenting as a sudden flow of bright red blood from a single nostril. The bleeding resolves either spontaneously or with simple digital compression, and the patient's hemodynamic status remains entirely stable.

Endonasal examination frequently identifies a localized source of bleeding within the anterior vascular network (Kiesselbach's plexus). This presentation rarely requires extensive investigation, baseline hemostasis testing is occasionally performed for reassurance, and management focuses primarily on patient education regarding nasal hygiene and local topical treatments.

5.2. Severe Epistaxis

Severe epistaxis is defined by profuse or recurrent bleeding. The hemorrhage is typically massive from the onset, bilateral, and presents with concurrent anterior flow and posterior oral expulsion. It is accompanied by signs of hemodynamic instability and poor systemic tolerance, including mucocutaneous pallor, severe asthenia, tachycardia with a weak pulse, and arterial hypotension. This critical scenario demands immediate resuscitation alongside local management. The patient must be placed in a semi-sitting position to prevent blood aspiration, intravenous access must be established promptly, and hemostatic measures initiated. Hemostasis is then tailored on a case-by-case basis, guided by the suspected or identified etiology.

6. Clinical Assessment of Severity

Assessing the severity of epistaxis is a critical step that dictates the urgency of clinical interventions, the escalation of hemostatic procedures, and the necessity for hospitalization or specialized care (intensive care unit admission or interventional radiology). The primary challenge in this assessment lies in the lack of objective, consensus-based, and reproducible standardized criteria.

Clinical indicators of severity can be categorized into four main dimensions:

- Hemodynamic and Systemic Shock: Signs of hypovolemic shock, including systolic blood pressure below 80 mmHg, shallow tachypnea, oliguria or anuria, psychomotor agitation, anxiety, confusion, cold extremities, and skin mottling.

- *Clinical Context and Precipitating Factors*: Epistaxis resulting from severe maxillofacial trauma, post-surgical complications, or occurring in patients with pre-existing congenital or acquired coagulopathies.

- *Underlying Comorbidities*: Associated conditions that lower the threshold for decompensation, such as coronary artery disease, congestive heart failure, severe chronic respiratory failure, brittle diabetes, advanced age, or major cognitive impairment.

Prognostic Severity Indicators: Specific clinical markers predictive of a worse outcome, which include:
-Persistent or recurrent bleeding episodes spanning over the preceding 15 days.
-Recurrent hemorrhage following technically sound nasal packing.
-Hemoglobin levels dropping below 10 g/dL (or below 8 g/dL according to some protocols), or signs of poor hemodynamic tolerance.
-Underlying severe medical conditions, such as advanced hepatic insufficiency or hematological malignancies.

Coagulation Anomalies and Antithrombotic Therapy: Underlying hemostasis disorders, active use of antithrombotic medications, and an elevated International Normalized Ratio (INR).

Vascular Fragility and Genetic Predispositions: Known hereditary hemorrhagic disorders, such as Hereditary Hemorrhagic Telangiectasia (Rendu-Osler-Weber disease).
Advanced Intervention Requirements: Cases necessitating complex hemostatic procedures, such as the placement of a double-balloon catheter.

Social and Environmental Constraints: Situations where safe or reliable home monitoring is unfeasible due to patient isolation or cognitive limitations.

We have proposed a simple and reproducible severity assessment score for epistaxis, the main interest of which is its practicality (Table 1) [8].

EPISTAXIS SEVERITY EVALUATION CRITERIA.

CLINICAL CRITERIA (5 POINTS).
-Duration: Epistaxis lasting longer than 24 hours.
-Onset and Flow: Sudden, bilateral, and posterior hemorrhage with pharyngeal expulsion.
-Systemic Tolerance: Hemodynamic instability characterized by hypotension, cutaneous pallor, and tachycardia.
-Age: Elderly patient aged over 70 years.
-Frequency: Recurrent episodes exceeding 3 recurrences per day.

BIOLOGICAL CRITERIA (4 POINTS).
-Hematocrit: Hematocrit level below 24%.
-Hemoglobin: Hemoglobin level below 6 g/dL in healthy individuals, or below 8 g/dL in patients with significant comorbidities.
-Platelet Count: Severe thrombocytopenia with a platelet count below 50,000/µL.
-Coagulopathy: Severe, laboratory-confirmed coagulation disorders.

GENERAL CRITERIA (5 POINTS).
-Iatrogenic Factors: Drug-induced hemostasis disorders (anticoagulants, antiplatelet therapies).
-Hypertensive Emergency: Malignant hypertension with a diastolic blood pressure exceeding 120 mmHg.
-Vascular Pathology: Structural vascular rupture or known vascular anomalies
(aneurysms, HHT).
-Refractory Hemorrhage: Failure of standard hemostatic interventions, including anterior/posterior packing or balloon catheterization.
-Comorbidities: Severe underlying systemic conditions, including coronary artery disease, brittle diabetes, or renal failure.

CLINICAL SCORING SYSTEM
-Mild Epistaxis (Score bellow to 2): Indication for outpatient management and home discharge.
-Moderate Epistaxis (Score 3–7): Indication for standard hospitalization or day-hospital monitoring, depending on clinical tolerance.
-Severe Epistaxis (Score 8–14): Mandatory admission to a specialized unit or intensive care, requiring advanced interventions such as interventional radiology or surgical ligation.

Table 1: Epistaxis Severity Scoring System [8].

7. Etiology of Epistaxis

The classic causes of epistaxis are well-documented, presenting with variable frequencies and severities, and are conventionally categorized into local and systemic etiologies. Local etiologies include trauma (nasal bone and midfacial fractures, sinonasal surgery, or iatrogenic trauma from diagnostic and therapeutic endonasal procedures), infectious or inflammatory rhinological conditions, foreign bodies, and benign or malignant neoplasms (such as juvenile nasopharyngeal angiofibromas, hemangiomas, undifferentiated nasopharyngeal carcinoma -UNCC, and sinonasal carcinomas). Systemic etiologies encompass infectious and autoimmune inflammatory diseases, hematological disorders (congenital and acquired coagulopathies, hematological malignancies), and vascular or metabolic conditions (severe hypertension, chronic kidney disease).

Despite this extensive list, idiopathic forms remain the most frequently encountered in clinical practice.

In addition to these traditional pathologies, recent etiological discoveries have expanded the spectrum of potential causes of epistaxis and warrant careful systematic investigation:

1. Iatrogenic Effects of modern oncological therapies: The advent and widespread clinical use of targeted therapies and immunotherapies have led to novel hemorrhagic side effects. Angiogenesis inhibitors, such as Bevacizumab, alter the nasal microcirculation and weaken capillary networks, inducing recurrent and sometimes severe epistaxis. Furthermore, modern immune checkpoint inhibitors can provoke bleeding by inducing localized nasal vasculitis or secondary immune thrombocytopenia.

2. Direct Oral Anticoagulants (DOACs): The widespread prescription of DOACs
(Rivaroxaban, Apixaban) frequently causes sudden-onset, posterior epistaxis. Unlike vitamin K antagonists, standard monitoring parameters like the International Normalized Ratio (INR) cannot assess their activity. While emergency reversal strategies exist, including nonspecific prothrombin complex concentrates (PCC) or specific antidotes like Andexanet alfa (for factor Xa inhibitors) and Idarucizumab (for dabigatran),management remains highly challenging in emergency settings.

3. Dyslipidemia and arterial stiffness: Recent clinical data strongly correlate non-septal hemorrhages (originating from the

lateral or posterior walls of the nasal cavity) with dyslipidemia and increased arterial stiffness (9). Atherosclerotic remodeling in these areas impairs local vascular vasoconstriction following mucosal rupture.

4. Latrogenic mucosal trauma from Non-Invasive Ventilation: The increasing global prevalence of obstructive sleep apnea (OSA) has led to widespread use of Continuous Positive Airway Pressure (CPAP) and high-flow nasal oxygen therapies. The constant airflow delivers direct micro-trauma via the nasal cannula and induces severe mucosal drying, significantly elevating the risk of epistaxis.

5. Topical nasal therapies: Similarly, the widespread and chronic use of nasal corticosteroid sprays triggers epistaxis through an identical mechanism of localized mucosal irritation and mucosal drying [10].

6. Herbal medicine and dietary Supplements: the growing global trend toward self-prescribed phytotherapy and nutritional supplements significantly influences bleeding risks. Several widely used substances possess antiplatelet or anticoagulant properties often unrecognized by patients, which can dangerously potentiate standard antithrombotic drugs like aspirin. Notably, high doses of Ginkgo Biloba, Ginseng, concentrated Turmeric, and vitamin E are documented causes of mucosal hemorrhage [11].

7. Synthetic substance abuse: An increasing number of young patients present to ENT emergency departments with sudden, severe epistaxis caused by acute chemical necrosis and septal perforation from intranasal drug abuse. Beyond classic cocaine use, contemporary clinical assessments must account for new synthetic psychoactive substances (NPS) taken via insufflation "snorting", including synthetic cathinones, ketamine derivatives, crystal meth, and highly caustic chemical cutting agents [11].

8. Environmental and lifestyle factors: Indoor air pollution and the intensive use of modern heating and air conditioning systems without integrated humidifiers aggressively dehydrate the nasal mucosa. This prolonged exposure renders the anterior vascular network (Kiesselbach's plexus) extremely friable and highly susceptible to spontaneous rupture [9].

8. Risk Factors for Epistaxis

Analyzing the specific risk factors for epistaxis is crucial for predicting clinical outcomes, optimizing early diagnostic strategies, preventing recurrences, and identifying potentially high-risk cohorts. Comprehensive clinical trials and literature reviews have firmly established the primary risk factors as follows:

-Cardiovascular and Metabolic Comorbidities: Advanced coronary artery disease or thromboembolic disorders requiring active antithrombotic therapies (anticoagulants or antiplatelet agents).

Anatomical Variations*:* The presence of a severe septal deviation, which disrupts normal laminar nasal airflow, induces localized turbulence, and creates a state of persistent mucosal inflammation and structural fragility.

-Surgical History: A documented history of recent or complex functional endoscopic sinus surgery (FESS) or rhinoplasty.

Traumatic Factors: Direct accidental maxillofacial or nasal trauma, as well as digital micro-trauma (nose picking).

Respiratory Support Devices: Routine use of supplemental oxygen via a nasal cannula or a Continuous Positive Airway Pressure (CPAP) system for obstructive sleep apnea.

Topical Therapies: Chronic administration of local nasal treatments, particularly corticosteroid or vasoconstrictor sprays.

Systemic Pharmacotherapy: Active use of antithrombotic medications (anticoagulants or antiplatelet agents).

Hematological Disorders: Congenital or acquired platelet function disorders and thrombocytopenia.

End-Stage Systemic Pathologies: Chronic kidney disease (CKD) and severe liver failure, which inherently impair standard coagulation pathways.

9. Epistaxis and Antithrombotic Therapy

Epistaxis induced by antithrombotic medications, encompassing both anticoagulants and antiplatelet agents, represents an increasingly common etiology for emergency department (ED) consultations.

The active use of these therapies substantially elevates the risk of severe epistaxis, often requiring aggressive and escalated hemostatic interventions. In the United States, an estimated 8 million individuals are currently prescribed antithrombotic drugs [12]. Hemorrhagic events related to vitamin K antagonists (VKAs) necessitate approximately 60,000 ED visits annually, with a comparable incidence observed among aspirin users [13].

Furthermore, epistaxis accounts for 30% to 50% of all hemorrhagic complications associated with VKAs, while the concurrent use of antiplatelet agents (e.g., aspirin or clopidogrel) multiplies the baseline risk of developing epistaxis by a factor of 5 to 10 [14].

9.1 Antiplatelet Agents

Therapeutic Molecules: This class includes Aspirin, Clopidogrel (Plavix), Prasugrel (Effient), Ticagrelor (Brilique), and Ticlodipine (Ticlid). All of these molecules significantly elevate the risk of epistaxis, which increases linearly in the following order of potency: aspirin < clopidogrel < prasugrel < ticagrelor. Dual antiplatelet therapy (DAPT), such as combining aspirin with clopidogrel, further exacerbates this risk exponentially.

Pharmacokinetic Reversibility: Understanding the precise window of antiplatelet reversibility is critical for managing

treatment regimens during a hemorrhagic crisis. The antiplatelet effect diminishes between 48 and 72 hours for aspirin, 3 to 5 days for clopidogrel and ticagrelor, and 5 to 7 days for prasugrel. Complete and immediate clinical reversal during an uncontrollable hemorrhage requires targeted platelet transfusions.

9.2 Anticoagulants

Therapeutic Molecules:
Vitamin K Antagonists (VKAs): Warfarin (Coumadin).
Heparins / Low-Molecular-Weight Heparins (LMWHs): Enoxaparin (Lovenox), dalteparin (Fragmin).
Direct Oral Anticoagulants (DOACs): Dabigatran (Pradaxa), edoxaban (Lixiana), apixaban (Eliquis), and rivaroxaban (Xarelto).

9.3 Reversal Protocols

Management under VKAs: In mild epistaxis, management focuses on investigating a potential overdose via International Normalized Ratio (INR) monitoring, correcting the anticoagulation level if necessary, and achieving local hemostasis (e.g., absorbable packing with oxidized regenerated cellulose/Surgicel).

In severe or life-threatening hemorrhage, immediate VKA reversal is mandatory using intravenous Vitamin K and Prothrombin Complex Concentrates (PCC) or Fresh Frozen Plasma (FFP). Concurrently, blood loss must be corrected, local hemostasis performed, and the subsequent therapeutic window for restarting anticoagulation via LMWH carefully evaluated.

Management under DOACs: In mild epistaxis secondary to dabigatran or other DOACs, management involves monitoring hemodynamic parameters and delaying or omitting subsequent doses. Conversely, severe or life-threatening hemorrhage requires aggressive volume resuscitation (packed red blood cells, FFP, and platelets if indicated) along with specific reversal agents. For dabigatran, Idarucizumab (Praxbind) is the first-line specific antidote. For factor Xa inhibitors (apixaban, rivaroxaban), Andexanet alfa (Ondexxya) or high-dose non-specific PCC/activated PCC (FEIBA) should be administered.

10. Epistaxis and Internal Carotid Artery Pathology

Internal carotid artery (ICA) pathologies associated with epistaxis typically involve either the intrapetrous or the cavernous segment of the artery. In these critical scenarios, the clinical presentation of epistaxis is highly variable, ranging from catastrophic, exsanguinating hemorrhage to recurrent, intermittent bleeding, or even occult micro-bleeding.

Diagnostic suspicion should be raised by a history of maxillofacial or cranioencephalic trauma, known congenital or acquired vascular diseases, and associated neurological symptoms, including trigeminal neuralgia or atypical headaches.

An internal carotid artery aneurysm located within its intrapetrous segment is an exceedingly rare but life-threatening condition. While its precise etiologies often remain obscure, established predisposing factors documented in the literature include long-standing arterial hypertension, severe head trauma, previous otological or mastoid surgery, and localized skull base infections.

Predisposing factors and demographics: middle ear infections, autosomal dominant polycystic kidney disease, Ehlers-Danlos syndrome, and Marfan syndrome are strongly associated with this vascular condition. The peak age of onset ranges between 30 and 40 years, with no sex predilection (15).

Clinical presentation and diagnosis: Presenting symptoms primarily encompass trigeminal neuralgia, atypical headaches, and intermittent epistaxis. Otoscopy may characteristically reveal a pulsatile red mass behind an intact tympanic membrane. Definitive diagnosis relies on computed tomography (CT), magnetic resonance imaging (MRI), and magnetic resonance angiography (MRA), while definitive management is strictly endovascular.

10.1. Cavernous segment internal carotid artery aneurysms

Aneurysms involving the cavernous portion of the internal carotid artery account for 2% to 9% of all intracranial aneurysms. In 1924, Barth described the classic symptomatic triad characterizing this condition: blindness, epistaxis, and previous maxillofacial trauma [16]. While these lesions can remain completely asymptomatic and be discovered incidentally on imaging, significant expansion leads to the compression of adjacent cavernous sinus structures, resulting in progressive cranial nerve palsies (CN III, IV, V, VI).

The terminal stage involves structural erosion of the sphenoid sinus wall, culminating in aneurysmal rupture and catastrophic, life-threatening epistaxis. Mortality rates associated with these ruptured lesions can reach up to 50%.

10.2. Carotid-cavernous fistulas

Carotid-cavernous fistulas constitute an abnormal shunt between the internal carotid artery and the surrounding cavernous sinus, representing approximately 1% of all intracranial vascular anomalies. Traumatic etiologies predominate in young adult males, whereas spontaneous forms typically occur in postmenopausal women with chronic hypertension. Clinical presentation is dominated by ophthalmological manifestations, including pulsatile exophthalmos, conjunctival injection, chemosis, decreased visual acuity, and ophthalmoplegia.

Comprehensive diagnostic workup requires CT, MRI, MRA, and definitive digital subtraction angiography (DSA). Treatment is exclusively endovascular [17].

10.3. Post-surgical Iatrogenic pseudoaneurysms

Iatrogenic pseudoaneurysms following endoscopic endonasal skull base surgery are increasingly reported in contemporary literature. They characteristically present as delayed epistaxis occurring anywhere from 3 days to several months post-operatively, a latency period that frequently delays accurate diagnosis.

Consequently, early diagnostic intervention is vital. A high-resolution CT angiography (CTA) or cerebral MRA must be

performed immediately for any "sentinel" posterior epistaxis in a patient with a documented history of skull base surgery or previous facial trauma, regardless of how remote the event [18].

10.4. The Trigeminocardial reflex during packing

Posterior nasal packing performed during an active rupture of an internal carotid aneurysm can trigger profound bradycardia and severe hemodynamic instability. This phenomenon is mediated by the trigeminocardial reflex (TCR), resulting from the mechanical pressure exerted by the packing onto adjacent cavernous nerve structures. Therefore, posterior packing in this context must only be a temporary, life-saving bridge pending immediate transfer to a neurovascular interventional suite [19].

10.5. Revolution of endovascular management

The advent of flow-diverting stents has revolutionized the endovascular management of ruptured internal carotid aneurysms and carotid-cavernous fistulas. These specialized stents redirect laminar blood flow along the parent artery, promoting progressive thrombosis and exclusion of the aneurysmal sac.

They are frequently coupled with endovascular coiling to achieve rapid occlusion, which is crucial since these patients must immediately initiate dual antiplatelet therapy to prevent acute stent thrombosis and secondary ischemic stroke [20].

11. Epistaxis and Hypertension

The World Health Organization (WHO) defines hypertension (HTN) as a persistent elevation of systemic arterial blood pressure, characterized by a systolic blood pressure (SBP) ≥ 140 mmHg and/or a diastolic blood pressure (DBP) ≥ 90 mmHg. HTN is a ubiquitous global health issue affecting an estimated 1.4 billion adults aged 30 to 79 years, and it remains a primary driver of cardiovascular morbidity and mortality, including myocardial infarction and stroke.

From a pathogenic perspective, the relationship between HTN and epistaxis involves dual microvascular mechanisms: structural stiffening of the vascular walls and biochemical alterations of endothelial wall proteins, specifically involving angiotensinogen and Vascular Endothelial Growth Factor (VEGF).

The association between epistaxis and hypertension is deeply entrenched in both clinical tradition and the public consciousness. In reality, establishing a direct causal relationship using rigorous scientific methodology remains challenging, and contemporary epidemiological data are frequently contradictory.

Excluding confounding variables such as concurrent antithrombotic therapy or localized rhinological pathologies, current clinical evidence regarding the interplay between hypertension and epistaxis can be synthesized as follows:

Risk and Causality

Epidemiological association: hypertension is statistically correlated with a higher baseline risk of epistaxis, even in the absence of identifiable local mucosal pathologies.

The Stress confounder: elevated blood pressure captured during an acute bleeding episode may not reflect baseline hypertension; rather, it often represents a secondary manifestation of acute stress syndrome and sympathetic activation induced by the hemorrhage itself. Consequently, a direct causal link remains difficult to formally establish clinical severity and control.

Impact on hemostasis: epistaxis occurring in hypertensive patients is characteristically more difficult to control than in normotensive individuals, frequently requiring more invasive hemostatic interventions.

Severity paradox: while the presence of hypertension does not independently dictate the intrinsic severity of the hemorrhage, severe epistaxis can often serve as the initial clinical presentation that reveals undiagnosed hypertension.

Hypertension complicated by hypertensive retinopathy serves as a documented independent risk factor for the development of severe epistaxis.

Hospitalization rates: epistaxis episodes in patients with documented hypertension are significantly more likely to result in hospital admission and more frequently necessitate the escalation to posterior nasal packing.

The recurrence misconception: Hypertension does not appear to be correlated with an increased risk of long-term recurrent epistaxis. Recurrence is more accurately attributed to a failure to localize and definitively treat the primary bleeding site.

Blood pressure management: actively controlling and lowering blood pressure during an acute episode has been shown to shorten the overall duration of the nosebleed.

Regardless of these ongoing pathophysiological debates, it is strongly recommended that patients presenting with concurrent epistaxis and elevated blood pressure be referred for comprehensive medical evaluation and specialized long-term management of their hypertension.

12. Epistaxis and Rendu-Osler Disease

Rendu-Osler disease is a constitutional vascular dysplasia without coagulation disturbances. It is a rare disease, affecting 1 to 2 people per 100,000 inhabitants, sometimes with a specific distribution (endemic areas). Diagnosis is primarily clinical.

This diagnosis is based on three semiological elements: epistaxis, a cardinal sign of the disease, with repeated, chronic bleeding, often neglected by the patient and those around them; telangiectasias (skin and mucous membranes); and the autosomal dominant inheritance pattern, found in more than 86% of cases at the time

of diagnosis.

Classically, the diagnosis is based on the Curacao criteria, with the presence of 3 out of 4 signs (epistaxis, telangiectasias, familial involvement, and visceral manifestations such as arteriovenous fistulas).

In reality, although the diagnosis remains clinical (and therefore simple) and well-defined (CURUACO criteria), it only allows for the identification of this condition in 10% of cases [21].

Recent advances in the diagnosis of this condition demonstrate a low level of diagnostic evidence for the triad, favoring the designation of "probable diagnosis" rather than "definite diagnosis" when all three criteria are met.

Furthermore, genetic sequencing is now mandatory, not optional. These primarily involve high-sensitivity NGS panels (ENG for THH1, ACVRL1/ALK1 for THH2, and SMAD4 for combined juvenile polyposis syndrome) and somatic "second hit" (secondary local somatic mutations leading to a loss of heterogeneity in the vascular endothelium) [21].

In terms of diagnostic testing, non-invasive investigations are increasingly preferred for arteriovenous malformations: contrast-enhanced ultrasound (lungs) and abdominal vascular Doppler ultrasound (liver).

Regarding therapeutic management, which is classically considered difficult and complex, there is a shift from a purely symptomatic and surgical approach (cauterization, vascular packing) to a phase of targeted and disease-modifying medical therapies.

Therapeutically, the role of anti-angiogenics is validated (bevacizumab and pomalidomide) with the arrival Vitamin K antagonists (VKAs) act on the abnormal overactivation of the PI3K-ATK pathway that causes aretiovenous malformations (following mutations in the ENG or ALK1 disease genes).

Engasertib (an oral ATK inhibitor) significantly reduces the frequency and duration of epistaxis [22].

Furthermore, efforts are made to better manage iron levels and anemia; ferritin levels should be above 50 ng/mL and transferrin saturation above 20%; intravenous iron infusions are preferred (ferric carboxymaltose should be avoided).

Anticoagulants remain an option when indicated (cardiac or thromboembolic risk factors).

13. Epistaxis and Pregnancy

The frequent occurrence of epistaxis during pregnancy is pathophysiologically linked to profound hormonal fluctuations. Elevated gestational estrogen levels induce a state of hypervascularization within the nasal mucosa, while progesterone increases circulating blood volume, thereby exacerbating venous and capillary congestion.

Furthermore, these gestational hormones upregulate the expression of histamine H1 receptors in both the nasal epithelium and microvascular endothelial cells, playing a critical role in localized inflammatory pathways.

The prevalence of epistaxis in pregnant women is 20.3%, compared to 6.2% in the general female population [23]. These episodes are predominantly moderate and rarely require emergency medical intervention. Severe or refractory epistaxis during gestation remains rare, except in the presence of compounding risk factors such as concurrent antithrombotic therapy or pre-existing inherited coagulopathies.

A classic localized lesion is the pregnancy hemangioma (granuloma gravidarum), a lobular capillary hemangioma that characteristically develops within the anterior vascular network (the anteroinferior segment of the nasal septum).

It accounts for approximately 5% of benign septal tumors in pregnant women, typically emerging during the second trimester and frequently undergoing spontaneous regression postpartum. Additionally, systemic gestational pathologies can present with or exacerbate epistaxis; these include gestational hypertension, preeclampsia, gestational thrombocytopenia, and HELLP syndrome.

Clinical Management: management of gestational epistaxis requires rigorous blood pressure control, specific rhinological care, and the topical application of emollient ointments. First-line interventions encompass targeted anterior nasal packing, intravenous tranexamic acid, and localized chemical or electrical cauterization. Refractory cases require escalated, invasive interventions such as endoscopic sphenopalatine artery ligation, anterior ethmoidal artery ligation, or endovascular embolization.

Beyond immediate rhinological hemostasis, the systemic impact of recurrent epistaxis on maternal anemia, labor outcomes, and potential neonatal anemia must be managed via close multidisciplinary collaboration with the obstetrician [24].

14. Pediatric Epistaxis

Epistaxis is a major clinical entity in pediatrics, accounting for 30% to 50% of all rhinological consultations in children under 5 years of age, and representing 0.5% of all pediatric emergency department visits with only 6% of cases necessitating specialized medical intervention. Epistaxis is exceedingly rare in infants under 2 years of age; its incidence peaks bimodally between 3 and 8 years and between 10 and 12 years, displaying no sex predilection.

14.1 Local and Systemic Factors

Local etiologies are predominantly driven by digital micro-trauma (nose picking), foreign bodies, and acute infectious or inflammatory sinonasal conditions. Environmental or anatomical factors that induce mucosal dryness and localized vascular

friability further exacerbate this risk. Allergic rhinitis is heavily implicated as a frequent and significant comorbidity that promotes recurrent pediatric epistaxis due to chronic mucosal inflammation and subsequent rubbing.

From a systemic standpoint, thrombocytopenia (including immune thrombocytopenia) is a relatively frequent etiology in recurrent pediatric cases, warranting a low threshold for a complete blood count (CBC) workup.

Environmental Influence: epidemiological data strongly correlate specific environmental and seasonal factors with a high incidence of pediatric epistaxis. Episodes peak significantly during the summer months, driven by elevated ambient temperatures, prolonged solar exposure, low relative humidity, low atmospheric pressure, and elevated ambient ozone (O_3) levels.

Refractory risk factors: Specific indicators are strongly associated with refractory forms of pediatric epistaxis, including a documented family history of coagulopathy, daily bleeding episodes, and the baseline presence of anemia.

Microbial colonization and pathogenesis: In pediatric idiopathic epistaxis, studies have demonstrated a high rate of nasal cavity colonization by Staphylococcus aureus. This bacterial colonization induces chronic crust formation and secondary ectasia of the microvasculature within the anterior nasal septum. This localized irritation prompts persistent digital micro-trauma (nasal scratching), directly triggering hemorrhage (25).

Recurrent pediatric epistaxis: recurrent epistaxis, a highly prevalent clinical scenario in pediatrics, is defined as five or more distinct bleeding episodes per year, accounting for approximately 9% of all pediatric cases. These hemorrhages typically originate from the anterior vascular network (Kiesselbach's plexus) and are primarily driven by repeated digital trauma or chronic mucosal inflammation.

While most recurrent cases are benign, underlying systemic coagulation abnormalities are identified in up to 10% of these patients, necessitating a thorough hematological workup. The primary hematological etiologies include von Willebrand disease, chronic liver disorders, and congenital or acquired platelet aggregation defects. Less frequently, these recurrences reveal rare but severe structural or systemic pathologies, such as a juvenile nasopharyngeal angiofibroma or Hereditary Hemorrhagic Telangiectasia (HHT/Rendu-Osler disease) [26].

14.2 Therapeutic escalation in pediatrics

The vast majority of recurrent episodes respond favorably to intensive, adequate hydration of the nasal mucosa using saline sprays or emollient ointments.

For resistant or refractory cases, modern therapeutic alternatives have expanded to include:

-Targeted chemical (silver nitrate) or electrical nasal cauterization.
-Potassium titanyl phosphate (KTP) laser photocoagulation or microwave ablation.
-Oral propranolol therapy, considered for its anti-angiogenic properties in vascular malformations.

Advanced hemostatic interventions: standard interventions for acute or severe pediatric epistaxis comprise anterior/posterior nasal packing, topical application of gelatin-thrombin matrix sealants (FloSeal), endoscopic sinonasal surgery, and, in rare exsanguinating scenarios, superselective endovascular embolization.

Concurrently, topical and systemic tranexamic acid (TXA) is gaining substantial clinical traction.
Accumulating evidence demonstrates its efficacy and safety profile compared to aggressive nasal packing or extensive cauterization, positioning TXA as an important agent in modern pediatric algorithms. Intravenous (IV) administration may be used for managing bleeding as determined by a healthcare provider.

15. Idiopathic Epistaxis

Idiopathic (essential) epistaxis remains the most common etiology of nasal hemorrhage in both adults and children. As a diagnosis of exclusion, it requires a rigorous yet cost-effective clinical approach to ensure high-morbidity or life-threatening secondary pathologies are not overlooked. A thorough medical history and comprehensive intercritical endoscopic examination are mandatory before initiating complex diagnostic investigations.

Chronobiological and Metabolic Associations:
Seasonal predilection: spontaneous episodes exhibit a significant bimodal seasonal peak during winter (December to February) and mid-summer (August) [27].

Nocturnal onset: bleeding occurring predominantly at night is a hallmark characteristic of posterior epistaxis, often correlated with nocturnal hypertensive surges.

Metabolic indicators: long-standing diabetes mellitus is frequently documented in these cohorts; it directly accelerates microvascular capillary fragility, thereby increasing the frequency and recurrence of bleeding episodes [28].

Anatomical and Endoscopic Characteristics:
Localization: approximately 80% to 90% of episodes originate from the anteroinferior segment of the nasal septum (Kiesselbach's plexus). Conversely, posterior epistaxis accounts for roughly 5% of cases and is predominantly observed in older populations. Statistically, a right-sided predominance has been documented [29].

Endoscopic findings: endoscopy typically reveals anatomical predisposing factors, such as a deviated nasal septum, along with prominent septal neovascularization or telangiectasias. Crucially, it serves to definitively rule out occult space-occupying lesions

within the posterior nasal cavities and nasopharynx.

Laboratory testing: routine baseline hemostasis screening is not recommended for idiopathic forms unless justified by clinical history. Pande et al. identified subclinical coagulation abnormalities in only 7% of unselected idiopathic epistaxis cohorts [30].

Modern management paradigms: the therapeutic approach to essential epistaxis has shifted dramatically. Current international guidelines prioritize the immediate use of first-line topical tranexamic acid, followed by the selection of bioresorbable fragmentable dressings, widespread utilization of procoagulant balloons, and low-temperature bipolar cauterization techniques. Regarding long-term antithrombotic therapy, modern recommendations heavily favor the strict continuation of the medication regimen, rather than routine discontinuation.

16. Geriatric Epistaxis

Geriatric epistaxis constitutes a distinct, high-risk clinical entity in otorhinolaryngology and emergency medicine. Hemorrhage in patients aged 65 to 70 and older frequently represents a complex, critical medical-surgical emergency characterized by rapid recurrence and a profound risk of systemic decompensation.

Statistically, epistaxis represents the second leading cause of emergency department consultations within the geriatric demographic, surpassed only by maxillofacial trauma.

Pathophysiological challenges and airway risks: advanced age functions as an independent risk factor for high morbidity and mortality during epistaxis episodes. In geriatric populations, the incidence of posterior epistaxis rises significantly to approximately 10%.

The clinical severity of these episodes is critically compounded by airway risks:
Aspiration hazards: posterior bleeding creates a high risk of blood aspiration, choking, asphyxia, and secondary aspiration pneumonia. This airway vulnerability is significantly exacerbated in bedridden patients or those suffering from cognitive decline (Alzheimer's disease) or post-stroke dysphagia.

Systemic decompensation and hemodynamic fragility: systemic decompensation occurs rapidly in elderly individuals. A sudden drop in hemoglobin levels (acute hemorrhagic anemia) can immediately trigger or exacerbate underlying ischemic heart disease, precipitating acute myocardial infarction, unstable angina, or acute heart failure. Similarly, sudden hemodynamic shifts drastically elevate the risk of secondary ischemic or watershed strokes.

Specialized geriatric protocols: therapeutic management prioritizes immediate systemic stabilization and intensive monitoring of vital functions (blood pressure, heart rate, and continuous cardiac tracking), alongside urgent laboratory workups (hemoglobin levels, complete blood count, and PT/INR coagulation profiles).

Furthermore, current guidelines mandate the use of atraumatic, non-invasive packing modalities to preserve the fragile senile mucosa. Recommended materials include gelatin-thrombin matrix sealants (FloSeal), bioresorbable fragmentable packing (NasoPore), and specialized devices like the CAVIT-T catheter, which features a central airway channel to ensure continuous patient oxygenation during bilateral packing.

Concurrently, antithrombotic therapies must be carefully tailored to the patient's overall thromboembolic risk, strictly avoiding unnecessary or prolonged treatment interruptions.

17. Mortality in Epistaxis

While the global mortality rate directly attributable to epistaxis remains exceedingly low, estimated at 0.01% in the general population, contemporary multicenter data reveal a higher burden of all-cause mortality among hospitalized cohorts.

The landmark "Epistaxis 2016: National Audit of Care" study, which prospectively collected data over a 30-day window across 113 centers, documented a significant 30-day all-cause mortality rate of 3.4% among admitted patients [31].

Fatal outcomes associated with epistaxis occur predominantly in elderly populations (aged 77 years and older) characterized by specific baseline biomarkers and clinical indicators, including a hemoglobin level below 12.8 g/dL, underlying coagulopathies, hypoalbuminemia, and prolonged hospital stays [32]. Crucially, mortality during an acute epistaxis episode is rarely driven by exsanguination alone.

Instead, deaths are primarily precipitated by severe cardiovascular, metabolic, or respiratory complications triggered by the acute physical stress of the hemorrhage.

Additional independent risk factors implicated in higher mortality include comorbid cardiovascular disease, diabetes mellitus, active antithrombotic therapy, and the requirement for allogeneic blood transfusions.

18. Risk Factors for Recurrence

Identifying the specific clinical indicators associated with early recurrence within hours or days following the initial hemorrhagic event is essential for risk-stratifying patients at the time of admission.

Comprehensive clinical trials have clarified the primary drivers of recurrent epistaxis:
Bimodal age extremes: age acts as a key risk factor, with recurrence rates peaking at both ends of the demographic spectrum: pediatric patients and geriatric populations aged over 70 years.

Male predominance: various epidemiological studies consistently correlate male sex with an elevated risk of recurrent bleeding. This association is hypothesized to be linked to higher physical activity levels, distinct behavioral factors, or potentially sex-linked

microvascular genetic influences [33].

Vascular and structural remodeling: chronic arterial hypertension, along with its associated structural and functional microvascular remodeling, increases capillary fragility and promotes spontaneous secondary rupture. Similarly, localized hypervascularization of the anterior nasal septum (Kiesselbach's plexus) and specific gestational vascular shifts (postpartum bleeding) are strong drivers of early recurrence.

Etiological susceptibility: certain underlying pathologies inherently predispose patients to intractable, multi-episodic bleeding. These include congenital or acquired hemostasis and coagulation disorders, the mandatory use of antithrombotic medications, and genetic vascular dysplasias such as Hereditary Hemorrhagic Telangiectasia (HHT/Rendu-Osler-Weber disease) [34]

19. Updates on Hemostasis Modalities

19.1. Pharmacological and Biological Innovations:

Propranolol in Pediatric Care: Recent literature highlights promising outcomes using oral propranolol in pediatric cohorts suffering from severe, recurrent epistaxis (weekly episodes). A dosage of 1.5–2.0 mg/kg/day, divided into three daily doses for three weeks to one month, has demonstrated a significant reduction or complete resolution of recurrence, likely mediated by its anti-angiogenic properties [35].

Fibrin Glue: The application of a localized fibrin sealant spray (Quixil) via a 0.3 mL nasal delivery system serves as an effective, atraumatic alternative to traditional nasal packing, achieving definitive hemostasis in 92.5% of cases [36].

Tranexamic Acid (TXA): This antifibrinolytic agent is widely utilized for acute epistaxis, particularly during persistent hemorrhages lasting longer than 10 minutes. While parenteral and oral administrations yield substantial clinical results, data on isolated topical application show variable superiority compared to active control treatments (Table 3). For topical delivery, a 5 mL ampoule (100 mg/mL) is applied directly to the bleeding site using a delivery matrix (e.g., cotton pledge, polyvinyl alcohol sponge/ Merocel) or via a targeted atomized spray [37].

Gelatin-Thrombin Matrix Sealant: This topical biological hemostatic agent (FloSeal) is applied directly to the bleeding site and represents a major alternative to traditional anterior or posterior nasal packing in acute, severe epistaxis. It combines a bovine gelatin matrix with an active human thrombin component, operating through a dual mechanism: mechanical volume tamponade and biological coagulation cascade activation. Absolute contraindications include known hypersensitivity to animal-derived proteins, intravascular injection, and active localized infection.

Application requires delivering the matrix from the deepest aspect of the wound toward the periphery, followed by local digital compression with a sterile gauze pad for 2 to 3 minutes.

Micronized Purified Flavonoid Fraction (MPFF): Oral administration of flavonoid-based venotonic and vascular protective agents (Daflon, 500 mg or 1000 mg) effectively combats microvascular capillary fragility. A continuous 3-month therapeutic course is statistically associated with excellent long-term control of idiopathic epistaxis, significantly reducing secondary emergency department readmissions [39].

19.2. Advanced Interventional and Thermal Ablation Techniques

Microwave Ablation (MWA): Microwave ablation utilizes a specialized miniature antenna typically coupled to a 2450 MHz generator inserted into the nasal cavity under direct endoscopic guidance. The emitted electromagnetic waves agitate water molecules within the surrounding mucosal tissue, creating friction and intense localized thermal energy that induces immediate, irreversible coagulation of the culprit vessel. This outpatient procedure is relatively painless, significantly minimizes long-term recurrence, and is highly suited for refractory idiopathic anterior epistaxis (following failed chemical or electrical cauterization) as well as localized arterial hemorrhages [38].

Coblation (Controlled Plasma Ablation): This advanced modality utilizes low-temperature bipolar radiofrequency energy to excite electrolytes in a conductive medium, creating a focused plasma layer. This layer simultaneously dissects and coagulates tissue at a lower thermal threshold than conventional electrocautery under direct endoscopic control, making it an ideal, tissue-sparing option for managing recurrent mucosal bleeding.

19.3. Future Technological Horizons

Bioactive hydrogels and nanotechnology: contemporary research is heavily focused on photoreactive bioactive hydrogels that achieve instantaneous polymer cross-linking and coagulation within 5 seconds when exposed to a targeted blue laser light. Additionally, advanced porous nanomaterials are being developed to maximize the functional contact surface area, instantly concentrating endogenous coagulation factors directly at the site of mucosal rupture [40].

20. Therapeutic Paradigms and Packing Modalities

The modern management of epistaxis is strictly based on the anatomical localization of the definitive bleeding source or area. Localized hemorrhage originating from the anterior vascular network (Kiesselbach's plexus) the most frequent clinical scenario is primarily managed with the topical application of a local vasoconstrictor, followed by targeted chemical, electrical, radiofrequency, or laser cauterization.

If first-line cauterization fails, or if the bleeding site cannot be precisely localized, nasal packing (vestibular, anterior, or

posterior) is indicated. While contemporary guidelines heavily promote a shift toward atraumatic, bioresorbable solutions, the choice between absorbable and non-absorbable packing materials remains largely dictated by emergency room availability, specific clinical constraints, and the practitioner's technical experience.
Traditional petroleum jelly (vaseline-impregnated) gauze or compresses remain widely utilized as first-line packing devices due to their simplicity, ease of use, and low cost.

In clinical trials, demonstrating the definitive superiority of one packing matrix over another remains challenging. However, a recent meta-analysis suggested that petroleum jelly gauze may be associated with a statistically lower rate of recurrent bleeding compared to polyvinyl alcohol (PVA) sponges (Merocel), though its removal is significantly more painful for the patient and the procedure is more time-consuming for the clinician [41].

Classification of Nasal Packing Materials (Figure 1): depending on availability and clinical severity, nasal packing is performed using various specialized materials classified into five main categories:
-Impregnated and non-adherent gauze: vaseline- or paraffin-impregnated meshes, including petrolatum gauze (Jelonet, Grasol, Biogaze, Antibiotulle).

Topical hemostatic alginate wicks: calcium alginate fibers that exert a local procoagulant effect upon contact with blood (Algosteril, Coalgan).

Non-absorbable expandable tampons: polyvinyl alcohol (PVA) expandable sponges or specialized carboxymethylcellulose (CMC) hydrocolloid fabric balloons (Merocel, Rapid Rhino).

Bioresorbable and absorbable matrices: advanced biomaterials designed to degrade spontaneously without requiring secondary removal, minimizing mucosal trauma. These include oxidized regenerated cellulose, gelatin-thrombin matrix sealants, and fragmentable polyurethane foams (Surgicel, Surgiflo, NasoPore, Hemopore, Gelfoam, MeroGel, Sinu-Foam, Nasastent, Sorbacel).

Dual-balloon catheters: specialized internal balloon probes engineered to exert targeted mechanical pressure on both the anterior and posterior nasal cavities simultaneously (Epimax, Epistop, Epistax, PostStop, Epistat).



Figure 1: Equipment for hemostasis.

From Top to Bottom: absorbable gauze (Surgicel), expandable polyvinyl alcohol (PVA) sponge (Merocel), oily (paraffin-impregnated) gauze, cotton pledge soaked in 5% lidocaine-naphazoline solution, Spencer nasal speculum, and angled dressing forceps.

The CAVIT-T, introduced in 2020, is a non-absorbable polyurethane balloon device with a polyvinyl chloride deployment guide designed to stop epistaxis via targeted direct pressure. It is less invasive and painful than traditional packing, preserves airway patency with a central breathing tube, and offers an alternative to double-balloon catheters (Figure 2). The materials shown include oxidized regenerated cellulose (Surgicel), expandable PVA sponge (Merocel), petroleum jelly gauze, and specialized nasal instrumentation.

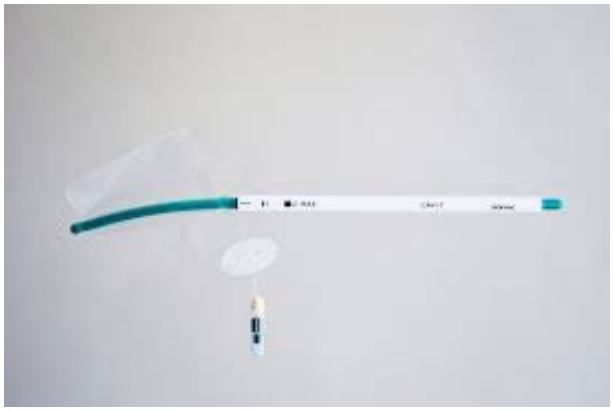


Figure 2: Modern nasal packing devices: CAVIT-T.

21. Surgical Interventions and Advanced Hemostasis

21.1. Endoscopic arterial ligation techniques

When nasal packing fails, immediate endoscopic hemostasis of the sphenopalatine artery (SPA) or the anterior/posterior ethmoidal arteries (via an endoscopic or transcanthal approach) is mandatory. The contemporary surgical paradigm favors the "modified arterial ligation" technique. This approach involves the total exposure of the SPA trunk by drilling away the posterior lip of the bony sphenopalatine foramen. Implementing this modification has significantly reduced failure rates to 7.8%, compared to 28% associated with traditional ligation techniques.

Furthermore, comprehensive hemostasis of all secondary arterial branches is strongly recommended, as the SPA frequently bifurcates into 3 to 5 distinct branches upon emerging from the foramen. Direct endoscopic bipolar cauterization is currently preferred over the application of titanium vascular clips. In scenarios where the initial ligation fails, clinicians are advised to perform direct cauterization of Stamm's S-point (located at the superior and anterior segment of the nasal septum) before attempting a surgical revision of the sphenopalatine region [42].

21.2. Interventional radiology and embolization

Superselective endovascular embolization serves as a critical last-resort intervention and is occasionally the most appropriate first-line modality for intractable or severe posterior epistaxis.

This technique is primarily reserved for geriatric patients, individuals with multiple severe systemic comorbidities, or those presenting with profound, uncorrectable coagulopathies. However, the decision to embolize must always carefully weigh the potential ischemic and neurovascular morbidities associated with the procedure.

22. Post-Intervention Management and Complications

1. Recurrent hemorrhage following packing removal: approximately 20% of patients experience a recurrence of bleeding immediately following the removal of non-absorbable nasal packing. Statistically, 90% of these secondary hemorrhagic episodes occur within four (4) hours post-removal. This recurrence is entirely independent of the specific packing material utilized.

2. Fortunately, post-removal epistaxis is predominantly benign and rarely necessitates re-packing; it is efficiently managed with simple clot aspiration and the temporary placement of a cotton pledge soaked in a local anesthetic-vasoconstrictor solution (lidocaine-naphazoline 5%).

These data highlight the absolute clinical necessity of monitoring patients in a clinical setting for a few hours immediately following packing removal.

Criteria for hospital admission: epistaxis necessitates formal hospital admission in 10% to 30% of cases. In patient care is explicitly indicated for elderly patients, severe or posterior hemorrhages, individuals on active anticoagulant therapy, patients with multiple systemic comorbidities, those exhibiting clinical signs of acute hemorrhagic anemia, or following the failure of first-line emergency interventions.

Beyond strictly medical parameters, critical socio-environmental and cognitive factors must dictate the admission decision. These include patients living alone or in geographic isolation, significant cognitive impairment, and active refusal of care or psychomotor agitation (advanced Alzheimer's disease or dementia) that renders the patient unable to maintain nasal packing or comply with mandatory post-procedural instructions.

3. Post-Hemostasis Follow-Up and patient education: following successful hemostasis, it represents excellent clinical practice to schedule a formal follow-up evaluation between the 3rd and 4th post-procedural weeks, particularly for patients who experienced moderate, recurrent, or poorly controlled episodes.

This delayed evaluation allows for a comprehensive etiological workup under optimal conditions specifically ruling out occult sinonasal or nasopharyngeal malignancies and enables the clinician to refine or complete initial treatments, such as performing a touch-up chemical or electrical cauterization.

A thorough ENT examination paired with high-resolution rigid or flexible nasal endoscopy is typically sufficient. Additional cross-

sectional imaging or specialized hematological assays should be ordered selectively based on the patient's updated clinical presentation.

Furthermore, patients who presented with severe hypertension upon admission must be systematically referred to their primary care physician for long-term blood pressure optimization once the acute bleeding episode has completely resolved.

Finally, structured patient health education is a cornerstone of the post-treatment phase to prevent long-term recurrence. Patients must be instructed to perform intensive topical mucosal hydration using saline sprays, petroleum jelly, or localized antibiotic ointments applied three times daily for 7 to 10 days to facilitate healthy mucosal re-epithelialization.

Crucially, patients must be explicitly counseled to strictly avoid the use of systemic non-steroidal anti-inflammatory drugs (NSAIDs), which impair platelet aggregation and significantly elevate the risk of recurrent hemorrhage [43].

Conclusion

Epistaxis is a frequent emergency in daily medical practice requiring accurate diagnosis, identification of the bleeding site and whether it is localized or diffuse, and, above all, recognition of patients in precarious situations (age, comorbidities, severity of bleeding, failure of previous treatments) in order to hospitalize them and implement appropriate therapeutic measures.

Current trends favor the application of atraumatic methods, prioritizing fragmentable dressings, bioresorbable fluid matrices, and procoagulant balloons. Endoscopic ligation of the sphenopalatine artery and embolization complete the hemostasis options for epistaxis, each with its respective indications.

The importance of an ENT examination after the bleeding has subsided (malignant tumors and aneurysms of the internal carotid artery) and the impact of new etiologies to be considered (toxic, iatrogenic) should not be overlooked.

Future efforts to reduce the number of consultations for epistaxis could focus on educating the elderly and parents of young children about nasal mucosal care and simple home first aid techniques [44,45].

Conflicts of Interest

The author declares no conflicts of interest related to this work.

References

1. Passali, D., Damiani, V., Passali, F. M., Tosca, M. A., Motta, G., & Ciprandi, G. (2020). An international survey on the pragmatic management of epistaxis. *Acta Biomedica, 91*, 5–10.
2. Hong, S. P., Yang, M. J., Bae, J. H., Choi, D. R., Kim, Y. C., Yang, M. S., Oh, B., Kang, K. W., Lee, S. M., Kim, B., Kim, Y. D., Ahn, J. H., & Koh, G. Y. (2023). Three-dimensional morphologic and molecular atlases of nasal vasculature. *Nature Cardiovascular Research, 2*, 449–466.
3. Kallenbach, M., Dittberner, A., Boeger, D., Buentzel, J., Kaftan, H., Hoffmann, K., Jecker, P., Mueller, A., Radtke, G., & Guntinas-Lichius, O. (2020). Hospitalization for epistaxis: A population-based healthcare research study in Thuringia, Germany. *European Archives of Oto-Rhino-Laryngology, 277*(6), 1659–1666.
4. Ruhela, S., Mittal, H. K., Bist, S. S., Luthra, M., Kumar, L., & Agarwal, V. K. (2023). Clinico-etiological evaluation of epistaxis. *Indian Journal of Otolaryngology and Head & Neck Surgery, 75*(Suppl. 1), 828–835.
5. Mauer, A. C., Khazanov, N. A., Levenkova, N., Tian, S., Barbour, E. M., Khalida, C., Tobin, J. N., & Coller, B. S. (2011). Impact of sex, age, race, ethnicity, and aspirin use on bleeding symptoms in healthy adults. *Journal of Thrombosis and Haemostasis, 9*(1), 100–108.
6. Pallin, D. J., Chng, Y. M., McKay, M. P., Emond, J. A., Pelletier, A. J., & Camargo, C. A. (2005). Epidemiology of epistaxis in US emergency departments, 1992 to 2001. *Annals of Emergency Medicine, 46*(1), 77–81.
7. Merland, J. J., Herbreteau, D., & Tran Ba Huy, P. (1994). Diagnostic and interventional angiography in ENT. In Modern ENT imaging (pp. 497–542). Arnette.
8. Kharoubi, S. (2024). Epistaxis. Universitary Office of Publication (OPU).ISBN: 978-9961-0-1181-2.
9. Kuo, C. L. (2019). Updates on the management of epistaxis. *Clinical Medicine and Therapeutics, 1*(1), 5.
10. Sastre, J., & Mösges, R. (2012). Local and systemic safety of intranasal corticosteroids. *Journal of Investigational Allergology and Clinical Immunology, 22*(1), 1–12.
11. Tabassom, A., & Dahlstrom, J. J. (2022). Epistaxis. In *StatPearls*. StatPearls Publishing.
12. National Blood Clot Alliance. (2024). *Bleeding risk awareness survey*.
13. Shehab, N., Sperling, L. S., Kegler, S. R., & Budnitz, D. S. (2010). National estimates of emergency department visits for hemorrhage-related adverse events from clopidogrel plus aspirin and from warfarin. Archives of Internal Medicine, 170, 1926–1933.
14. Rainsbury, J. W., & Molony, N. C. (2009). Clopidogrel versus low-dose aspirin as risk factors for epistaxis. Clinical Otolaryngology, 34, 232–235.
15. Asla, C., Bal, K. K., Gur, H., & Gorur, K. (2024). Aneurysm of petrous and cavernous parts of the internal carotid artery: A case report. *Indian Journal of Otolaryngology and Head & Neck Surgery, 76*, 3637–3640.
16. Hassan, A. M., Donley, C. W., & Venkatachalam, P. (2024). Post-traumatic intracranial pseudoaneurysm presenting as epistaxis. Open Access Emergency Medicine, 16, 75.
17. Harake, E. S., Nieblas-Bedolla, E., Wilseck, Z., Chaudhary, N., Armonda, R. A., Pandey, A. S., & Dowlati, E. (2026). Endovascular approaches for the treatment of dural carotid-cavernous fistulas: A systematic review. *Interventional Neuroradiology, 32*(4), 1714–1728.

18. Kim, H., & Choi, J. Y. (2024). Internal carotid artery pseudoaneurysm in a patient presenting with recurrent epistaxis: A case report and literature review. *Journal of Rhinology, 31*, 46.
19. Byrne, C., Flynn, L., & Carroll, C. (2023). Unstable bradycardia induced by posterior nasal packing: A rare activation of the trigeminocardiac reflex. *BMJ Case Reports CP, 16,* Article e258537.
20. Ghali, M. G. Z., & Binning, M. (2019). Flow diversion for the treatment of petrous internal carotid artery aneurysms. *Asian Journal of Neurosurgery, 14*(4), 1058–1062.
21. Locke, T., Miguel, B., Campos, S., & Chen, P. (2026). Hereditary hemorrhagic telangiectasia (HHT). In StatPearls. StatPearls Publishing.
22. Kasthuri, R. S. (2025). What's new in hereditary hemorrhagic telangiectasia? *Hematology, 2025*(1), 131–136.
23. Dugan-Kim, M., Connell, S., Stika, C., Wong, C. A., & Gossett, D. R. (2009). Epistaxis of pregnancy and association with postpartum hemorrhage. *Obstetrics & Gynecology, 114*(6), 1322–1325.
24. Piccioni, M. G., Derme, M., Salerno, L., et al. (2019). Management of severe epistaxis during pregnancy: A case report and review of the literature. *Case Reports in Obstetrics and Gynecology, 2019*, 1–3.
25. Kamble, P., Saxena, S., & Kumar, S. (2015). Nasal bacterial colonization in cases of idiopathic epistaxis in children. *International Journal of Pediatric Otorhinolaryngology, 79*(11), 1901–1904.
26. Yan, T., & Goldman, R. D. (2021). Recurrent epistaxis in children. *Canadian Family Physician, 67*(6), 427–429.
27. Yu, J., Wang, S., & Zhong, Z. (2024). Frequency and severity of idiopathic epistaxis relative to time of day. *Scientific Reports, 14*(1), Article 29852.
28. Song, S. O., Jung, C. H., Song, Y. D., et al. (2014). Background and data configuration process of a nationwide population-based study using the Korean National Health Insurance System. *Diabetes & Metabolism Journal, 38*(5), 395–403.
29. Bhatta, R. (2012). Clinical profile of idiopathic epistaxis in a hospital. Journal of Nepal Medical Association, 52(188), 167–171.
30. Pande, N. M., Jain, S. K., Mahore, D. M., Nitnaware, A. Z., Bokare, B. D., & Sakhare, P. T. (2006). Role of routine coagulation studies in idiopathic epistaxis. *Indian Journal of Otolaryngology and Head & Neck Surgery, 58*(2), 160–161.
31. Integrate (The National ENT Trainee Research Network). (2018). Epistaxis and mortality. *The Journal of Laryngology & Otology, 132*(12), 1061–1066.
32. Corr, M. J., Tikka, T., Douglas, C. M., & Marshall, J. (2019). One-year all-cause mortality for 338 patients admitted with epistaxis in a large tertiary ENT centre. *The Journal of Laryngology & Otology, 133*(6), 487–493.
33. Hadar, A., Shaul, C., Ghantous, J., Tarnovsky, Y., Cohen, A., Zini, A., & Peleg, U. (2026). Risk factors for severe clinical course in epistaxis patients. *Ear, Nose & Throat Journal, 105*(2), NP135–NP140.
34. Ross, A., Engebretsen, S., Mahoney, R., & Bathula, S. (2022). Risk factors and management for epistaxis in a hospitalized adult sample. *Spartan Medical Research Journal, 7*(2), Article 37760.
35. Bjelakovic, B., Bojanovic, M., Lukic, S., Saranac, L., Vukomanovic, V., Prijic, S., Zivkovic, N., & Randjelovic, D. (2013). The therapeutic efficacy of propranolol in children with recurrent primary epistaxis. *Drug Design, Development and Therapy, 7,* 127–129.
36. Vaiman, M., Segal, S., & Eviatar, E. (2002). Fibrin glue treatment for epistaxis. Rhinology, 40(2), 88–91.
37. Zahed, R., Moharamzadeh, P., Alizadeharasi, S., Ghasemi, A., & Saeedi, M. (2013). A new and rapid method for epistaxis treatment using injectable form of tranexamic acid topically: A randomized controlled trial. *The American Journal of Emergency Medicine, 31*(9), 1389–1392.
38. Lou, Z. C., Dong, Y., & Lou, Z. H. (2019). Microwave ablation for the treatment of arterial epistaxis: "How I do it". *International Forum of Allergy & Rhinology, 9*(6), 702–706.
39. Attia, T. M. (2019). Efficacy and safety of Daflon in the treatment of idiopathic epistaxis. *American Journal of Rhinology & Allergy, 33*(1), 62–68.
40. Wu, Y., Ge, X., Yang, G., Bai, C., Pan, M., Xue, L., & Gu, N. (2026). Interventional hemostatic materials: Innovations in bleeding control, coagulation modulation, and vascular healing. *Advanced Interventional Materials, 1*(1), Article 100009.
41. Meccariello, G., Cammaroto, G., Gallo, O., et al. (2019). Management of idiopathic epistaxis in adults: What's new? *Acta Otorhinolaryngologica Italica, 39*(4), 211–219.
42. Dispenza, F., Lorusso, F., Di Vincenzo, S. A., Dolce, A., Immordino, A., Gallina, S., Maniaci, A., Lechien, J. R., Calvo-Henriquez, C., Saibene, A. M., & Sireci, F. (2024). Management of uncontrolled/recurrent epistaxis by ligation or cauterization of the sphenopalatine artery: A scoping review. *European Archives of Oto-Rhino-Laryngology, 281*, 6229–6238.
43. Mylonas, S., Skoulakis, C., Nikolaidis, V., & Hajiioannou, J. (2023). Epistaxis treatment options: Literature review. *Indian Journal of Otolaryngology and Head & Neck Surgery, 75*, 2235–2244.
44. Sorrentino, J. M., DiNardo, L. A., Kassem, A. A., Reese, A., Nanu, D. P., Nagy, R., & Carr, M. M. (2026). Public education on epistaxis management: Does a video help? *Cureus, 18*(4), Article e106436.
45. Dunne, H., Abouabdallah, M., Roscamp, J., Birks, S., McGibbon, K., Dewhurst, S., Strachan, D., & Sharma, R. (2025). Exploring knowledge of first aid in epistaxis—25 years on. *PLOS ONE, 20*, Article e0315092.

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