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Original Article

Determinants of infarction patterns in posterior cerebral circulation

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Abstract

Background

Ischemic stroke is defined as focal or global neurological dysfunction lasting longer than 24 h or leading to death that is caused by vascular insult, either stricture or occlusion of a specific vascular territory.

Aim of the study

This study aimed to determine the relationship between different risk factors and different infarction patterns in posterior circulation such single small lacunar lesion, single large lesion, and multiple scattered lesions.

Patients and methods

This study included 60 patients recruited from the stroke unit of Ain Shams University and El Sahel Teaching Hospitals during the period from April to October 2018 with the diagnosis of posterior circulation ischemic stroke. The study population was divided into three groups according to the infarction pattern. Infarction patterns were categorized into a single small lacunar lesion (20 patients) (group I), a single large lesion (20 patients) (group II), and multiple scattered lesions (20 patients) (group III).

Results

There was no significant difference between the three groups in the presence of vascular risk factors such as hypertension ($P = 0.153$), diabetes ($P = 0.317$), dyslipidemia ($P = 0.420$), presence of cardiac diseases ($P = 0.180$), and smoking ($P = 0.931$). The only significant difference in terms of vascular risk factors was atrial fibrillation (AF). AF was present in six patients in group II and six patients in group III, and not present in group I patients ($P = 0.024$).

Conclusion

Different vascular risk factors such as hypertension, diabetes, dyslipidemia, and smoking are present in all infarction patterns of posterior cerebral circulation, either single or multiple infarctions, and there were no significant differences in the presence of these vascular risk factors in relation to the type of the vascular lesion. AF and significant vertebrobasilar stenosis were mostly associated with large and multiple infarct lesion patterns. Small-vessel disease was the most common stroke etiology for a single small lacunar lesion, whereas large artery atherosclerosis was mostly present in a single large lesion and multiple lesions. Strict control of vascular risk factors is highly recommended in all infarction patterns of posterior circulation.

Keywords: Infarction patterns, ischemic stroke, posterior cerebral circulation

Introduction

Stroke is the second most common cause of death among individuals older than 60 years of age. Stroke is the most common cause of adult disability and the second most important cause of dementia worldwide [1].

Approximately 16 million first-ever strokes occur in the world, causing a total of 5.7 million deaths per year [2]. About 85% of all stroke deaths are registered in low-income and middle-income countries that additionally account for 87% of total losses because of stroke concerning disability-adjusted life years, calculated worldwide in 72 million per year clear [3].

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Stroke accounts for 6.4% of all deaths in Egypt and therefore ranks third after heart diseases and liver diseases [4]. The incidence rate for all strokes in Egypt is 250 per 100 000 population annually in Egypt. It ranges between 22.7 and 180 per 100 000 population each year for first-ever and recurrent stroke, and between 29.8 and 250 per 100 000 population each year for first-ever stroke. The prevalence rate in Egypt for first ever and recurrent stroke ranged between 508 and 777 per 100 000 population in 1992 and 2001–2013, respectively [5].

Early predictors of functional outcome after stroke are necessary for better planning of treatment and care [6].

Posterior circulation ischemic stroke is a clinical syndrome that is classically defined by infarction occurring within the vascular territory supplied by the vertebrobasilar arterial system. It accounts for ~20–25% of all strokes with different clinical presentations that differ from anterior circulation stroke with reference to etiology, clinical features, and prognosis [7].

Posterior circulation infarctions are known to have different patterns such as single small lacunar lesion, a single large territorial lesion, or multiple scattered infarct lesions that are caused by several risk factors. However, it remains unknown which factors contribute to these different infarctions in posterior circulation [8].

The prognosis and clinical outcome after posterior circulation infarction are more dangerous than anterior circulation infarction according to the modified Rankin scale (mRS) 3 months after the occurrence of infarction. Disability was 32.3% in minor posterior circulation infarct compared with 30.3% in minor anterior circulation infarct, and death was 1.3 and 1.5%, respectively [9].

Aim of the study

In this study, we aimed to determine the relationship between different risk factors and different infarction patterns in posterior circulation such as single small lacunar lesion, single large lesion, and multiple scattered lesions.

Patients and methods

Type of study

This was a cross-sectional observational prospective hospital-based study.

Study setting

Patients were recruited from the stroke unit of Ain Shams University and El Sahel Teaching Hospitals.

Study period

This study was carried out for about 6 months.

Inclusion criteria

Patients with acute cerebral infarction admitted within 48 h of onset of symptoms were included. The diagnosis of ischemic stroke was made on the basis of the clinical features in combination with brain imaging. The infarction will be within the territory of posterior circulation.

Exclusion criteria

The exclusion criteria were as follows:

(1) Patients with infarction within anterior circulation.
(2) Patients with cerebral venous thrombosis.
(3) Patients who refused to participate in the study.

Sampling method

Simple random sample.

Sample size

Sixty patients were included.

Ethical considerations

Written consent was obtained from the patients or their guardians.

Study tools

MRI brain and magnetic resonance angiography (MRA) imaging were used.

Study procedures

Patients in this study were subjected to the following:

(1) Assessment of detailed medical history including personal history, family history, and vascular risk factors such as:
   (a) Hypertension:

Defined as a history of use of antihypertensive medications, or if systolic blood pressure less than 140 mmHg, diastolic blood pressure less than 90 mmHg, or both during admission for 4 days at least.

   (b) Diabetes mellitus:

Defined as a history of use of insulin or oral hypoglycemic agents, or if blood glucose level was more than or equal to 126 mg/dl after an overnight fast, or if more than or equal to 200 mg/dl after 2 h from ingestion of 75 g of oral glucose on at least two separate occasions.

   (c) Previous history of cardiac diseases:

Myocardial infarction, angina, coronary revascularization, rheumatic heart diseases, and atrial fibrillation (AF).

   (d) Previous history of cerebrovascular events:

(1) Ischemic stroke, transient ischemic attack (TIA), and intracerebral hemorrhage.
(2) The time from the onset of symptoms till admission to the hospital was determined.
(3) General medical examination including blood pressure measurement was performed at the time of admission.
(4) Detailed neurological examination with assessment of stroke severity using the NIHSS score was performed on admission, 24 h after admission, and at 7 days from the onset of symptoms.
(5) The patients' functional status was assessed using the mRS at admission and on discharge from hospital and at the 7-day follow-up from the onset of symptoms.
(6) The duration of admission of the patients in the hospital was determined.
(7) The degree of improvement from patients' admission to discharge was assessed.

(8) The laboratory investigations included the following:
   (a) Random blood sugar (RBS) at the time of admission and glycated hemoglobin
   (b) Complete blood picture.
   (c) Erythrocyte sedimentation rate (ESR).
   (d) Coagulation profile.
   (e) Lipid profile (total cholesterol, low-density lipoproteins, high-density lipoproteins, and triacylglyceride).
(9) Computed tomography brain was performed to obtain information on the presence or absence of cerebral infarction and intracerebral hemorrhage.
(10) MRI of the brain was performed for all patients including Diffuse Weighted Imaging (DWI), Fluid Attenuation Inversion Recovery (FLAIR), T1-weighted imaging (T1W1), T2 weighted imaging (T2W1), and gradient-echo T2*-weighted MRI scans. Assessment of the site and the size of infarction was performed. The volume of infarction was determined roughly by multiplying the largest diameter of the lesion by the largest length of the lesion by the number of the cuts and then dividing the result over 2. MRA was visualized for the presence of intracranial stenosis or occlusion. Significant intracranial stenosis was assumed if more than or equal to 50%.
(11) An ECG was performed for all patients on admission or whenever needed for the assessment of any cardiac arrhythmia.
(12) Transthoracic echocardiography was performed for all patients and the ejection fraction, any segmental wall motion abnormalities, diameter of chambers, valvular heart diseases, and any intracardiac masses and transesophageal echocardiography were determined only for selected cases. Data from these were useful to determine the presence of cardiac diseases.
(13) Stroke subtypes were defined using the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) criteria as one of five categories on the basis of risk factors as well as clinical and brain imaging features: large artery atherosclerosis, cardioembolism, small-vessel occlusion (lacunar), undetermined etiology stroke, or other etiology [10].
(14) The outcome was assessed using the mRS 7 days from the onset of symptoms and was categorized as favorable (mRS 0–2; patients can perform his or her daily activities independently) or unfavorable (mRS 3–6; dependent patient or dead) [11].
(15) Infarction patterns were categorized into a single large lesion (20 patients), a single small lacunar lesion (20 patients), and multiple scattered lesions (20 patients). The relationships between infarction patterns and clinical and investigational data were investigated.

Statistical analysis

Recorded data were analyzed using the statistical package for social sciences, version 20.0 (SPSS Inc., Chicago, Illinois, USA). Quantitative data were expressed as mean ± SD. Qualitative data were expressed as frequency and percentage.

The following tests were done:
(1) A one-way analysis of variance was used when comparing between more than two means.
(2) Post-hoc test: least significant difference was used for multiple comparisons between different variables.
(3) χ^2 test of significance was used to compare proportions between qualitative parameters.
(4) The confidence interval was set to 95% and the margin of error accepted was set to 5%. Therefore, the *P* value was considered significant according to the following:
(5) *P* value:
   (a) *P* value less than or equal to 0.05 was considered significant.
   (b) *P* value less than or equal to 0.001 was considered highly significant.
   (c) *P* value more than 0.05 was considered insignificant.

Results

Table 1 shows no statistically significant difference between the groups according to demographic data.

Table 2 shows a statistically significant difference between the groups according to AF.

Table 3 shows a statistically significant difference between the groups according to previous TIAs.

Table 4 shows no statistically significant difference between the groups according to blood pressure.

Table 5 shows no statistically significant difference between the groups according to echo ejection fraction%.

Tables 6 and 7 shows a statistically significant decrease in the mean NIHSS in group I, single lacunar lesion, compared with group II,

Table 1: Comparison between groups according to demographic data

Demographic data	All patients (*n*=60)	Group I (single small lacunar lesion) (*n*=20)	Group II (single large lesion) (*n*=20)	Group III (multiple lesions) (*n*=20)	*F*/χ^2#	*P*
Age (years)						
Mean±SD	59.20±9.63	56.60±9.78	61.70±8.82	59.30±10.03	1.425	0.249
Range	38-79	38-71	38-77	40-79		
Sex [*n* (%)]						
Female	27 (45.0)	11 (55.0)	7 (35.0)	9 (45.0)	1.616#	0.446
Male	33 (55.0)	9 (45.0)	13 (65.0)	11 (55.0)		

F, analysis of variance test. #χ^2, χ^2 test. *P* value more than 0.05 (NS).

Table 2: Comparison between groups according to vascular risk factors

Vascular risk factors	All patients (*n*=60) [*n* (%)]	Group I (single small lacunar lesion) (*n*=20) [*n* (%)]	Group II (single large lesion) (*n*=20) [*n* (%)]	Group III (multiple lesions) (*n*=20) [*n* (%)]	χ^2	*P*
Hypertension	52 (86.7)	19 (95.0)	15 (75.0)	18 (90.0)	0.375	0.153
Diabetes	38 (63.3)	14 (70.0)	14 (70.0)	10 (50.0)	2.297	0.317
Dyslipidemia	31 (51.7)	12 (60.0)	8 (40.0)	11 (55.0)	1.735	0.420
Cardiac diseases						
Positive	25 (41.7)	5 (25.0)	10 (50.0)	10 (50.0)	3.429	0.180
AF	12 (20.0)	0 (0.0)	6 (30.0)	6 (30.0)	7.500	0.024*
Ischemic heart disease	21 (35.0)	5 (25.0)	8 (40.0)	8 (40.0)	1.319	0.517
Rheumatic heart disease	2 (3.3)	0 (0.0)	2 (10.0)	0 (0.0)	4.138	0.126
Coronary revascularization	6 (10.0)	1 (5.0)	2 (10.0)	3 (15.0)	1.111	0.574
Smoking						
Nonsmoker	38 (63.3)	13 (65.0)	13 (65.0)	12 (60.0)	0.144	0.931
Smoker	22 (36.7)	7 (35.0)	7 (35.0)	8 (40.0)		

χ^2, χ^2 test; AF, atrial fibrillation. *P* value more than 0.05 (NS). **P* value less than 0.05 (S).

Table 3: Comparison between groups according to previous transient ischemic attacks and previous stroke

	All patients (*n*=60) [*n* (%)]	Group I (single small lacunar lesion) (*n*=20) [*n* (%)]	Group II (single large lesion) (*n*=20) [*n* (%)]	Group III (multiple lesions) (*n*=20) [*n* (%)]	χ^2	*P*
Previous TIA	29 (48.3)	8 (40.0)	7 (35.0)	14 (70.0)	5.740	0.037*
Previous stroke	19 (31.7)	5 (25.0)	6 (30.0)	8 (40.0)	1.078	0.583

χ^2, χ^2 test; TIA, transient ischemic attack. *P* value more than 0.05 (NS). **P* value less than 0.05 (S).

Table 4: Comparison between groups according to blood pressure (mmHg)

Blood pressure (mmHg)	All patients (*n*=60)	Group I (single small lacunar lesion) (*n*=20)	Group II (single large lesion) (*n*=20)	Group III (multiple lesions) (*n*=20)	*F*	*P*
SBP						
Mean±SD	157.17±30.81	159.50±31.20	155.00±29.11	157.00±33.42	0.104	0.902
Range	110-230	110-230	110-220	110-220		
DBP						
Mean±SD	89.17±12.25	88.50±11.37	88.50±13.09	90.50±12.76	0.173	0.842
Range	70-120	70-120	70-120	70-110		

DBP, diastolic blood pressure; *F*, analysis of variance test; SBP, systolic blood pressure. *P* value more than 0.05 (NS).

Table 5: Comparison between groups according to echo

Echo EF%	All patients (*n*=60)	Group I (single small lacunar lesion) (*n*=20)	Group II (single large lesion) (*n*=20)	Group III (multiple lesions) (*n*=20)	*F*	*P*
Mean±SD	51.50±10.82	56.65±11.90	48.93±10.27	43.78±9.19	1.076	0.296
Range	20-80	30-80	25-70	20-65		

EF, ejection fraction; *F*, analysis of variance test. *P* value more than 0.05 (NS).

single large embolic lesion, and also a significant decrease in the mean NIHSS score in group II compared with group III.

Tables 8–12 shows a statistically significant difference between the groups according to the duration of admission of hospital (days).

Tables 13–16 shows no statistically significant difference between groups in the coagulation profile (prothrombin time, international normalized ratio, and partial thromboplastin time).

Table 17 shows a statistically significant difference between the groups according to occipital, medical temporal, cerebellum, thalamic posterior, and pons.

Discussion

Posterior circulation ischemic stroke is a clinical syndrome associated with ischemia related to stenosis, in-situ thrombosis,

Table 6: Comparison between groups according to the NIHSS score

NIHSS score	All patients (*n*=60)	Group I (single small lacunar lesion) (*n*=20)	Group II (single large lesion) (*n*=20)	Group III (multiple lesions) (*n*=20)	*F*	*P*
On admission						
Mean±SD	7.03±4.46	5.30±2.62	6.45±3.56[a]	9.35±5.74[ab]	4.977	0.010*
Range	1-18	1-10	2-17	2-18		
24 h after admission						
Mean±SD	6.20±2.25	4.05±2.09	5.30±3.88[a]	9.25±3.12[ab]	6.301	0.003*
Range	1-20	1-7	1-19	1-20		
At 7 days from the onset of symptoms						
Mean±SD	5.02±2.12	1.45±0.99	2.95±1.00[a]	10.65±5.03[ab]	7.063	0.002*
Range	0-34	0-3	0-28	0-34		

F, analysis of variance test. [a]Significant difference from group I. [b]Significant difference from group II. **P* value less than 0.05 (S; post hoc).

Table 7: Comparison between groups according to the modified Rankin score

Modified Rankin score	All patients (*n*=60)	Group I (single small lacunar lesion) (*n*=20)	Group II (single large lesion) (*n*=20)	Group III (multiple lesions) (*n*=20)	*F*	*P*
On admission						
Mean±SD	2.95±1.42	2.45±1.28	2.95±1.32	3.45±1.54	2.618	0.082
Range	1-5	1-4	1-5	1-5		
At discharge						
Mean±SD	1.90±1.09	1.00±0.98	1.75±0.52[a]	2.95±1.31[ab]	6.616	0.003*
Range	0-6	0-3	0-6	0-6		
Degree of improvement on admission and at discharge (%)	35.6	59.2	40.7[a]	14.5[ab]	8.498	<0.001**
At 7th days of follow up						
Mean±SD	1.82±1.05	0.96±0.94	1.68±0.50[a]	2.83±1.26[ab]	4.291	0.024*
Range	0-6	0-3	0-6	0-6		

F, analysis of variance test. [a]Significant difference from group I. [b]Significant difference from group II. *P* value more than 0.05 (NS).**P* value less than 0.05 (S; post hoc).

Table 8: Comparison between groups according to the duration of admission of hospital (days)

Duration of admission to the hospital (days)	All patients (*n*=60)	Group I (single small lacunar lesion) (*n*=20)	Group II (single large lesion) (*n*=20)	Group III (multiple lesions) (*n*=20)	*F*	*P***
Mean±SD	8.76±1.84	3.61±0.76	7.21±1.51[a]	11.33±2.38[ab]	13.819	<0.001
Range	2-15	2-5	4-10	7-15		

F, analysis of variance test. [a]Significant difference from group I. [b]Significant difference from group II. ***P* value less than 0.001 (HS; post hoc).

or embolic occlusion of the vertebrobasilar arterial system. It accounts for 20–25% of ischemic strokes [12].

Early recognition of posterior circulation stroke or TIAs may prevent functional disability and save lives, but it remains more difficult to recognize and treat effectively than other stroke types. Delayed or incorrect diagnosis may have catastrophic consequences, including potentially preventable death or severe functional disability, if acute treatment or secondary prevention is delayed [13].

This study aimed to determine the relationship between different risk factors and different patterns of infarctions in posterior circulation. This was across a sectional observational prospective hospital-based study carried out on 60 patients with first ever acute posterior circulation ischemic stroke. Patients were recruited from the stroke unit of Ain Shams University and El Sahel Teaching Hospitals.

The study population was divided into three groups according to the infarction pattern. Infarction patterns were categorized into a single small lacunar lesion (20 patients) (group I), a single large lesion (20 patients) (group II), and multiple scattered lesions (20 patients) (group III).

In this study, there were no significant differences between the three groups in the age and sex of the patients.

Similar findings were reported by many previous studies and no significant differences were found between patients with single infarction and those with multiple infarctions on DWI

Table 9: Comparison between groups according to the early functional outcome (on the basis of modified Rankin scale at 7 days from onset of symptoms)

Study population	All patients (*n*=60) [*n* (%)]	Group I (single small lacunar lesion) (*n*=20) [*n* (%)]	Group II (single large lesion) (*n*=20) [*n* (%)]	Group III (multiple lesions) (*n*=20) [*n* (%)]	χ^2	*P*
Favorable outcome (0-2)	44 (73.3)	19 (95.0)	15 (75.0)	10 (50.0)	10.398	0.006*
Unfavorable Outcome (3-6)	16 (26.7)	1 (5.0)	5 (25.0)	10 (50.0)		

χ^2, χ^2 test. **P* value less than 0.05 (S).

Table 10: Comparison between groups according to MRI and magnetic resonance angiography

MRI and MRA	All patients (*n*=60)	Group I (single small lacunar lesion) (*n*=20)	Group II (single large lesion) (*n*=20)	Group III (multiple lesions) (*n*=20)	F/χ^2#	*P*
Volume of infarction (cm^3)						
Mean±SD	26.41±14.24	0.48±0.14	41.77±15.32[a]	36.97±14.08[a]	6.114	0.004*
Range	0.1-200	0.1-1.1	2-200	0.37-148		
Presence of significant intracranial stenosis [*n* (%)]						
Negative	34 (56.7)	15 (75.0)	10 (50.0)	9 (45.0)	4.208#	0.122
Positive	26 (43.3)	5 (25.0)	10 (50.0)	11 (55.0)		

F, analysis of variance test; MRA, magnetic resonance angiography. #χ^2, χ^2 test. [a]Significant difference from group I. [b]Significant difference from group II. *P* value more than 0.05 (NS). **P* value less than 0.05 (S; post hoc).

Table 11: Comparison between groups according to the type of stroke

Type of stroke	All patients (*n*=60) [*n* (%)]	Group I (single small lacunar lesion) (*n*=20) [*n* (%)]	Group II (single large lesion) (*n*=20) [*n* (%)]	Group III (multiple lesions) (*n*=20) [*n* (%)]	χ^2	*P**
Large artery atherosclerosis	32 (53.3)	0 (0.0)	16 (80.0)	16 (80.0)	34.286	<0.001**
Cardioembolic	5 (8.3)	0 (0.0)	3 (15.0)	2 (10.0)	3.055	0.217
Small-vessel disease	20 (33.3)	20 (100.0)	0 (0.0)	0 (0.0)	60.00	<0.001**
Stroke of undetermined etiology	3 (5.0)	0 (0.0)	1 (5.0)	2 (10.0)	0.002	0.963

χ^2, χ^2 test. *P* value more than 0.05 NS **P* value less than 0.05 (S). ***P* value less than 0.001 (HS).

Table 12: Comparison between groups according to complete blood count

CBC	All patients (*n*=60)	Group I (single small lacunar lesion) (*n*=20)	Group II (single large lesion) (*n*=20)	Group III (multiple lesions) (*n*=20)	*F*	*P*
Hemoglobin						
Mean±SD	13.38±1.63	12.94±1.80	13.54±1.56	13.68±1.51	1.171	0.317
Range	9-16.9	9-15.6	10-16.9	10.5-15.7		
Total leukocyte count						
Mean±SD	9.53±4.04	9.91±3.98	7.95±3.61	10.72±4.19	2.614	0.082
Range	3.7-22.5	4.2-16.7	3.7-20.9	4.7-22.5		
Platelet count						
Mean±SD	259.53±101.59	310.50±125.02	233.85±86.72[a]	234.25±69.27[a]	4.182	0.020*
Range	80-555	80-555	88-392	109-412		

CBC, complete blood count; *F*, analysis of variance test. [a]Significant difference from group I. [b]Significant difference from group II. *P* value more than 0.05 (NS). **P* value less than 0.05 (S; post hoc).

in terms of age and sex [14], whereas other studies showed that vertebrobasilar artery tortuosity was significantly more common in elderly patients aged more than 60 years than less than 60 years and there was a strong association between vertebral artery tortuosity and multiple small posterior circulation infarctions in the brainstem and the cerebellum [15].

In this study, there were no statistically significant differences in the presence of vascular risk factors such as hypertension, diabetes, dyslipidemia, presence of cardiac diseases, and smoking in relation to the type of vascular lesion. The only significant difference in vascular risk factors was AF. AF was present in six patients in group II

Table 13: Comparison between groups according to the coagulation profile

Coagulation profile	All patients (*n*=60)	Group I (single small lacunar lesion) (*n*=20)	Group II (single large lesion) (*n*=20)	Group III (multiple lesions) (*n*=20)	*F*	*P*
Prothrombin time						
Mean±SD	13.78±1.23	13.54±0.91	14.02±1.06	13.79±1.61	0.777	0.465
Range	11.5-19	11.5-15	12.5-16	12-19		
Partial thromboplastin time						
Mean±SD	31.37±2.99	31.61±3.18	30.70±2.45	31.80±3.29	0.765	0.470
Range	25.3-42	26-40	26-35	25.3-42		
INR						
Mean±SD	1.12±0.13	1.10±0.09	1.14±0.12	1.13±0.17	0.339	0.714
Range	1-1.7	1-1.24	1-1.37	1-1.7		

INR, international normalized ratio. *P* value more than 0.05 (NS).

Table 14: Comparison between groups in the lipid profile

Lipid profile	All patients (*n*=60)	Group I (single small lacunar lesion) (*n*=20)	Group II (single large lesion) (*n*=20)	Group III (multiple lesions) (*n*=20)	*F*	*P**
Triglycerides (mg/dl)						
Mean±SD	192.20±104.66	206.05±105.39	196.20±128.05	174.35±77.18	0.472	0.626
Range	61-575	61-483	88-575	84-345		
Total cholesterol (mg/dl)						
Mean±SD	184.62±48.21	196.05±51.33	185.40±39.35	172.40±52.33	1.216	0.304
Range	77-323	108-323	128-266	77-245		
LDL (mg/dl)						
Mean±SD	128.12±23.29	132.40±24.70	128.30±25.54	123.65±19.53	0.700	0.501
Range	90-195	94-178	95-195	90-165		
HDL (mg/dl)						
Mean±SD	43.28±3.14	44.05±3.27	42.70±2.94	43.10±3.19	0.977	0.383
Range	37-50	37-50	38-48	38-49		

F, analysis of variance test; HDL, high-density lipoproteins; LDL, low-density lipoprotein. *P* value more than 0.05 (NS). **P* value less than 0.05 (S).

Table 15: Comparison between groups in random blood sugar on admission and glycated hemoglobin

	All patients (*n*=60)	Group I (single small lacunar lesion) (*n*=20)	Group II (single large lesion) (*n*=20)	Group III (multiple lesions) (*n*=20)	*F*	*P*
Random blood sugar at admission (mg/dl)						
Mean±SD	237.88±94.74	209.25±84.08	278.90±103.10a	225.50±85.89ab	3.177	0.049*
Range	32-452	32-367	132-452	133-396		
Glycated hemoglobin						
Mean±SD	6.72±1.65	6.76±1.40	7.05±1.70	6.36±1.83	0.889	0.417
Range	4-9.6	4.3-9	4.2-9.6	4-9.5		

F, analysis of variance test. *P* value more than 0.05 (NS). **P* value less than 0.05 (S; post hoc). [a]Significant difference from group I. [b]Significant difference from group II.

Table 16: Comparison between groups in the erythrocyte sedimentation rate at 1 h

ESR	All patients (*n*=60)	Group I (single small lacunar lesion) (*n*=20)	Group II (single large lesion) (*n*=20)	Group III (multiple lesions) (*n*=20)	*F*	*P*
Mean±SD	12.97±5.09	11.25±3.97	12.35±4.40	15.30±6.01ab	3.691	0.031*
Range	5-30	5-18	5-25	5-30		

ESR, erythrocyte sedimentation rate; *F*, analysis of variance test. [a]Significant difference from group I. [b]Significant difference from group II. *P* value more than 0.05 (NS). **P* value less than 0.05 (S; post hoc).

versus six patients in group III, and not present in any of the patients in group I.

Previous studies are in agreement with these results and found that patients with AF were more prone to develop multiple

Table 17: Comparison between groups according to the site of infraction

Site of infarction	All patients ($n=60$)	Group I (single small lacunar lesion) ($n=20$)	Group II (single large lesion) ($n=20$)	Group III (multiple lesions) ($n=20$)	χ^2	*P*
Occipital	28 (46.7)	2 (10.0)	16 (80.0)	10 (50.0)	19.821	<0.001**
Medial temporal	9 (15.0)	0 (0.0)	4 (20.0)	5 (25.0)	6.490	0.044*
Cerebellum	15 (25.0)	2 (10.0)	1 (5.0)	12 (60.0)	19.733	<0.001**
Thalamic	7 (11.7)	0 (0.0)	0 (0.0)	7 (35.0)	15.849	<0.001**
Brain stem						
Midbrain	3 (5.0)	1 (5.0)	0 (0.0)	2 (10.0)	2.105	0.349
Pons	25 (41.7)	15 (75.0)	3 (15.0)	7 (35.0)	15.360	<0.001**
Medulla oblongata	2 (3.3)	0 (0.0)	0 (0.0)	2 (10.0)	4.138	0.126

χ^2, χ^2 test. *P* value more than 0.05 (NS). **P* value less than 0.05 (S). ***P* value less than 0.001 (HS).

lesions in posterior circulation than a single small lesion because of cardioembolism that block either a large territorial vessel or small penetrating arteries [16], whereas others did not report such a relationship [17].

Previous studies have found that the frequency of vascular risk factors such as hypertension, uncontrolled diabetes mellitus, dyslipidemia, and smoking was high in all types of posterior circulation infarctions, either single or multiple lesions; this could explain the absence of any statistically significant difference between groups in vascular risk factors [14]. This needs to be validated in larger scale trials studying specifically the association between vascular risk factors and different infarction patterns.

In this study, there was a statistically significant difference between the groups in the occurrence of previous TIAs and the occurrence of posterior circulation stroke, especially of multiple scattered lesion patterns. Previous studies have confirmed this as it was found that the presence of vertebrobasilar stenosis was more common among patients with multiple scattered lesions than other types, and hence these patients were more prone to recurrent TIAs and multiple microembolic signals that can lead to multiple acute infarctions [18].

Also, previous studies showed that patients with vertebrobasilar TIA or minor stroke have a high prevalence of vertebrobasilar stenosis, which is associated with a high risk of recurrent stroke, and in patients with clinically definite vertebrobasilar TIA, the absolute risk of stroke at 1 year was 17.1% [7].

There were no significant differences between the three groups in the result of echocardiography ejection fraction as a risk of posterior circulation stroke. Previous studies have shown that heart failure and low ejection fraction were risk factors for ischemic stroke and associated with poor outcome in acute ischemic stroke [19]. However, others reported that preserved ejection fraction was associated with early favorable outcome of acute ischemic stroke patients [20].

In this study, there were statistically significant differences between the three groups in the NIHSS score on admission, after 24 h, and at 7 days from admission. It was found that the patients in group III had significantly higher scores of NIHSS compared with the patients in group I and group II. Some previous studies have supported these results [21]. This can be explained by multiple br ain areas affection that may affect stroke severity assessment by NIHSS score.

On comparing the NIHSS score on admission with the scores after 7 days, it was observed that the patients in group I and the patients in group II showed improvements unlike the patients in group III, who developed deterioration in their neurological status manifested by an increase in the mean NIHSS score; this was explained by multiple brain areas affection that might worsen the functional outcome and prolonged hospital stay, which may be associated with an increased risk of neurological and medical complication during hospitalization. Also, early neurological worsening was associated with extracranially and intracranially large-vessel stenosis or occlusions that were usually more common in the patients in group III than other patterns of posterior circulation stroke. Some previous studies have supported this [22].

It was found that there were statistically significant differences between the three groups in the mRS score at discharge, and at the 7-day follow-up from the onset of symptoms and the degree of improvement from admission to discharge. It was observed that the degree of improvement and hence favorable functional outcome were the highest among the patients in group I, followed by the patients in group II, and finally, group III, which showed the least degree of improvement and hence poor functional outcome.

There were statistically significant differences between the three groups in the duration of admission at hospital; the patients in group III tended to have a longer duration of admission. Previous studies have supported this and shown that in-hospital medical complications (vascular, urinary, and infections) are relevant factors that influence the duration of hospitalization after acute stroke. Therefore, prevention of potentially modifiable risk factors for medical complications is an important aspect of the early management of patients with this type of stroke [23].

Similar findings were reported by Saxena *et al.* [24] and Jaul *et al.* [25], and showed that prolonged hospitalization was associated with pressure sores and sepsis.

In terms of the MRI findings of the study population, it seemed logical that the patients in group II and the patients in group III had larger volumes of infarction compared to the patients in group I, and this was statistically significant.

Previous studies have shown similar results and reported that in patients with hyperacute posterior circulation ischemic strokes, the volume of lesions assessed by DWI was correlated with the clinical outcome, irrespective of the initial neurological status. DWI was an effective initial imaging tool for assessment of the extent of lesions and clinical functional outcomes in patients with hyperacute posterior circulation ischemic stroke [17].

In contrast to this study, many studies have reported that DWI lesion volume did not correlate significantly with the NIHSS score and hence large lesions or multiple lesions may have lower NIHSS scores than a single small lacunar lesion. For example, a relatively large infarct in the occipital cortex might only cause a hemianopia, whereas small (<1 cm^3) pontine or midbrain infarction can cause severe deficits. Therefore, although NIHSS represents an easy-to-administer and widely validated scale, it seems more useful in patients with anterior circulation stroke than posterior circulation stroke [26].

Previous studies had different points of view and reported that infarct volume and stroke severity, but not infarct location, were associated with poor long-term outcomes in first-ever posterior fossa strokes [27]. So that the greater volume of infarction on DWI and severe neurological deficits the more predilection for poor and unfavorable functional outcome. The only exception was in the case of pontine infarcts, which tended to be associated with poorer long-term outcome compared with infarcts in other posterior fossa regions [28].

Comparison of MRA data of the three groups showed that the presence of intracranial stenosis was not significantly different among the study groups. Intracranial stenosis played a role in posterior circulation stroke etiology by one of three mechanisms (hypoperfusion, artery-to-artery embolism, and plaque extension over small penetrating artery ostia) [29]. This could point to the fact that intracranial stenosis played a role in the etiology of different patterns of posterior circulation infarctions.

Similar findings were reported by previous studies and showed that severe intracranial atherosclerotic disease may be a potential mechanism for ischemic lacunar infarcts in the absence of small artery disease by extension over small penetrating artery ostia and these non-Small-artery disease (SAD) lacunar strokes had a worse clinical outcome [30]. Also, other studies showed that the presence of vertebrobasilar intracranial stenosis was associated with recurrent TIAs and multiple microembolic signals that can lead to acute multiple posterior circulation ischemia and infarctions [8].

In terms of stroke etiology according to the TOAST classification, the comparison between the three groups showed that large artery atherosclerosis was the most common stroke etiology in the patients in group II (80.0% of the patients in group II) and the patients in group III (80.0% of the patients in group III). Previous studies have supported that large artery atherosclerotic disease was frequently associated with large size of infarction and an acute multiple brain infarction pattern on diffusion-weighted imaging in the posterior circulation [31].

Cardioembolic stroke was present only in three patients in group II (15.0% of the patients in group II) and among two patients of group III (10.0% of the patients in group III). Some previous studies have supported that AF might be an etiology for large territorial and multiple posterior circulation infarcts [32].

The laboratory results of the study groups showed that the significant statistical differences between the three groups were the platelet count, RBS on admission, and ESR.

The platelet count was significantly higher among the patients in group I compared with the patients in group II and group III. Previous studies showed that elevated platelet count increases the risk for ischemic stroke [33]. The pathology of lacunar infarctions is mainly thrombus formation in the small vessels. As the thrombus formation depends mainly on platelets, this finding seems logical.

In terms of the RBS, it was observed that the patients in group II had the highest mean of RBS on admission, followed by the patients in group III, followed by the patients in group I. Similar studies supported these results and found that elevated blood glucose levels induce anaerobic metabolism, lactic acidosis, and free radical production, which in turn result in disruption of BBB, and hence larger area of infarction [34].

For ESR, it was observed that the patients in group III had the highest mean of ESR at 1 h, followed by the patients in group II, followed by the patients in group I. Previous studies had shown similar results and reported that the increase in ESR correlates with early brain damage in acute ischemic stroke [35]. A possible explanation for this finding might be the presence of multiple areas of infarction that predispose to more brain damage and hence more elevation of ESR.

The strengths of this study were the correlation between the clinical criteria and the investigation modalities; MRI, MRA, and different risk factors in different patterns of infarctions in posterior circulation for better prognostic evaluation.

The limitations of this study were the relatively small sample size and the relatively short duration of follow-up.

Conclusion

Different vascular risk factors such as hypertension, diabetes, dyslipidemia, and smoking are present in all infarction patterns of posterior cerebral circulation, either single or multiple infarctions, and there were no significant differences in the presence of these vascular risk factors in relation to the type of the vascular lesion. AF and significant vertebrobasilar stenosis were mostly associated with large and multiple infarct lesion patterns. Small-vessel disease was the most common stroke

etiology for a single small lacunar lesion, whereas large artery atherosclerosis was mostly present in a single large lesion and multiple lesions. Strict control of vascular risk factors is highly recommended in all infarction patterns of posterior circulation.

Recommendations

(1) MRI and MRA in the acute phase should be performed, especially if posterior ischemic circulation stroke is suspected, because these modalities have high sensitivity for identifying small and hyperacute ischemic lesions.
(2) Transfer of patients who are at risk of deterioration in the acute phase of posterior circulation ischemic stroke to a specialized neurological center should be considered.
(3) Strict control of the vascular risk factors such as hypertension, diabetes, and dyslipidemia is highly recommended to improve functional outcome.
(4) Transthoracic echocardiography should be performed routinely, especially for patients with a single large lesion or multiple lesions, to search for possible cardiac sources of emboli such as AF. Transesophageal echocardiography is a more sensitive test. It is more effective than transthoracic echocardiography in identifying valvular degenerative lesions, aortic arch atheroma, and interatrial shunts. Patent foramen ovale and any abnormal anatomy associated with it (such as atrial septal aneurysms) are detected more frequently by transesophageal echocardiography.
(5) Carotid duplex and MRA should be performed routinely in all patients with acute posterior circulation ischemic stroke to detect any probable vertebrobasilar stenosis.
(6) If a patient is a candidate for thrombolytic therapy, brain and vessel imaging with a computed tomography angiography is essential to identify basilar artery occlusion. It should be performed without delay because minimizing the time between stroke onset and the start of thrombolysis is associated with a good outcome. Computed tomography angiography is more readily available in the acute phase than MRI, and is useful if MRI is contraindicated or unavailable. Computed tomography angiography in comparison with MRA had high sensitivity for identifying vessel occlusion, 100 and 87% sensitivity, respectively.
(7) Patients with high NIHSS and mRS scores on admission, high serum levels of RBS on admission, intracranial stenosis, large volume of infarction, and large artery atherosclerosis stroke, and cardioembolic stroke should be monitored carefully in the hospital because of the high incidence of unfavorable outcome and mortality.
(8) Stroke severity can be judged clinically on the basis of the degree of neurologic impairment (e.g. disturbance in conscious level, language, behavior impairment, visual field deficit, motor deficit), and the size and location of the infarction on neuroimaging with MRI or computed tomography. Factors affecting the functional outcome include location of infarctions, mechanism of ischemic stroke, associated comorbid conditions, and stroke complications such as DVT and chest infections from prolonged admission at hospital.

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Conflicts of interest

There are no conflicts of interest.

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