Factors Associated with Hemorrhagic Transformation in Infarctions Involving the Posterior Circulation System

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Backgrounds and Purpose: Hemorrhagic transformation (HT) following stroke of the posterior circulation is a rare occurrence, and its risk factors remain relatively unknown. This study aimed at examining the rate of HT and its risk factors in patients enduring acute ischemic stroke in the territories of the vertebral, basilar, and posterior-cerebral arteries. *Materials and Methods:* A total of 217 consecutive patients the risk factors for ischemic stroke were recorded and comprehensive biochemical, cardiac assessments, and neuroimaging were performed. National Institutes of Health Stroke Scale (NIHSS) scores were calculated for each patient. Those with HT as documented with neuroimaging based on the European Cooperative Acute Stroke Study criteria and potential risk factors were assessed. *Results:* There were 217 participants with a mean age of 67.33 ± 12.44 years. Among 17 patients (7.8%) developing HT, 8 (47%) had parenchymal hematoma, and 9(53.5%) had hemorrhagic infarction. Cardioembolism was the most frequent etiological factor both in the overall group (31.0%) as well as in those with HT (41.2%). Factors that emerged as significant predictors of HT included high systolic (odds ratio [OR] 1.14; 95% confidence interval [CI] 1.08-1.20; $P< .001$) and diastolic blood pressure ($P= .001$) on the day of admission and the infarction volume of greater than or equal to 3.60 ± 3.29 cm^3(OR 1.00, 95% CI 1.00-1.01; $P< .001$). While NIHSS scores were not significantly different on Day 1, HT patients had higher NIHSS scores at Day 10(OR 1.22; 95% CI 1.09-1.36; $P< .001$), and this difference was also reflected in mRS at the end of the 3 month period. *Conclusion:* HT is a rare complication of the infarction of the posterior circulation that is associated with increased morbidity and mortality. Identification of predictive factors for HT in patients with the acute infarction of the posterior circulation may facilitate patient selection for thrombolytic treatment.

Key Words: Stroke—hemorrhagic transformation—posterior circulation—neuroimaging

Introduction

Hemorrhagic transformation (HT), which occurs spontaneously after an acute ischemic stroke (AIS) or following thrombolytic treatment of AIS, is associated with poor prognosis. Hemorrhage occurring after an infarction may vary from minor petechial bleedings to extensive areas of hemorrhage. Mechanisms thought to be responsible for the pathogenesis AIS include impaired blood-brain barrier together with cytotoxicity occurring proximal to the site of blockade. Blood products entering into the parenchyma and toxic alterations arising from reperfusion lead to clinical manifestations of HT.[1,2]

Approximately 80% of cerebrovascular diseases result from ischemic events and of these, 20% originate from the posterior circulation.[3,4] Infarctions of the anterior and posterior systems exhibit certain differences in terms of risk factors, etiology, clinical manifestations, and prognosis.[5] For instance, ischemic infarctions of the anterior circulations are mostly secondary to cardio-embolism, and

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delayed reperfusion is generally implicated in the occurrence of HT. Other risk factors include age, size of the infarct, and thrombolytic treatment. Previously, atherosclerosis of the vertebra-basilar arteries with consequent hypoperfusion, or local atherosclerotic emboli caused by both of these factors has been implicated in the etiology of the ischemic infarctions of the posterior circulation. However, advances in the field of medical imaging have actually established the role of cardio-embolic events as a common cause of such infarctions.[6-10] Until now, only few studies have examined HT in infarctions of the posterior circulation, mainly due to their relatively lower incidence and high mortality. Identification of the predictive factors for the infarctions involving this area as well as for their HTs will facilitate the treatment decisions for clinicians. Therefore, in this study we examined the HT incidence, risk factors, and clinical manifestations over a 3 month period in a group of patients with AIS of the posterior circulation who did not receive thrombolytic therapy.

Materials and Methods

Patients Characteristics

A total of 482 patients admitted to the neurology unit between July 2013 and April 2015 upon a diagnosis of acute infarction in the vertebral, basilar, or posterior-cerebellar artery based on neurological examination, computed tomography (CT) and/or diffusion weighted imaging-magnetic resonance imaging (DWI-MRI) performed at the emergency unit were screened for study eligibility. Exclusion criteria included the following: thrombolytic therapy, use of antithrombotic or anticoagulant agents prior to stroke, presence of hemorrhage in the initial CT scan or absence of AIS in the initial CT or DWI-MRI, presence of other etiological factors such as arterial dissection or vasculitis, a delay of more than 24 hours between onset of symptoms and presentation at the emergency unit, death within the first 24 hours, and bleeding secondary to surgery or shunt procedure due to decompression. Based on this exclusion criteria, a total of 217 eligible patients were enrolled into the study.

Detailed physical and neurological examinations were repeated in all patients. Age, gender, history of hypertension, cardiac disease, diabetes mellitus, chronic renal failure, stroke, or transient ischemic attacks were recorded. Blood pressure at the presentation was measured. Complete blood count, renal function tests, fasting blood glucose, HbA1c, INR, low-density lipoprotein cholesterol (LDL-C), triglycerides, total cholesterol, high-density lipoprotein cholesterol (HDL-C), electrocardiography, and cardiac echocardiography findings were recorded. Etiological assessment of the AIS was based on TOAST classification,[11] and antithrombotic and anticoagulant agents were commenced in those with an atherosclerotic or cardio-embolic etiology, respectively. During the follow-up period, neurological examination was repeated in all patients at Day 10. NIHSS (National Institutes of Health Stroke Scale) scores were calculated. Those with HT were categorized as asymptomatic (asHT, no change in NIHSS score despite HT) or symptomatic (sHT, in conjunction with increased NIHSS score) based on NIHSS assessment. Modified Rankin Scores (mRS) were also calculated using the neurological findings at 3 months of follow up. A mRS lesser than or equal to 2 was considered good functional outcome, while a mRS greater than 2 was considered poor functional outcome based on the assessments at 3 months. We performed the study after obtaining the approval of local Ethics Committee.

Imaging Analysis

In order to calculate the volume of infarction, the following formula was used in MRI images: axbxcx 0.5 mm^3. Infarctions were classified as occipital, medial-temporal, cerebellar, mesencephalon, pons, or bulbus, according to their site. Neuroimaging studies (cranial CT and/or cranial MRI) were repeated immediately in those with a worsening clinical condition, at 10 days in the remaining patients, and at 3 months in all surviving study subjects. Those with HT as documented by the repeat neuroimaging studies were classified into the 2 following groups based on European Cooperative Acute Stroke Study[12]: parenchymal hematoma (PH), and hemorrhagic infarction (HI). Associated risk factors were evaluated. Cranial imaging studies were interpreted by a senior radiologist blinded to the patients' clinical condition.

Statistical Analysis

Statistical analyses were performed with Predictive Analytics Software Statistics for Windows, Version 18.0 (SPSS Inc., Chicago, USA, Releaset 2009). Descriptive statistics included mean, standard deviation, median, minimum-maximum, rate, and frequency. The distribution of the variables was evaluated with Kolmogorov-Smirnov test. For the analysis of quantitative data chi-square test was used, while Fisher's test was used when the conditions for chi-square test were not met. For statistically significant ($P < .05$) parameters in univariate analysis, a multiple logistic regression analysis was performed. Significance was set at a $P < .05$ level, and confidence intervals were presented.

Results

A total of 217 patients with a mean ± SD age of 57.33 ± 12.44 years (28-93 years) were included; of these 122 (56.2%) were male and 95 (43.8%) were female. Among 17 patients (7.8%) with HT, 8 (47%) had PH and 9 (53%) had HI, with all PH patients being categorized in the symptomatic group. Of the 13 patients with symptomatic HT (sHT), 5 had HI and 8 had PH. Following AIS, HT occurred within 10 days in all patients, with a peak occurrence at day 2 (9 patients). Patients with or without HT did not differ significantly with regard to age or gender, while among HT patients, those with PT were younger (P= .002). Again, patients with or without HT were not significantly different in terms of risk factors (P> .05). Hypertension was the most frequent risk factor in the overall group (31.8), while transient ischemic attacks were the least frequent (9.7% overall, 17.6% in HT group). The most frequent cause of cerebral infarction in those with HT was cardioembolism (31%). On the other hand, HI rather than PH was more frequent among those with cardioembolism (P= .03). In those with no identified etiology, all 4 subjects had PH (P= .02). Also, another significant finding was the absence of HT among all cases with lacunar infarction (P= .003). Table 1 shows the demographic and clinical characteristics of the patients.

The most frequent clinical sign in the overall patient group was arterial hypertension (72.8%), with 88.2% of patients in HT group having this condition. The mean ± SD systolic blood pressure (SBP) on the day of admission was 182.06 ± 11.32 mmHg, with significantly higher values in HT group (odds ratio [OR] 1.14; 95% confidence interval [CI] 1.08-1.20; P< .001). In HT group SBP was statistically significantly higher in PH group than HI group, and in sHT group than asymptomatic HT group (P= .032, P= .003 respectively). Diastolic blood pressure on the day of admission was 86.47 ± 9.80 mm Hg among the overall study subjects, while it was significantly higher in those with HT (P = .001), although clinical and radiological findings were comparable. Neuroimaging on the day of admission showed an infarction volume of 3.60 ± 3.29 cm^3 in those with HT, and this difference was significant (OR 1.00; 95% CI 1.00-1.01; $P\leq$ .001). Patients with or without HT were comparable in terms of complete blood count and biochemistry analyses (Table 2).

Localizations of infarction among 217 study subjects included cerebellum (n = 65), occipital lobe (n = 56), pons (n = 37), medial-temporal areas supplied by the posterior-cerebral artery, mesencephalon (n = 16), and medulla oblongata (n = 11). The most frequent site involved in HT patients (n = 9) was the occipital area (52.9%), and this site was significantly more frequently involved in HT patients (P= .01). No HT patients had infarction in the brainstem, that is, mesencephalon, pons, and medulla. During the follow-up, 169 patients (77.89%) received antiaggregating agents, and 48 (22.11%) had anticoagulant agents. The groups were comparable with regard to HT. When patients were reassessed in terms of the agents used, those receiving treatment with low-molecular weight heparin (LMW heparin) and ASA were significantly more likely to have HT (P = .001 and P= .01, respectively). The localization of infarcts as well as the treatments received by the patients are shown in Table 3.

NIHSS scores at the day of admission in groups with or without HT were similar. At Day 10, HT patients had significant increase in NIHSS scores (OR 1.22; 95% CI 1.09-1.36; P< .001), although PH and HI patients did not differ significantly in terms of NIHS scores (P= .548). At month 3, a total of 10 patients (4.60%) had died, 1 with PH (5.9%) and 9 without HT (4.8). The causes of death included immobilization and intercurrent infection. mRS at 3 months was significantly worse in HT group (P = .001) (Table 4). Patients with sHT had worse mRS scores than asHT at 3 months of follow-up (P= .003).

Discussion

Posterior and anterior cerebral circulation systems exhibit certain differences with regard to collateral vessel formation and vascular resistance to bleeding, leading to differences in rates of HT and response to similar treatments.[13] The small volume of the posterior fossa along with the predisposition to poorer neurological outcomes and higher mortality represent major challenges for investigators.[13,14] In a recent study by Valentino et al, the reported rates of spontaneous HT and PH following posterior ischemic strokes were 5.9%, and 2.5%, respectively.[15] In our study, the corresponding figure for spontaneous HT was 7.8%, which is in line with their findings. Reduced occurrence of HT in infarctions of the posterior circulation has explained on the basis of smaller dimensions of infarcts or better functioning of the collateral vessels of the posterior circulation.[16,17] The brainstem is supplied by small penetrating end-arteries, which may also reduce the likelihood of HT. This is consistent with the absence of HT among our patients with brainstem infarction. In a study by Sakamato et al, HT was found in 43% of the patients with acute cerebellar infarction, and these authors emphasized the significant correlation between HT and the diameter and volume of the infarct.[18] Infarctions involving the occipital lobe, mesial-temporal area, and cerebellum tend to occupy larger volumes, and this is again in line with our observation that HT occurred only in those anatomical locations in our study. Also, the volume of the infarction in those with PH was significantly larger than those with HI. Infarctions with larger volume are thought to affect more extensive areas of the blood-brain barrier, with more severe reperfusion injury and increased likelihood of HT.[1,19]

Currently, cardiac embolism is thought to represent the most frequent cause of acute ischemic infarctions of the posterior circulation. Also, embolism from artery to artery is not uncommon. Reperfusion due to peripheral propagation of the fragmented emboli is thought to result in HT. In infarctions of the posterior cerebral artery or cerebellum, embolism represents a more common etiological mechanism. On the other hand, atherosclerosis is a main cause of ischemic strokes of the penetrating arteries supplying the brainstem. Therefore, hypertension and DM also represent important risk factors for infarctions involving this anatomical location, and this explains the much less frequent occurrence of HT in the ischemic

Table 1. *Clinical and demographic data of patients*

	Hemorrhagic transformation		*P**	Radiological classification		*P*	Clinic classification		*P*
	HT(+) (n: 17)	HT(−) (n: 200)		PH (n:8)	HI (n: 9)		sHT (n: 13)	asHT (n: 4)	
Age (mean ± SS)	68.5 ± 10.5	67.2 ± 12.6	0.618	63.5 ± 10.5	74.2 ± 7.5	**0.02**	69.8 ± 11.5	64.5 ± 5.0	0.215
Gender (E/K)	5/12	90/110	0.214	6/2	6/3	0.706	8/5	0/4	0.08
Risk factors (%)									
Hypertension	88.2	71.5	0.137	100	77.8	0.188	92.3	75.0	0.187
DM	52.9	54.0	0.933	50	55.6	0.279	61.5	25.0	0.429
TİA	17.6	9	0.247	25	11.1	0.224	15.4	25.0	0.520
SVD	23.5	43.0	0.118	25	22.2	0.569	30.8	0	0.07
AF	35.3	26.0	0.406	37.5	33.3	0.224	38.5	25.0	0.635
CRF	17.6	18.0	0.971	25.0	11.1	0.519	15.4	25.0	0.912
TOAST n (%)									
Large artery aterosclerosıs	5(29.4)	56(28.0)	0.901	3(37.5)	2(22.2)	0.437	4(30.8)	1(25.0)	0.968
Cardio embolic	7(41.2)	62(31.0)	0.387	1(12.5)	6(66.7)	**0.03**	4(30.8)	3(75)	201
Lacunar infarct	0	62(31.0)	**0.003**	0	0	-	0	0	-
Other reasons	1(5.9)	3(1.5)	0.208	0	1(11.1)	0.529	1(7.7)	0	0.434
Indeterminate	4(23.5)	20(10)	0.102	4(50)	0	**0.02**	4(30.8)	0	0.05

AF, atrial fibrillation; asHT, asymptomatic HT; DM, diabetes; HI, hemorrhagic infact; HT, hemorrhagic transformation; KBY, chronic renal failure; PH, Parenchymal hematoma; sHT, symptomatic HT; TOAST, Trial of Stroke Org 10172 in Acute Stroke Treatment.

*In the analysis of qualitative data, independent sample t test, chi-square test in the analysis of qualitative data and Fischer test were used when the chi-square conditions were not provided. *P* value of <.05 was considered as significant and indicated bold in the table.

Table 2. *Clinical and laboratory findings of patients*

	Hemorrhagic transformation		*P**	Radiological classification		*P**	Clinic classification		*P**
	HT(+) (n: 17) Mean ± SD	HT(−) (n: 200) Mean ±SD		PH (n:8) Mean ± SD	HI (n: 9) Mean ± SD		sHT (n: 13) Mean ± SD	asHT (n: 4) Mean ± SD	
CBP	182.06 ± 11.3	159.10 ±10.9	**≤0.001**	188,13 ± 7.9	176,67 ± 11.4	**0.032**	186.10 ± 7.1	168.73 ± 13.1	**0.003**
DBP	86.47 ± 9.8	77.13 ±8.2	**0,001**	91,25 ± 7.4	82,22 ± 10.0	0.051	87.69 ± 9.0	82.50 ± 12.5	0.486
GLUCOSE	160.2 ± 79.5	146.5 ± 68.1	0.433	166.1 ± 59.0	155.1 ± 97.7	0.786	162.7 ± 67.9	152.2 ± 123.3	0.879
CRE	1.16 ± 0.2	1.25 ± 0.9	0,704	1.15 ± 0.2	1.17 ± 0.2	0.805	1.17 ± 0.2	1.12 ± 0.26	0.736
URE	47.4 ± 13.4	50.9 ± 27.9	0,612	46.0 ± 11.8	48.7 ± 15.3	0.685	47.0 ± 14.6	48.7 ± 10.0	0.803
T.C	202.5 ± 32.6	195.4 ± 52.4	0,582	192.1 ± 25.1	211.8 ± 37.0	0.224	194 ± 31.6	230 ± 18.0	**0.017**
TG	138.8 ± 55.2	162.1 ± 90.2	0,298	140.3 ± 57.0	137.5 ± 57.0	0.920	136.3 ± 49.2	147.2 ± 80.2	0.810
LDL-C	131.1 ± 27.1	127.9 ± 50.9	0.798	124.2 ± 24.7	137.3 ± 29.0	0.332	125.4 ± 28.6	149.7 ± 6.1	**0.012**
HDL-C	42.1 ± 9.8	38.8 ± 10.6	0.227	39.7 ± 7.9	44.2 ± 11.4	0.369	39.2 ± 8.7	51.5 ± 7.9	**0.042**
Hb	13.4 ± 1.5	12.8 ± 1.7	0,240	13.6 ± 1.5	13.1 ± 1.6	0.546	13.0 ± 1.4	14.7 ± 0.98	0.051
PLT	241.0 ± 92.2	253.2 ± 97.6	0.619	239.1 ± 82.0	242.6 ± 105.4	0.940	238.6 ± 88.4	248.5 ± 118.1	0.885
İnfarct volume	3.60 ± 3.29	1.25 ± 1.99	**≤0.001**	4.43 ± 4.08	2.86 ± 2.41	0.362	3,61 ± 3.41	3.57 ± 3.35	0.985

asHT, asymptomatic HT; CBP, cystolic blood pressure; CRE, creatinin; DBP, diastolic blood pressure; Hb, hemoglobin; HDL-C, high-density lipoprotein-cholesterol; HI, hemorrhagic infact; HT, hemorrhagic transformation; LDL-C, low-density lipoprotein-cholesterol; PH, parenchymal hematoma; PLT, platelet; sHT, symptomatic HT; T.C, total cholesterol; TG, triglycerid.

*Independent sample t test was used in the analysis of qualitative data. *P* value of <.05 was considered as significant and indicated bold in the table.

infarctions of the brainstem.[7,20-22] The association between arterial blood pressure and HT is controversial. Although elevation of mean blood pressure during the course of the follow-up is considered to increase the HT risk, the link between high blood pressure on the first day admission and HT is unclear.[23,24] In our study, the systolic and diastolic blood pressure values measured on the first day were significantly higher in HT patients. In other groups, no significant blood pressure difference was detected during the follow-up. These observations seem to support previous reports suggesting that elevated arterial blood pressure may be an important factor in HT. Although atrial fibrillation and DM were more common in HT patients, the differences did not reach significance.

Cholesterol has major roles in maintaining the integrity of the vascular structures, and reduced cholesterol levels are believed to increase the risk of HT by causing rupture at microvascular level.[25,26] However, the association of HT with low total cholesterol and low-density lipoprotein cholesterol is far from being clear.[25,27-29] In our study, patient groups had similar lipid profiles. In a study by Lee et al among a total of 14 patients with acute posterior circulation infarction who underwent treatment with pharmacological thrombolysis and/or mechanical device, 1 had symptomatic PH, 4 had asymptomatic parenchymal hemorrhage, and 1 had ventricular hemorrhage, with no difference in risk factors between t with or without risk factors.[9] Again, these findings are consistent with our results.

HT may spontaneously develop at any time after an AIS. Although drugs used for the treatment of AIS such as acetylsalicylic acid (ASA), heparin, and thrombolytic agents have been proposed to increase the risk of HT,[23,30] the benefits of treatment are generally considered to overweight its risks.[23,30] Some authors have suggested that ASA and LMW heparin used prophylactically or to prevent deep venous thrombosis may not have a significant impact on the risk of HT.[22,31] However, in our patient group, those receiving treatment with ASA or ASA plus LMW heparin had increased risk of HT.

HT may manifest itself in a wide clinical spectrum from minor petechial bleeding to large intracerebral hematomas. The former lesions are generally asymptomatic, and can only be identified in serial neuroimaging studies.[32] On the other hand, sHT patients frequently worsen over time. One potential reason for the lack of a dramatic effect on the clinical manifestations is the occurrence of bleeding into necrotic brain tissue. In symptomatic cases of HT, a dense intracerebral hematoma is believed to result in clinical worsening due to mechanical compression effect.[2,30,32] In our patients, although NIHSS scores on day 1 were not significantly different between the study groups, a significantly higher NIHSS score was found in those with HT, as previously reported in other studies. This difference was reflected in the recovery rates at the end of the 3 month follow-up period. Accordingly, patients with HT had

Table 3. *İnfarctlocalization and folow-up treatment of patients*

	Hemorrhagic transformation		*P**	Radiological classification		*P*	Clinical classification		*P*
	HT(+) (n: 17)	HT(-) (n: 200)		PH (n: 8)	HI (n: 9)		sHT (n: 13)	asHT (n: 4)	
Lesion Localization (%)									
M.T.L	29.4	13.5	0.076	37.5	22.2	0.490	30.8	25.0	0.262
Occipital L.	52.9	23.5	**0.01**	50	55.6	0.819	53.8	50	0.504
Cerebellum	17.6	31	0.249	12.5	22.2	0.596	15.4	25.0	0.440
Mesencephalone	0	8	0.226	0	0	-	0	0	-
Pons	0	18.5	0.08	0	0	-	0	0	-
Bulbus	0	5.5	0.321	0	0	-	0	0	-
Treatment (%)									
ASA	76.5	45.5	**0.01**	75	77.8	0.893	76.9	75	0.607
ASA + CLP	5.9	8.0	0.755	0	11.1	0.529	7.7	0	0.755
LMWH	11.8	0	**0.001**	25	0	0.216	15.4	0	**0.001**
LMWH + ASA	0	1.5	0.782	0	0	-	0	0	-
WARFARİN	5.9	21	0.133	0	1	0.529	0	25	0.05
ANTIAGGREGANT n (%)	14 (82,4)	155 (77,5)	0,770	6 (75,0)	8 (88,9)	0,576	11 (84,6)	3 (75,0)	0,828
ANTICOAGULANT n (%)	3 (17,6)	45 (22,5)	0,457	2 (25,0)	1 (11,1)	0,453	2 (15,4)	1 (25,0)	0,816

ASA, acetylalicic acid; asHT, asemptomatik HT; DMAH, low molecularl weight heparin; HD, hemorajik dönüşüm; HI, hemorajikinfarkt; KLP, clopidogrel; M.T.L, medial temporal lobe; n: patien t number; PH, parenkimalhematom; sHT, semptomatik.

*In the analysis of qualitative data, independent sample t test, chi-square test for the analysis of qualitative data and Fischer test were used when the chi-square conditions were not provided. *P* value of <.05 was considered as significant and indicated bold in the table.

Table 4. *Patient and functional evaluation of patients*

	Hemorrhagic Transformation		P*	Radiological Classification		P*	Clinical Classification		P*
	HT(+) (n: 17)	HT(-) (n: 200)		PH (n: 8)	HI (n: 9)		sHT (n: 13)	asHT (n: 4)	
NIHSS (Mean ± SD)									
1.DAY	5.8 ± 2.3	5.8 ± 2.9	0.981	4.8 ± 1.4	6.7 ± 2.7	0.099	6.1 ± 2.3	5.0 ± 2.5	0.463
10.DAY	8.1 ± 3.9	4.5 ± 3.1	**≤0.001**	8.7 ± 4.8	7.5 ± 3.0	0.548	9.0 ± 3.8	5.0 ± 2.5	**0.042**
mRS(90. Gün) n, (%)									
≤2	6(35.3)	137(72.5)	**0.001**	2(25.0)	4(44.4)	0.399	3(23.1)	3(75)	0.002
3-5	10(58.8)	43 (22.7)	**0.001**	5(62.5)	5(55.6)	0.771	9(69.2)	1(25.0)	0.003
6 (Mortalite)	1(5.9)	9(4.8)	0.837	1	0	0.471	1	0	0.742

asHT, asymptomatic HT; HI, hemorrhagic infact; HT, hemorrhagic transformation; n, Patient number; PH, parenchymal hematoma; sHT, symptomatic HT.

*In the analysis of qualitative data, independent sample t test, chi-square test in the analysis of qualitative data and Fischer-test were used when the chi-square conditions were not provided. *P* value of $<.05$ was considered as significant and indicated bold in the table

significantly worse functional status. We believe that, NIHSS score at day 10 after admission may represent a significant predictor for longer term outcomes. The mortality rates in patients with HT, without HT, and in the overall study group were 5.9%, 4.8%, and 4.6%, respectively. In the few studies published until now, the reported mortality rates for infarctions of the posterior circulation were 8.1% and 11.5%.[27,23] Exclusion of patients who died within the first 24 hour period as well as those who had undergone thrombolytic therapy may explain the lower rates of mortality in our study.

The frequency and incidence of stroke subtypes exhibit significant differences between countries, and even between different regions in the same country. This is explained by the differences in lifestyle, genetic factors, and environmental factors etc. Our literature search for studies examining spontaneous HT rates in patients with the infarction of the posterior circulations has revealed only few studies. The association of HT with thrombolytic therapy has attracted recent attention due to increasing use of thrombolytic therapy. In this study, SBP on the day of admission and the stroke volume emerged as predictive risk factors for HT, while poorer NIHSS scores at day 10 were associated with poor functional outcomes. One limitation of our study was the fact that not all patients could undergo a gradient-echo MR imaging study. We believe that further studies may shed more light on predictive risk factors for HT in AISs of the posterior circulation, and alternative treatments instead of thrombolysis may be administered to high risk patients.

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