 

ORIGINAL ARTICLE

# CLINICAL AND IMAGING FEATURES OF LACUNAR AND NON-LACUNAR SUBTYPES OF ISCHEMIC POSTERIOR CIRCULATION STROKE

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## ABSTRACT

**The aim:** The purpose of this study is to determine clinical and imaging features of etiological subtypes of posterior circulation stroke in a prospective hospital-based cohort study.
**Materials and methods:** We prospectively recruited 120 acute posterior circulation stroke patients, admitted to the Neurological Center of the University Hospital (Oleksandrivska Clinical Hospital) in Kyiv, Ukraine, within 6 to 24 hours from the onset of the stroke symptoms. Comprehensive neurological, clinical, laboratory, ultrasound, and imaging examination was performed on all patients.
**Results:** MRI/CT-proven etiological subtypes of ischemic posterior circulation stroke were defined - atherothrombotic (n = 59), cardioembolic (n = 24), lacunar (n = 27), and definitively indeterminate (n = 7). Two main study groups were formed - lacunar (n = 27) and non-lacunar (n = 90) subtypes of posterior circulation stroke.
**Conclusions:** Specific clinical and imaging features of etiological subtypes of posterior circulation stroke were determined, analyzed, compared, and described.

**KEY WORDS:** stroke, imaging, etiological subtype, lacunar, non-lacunar, posterior circulation stroke.

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## INTRODUCTION

Over 165,000 strokes a year are misdiagnosed in U.S. emergency departments [1, 2]. Globally, over 26% of strokes are misdiagnosed, making a stroke the fourth most common misdiagnosis among the major medical diagnostic errors [3, 4]. Posterior strokes are twice more likely to be misdiagnosed compared to anterior circulation strokes [5].

Posterior circulation stroke (PCS), caused by infarction within the vertebrobasilar arterial system, is a potentially life-threatening condition that is misdiagnosed 30-60% of the time [6-8]. Failure to rapidly diagnose stroke delays time-sensitive treatments, resulting in higher risks of disability and mortality [1, 9-17].

Early etiological treatment minimizes brain damage in stroke patients [18-23]. For that, etiological subtypes of PCS should be promptly recognized and diagnosed. Otherwise, patients with delayed diagnosis may do worse due to a stroke progression, brainstem compression from posterior fossa edema, or recurrent stroke. That is why it is critical to promptly determine clinical and imaging features of etiological subtypes of PCS to prevent its severe consequences promptly.

## THE AIM

We aimed to determine clinical and imaging features of lacunar and non-lacunar subtypes of ischemic posterior circulation stroke in a prospective hospital-based cohort study.

## MATERIALS AND METHODS

### STUDY SETTING AND PATIENTS

We have conducted a prospective, hospital-based, cohort study of acute PCS patients. All study participants were admitted to the Neurological Center of the University Hospital (Oleksandrivska Clinical Hospital, Kyiv, Ukraine), within the first 6 to 24 h since the first stroke symptoms occurred.

The Neurological Center of Oleksandrivska Clinical Hospital consists of an admission department, clinical department of neurology, department of cerebrovascular pathology with intensive care/stroke unit, and a research department of neurology. The Hospital represents the largest tertiary care center in Kyiv. It has a catchment population of approximately two million. Healthcare is provided free of charge to all citizens and registered long-term residents. Institutional ethics board approval was obtained and written informed consent was received from all participants or legally authorized representatives for this study.

Study subjects were recruited from the hospital's emergency departments and in-hospital wards between 2011 and 2020. All stroke patients were reviewed by at least two board-certified neurologists with training in cerebrovascular diseases. Clinical history, 12-lead electrocardiogram,

blood testing, carotid ultrasound (Toshiba, Japan), head CT and/or brain MRI (Siemens, USA, 1.5 T) were obtained for all study participants.

The size of the PCS foci was determined by direct measurement, taking into account the magnification of the tomogram. The section with the largest foci size was chosen and the measurement of the foci was determined by the formula of the wrong ellipsoid:

$V = 0{,}52 \cdot A \cdot D \cdot C$,

Where V is the volume of the lesion, A, B, C is its diameters, and 0.52 is the coefficient for calculating the wrong ellipsoid.

## PATIENT INCLUSION AND EXCLUSION

The methods of the study, inclusion and exclusion criteria have been reported in detail previously [7, 24, 25]. In brief, only acute PCS patients aged 18 years or older were included.

## STUDY ENDPOINTS AND RISK FACTORS DEFINITIONS

Study endpoints of interest were acute ischemic PCS. Stroke and TIA were defined according to the criteria of the World Health Organization, AHA/ASA guidelines for adult stroke, and was confirmed by imaging [26, 27]. The etiology of stroke was classified according to the TOAST criteria [28]. The National Institutes of Health Stroke Scale (NIHSS), the Modified Rankin Scale (mRS), the Barthel index (BI), and the Charlson Comorbidity Index (CCI) were determined for all participants. Secondary stroke prevention was prescribed according to the American Heart Association/American Stroke Association and the European Stroke Organization (ESO) Guidelines, immediately after the stroke diagnosis was made [29-33]. Stroke education programs were provided to all study participants [34-38].

## STATISTICAL ANALYSIS

Parametric and non-parametric statistic methods were applied. The log-rank test was used for univariate comparisons of event-free survival between groups. A two-sided $p<0.05$ was considered significant for all analyses. All statistical analyses were performed using IBM SPSS Statistics, Version 24.

# RESULTS

## BASIC CHARACTERISTICS OF THE STUDY POPULATION

In total, 120 adult patients (68 men, 52 women aged 28 to 89 years; average age 60.7 ± 12.1 years) with an acute ischemic MRI/CT-proven PCS were screened. The breakdown for study group by stroke type was as follows:

- 10.9% (n=13) of patients were diagnosed with acute midbrain infarctions;
- 18.3% (n=22) of patients diagnosed with acute medulla oblongata infarctions;
- 18.3% (n=22) of patients diagnosed with acute thalamic infarctions;
- 20.8% (n=25) of patients were diagnosed with acute cerebellar infarctions;
- 31.7% (n=38) of patients have a proven diagnosis of acute pons infarctions.

There was no statistically significant difference between patients with infarcts of the proximal, middle, and distal territory of the posterior circulation by the main demo-

**Table I.** Comparative characteristics of patients by basic demographic indicators and vascular risk factors depending on the location of the acute posterior circulation stroke

| Indicator | Localization of the brain infarction | | | | |
|---|---|---|---|---|---|
| | Midbrain (n=13) | Medula Oblongata (n=22) | Thalamus (n=22) | Cerebellum (n=25) | Pons (n=38) |
| Men | 5(38,5%) | 12(54,5%) | 12(54,5%) | 16(64,0%) | 23(60,5%) |
| Women | 8(61,5%) | 10(45,5%) | 10(45,5%) | 9(36,0%) | 15(39,5%) |
| Age, years | 56,8±9,1 | 61,3±10,4 | 61,9±10,2 | 57,9±14,7 | 62,5±12,2 |
| Vascular risk factors | 10(76,9%) | 18(81,8%) | 15(68,2%) | 18(72,0%) | 28(73,7%) |
| Hypertension + atherosclerosis | 10(76,9%) | 15(68,2%) | 16(72,7%) | 16(64,0%) | 30(78,9%) |
| Ischemic heart disease | 1(7,7%) | 3(13,6%) | 2(9,1%) | 3(12,0%) | 6(16,8%) |
| Myocardial infarction history | 3(23,1%) | 5(22,7%) | 2(9,1%) | 7(28,0%) | 8(21,1%) |
| Atrial fibrillation | 2(13,4%) | 4(18,2%) | 3(13,6%) | 4(16,0%) | 13(34,2%) |
| Diabetes mellitus | 2(13,4%) | 3(13,6%) | 4(18,2%) | 4(16,0%) | 6(15,8%) |
| Excessive weight | 3(23,1%) | 7(31,8%) | 7(31,8%) | 9(36,0%) | 14(36,8%) |
| Smoking | 1(7,7%) | 2(9,1%) | 3(13,6%) | 3(12,0%) | 5(13,2%) |
| Transient ischemic attack history | 5(38,5%) | 12(54,5%) | 12(54,5%) | 16(64,0%) | 23(60,5%) |

**Table II**. Distribution of patients by etiological subtype of ischemic infarction depending on the affected intracranial anatomical area of the posterior circular basin

| Etiological Subtype of Ischemic Stroke | Localization of the brain infarction | | | | |
|---|---|---|---|---|---|
| | Midbrain (n=13) | Medula Oblongata (n=22) | Thalamus (n=22) | Cerebellum (n=25) | Pons (n=38) |
| Atherothrombotic (n = 59) | 9(69,2%) | 14(63,6%) | 11(50,0%) | 11(44,0%) | 18(47,4%) |
| Cardioembolic (n = 24) | 3(23,1%) | 6(27,3%) | 4(18,2%) | 7(28,0%) | 4(10,5%) |
| Lacunar (n = 27) | - | - | 7(31,8%) | 5(20,0%) | 15(39,5%) |
| Definitely Indeterminate (n = 7) | 1(7,7%) | 2(9,1%) | - | 2(8,0%) | 1(2,6%) |

graphic parameters and vascular risk factors (Table I).

In 49.2% of patients, PCS was accompanied by multiple lesions in different parts of the brain, such as cerebellar hemispheres, thalamus, occipital and temporal lobes of cerebral hemispheres.

Unilateral cerebellar infarctions in the area of vascularization of the superior cerebellar artery (n = 4) and posterior inferior cerebellar artery (n = 1) caused the development of ischemic foci in the cortex and white matter of the frontal and frontoparietal lobes of the contralateral hemisphere of the brain - *crossed cerebellar hemispheric diaschisis* [39-42]. At the same time, the infarctions of the upper and middle parts of the pons (n = 3) were accompanied by *crossed pontine-cerebellar diaschisis* with the development of ischemic foci on the territory of vascularization of the contralateral cerebral hemisphere [43-45].

## ETIOLOGICAL SUBTYPES OF POSTERIOR CIRCULATION STROKE

Based on the clinical, imaging, ultrasound data, laboratory methods, and taking into account the TOAST criteria we identified four etiological subtypes of ischemic PCS [28]. They were as follows:

- atherothrombotic (n = 59),
- cardioembolic (n = 24),
- lacunar (n = 27),
- and definitively indeterminate (n = 7).

Based on the etiological subtype and location of PCS patients distributed differently (Table II).

Consequently, according to the TOAST criteria, we distinguish lacunar (n = 27) and non-lacunar (n = 90) subtypes of brainstem and PCS infarctions. Patients with arterial stenosis >50% ipsilateral to a PCS and/or source of cardioembolism were classified as a non-lacunar infarction.

In accordant with our data, lacunar PCS (LPCS) was localized mostly in the area of the pons (n = 15), thalamus (n = 7), and cerebellum (n = 5) but were not detected in the medulla oblongata or midbrain. They arose as a result of the defeat of a separate paramedian branch of the basilar artery, one of the perforating branches of the thalamogenicular artery, or a medial or lateral branch of a superior cerebellar artery. LPCSs were caused by thrombotic microangiopathies in patients with arterial hypertension, diabetes mellitus, and in the absence of sources of cardioembolism and stenosis of the large vertebrobasilar arteries.

The clinical characteristics of lacunar syndromes, verified by imaging methods, were different. Most often foci of LPCS were found in the area of pons (n = 15), thalamus (n = 7), and less often in the area of the cerebellum (n = 5). *Pure motor stroke*, also known as pure motor hemiparesis or pure motor syndrome, occurred in 11 patients, *ataxic hemiparesis* - in 3 patients, *dysarthria-clumsy hand syndrome* - in one patient with the stroke lesion in the area of the pons. *Pure sensory stroke* was detected in 5 patients (thalamic lesions), 2 more patients were diagnosed with *mixed sensorimotor stroke*; in this case, the stroke accordingly spreads towards the inner capsule.

Non-lacunar infarcts were formed due to the defeat of short and/or long bypass (circular) arteries (vertebral artery and basilar artery) in the presence of sources of cardioembolism and the absence of stenosis of the large vertebrobasilar arteries (n = 90). They also arose as a result of occlusive lesions of large arteries (vertebral and basilar arteries) in the extra- or intracranial departments, ie they were caused by macroangiopathies (n = 59).

## DISCUSSION

Along with the presence of typical neurological syndromes, the atypical clinical course is observed in patients with PCS, which makes it difficult to determine the nature of stroke, its location, and the choice of adequate therapy [46, 47]. Consequently, the majority of PCSs may not be accurately classified only by symptoms or signs. Misdiagnosis, which commonly occurs in the initial phase of patient evaluation, leads to erroneous clinical decision-making [2, 5, 9, 12].

PCS can cause unilateral or bilateral deficits and is more likely to affect consciousness, especially when the basilar artery is involved [7, 24]. The presence of arteries of different caliber, structure, anastomotic potential, and blood supply zones determines the variety of locations, size, and clinical course of PCS [8]. At the same time, individual differences in the location of arteries and a variety

of etiological mechanisms determine the individual characteristics of clinical and imaging features of acute PCS. In such a clinical situation, of course, only brain imaging techniques are helpful.

According to our data, the pontine infarcts were found most often (31.7% of patients), while in the midbrain three times less often (10.9%). Infarcts in the medulla oblongata, thalamus, and cerebellum were found with almost equal frequency. In many subjects (49.2%) PCS were accompanied by acute multiply lesions in other parts of the brain fed with posterior circulation blood supply.

## LACUNAR SUBTYPE OF POSTERIOR CIRCULATION STROKE

The word "lacuna" (French lacune - lake, or lacunar - cavity) entered medicine after Durand-Fardel (1842), and later Pierre Marie (1901) found in the brain of the elderly a large number of small cavities and for the first time described the clinical course of lacunar stroke [48, 49]. The manifestations of lacunar stroke were described in more detail by SM Fisher (1982) [50].

### *DEFINITION*

Lacunar stroke is a special form of acute or slowly progressing ischemic circulatory brain disorder. It is caused by primary lesions in the areas of blood supply by perforated arteries and the development of small foci of necrosis in the deep brain in patients with arterial hypertension. Lacunar strokes foci are small in size (0.5-1.0 cm) cavities of round or irregular shape - gaps.

### *INCIDENCE AND RISK FACTORS*

The incidence of LPCS ranges from 16 to 35% of all cases of ischemic strokes [51, 52]. In the United States and Western Europe, lacunar strokes account for 15-25% of all ischemic strokes. Recurrent LPCS forms the lacunar state of the brain, i.e. the morphological substrate of hypertensive encephalopathy.

The leading risk factor for the development of LPCS is arterial hypertension, which is often complicated by diabetes mellitus [53]. Occlusions of small penetrating arteries, if there is no cardioembolism, can be caused by micro thrombosis, microatheroma, local spasm, or a combination of these factors of varying degrees of expression.

### *MORPHOLOGY*

According to the morphology, LPCS is a kind of white stroke. Inside and around formed LPCS, there are foci of incomplete necrosis of white and gray matter, axonal dystrophy, Waller's degeneration of white matter fibers, glial cell proliferation, and edema. Lacunar stroke is mostly localized in the deep parts of its hemispheres, subcortical nodes, thalamus, inner capsule, basal parts of the brain, white matter, and nuclei of the cerebellum.

### *CLINICAL PRESENTATION*

More than 25 brain syndromes of lacunar stroke have been described in the literature. In clinical practice, the four most common syndromes, described by SM Fisher (1965), are recognized: *pure motor stroke, pure sensor stroke, ataxic hemiparesis, and dysarthria-clumsy hand syndrome* [54].

*Pure motor stroke*, or pure motor hemiplegia, is the most common. The lacunas are mostly localized in the area of the pons. Patients have a complete motor syndrome or paresis of the arm and leg, paresis of the lower facial muscles. This group does not include syndromes that are characterized by isolated weakness of the arm or leg as these cases are considered incomplete stroke in the pool of the middle or anterior cerebral arteries.

*Pure sensory stroke*, or pure hemianesthesia, occurs in 20% of lacunar stroke cases. Sensory syndrome develops when the affected posterior ventral nucleus of the thalamus is the main sensitive (sensory) nucleus. Hemisensory syndrome is complete in the case of decreased superficial and/or deep sensitivity or numbness of the skin by hemitype in the absence of homonymous hemianopsia, aphasia, agnosia, and apraxia. Sensitivity disorders can be manifested by dysesthesia, hyperpathy, and pain. In the case of incomplete hemisensory syndrome, sensitive disorders registered not on the whole half of the body, but only on the face, arm, or leg: *cheiro-oral* syndrome is detected when the sensitivity disorder occurs in the corner of the mouth and palm homolaterally; *cheiro-pedo-oral* syndrome manifested by hypoalgesia in the corner of the mouth, palms, and feet on the one hand without motor disorders. If stroke forms in the thalamus and spreads to the inner capsule, - sensorimotor syndrome develops.

*Ataxic hemiparesis* forms in the case of stroke localization in the basal parts of the pons. Neurological findings, in this case, include moderate leg weakness, mainly in the distal parts with minimal signs of hand paresis, and hemiataxia. Stroke in the upper cerebellum causes ataxic disorders.

*Dysarthria-clumsy-hand syndrome* accounts for about 6% of all lacunar stroke cases. It develops due to the localization of LPCS's foci mainly in the basal parts of the pons. Neurological symptoms include speech disorders such as dysarthria in combination with contralateral dysmetria of the arm and leg. Weakness in the limbs and muscles of the face is also possible. Dysarthria occurs when the basal parts of the pons are affected, more often on the left (100%) than on the right (73%) side of the pons [55].

A *hyperkinetic syndrome* is caused by the development of LPCS in the thalamus and subthalamic nucleus. Neurological deficit includes hemichorea, hemibalism, and dystonic disorders.

## NON-LACUNAR SUBTYPE OF POSTERIOR CIRCULATION STROKE

Patients with arterial stenosis >50% ipsilateral to an LPCS and/or forces of cardioembolism, and/or undefined etiology were classified as patients with non-lacunar infarctions according to the TOAST criteria. These were patients with

large-artery atherosclerosis, cardioembolism, PCS of other determined etiology, and undetermined etiology [56, 57].

An *atherothrombotic* subtype of infarction was diagnosed in 59 patients. This variant of the PCS subtype was mostly caused by the atherosclerotic process of extra- and/or intracranial arteries, causing their stenosis, obstruction, thrombosis, ie macroangiopathy. In the majority of cases (69.5%), this subtype of PCS occurred during the patient's sleep or immediately after it. The clinical course was characterized by a subacute, gradual undulating increase in neurological deficits during the first hours occurred.

A *cardioembolic* subtype of PCS (n = 24) usually occurs suddenly, without any predictors, during a patient's physical activity, often accompanied by the loss of consciousness. Clinical course ranged from moderate to significant cerebral symptoms. The main ethnological cause was a permanent or paroxysmal form of atrial fibrillation, myocardial infarction, and dilated cardiomyopathy [20, 23, 28].

*Ultimately undefined* subtype of PCS was diagnosed in 7 patients. This subtype of PCS manifested as a vascular insufficiency and a disruption of the autoregulatory response of cerebral circulation. It occurred due to a rapid decrease in blood pressure or decreased cardiac output due to acute myocardial ischemia.

## CONCLUSIONS

Clinical and imaging examinations of the patient give the ability to verify the lesion and the corresponding arterial area involved in the pathological process of stroke of various anatomical parts of the brainstem and the basin of the posterior circulation.

PCS is most often caused by microangiopathy, macroangiopathy, and cardioembolic mechanisms. The presence of arteries of different caliber in the posterior circular basin, with different anastomotic potential and different areas of blood supply in the case of acute ischemic strokes determine the heterogeneity of the neurological clinic.

Knowledge of the features of the neurological clinical course of PCS is important for the medical doctor, helping to diagnose the PCS promptly, timely, and choose adequate methods of therapy and assessment of long-term functional prognosis.

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**Conflict of interest:**
*The Authors declare no conflict of interest.*


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