Epistaxis: An Overview

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Summary:

Epistaxis is a common clinical problem with varied etiological factors. Careful history taking and examination under good and adequate lighting with appropriate instruments is essential in identifying the bleeding site. Management includes resuscitation and followed by arrest of bleeding through cautery/nasal packing. Refractile cases will need surgical intervention. Investigations are usually tailored towards identifying the cause in the individual patient especially if other bleeding sites are apparent or the cause is not known.
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INTRODUCTION AND HISTORY

EPISTAXIS is Greek word for nosebleed. It is a clinical problem of common occurrence and may present as an emergency, as a chronic problem of recurrent bleeds or may be an indicator of a generalized disorder.

Folklore and Myth has been associated with epistaxis such as its occurrence indicating that you are in "love", "one drop of blood from the nose commonly foretells death or a very severe illness"[1], while some believe its cause to be "spiritual"[2]

Lupton in 1601 has been cited as advocating writing magical words on the patient's forehead using his/her blood while Moncrief in 1716 had the patients sniff their own fried blood and wear scarlet silk thread with nine knots down the front all in an attempt to prevent further bleeds[1,3].

Ancient Egyptian history had it that ashes of papyrus mixed with vinegar were used to treat nosebleed, while powder made from mummies (Mummia Vera) was also used even to the end of the 17th century[4]. In the South West of Nigeria, the use of "effirin leaf," a local herb (Occimum gratissimum) in various forms in the arrest of epistaxis had been an age long practice[2,5,6].

Pressure on the alae nasi as an effective way of controlling anterior epistaxis had been recognised by earliest physicians including Hippocrates. Cave Michael (1871), James Little (1879) and Wilhelm Kiesselbach were the first to identify the anterior septal vascular plexus as the source of nasal bleed[1,3].

Prevalence of Epistaxis in the United States of American is about 5 – 14%[1,3]. Some workers are of the opinion that the incidence of epistaxis increases with advancing age, during winter months[7] and hot dry climates with low humidity[1,7] but appears to be more common during the rainy season in Nigeria[5]. The increased incidence of epistaxis during the cold and hot dry seasons may be due to the increased incidence of upper respiratory tract infections. Forceful blowing of the inflamed congested nose may lead to epistaxis[3,5]. Nasal mucosal injury may lead to an inflammatory reaction with granulation tissue formation, which bleeds easily on slight contact. The resulting nosebleed may be worsened by the fibrinolytic enzymes (e.g. staphylokinase and streptokinase) which are elaborated by the invading bacteria[1].

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Though Epistaxis generally has a bimodal incidence, with peaks in the 2 – 10 and 60 – 80 years age groups[8], studies from Nigeria show the 21 – 30 years age group to be more affected[5,9]. It is thought that the predominance of the young in clinic attendances in the tropics may be responsible for this[5]. The male gender is more affected than the female. Nosebleed is unusual in infants in the absence of a coagulopathy or nasal pathology (e.g. choanal atresia, neoplasm) [1,10].

Two areas often implicated in epistaxis are the Little's area and Woodruff's plexus for anterior and posterior nasal bleeds respectively[1,3]. Anterior epistaxis is more common in children and adolescents while posterior epistaxis is more likely in older adults with hypertension and arteriosclerosis[3].

Vascular Supply of the Nasal Cavity

The rich vascular supply underlying the nasal mucosal covering is important for the function of humidification, temperature regulation and immune defense. These vessels arise from either the internal carotid (ICA) or from the external carotid artery (ECA).

The ophthalmic artery, which is the first division of the internal carotid artery, gives off both the anterior and posterior ethmoidal arteries. The anterior artery is bigger than the posterior division and both course medially to cross the roof of the nasal cavity to supply the superior part of both the nasal septum and lateral nasal wall. The ECA supplies the nose via its facial and internal maxillary artery branches. The facial artery supplies the anterior part of the nose via the superior labial artery.

The internal maxillary artery in the pterygopalatine fossa gives off about five main branches including the sphenopalatine, posterior nasal and greater palatine arteries. The sphenopalatine enters the nose at about the level of the posterior end of the middle turbinate supplying the posterior part of the septum and most of the lateral wall. The other branches also contribute to the vascular supply of the nasal cavity.

In the anterior part of the septum anastomosis of the sphenopalatine, Greater palatine, Superior labial artery (branch of the facial artery) and the anterior ethmoidal artery form the Kiesselbach's plexus at Little's area.

In the posterior part of lateral wall at the posterior end of the middle turbinate is the Woodruff's plexus formed by the anastomosis of the sphenopalatine, the posterior nasal and the ascending pharyngeal arteries[1,3]. It does seem however that there is a venous component of Woodruff's plexus, which is situated on the lateral wall of the nose at the posterior end of the inferior turbinate[10]. Venous bleeds from the anterior nasal septum comes from the retrocolumellar vein and not from the Kiesselbach plexus as was erroneously believed in the past.

Anterior bleeds can be accessed easily in contrast to posterior bleeds, which are rather more difficult to see and treat[1,3]; the dividing line between anterior and posterior epistaxis being the ostium of the maxillary sinus.

Etiology of Epistaxis

The etiology of epistaxis is multi factorial but can be divided into two broad groups – Local and Systemic. It must be noted that most cases of epistaxis do not have an easily identifiable cause.

Local factors:

Local factors tend to alter the normal physiologic function of the nose. Vascular injuries with bleeding, results secondary to the effect of environmental factors on the exposed vascular network. Nose picking in children is a known and common cause of epistaxis. Cranio-facial trauma may cause deformation and fracture of the facial structures with resultant nosebleed. Nasal bleed may thus follow trauma to the paranasal sinuses, orbits, middle ear and base of skull[1,3,5,9,11].

Foreign bodies insertion into the nose, nasogastric tube and naso-tracheal intubation and sinonasal surgeries are other forms of trauma that may result in nosebleed. Epistaxis may result from abuse of topical decongestants, cocaine, cannabis or heroin.

Anatomic deformities such as septal spurs and deviations, result in air turbulence, which in turn leads to crusting. The vessels rupture with the slightest trauma such as rubbing of the nose. Upper respiratory tract infection, chronic sinusitis, allergy and environmental irritants damage the mucosa thus encouraging the overgrowth of virulent bacteria, friable mucosa and epistaxis. Chronic specific infections (Tuberculosis, Leprosy, nasal diphtheria and Syphilis) may present with epistaxis[3,12].

Tumours, extra nasal aneurysms of the ICA are another group of local factors that give rise to epistaxis. These tumours include those in the sinonasal and nasopharyngeal regions. Bleeding may be fatal.

Systemic factors:

Inspite of a strong association with epistaxis,

hypertension is not a direct cause of epistaxis. Arteriosclerosis in the vessels of hypertensives leads to decreased haemostatic abilities and increased fragility of the vessels.

Osler – Rendu Weber disease (hereditary hemorrhagic telangectasia) is an autosomal dominant disease. The elastic and muscular tissue elements of the vessel lack contractile elements. Arterio-venous fistulae are formed. Minimal injury to the nasal mucosa leads to a significant bleed.

Patients with lymphoproliferative disorders, blood dyscrasias, immune deficiency states or alcoholic state can present with epistaxis. Drugs such as the NSAIDS, chloramphenicol and patients with vitamins C and K deficiencies can result in epistaxis.

Clotting factors that can result in frequent recurring epistaxis include: VonWillibrand's disease (most common), Factor VIII deficiency (Haemophilia A), Factor IX deficiency (Haemophilia B) and factor XI[3].

It has been suggested that epistaxis seen in people undergoing chronic haemodialysis may be due to a prostaglandin (prostacyclin) with platelet anti-agg regatory properties[12]. Thrombocytopaenia may be seen in pregnancy especially if there is folic acid deficiency[12].

Epistaxis can occur with heavy metal (such as phosphorus and chromium) as well as infections like smallpox, typhoid fever, rheumatic fever[3, 12].

Cardiovascular factors such as congenital heart failure, mitral stenosis, coarctation of the aorta and superior vena cava obstruction have been implicated in the cause of epistaxis. They are associated with increased systemic venous pressure, which can lead to rupturing of the vessels in the Little's area[12] and Woodruff's plexus[13].

Management of Epistaxis

This should be individualized according to the degree, site, clinical state and the etiology of the epistaxis. An intravenous access is established, and blood taken for full blood count, clotting profile (PT, PTTK) and grouping and cross-matching (as needed). Initial measures are directed towards the immediate control of bleeding and correction of hypovolaemia. Generally speaking for every 100mls of blood loss, 300mls of crystalloid fluid is given. If blood volume loss in the adult is greater than 30% (1500mls in the adult) or on going loss exceeds 100mls/min, transfusion should be considered[1, 14]. The patient with severe blood loss is haemodynamically unstable with hypotension, a pulse rate > 120/min and respiratory rate of above 301/min. The American College of Surgeons established a simple guideline in which blood loss is classified into class I to IV in their *Advanced Trauma Life Support Instructor Manual.* This is aimed at preventing hypovolemia and irreversible shock in epistaxis patients. Table 1.

Having controlled bleeding and stabilized the patient, a meticulous and comprehensive history is taken and clinical examination is performed.

Conservative treatment:

Most nosebleeds may cease spontaneously or following simple measures. The modified Trotter's maneuver is usually employed. This involves the patient sitting quietly, leaning

Table 1

Classification of Blood Loss, Physiologic Alterations and Fluid Replacement (Based on 70-Kg Adult)

	Class I	Class II	Class III	Class IV
Percentage of blood loss	10%-15%	15%-30%	30%-40%	>40%
Amount of blood loss (mL)	700-750	750-1500	1500-2000	>2000
Pulse rate	<100/min	100-120/min	>120/min	Usually >140/min
Respiratory rate	14-20/min	20-30/min	30-40/min	Usually >35/min
Blood pressure	Normal	Normal	Decreased	Further decrease in systolic pressure
Fluid replacement	Lactated Ringer's and blood	Lactated Ringer's	Lactated Ringer's	Lactated Ringer's

Culled from The American College of Surgeons Advanced trauma life support instructor manual 1989 as contained in Bailey Head & Neck Surgery-Otolaryngology.

forward; the mouth is left open for breathing with the patient holding a kidney receiver to collect any blood, which may have entered the oropharynx. A direct two-finger pressure is applied over the anterior nose. Ice packs may be placed over the midface. Undue straining, sneezing nose picking, nose blowing and medications that inhibit coagulation should be avoided.

Chemical cautery using either silver nitrate or trichloroacetic acid can be used to arrest a bleed. An actively bleeding vessel is best arrested using an electrocautery. The depth of tissue penetration is more and can expose septal cartilage or cause septal perforation. A local anesthetic is always desirable before cautery, while antibiotic ointment application to the cauterized area until healing is needed[12].

Cryotherapy has been in use since the days of Hippocrates[12]. Though it has a low complication rate, it is infrequently used now[1].

Anterior nasal packs are used in the case of failure of the above measures or a seeming anterior nosebleed, which cannot be localized. The nose is adequately anaesthetized using either a 5% cocaine solution or 4% lignocain. A vaseline gauze impregnated with an antimicrobial ointment or ribbon gauze soaked with Bismuth Iodoform Paraffin Paste (BIPP) is layered in fashion as far back as possible toward the posterior choana starting from the floor of the nasal cavity. Cut-fracture of the inferior turbinate may be necessary to allow for adequate packing. The pack is left in place for 2 – 7 days. Prophylactic broad-spectrum antibiotics should be given in all cases of nasal packing because of the risk of sinusitis and toxic shock syndrome due to staphylococcus aureus. Merocel sponge, gel foam and surgical oxycel are nasal tampons that can be used in place of anterior nasal pack. They dissolve within 2 – 3 weeks.

A posterior bleed that is refractive to cautery or properly placed anterior nasal packing calls for a posterior nasal pack. For this purpose, a standard posterior nasal pack (made up of finely rolled gauze tied in the center with two long pieces and one short piece of umbilical tape or O-silk) or foleys catheter is used. An anterior nasal pack is also inserted in addition. Adequate external nasal padding is ensured to avoid skin necrosis. Pack is removed after 4 – 7 days.

Epi-stat, an epistaxis balloon tampon is a double balloon system that serves as both anterior and posterior pack. It is easy to insert, less traumatic while allowing a partial airway. It is however less effective than a standard pack since the pressure applied to the nose is not equal. Patients with nasal packs should be on admission while the elderly with cardiopulmonary past medical history or blood gas changes should be monitored in the intensive care unit[1, 12]. The greater palatine foramen block in which about 3mls of 1% or 2% lignocain with 1:100,000 or 1:200,000 epinephrine is injected into the pterygopalatine fossa is used to control nasal haemorrhage involving the distribution of the sphenopalatine artery. The control of epistaxis, which is temporary (3 hours) acts by volume compression of the vascular structures in the pterygopalatine fossa.

Surgical treatment:

For uncontrolled epistaxis the surgical options are arterial ligation and septoplasty/septal dermoplasty. Before deciding on which artery to ligate the site of the epistaxis must be determined first. Ligation should be as close as possible to the site of bleed to minimize the risk of collateral circulation[1]. Other factors accounting for failures in controlling the bleed include improper identification of the vessel and alternative dominance of vessels[14].

Recurrence rates for bleeding after internal maxillary artery ligation range from 5% to 13%. The transantral approach into the pterygopalatine fossa for IMA ligation has the demerit of n[illegible]g used in children, patients with hypoplastic maxillary sinuses or those with comminuted facial fractures. It may be also complicated by damage to the Vidian nerves or sphenopalatine ganglion, infra orbital nerve and oro-antral fistula.

The intra oral approach to the infra temporal portion of the IMA is useful in children as an alternative to embolization and external carotid artery ligation. It is also ideal in patients with hypoplastic maxillary sinus, comminuted fractures of the maxilla and to control severe bleeding during maxillectomy. The tendency to the development of collateral circulation is high and the manipulation of the temporalis muscle results in trismus.

In patients with re-bleed after IMA ligation or those with superior nasal cavity epistaxis, ligation of the ethmoidal vessels may be considered. They are often ligated in conjunction with the IMA when the bleeding site is ill- defined.[1,7]. Reported complications include cerebrovascular accident, blindness, ophthalmoplegia and epiphora.[7]

External carotid artery ligation decreases flow throughout the vessel by 50%. Collaterals from the ethmoidal vessels make it imperative for them to be ligated in conjunction with the ECA especially in severe facial fractures[12]. During the

isolation and identification of the ECA, few drops of 1% lignocain are dripped onto the carotid sinus to checkmate bradycardia. The suture ligation is placed distal to the superior thyroid artery origin.

An innovative approach in the management of posterior epistaxis is Endoscopic cauterization of a specific site using an insulated malleable suction electrocautery apparatus through an Endoscopic sinus surgery set or with the aid of a flexible nasopharyngoscope[12].

Selective arteriography is very helpful as a diagnostic and therapeutic tool in the management of bleeding sites responsible for intractable epistaxis. Identifying the site of the bleed enables it to be embolized using either polyvinyl alcohol, gel-foam particles or coiled springs. The procedure is effective when the bleeding rate is greater than 0.5mls/min. Its complications include hemiplegia, facial paralysis, skin necrosis, grand mal seizure and trismus[1, 15]. It has been reported that risk factors for intractable epistaxis include hypertension, alcohol or tobacco use and anticoagulant medication.[15, 16].

Septodermoplasty is often the mode of treatment for patients with hereditary haemorrhagic telangiectaesia but can also be used in patients with chronic septal ulcers and septal perforations. A total excision of the telangiectatic area is followed with placement of a split-thickness skin graft to the area. Cutaneous, myocutaneous or micro vascular free flaps can also be used in place of the skin graft. Complications may result from shrinkage of graft with exposed telangiectatic areas or in growth of the telangiectasia into the skin graft itself[1, 12]. The carbon dioxide and Nd – YAG lasers have shown encouraging results in the treatment of superficial telangiectatic and subepithelial arteriovenous malformations respectively[17].

Septoplasty/submucous resection of the nasal septum corrects deformities, thus minimizing turbulence and also decreases the vascularity of the nasal septum by promoting scarring of the bleeding tissues.

Drugs:

The use of drugs in the management of epistaxis has a limited application. It is reported that estrogen has been used with good success in the treatment of difficult cases of hereditary haemorrhagic telangiectasia in women. This is due to alteration of the nasal mucosa to a more protective squamous variety for the damaged telangiectatic areas. This is in addition to reconstituting the normal continuity of the abnormal endothelial lining of the affected vessel[2, 5]. Desmopressin (DDAVP) a synthetic analogue of arginine vasopressin has been used to decrease the bleeding time in patients with hemophilia A, Von Willebrand's disease and uraemia [18]. Ethamsylate (dicynone) is an antihaemorrhagic and vasoprotective agent that acts by increasing platelet adhesiveness and restores capillary-wall resistance.

Epistaxis is thus a common clinical problem with varied etiological factors. Careful history taking and examination under good and adequate lighting with appropriate instruments is essential in identifying the bleeding site. Management includes resuscitation and followed by arrest of bleeding through cautery/nasal packing. Refractile cases will need surgical intervention. Investigations are usually tailored towards identifying the cause in the individual patient especially if other bleeding sites are apparent or the cause is not known. This brings to the forefront the multidisciplinary nature in the management of epistaxis. The need may arise where the expertise of the Haematologist, Nephrologist or the Endocrinologist comes into play in the patient with coagulopathy/blood dyscrasia, hypertension or diabetes respectively. In the same vein the skill of the Radiologist, Plastic surgeon, Neurosurgeon and the Cardiothoracic Surgeon are required in selective arteriography/embolisation, Septodermoplasty, resection of intracranial aneurysm with intranasal extension and relief of superior vena cava obstruction respectively.

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