

Relationship between epistaxis and hypertension: A cause and effect or coincidence?

Nabil Abdulghany Sarhan^a, Abdulsalam Mahmoud Algamal^{b,*}

^a Department of Otorhinolaryngology, Al-Azhar University

^b Department of Cardiology, Mansoura University

^{a,b} Egypt

Introduction: Epistaxis is the most common otorhinolaryngological emergency. Whether there is an association or cause and effect relationship between epistaxis and hypertension is a subject of longstanding controversy.

Objective: The aim of our study is to evaluate the relationship between epistaxis and hypertension.

Materials and methods: This study was conducted at Olaya Medical Center (Riyadh) during the period between May 2013 and June 2014. A total of 80 patients were divided into two groups: Group A consisted of 40 patients who presented with epistaxis, and Group B consisted of 40 patients who served as a control group. Twenty-four-hour ambulatory blood pressure monitoring (ABPM) was performed for all patients. Patients were followed up for a period of three months.

Results: Readings of blood pressure (BP) were similar between the two groups regarding BP at presentation, ABPM, and BP at three months. There was a higher number of attacks in patients with history of hypertension. There was highly significant positive correlation between number of attacks of epistaxis and BP readings. Systolic BP at presentation was higher in patients who needed more complex interventions such as pack, balloon or cautery than those managed by first aid.

Conclusion: We found no definite association between epistaxis and hypertension. Epistaxis was not initiated by high BP but was more difficult to control in hypertensive patients.

© 2014 King Saud University. Production and hosting by Elsevier B.V. All rights reserved.

Keywords: Hypertension, Epistaxis, Ambulatory monitoring

Introduction

The term 'epistaxis' is Latin, derived from the Greek, epistazein (epi – above, over; stazein – to drip) [1]. Epistaxis is a common symptom of diverse conditions which may present as mild recurrent bleeds or severe life threatening rhinological emergency and may pose a challenge to

even a skilled otolaryngologist [2]. Globally, the true incidence remains unknown, but it is estimated that 60% of the population will have at least one episode of epistaxis in their lifetime, and 6% of them will seek medical attention. A slight male preponderance with 55% male and 45% female has been reported. Epistaxis is rare in neonates but common among children and

Disclosure: Authors have nothing to disclose with regard to commercial support.

Received 1 July 2014; revised 16 August 2014; accepted 5 September 2014. Available online 16 September 2014

* Corresponding author.

E-mail address: abdo75gamal@gmail.com (A.M. Algamal).



P.O. Box 2925 Riyadh – 11461KSA Tel: +966 1 2520088 ext 40151 Fax: +966 1 2520718 Email: sha@sha.org.sa URL: www.sha.org.sa



1016–7315 © 2014 King Saud University. Production and hosting by Elsevier B.V. All rights reserved.

Peer review under responsibility of King Saud University. URL: www.ksu.edu.sa http://dx.doi.org/10.1016/j.jsha.2014.09.002



Production and hosting by Elsevier

J Saudi Heart Assoc 2015;27:79–84

young adults, and peaks in the sixth decade giving a bi-modal age presentation [3].

Hypertension is increasing in prevalence in Saudi Arabia, affecting more than one fourth of the adult Saudi population [4]. It is still doubtful whether a connection exists between epistaxis and hypertension [6]. The prevalence rates of hypertension among patients with epistaxis range from 17 to 67% [6]. Whether there is an association or cause and effect relationship between epistaxis and hypertension is a subject of longstanding controversy [7].

Twenty-four-hour ambulatory BP monitoring (ABPM) is more valuable for predicting prognosis than other measures, as it more accurately assesses the risk of cardiovascular disease than measurements of BP made during clinic or office visits, and also ABPM is closely related to damage of target organs [8]. Twenty-four-hour ABPM enables the continuous observance of changes in BP during activities of daily life, measuring automatically at specific time intervals, and therefore allowing for more accurate BP measurements [9]. Serious spontaneous epistaxis may also be the presenting sign of underlying true hypertension in 43% of patients with no history of hypertension. However, hypertension per se does not appear to be a significant causal factor and/or factor of severity in serious spontaneous epistaxis [10].

Blood vessels in the nose run superficially through the easily-damaged mucosa and are therefore relatively unprotected [11]. The arterial hypertension would determine structural alterations of the nasal vessels similar to those verified in the cerebral circulation and retinal examination [12]. The etiologic role of hypertension in epistaxis is not certain. It is possible that hypertension causes arteriolosclerotic nasal vascular changes that predispose hypertensives to increased susceptibility to epistaxis [7]. Fundus examination of hypertensive epistaxics has demonstrated high prevalence of hypertensive retinal arteriolosclerosis in patients with epistaxis, which is an index of arteriolosclerotic changes in other parts of the body [13]. Similarly, an association between duration of hypertension and left ventricular hypertrophy and nasal artery enlargement determined by rhinoscopy has been described among hypertensives with history of epistaxis, indicating that long lasting hypertension might contribute to epistaxis [14].

The aim of our study is to evaluate the relationship between epistaxis and hypertension, its recurrence and control.

Abbreviations

ABPM	ambulatory blood pressure monitoring
BMI	body mass index
BP	blood pressure
d	diastolic
dBP	diastolic blood pressure
DM	diabetes mellitus
ENT	ear, nose and throat
HTN	hypertension
s	systolic
sBP	systolic blood pressure

Patients and methods

This is a prospective observational study conducted in Olaya Medical Center (Riyadh) during the period from May 2013 to June 2014. The study protocol was approved by the center's ethics committee.

Patients older than 18 years presented to ear, nose and throat (ENT) clinic were enrolled in the study after a written consent to participate in the study. A total of 80 patients were divided into two groups. Group A consisted of 40 patients who presented with idiopathic epistaxis. Group B consisted of 40 patients who served as a control group. These had presented with other reasons such as ear pain, headache, and dizziness. Patients with history of trauma to nose, local pathology, systemic diseases, bleeding disorders, patients on aspirin, clopidogrel or anticoagulants, and children were excluded from the study. None of the patients was lost to follow-up.

Rhinoscopy

Anterior rhinoscopy was done using a nasal speculum, light source, and a head mirror with simple inspection. For posterior rhinoscopy, a tongue depressor was placed on the center of the base of the tongue with one hand, and the base of the tongue was pressed downward. A small warmed mirror was then introduced into the space between the soft palate and posterior pharyngeal wall to inspect the choana, the posterior ends of the turbinates, the posterior margin of the septum, and the nasopharynx, together with its roof and the ostia of the Eustachian tubes. Nasal sinoscopy was done using 1.7 mm rigid endoscope (30°), light source, camera, and monitor to evaluate all cases, and to detect site, severity and management method of epistaxis. Most patients underwent anterior rhinoscopy and sinoscopy, whereas posterior rhinoscopy was used only in a limited number of patients.

Management of epistaxis in our patients included four methods: first aid (including anterior flexion of the head, control of blood pressure and fluid replacement if needed and nose pinching after packing with xylometazoline, provided the blood pressure is not high), nasal packing with Merocel, electrocautery, and nasal balloon.

BP measurement

The patient was rested, and then BP was measured by the authors using a mercuric manometer in supine position. The first measurement was taken at presentation before rhinoscopy; the two other readings were taken 20 min and one hour after epistaxis control; the first value was rejected and the final result was calculated as the mean of the second and the third value.

During the following week, ABPM was initiated on a 24-h basis by using an Oscar 2, SunTech Medical, Inc. USA apparatus. The diagnosis of hypertension was made on the basis of BP \ge 140 mmHg systolic and/or \ge 90 mmHg diastolic or use of antihypertensive medications. Hypertension by 24-h ambulatory BP was defined when the mean daytime systolic BP was equal to or greater than 135 mmHg or when the mean daytime diastolic BP was equal to or greater than 85 mmHg, according to the report of seventh report of the 2003 US Hypertension Joint National Committee, European Society of Hypertension and European Society of Cardiology guidelines for hypertension [15].

Patients were followed up for a period of three months for recurrent attacks of epistaxis and BP measurement in the same method as mentioned before. BP values after three months were used for statistical analysis as an indicator of BP control.

Statistical analysis

Data entry and analysis was performed using SPSS version 15 software. Continuous and cate-

gorical variables are presented as mean plus or minus standard deviation and percentages, respectively. Mean values between the two groups were compared using *t*-test. Comparison between groups was done by Chi-square test. Pearson's correlation coefficient was used to test correlation between variables. *F*-test (One-Way Anova) was used to compare between more than two groups. A *p* value ≤ 0.05 was considered statistically significant.

Results

We enrolled 80 patients in this study with mean \pm SD age of 47.86 \pm 16.01. There were 55 males (68.8%) and 25 females (31.2%), and the study included 29 diabetic patients (36.3%), 32 smokers (40%) and 23 hypertensive patients (28.8%). Patients were divided into two groups: epistaxis group with 40 patients, and control group with 40 patients.

Table 1 shows non-significant difference between the two groups regarding all parameters assessed including age, sex, diabetes, smoking, BMI, history of hypertension and its duration in years. Table 2 showed that readings of BP were similar between the two groups regarding BP at presentation, ABPM and BP at three months. BP at presentation was not significantly higher in patients with epistaxis than control group. Results of ABPM readings classified patients into stressinduced hypertension (initial high and normal ABPM), masked hypertension (initial normal and high ABPM), pre-existing hypertension, newly diagnosed hypertension and normal BP. There were no significant differences between patient and control groups regarding the final diagnosis of hypertension.

Management of epistaxis in our patients included four methods, starting with first aid

Tahla 1	Clinical	data	of	nationte	and	control	aroune
Tuble 1.	Cunicai	иши	ΟJ	putients	ипи	common	groups.

		Epistaxis group (40)	Control group (40)	P value
Age		50.23 ± 16.62	45.5 ± 15.23	0.189
Sex	Male patients	27 (67.5%)	28 (70%)	0.809
	Female patients	13 (32.5%)	12 (30%)	
DM	-	15 (37.5%)	14 (35%)	0.816
Smoking		17 (42.5%)	15 (37.5%)	0.648
History of HTN		10 (25%)	13 (32.5%)	0.459
Duration of HTN in years		13.4 ± 7.63	10.38 ± 6.19	0.307
BMI		29.56 ± 4.59	28.51 ± 4.98	0.331

DM = diabetes mellitus, HTN = hypertension, BMI = body mass index.

1 1 1 1 1 1 1 1 1 1	Table 2	2.	Blood	pressure	readings	and	final	diagnosis	of	patients	and	control	groi	ıp.
---------------------------------------	---------	----	-------	----------	----------	-----	-------	-----------	----	----------	-----	---------	------	-----

		Epistaxis group (40)	Control group (40)	P value
sBP at presentation		138.13 ± 22.47	135.63 ± 19.91	0.6
dBP at presentation		85.38 ± 9.57	83 ± 10.11	0.284
ABPM s day		146.57 ± 18.8	143.6 ± 17.59	0.467
ABPM d day		88.63 ± 9.31	86.58 ± 8.4	0.304
ABPM s night		137.53 ± 21.22	133.23 ± 20.21	0.356
ABPM d night		81.05 ± 12.6	79.15 ± 10.45	0.465
ABPM s 24 Hours		145.78 ± 19.33	142.35 ± 17.53	0.409
ABPM d 24 Hours		89.38 ± 12.07	86.3 ± 11.72	0.251
sBP at 3 months		128.75 ± 12.49	125 ± 10.06	0.143
dBP at 3 months		82 ± 7.32	80.63 ± 6.62	0.381
Final diagnosis	Normal	20 (50%)	21 (52.5%)	0.782
-	Stress HTN	2 (5%)	2 (5%)	
	Pre-existing HTN	10 (25%)	13 (32.5%)	
	Masked HTN	6 (15%)	3 (7.5%)	
	Newly diagnosed HTN	2 (5%)	1 (2.5%)	

sBP = systolic blood pressure, dBP = diastolic blood pressure, ABPM = ambulatory blood pressure monitoring, s = systolic, d = diastolic, HTN = hypertension.

Table 3. Clinical data of patients group in relation to the way of management.

		First aid (15)	Pack (12)	Balloon (6)	Electrocautery (7)	P value
Age		52.27 ± 18.27	48.75 ± 17.97	45.67 ± 16.61	52.29 ± 12.38	0.842
Sex	Male	10 (66.7%)	8 (66.7%)	4 (66.7%)	5 (71.4%)	0.966
	Female	5 (33.3%)	4 (33.3%)	2 (33.3%)	2 (28.6%)	
Smoking		7 (46.7%)	6 (50%)	3 (50%)	1 (14.3%)	0.423
HTN history		1 (6.7%)	4 (33.3%)	2 (33.3%)	3 (42.9%)	0.208
BMI		30.43 ± 4.57	31.5 ± 4.82	26.6 ± 2.53	26.89 ± 3.7	0.049
DM		7 (46.7%)	4 (33.3%)	2 (33.3%)	2 (28.6%)	0.823
Number of attacks	0	10 (66.7%)	2 (16.7%)	1 (16.7%)	2 (28.6%)	0.041
	1	3 (20%)	2 (16.7%)	0	1 (14.3%)	
	2	2 (13.3%)	5 (41.7%)	5 (83.3%)	3 (42.9%)	
	3	0	3 (25%)	0	1 (14.3%)	

DM = diabetes mellitus, HTN = hypertension, BMI = body mass index.

Table 4. Blood pressure readings of patients group in relation to the way of management.

	First aid (15)	Pack (12)	Balloon (6)	Electrocautery (7)	P value
sBP at presentation	126 ± 14.29	143.33 ± 21.57	156.67 ± 29.27	139.29 ± 21.68	0.021
dBP at presentation	80.67 ± 7.04	87.92 ± 9.64	90.83 ± 11.58	86.43 ± 9.88	0.083
ABPM s day	133.13 ± 6.59	160.42 ± 21.67	149.83 ± 16.83	148.86 ± 15.73	0.001
ABPM d day	83 ± 5.3	92.08 ± 8.89	92 ± 11.63	91.86 ± 10.65	0.026
ABPM s night	122.2 ± 9.25	153.75 ± 23.72	141.5 ± 18.01	135.38 ± 20.7	< 0.0001
ABPM d night	72.67 ± 7.72	87.58 ± 11.55	85.33 ± 14.21	84.14 ± 13.79	0.007
ABPM s 24 h	131.4 ± 7.37	159.92 ± 21.63	150 ± 16.31	148.74 ± 16.71	< 0.0001
ABPM d 24 h	85.67 ± 13.54	90.58 ± 11.19	92 ± 11.56	93 ± 10.91	0.5
sBP 3 months	120.67 ± 11	134.17 ± 13.11	135 ± 5.48	131.43 ± 11.07	0.01
dBP 3 months	76.33 ± 6.67	85.83 ± 5.97	87.5 ± 2.74	82.86 ± 5.67	< 0.0001

sBP = systolic blood pressure, dBP = diastolic blood pressure, ABPM = ambulatory blood pressure monitoring, s = systolic, d = diastolic, HTN = hypertension.

[15], then nasal packing with Merocel [12], electrocautery [7], and nasal balloon [6].

Table 3 shows non-significant differences between the various ways of epistaxis management regarding age, sex, diabetes, smoking, and hypertension history. There was a significantly higher number of attacks in patients managed by more complex interventions such as pack, electrocautery and balloon than those managed by first aid. Table 4 shows significantly higher BP readings in patients managed by more complex interventions such as pack, electrocautery and balloon than those managed by first aid, except for diastolic BP at presentation.

Discussion

Association between epistaxis and hypertension is controversial [16]. Our study was designed to provide an answer as to whether epistaxis may be a symptom related to the underlying presence of arterial hypertension, and to assess the effect of blood pressure control on epistaxis management.

This study included 80 patients who were divided into two groups; an epistaxis group and a control group. Both groups were well matched for gender, age, smoking habits, BMI and DM. The BP at presentation in both groups was in the high normal range, and initial hypertension was found in 14 patients with epistaxis (35%) and in 16 control patients (40%). Increased blood pressure at presentation may be due to patients' apprehension at the sight of blood [17]. Kikidis et al. [18] concluded that the presence of high arterial blood pressure during the actual episode of nasal bleeding cannot establish a causative relationship with epistaxis due to confounding stress and possible white coat phenomenon, but may lead to initial diagnosis of an already installed arterial hypertension.

In patients with epistaxis, the final diagnosis of hypertension was made in 18 patients (45%), with eight of them unaware of this diagnosis. Two patients who presented with high BP eventually had normal BP, whereas in the control group, 17 patients (42.5%) were found to have hypertension, with four of them unaware of the disease. Another two patients with initial high BP were found not to have hypertension. There was no significant difference between the two groups. These findings indicate no connection between epistaxis and hypertension.

The prevalence of hypertension in patients with epistaxis reportedly ranges from 24% to 64% [19]. Theodosis et al. [5] found that the final diagnosis of hypertension was set in 42.9% of patients admitted with epistaxis and in 28.9% of controls, which was not a statistically significant difference. Also, Nash and Field [11] found that history of hypertension was noted in 43.7% of patients, of whom 40.5% were receiving antihypertensive medications. Similarly, Page et al. [10] found that 55% of patients with epistaxis had a history of hypertension versus 48% for Viducich et al. [20] and 47% for Pollice and Yoder [21].

Our study showed that, in patients with epistaxis, the final there was no significant difference between male and female patients regarding BP readings. Further, the number of attacks over three months showed no significant correlation with age, sex, BMI, or smoking. The number of attacks was significantly higher in hypertensive patients; and in addition, there was a highly significant positive correlation between the number of attacks and BP readings including BP at presentation, ABPM and BP at three months. This indicates that uncontrolled hypertension is associated with more attacks of epistaxis and also that epistaxis may be difficult to control in patients with uncontrolled hypertension.

Systolic BP at presentation was significantly higher in patients who needed more complex interventions such as pack, balloon or cautery than patients managed by first aid. This indicates that hypertension renders the management of epistaxis more difficult. Diastolic pressure was not significantly different. Similar results were found for ABPM readings, except for diastolic BP over the 24 h.

Our results were in agreement with Theodosis et al. [5] who found that patients admitted with epistaxis had elevated systolic pressures compared to controls, but no difference regarding the final diagnosis of hypertension, which indicates no connection between epistaxis and hypertension. Our results are also in agreement with Fuchs et al. [22] who found that hypertension is not associated with history of epistaxis in the adulthood. Similar results were drawn by Karras et al. [23] in a population of 1908 individuals. Lubianca Neto et al. [14] found no definite association between blood pressure and history of adult epistaxis in hypertensive patients. Yüksel et al. [24] found that the evidence available was insufficient to prove a significant association between hypertension and epistaxis. Lima and Knopfholz [25] reported that epistaxis was unlikely to be a hypertensive emergency. Gifford and Orlandi [26] found that the control of epistaxis may be more difficult in patients with hypertension.

Our results were in contrast with the results of Herkner et al. [27] who found that patients with epistaxis have a higher blood pressure compared to that of control patients. Isezuo et al. [7] also found an association between epistaxis and hypertension.

In conclusion, we found no definite association between epistaxis and hypertension. The initial high BP may be explained by confounding stress and white coat effect; however, we found no difference between the patients and control groups, and no difference regarding BP readings and the final diagnosis of hypertension. All these findings clearly show a non-association between epistaxis and hypertension. We further concluded that the recurrence of epistaxis was higher in hypertensive patients, and higher BP made the management of epistaxis more complex, indicating that epistaxis was more difficult to control in hypertensive patients.

To the best of our knowledge, data assessing the correlation between blood pressure readings and management of epistaxis is scarce, and our study may be the first to address this correlation.

Our study limitations include a small number of patients and the short duration of follow-up. A larger study with more prolonged follow-up is needed to address the link between hypertension and epistaxis and whether a cause and effect relation exists.

Conclusion

We demonstrated that there is no association between hypertension and epistaxis, and that epistaxis was not initiated by high BP. However, epistaxis was more difficult to control in hypertensive patients. Due to the limited number of patients and short duration of follow-up, larger studies are needed to fully address this problem.

References

- [1] Swift AC. Epistaxis. Otorhinolaryngologist 2012;5(3): 129–32.
- [2] Nnennia CM. Epistaxis in Enugu: a 9 year review. Niger J Otorhinolaryngol 2004;1(1):11–4.
- [3] Varshney S, Saxena RK. Epistaxis: a retrospective clinical study. Indian J Otolaryngol Head Neck Surg 2005;57(2): 125–9.
- [4] Al-Nozha MM, Abdullah M, Arafah MR, Khalil MZ, Khan NB, Al-Mazrou YY, et al.. Hypertension in Saudi Arabia. Saudi Med J 2007;28(1):77–84.
- [5] Theodosis P, Mouktaroudi M, Papadogiannis D, Ladas S, Papaspyrou S. Epistaxis of patients admitted in the emergency department is not indicative of underlying arterial hypertension. Rhinology 2009;47(3):260–3.
- [6] Olatoke F, Ologe FE, Alabi BS, Dunmade AD, Busari SS, Afolabi OA. Epistaxis. A five-year review. Saudi Med J 2006;27(7):1077–9.
- [7] Isezuo SA, Segun-Busari S, Ezunu E, Yakubu A, Iseh K, Legbo J, et al.. Relationship between epistaxis and hypertension: a study of patients seen in the emergency units of two tertiary health institutions in Nigeria. Niger J Clin Pract 2008;11(4):379–82.
- [8] Mancia G, De Backer G, Dominiczak A, Cifkova R, Fagard R, Germano G, et al.. Guidelines for the management of arterial hypertension: the task force for the management of arterial hypertension of the European society of hypertension (ESH) and of the European society of cardiology (ESC). J Hypertens 2007;25(6):1105–87.
- [9] Kim SK, Bae JH, Nah DY, Lee DW, Hwang TY, Lee KS. Frequency and related factors of masked hypertension at a

worksite in Korea. J Prev Med Public Health 2011;44(3): 131–9.

- [10] Page C, Biet A, Liabeuf S, Strunski V, Fournier A. Serious spontaneous epistaxis and hypertension in hospitalized patients. Eur Arch Otorhinolaryngol 2011;268(12):1749–53.
- [11] Nash CM, Field SMB. Epidemiology of epistaxis in a canadian emergency department. Israeli J Emerg Med 2008;8(3):24–8.
- [12] Dal Secchi MM, Indolfo MLP, Rabesquine MM, de Castro FB. Epistaxis: prevailing factors and treatment. Int Arch Otorhinolaryngol 2009;13(4).
- [13] Ibrashi F, Sabri N, Eldawi M, Belal A. Effect of atherosclerosis and hypertension on arterial epistaxis. J Laryngol Otol 1978;92:877–81.
- [14] Lubianca Neto JF, Fuchs FD, Facco SR, Gus M, Fasolo L, Mafessoni R, et al.. Is epistaxis evidence of end-organ damage in patients with hypertension? Laryngoscope 1999;109(7 Pt. 1):1111–5.
- [15] Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo Jr JL, et al.. The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report. JAMA 2003;289(19):2560–72.
- [16] Knopfholz J, Lima-Junior E, Précoma-Neto D, Faria-Neto JR. Association between epistaxis and hypertension: a one year follow-up after an index episode of nose bleeding in hypertensive patients. Int J Cardiol 2009;134(3): e107–9.
- [17] Bhatta R. Clinical profile of idiopathic epistaxis in a hospital. JNMA J Nepal Med Assoc 2012;52(188):167–71.
- [18] Kikidis D, Tsioufis K, Papanikolaou V, Zerva K, Hantzakos A. Is epistaxis associated with arterial hypertension? A systematic review of the literature. Eur Arch Otorhinolaryngol 2014;271(2):237–43.
- [19] Herkner H, Havel C, Müllner M, Gamper G, Bur A, Temmel AF, et al.. Active epistaxis at ED presentation is associated with arterial hypertension. Am J Emerg Med 2002;20(2):92–5.
- [20] Viducich RA, Blanda MP, Gerson LW. Posterior epistaxis: clinical features and acute complications. Ann Emerg Med 1995;25(5):592–6.
- [21] Pollice PA, Yoder MG. Epistaxis: a retrospective review of hospitalized patients. Otolaryngol Head Neck Surg 1997;117(1):49–53.
- [22] Fuchs FD, Moreira LB, Pires CP, Torres FS, Furtado MV, Moraes RS, et al.. Absence of association between hypertension and epistaxis: a population-based study. Blood Press 2003;12(3):145–8.
- [23] Karras DJ, Ufberg JW, Harrigan RA, Wald DA, Botros MS, McNamara RM. Lack of relationship between hypertension-associated symptoms and blood pressure in hypertensive ED patients. Am J Emerg Med 2005;23(2):106–10.
- [24] Yüksel A, Kurtaran H, Kankiliç ES, Ark N, Uğur KS, Gündüz M. Epistaxis in geriatric patients. Turk J Med Sci 2014;44:133–6.
- [25] Lima E, Knopfholz J. Co30 Relationship between epistaxis and arterial pressoric blood levels: is epistaxis a hypertensive emergency? Am J Hypertens 2000;13(S2): 220A.
- [26] Gifford TO, Orlandi RR. Epistaxis. Otolaryngol Clin North Am 2008;41(3):525–36.
- [27] Herkner H, Laggner AN, Müllner M, Formanek M, Bur A, Gamper G, et al.. Hypertension in patients presenting with epistaxis. Ann Emerg Med 2000;35(2):126–30.