

Management of epistaxis

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Epistaxis, literally nose bleeding, can be a life-threatening emergency. It occurs in 10-12% of the general population. Adequate knowledge of first-line management is important for every medical practitioner, to avoid unnecessary referral to otorhinolaryngologists who are sparsely distributed, particularly in developing countries. The management of epistaxis depends on the clinical state of the patient at presentation; the simplest method that can control the bleeding is followed, but difficult cases should promptly be referred to the appropriate specialists, to reduce morbidity and mortality from this common emergency.

INTRODUCTION

Epistaxis literally means nose bleeding. This potentially life-threatening common condition is not a disease but a symptom of underlying disease. Whereas an adequate knowledge of its first-line management is important for every medical practitioner, definitive management should be reserved, where logistics and manpower permit, for a multidisciplinary team involving the otorhinolaryngologist, physician or paediatrician, and haematologist.

HISTORICAL PERSPECTIVES

Epistaxis has been known and described as far back as the period of Hippocrates. From that time on, various workers have contributed to advances and development in the understanding and management of epistaxis.

Ali Ibn-Rabban Al-Tabiri (AD 850) devoted a chapter of his massive work *The Paradise of Wisdom* to epistaxis, and he wrote: 'The complaint of nose bleeding is due to swelling and rupture of a vein or perhaps a reduction in the force which confines the blood within'.^{1,2}

Morgagni (1769) recognized 'the

extremely turgid blood vessels about that part where the alae nasi are formed with the bone about a finger breath from the bottom of nostrils', and stopped the bleeding with finger pressure on this part.^{3,5} This inspiration was drawn from his teacher Valsalva, and he therefore named the area *locus Valsalvae*.

Frederick Mahomed, a very perceptive physician at Guy's Hospital, in 1870 suggested epistaxis as a clinical feature of high blood pressure.⁴ In 1880, he noted that: 'The frequency with which severe epistaxis occurs in old people with high arterial pressure is striking and for them very fortunate for if their nose did not bleed their brain would bleed'.²

James Little (1879) published his case report in the hospital gazette, identifying the site of bleeding as the caudal end of the septum; and Kisselbach made a similar observation a year later.²

Over the years, different forms of arterial ligation have been performed for epistaxis control. Better results are now achieved with the advent of endoscopic and microvascular surgery.

ANATOMY

The nose is supplied by both the external carotid (lateral branches of sphenopalatine and greater palatine) and internal carotid (anterior and posterior ethmoidal) arteries, both forming numerous anastomoses. The medial wall of the nose is supplied by the septal branches of the anterior and posterior ethmoidal, the superior labial, and the septal branches of the sphenopalatine and greater palatine arteries. These medial arteries, with the exception of the posterior ethmoidal, meet at a point 1.5 cm above the floor of and posterior to the nares. This, Little's area, is easily traumatized. The lateral wall of the nose is supplied mainly by branches of the sphenopalatine artery.

EPIDEMIOLOGY

The frequency of epistaxis is difficult to determine because most episodes resolve with self-treatment and, therefore, are not reported.⁵ However, epistaxis is a common condition as well as a frequent otorhinolaryngological emergency, with up to 60% of the population experiencing one episode in their lifetime, 10-12% repeatedly, and 6% seeking medical attention.^{2,6} Epistaxis occurs in all age groups, with peak incidences between 15-24 and 45-65 years. There is no evidence of gender or racial predilection.

Anterior epistaxis, occurring in the anterior two-thirds of the nose, is the most common type, and is most frequently seen in children and young adults. It can often be resolved by pinching the cartilaginous part of the nose. Posterior epistaxis, occurring in the posterior third of the nose, accounts for 10% of nosebleeds and

is usually more profuse, because it originates from larger vessels near the sphenopalatine artery, behind the middle turbinate bone. It usually occurs in older persons, who have fragile vessels because of hypertension, atherosclerosis, coagulopathies or weakened tissue. Posterior bleeds require aggressive treatment and probably admission to hospital.

Epistaxis is usually more frequent in areas where there is increased prevalence of upper respiratory tract infections and drastic fluctuations in temperature and humidity.

AETIOLOGY AND PATHOGENESIS

Epistaxis can be due to local or general causes, which may be congenital or acquired.

Local causes

Congenital local causes

These are found in neonates and children, and include congenital central nervous system tumours such as meningocele, aneurysms, congenital nasal deformities such as septal deviation, or septal spurs. Septal deviations disrupt the normal nasal airflow, leading to dryness and hence epistaxis. The bleeding sites usually are located anterior to the deviation or spurs.

Acquired local causes

These are mostly idiopathic; accounting for more than 60% of cases of epistaxis.

Trauma may be external or internal. External trauma may be blunt injury, as in skull-base fracture following road traffic injury, or penetrating injury as in gunshot or surgery. Internal trauma includes nose picking, foreign bodies in the nose and intranasal surgery. Nose picking, nose blowing or sneezing can tear or abrade the mucosa and cause bleeding. Other forms of trauma causing nosebleeds include nasal fracture and nasogastric and nasotracheal intubation.

Septal perforations due to various

causes (septal surgery, nose picking, trauma, granulomatous disease, non-medical use of cocaine) also can cause epistaxis. Granulation and crusting occur on the margins of the perforations, which bleed easily.⁵

Infection leading to inflammation of the mucosa can result in friability, vascular rupture and bleeding. Such infection may be acute or chronic, specific or non-specific, bacterial, viral or fungal. Other inflammatory diseases, due to allergy or vasomotor changes as in nasal polyposis or rhinitis, can have similar effects.

Climatic changes, or even change from a dry or warm room to a damp or cold room, lead to variation in the normal nasal cycle and alternating congestion and decongestion. This in turn results in sinonasal congestion and infection, engorgement of the nasal mucosa and ultimately epistaxis.

Pseudotumours such as nasal granuloma, granuloma gravidarum or nasal polyp can cause vascular engorgement, weakness and rupture. Neoplasia can be benign (e.g. transitional cell papilloma or juvenile angiofibroma) or malignant, as in squamous cell carcinoma, adenocarcinoma, melanoma, nasopharyngeal carcinoma, antral carcinoma, etc.

Inhalation of, for example, tobacco, cannabis, heroin, wood dust, chromium or mercury may also cause epistaxis.

General causes

These are usually associated with pathology affecting vessel walls, platelets or coagulating factors. In addition, medications such as anticholinergic drugs, topical steroids, antihistamines, non-steroidal anti-inflammatory drugs (NSAIDs) and cytotoxins (methotrexate) can cause rhinitis medicamentosa and thus epistaxis.

Vessel walls

Hypertension predisposes to epistaxis by causing a replacement of the tunica media with collagen fibres.

This varies from interstitial fibrosis to almost complete replacement of muscle by scar tissue, so that the blood vessel fails to contract. Active epistaxis at emergency presentation has been shown to be associated with arterial hypertension. However, except in individuals with known or obvious hypertension, it can be difficult to determine whether the elevated blood pressure is causing the bleeding or if the active bleeding is causing the blood pressure to increase as a result of associated anxiety and stress.

Other vessel wall diseases include familial haemorrhagic telangiectasia, connective tissue diseases such as Ehler-Danlos syndrome, and vitamin C deficiency.

Platelet disorders

These can be due to acute myeloid or lymphocytic leukaemia, or autoimmune disease (autoimmunothrombocytopenia, Glanzman's thrombocytopenia, Bernard-Soulier syndrome).

Coagulating factors

Diseases affecting coagulating factors are congenital (haemophilia A or B, von Willebrand's disease) or acquired. Acquired causes include chronic liver disease, uraemia, renal dialysis, anticoagulant therapy (warfarin, heparin), consumptive coagulopathy, septicaemia, haemolytic transfusion reaction and snake envenomation.

INVESTIGATIONS

Prompt general examination is of primary importance. Haemodynamic evaluation for tachycardia, hypotension or light-headedness should be completed immediately. Hypovolaemic patients should be resuscitated with fluids and packed red blood cells.

Further investigations are requested at various stages, depending on the clinical condition of the patient. In acute severe epistaxis, blood grouping and cross-matching is paramount for immediate blood replace-

ment. There should be urgent estimation of packed cell volume (to assess baseline profile), full blood count, clotting time (normal is 2-7 minutes), bleeding time (normal is 2-4 minutes), prothrombin time (normal is 10-14 seconds), partial thromboplastin time and kaolin (normal is 35-45 seconds).

In chronic mild to moderate epistaxis, as well as in stabilized cases of acute severe epistaxis, plain radiography should follow and include the paranasal sinuses (usually 2 weeks after bleeding has stopped) and post-nasal space. When facilities are available, computerized tomography and magnetic resonance imaging (MRI) of the nose, nasopharynx, paranasal sinuses and adjacent structures yield useful information on both soft-tissue and bony lesions (MRI produces better soft-tissue resolution). Endoscopy, biopsy and histopathological studies of tumours may be helpful.

MANAGEMENT

Traditionally, management involves detailed history taking, examination, investigation and treatment. Patients may present as emergencies with acute severe epistaxis or as cold cases with recurrent mild epistaxis.

The traditional management protocol may not be followed in acute moderate and severe epistaxis. The priorities are resuscitation, identification of the site of bleeding, control of bleeding, treatment of identifiable causes and follow up.

Frequently, recurrent mild epistaxis has already been controlled before arrival at hospital. What the patient needs is usually reassurance and haematological investigations to determine the cause of the bleeding. Occasionally, radiological and endoscopic investigations may be required. Treatment is usually as an outpatient; in all cases, it is wise arrange follow up for 3-6 months, after which the patient can be discharged if there are no further incidents.

Resuscitation

Individuals presenting with acute bleeding undergo a brief assessment according to their clinical state. In the critically ill who have lost a significant amount of blood, a patent airway and adequate breathing and circulation are the first priorities, requiring efficient suctioning and bright illumination. An epistaxis tray is important, normally containing all the items necessary to control bleeding and to ensure universal precautions regarding blood and body fluids, since patients with acute epistaxis are potential hazards in terms of spread of hepatitis B and AIDS. Good venous access should be established for collection of blood specimens and for fluid replacement therapy, which can initially be in form of normal saline, Ringer's lactate and isoplasma. Observations of pulse, blood pressure and general condition are made to gauge the extent of blood loss and the need for blood replacement.

The nose is examined with the patient in Trotter's position (i.e. sitting upright and slightly inclined forward with the mouth open), breathing quietly through it and spitting out any blood in the throat. Swallowing is discouraged. The cavity is examined (nasopharyngoscopy is best, if available). Suction clearance of the nasal cavity should be performed to ensure proper identification of the bleeding site.

Control of bleeding

Non-surgical methods

These interventions are usually adequate.⁷

Initial measures involve gentle medial pressure on both alae of the nose, enough to compress vessels on the septum, for bleeding from Little's area. An ice pack applied to the forehead and nasal bridge may be helpful, as this produces reflex vasoconstriction. If these simple methods do not control the bleeding, anterior rhinoscopy can be carried out to visualize and cauterize the bleeding

point, using either a chemical or an electrical cautery. When cauterizing with silver nitrate sticks, these should be held in place for approximately 30 seconds, but should not be used for bilateral septal bleeds.⁷ Complications of cauterization include nasal synechia and septal perforation.⁵

Nasal packing is the placement of an intranasal device that will apply constant local pressure. Anterior and/or posterior nasal packing, depending on the identified bleeding site, may be carried out. Vaseline gauze, gauze-stuffed glove fingers or gauze impregnated with bismuth iod-oform paraffin paste may be employed, or balloons such as Simpson's, Brighton's or Foley's catheters. The gauze (2 m) is placed with forceps. For an anterior pack, the first layer is positioned on the nasal floor and subsequent layers on top, until the nose is tightly packed. The posterior pack consists of a gauze pad launched into the posterior nasopharynx and lodged against the choana via the oral cavity (a gauze pad sutured to a rubber catheter is inserted through the nose, and the suture is used to pull it into the back of the nose).⁷ Avoiding over-inflation of the balloon is important, because this can cause pain and displacement of the soft palate inferiorly, interfering with swallowing.⁸ The pack is removed after 48-72 hours. If bleeding reoccurs, the pack can be re-inserted for another 48-72 hours.

Antibiotic prophylaxis, analgesics and decongestants are administered to reduce complications of nasal packs. The complications of anterior packing include synechia, rhinosinusitis, toxic shock syndrome and eustachian tube dysfunction; those of posterior packing are synechia, rhinosinusitis, toxic shock syndrome, eustachian tube dysfunction, dysphagia, scarring of nasal ala and columella, hypoventilation and sudden death.⁵ Any patient receiving a posterior pack should be admitted to the hospital. It is generally understood

that older people, children and those with co-morbidities should be admitted to an intensive care unit for cardiopulmonary monitoring if a posterior pack is being placed. Newer devices have airways built in, which allow airflow through the nasal cavity despite the packing.⁷ If bleeding persists despite nasal packing, the patient is reassessed with a detailed history and examination.

At this time some of the results of investigations requested may be available, and these may give an additional lead to the likely cause of the epistaxis. Treatment of these causes may include replacement of deficient clotting factors in bleeding disorders with cryoprecipitates or fresh whole blood; withdrawal of drugs such as anticoagulants; and administration of haemostatic agents such as epsilon aminocaproic acid, ethamsylate or vitamin K.

Surgical methods

Indications for surgery have been widely debated, but usually include failure of medical treatment after 72 hours, nasal anatomy that precludes local treatments and patient refusal of medical management.⁷

The surgical options include examination under anaesthesia, transmaxillary internal maxillary artery ligation, transoral internal maxillary artery ligation, anterior/posterior ethmoid ligation, external carotid ligation, transnasal endoscopic sphenopalatine artery ligation and submucosal suprapericardial septoplasty.⁸

Careful endoscopic examination under general anaesthesia may be considered if packing fails because of inadequate placement (owing to either lack of cooperation by the patient, who may be a child, or anatomical factors such as a deviated septum). Bleeding sites can be cauterized under endoscopic guidance, a deviated septum can be straightened, spurs can be removed and meticulous packing can be placed.⁵

Transmaxillary internal maxillary

artery ligation was first proposed in the 1920s.⁸ It soon became the surgical option of choice for recalcitrant epistaxis. Traditionally, the internal maxillary artery is accessed transantrally via a Caldwell-Luc approach. With the help of an operating microscope, the posterior sinus wall is removed in a piecemeal fashion, and the posterior periosteum is carefully opened. The pterygopalatine fossa contents are exposed. The internal maxillary artery and three of its terminal branches (i.e. sphenopalatine, descending palatine and pharyngeal) are elevated using nerve hooks, and then clipped. The posterior sinus wall is packed with gelfoam, and the gingivobuccal incision is closed. Disadvantages of the procedure include unsuitability for children and individuals with hypoplastic maxillary sinuses or comminuted facial fractures, and the requirement for a general anaesthetic. There is a 10-15% technical failure rate. Other complications (25-30%) include anaesthetic risks, pain in teeth, damage to teeth, damage to the sphenopalatine ganglion or vidian nerve, damage to the infraorbital nerve, oroantral fistula and sinusitis.^{5,8,9}

Transmaxillary internal maxillary artery ligation by the transoral approach is indicated in cases of mid-face trauma, hypoplastic antra or maxillary tumours. The posterior portion of the maxilla is exposed through a posterior gingivobuccal incision, beginning at the second molar; blunt dissection is performed with fingers and the buccal fat is dissected or retracted. After the temporalis muscle has been split and partially dissected, the internal maxillary artery is visualized at the base of the wound or brought into the field by a nerve hook, and is clipped and divided. The ligation site is more proximal than with the transantral approach, resulting in a greater chance of failure due to collateral circulation. The procedure frequently results in trismus that may take up to 3 months to

resolve, due to manipulation of the temporal muscle, and there may be damage to the infraorbital nerve. Other complications include anaesthetic risks, cheek numbness and tongue paresthesiae.^{5,8,9}

The endoscopic method of internal maxillary artery ligation involves a large, middle-meatal antrostomy to expose the posterior sinus wall. The middle turbinate can be resected partially to ensure adequate exposure. The remaining steps are similar to the traditional transantral approach, with the disadvantage that skills with endoscopic instruments are required.^{5,8,9}

Anterior/posterior ethmoid artery ligation is performed for patients with superior bleeding or, in conjunction with internal maxillary artery surgery, in cases where the source of bleeding is not determined. The approach is via a Lynch incision, and the periosteum is carefully dissected from the medial orbital wall. The anterior ethmoid artery usually is found 22 (range, 16-29) mm from the anterior lacrimal crest. If clipping the artery does not stop the bleeding, the posterior ethmoid artery may be ligated. This artery is found approximately 12 mm posterior to its anterior counterpart. It should be clipped, not cauterized, because it is only 4-7 mm anterior to the optic nerve. The effectiveness of this procedure is uncertain. Complications follow in 25-30% of cases, and include anaesthetic sequelae, rhinosinusitis, lacrimal duct injury, telecanthus, stroke, blindness, ophthalmoplegia and epiphora.^{5,8,9}

The technique of external carotid ligation is simple and the anatomy familiar to most otolaryngologists. Ligation can be performed under local or general anaesthesia. A horizontal skin incision is made between the hyoid bone and the superior border of the thyroid cartilage. Subplatysmal skin flaps are raised, and the sternocleidomastoid muscle is retracted posteriorly. The carotid sheath is opened and its contents

exposed. The external carotid artery is identified by following the internal carotid for a few centimetres and dissecting the external carotid beyond its first few branches. After the external carotid has been positively identified, it usually is ligated just distal to the superior thyroid artery. Continued bleeding after ligation may be from anastomoses with the contralateral carotid system or with the ipsilateral internal carotid artery. However, this procedure is seldom performed because, being just proximal to the bleeding site, it is less effective than other ligations because of greater collateral blood flow; in addition, it is associated with more severe complication risks.^{5,8,9}

Transnasal endoscopic sphenopalatine artery ligation has received more attention in the recent literature. (The endoscopic technique also can be used to ligate the sphenopalatine artery at its exit from the sphenopalatine foramen.) An incision is made just posterior to the posterior attachment of the middle turbinate bone, in a subperiosteal plane. The mucosal flap is carefully elevated to reveal the sphenopalatine artery, which is clipped and ligated. The mucosa is then replaced. This is perhaps the most distal treatment and is very effective in bleeding from this artery. Many studies report an efficacy rate of 87-100%, with no complications. Although early authors found the approach fairly difficult, more recent reports indicate that it should be fairly straightforward for the skilled endoscopist.^{5,8,9} Audit shows that endoscopic sphenopalatine artery diathermy also is a safe, successful and effective management option for patients with refractory epistaxis. Morbidity is reduced and the hospital stay is shortened. Sphenopalatine artery diathermy can be combined with anterior ethmoid surgery when necessary.^{8,10}

Submucosal supraprericchondrial septoplasty may be indicated for patients whose nasal deformity is

PRACTICAL POINTS

- History: ask specific questions about severity, frequency, duration and laterality of the nosebleed.
- Physical examination: perform a thorough head-and-neck examination if the patient's condition permits.
- Resuscitation: ensure patent airway, adequate breathing, restoration of optimal circulation, appropriate drugs.
- Identify the bleeding site by traditional or endoscopic methods.
- Control bleeding by first aid, medical and surgical measures.
- Follow up, with definitive investigations, patient counselling and monitoring, and prognostication.

judged to be the cause of their epistaxis. Fibrosis results in decreased vascularity.^{5,8,9}

Selective embolization of the of the external carotid system, internal maxillary artery and sometimes the facial artery may be performed as either a first-line treatment for poor surgical candidates or as a second-line treatment where surgery has failed. Suitable cases include patients who cannot tolerate anaesthesia, those who have had failed surgery or who have generalized bleeding disorders, or patients with bleeding sites that are difficult to access. Embolization is most effective in cases of epistaxis refractory to arterial ligation, and is contraindicated in severe atherosclerosis. The procedure is effective, safe and quick. A catheter is introduced via the femoral artery and advanced to the level of the external carotid artery. Angiography identifies the target arteries, and may show the location of the nasal bleed if this is more than 0.5 ml/min. Platinum coils, polyvinyl alcohol, gelfoam particles or coiled springs are then released to cause embolization of the target arteries. Initially, this intervention was thought to be indicated only in cases of failed surgery. However, recent literature supports embolization as first-line treatment with or without surgical management. This change is primarily due to safer procedures (coils and pre-embolization angiography), and accumulation of experience. Currently, embolization is effective

in 82-100% of cases where rate of blood loss is more than 0.5 ml/min. Minor complications occur in 0-45% of cases (facial pain, paraesthesiae, trismus, facial paralysis, skin necrosis, groin haematoma, etc.) and resolve in 1 week. Major complications have ensued in 0-3% of cases (cerebral vascular accident, paraplegia, blindness), all of which resolved.^{5,8,11} In many centres, embolization has become the procedure of choice for patients with severe refractory epistaxis. However, not all hospitals employ an interventional radiologist who can perform it, and in such situations surgical management is the only option.

Coagulation with potassium-titanyl-phosphate or neodymium:yttrium-aluminum-garnet lasers in the treatment of hereditary haemorrhagic telangiectasia is palliative, because the underlying defect is not curable.^{5,9}

This review suggests that a better success rate, a comparable complication rate, and cost savings can be achieved with surgical intervention as the first-line treatment for intractable epistaxis, compared with traditional anterior-posterior packing and embolization.¹¹

Follow up

Patients should be counselled to avoid strenuous activity, hot and dry environments, hot and spicy foods, digital trauma, nose blowing and excessive sneezing, to sneeze gently with the mouth open and, for 24

hours after epistaxis, to sleep with their head raised, either in a recliner or propped up in bed. Aspirin and other NSAIDs should be avoided.¹²

Prognosis

The prognosis is good but variable. With adequate supportive care and control of underlying medical problems, most patients may not experience any rebleeding. Others may have minor recurrences that resolve spontaneously or with minimal self-treatment. A small percentage of individuals may require repacking or more aggressive treatments. Those with hereditary haemorrhagic telangiectasia tend to have multiple recurrences, regardless of treatment method.¹²

CONCLUSION

The management of epistaxis depends on the clinical state of the patient at presentation. The simplest method that can control the bleeding is employed; difficult cases should promptly be referred to the appropriate specialists to reduce morbidity and mortality from this common emergency.

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