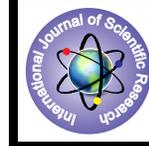


Role of Hypertension in Epistaxis



Medical Science

KEYWORDS :

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Introduction;

Epistaxis is one of the commonest ENT emergencies requiring hospital admission (15% of cases according to Timsit) [1]. Although predominantly a benign condition regardless of its etiology, epistaxis can be severe, particularly in elderly and/or frail patients, requiring hospitalization and "aggressive" management including repeated nasal packing, blood transfusion, arterial embolization or surgery [2-6]

Elevated blood pressure can contribute to epistaxis in two different ways. First, the high pressure causes chronic damage of a blood vessel wall in the nasal or sinus mucosa. Second, 20% of epistaxis patients experience elevated blood pressure because the natural response to seeing blood from one's nose is to get agitated, which can directly lead to elevation of the blood pressure. Practically, active bleeding patients in emergency department were related to hypertension and patients without active nasal bleeding had less related to hypertension. Hypertension has been considered to be a major cause of spontaneous epistaxis for a long time [7,8]. However, particularly in the recent medical literature, the relationship between hypertension and epistaxis appears to be more controversial [9-16]

MATERIAL AND METHOD;

This retrospective study was performed in the Department of ENT and Head Neck Surgery Government Medical College Srinagar J&k. A total of 140 patients were included in the study. The study inclusion criteria were:

male and female patients of any age, patients admitted to the ENT and Head and Neck surgery department with a diagnosis of serious spontaneous epistaxis requiring at least one nasal pack. The exclusion criteria were: patients with minor epistaxis easily managed by first aid measures and/or immediately successful local treatment (cauterization), post traumatic epistaxis (including iatrogenic epistaxis after nasal surgery) and patients usually followed for Hereditary Hemorrhagic Telangiectasia.

Management of epistaxis ranged from unilateral nasal packing to endoscopic surgery depending on the severity of epistaxis. Patients with systolic blood pressure (BP) higher than 160 mmHg also received Labetolol intravenous injection to control BP. Anterior and posterior nasal packing was performed with Medicated Guaze Pack/roll. Nasal packs were removed after 24-48 h if the patient did not re-bleed. In cases of re-bleeding after removal of nasal packs, nasal packing was repeated. Surgery was only indicated in the case of anemia or persistent bleeding after 2 or 3 nasal packs.

Study protocol

A history of hypertension was defined as patients treated with antihypertensive drugs. Patients with a self-reported history of epistaxis or who had been already hospitalized for at least one episode of epistaxis were considered to have a history of epistaxis. The mean of all BP measurements during hospitalization was determined, and BP that was difficult to control was defined as BP higher than 140/80 mmHg. International Normalized Ratio (INR) for prothrombin time was evaluated for 72 patients treated with anticoagulants. Epistaxis was classified as grade 1 (serious) or grade 2 (severe). Serious epistaxis was de-

defined by the need for medical management requiring hospitalization of the patient for epistaxis. Severe epistaxis (grade 2) was defined as follows:

- length of hospital stay 3 days
- nasal packing, both anterior and posterior nasal packing
- two or more nasal packs
- presence of hematologic consequences: anemia (hemoglobin < 10 g/dl) and/or blood transfusion
- patients treated by surgery

Statistical analysis

Data were expressed as mean \pm S.D., and range or frequency, as appropriate. For descriptive and analytical purposes, patients were stratified by severity of epistaxis, defined as Grade 1 or Grade 2.

Table 1

Clinical and Demographic characteristics of Study population	All N=140	Grade 1 N=62	Grade 2 N=78	P
Age (years)	52 \pm 15	50 \pm 15	54 \pm 15	0.5
Male gender, n(%)	84 (57)	32 (51)	52 (66)	0.6
Systolic blood pressure on admission (mmHg)	144 \pm 6	142 \pm 4	146 \pm 6	0.4
Diastolic blood pressure on admission (mmHg)	80 \pm 4	82 \pm 4	82 \pm 6	0.6
Number of patients in whom blood pressure was difficult to control, n(%)	77 (55)	23 (37)	54 (69)	0.3
Systolic blood pressure on discharge (mmHg)	126 \pm 6	124 \pm 4	126 \pm 4	0.4
Diastolic blood pressure on discharge (mmHg)	72 \pm 6	74 \pm 4	74 \pm 4	0.5
History of hypertension, n(%)	78 (55)	36 (58)	42 (53)	0.5
History of epistaxis, n(%)	44 (31)	13 (20)	31 (40)	0.2
Vitamin K antagonist, n(%)	27 (19)	09 (14)	18 (23)	0.3
INR (n=44)	3.1 \pm 1.6	2.9 \pm 1.2	3.1 \pm 1.4	0.4
Length of hospital stay (days)	5 \pm 2	3 \pm 2	4 \pm 3	0.3
Hemoglobin (g/dl)	11.7 \pm 1.9	12.4 \pm 1.2	10.2 \pm 1.3	0.1
Treatment with sphenopalatine artery surgical ligation or embolization, n(%)	3	0	3	NA
Treatment with more than 2 nasal packs, n(%)	22	0	22	NA

Result;

A total of 140 patients admitted to the ENT and Head and Neck Surgery Department were included. Table 1 shows the clinical characteristics of the patients according to the severity of epistaxis. No significant differences were observed between the two groups in terms of age, sex ratio, history of epistaxis and BP characteristics including history of hypertension, mean BP on admission, mean BP on discharge and number of patients in whom BP was difficult to control. Patients with more severe epistaxis had a similar exposure to anti-coagulant and platelet antiaggregant medications as patients with less severe epistaxis.

On univariate logistic regressions, no factors were independently associated with severity of epistaxis.

Discussion:

Nasal bleeding can conveniently be divided into anterior and posterior epistaxis. Anterior bleeds come out the front of the nose, whereas posterior bleeds run down the back of the nose into the pharynx. Roughly 90% of cases of epistaxis can be classified as anterior. The common sites of anterior bleeding include the anterior aspect of the nasal septum, anterior edge of the inferior turbinate and the anterior ethmoid sinus. Among them, the anterior aspect of the nasal septum is the single most common site, where sometimes referred to as Kisselbach's plexus (Little's area). Kisselbach's plexus contains a rich capillary blood supply that is at the confluence of four different arterial blood supplies, which are sphenopalatine artery, greater palatine artery, superior labial artery, and anterior ethmoid artery. Posterior epistaxis typically arises from vessels on the posterior septum, on the floor of the nose in the posterior choana, or from the back of the middle or inferior turbinate. The area at the back of the inferior turbinate is specified as Woodruff's plexus. Recently, it is known that the Woodruff plexus is a venous plexus located at the back of the inferior meatus, not an arterial plexus.

Hypertension and epistaxis commonly occur simultaneously among adults of general population. It is uncertain whether the hypertension is an etiologic factor in all of these patients. It is known that hypertension in epistaxis patients is caused by anxiety. However, one study that analyzed 200 epistaxis patients reported that 75% showed elevated blood pressure during nose bleeding and 30% was severe hypertension patients. (Herkner et al., 2000) Elevated blood pressure can contribute to epistaxis in two different ways. First, the high pressure causes chronic damage of a blood vessel wall in the nasal or sinus mucosa. Second, 20% of epistaxis patients experience elevated blood pressure because the natural response to seeing blood from one's nose is to get agitated, which can directly lead to elevation of the blood pressure. Practically, active bleeding patients in emergency department were related to hypertension and patients without active nasal bleeding had less related to hypertension.

Epistaxis has been considered to be related to hypertension for a long time. In 1959, Mitchell [7] statistically compared two groups of patients (one group of 374 patients with a history of hypertension and another group of 162 patients with acute epistaxis but with no history of hypertension and concluded that, in the absence of local nasal disease, epistaxis was associated with high BP. In 1977, Charles and Corrigan [8] confirmed this conclusion in a series of 194 patients and, more recently, Isezuo et al. [9] also showed a statistically significant association

between epistaxis and hypertension in a series of 62 patients. However, Fuchs et al. [10] in a sample of 1,174 subjects over the age of 18 years representative of Porto Alegre general population, found no association between epistaxis and hypertension. The same group, however, [11] in a study based on 323 adults with hypertension, pointed out however that duration of hypertension and left ventricular hypertrophy were associated with epistaxis, suggesting that epistaxis might be a consequence of long-lasting hypertension. In another study concerning 372 patients, Herkner et al. [14] concluded that active epistaxis in the emergency department was associated with hypertension, suggesting that hypertension was a predisposing factor for acute nose-bleed.

Knopfholz et al. [15] in a series of 36 patients, concluded that the incidence of epistaxis in hypertensive patients was not associated with the severity of hypertension. Moreover, BP readings in the setting of epistaxis were similar to those obtained in routine settings. The present series is fairly similar to other series of patients hospitalized for epistaxis published in the medical literature, but the patients of this series were slightly younger with a mean age of 52 years versus 64.3 years for Vidulich [5] and 60 years for Pollice (with 70% of patients [50 years]) [6]. 55% of the patients in the present series had a history of hypertension versus 48% for Vidulich [5] and 47% for Pollice [6]. However, this study reveals interesting results, as no significant differences were observed between the two patient groups concerning the role of hypertension. A history of hypertension was not associated with severe epistaxis versus serious epistaxis. No statistically significant difference was observed between patients with a history of epistaxis in the two patient groups (with or without a history of hypertension). No statistically significant difference was observed between BP recordings on admission between the two patient groups (with or without a history of hypertension). The severity of hypertension (BP [160/100 mmHg]) was not associated with severe epistaxis versus serious epistaxis. Blood pressure recordings were also not significantly different between the two groups of hypertensive versus non-hypertensive patients, which may indicate that, as suggested by Tan and Calhoun [3], hypertension at the time of treatment of epistaxis maybe anxiety-related, and BP returns to normal after control of epistaxis and reassurance. We can conclude like Celik et al. [16] that: the debate on BP and epistaxis will continue. The potential influence of BP levels on acute episodes of epistaxis still requires investigations. Our clinical data are not sufficient to support or dismiss the idea that epistaxis is secondary to end-organ damage caused by hypertension. This is corroborated by nasal examinations after the episode of epistaxis, since they were normal in almost all cases in our ENT experience.

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