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Original Article

Evaluation of the Relationship Between Blood Pressure Control and Epistaxis Recurrence After Achieving Effective Hemostasis in the Emergency Department

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Background: Epistaxis is the most common cause of otorhinolaryngologic emergencies. There is a longstanding controversy regarding the relationship between epistaxis and hypertension (HTN), in terms of blood pressure (BP) control in the emergency department (ED) setting. The objective of this study is to evaluate the association between HTN, BP control, and recurrent epistaxis among patients initially admitted to the ED for epistaxis.
Methods: This retrospective cohort study was conducted in the EDs of three different hospitals in Taiwan and included a total of 739 patients admitted for epistaxis.
Results: Among ED patients with epistaxis, older age was significantly associated with a history of HTN, and a statistically significant difference in age was noted between groups classified according to the systolic BP/diastolic BP (SBP/DBP) at triage. Patients with a history of HTN had higher BP values at triage than did patients without a history of HTN (SBP: 175.68 ± 32.30 mmHg vs. 148.00 ± 26.26 mmHg, DBP: 95.04 ± 20.98 mmHg vs. 83.30 ± 16.65 mmHg; $p < 0.0001$). Antihypertensive medications were more commonly administered to patients with a history of HTN ($p < 0.0001$) and in those patients with SBP/DBP: ≥ 140/≥ 90 mmHg at triage ($p < 0.0001$). Among patients receiving antihypertensive medications, reductions in SBP by the time of discharge were significantly greater in patients with a history of HTN and in patients with SBP/DBP: ≥ 160/≥ 100 mmHg at triage. ED revisits due to recurrent epistaxis within 72 hours were significantly associated with male sex, a positive history of HTN, level of GOT, observation for recurrent epistaxis at ED, and duration of recurrent bleeding.
Conclusion: A positive history of HTN is related to recurrent epistaxis among ED patients. The effectiveness of administering antihypertensive agents before achieving hemostasis in patients admitted to the ED for epistaxis warrants further study.

Key words: *hypertension, emergency medicine, epistaxis*

Introduction

Epistaxis, as a common cause of emergency department (ED) visits, often presents as a mild and self-limited nasal bleeding, or more rarely as a severe, life-threatening, hemorrhagic emergency. Globally, the true incidence of epistaxis remains unknown; it is estimated that 60% of the population will have at least one episode of epistaxis in their lifetime,[1] and that 6% will seek medical attention. A small minority

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(less than 0.2%) will require hospitalization.[2-5] Among patients in the ED, hypertension (HTN) is a common presenting feature, with a reported prevalence ranging from 17 to 64%.[6] The connection between epistaxis and HTN has been widely discussed. Patients with sustained arterial HTN had significantly more episodes of epistaxis when compared with patients with only elevated blood pressure (BP) during epistaxis.[7] A 2017 systematic review and meta-analysis showed that HTN was significantly associated with an increased risk for epistaxis among ED and other hospitalized patients, although a causal relationship could not be demonstrated.[8] In hospitalized patients, serious and spontaneous epistaxis may be a presenting sign of underlying true HTN in 43% of patients with no previous history of HTN.[9] Though the severity of HTN and history of epistaxis were not associated in a cohort of hypertensive patients,[10] active epistaxis at ED presentation was associated with arterial HTN[6] and in adult patients, with idiopathic epistaxis from Kiesselbach's area. Although management of BP for the prevention of persistent epistaxis in the ED has been proposed,[11] there is a lack of studies evaluating the effectiveness of controlling BP for managing epistaxis. Thus, the aim of our study was to evaluate the association between HTN, BP control, and recurrent epistaxis after effective hemostasis, among patients presenting with epistaxis in the ED setting.

Methods

This retrospective case-control study was approved through an expedited review by the institutional review board (case number: 104-5371B).

Data Acquisition

Patients with epistaxis who visited the EDs of three hospitals (including one metropolitan hospital in Keelung and two medical centers in Taipei and Linkou, all members of a Taiwanese medical foundation) were retrospectively identified by searching electronic medical records for the International Classification of Diseases (ICD)-9 code for epistaxis (784.7). Records from a 12-month period, between January 1, 2014, and December 31, 2014, were surveyed.

The study inclusion criteria were: age above 18 years, a diagnosis of atraumatic epistaxis, and a history of an ED visit for reasons other than epistaxis but requiring treatment for epistaxis. The exclusion criteria included: age below 18 years, epistaxis caused by trauma, and a misdiagnosis of epistaxis. All medical charts of the enrolled patients were available and independently reviewed by two authors (Cheng-Jung Lee and Pin-Chieh Liao) to confirm the diagnosis of epistaxis and to exclude patients who were misclassified using the ICD code.

The following variables were collected using a predefined abstraction form (Microsoft Access 2010, Microsoft Corp., Redmond, WA, USA) and independently reviewed by two naive reviewers: BP at triage, patient demographics (age, gender), associated medical diseases (medical history), and current medications (e.g., antiplatelet drugs, anticoagulant drugs, or antihypertensive drugs). Laboratory test values, such as white blood cell counts, platelet counts, hemoglobin levels, prothrombin time, activated partial thromboplastin time, and blood creatinine or blood urea nitrogen levels were also recorded. Consultation with an otorhinolaryngologist (certified ear-nose-throat [ENT] doctor), hemostasis methods used, length of ED stay, and associated patient management steps (e.g., cool compression of the nose, pinching of the nasal alae, etc.) were also recorded. Effective hemostasis was defined as an inactive nasal bleeding and eligibility for discharge as confirmed by an ED doctor. Outcomes included the dispositions of patients (i.e., discharge upon effective hemostasis: may be discharged [MBD], discharge against medical advice [AMA], admission and escaped [self-discharge]), and subsequent ED revisits due to recurrent epistaxis within 72 hours of discharge.

We classified patients based on their history of HTN (category I) and BP at triage (category II). In category I, Group A and group B included patients with, and without a history of HTN, respectively. In category II, patients were classified into four groups based on BP (systolic BP [SBP]/diastolic BP [DBP]) at triage in accordance with previously established guidelines[12] for the management of arterial HTN: group 1 (< 120/< 80 mmHg), group 2 (120–139/80–89 mmHg), group 3 (140–159/90–99 mmHg), and group 4 (≥ 160/≥ 100 mmHg).

Statistical Analyses

All data were computed using Microsoft Excel 2010 (Microsoft Corp., Seattle, WA, USA) and the Statistics Analysis System (SAS v. 9.4, TS1M3; SAS Institute, Inc., Cary, NC, USA). The chi-square and

Fisher's exact tests for used for comparing dichotomous variables, and the two-sample t tests were used to analyze continuous variables. A multivariate logistic analysis was conducted to assess the potential association of background factors with ED revisits due to recurrent epistaxis, and a receiver operating characteristic (ROC) curve (and area under the curve [AUC]) was determined. The level of statistical significance was set at $p = 0.05$.

Results

A total of 739 patients with epistaxis were included. In category I, group A patients (314/739, 42%) had a positive history of HTN, with 127 being on antihypertensive medication(s). Group B patients (411/739, 56%) did not have HTN, but 2% (14/739) of the patients claimed to have an undocumented history of HTN. Patients in group A were significantly older than those in group B (average age: 60.93 years vs. 48.21 years, $p < 0.0001$; Table 1). In category II, a significant difference in age was noted between groups 1 to 4 ($p = 0.003$; Table 1). A total of 8% (62/739) of the patients were using aspirin, and this was more common in group A than in group B ($p < 0.0001$). The proportion of anticoagulant (warfarin or a novel oral anti-coagulant [NOAC]) users did not differ between groups in either category.

Diabetes mellitus (DM), end stage renal disease (ESRD), and cerebrovascular accident (CVA) were more prevalent among patients in group A than group B ($p < 0.0001$; Table 1). Significant differences related to the history of ENT surgery were also observed between the groups in category I ($p < 0.001$; Table 1).

SBP and DBP values at triage were significantly greater among patients in group A compared to group B ($p < 0.0001$; Table 2). Significant differences in SBP and DBP were also noted between groups 1–4 ($p < 0.0001$; Table 2). Similarly, both the SBP ($p < 0.0001$) and DBP ($p = 0.008$) were higher in group A than in group B before discharge (Table 2). A significant difference in SBP and DBP before discharge between groups 1–4 was also observed ($p < 0.0001$; Table 2).

With regards to the blood laboratory tests, no significant differences between groups were noted, except for the levels of glutamic oxaloacetic transaminase (GOT) between groups 1–4 ($p = 0.01$; Table 2). In terms of the interventions used to control epistaxis (Table 3), 70% (518/739) of the patients applied direct pressure by pinching the nose, and this significantly differed between the groups in category II ($p = 0.02$). Cool compression with ice packs (399/739, 55%) was provided fewer times in group B ($p = 0.0009$) and group 1 ($p = 0.02$).

An oral or intravenous (IV) antihypertensive agent was administered to 27% (197/739) of the participants to lower BP before hemostasis was achieved (Table 3). The use of antihypertensive agents was significantly different between groups in both categories ($p < 0.0001$). In category I, 46% of group A patients were prescribed BP-lowering medication. In category II, the use of antihypertensive agents increased significantly with elevated BP from groups 1–4. While the majority (97%) of patients ceased using antihypertensive agents after hemostasis, patients in group A and group 1 still continued to receive this medication more often (Table 3).

Epistaxis was documented to have spontaneously stopped in 72% (469/649) of patients. The incidence of epistaxis was similar on both sides of the nostril (41%, right vs. 40%, left), and the Kiesselbach's plexus (Little's area) was the most commonly involved anatomical site for all groups in both categories, except for group 4 in which posterior bleeding was more common ($p = 0.02$; Table 3). Surgicel gauze (oxidized regenerated cellulose, Ethicon, Medline Industries, Inc., Waukegan, IL, USA) was used for hemostasis in most cases (255/739, 35%), followed by the application of Bosmine (adrenaline, Daiichi Seiyaku Co., Ltd., Tokyo, Japan), a vasoconstrictive spray, in 18% (134/739) of cases. Combined use of Surgicel and Bosmine was more common in group B ($p < 0.0001$) and group 1 ($p < 0.0001$; Table 3). The time until hemostasis achieved by the ENT doctor was similar, and not significantly different between groups in either category (Table 3).

Tranexamic acid was prescribed in 18% (135/739) of participants before achieving hemostasis and was more commonly used in group 1 and 4 in category II ($p = 0.02$; Table 3). After hemostasis, tranexamic acid was administered commonly in both categories (Table 3). The average stay in the ED was 4.62 ± 13.31 hours, and the median duration of ED stay was 1.58 hours (min: 0.18 hours, max: 192.2 hours; Table 4). The time from triage until consultation with an ENT specialist was longer in group A than group B ($p = 0.04$; Table 4). The time of achieving hemostasis by ENT doctor did not differ between groups in either category. Overall,

Table 1. Demographic data and medical history of patients with epistaxis in the emergency department, according to a positive history of hypertension and the blood pressure at triage

	Total patients (n = 739)	Category I Comparison of patients with a positive history of HTN			Category II Comparison of patients according to the BP at triage				
		Group A Yes 314 (42%)	Group B No 411 (56%)	*p*-value	Group 1 < 120/< 80 mmHg 51 (7)	Group 2 120–139/ 80–89 mmHg 271 (37)	Group 3 140–159/ 90–99 mmHg 202 (27)	Group 4 ≥ 160 /≥ 100 mmHg 215 (29)	*p*-value
Demographic data									
Age (years) mean ± SD	53.61 ± 17.87	60.93 ± 14.82	48.21 ± 18.02	< 0.0001	49.69 ± 19.87	51.1 ± 18.55	56.15 ± 17.61	55.33 ± 16.22	0.003
Gender				0.89					0.39
Male	498 (67)	213 (68)	274 (67)		30 (59)	179 (66)	137 (68)	152 (71)	
Female	241 (33)	101 (32)	137 (33)		21 (41)	92 (34)	65 (32)	63 (29)	
Medical history									
Currently taking aspirin	62 (8)	42 (13)	20 (5)	< 0.0001	5 (10)	30 (11)	14 (7)	13 (6)	0.19
Currently taking of warfarin	19 (3)	10 (3)	9 (2)	0.51	1 (2)	3 (1)	8 (4)	7 (3)	0.23
Currently taking a NOAC	2 (< 1)	0	2 (< 1)	0.51	0 (0)	2 (1)	0 (0)	0 (0)	0.32
Diabetes mellitus	79 (11)	54 (17)	25 (6)	< 0.0001	6 (12)	33 (12)	20 (10)	20 (9)	0.74
Coronary artery disease	39 (5)	26 (8)	13 (3)	0.06	3 (6)	15 (6)	10 (5)	11 (5)	0.99
End stage renal disease	23 (3)	16 (5)	7 (2)	0.01	4 (8)	9 (3)	7 (3)	3 (1)	0.11
Malignant diseases	41 (6)	15 (5)	26 (6)	0.53	4 (8)	14 (5)	13 (6)	10 (5)	0.75
Liver cirrhosis	25 (3)	12 (4)	13 (3)	0.72	2 (4)	10 (4)	7 (3)	6 (3)	0.95
Hematologic diseases	17 (2)	5 (2)	12 (3)	0.39	0 (0)	7 (3)	5 (2)	5 (2)	0.72
Rheumatologic diseases	7 (1)	4 (1)	3 (1)	0.47	0 (0)	2 (1)	3 (1)	2 (1)	0.74
Pulmonary diseases	26 (4)	15 (5)	11 (3)	0.16	3 (6)	6 (2)	8 (4)	9 (4)	0.46
Cerebrovascular accident	28 (4)	18 (6)	10 (2)	0.03	5 (10)	12 (4)	7 (3)	4 (2)	0.05
ENT diseases				< 0.0001					0.18
Allergic rhinitis	8 (1)	8 (2)	0 (0)	0.02	2 (4)	4 (1)	2 (1)	0 (0)	0.07
Nasal septum deviation	26 (4)	8 (2)	18 (6)	0.01	1 (2)	11 (4)	5 (2)	9 (4)	0.68
History of surgery in ENT field	111 (15)	82 (19)	29 (9)	0.0002	9 (18)	49 (18)	27 (13)	26 (12)	0.25

Data were given as number (%) or mean ± SD.
BP: blood pressure; ENT: ear-nose-throat; HTN: hypertension; NOAC: novel oral anticoagulant; SD: standard deviation.

Table 2 Physical parameters and laboratory data of patients with epistaxis in the emergency department according to a positive history of hypertension and the blood pressure at triage

	Total patients (n = 739)	Category I Comparison of patients with a positive history of HTN		
		Group A Yes 314 (42)	Group B No 411 (56)	*p*-value
Vital signs in the ED				
BT at triage (°C)	36.41 ± 0.59	36.39 ± 0.64	36.41 ± 0.55	0.68
HR at triage (beats/min)	87.70 ± 17.27	91.11 ± 17.81	89.15 ± 16.83	0.13
RR at triage (breaths/min)	18.46 ± 1.64	18.40 ± 1.66	18.51 ± 1.63	0.37
SBP at triage (mmHg)	159.69 ± 32.04	175.68 ± 32.30	148.15 ± 25.39	< 0.0001
DBP at triage (mmHg)	88.39 ± 19.51	95.04 ± 20.98	83.47 ± 16.75	< 0.0001
SBP before discharge (mmHg)	138.40 ± 23.15	145.00 ± 23.21	132 ± 21.26	< 0.0001
DBP before discharge (mmHg)	83.17 ± 15.76	85.29 ± 16.50	81.19 ± 14.76	0.008
Laboratory values				
WBC count (/mm^3)	9008.94 ± 13183.56	9201.50 ± 12966.00	8861.40 ± 13553.80	0.423
Hb (g/dL)	13.20 ± 6.96	13.58 ± 9.87	12.87 ± 2.34	0.11
Platelet (/mm^3)	211600.46 ± 131668.18	209309.00 ± 115935.00	211507.00 ± 138109.00	0.26
BUN (mg/dL)	20.22 ± 13.36	24.85 ± 16.90	17.05 ± 9.54	0.05
Cr (mg/dL)	1.12 ± 1.29	1.42 ± 1.79	0.83 ± 0.30	0.06
GOT (U/L)	44.33 ± 41.60	50.00 ± 32.7	41.50 ± 46.50	0.06
GPT (U/L)	26.68 ± 18.07	27.59 ± 18.66	24.45 ± 14.51	0.25
PT (sec)	12.76 ± 4.04	12.92 ± 4.81	12.62 ± 3.20	0.93
INR	1.14 ± 0.34	1.16 ± 0.41	1.13 ± 0.27	0.18
aPTT (sec)	27.73 ± 3.64	27.46 ± 0.34	27.93 ± 0.42	0.36

Table 2 Physical parameters and laboratory data of patients with epistaxis in the emergency department according to a positive history of hypertension and the blood pressure at triage (continued)

	Category II Comparison of patients according to the BP at triage				
	Group 1 < 120/< 80 mmHg 51 (7)	Group 2 120–139/80–89 mmHg 271 (37)	Group 3 140–159/90–99 mmHg 202 (27)	Group 4 ≥ 160 /≥ 100 mmHg 215 (29)	*p*-value
Vital signs in the ED					
BT at triage (°C)	36.35 ± 0.54	36.39 ± 0.54	36.53 ± 0.55	36.31 ± 0.68	0.2
HR at triage (beats/min)	86.57 ± 15.79	87.29 ± 16.1	89.17 ± 16.51	94.95 ± 18.70	< 0.0001
RR at triage (breaths/min)	18.53 ± 2.00	18.36 ± 1.37	18.34 ± 1.39	18.70 ± 2.01	0.07
SBP at triage (mmHg)	111.69 ± 10.36	145.72 ± 22.16	158.75 ± 18.36	190.12 ± 27.47	< 0.0001
DBP at triage (mmHg)	64.41 ± 8.59	79.41 ± 9.57	86.30 ± 12.77	107.34 ± 20.56	< 0.0001
SBP before discharge (mmHg)	118.77 ± 22.86	131.38 ± 21.30	137.14 ± 19.58	148.57 ± 22.83	< 0.0001
DBP before discharge (mmHg)	69.86 ± 10.27	79.77 ± 13.23	80.94 ± 14.88	89.83 ± 16.54	< 0.0001
Laboratory values					
WBC count (/mm^3)	8084.74 ± 4092.84	11181.13 ± 24507.57	7454.27 ± 3.60.22	9201.14 ± 12156.84	0.56
Hb (g/dL)	11.67 ± 2.77	12.87 ± 2.29	12.26 ± 2.36	14.04 ± 9.38	0.36
Platelet (/mm^3)	204536.00 ± 157174.00	182452.00 ± 92210.00	182580.00 ± 122936.00	234590.00 ± 137713.00	0.2
BUN (mg/dL)	19.60 ± 10.83	19.00 ± 22.23	20.00 ± 10.15	20.83 ± 11.81	0.12
Cr (mg/dL)	1.23 ± 0.66	1.25 ± 1.93	0.88 ± 0.41	1.16 ± 1.38	0.07
GOT (U/L)	78.67 ± 91.24	33.40 ± 18.37	47.67 ± 42.77	36.00 ± 22.77	0.01
GPT (U/L)	21.20 ± 12.34	30.14 ± 28.87	23.20 ± 9.12	29.18 ± 19.83	0.36
PT (sec)	13.22 ± 3.22	13.09 ± 4.52	12.47 ± 2.55	12.73 ± 4.57	0.17
INR	1.20 ± 0.27	1.18 ± 0.40	1.09 ± 0.16	1.15 ± 0.38	0.12
aPTT (sec)	28.84 ± 3.68	28.55 ± 5.01	27.74 ± 3.38	27.30 ± 3.23	0.22

Data were given as number (%) or mean ± SD.

aPTT: activated partial thromboplastin time; BT: body temperature; BP: blood pressure; BUN: blood urea nitrogen; Cr: creatinine; DBP: diastolic blood pressure; ED: emergency department; GOT: glutamic oxaloacetic transaminase; GPT: glutamate pyruvate transaminase; Hb: hemoglobin; HR: heart rate; INR: international ormalized ratio; PT: prothrombin time; RR: respiratory rate; SBP: systolic blood pressure; WBC: white blood cell.

Table 3. Interventions for patients with epistaxis in the emergency department according to a positive history of hypertension and the blood pressure at triage

	Total patients (n = 739)	Category I Comparison of patients with a positive history of HTN			Category II Comparison of patients according to the BP at triage				
		Group A Yes 314 (42)	Group B No 411 (56)	*p*-value	Group 1 < 120/< 80 mmHg 51 (7)	Group 2 120–139/80–89 mmHg 271 (37)	Group 3 140–159/90–99 mmHg 202 (27)	Group 4 ≥ 160 /≥ 100 mmHg 215 (29)	*p*-value
Pinching the nasal alae	518 (70)	229 (73)	289 (68)	0.19	30 (5)	182 (67)	137 (68)	169 (79)	0.005
Cool compression on the nose	399 (55)	192 (62)	207 (49)	0.0009	17 (33)	148 (55)	115 (58)	119 (56)	0.02
Anti-HTN use before hemostasis				< 0.0001					< 0.0001
IV	22 (3)	14 (4)	5 (1)	0.007	0 (0)	1 (< 1)	2 (1)	19 (9)	< 0.0001
Oral	175 (24)	132 (42)	42 (10)	< 0.0001	0 (0)	33 (12)	37 (18)	105 (49)	< 0.0001
None	542 (73)	168 (54)	364 (89)	< 0.0001	51 (100)	237 (87)	163 (81)	91 (42)	< 0.0001
Use of tranexamic acid before hemostasis				0.13					0.002
Yes	135 (18)	64 (20)	67 (16)		11 (22)	37 (14)	28 (14)	55 (26)	
No	604 (82)	249 (80)	355 (84)		40 (78)	233 (86)	172 (86)	159 (74)	
Hemostasis by consulting an ENT Dr	649 (88)	280 (89)	369 (87)	0.39	44 (86)	236 (87)	176 (87)	193 (90)	0.78
Site of bleeding				0.37					0.02
Anterior (Kiesselbach's plexus)	513 (69)	224 (71)	289 (68)		19 (37)	93 (34)	66 (33)	48 (22)	
Posterior	226 (31)	90 (29)	136 (32)		32 (63)	178 (66)	136 (67)	167 (78)	
Status of bleeding (inactive) inspected by an ENT Dr	469 (72)	206 (66)	263 (62)	0.36	39 (76)	170 (63)	127 (63)	133 (62)	0.26
Method for hemostasis by an ENT Dr				< 0.0001					< 0.0001
Bosmine	134 (18)	61 (19)	73 (17)	0.49	9 (18)	45 (17)	42 (21)	38 (18)	0.70
Surgicel	255 (35)	109 (35)	146 (34)	0.98	16 (31)	102 (38)	68 (34)	69 (32)	0.57
Bosmine + surgicel	99 (14)	11 (4)	88 (21)	< 0.0001	18 (35)	40 (15)	29 (14)	12 (6)	< 0.0001
Merocel	64 (9)	34 (11)	30 (7)	0.1	1 (2)	22 (8)	14 (7)	27 (13)	0.05
Vaseline gauze packing	25 (3)	9 (3)	16 (4)	0.64	0 (0)	9 (3)	6 (3)	10 (5)	0.4
H_2O_2	15 (2)	7 (2)	8 (2)	0.95	1 (2)	2 (1)	3 (1)	9 (4)	0.06
Use of tranexamic acid after hemostasis	563 (76)	236 (75)	301 (73)	0.10	39 (76)	199 (73)	157 (78)	168 (78)	0.6
Anti-HTN drug use after hemostasis									0.35
Yes	19 (3)	15 (5)	3 (< 1)	0.003	1 (2)	2 (1)	0 (0)	16 (7)	< 0.0001
No	720 (97)	299 (95)	408 (99)		64 (98)	149 (98)	185 (100)	322 (96)	

Data were given as number (%).
anti-HTN: anti-hypertensive drug; BP: blood pressure; ENT Dr: ear-nose-throat doctor; HTN: hypertension; H_2O_2: hydrogen peroxide; IV: intravenous.

Table 4. Duration in the emergency department (ED), blood pressure (BP) changes, and measurement outcomes of patients with epistaxis in the ED according to a positive history of hypertension and the BP at triage

	Total patients (n = 739)	Category I Comparison of patients with a positive history of HTN			Category II Comparison of patients according to the BP at triage				
		Group A Yes 314 (42)	Group B No 411 (56)	*p*-value	Group 1 < 120/< 80 mmHg 51 (7)	Group 2 120–139/80–89 mmHg 271 (37)	Group 3 140–159/90–99 mmHg 202 (27)	Group 4 ≥ 160 /≥ 100 mmHg 215 (29)	*p*-value
Duration in the ED									
Total time in the ED (h)	4.62 ± 13.31	5.43 ± 16.82	4.17 ± 9.93	0.18	5.93 ± 12.43	4.02 ± 13.75	3.31 ± 7.64	6.28 ± 16.61	0.09
Time from triage until consultation with an ENT Dr (h)	0.47 ± 2.38	0.81 ± 0.38	0.33 ± 0.38	0.04	0.37 ± 0.52	0.5 ± 3.83	0.41 ± 0.92	0.51 ± 0.52	0.95
Time until the ENT Dr achieved hemostasis	1.88 ± 6.45	1.45 ± 2.97	1.82 ± 7.11	0.33	2.05 ± 6.78	1.45 ± 5.11	1.26 ± 4.38	2.21 ± 7.11	0.31
Observation for recurrent epistaxis in the ED				0.11					0.002
Yes	155 (21)	75 (24)	78 (19)		13 (25)	43 (16)	36 (18)	63 (29)	
No	584 (79)	239 (76)	333 (81)		38 (7)	228 (84)	166 (82)	152 (71)	
Duration of observation for recurrent epistaxis in the ED after hemostasis (h)	2.88 ± 12.94	3.57 ± 16.64	2.36 ± 9.31	0.25	4.34 ± 12.44	2.52 ± 13.52	1.71 ± 7.38	4.07 ± 16.02	0.23
BP changes at triage and before discharge									
Reduction in SBP									
HTN at triage s/p anti-HTN use	47.45 ± 26.31	51.54 ± 29.88	39.11 ± 24.02	0.03	—	—	12.14 ± 12.78	49.87 ± 23.53	< 0.0001
HTN at triage s/p no anti-HTN use	24.35 ± 17.93	22.05 ± 23.47	27.43 ± 21.64	0.11	—	—	16.23 ± 16.31	30.62 ± 19.12	< 0.0001
Reduction in DBP									
HTN at triage s/p anti-HTN use	24.12 ± 16.64	25,14 ± 18.6	24.83 ± 19.57	0.94	—	—	3.67 ± 12.79	20.15 ± 16.55	< 0.0001
HTN at triage s/p no anti-HTN use	12.34 ± 14.89	11.32 ± 15.6	13.08 ± 14.91	0.65.	—	—	14.9 ± 13.89	15.04 ± 14.98	0.19

Table 4. Duration in the emergency department (ED), blood pressure (BP) changes, and measurement outcomes of patients with epistaxis in the ED according to a positive history of hypertension and the BP at triage (continued)

	Total patients (n = 739)	Category I Comparison of patients with a positive history of HTN			Category II Comparison of patients according to the BP at triage				
		Group A Yes 314 (42)	Group B No 411 (56)	*p*-value	Group 1 < 120/< 80 mmHg 51 (7)	Group 2 120–139/80–89 mmHg 271 (37)	Group 3 140–159/90–99 mmHg 202 (27)	Group 4 ≥ 160 /≥ 100 mmHg 215 (29)	*p*-value
Measurement outcomes									
Disposition									
MBD	692 (93)	294 (94)	383 (93)	1	47 (92)	256 (94)	187 (9)	203 (94)	0.78
AMA	8 (1)	6 (2)	6 (1)	0.34	1 (2)	5 (2)	2 (1)	2 (1)	0.67
Admission	28 (4)	10 (3)	17 (4)	0.59	36 (71)	9 (3)	8 (4)	8 (4)	0.85
Escaped	11 (2)	5 (2)	5 (1)	0.73	0 (0)	1 (< 1)	5 (2)	2 (1)	0.2
Re-visit to the ED within 72 h due to recurrent epistaxis	64 (9)	39 (12)	25 (6)	0.04	7 (42)	18 (7)	20 (10)	20 (9)	0.32
Duration of recurrent bleeding (h)	50.36 ± 21.12	44.45 ± 24.51	56.72 ± 23.17	0.67	17.60 ± 9.54	49.72 ± 21.09	60.44 ± 27.31	49.40 ± 25.61	0.68

Data were given as number (%) or mean ± SD.

AMA: (discharge) against medical advice; anti-HTN: anti-hypertensive drug; BP: blood pressure; DBP: diastolic blood pressure; ED: emergency department; ENT Dr: ear-nose-throat doctor; HTN: hypertension; SBP: systolic blood pressure; s/p:status post; MBD: may be discharged.

21% (155/739) of the patients were observed in the ED due to recurrent bleeding concerns. The average observation time was 2.88 ± 12.94 hours, and was similar between groups in each category (Table 4). With regards to BP by the time of discharge, among patients receiving anti-hypertensive medications (197/739, 26.7%), reductions were significantly greater in group A (SBP, $p = 0.03$) and group 4 (SBP and DBP, $p < 0.0001$; Table 4).

Most patients (692/739, 93%) left the hospital under MBD orders, with 4% admitted to the medical floor, and the rest having left the hospital against recommendations (AMA and escaped). No significant differences in disposition were observed between groups in either category (Table 4). Just under a tenth (9%) of patients with epistaxis revisited the ED due to recurrent epistaxis within a mean duration of 50.36 ± 21.12 hours. ED revisit rates were more common in group A ($p = 0.04$) in category I, and similar among groups in category II (Table 4).

As determined by multivariate analysis, ED revisits due to recurrent epistaxis were significantly associated with male sex, a positive history of HTN, level of GOT, observation for recurrent epistaxis at ED, and duration of recurrent bleeding (Table 5). With regards to the ROC, the AUC was determined to be 0.857, which indicated a strong association for the factors in the model (Fig. 1).

Discussion

Our study found that ED patients with epistaxis and a history of HTN had higher BP values at triage than those without HTN. Antihypertensive medications were more commonly administered to patients with a positive history of HTN, and those with SBP/DBP: ≥ 140/≥ 90 mmHg at triage.

Confirming the results of prior studies, this study found higher average BP values at triage among patients with epistaxis.[5,10,11,13,14] The present study, however, documented a higher proportion of patients without HTN than with HTN, which is contrary to previous reports.[10,11,13,14]

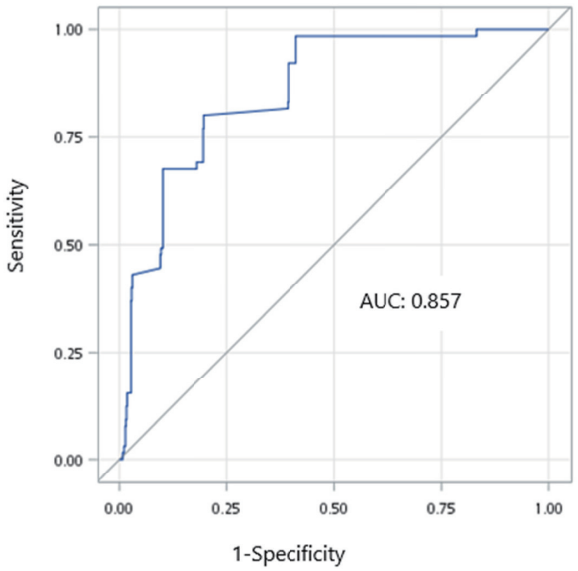

Fig. 1. Receiver operating characteristics curve constructed from logistic analysis of significant multivariate factors of visits of emergency department due to recurrent epistaxis in 72 hours.
AUC: area under the curve.

Table 5. Logistic regression analysis of factors associated with emergency department revisits within 72 hour due to recurrent epistaxis[a]

	Univariate	Multivariate	
	p-value	Odds ratio (95% CI)	*p*-value
Gender (male : female)	0.003	2.476 (1.164–5.266)	0.0186
Positive history of HTN (presence : absence)	0.004	2.291 (1.302–4.030)	0.004
GOT (U/L)	0.047	0.998 (0.997–0.999)	0.0031
Duration of recurrent bleeding (hours)	< 0.0001	1.023 (1.014–1.031)	< 0.0001
Observation for recurrent epistaxis in the ED (presence : absence)	< 0.0001	3.111 (1.760–5.500)	< 0.0001

[a]Univariate and multivariate logistic regression analyses were conducted for emergency department (ED) revisits within 72 hours due to recurrent epistaxis as the statistically significant outcome. Demographic data, medical history, vital signs in the ED, laboratory values, interventions, duration in the ED, blood pressure changes, and other measurement outcomes were analyzed using univariate logistic regression analysis. Statistically significant adjusted variables from univariate logistic regression analysis, including gender, positive history of hypertension, glutamic oxaloacetic transaminase, duration of recurrent bleeding, and observation for recurrent epistaxis in the ED, were analyzed using multivariate logistic regression analysis.
CI: confidence interval; ED: emergency department; GOT: glutamic oxaloacetic transaminase; HTN: hypertension.

Ideally, the diagnosis of HTN should be made based on the average of two or more correctly measured, BP readings obtained in the sitting position, on each of two or more office visits.[12] These criteria are difficult to meet in most patients seeking ED treatment for active epistaxis. This may have caused our study to record a fewer number of hypertensive patients than those reported by earlier studies. Furthermore, clinical guidelines for the management of HTN, such as Joint National Committee 7[12] (in effect from 2003 to 2014), JNC 8[15] (in effect since 2014), and the latest European Society of Cardiology/European Society of HTN Guidelines[16] (in effect since 2018), recommend establishing and maintaining the patient's target BP (based on patient's age, race, physical comorbidities, and specific circumstances) within one month of commencing treatment. In the ED, however, aggressive BP control is enacted in response to the presence of hypertensive crisis, rather than achieving an ideal target BP over the longer term.

Previous proposals to classify epistaxis as end-organ damage and vascular changes caused by HTN have been extensively discussed. The presence of enlarged septal vessels was strongly and independently associated with a history of epistaxis in a logistic regression model.[17] Nasal artery enlargement, as determined by rhinoscopy, has been reported among HTN patients with a history of epistaxis, indicating that long-term HTN might contribute to epistaxis.[18] The concept of epistaxis as a hypertensive emergency has not been supported by recent studies.[5,14,17,19] Given this lack of understanding regarding the relationship between BP control and epistaxis in the ED, our analysis demonstrates important and practical aspects for managing these cases.

In our study, antihypertensive medications were often administered before hemostasis, when SBP/DBP at triage was > 160/100 mmHg. According to our findings, administration of antihypertensive medications, and the reduction of BP, differed between groups with elevated BP at triage. Nevertheless, no significant differences were observed with regards to the time required for ENT doctors to achieve hemostasis; patient disposition; or ED visits due to recurrent epistaxis. This suggests that the effectiveness of aggressive BP control for epistaxis (in accordance with BP determination at ED triage), enacted before hemostasis is achieved, warrants further investigation, preferably by prospectively controlled studies.

A positive history of HTN was associated with ED revisits due to recurrent epistaxis. Higher rates of aspirin use, as well as an increased prevalence of DM, ESRD, and CVA were observed among patients with a history of HTN. This suggests the importance of additional analyses for patients with specific medical comorbidities in future studies. Although rhinologic comorbidities, such as rhinitis and nasal septum deviation, were not found to increase the risk of recurrent epistaxis,[20,21] studies with larger sample sizes are required to further elucidate the potential influence of rhinologic comorbidities on the effectiveness of BP control for epistaxis.

Up to 90% of epistaxis cases occur at the Kiesselbach's plexus as anterior epistaxis,[22] and this type accounted for about 70% of the cases in our study. Posterior nosebleeds are more common in older patients, with a mean age of 64 years reported in a previous prospective study.[23] When compared with earlier studies, the mean age of patients in our study was lower. Posterior bleeding was more common in patients with elevated BP at triage, as well as older age. This result demonstrates the difference between the epidemiology of epistaxis in the ED and the general population, indicating the need for further studies specifically targeting posterior bleeding among ED cases.

A number of limitations are acknowledged in the present study. A high ratio of inactive epistaxis, as determined by an ENT specialist, was noted among the patients in our study. The most eligible cases, according to physical parameters at triage, were not serious enough to affect the patient's hemodynamic status. Moreover, the severity of epistaxis was neither formally evaluated nor documented in the patient's medical chart. The accuracy of pinching the nasal alae using the recommended maneuver[24] was also difficult to determine while retrospectively reviewing the medical charts. Moreover, cold compression of the nose, which is commonly suggested by ENT doctors to help stop nasal bleeding, showed no statistically significant effects on the blood vessels of the nasal mucosa.[25] However, its effect on clinical outcomes has not been determined yet.

Although most patients with epistaxis who visited the ED in the present study left after effective hemostasis was established, the duration of time spent in the ED was surprisingly prolonged. Several patients with ESRD and/or DM had a disproportionately prolonged ED stay. However, we failed to demonstrate whether these comorbidities affected the process of

achieving effective hemostasis, given the limited number of cases enrolled, and the retrospective nature of this study. Thus, further studies evaluating the effect of these comorbidities on epistaxis management are needed.

In summary, BP was higher in patients who visited the ED due to epistaxis with a history of HTN at triage. Male sex, a positive history of HTN, level of GOT, observation for recurrent epistaxis at ED, and duration of recurrent bleeding were associated with returns to the ED due to recurring epistaxis within 72 hours. Antihypertensive medication was more frequently administered to patients with elevated BP at triage or a history of HTN, and these patients also exhibited the greatest reductions in BP prior to discharge. Concerns for recurrent epistaxis should be raised in patients with a history of HTN. Further studies are required to determine the effectiveness of aggressive control of BP for epistaxis, prior to achieving hemostasis.

Conflicts of Interest Statement

We declare that we have no financial or personal relationships with other people or companies that would inappropriately influence our study. There is no professional or other personal interest of any kind in any product, service, and/or company that could be construed as influencing the position presented in the manuscript entitled.

References

1. Petruson B, Rudin R. The frequency of epistaxis in a male population sample. *Rhinology* 1975;13:129-133.
2. Christensen NP, Smith DS, Barnwell SL, Wax MK. Arterial embolization in the management of posterior epistaxis. *Otolaryngol Head Neck Surg* 2005;133:748-753. doi:10.1016/j.otohns.2005.07.041
3. Cullen MM, Tami TA. Comparison of internal maxillary artery ligation versus embolization for refractory posterior epistaxis. *Otolaryngol Head Neck Surg* 1998;118:636-642. doi:10.1177/019459989811800512
4. Villwock JA, Jones K. Recent trends in epistaxis management in the United States: 2008-2010. *JAMA Otolaryngol Head Neck Surg* 2013;139:1279-1284. doi:10.1001/jamaoto.2013.5220
5. Kucik CJ, Clenney T. Management of epistaxis. *Am Fam Physician* 2005;71:305-311.
6. Herkner H, Havel C, Müllner M, et al. Active epistaxis at ED presentation is associated with arterial hypertension. *Am J Emerg Med* 2002;20:92-95. doi:10.1053/ajem.2002.31577
7. Herkner H, Laggner AN, Müllner M, et al. Hypertension in patients presenting with epistaxis. *Ann Emerg Med* 2000;35:126-130. doi:10.1016/s0196-0644(00)70131-4
8. Min HJ, Kang H, Choi GJ, Kim KS. Association between hypertension and epistaxis: systematic review and meta-analysis. *Otolaryngol Head Neck Surg* 2017;157:921-927. doi:10.1177/0194599817721445
9. Page C, Biet A, Liabeuf S, Strunski V, Fournier A. Serious spontaneous epistaxis and hypertension in hospitalized patients. *Eur Arch Otorhinolaryngol* 2011;268:1749-1753. doi:10.1007/s00405-011-1659-y
10. Lubianca-Neto JF, Bredemeier M, Carvalhal EF, et al. A study of the association between epistaxis and the severity of hypertension. *Am J Rhinol* 1998;12:269-272. doi:10.2500/105065898781389985
11. Terakura M, Fujisaki R, Suda T, Sagawa T, Sakamoto T. Relationship between blood pressure and persistent epistaxis at the emergency department: a retrospective study. *J Am Soc Hypertens* 2012;6:291-295. doi:10.1016/j.jash.2012.05.001
12. Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC7 report. *JAMA* 2003;289:2560-2572. doi:10.1001/jama.289.19.2560
13. Pallin DJ, Chng YM, McKay MP, Emond JA, Pelletier AJ, Camargo CA Jr. Epidemiology of epistaxis in US emergency departments, 1992 to 2001. *Ann Emerg Med* 2005;46:77-81. doi:10.1016/j.annemergmed.2004.12.014
14. Isezuo SA, Segun-Busari S, Ezunu E, 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;11:379-382.
15. James PA, Oparil S, Carter BL, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC8). *JAMA* 2014;311:507-520. doi:10.1001/jama.2013.284427
16. Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. The task force for the management of arterial hypertension of the European Society of Cardiology and the European Society of Hypertension. *J Hypertens* 2018;36:1953-2041. doi:10.1097/HJH.0000000000001940
17. Lubianca Neto JF, Fuchs FD, Facco SR, et al. Is epistaxis evidence of end-organ damage in patients with hypertension? *Laryngoscope* 1999;109(7 Pt 1):1111-1115. doi:10.1097/00005537-199907000-00019
18. Sarhan NA, Algamal AM. Relationship between epistaxis and hypertension: a cause and effect or coincidence?

J Saudi Heart Assoc 2015;27:79-84. doi:10.1016/j.jsha.2014.09.002

19. 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;271:237-243. doi:10.1007/s00405-013-2450-z
20. Abrich V, Brozek A, Boyle TR, Chyou PH, Yale SH. Risk factors for recurrent spontaneous epistaxis. *Mayo Clin Proc* 2014;89:1636-1643. doi:10.1016/j.mayocp.2014.09.009
21. Khan M, Conroy K, Ubayasiri K, et al. Initial assessment in the management of adult epistaxis: systematic review. *J Laryngol Otol* 2017;131:1035-1055. doi:10.1017/S0022215117002031
22. Schlosser RJ. Clinical practice. Epistaxis. *N Engl J Med* 2009;360:784-789. doi:10.1056/NEJMcp0807078
23. Waddell AN, Patel SK, Toma AG, Maw AR. Intranasal steroid sprays in the treatment of rhinitis: is one better than another? *J Laryngol Otol* 2003;117:843-845. doi:10.1258/002221503322542818
24. McGarry GW, Moulton C. The first aid management of epistaxis by accident and emergency department staff. *Arch Emerg Med* 1993;10:298-230. doi:10.1136/emj.10.4.298
25. Teymoortash A, Sesterhenn A, Kress R, Sapundzhiev N, Werner JA. Efficacy of ice packs in the management of epistaxis. *Clin Otolaryngol Allied Sci* 2003;28:545-547. doi:10.1046/j.1365-2273.2003.00773.x