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Original Article

Khorasan Posterior Circulation Stroke Registry: a Hospital-Based Study

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Abstract

Background: Clinical information about stroke in the vertebrobasilar territory has lagged behind that for anterior circulation syndrome. This is the first report from posterior circulation syndrome registry in Iran.

Methods: Consecutive patients with brain infarction in vertebrobasilar territory admitted to Ghaem hospital, Mashhad were enrolled in a prospective study during 2006-2007. Diagnosis of ischemic stroke in the posterior circulation was made by a stroke neurologist based on the clinical manifestations and neuroimaging. Vertebrobasilar territory infarcts were classified into five groups according to the location involved: brainstem, thalamus, cerebellum, posterior cerebral artery, and mixed categories. All of the stroke patients underwent a standard battery of diagnostic investigations and the etiology of ischemic stroke was determined by the Practical Iranian Criteria classification. The 72-hour stroke course determined as regressive, stable, and deteriorative.

Results: Total of 302 patients (147 females, 155 males) with mean age 62.5 years (±17.2) were investigated. Posterior cerebral artery, thalamus, brain stem, cerebellum, and mixed categories consisted 31.3%, 4.3%, 32.8%, 17.9%, and 13.9% of the stroke topographies respectively. Atherosclerosis consisted 50.6% of etiologies in our patients followed by uncertain (25.5%), cardioembolism (12.5%), both atherosclerosis and cardioembolism (6.3%), and miscellaneous causes (4.6%). Rheumatic mitral stenosis was the cause in 34.2% of our patients with cardiac emboly. The distribution of stroke etiologies based on its localization was not significantly different (df=16, and P=0.421). Stable status was the most common early stroke course (57.7%) followed by deteriorative (22.1%), and regressive (20.2%). A significant association between stroke localization in the vertebrobasilar territory and its course was not found (df=8, and P=0.901).

Conclusion: Atherosclerosis is the most common cause of posterior circulation syndrome in Iranian patients. The cause of stroke in the posterior circulation could not reliably be derived from infarct topography.

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Keywords ● Stroke ● vertebrobasilar ● posterior circulation

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Introduction

Vertebrobasilar territory nourishes one-fifth of the brain. Posterior circulation syndromes could be due to hemorrhagic or ischemic cerebrovascular pathologies. Hypertensive intracerebral hemorrhage is the most common cause of hemorrhagic posterior circulation syndrome, which is usually localized in the pons and cerebellum.[1] Etiologies of ischemic posterior circulation syndromes is similar to the carotid territory infarctions.[2]

Clinical information about the management of patients with posterior circulation ischemia has lagged behind that for anterior circulation ischemia.[1,2] Posterior circulation syndrome often has been attributed to hemodynamically significant vertebrobasilar artery stenosis leading to low flow ischemia or penetrating artery disease, whereas anterior circulation ischemia most often is attributed to embolism from the heart or extracranial internal carotid arteries.[1,2] Patients with carotid territory ischemia usually undergo cardiac and internal carotid artery evaluation, whereas patients with vertebrobasilar territory ischemia infrequently undergo extensive cardiac or vascular ultrasound evaluations.[3] Because of these different clinical practices, much more is known about anterior circulation than posterior circulation diseases.[3]

Clinical differentiation of posterior and anterior circulation syndromes is sometimes difficult. Turkish Posterior Circulation Stroke Registry (TPCSR) is the largest reported series of patients (1296 patients) with vertebrobasilar territory ischemia to date.[4] Khorasan Stroke Registery (KSR) is the only Iranian stroke registry that compares anterior and posterior circulation infarctions based on the age, gender, etiology, and small or large vessel territory involvement.[5] Khorasan Posterior Circulation Stroke Registry (KPCSR**)** is the first posterior circulation syndrome registry in Iran, which deals with clinical course and etiology of stroke based on the different topographies of the vertebrobasilar territory.

Patients and Methods

Consecutive patients with ischemic posterior circulation syndrome admitted to Ghaem hospital, Mashhad, north of Iran, during 2006-2007 were enrolled in a prospective study. Stroke was defined as an ischemic focal neurological deficit that persisted at least 24 hours.[6] Transient ischemic attack (TIA) was determined by taking history from the patients or their relatives. TIA was defined as an acute unilateral blindness or focal cerebral deficit related to ischemia lasting less than 24 hours.[6]

Patients died before admission or evaluation by stroke neurologist, were excluded from the study. A signed informed consent was obtained from the patients or their first degree relatives. The patients who refused the consents were excluded from the study. Patients with head trauma, primary intracerebral hemorrhage, and subarachnoid hemorrhage were excluded as well.

The diagnosis and etiologic investigations of stroke were made by a stroke neurologist. The 72-hour stroke course determined as regressive, stable, and deteriorative. For all the patients with ischemic stroke at least one control brain computerized tomography (CT) was requested 48 hours after the stroke. Magnetic resonance imaging (MRI) of the brain was performed in patients who had unreliable CT for localization of the stroke in the posterior circulation. Brain MRI was also requested for patients with clinially definite vertebrobasilar territory stroke and non-diagnostic brain CT. Magnetic resonance angiography were also performed in patients suspicious of having arterial dissection, arteriovenous malformation, or aneurysm. These differential levels of neuroimaging assessment are the standard protocol in diagnostic work up of patients with stroke.[4,7,8]

The territory of infarct was determined by topographic maps of cerebrovascular territories in the neuroimaging.[9] Vertebrobasilar territory infarctions were classified into five groups according to the location involved: brainstem, thalamus, cerebellum, posterior cerebral artery (PCA), and mixed categories. Secondary hemorrhagic transformation of vertebrobasilar territory brain infarctions were also evaluated. History of TIA and stroke in the patients were recorded. History of strokes in-parents and siblings which were diagnosed by a neurologist or an internist were also recorded. The presence of headache and lacunar syndromes were also evaluated in the patients. All of the patients underwent a standard battery of diagnostic investigations, including electrocardiography (ECG), complete blood count, coagulation profile, serum electrolytes, fasting blood sugar and lipid profile, as well as duplex sonography of supra-aortic trunks, transcranial doppler ultrasonography, and transthoracic echocardiography. A 24-hour Holter monitoring was performed in patients with the history of syncope and/or palpitation with non-diagnostic ECG. Transesophageal echocardiography was performed in whom transthoracic echocardiography was non-diagnostic despite high suspicion of cardioembolism.

Three serial blood cultures were requested for any stroke patient with fever and heart murmur or valvular vegetation detected by

echocardiography. Cardiac enzymes were measured if the history or electrocardiographic evidence of recent myocardial infarction was present. An extended coagulation profile (antithrombin III, protein C, protein S) was requested in young adult patients without identifiable cause of stroke, who had personal or family history of venous thrombosis and in the patients with multiple unexplained stroke and abnormalities on routine screening coagulation tests. Antinuclear and anticardiolipine antibodies were checked in cryptogenic stroke patients with personal or family history of venous thrombosis, recurrent miscarriage, thrombocytopenia, cardiac valve vegetations, livedo reticularis or raised sedimentation rate. The sedimentation rate was requested in patients with suspected vasculitis. Hypertension was defined as using antihypertensive medications or detecting two blood pressure values (at least 1 week apart) >140/90 mm/Hg. Receiving antidiabetic medications or a fasting blood glucose>6.4 mmol/l or >126 mg/dl were considered as diabetes mellitus. Hypercholestrolemia assumed as using lipid lowering medications or fasting serum cholestrole level> 5.2 mmol/l or >200 mg/dl. Fasting blood sugar and lipid profile were part of the routine investigations performed within the first 48 hours of stroke. Patients who smoked more than 5 cigaretts per day in the recent year were defined as smoker. Migraine induced stroke was defined based on the revised criteria developed by the International Headache Society.[10] Diagnosis of the stroke etiologies was made using Practical Iranian Criteria for classification of brain infarction (table 1).[7,11] Data on patients demographics, clinical presentations, course, and results of investigations were kept in KPCSR data bank. SPSS software version 11.5 was used for analysis the data. The Pearson X^2, t, and Avona tests were used for statistical analyses and $P<0.05$ was considered as significant.

Results

Total of 302 patients (147 female, 155 male; mean age 62.5; SD: 17.2) with ischemic stroke in the posterior circulation were investigated during a 3-year period. The mean age of women and men with ischemic stroke was 60.28; SD=15.21 *v* 65.12; SD=13.49; t=1.91, P=0.059. 14 patients died before admission or evaluation by stroke neurologist. All of these patients were residents of Khorasan province in northeaster Iran.

Right sided stroke was found in 50.1% of our patients and 41.9% of the patients had left sided stroke. Bilateral localization of brain infarction was found in 8% of the patients. Topographic localization of brain infarction in the vertebrobasilar territory was determined by MRI in 58% of the patients. PCA, thalamus, brain stem, cerebellum, and mixed categories consisted 31.1%, 4.3%, 32.8%, 17.9%, and 13.9% of the stroke topographies in the posterior circulation respectively. Table 2 demonstrates the details of stroke topography in our 42 patients with mixed localization. Although 65.2% of cerebellar infarctions occurred in men, the effect of gender on distributions of infarct localization was not significant (X^2=1.756, df=1, P=0.65). Differences in distribution of stroke localization in the age groups were not significant (F=1.587, df=9-118, P=0.127). Headache was present in 32.8%

Table 1: The Practical Iranian Criteria (PIC) for Classification of Brain Infarction

Etiologic Classification:

I -Atherosclerosis Grade 1: A and /or B

A: at least two of the following risk factors

aged ≥ 60 years, hypertension, diabetes mellitus, smoking, hyperlipidemia

B: < 50 % stenosis of the corresponding large intracranial artery, < 70 % stenosis of the corresponding extracranial artery, aortic arch atheroma >4 mm without mobile component

I -Atherosclerosis Grade 2:

≥ 50 % stenosis of the corresponding large intracranial artery, ≥ 70 % stenosis of the corresponding extracranial artery, aortic arch atheroma with mobile component

II -Cardioembolism Grade 1:

Right to left heart shunt with deep vein thrombosis (DVT) or right heart thrombus, bioprosthetic mitral or aortic valve, mitral valve prolapse with mitral regurgitation, sever mitral regurgitation, left ventricular aneurysm after acute myocardial infarction (MI), left ventricular akinetic segment after acute MI

II -Cardioembolism Grade 2:

Atrial fibrillation (AF), mechanical mitral or aortic valve, acute MI < 4 weeks, left heart thrombus, bacterial and non-bacterial endocarditis, congestive heart failure, dilative cardiomyopathy, rheumatic mitral stenosis, atrial myxoma

III -Miscellaneous Grade 1:

Hypercoagulability, migraine induced stroke, fibromuscular dysplasia *, aneurysmal sac*

III -Miscellaneous Grade 2:

Arterial dissection*, moyamoya syndrome*, arteriovenous malformation*, vasculitis*, cerebral venous thrombosis*

IV -Mixed: any combination of the above etiologies

V -Undetermined: none of the above causes could be determined by complete diagnostic investigation

VI -incomplete diagnostic investigation

*compatible with stroke manifestations

Table 2: Details of stroke localization in 42 patients with mixed topography.

Localization	Number	Percentage
PCA+cerebellum	1	2.4%
PCA+brain stem	9	21.4%
PCA+thalamus	17	40.4%
Cerebellum +thalamus	2	4.8%
Brain stem+thalamus	6	14.3%
PCA+thalamus+cerebellum	2	4.8%
PCA+brain stem+cerebellum	3	7.1%
PCA+brain stem+ cerebellum+thalamus	2	4.8%
Total	42	100%

PCA: Posterior cerebral artery

(n=99) of our patients and 6.3% (n=19) of them had lacunar syndromes. History of stroke and TIA and family history of stroke were found in 20.1% (n=61), 4.6% (n=14) and 11.6% (n=35) of our patients respectively. Hemorrhagic transformation of the brain infarction was found in 4.6% of our patients and half of the hemorrhagic transformations occurred in the PCA territory infarctions (n=14). Hypertension, hyperlipidemia, diabetes mellitus, and smoking were found in 22.5% (n=68), 7.9% (n=24), 3.9% (n=12), and 4.6% (n=14) of the patients in isolation, and 37.4% (n=113) of our patients had multiple risk factors. The effect of gender in distribution of risk factors was not significant (X^2=14.368, df=18, P=0.705). Of the patients with posterior circulation stroke, 28.1% (n=85) had no risk factor. Differences in frequency rate of risk factors among various stroke localizations were not significant (X^2=183.10, df=162, P=0.123). Presence of multiple risk factors was the most common probability in brain stem, cerebellum, thalamus and mixed localizations. Atherosclerosis consisted 50.6% (n=153) of etiologies in our patients followed by uncertain 25.5 % (n=77), cardioembolism 12.5% (n=38), both atherosclerosis and cardioembolism 6.3% (n=19), and miscellaneous causes 4.6% (n=14). Atherosclerotic stenosis was found in 42 (10.6%) patients. V1, V2, V3, V4, Basilar, and PCA stenosis were found in 26, 1, 1, 8, 4, and 2 patients respectively. Atherosclerosis was the most common etiology in the age groups 60-69 and 70-79 years. Coexistence of cardioembolism and atherosclerosis was found in 50% of the patients aged 80-89 years. Uncertain etiology consisted 38% of stroke subtype in patients younger than 50 years. Migraine induced stroke consisted 33.3% of our miscellaneous etiologies followed by dural sinus thrombosis 33.3% and extracranial vertebral artery dissection (22.2%).

The distribution of stroke etiologies in the age groups was significantly different (F=4.569, df=4-123, P=0.002). The effect of gender in frequency rate of stroke etiologies was not significant (X^2=5.159, df=4, P=0.271). Table 3 compares the frequency rate of stroke etiologies based on its localization in our study group. The distribution of stroke etiology based on its localization was not significantly different (X^2=16.458, df=16, P=0.421). Atherosclerosis was the most common etiology in all localization of stroke in the posterior circulation. Atrial fibrillation was present in 50% (n=19) of patients with cardioembolic mechanism in KPCSR. KPCSR revealed that 34.2% (n=13) of the patients with cardioembolism had rheumatic mitral stenosis. rheumatic mitral stenosis was found in 26.3% (n=5) of the patients with atrial fibrillation.

Stable status consisted the most common early stroke course (57.6%, n=174) followed by deterioration (22.2%, n=67) and regression (20.2%, n=61). Table 4 represents frequency rate of early course of stroke based on its topographic localization. The effect of gender on the frequency rate of stroke course was not significant (X^2=4.222, df=2, P=0.121). Of the patients in deteriorative state, 43.3% (n=29) had atherosclerotic etiology and 35.8% (n=24) of them had uncertain cause of stroke. The distribution of course subtypes in the age

Table 3: Frequency rate of stroke etiologies based on its localization in the KPCSR

Etiology/Localization	PCA	Thalamus	Brain stem	Cerebellum	Mixed
Atherosclerosis	52-55.3%	8-61.5%	40-40.4%	28-51.8%	25-59.5%
Cardioembolism	14-14.9%	1-7.7%	12-12.1%	6-11.2%	5-11.9%
Atherosclerosis+Cardioembolism	4-4.2%	1-7.7%	8-8.1%	4-7.4%	2-4.8%
Uncertain	20-21.4%	2-15.4%	36-36.4%	14-25.9%	6-14.3%
Miscellaneous	4-4.2%	1-7.7%	3-3%	2-3.7%	4-9.5%
Total	94-100%	13-100%	99-100%	54-100%	42-100%

PCA: Posterior cerebral artery

Table 4: Frequency rate of early course of stroke based on its localization in the KPCSR

Course/Localization	PCA	Thalamus	Cerebellum	Brain stem	Mixed
Stable status	58-61.7%	7-53.8%	33-61.1%	55-55.6%	21-50%
Regressive	20-21.3%	3-23.1%	9-16.7%	20-20.2%	9-21.5%
Deterioration	16-17%	3-23.1%	12-22.2%	24-24.2%	12-28.5%
Total	94-100%	13-100%	54-100%	99-100%	42-100%

PCA: Posterior cerebral artery

groups was not significantly different (F=0.385, df=2-125, P=0.081). The distribution of course subtypes was not significantly different based on of the risk factors (X^2= 33.133, df=36, P=0.606). The distribution of stroke course based on its etiologies was not significantly different (X^2=5.558, df=8, P=0.697). Mortality of our patients with posterior circulation stroke within the first week after the stroke was 10.9% (n=33). And 51.5% of the deceased patients had brain stem involvement. Atherosclerosis consisted 63.6% of etiologies in the deceased patients (n=21) followed by uncertain etiology, 15.2% (n=5).

Discussion

The KSR revealed that vertebrobasilar territory infarctions are seen in 17% of women and 20% of men with brain infarction.[5,7] Similar gender distribution was found in the KPCSR. The most common site of stroke in KPCSR was brain stem followed by PCA, cerebellum, and mixed topographies. Brain stem was the most common site of stroke (59%) in the posterior circulation followed by cerebellum (47%), and mixed topographies (16%) in the Laussane Stroke Registry (LSR).[12] Other topographic studies have shown that brain stem and cerebellum are the most common sites for posterior circulation infarctions.[13] Topographic classification of New England Medical Center-Posterior Circulation Registry (NEMC-PCR) includes proximal, middle, and distal intracranial posterior circulation territories.[14] TPCSR topographic classification is similar to the NEMC-PCR and categorizes: 1-proximal territory infarctions including the medulla and posterior inferior cerebellar artery territory, 2-middle territory infarctions including the pons and anterior inferior cerebellar artery territory, 3-distal territory infarctions including mesencephalon, thalamus, medial temporal, occipital lobes, and superior cerebellar artery territory.[4,14] Topographic classification of KPCSR is similar to the Besancon Stroke Registry and is not categorized as proximal, middle, and distal vertebrobasilar territories.[7]

The effect of gender on frequency rate of stroke etiologies was not significant in KPCSR. Distribution of stroke with cardioembolic mechanism was not significantly different based on the gender in KSR.[5,7] Women were significantly more susceptible to stroke with atherosclerotic mechanism and miscellaneous etiology, while men were significantly more preponderant for stroke with uncertain causes in KSR.[5,7] Atherosclerosis was the etiology of stroke in 50.6% of our patients in KPCSR. KSR confirmed that distribution of stroke with atherosclerotic and cardioembolic mechanisms was not significantly different between carotid versus vertebrobasilar territory involvement in the Iranian stroke patients,[5,7] while stroke with uncertain causes was significantly more frequent in vertebrobasilar territory involvement.[5,7] TPCSR reported large artery disease (39%), small artery disease (24%), and cardioembolism (20%) as the most frequent etiologies of posterior circulation stroke in Turkish population.[4] Etiologic classification of KPCSR is not based on the large or small artery territory involvement.[5,7,11] Since various etiologies can lead to brain infarction in small vessel as well as large vessel territories,[15,16] etiologic classification of the KSR and KPCSR includes all vascular territories. Atherosclerotic etiology in KSR and KPCSR includes small and large artery diseases.[5,7,11] Two-thirds of the patients with posterior circulation brain infarction in LSR had atherosclerotic stenosis or occlusion of vertebrobasilar arteries and 20% of them had cardioembolic mechanism which co-existed with large-artery disease in one-third of these patients.[12] The distribution of stroke etiology based on its localization was not significantly different in KPCSR. In KPCSR, 51.8% of cerebellar infarctions were due to atherosclerosis. Cardioembolic mechanism was found in 67% of isolated cerebellar infarctions in the LSR.[12] However, a Japanese study on patients with multiple cerebellar infarctions has shown that 60% of these patients had atherosclerotic mechanism and 20% had cardioembolism.[17] In TPCSR, 52% of reported cerebellar infarctions were due to large artery atherosclerotic disease and co-existing large artery disease. Cardioembolic mechanisms were present in 17% of these patients.[18] 55.3% of our patients with infarction in PCA territory had atherosclerotic mechanism. A German multi-center study reported cardioembolism as the most common cause of PCA territory infarction.[19] Evaluation of infarctions in PCA territory in Düsseldorf showed cardioembolism, large artery disease, uncertain, and miscellaneous etiologies in 31%, 30%, 24% and 5% of the patients respectively.[20]

Embolic mechanism in NEMC-PCR included cardioembolism and artery-to-artery embolism which was often due to atherosclerosis.[14] PCA infarctions with or without thalamic involvement were mostly caused by cardiac origin and intra-arterial embolism in NEMC-PCR.[14] Atherosclerosis constituted 40.4% of etiologies in brain stem infarctions in the KPCSR. Large artery atherosclerotic disease has been reported as the most common etiology of brain stem infarctions in the TPCSR,[4] and NEMC-PCR.[14] 61.5% of our patients with thalamic infarctions had atherosclerotic etiology. Isolated thalamic infarctions in

the NEMC-PCR were most often attributable to penetrating artery disease.[14] Most of the infarctions with mixed topography in KPCSR were caused by atherosclerosis. A Portuguese stroke registry reported that multiple vertebrobasilar territory infarctions from a first ever stroke event was frequently due to cardioembolic etiology, although this was not statistically significant.[21]

Rheumatic valvular disease is an important cause of cardioembolism in Iran and other developing countries.[22-24] Rheumatic mitral stenosis consisted 35.7% of cardioembolic mechanisms in KPCSR. Rheumatic mitral valve disease was found in 44.8% of the patients with cardioembolic mechanism and in 63.5% of stroke patients with atrial fibrillation in KSR.[5,7] Rheumatic mitral valve disease comprised 32% of ischemic stroke etiology and 59.7% of cardioembolic strokes in Iranian young adults.[25] In-hospital mortality of KPCSR is similar to what reported in KSR. However early deteriorative course is more common in KPCSR than KSR.[5,7] In-hospital mortality of vertebrobasilar territory infarctions is reported up to 25%.[13] The death rate of TPCSR and NEMC-PCR was reported 7% and 2.3%, respectively.[26,27] Mortality in the NEMC-PCR was the highest among the patients who had cardioembolism and atherosclerotic artery-to-artery embolism.[28] Atherosclerosis consisted most of the etiologies in the deceased patients followed by uncertain etiology in KPCSR. Mixed territory infarctions were more probable to have deteriorative course than other localizations in KPCSR. Multiple posterior circulation infarctions and cardiac embolism were reported as predictors of poor outcome.[29,30]

Conclusion

Ischemic posterior circulation syndromes do not differ much from the anterior circulation syndromes in risk factors and etiology. The cause of stroke in the posterior circulation could not reliably be derived from infarction topography. The course of the brain infarction in vertebrobasilar territory is not related to its etiology and localization.

Conflict of Interest: None declared

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