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Dr. Ajaz Ul Haq

Assistant Professor, Department of ENT, Shri Guru Ram Rai Institute of Medical & Health Sciences, Dehradun, Uttarakhand, India

Dr. Aneesa Afzal

Senior Resident, Department of ENT, Government Medical College, Srinagar, Jammu and Kashmir, India

Dr. Rishabh Dogra

Senior Resident, Department of ENT, Shri Guru Ram Rai Institute of Medical & Health Sciences, Dehradun, Uttarakhand

Dr. Akanksha Bisht

Department of ENT, Shri Guru Ram Rai Institute of Medical & Health Sciences, Dehradun, Uttarakhand, India

Dr. Fatma Anjum

PG ENT, Department of ENT, Shri Guru Ram Rai Institute of Medical & Health Sciences, Dehradun, Uttarakhand, India

Dr. Sonal Kala

PG ENT, Department of ENT, Shri Guru Ram Rai Institute of Medical & Health Sciences, Dehradun, Uttarakhand, India

Dr. Sajad Majid Qazi

MS ENT, Professor, Department of ENT, Government Medical College, Srinagar, Jammu and Kashmir, India

Corresponding Author:

Dr. Ajaz Ul Haq

Assistant Professor, Department of ENT, Shri Guru Ram Rai Institute of Medical & Health Sciences, Dehradun, Uttarakhand, India

Role of hypertension in epistaxis

Ajaz Ul Haq, Aneesa Afzal, Rishabh Dogra, Akanksha Bisht, Fatma Anjum, Sonal Kala and Sajad Majid Qazi

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Abstract

Background: The aim of the study was to study the role of hypertension in patients hospitalized for serious epistaxis.

Methodology: This is a retrospective study conducted over duration of 2 years from August 2014 to July 2016 over 140 patients hospitalized for serious epistaxis. Epistaxis was classified into serious and severe type. The following parameters were recorded: age, gender, blood pressure recordings, patients in whom blood pressure is difficult to control, duration of hospital stay, history of hypertension, severity of epistaxis, including medical and/or surgical management, medications affecting clotting.

Results: Mean age of our patients was 52 years, 55% of the patients had a history of hypertension. There was no statistically significant difference between patients with a history of epistaxis in the two groups (with or without a history of hypertension). There was no statistical significance between blood pressure recordings on admission between the two groups. The severity of hypertension was also not associated with severe epistaxis versus serious epistaxis.

Conclusion: Hypertension does not appear to be causal factor of serious epistaxis.

Keywords: Epistaxis, hypertension, blood pressure

1. Introduction

Epistaxis or nose-bleed is one of the commonest Otorhinolaryngology emergencies requiring hospital admission. It can be post traumatic, iatrogenic or spontaneous. Timsit *et al.* [1] reported 15% of cases of epistaxis amongst the hospital admissions. Although nose-bleeds are predominantly benign, but can be severe, especially in elderly and/or debilitated patients, requiring hospitalization and aggressive treatment, including repeated nasal tamponades, blood transfusions, arterial embolization or surgery [2-5]. Hypertension and epistaxis commonly occur simultaneously among adults and hypertension has been considered as a major cause of spontaneous nose-bleed. It is not known whether hypertension is an etiological factor in all patients of epistaxis. However, it is known that hypertension in nose-bleeds is caused by anxiety. Herkner *et al.* [6] found that maximum patient of epistaxis showed elevated blood pressure and some patients had severe hypertension. High blood pressure can cause epistaxis in two different ways. Firstly, high blood pressure causes chronic damage to the blood vessel wall of sinuses or nasal mucosa. Secondly, 20% of patients with nose-bleeds experience high blood pressure because the natural reaction to nose-bleeds is agitation, which can directly lead to elevation of blood pressure. In practice, patients with active bleeding in the emergency department were associated with hypertension, while patients without active epistaxis were less associated with hypertension. Although, in few medical literatures, [7-11] the relationship between epistaxis and hypertension appears to be controversial. However, there are studies which showed that epistaxis is a true symptom of hypertension and it has been considered to be a major cause of spontaneous epistaxis [12]. We conducted this retrospective study on 140 patients who were admitted for serious epistaxis with an aim to investigate the role of hypertension in these patients.

2. Material and Methodology

This study was conducted over a duration of 2 year from August 2014 to July 2016 after the approval from institutional ethical committee. A total of 140 patients were included in the study. All patients irrespective of any age and gender, who were admitted to the Otorhinolaryngology department with a diagnosis of serious epistaxis requiring at least one nasal pack were included in the study.

The patients with minor epistaxis who were easily managed by the first aid measures and/or immediately successful local treatment (cauterisation), post traumatic epistaxis (including iatrogenic epistaxis after nasal surgery) and follow up patients of Hereditary Hemorrhagic Telangiectasia were excluded from the study. Depending on the severity, epistaxis was managed either by unilateral nasal packing or endoscopic surgery or embolisation. Patients with systolic blood pressure (BP) more than 160mmHg were given intravenous Labetelol. Anterior and posterior nasal packing was performed with medicated gauze pack/roll. If the patient did not re-bleed, the nasal packs were removed after 24-48 hours. In cases of re-bleeding after removal of nasal packs, nasal packing was repeated. Surgery was only indicated in cases of persistent bleeding after 2 or 3 nasal packs and in the case of anemia. Hypertension in epistaxis was considered in patients who were treated with anti-hypertensive drugs with a self-reported history of epistaxis or who had been already hospitalized for at least one episode of epistaxis. The mean of all blood pressure measurements during hospitalization was determined and blood pressure that was difficult to control was defined as blood pressure 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 or serious epistaxis and grade 2 or severe epistaxis. Serious epistaxis was defined by the need for

medical management requiring hospitalization for the patient of epistaxis. Severe epistaxis was considered in the following situation- Length of hospital stay 3 days or nasal packing using double balloon nasal catheter or two or more nasal packs required or presence of hematologic consequences: anemia (hemoglobin 10g/dl) and/or blood transfusion or epistaxis controlled by embolization or surgery. Data were expressed as mean±standard deviation and range or frequency, as appropriate. For descriptive and analytical purposes, patients were categorized as Grade 1 or Grade 2.

3. Result

A total of 140 patients were included in the study amongst which 57% were male. 62 patients belong to grade 1 and rest were of grade 2. Mean age was 52±15 year. No significant differences were observed between the two groups in terms of age, gender, history of epistaxis and blood pressure characteristics including history of hypertension, mean blood pressure on admission, mean blood pressure on discharge and number of patients in whom blood pressure was difficult to control. Patients with more severe epistaxis had a similar exposure to anti-coagulant and antiplatelet medications as patients with less severe epistaxis.(Table 1) None of the factors were independently associated with severity of epistaxis.

Table 1: Clinical and demographic characteristics of study population

Clinical and Demographic characteristics of study population	All n=140	Grade 1 n=62	Grade 2 n=78	P value
Age (in years)	52±15	50±15	54±15	0.5
Male gender, n (%)	84 (57)	32 (51)	52 (66)	0.6
Systolic blood pressure on admission (mmHg)	144±6	142±4	146±6	0.4
Diastolic blood pressure on admission (mmHg)	80±4	82±4	82±6	0.6
Number of patients in whom blood pressure is difficult to control, n (%)	77 (55)	23 (3)	54 (69)	0.3
Systolic blood pressure on discharge (mmHg)	126 ±6	124±4	126±4	0.4
Diastolic blood pressure on discharge (mmHg)	72±6	74±4	74±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±1.6	2.9±1.2	3.1±1.4	0.4
Duration of hospital stay (days)	5±2	3±2	4±3	0.3
Hemoglobin (g/dl)	11.7±1.9	12.4±1.2	10.2±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

4. Discussion

Epistaxis is a very common disease and can occur due to trauma, disorders in mucosa or vessels, or coagulopathy. 10% of all population experience severe epistaxis.^[13] On analyzing age wise distribution, about 30% of children aged 0-5 year, 56% of children aged 6-10year and 64% of children aged 11-15 year experience one or more episode of epistaxis.^[13] Epistaxis is classified into anterior and posterior epistaxis. Approximately 90% of nosebleeds can be classified as anterior. Anterior bleeding flows from the front of the nose, while posterior bleeding flows down the back of the nose into the pharynx. Common sites of anterior epistaxis include the anterior nasal septum, which is the most common site and also known as the Kesselbach plexus (Little region), the anterior margin of the inferior turbinate, and the anterior ethmoid sinus. The Little area contains a rich capillary blood supply that resides at the confluence of four different arterial blood supply, which include the sphenopalatine artery, the greater palatine artery, the superior labial artery, and the anterior ethmoid artery. Posterior epistaxis arise from vessels in the posterior septum, posterior

choanae, or in the posterior part of the middle or inferior turbinate. The area at the back of the inferior turbinate is termed as Woodruff plexus. A number of factors and conditions contribute to the development, severity and recurrence of epistaxis. Various possible etiologic factors for epistaxis are- environmental factors (humidity and temperature), local factors (inflammation, deviated nasal septum, perforations, foreign body, aneurysm, tumors), systemic factors (hypertension, hematological abnormalities, renal failure, arteriosclerosis, hereditary telangiectasias) and several medications affecting clotting (anticoagulants, non-steroidal anti-inflammatory drugs)^[14]. Hypertension and epistaxis usually occur at the same time in adults. It is not known whether hypertension is an etiological factor in all of these patients. For a long time, epistaxis was thought to be associated with hypertension. Mitchell^[15] compared two groups of patients (one with a history of hypertension and another group of acute epistaxis patients but with no history of hypertension) and concluded that in the absence of local nasal disease, epistaxis was associated with high blood pressure. 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. Later, Charles *et al.* [12] and Isezuo *et al.* [7] also showed similar association between nosebleeds and hypertension. Herkner *et al.* [9] also suggested that hypertension was a predisposing factor for acute epistaxis. On the contrary, Fuchs *et al.* [8], found no association between epistaxis and hypertension. However, Lubianca Neto JF *et al.* [16] conducted a study on 323 adults with hypertension and suggested that epistaxis might be a consequence of long-lasting hypertension. Knopfholz *et al.* [10] worked on the severity of hypertension and concluded that the incidence of epistaxis in hypertensive patients was not associated with the severity of hypertension and the blood pressure readings were almost similar to the routine settings. Our findings were almost similar to other studies, but our patients had a mean age of 52 years. Viducich *et al.* [5] had mean age of 64.3 years Pollice *et al.* [17] had mean age of 60 years. In our study 55% of the patients had a history of hypertension which was almost similar to Viducich *et al.* [5] and Pollice *et al.* [17] who observed that 48% and 47% had history of hypertension respectively. However, we observed that there was no statistically significant difference was observed between patients with a history of epistaxis in the two groups (with or without a history of hypertension). There was no statistical significance between blood pressure recordings on admission between the two groups.

5. Conclusion

The severity of hypertension was also not associated with severe epistaxis versus serious epistaxis. Considering the blood pressure recordings, they were also not significantly different between the hypertensive and non-hypertensive groups. The possible effect of blood pressure on acute episodes of epistaxis still needs research. Our clinical data are insufficient to support or disprove the idea that epistaxis is secondary to target organ damage caused by hypertension.

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7. References

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