Features of lacunar pontine stroke: results of a prospective hospital-based cohort study

Abstract. Background. Little to no new data are published on the prospective hospital-based cohort studies that report and analyze clinical and imaging features of lacunar pontine infarction. This study aims to determine clinical and imaging features of lacunar pontine infarction at an early stage in a prospective hospital-based cohort study.

Materials and methods. We prospectively recruited 120 patients with acute MRI-positive posterior circulation stroke who were admitted to the Neurological Center of the University Hospital. Patients with pontine stroke were enrolled in the study within 6 hours from the onset of stroke symptoms. Comprehensive neurological, clinical, laboratory, ultrasound, and imaging examinations were performed for all participants. Results. Out of 120 adult patients aged 28 to 89 years, 15 (12.5 %) were diagnosed with lacunar pontine infarction and formed a study group. We provided a comprehensive clinical, neurological, laboratory, and instrumental analysis of lacunar pontine infarction. Conclusions. Specific clinical and imaging features of lacunar pontine infarction were determined, analyzed, and described.

Keywords: stroke; posterior circulation stroke; pons; lacunar pontine infarction; imaging

Introduction
Pontine stroke is the most common ischemic stroke of the brainstem [1–3]. Isolated pontine infarctions are classified as either paramedian or lacunar pontine infarctions (LPI) [4–6]. It is widely accepted that paramedian pontine infarction is caused by the occlusion of basilar perforating branches, whereas LPI is caused by small vessel disease [7].

Clinical presentation of pontine stroke ranges from the classical crossed syndromes (such as Millard-Gubler, Foville, and Raymond-Cestan) to the less common pure motor, pure sensory stroke, or respiratory and cardiac dysfunction [8–12]. Early diagnosis and adequate understanding of the clinical presentation of LPI are essential for evaluating and managing the disease [13–15].

The purpose: to determine clinical and imaging features of lacunar pontine infarction at an early stage in a prospective hospital-based cohort study.

Materials and methods
The study settings, patterns, definitions, inclusion, and exclusion criteria have been reported in detail previously [16–24]. Briefly, 120 patients with MRI-positive acute posterior circulation stroke were consecutively selected. All of them were admitted to the Neurological center of the University Hospital, Oleksandrivska Clinical Hospital, between 2011 and 2020. The Hospital represents the largest tertiary care center in the capital of Ukraine, Kyiv.

Results
Among 120 consecutively selected patients with MRI-positive acute posterior circulation stroke, 38 were diagnosed with pontine infarctions. Of them, 15 patients, admitted within 6 hours after onset, were diagnosed with LPI and formed a study group. All 15 MRI-positive patients with LPI had a history of hypertension, and 40.0 % of them had diabetes. All LPI were isolated.
Most often, in 8 (53.3 %) cases, a pure motor LPI was detected, caused by damage to the pyramidal pathways in the area of the base of the pons. Pronounced hemiparesis in the acute period of a stroke was observed in one patient, moderate in two, and mild in five. Equal by the strength, weakness in the arm and leg was presented in three cases; more pronounced weakness in the arm was detected in five patients. Dysarthria was moderate in three cases and mild in two, central facial palsy was found in two patients. Pure motor hemiparesis was not accompanied by any impairment of speech, sensitivity, vision, brain stem function, hearing loss, tinnitus, diplopia, and gross nystagmus.

According to the MRI results, brain lesions in LPI patients were more often localized in the caudal part of the pons (n = 4). LPI was located in the middle part of the pons in two patients, and another two patients had LPI in the rostral part of the pons. All lesions were below 1 cm³. For illustration, we present brain MRI of patient K. (Fig. 1).

Pure sensory pontine strokes occurred in two (13.3 %) patients. In one person, a complete hemisensory syndrome was observed. It manifested by the loss of all types of sensitivity by the conductive type and was due to damage to the dorsal part of the pons. Another patient was diagnosed with incomplete hemisensory (chiro-oral-pedal) syndrome in the presence of clinical symptoms of hypalgesia in the angle of the mouth, palm, and foot on one side without motor impairment.

Ataxic hemiparesis was diagnosed in four (26.7 %) patients. The typical localization of LPI lesion, in this case, was the rostral part of the pons, closer to the midline. The neurological status showed hemiataxia, moderate weakness of the leg, and slight paresis of the hand.

Dysarthria-clumsy hand pontine syndrome was detected in one (7 %) patient. The syndrome of dysarthria and clumsy hand was detected, which was accompanied by dysarthria and pronounced dysmetria of the arm and leg. The LPI lesion was localized in the paramedian area of the basal parts of the pons.

The neurological deficit in patients with LPI by the National Institutes of Health Stroke Scale (NIHSS) and B. Hoffenberth scale was 6.3 ± 1.1 points and 8.7 ± 1.2 points, respectively. LPI patients' reliable recovery according to the NIHSS occurred on the 7 th day and by the scale of B. Hoffenberth, on the 14 th day (0—1 points on the modified Rankin scale (mRS)).

**Discussion**

Lacunar stroke is an ischemic infarction of less than 15 mm in diameter located in the territory of the cerebral penetration arteriole [25–27]. Lacunar infarctions most commonly occur in the lentilacleral nucleus, thalamus, frontal lobe white matter, pons, basal ganglia, internal capsule, and caudate nucleus and are caused by occlusion of the deep perforating blood vessels [28]. Small vessel disease is most commonly associated with hypertension and diabetes [29, 30].

The most common causes of LPI are lesions of the basal artery branches, long and short bypass arteries (43 %), of small paramedian arteries (34 %), and less often — occlusion of the basal artery (21 %) [31]. LPI is caused by lesions of the corticospinal, corticocuneal, corticopontine, and pontocerebellar tracts [16, 32]. Patients with LPI have a history of diabetes mellitus more often (50 %) compared to those with anterior circulation strokes [33]. Diabetes and arterial hypertension cause damage to the vessels of the microcirculatory channel (arteries and arterioles), the development of microangiopathy, and therefore LPI or microcirculatory pontine infarctions [34].

The pyramidal pathway at different levels of the pons has its own characteristics, and therefore the severity of the paresis depends on the level of pontine damage: movement disorders are more significant if an ischemic lesion affects the caudal part of the pons, less significant — the paramedian ventral area, and the rostral part of the pons, where the fibers of the pyramidal pathway pass diffusely.

Clinical presentation of LPI includes pure motor, pure sensory, and dysarthria-clumsy hand pontine syndromes [35]. Pure motor pontine stroke with hemiparesis or hemiplegia accounts for 10.2 % of all primary ischemic strokes and prevails among other pontine lacunar infarctions [36]. Pure sensory pontine strokes occur in case of ischemic lesions in the dorsal part of the pons [37]. Because the medial loop and the spinothalamic pathways are compatible here, the lesions lead to a disturbance of superficial and proprioceptive sensitivity. Often such patients complain of dysesthesia. Dysarthria-clumsy hand pontine syndrome occurs in case of lacunar infarction development in the basal parts of the pons and is accompanied by dysarthria and severe dysmetria of the arm and leg. It is believed that dysarthria is more common in patients with lesions of the left half of the pons [38]. In all LPI cases, the neurological deficit might be transient, lasting from seconds to 24 hours, causing transient ischemic attack [33, 39–41]. Effective secondary stroke pre-
vention and early rehabilitation programs should be applied to all LPI patients to improve their quality of life and prevent disability and mortality [42–50].

A comparison of the neurological and functional recovery showed that on the 21st day a favorable outcome (0–2 points) was achieved in all LPI patients: in 4 patients, there was a complete recovery of neurological functions (0 points by the mRS), in the other 11, insignificant neurological microsymptoms persisted, which did not affect daily household activities (1 point on the mRS), and the recovery of neurological functions occurred within the first 7–14 days. Knowledge of the features of LPI is important, helping to promptly diagnose, select and apply adequate therapy and increase long-term functional prognosis.

Conclusions

The clinical course of LPI is extremely important when making a decision about a patient’s diagnosis, management, and prognosis. We provided a comprehensive narrative review of the clinical features of LPI. Specific clinical and imaging features of LPI were determined, analyzed, compared, and described. LPI must be promptly diagnosed and treated to avoid high morbidity and mortality associated with it.

Prospects for further research. Future studies are needed on a larger number of patients to determine early clinical and imaging features of LPI, to promptly increase diagnosing and treatment of such patients to avoid high morbidity and mortality. It is also important to promote awareness of stroke prevention programs among patients and medical personnel.

References


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Особливості лакунарного інсульту варолієвого моста: результати госпітального проспективного когортного дослідження

Резюме. Вступ. Нових даних про проспективні госпітальні когортні дослідження, у яких проаналізовано клінічні та візуалізаційні ознаки лакунарного інфаркту моста, майже не опубліковано. Мета: визначити клінічні та нейровізуалізаційні особливості лакунарного інфаркту варолієвого моста на ранній стадії в госпітальному проспективному когортному дослідженні. Матеріали та методи. Ми проспективно відібрали 120 пацієнтів, госпіталізованих до неврологічного центру із гострим інсультом у вертебробазилярному басейні, підтвердженим магнітно-резонансною томографією. Особи з інсультом моста були включені в дослідження протягом 6 годин від розвитку симптомів інсульту. Усім пацієнтам проведено комплексне неврологічне, клінічне, лабораторне, ультразвукове та візуалізаційне обстеження. Результати. Із 120 пацієнтів віком від 28 до 89 років у 15 (12,5 %) був діагностований лакунарний інфаркт моста. Проведено комплексне клінічне, неврологічне, лабораторне та інструментальне дослідження особливостей лакунарного інфаркту моста. Висновки. Визначено, проаналізовано та описано специфічні клінічні та візуалізаційні ознаки раннього лакунарного інфаркту моста. Ключові слова: інсульт; вертебробазилярний басейн; міст; лакунарний інфаркт моста; нейровізуалізація.