10.5005/jp-journals-10001-1056

ORIGINAL ARTICLE

Comparison of Blind Nasal Packing *vs* Endoscopic Control of Epistaxis in an Emergency Setting

[1]Sunita Chhapola, [1]Inita Matta, [2]Pratima Marker

[1]Surgeon, Department of ENT, Mumbai Port Trust Hospital, Mumbai, Maharashtra, India

[2]Resident, Department of ENT, Mumbai Port Trust Hospital, Mumbai, Maharashtra, India

Correspondence: Sunita Chhapola, Surgeon, Department of ENT, Flat No. 4, Dhanvantari, Mumbai Port Trust Hospital Campus, Wadala (E), Mumbai-400037, Maharashtra, India, Phone: 91-22-66567904, Fax: 91-22-24145115, e-mail: drsunita7ent@yahoo.co.in

ABSTRACT

Aim: To compare the precision and efficacy of endoscopic control versus nasal packing in epistaxis presenting to the emergency room.

Design: Open labelled randomized controlled trial for comparison of precision and efficacy of emergency blind nasal packing with primary endoscopic control of epistaxis.

Subjects: A total of 160 consecutive patients of epistaxis in the age group of 40 to 70 years were randomized in two groups (A and B) of 80 patients each. Group A was subjected to blind nasal packing and group B to endoscopic procedure. About 48 (30%) patients were alcoholic, 64 (40%) were hypertensive and 48 (30%) patients did not have any overt predisposing factor. Bleeding time, clotting time, prothrombin time, partial thromboplastin time and international normalized ratio (INR) were done in all patients to rule out coagulation diseases.

Results: The nasal pack of patients in group A was removed after 48 hours. The nose was endoscopically examined on 3rd day, 7th day and then 1 month after the epistaxis. A total of 44 (55%) patients of group A had nasal mucosal abrasions ($p < 0.05$), two (2.5%) patients had secretory otitis media ($p > 0.05$) and 10 (12.5%) had synechiae formation ($p > 0.05$). A total of 28 (35%) patients from group A had one episode of rebleed after nasal pack removal. Group B had no complications.

Conclusion: Epistaxis presenting to the emergency room can be precisely and effectively controlled endoscopically. Clumsy nasal packing, complications and subsequent hospitalization costs are thereby reduced.

Keywords: Epistaxis, Nasal packing, Endoscopy.

INTRODUCTION

Epistaxis is one of the commonest otorhinolaryngological emergencies and broadly divided into 'anterior' and 'posterior' types. It is the latter, originating from the large caliber sphenopalatine artery or its branches, which is often difficult to control in the emergency room. The fact that the patient may be elderly and suffering from concomitant ischemic heart disease, hypertension, age related problems and on antiplatelet agents like aspirin, compounds the problem. This type of epistaxis is alarming both for the patient and the emergency staff and results in hurried, rough and blind nasal packing. As a result, a single bleeding point may be converted into a large abraded bleeding area. Given the nature of the nasal mucosa and its recesses, the packing often does not reach the bleeding point, resulting in repeated and ineffective repackings.

Endoscopic control of epistaxis as a primary measure is accurate and effective. Special attention should be given to areas prone to bleeding, e.g. above the middle turbinate, sphenopalatine foramen and Woodruff's plexus.

MATERIALS AND METHODS

Out of 12,305 patients attending ENT emergency at our hospital from 06 January to 09 December, 160 patients presented with epistaxis. Hence, the incidence of epistaxis in our set-up is 0.65%. Patients were randomly divided in two groups, group A–blind nasal packing was done and for group B–endoscopic control of epistaxis was done with either silver nitrate, 50% trichloroacetic acid, electrocautery or CO_2 laser. Patients included in our study belonged to age group between 40 and 70 years of which 116 (72.5%) were male and 44 (27.5%) were female. A total of 136 (85%) patients presented with bilateral epistaxis and 24 (15%) with unilateral epistaxis.

In 88 patients, septum was the site of bleed. Twelve (7.5%) patients bled from the anterior septum and 76 (47.5%) patients bled from the posterior septum. 72 (45%) patients presented with bleeding from lateral wall of nose, 36 (22.5%) were from middle turbinate (Fig. 1), 12 (7.5%) from inferior turbinate and 24 (15%) from Woodruff's plexus (Table 1).

Out of 160 patients of epistaxis, 48 (30%) patients were alcoholics, 64 (40%) were hypertensive and 48 (30%) patients did not have any obvious predisposing factor. On presentation, all patients underwent a detailed history alongwith thorough systemic and ENT examination. Hemogram, liver profile, ECG and specific coagulation profile were done for all patients, including bleeding time, clotting time, prothrombin time and partial thromboplastin time.

Fig. 1: Bleeding from middle turbinate

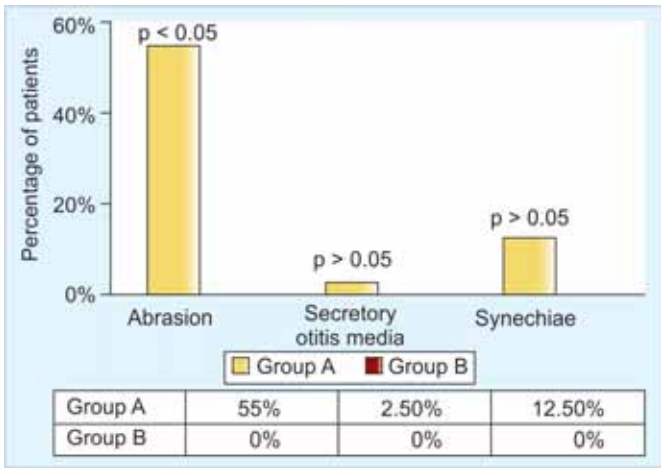

Fig. 2: Complications

Table 1: Site of epistaxis

		n (%)
Septum 88 (55%)	Anterior	12 (07.5%)
	Posterior	76 (47.5%)
Lateral wall of nose 72 (45%)	Middle turbinate	36 (22.5%)
	Inferior turbinate	12 (07.5%)
	Woodruff's plexus	24 (15.0%)

Patients were randomly divided into two groups. For group A, nasal packing with paraffin soaked ribbon gauze was done and antibiotic cover was given for 48 hours. The nasal packs were removed after 48 hours and repacking done where required. In group B, endoscopic control was achieved. Bleeding vessels were cauterized with silver nitrate, 50% tricholoroacetic acid, electrocautery or CO_2 laser. The nose was not packed and the patient was discharged. Endoscopic examination was done for all patients on 3rd day, 7th day and after 1 month. Associated medical conditions like hypertension and alcoholic coagulopathies were managed by the physicians.

RESULTS AND ANALYSES

A total of 48 (60%) patients of group A had a dry mouth, 68 (85%) had discomfort and 36 (45%) had halitosis due to mouth breathing. Nasal pack was removed for patients of group A on 3rd day. Total 28 (35%) patients had rebleeding, which was then controlled endoscopically, with no rebleeding thereafter. On the 3rd day, 44 (55%) patients of group A had abrasions ($p < 0.05$) on their nasal mucosa. Two (2.5%) patients of group A had secretory otitis media ($p > 0.05$), which was treated by antibiotics and decongestants. Ten (12%) patients of group A had synechiae ($p > 0.05$) at 1 month which was released under local anesthesia.

A total of 72 patients (90%) in group B had small ulcer at the site of cautery at 1 week. The ulcer healed completely within one month. Nasal mucosa abrasions, secretory otitis media and synechiae were not encountered in patients of group B (Fig. 2). None of the patients in either group had septal perforation, facial numbness or toxic shock syndrome.

DISCUSSION

Epistaxis can be primary (no causal factors) or secondary (trauma, surgery, anticoagulant overdose, hypertension, etc). The site of bleeding can be anterior nasal or posterior nasal. In anterior epistaxis, the source is anterior to the plane of pyriform aperture. This includes anterior septum, vestibular skin and mucocutaneous junction. Anterior epistaxis is usually from Kiesselbachs plexus[1] at the Little's area, which is an arterial plexus of four vessels on the septum, i.e. septal branch of sphenopalatine artery, superior labial artery, greater palatine artery and anterior ethmoidal artery. This type of epistaxis generally poses little problem and usually stops by pinching the ala nasi, called Hippocratic technique.

Posterior epistaxis is from vessels posterior to the pyriform aperture. These are commonly profuse and troublesome. The most common sites of posterior bleeding are on the lateral wall of nose, posterior septum, nasal floor, sphenopalatine foramen and Woodruff's plexus. Above the middle turbinate, there is anastomosis of branches of sphenopalatine artery and ethmoidal arteries. The sphenopalatine artery enters the nose through the sphenopalatine foramen and can bleed profusely. Woodruff's plexus[2] is a collection of large blood vessels in the posterior part of the inferior meatus. These vessels originate from the posterior pharyngeal wall and are venous in origin.[3] These areas can be seen easily with the endoscope.

The nose is a recessed cavity, made uneven by the presence of turbinates and the folding pattern of its lining mucosa. The mucosa and the turbinates are highly vascular and therefore tend to bleed. A profusely bleeding patient is often treated with rough and blind nasal packing in an attempt to stop the bleed. As the nose is not a flat area, blind nasal packing may often not reach the actual bleeding site. Gauze can be abrasive and gelfoam may simply be

inadequate in giving the required pressure at the bleeding point. The situation is made worse as patients are often elderly, anxious and have already swallowed considerable amount of blood. In addition, they may have hypertension, ischemic heart disease or altered coagulation profile due to use of antiplatelet drugs and aspirin.

Nonsteroidal anti-inflammatory drugs (NSAIDS) cause antiplatelet aggregation due to altered platelet membrane physiology.[4-6] Alcoholism can cause prolongation of the bleeding time despite normal platelet counts and coagulation profile with altered liver function tests. These patients have either dilated blood vessels (Fig. 3) or telangiectatic changes (Fig. 4) on the nasal mucosa. Increased blood pressure is observed in almost all epistaxis patients. However, studies have failed to show a relationship between hypertension and epistaxis.[7] Also, no correlation between epistaxis and secondary effects of hypertension or with the severity of hypertension have been proved.[8] Deviated nasal septum can also lead to recurrent epistaxis, especially during rhinitis when nasal mucosa is inflamed and edematous. This causes increased vascularity and greater friability of the vessels. One study has found an association between deviated nasal septum and recurrent epistaxis in young individuals.[9] Age related arterial muscle degeneration causes inability of bleeding vessels to contract. All these factors can result in a massive epistaxis, where the nose continues to bleed despite cold compresses and packing.

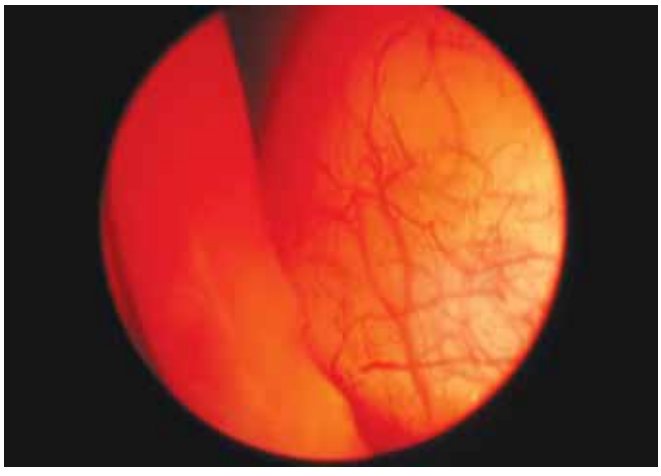

Fig. 3: Dilated blood vessels on the septum

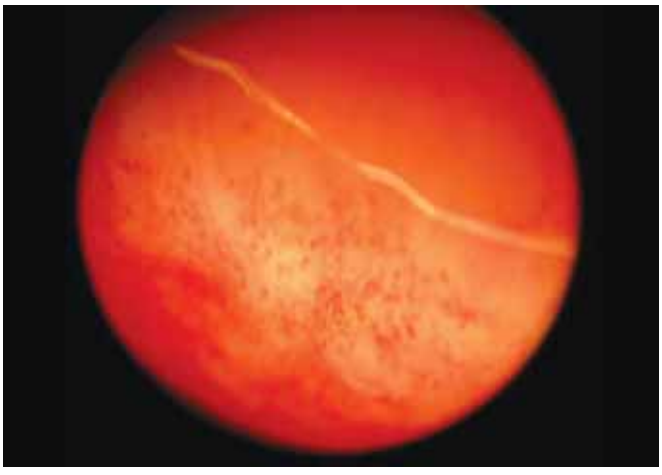

Fig. 4: Telangiectasia on the nasal septum

Management of epistaxis is multidimensional. Control of nasal bleed and treatment of co-existent medical condition should be dealt concomitantly. Direct treatment requires identification of bleeding point by nasal endoscope and stopping the bleed. Indirect methods involve nasal packing, hot water irrigation, systemic medical therapy, etc. If the above techniques fail, surgical management is required which consists of ligation techniques, septal surgeries or embolization. Since we have started doing primary endoscopic control of emergency epistaxis, we have not had a single instance where sphenopalatine clipping was required to be done.

As endoscopes are easily available at most centers now, it is advisable to use them as a primary method of epistaxis control. Control of the bleeding vessel can be done under direct vision using silver nitrate, trichloroacetic acid, electrocautery or carbon dioxide laser. Nasal packing is then not needed. However, if required, it can be done gently under endoscopic control. Only affected areas are targeted and trauma to healthy areas is avoided. Large raw areas are not created and rebleeds are minimized. We used cautery around the offending blood vessel in a circumferential manner to decrease blood flow from surrounding vessels. Bipolar diathermy is preferred as monopolar diathermy can cause blindness due to current propogation.[10] Endoscopy identifies source of posterior epistaxis in 80% cases.[11-14] A bleeding vessel lying on a septal spur, or hidden behind one, is easily tackled with an endoscope. Woodruffs plexus may be missed by blind nasal packing or even by a posterior nasal pack. These vessels are easily seen with an endoscope coupled with suction to enable vision. Control of epistaxis is immediate by endoscopy as reported in 90% cases.[13] Endoscopy management also facilitates outpatient management and decreases indoor stay.[13]

Nasal packing with paraffin gauze when used, can create raw areas in the nose. Other complications of nasal packing are:

1. Synechiae (Fig. 5)
2. Facial numbness
3. Blockage of nasolacrimal duct leading to epiphora
4. Blockage of sinus drainage leading to sinusitis
5. Blockage of nasal airway leading to hypoxia
6. Blockage of eustachian tube leading to suppurative otitis media
7. Nasovagal reflex: This reflex occurs during insertion of a pack or instrumentation of the nasal cavity, leading to vagal stimulation with consequent hypotension and bradycardia
8. Worsening of sleep apnea
9. Displacement of pack into oropharynx with risk of acute airway obstruction.

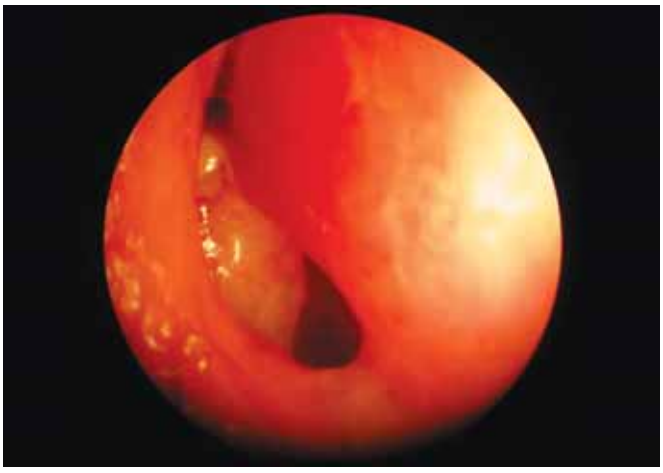

Fig. 5: Synechiae between inferior mucosa turbinate and nasal septum

Repeated packing and use of silver nitrate cautery can occasionally result in septal perforations. Other major complications of nasal packing are:

1. Cardiac arrythmias
2. Myocardial ischemia
3. Gram-negative sepsis.

Blind nasal packing if done with gelfoam may fail due to sheer inadequacy of pressure needed to stop the epistaxis. Prolonged nasal packing is undesirable in patients suffering from obstructive sleep apnea because of concern of serious life-threatening hypoxia. Continuous bleeding or rebleeding even after nasal packing is seen in 40% cases.[15] In our study, 28 (35%) patients had rebleed after nasal pack removal. 44 (55%) patients of group A had nasal mucosal abrasions ($p < 0.05$), two (2.5%) patients had secretory otitis media ($p > 0.05$) and 10 (12.5%) had synechiae formation ($p > 0.05$). The rate of complication of endoscopic control in our study was 0%. Avoidance of blind hasty nasal packing helps minimize trauma to nasal mucosa. We, therefore, feel that when epistaxis needs intervention, it should be done primarily under endoscopic vision.

CONCLUSION

Epistaxis is a very common ENT emergency and alarming for the patient. Epistaxis with accurate identification of bleeding points means good control and freedom from cumbersome prolonged nasal packing. Keeping in mind that the usual sites to look for are above the middle turbinate, at the sphenopalatine foramen, on a septal spur and in the region of Woodruff's plexus, bleeding can be dealt with by electrocautery, chemical cautery or alternatively by CO_2 laser. Precise visualization is an important prerequisite before effective control. The availability of endoscopes in almost every hospital has revolutionized the treatment of such nasal pathology. This is largely due to the superb visualization and consequent precise instrumentation it affords. Good and early control endoscopically in the first instance reduces patients discomfort, complication rates, hospital stay and consequent costs.

REFERENCES

1. Morgagni JP. The seats and causes of diseases. Alabama: Gryphon Editions Ltd 1983;1:312-54 (First published in 1761).
2. Woodruff GH. Cardiovascular epistaxis and the nasopharyngeal plexus. Laryngoscope 1949;15:1238-47.
3. Chiu TW, Shaw-Dunn J, McGarry GW. Woodruff's nasopharyngeal plexus: How important is it in posterior epistaxis? Clinical Otolaryngology 1998;23:272-79.
4. Watson MG, Shenoi PM. Drug-induced epistaxis. Journal of the Royal Society of Medicine 1990;83:162-64.
5. McGarry GW. Drug-induced epistaxis. Journal of the Royal Society of Medicine 1990;83:812.
6. Livesey JR, Watson MG, Kelly PJ, Kesteven PJ. Do patients with epistaxis have drug-induced platelet dysfunction? Clinical Otolaryngology 1995;20:407-10.
7. Lubianca-Neto JF, Bredemeier M, Carvahal EF, Arruda CA, Estrella E, Pletsch A, et al. A study of the association between epistaxis and severity of hypertension. American Journal of Rhinology 1998;12:269-72.
8. Lubianca-Neto JF, Fuch FD, Facco SR, Gus M, Fasolo L, Mafessoni R, et al. Is epistaxis evidence of end-organ damage in patients with hypertension? Laryngocope 1999;109:1111-15.
9. O'Reilly BJ, Simpson DC, Dharmeratnam R. Recurrent epistaxis and nasal septal deviation in young adults. Clinical Otolaryngology 1996;21:12-14.
10. Vandan Abeele D, Clemens A, Tassignon MJ, van der Heyning PH. Blindness due to electrocoagulation following functional endoscopic sinus surgery. Journal of Laryngology and Otology 1996;110:261-64.
11. Wurman LH, Sack JG, Flannery JV, Paulson JO. Selective endoscopic electrocautery for posterior epistaxis. Laryngoscope 1988;98:1348-49.
12. O'Leary-Stickney K, Makielski K, Weymuller EA. Rigid endoscopy for the control of epistaxis. Archives of Otolaryngology Head and Neck Surgery 1992;118:966-67.
13. O'Donnell M, Roertson G, McGarry GW. A new bipolar diathermy probe for the outpatient management of adult acute epistaxis. Clinical Otolaryngology 1999;24:537-41.
14. McGarry GW. Nasal endoscope in posterior epistaxis: A preliminary evaluation. Journal of Laryngology and Otology. 1991;105:428-31.
15. Stangerup SE, Dommerby H, Lau T. Hot-water irrigation as a treatment of posterior epistaxis. Rhinology 1996;34:18-20.