Epistaxis

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Abstract

Epistaxis is a common problem that affects the whole population. The majority of cases are self-limiting and do not require any medical assessment or treatment. Epistaxis may not resolve with first aid measures, or the episodes may be frequent and thus the patient presents to emergency medicine or their general practitioner. Some patients require assessment and treatment by ENT, either in the outpatients clinic or require an unscheduled (emergency) admission to hospital. We provide an overview of the management of epistaxis in the outpatients clinic and during an unscheduled admission in paediatric and adult patients. We highlight the key considerations in the history and management, and the rare conditions to be aware of that are associated with epistaxis. Medical and surgical management are discussed. We will briefly outline first aid measures that can be performed by the patient and expected management of the patient in the community.

Keywords Adult; antithrombotic medication; cautery; epistaxis; hypertension; nasal packing; neomycin; paediatric; sphenopalatine artery

Epistaxis is the most common cause of unscheduled (emergency) admission within otolaryngology.[1] Its true incidence in the general population is unknown as most cases are self-limiting and do not require any medical input. The recent British Rhinological Society consensus recommendations on epistaxis identified a presentation to hospital rate of 25,000 cases per year.[2] In the adult population there are at least 1400 admissions per month within the United Kingdom.[3] Epistaxis is a condition that affects all ages and may be managed by the patient without medical input, by the general practitioner, by the emergency department, within the ENT outpatients' setting or may require an unscheduled (emergency) inpatient admission. The common areas of assessment and treatment will be discussed below followed by a focus on specific treatment in each of the above scenarios.

History

The history taken by a clinician in general practice, the emergency department and by an ENT clinician all have common elements, which are described below. The side of epistaxis should be determined; however, both sides of the nose must be examined. The duration of a typical episode should be sought as well as the frequency of the episodes.

Epistaxis may be due to local causes within the nose such as facial or digital trauma, prolonged inhalation of dry and/or cold air, intranasal substance misuse, altered laminar flow due to septal abnormalities (e.g. septal deviation or septal perforation). Medication such as antithrombotic medication (Table 1)[4] or systemic causes that affect coagulation can also cause epistaxis. A past medical history should also be sought; in particular, any cardiovascular events, the indication for antithrombotic therapy and a history of hypertension or diabetes mellitus. Liver disease (associated with platelet dysfunction and impaired clotting factor production due to inadequate protein synthesis) and pregnancy (secondary to increased nasal blood flow) are also associated with epistaxis. A history of previous treatment or previous nasal surgery should also be determined. The presence of unilateral, permanent nasal obstruction may indicate a neoplastic cause for epistaxis.

The National Epistaxis audit of patients admitted with epistaxis, identified the common comorbidities of hypertension (55%), ischaemic heart disease (30.4%) and diabetes mellitus (14.4%).[3] Hypertension is the most common comorbidity in the United Kingdom and affected 7.4 million in 2011. The number of adults in the United Kingdom with multiple comorbidities is estimated to be 2.9 million in 2018.[5] Thus the potential burden of epistaxis is high due to the prevalence of hypertension and associated antithrombotic medication, and patients with multiple comorbidities.

A personal or family history of bleeding disorders, easy bruising or prolonged bleeding should be determined. Inherited disorders that are associated with increased frequency of epistaxis episodes include von Willebrand disease (VWD), haemophilia type A, haemophilia type B and hereditary haemorrhagic telangiectasia (HHT; formerly known as Osler-Weber-Rendu disease) (Box 1). HHT will be discussed in further detail, as it can be difficult to diagnose and treat.

Hereditary haemorrhagic telangiectasia (HHT)

HHT is a rare, autosomal dominant disorder caused by defects in a TGF-β superfamily receptor. The defect causes vascular dysplasia and the formation of telangiectasia that lack contractile tissue, arteriovenous malformations and aneurysms. Telangiectasia most commonly occurs on the mucous membranes of the lips, nasal mucosa and tongue. Investigation for lung, liver and brain arteriovenous malformations should be undertaken and the potential general anaesthetic risk assessed.

Ninety-five per cent of HHT patients experience epistaxis.[6] The Epistaxis Severity Score was developed to assess severity of the condition and also demonstrate the efficacy of treatment.[6] Instrumentation and packing of the nose should be avoided unless absolutely necessary in order to prevent further trauma and worsening haemorrhage. Conservative therapy includes adequate hydration, barrier creams, topical emollients and nasal obturators. Oral tranexamic acid in HHT may reduce the severity and frequency of episodes. Surgical treatment includes KTP LASER or electrocautery to telangiectasia, septodermoplasty or Young's procedure.

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Management of bleeding in patients on antithrombotic agents (After Makris *et al.*, 2013)[4]

Class of drug	Drug	Mechanism of action	Oral bioavailability	Onset of action	Peak plasma level/effect	Elimination half life	Effect on platelets	Restoration of platelet function	Management of bleeding		
									Reversal agent	Four factor prothrombin complex concentrate (e.g. Beriplex or Octaplex)	Other measures and considerations
Antiplatelet medication - Decrease platelet aggregation and inhibit thrombus formation in the arterial circulation where blood flow is faster thus thrombi are composed mainly of aggregated platelets with little fibrin	Aspirin	COX inhibitor	50–75%	<1 hour 3–4 hours with enteric preparations	1–3 hours	20 minutes	Irreversible platelet inhibition – lasts 4 days	5–7 days	No specific reversal agent		Efficacy of transfusion reduced General haemostatic measures Cessation of anti-platelet medication. Patients on antiplatelets usually at high risk of arterial thrombosis thus re-instate as soon as possible after haemostasis is achieved
	Clopidogrel	$P2Y_{12}$ antagonists – irreversible	>50 % plus active metabolite	4–8 hours		6 hours	Irreversible platelet inhibition – delayed onset due to two stage hepatic metabolism	5–7 days			
	Prasugrel	$P2Y_{12}$ antagonists – irreversible					Irreversible platelet activation – one-stage activation	5–7 days			
	Ticagrelor (active form of prasugrel)	$P2Y_{12}$ antagonists – potent but reversible action	30–42%	2–4 hours	2 hours	8–12 hours	Irreversible platelet inhibition	3–5 days			
Oral anticoagulants - Alter fibrin production	Warfarin	Reduced levels of factors II, VII, IX and X				4–5 days		3–4 days	Prothrombin complex concentrate i.e. Beriplex and Octaplex – have a short half life thus need to give Vitamin K iv (5 mg) Acts faster than FFP	Rapid reversal 25–50 u/kg– can reverse warfarin induced anticoagulation within 10 minutes but have a short half life thus need to give Vitamin K iv (5 mg)	
Direct oral-anticoagulants - Alter fibrin production - Do not require regular monitoring	Rivaroxaban	Direct oral Xa inhibitor			3 hours	7–11 hours			No antidote	Consider in life-threatening situations	**Minor bleeding** Withhold medication, supportive measures, surgery, fluid replacement **Life-threatening bleeding** Consider PCC/APCC and rFVIIa
	Apixaban	Direct oral Xa inhibitor			3 hours	12 hours					

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