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Features of non-lacunar pontine stroke: results of a prospective hospital-based cohort study and a report of a series of clinical cases in white European adults

Abstract. ***Background.*** *Pontine strokes are catastrophic. However, no new data are published on the prospective hospital-based cohort studies that report and analyze clinical and imaging features of non-lacunar pontine stroke. This study aims to determine clinical and imaging features of non-lacunar pontine stroke in a prospective hospital-based cohort study, illustrated with a series of clinical cases presented in white European adults.* ***Materials and methods.*** *We prospectively recruited 120 acute MRI-positive posterior circulation stroke patients, admitted to the Neurological Center of the University Hospital. Complex neurological, clinical, laboratory, ultrasound, and imaging examinations were performed for all patients.* ***Results.*** *Out of 120 adult patients (aged 28 to 89 years), 23 (19,2 %) patients were diagnosed with non-lacunar pontine infarction, forming a study group. We provided a complex clinical, neurological, laboratory, and instrumental analysis of non-lacunar pontine infarction.* ***Conclusions.*** *We provided a comprehensive narrative review and analysis of the clinical features of non-lacunar pontine stroke.*
Keywords: *stroke; posterior circulation stroke; pons; non-lacunar pontine infarction; tegmental; bilateral; medial; lateral; imaging; clinical case.*

Introduction

The pons is the largest component of the brain stem [1, 2]. Pontine stroke (PS) is catastrophic and the most common ischemic stroke of the brainstem [3–5]. PS has complex symptoms that often differ from the hallmark symptoms of a stroke, ranging from the classical crossed syndromes to severe respiratory and cardiac dysfunction [6–8]. Early diagnosis and adequate understanding of the clinical presentation of PS are essential for evaluating and managing the disease [9–11].

The purpose: to determine clinical and imaging features of non-lacunar pontine stroke in a prospective hospital-based cohort study.

Materials and methods

The work is a part of the posterior circulation stroke study. Its settings, parents, definitions, inclusion, and exclusion criteria have been reported in detail previously [12–21]. Briefly, in 120 consecutive selected MRI-positive patients with acute posterior circulation stroke. All patients were admitted to the Neurologica 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 and discussion

Among 120 consecutive selected MRI-positive patients with acute posterior circulation stroke, 38 patients were diagnosed with PS. Among them, 23 (19,2 %) patients, admitted within 6 hours after onset, were diagnosed with non-lacunar pontine stroke (NLPS), forming a study group. In the study group, four (17.4 %) patients had isolated (predominant ventral-medial and tegmental-medial infarcts) PS and 19 (82.6 %) had combined infarcts with existing ischemic foci in various structures of the brain or in other anatomical and

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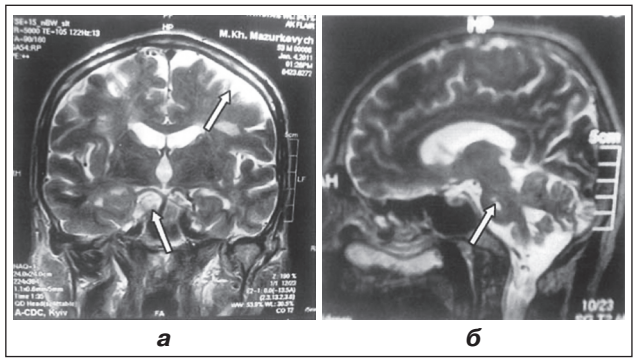

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Figure 1. Magnetic resonance imaging of the brain of patient M., 53 years old, T2-weighted image, performed 18 hours after the onset of the disease. Findings: in the right upper part of the pons, a weakly intense center of infarction (arrow) and in the subcortical parts of the frontal lobes of the cerebral hemisphere — an area of ischemia, measuring 18 × 7 × 8 mm (arrow)

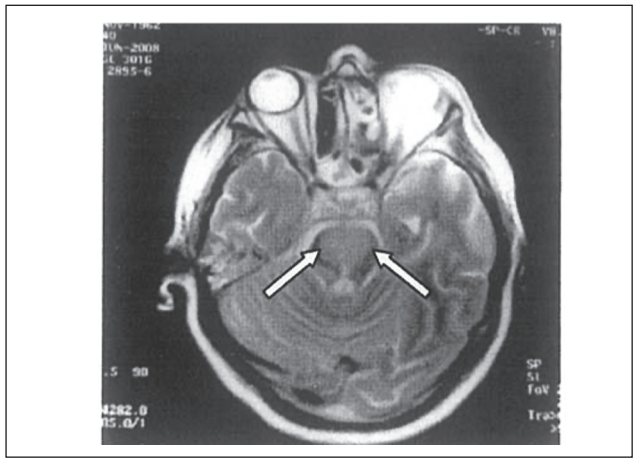

Figure 2. Magnetic resonance imaging of the brain of patient B., 67 years old, T2-weighted image, axial projection, performed 8 hours after the onset of the disease. In the area of the base of the pons, 2 extensive areas of ischemia are visualized (arrows)

vascular territories: 6 (26.1 %) in the cerebellar hemisphere, 4 (17.4 %) in the peduncles, 3 (13.1%) in the medulla oblongata, 2 (8.7 %) in the thalamus, 1 (4.3 %) in temporal, 2 (8.7 %) in occipital, and 1 (4.3 %) in frontal lobes of the brain.

In 4 (17.4 %) patients, the clinical picture corresponded to a ventral-medial infarction of the pons Varolii, in 8 (34.8 %) cases to a medial tegmental infarction, in 5 (21.7 %) patients to a bilateral medial infarction, in 2 (8.7 %) — to a lateral infarction, 4 (17.4 %) patients has total pontine infarction. The average size of the pontine lesion was 3.4 cm^3.

A ***ventral medial pontine infarction*** clinically manifested by dysarthria, dysphonia, and motor hemiparesis. Contralateral hemihypesthesia of pain and temperature sensitivity was detected in only 1 patient. As an example of a ventral-medial pontine infarction, we present a clinical case.

Clinical Case

Patient M., 53 years old, medical history No. 3231591, was presented and hospitalized at the Neurological Center of the University Hospital with complaints of difficulty speaking and general weakness. The speech impairment arose during the hypertensive crisis with the increase of the patient's blood pressure to 220/100 mm Hg after consuming alcohol. The patient has had a history of alcoholism and hypertension for the past 5 years and has not systematically taken antihypertensive therapy.

Objectively: pulse — 78 beats in min, rhythmic; blood pressure — 150/80 mm Hg.

In the neurological status: Severe dysarthria. Strongly positive Bekhterev's oral reflex (++), and bilateral Marinescu-Radovich symptoms. Hyperreflexia of tendon and periosteal reflexes on hands and knees S > D, Achilles — the right is not evoked, the left is reduced, and the Babinski reflex is positive on the left.

Scales at admission: NIHSS — 4 points, corresponding to a mild stroke; B. Hoffenberth — 12 points, corresponding to moderate impairment.

Ultrasound of the carotid revealed hemodynamically marked atherosclerotic stenoses of both carotid arteries (left ICA (LVCA) — 40–50 %, right ICA (PVSA) — up to 50 %).

Brain MRI is presented in Figure 1.

Diagnosis: ventral medial infarction of the pons, combined with an ischemic lesion in the left frontal lobe of the brain in a patient with hypertensive and atherosclerotic encephalopathy of the III degree.

Tegmental medial pontine infarction manifested by damage to VI and VII pairs of cranial nerves, motor hemiparesis, hemihypesthesia, and complete or incomplete alternating Foville syndrome.

Three patients have complete Foville syndrome with peripheral paresis of facial muscles and external eye muscle on the side of the lesion combined with contralateral hemiparesis of the limbs and anesthesia of all types of sensitivity on the opposite side. Such a clinical picture arose as a result of damage to the paramedian arteries with the localization of the ischemic lesion mainly in the dorsal part of the lower part of the pons.

Four patients had incomplete alternating Foville syndrome, manifested by ipsilateral paresis of the abductor eye muscle and contralateral motor and conductive sensory disorders. In this case, the center of ischemia was visualized mainly in the dorsal part of the upper part of the pons. The recovery of the motor functions of the limbs, in these patients, was better than the restoration of the range of motion of the eyeball.

One patient had Raymond-Sestan syndrome, manifested by paresis of gaze toward the stroke lesion, accompanied by hemiparesis, hemihypesthesia, and hemiataxia on the opposite of the stroke side.

Bilateral medial pontine infarctions occurred due to damage to the ventral part of the pons and were clinically manifested by pseudobulbar syndrome, tetraparesis due to damage to the pyramidal tracts, and impairment of surface and proprioceptive sensitivity. For illustration, we present a clinical case.

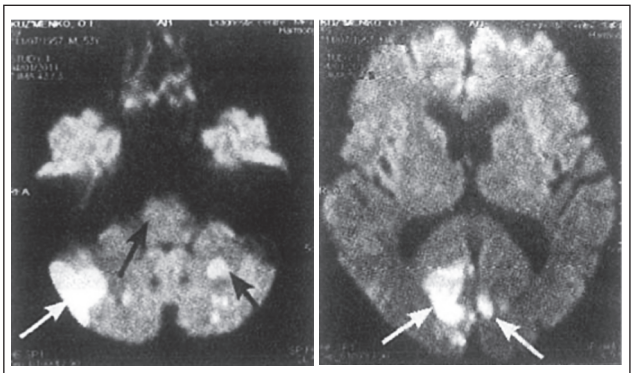

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Figure 3. Magnetic resonance imaging of the brain of patient K., 53 years old, T2-weighted image (axial projection). The study was conducted 5 days after the onset of the disease. A hyperintense lesions are: in the lower part of the pons on the right (d = 2.2 cm); in the right occipital area (2.6 × 4.6 × 2.1 cm); left occipital area (0.7 × 1.1 × 0.7 cm); in the projection of the hippocampus on the right (d = 0.8 cm); in the right and left hemispheres of the cerebellum (d = 1.8 and 0.7 cm, respectively) (arrows)

Clinical Case

Patient B., a female 67 years old, medical history No. 3226901, was hospitalized in the department of cerebrovascular pathology at the University Hospital in a serious condition. She suddenly developed weakness in all limbs and loss of consciousness. Two years prior to the hospitalization, she suffered a myocardial infarction. The patient has had a history of hypertension for the past ten years and has been taking antihypertensive therapy on a daily basis.

Objectively: consciousness is disturbed to a sopor state. Pulse — 74 beats/min, rhythmic, blood pressure — 175/80 mm Hg. Body temperature slightly elevated to 36.8 °C.

Meningeal signs were negative. Pupils were symmetrical, the tongue in the mouth with no deviation. Pharyngeal reflexes are reduced. Tetraparesis with low limbs' muscle tone. Tendon and periosteal reflexes from the limbs are depressed. Babinski's symptom is positive bilateral. Impossible to check the sensitivity and coordination upon admission due to the severity of the patient's condition. The patient does not control the function of the pelvic organs.

Scales at admission. The assessment of neurological status according to the NIHSS scale is 15 points, according to the scale of B. Hoffenberth — 22 points, corresponding to severe neurological impairment.

Ultrasound of the carotids and vertebral arteries: hemodynamically marked atherosclerotic stenoses of both carotid arteries (LVSA — 75 %, PVSA — 80 %) and both vertebral arteries (75–80 %).

Brain MRI results are shown in Figure 2.

Diagnosis: bilateral medial ischemic pontine infarction in a patient with hypertensive and atherosclerotic encephalopathy of the III degree.

Despite intensive therapy, the patient's condition worsened after 72 hours with mSHR > 1 point. The patient died on the 7th day of hospitalization. At autopsy, two large foci of ischemia were found in the area of the pons.

Lateral pontine infarction was found much less frequently (only 2 cases) compared to medial infarction. The lateral areas of the pons are mainly vascularized by the anterior inferior cerebellar artery, upper cerebellar artery, and their perforating branches. Clinically, lateral pontine infarctions were manifested by dysarthria, dysphonia, impairment of the VII and VIII pairs of cranial nerves, sharp noise in the ear, and cerebellar ataxia. For illustration, we present a clinical observation.

Clinical Case

Patient K., male 53 years old, medical history No. 3131594, hospitalized in the department of cerebrovascular pathology with complaints of headache, difficulty speaking, loss of the left lower quadrant of the visual field, decreased sensitivity on the left half of the face, and gait problems. The sudden onset of this stroke started with an acute feeling of congestion in the right ear, which preceded the development of the abovementioned complaints. For the past two years, he has been suffering from hypertension disease but did not systematically take antihypertensive therapy.

Objectively: pulse — 76 beats/min, rhythmic; blood pressure — 140/80 mm Hg.

Meningeal signs are absent. The patient is in consciousness but productive contact is difficult due to pronounced dysarthria. Bekhterev's oral reflex is pronounced (4+). Mild enophthalmos and ptosis on the right. Pupils are symmetrical. The patient had large-scale and medium-scale horizontal and vertical nystagmus. Limited downward movements of the left eyeball. Sensitivity on the face is impaired according to the segmental dissociated type in zones B1 and B2 on the left. The patient is unable to close his right eye (eyelash symptom). The right nasolabial fold is smoothed. The tongue is in the middle line. Swallowing and phonation are intact. The pharyngeal reflex is preserved. Bilateral pyramidal insufficiency in the form of reflex tetraparesis. Indistinctly performs the finger-nose test on the left. Intentional tremor during performing the heel-knee test on the left. Hypermetria, adiadochokinesis on the left.

Scales at admission: NIHSS scale — 11 points, Hoffenberth scale — 22 points, which corresponds to severe neurological impairment.

Ultrasound of the carotids, vertebral arteries, and transcranial dopplerography: hemodynamically marked atherosclerotic stenosis of the right carotid (50 %) and unmarked atherosclerotic stenosis of the left carotid (up to 40 %). Congenital hypoplasia of the right vertebral artery. Lack of visualization of the blood flow along the basal artery and the mosaic character of the blood flow along the left vertebral artery.

The results of the patient's brain MRI are presented in Figure 3.

With applied treatment, the patient's condition improved: the severity of vertigo decreased, the sensation of congestion in the right ear regressed, and speech articulation improved. In 25 days, the patient underwent a repeat MRI of the brain and MRA (Fig. 4).

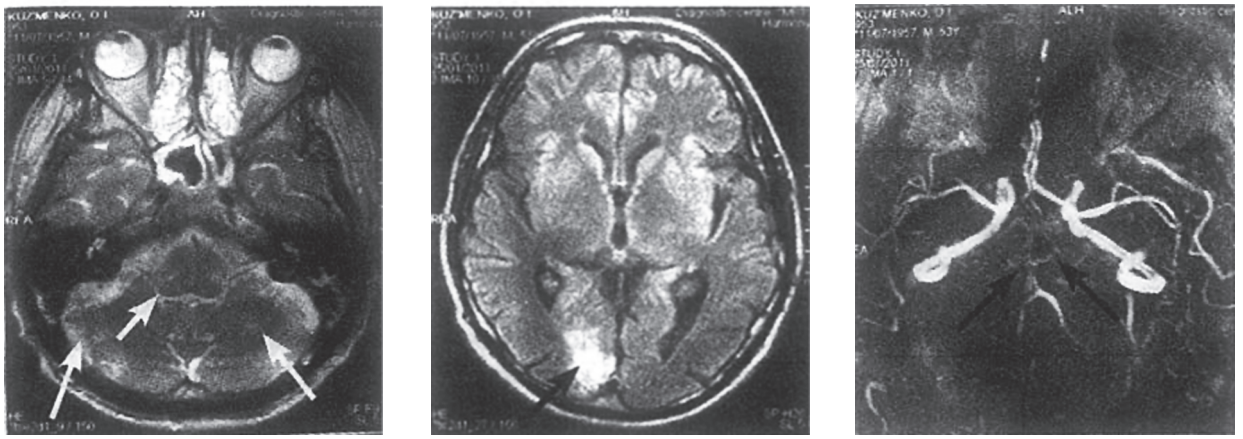

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Figure 4. Magnetic resonance imaging of the brain of patient K., 53 years old, T2-weighted image (axial projection). The study was conducted 25 days after the onset of the disease. When compared with the examination from 20 days ago, the sizes of the previously described lesion have decreased, some of them are not even visualized, and the size of the largest lesion in the right occipital region is now 2.3 × 4.1 × 1.9 cm (a, b, arrow); a sharp impoverishment of the vascular pattern of the intracranial main vessels is determined on the MR angiogram (c). Vertebral arteries are narrowed and deformed (c, arrows). The basal artery is also narrowed, its diameter is 0.25 cm (c). Only the initial sections of the posterior cerebral artery are visualized, and the vessels are not identified for the rest of the course

During the next 2 weeks, a positive neurological dynamic was observed: the function of facial muscles on the right significantly recovered, the severity of dysarthria decreased, and strength in the limbs increased.

In a month, an ultrasound of the carotids, vertebral arteries, and transcranial dopplerography was performed again: visualization of the blood flow through the basal artery improved, as a manifestation of recanalization of it.

Diagnosis: lateral pontine infarction, combined with ischemia in the occipital lobes of the brain, thalamus, hippocampus on the right, and in both hemispheres of the cerebellum, due to atherosclerotic stenosis of the basal artery.

Incomplete lateral lower pontine infarction was observed in a patient presented below.

Clinical Case

Patient E., 61 years old, was admitted to the department of cerebrovascular pathology. During a night of sleep, he suddenly developed systemic dizziness, repeated vomiting, impaired coordination, and weakness in the left limbs. In the morning, the patient developed deafness in the left ear and peripheral paralysis of the left facial muscles. Consciousness was not impaired.

Objectively: horizontal nystagmus, peripheral paresis of facial muscles on the left, deafness in the left ear. Hemihypesthesia of pain and temperature sensitivity on the right and cerebellar ataxia on the left. Tendon and periosteal reflexes from the upper limbs — S > D, from the lower limbs — D > S, positive bilateral Strümpel's symptom. Muscle tone in the limbs is impaired.

Scales at admission: the NIHSS scale is 9 points, which indicates an average degree of stroke severity; according to the Hoffenberth scale — 22 points — for severe neurological impairment.

Ultrasound of the carotids, vertebral arteries, and transcranial dopplerography: hemodynamically marked locally atherosclerotic stenosis of the left carotid — 55 % and hemodynamically marked stenoses of both vertebral arteries (75–80 %). Brain MRI data are shown in Figure 5.

Diagnosis: incomplete lateral lower infarction of pons in a patient with hypertensive and atherosclerotic encephalopathy of the III degree.

Over the next 3 weeks, the patient's nystagmus regressed, the severity of peripheral paresis of facial muscles significantly decreased, and only hearing loss in the left ear and impaired coordination of left limb movements remained.

A detailed clinical and radiological analysis showed that the center of the pontine infarction was localized in the lower lateral part, supplied with blood by the branches of the anterior infe-rior cerebella artery. Unilateral sudden hearing loss in the left ear was the result of occlusion of the internal auditory artery, which in 80 % of cases departs from the anterior inferior cerebella artery.

In 2 patients with a ***lateral infarction*** and in three patients with a ***ventral-tegmental infarction*** of the upper and middle parts of the pons, lesions were detected not only in the territory of blood supply by the small-diameter paramedian arteries of the pons, which depart from the basal artery but also in the territory of the vascularization of the contralateral superior cerebellar artery of cerebellar hemispheres. The simultaneous development of a pontine infarction and a lesion in the cerebellar hemisphere, contralateral to the primary pontine infarction, is due to a distant cross pontine-cerebellar diaschisis [22–26]. Its occurrence is explained by damage to the junction of afferent pontine-cerebellar pathways, most of which pass transversely in the upper and middle parts of the pons from its nuclei to the cortex of the

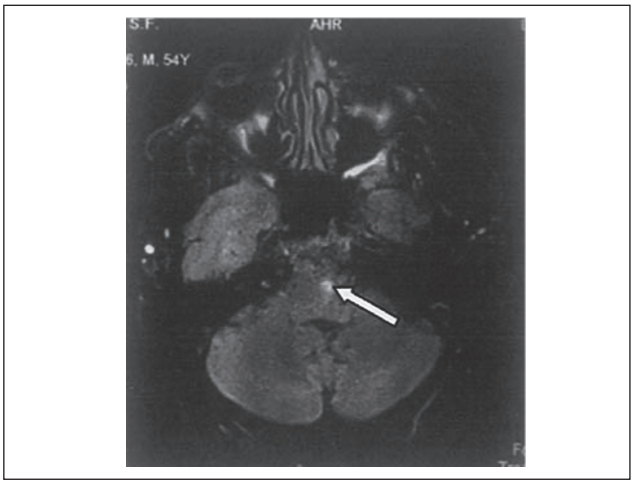

Figure 5. Magnetic resonance imaging of the brain of patient E., 61 years old, with an acute lateral infarction of the pons, T1-weighted tomogram (axial projection). The study was conducted 8 hours after the onset of the disease. In the lower part of the lateral part of the pons on the left, a hyperintense lesion (arrow), corresponding to an acute pontine infarction, is identified

contralateral cerebellar hemisphere. Therefore, in such clinical cases, neurologic symptoms were detected homolaterally and cerebellar impairment was caused by an ischemic lesion in the territory of the superior cerebellar artery vascularization of the opposite cerebellar hemisphere.

Bilateral (total) pontine infarction was characterized by a very serious condition with signs of impaired consciousness, oculomotor disorders, trismus, impaired VII pair of cranial nerves, tetraplegia, cerebellar dysfunction, and manifestations of hormetonial seizures.

Clinical Case

For illustration, we present a case of a patient M., 46 years old, who was treated in the general intensive care unit of the Hospital. The patient was delivered to the Hospital with impaired consciousness, oculomotor disorders, trismus, impaired VII pair of cranial nerves, tetraplegia, cerebellar dysfunction, manifestations of hormetonial seizures, and had a one-and-a-half syndrome, which was clinically manifested with ophthalmoplegia of the right eyeball in all directions when the left eyeball moved only outwards. A total combined pontine infarction was diagnosed.

The peculiarity of the course of this clinical case was the occurrence of transient right-sided hemiparesis, which was repeated three times during the day before the development of total pontine infarction. Such transient pontine hemiparesis M. Fisher (1988) [27] is called "hemiparesis-messenger" and, according to the author, it indicates thrombosis of the basal artery. At the same time, such neurological impairment also occurs in the case of bilateral vertebral artery damage [28], which is consistent with our observation. In a series of MRA of the patient, M. vertebral arteries were not reliably visualized (signs of loss of the MR signal).

Thus, among NLPS, bilateral medial and total infarcts manifested by impaired consciousness, pseudobulbar disorders, motor tetraparesis, and sensory dysfunction. The course of these strokes was were the most severe. Unilateral medial and lateral infarctions in NLPS patients were characterized by a more favorable clinical course and were manifested by a combination of damage to the nuclei of cranial nerves located in the pons and the central impairment of motor and sensory systems.

The neurological deficit in patients with NLPS according to the NIHSS scale was 11.7 ± 1.4 points and according to the scale of B. Hoffenberth et al. (1990) [29] — 18.2 ± 1.8 points, corresponding to the medium stroke severity. Reliable recovery of neurological functions occurred only on the 14^{th} day of observation. The neurological deficit within two weeks of treatment declined as followed: according to the NIHSS scale to 7.4 ± 1.1 points; according to the scale of B. Hoffenberth to 8.8 ± 1.9 points ($p < 0.05$).

Thus, the results of the study showed that non-lacunar pontine infarctions were mostly correlated with systemic dizziness (87 %), dysarthria (74 %), heterolateral hemiparesis (61 %), cerebellar ataxia (58 %), nystagmus (55 %), impaired sensitivity (50 %). Important are also some rare symptoms of pontine infarctions manifestation such as trismus and sharp tinnitus.

Based on the exact location of ischemia in patients with NLPS, presented symptoms were different. Thus, ventral pontine stroke mostly presented with purely motor hemiparesis due to the impairment of the pyramidal pathway. Ventral-tegmental pontine stroke predominantly manifested with ataxic hemiparesis. The following clinical manifestations were specific for the tegmental pontine stroke: purely sensory stroke due to the impairment of the medial loop and spinal-thalamic pathway with contralateral hemianesthesia of all types of sensitivity; signs of impairment of nuclei VI and VII pairs of cranial nerves; development of complete or incomplete alternating Foville syndrome. Lateral pontine infarction was characterized by the following clinical symptoms: impairment of V, VI, VII, and VIII pairs of cranial nerves; Bernard-Horner syndrome; homolateral cerebellar ataxia ("cerebellar hemiparesis" according to Marie and Fois), combined with pain and temperature anesthesia sensitivity on the opposite side. Bilateral medial NLPS and total NLPS were manifested by loss of consciousness, tetraplegia, and pseudobulbar syndrome.

Conclusions

NLPS can cause a variety of serious symptoms, including balance issues, dizziness, double vision, loss of sensation and coordination, vertigo, nausea, difficulty swallowing, weakness in one-half of the body, numbness, slurred speech, and so-called locked-in syndrome. We provided a comprehensive narrative review of the clinical features of NLPS. Knowledge of the features of NLPS is important, helping to promptly diagnose, select and apply adequate therapy and increase long-term functional prognosis. Stroke education influences the timely recognition of symptom onset and is critical in reducing the burden of a stroke worldwide.

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Особливості нелакунарного інсульту варолієвого моста: результати госпітального проспективного когортного дослідження і презентація серії клінічних випадків у білих дорослих європейців

Резюме. ***Актуальність.*** Мостові інсульти мають катастрофічні наслідки. Проте не опубліковано нових даних госпітальних проспективних когортних досліджень, у яких повідомляються й аналізуються клінічні й нейровізуалізаційні особливості нелакунарних мостових інсультів. ***Мета:*** дослідження спрямоване на визначення клінічних і нейровізуалізаційних особливостей нелакунарних мостових інсультів у госпітальному проспективному когортному дослідженні, яке проілюстровано серією клінічних випадків у дорослих білих європейців. ***Матеріали та методи.*** Ми проспективно набрали 120 пацієнтів із гострим інсультом вертебробазилярного басейну, підтвердженим магнітно-резонансною томографією, госпіталізованих до неврологічного центру університетської лікарні. Усім хворим проведено комплексне неврологічне, клінічне, лабораторне, ультразвукове й нейровізуалізаційне обстеження. ***Результати.*** Зі 120 дорослих пацієнтів (віком від 28 до 89 років) у 23 (19,2 %) було діагностовано нелакунарний інсульт моста. Проведено комплексний клінічний, неврологічний, лабораторний та інструментальний аналіз нелакунарних інсультів моста. ***Висновки.*** Ми подали всебічний огляд та аналіз клінічних особливостей нелакунарного мостового інсульту.
Ключові слова: інсульт; вертебробазилярний басейн; міст; нелакунарний інсульт моста; тегментальний; двосторонній; медіальний; бічний; візуалізація; клінічний випадок