

ORIGINAL ARTICLE

LATERAL MEDULLARY INFARCTION: A PROSPECTIVE HOSPITAL-BASED COHORT STUDY OF CLINICAL AND IMAGING FEATURES AND A CASE REPORT IN A WHITE ADULT

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ABSTRACT

The aim: The purpose of this study is to determine clinical and imaging features of lateral medullary infarction in a prospective hospital-based cohort study, illustrated with a clinical case presentation in a white adult.

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: Out of 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 posterior circulation stroke, 22 (18.3%) patients have acute medulla oblongata infarctions. We provided a complex clinical, neurological, laboratory, and instrumental analysis of lateral medullary infarction illustrated with a clinical case presentation.

Conclusions: Specific clinical and imaging features of lateral medullary infarction were determined, analyzed, compared, and described.

KEY WORDS: stroke; posterior circulation stroke; lateral medullary infarction; lateral medullary syndrome; imaging; prospective study; case report

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INTRODUCTION

Posterior circulation stroke is a life-threatening condition that is less represented in the scientific literature, is more difficult to diagnose, has a more severe clinical course, and has higher mortality compared to anterior circulation stroke [1-5]. Consequently, posterior circulation stroke is misdiagnosed 30-60% of the time [6].

The lateral medullary infarction (LMI) is the most common form of posterior ischemic stroke that is often misdiagnosed or missed. Failure to rapidly diagnose LMI delays time-sensitive treatments, resulting in higher risks of disability and mortality [7]. That is why it is critical to promptly determine clinical and imaging features of LMI to prevent its severe consequences promptly.

THE AIM

We aimed to determine clinical and imaging features of lateral medullary infarction in a prospective hospital-based cohort study, illustrated with a clinical case presentation in a white adult.

MATERIALS AND METHODS

STUDY SETTING AND PATIENTS

We have conducted a prospective, hospital-based, cohort study of acute posterior circulation stroke 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.

Oleksandrivska Clinical Hospital represents the largest tertiary care center in the capital of Ukraine, Kyiv. Started its work on November 26, 1875, as the city hospital named after Royal Tsarevich Oleksandr, it became the highest scientific and pedagogical institution with a catchment population of approximately two million [8]. There are 27 specialized departments with over a thousand beds in the Hospital. Cardiological, cardiovascular, neurological, surgical, neurosurgical, urological, otolaryngological, ophthalmological, gynecological, infectious diseases, and hemodialysis departments work around a clock, admitting patients and providing urgent care. 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. Healthcare is provided free of charge to all citizens and registered long-term residents.

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 cerebrovas-

cular 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.

PATIENT INCLUSION AND EXCLUSION

The methods of the study, inclusion and exclusion criteria have been reported in detail previously [9-11]. 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 [12, 13]. The etiology of stroke was classified according to the TOAST criteria [14]. The National Institutes of Health Stroke Scale, the Modified Rankin Scale, the Barthel index, and the Charlson Comorbidity Index were determined for all participants. Secondary stroke prevention was prescribed according to the American Heart Association/American Stroke Association and the European Stroke Organization Guidelines, immediately after the stroke diagnosis was made [15, 16]. Stroke education programs were provided to all study participants [9, 17-21].

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.

ETHICS

Institutional ethics board approval was obtained and written informed consent was received from all participants or legally authorized representatives for this study.

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.

In this article, we are focused on 22 patients diagnosed with acute medullary infarction.

ANATOMICAL FEATURES OF MEDULLARY INFARCTION

According to our data, out of 22 patients with acute medulla oblongata infarctions, LMI was identified in 15 (68.2%) patients, medial medullary infarction - in five (22.7%) subjects, combined lesions of the medial and lateral parts of the medulla oblongata were diagnosed in two (9.1%) patients. Therefore, isolated medullary infarcts were identified in 14 (63.6%) examined patients, combined - in eight (36.4%). Out of these eight patients with combined lesions, additional ischemic focus in the cerebellar hemispheres was diagnosed in six out of eight patients, in the thalamus - in one patient, and the cerebral peduncles - in another one patient [9]. In eight (53%) patients with LMI, neurogenic oropharyngeal dysphagia was detected as a component of the bulbar syndrome, which negatively affected the oral and pharyngeal phases of the swallowing process.

CLINICAL FEATURES OF LATERAL MEDULLARY INFARCTION

In the study patients, LMI most often manifested by systemic dizziness, nausea, vomiting (80%), nystagmus (80%), cerebellar ataxia on the affected side (80%), Bernard-Horner syndrome (73%) on the side of the lesion (dysfunction of the descending sympathetic fibers), impaired sensitivity on the face (60%) by the segmental dissociated type in the areas of Zelder that is located on the stroke side (due to damage to the descending tract and the spinal nuclei of V pair of cranial nerves), and hypoesthesia of pain and temperature sensitivity by the Hemi type (60%) on the opposite to the stroke side (due to dysfunction of the spinal-thalamic pathway). These neurological symptoms are part of the alternating Wallenberg syndrome.

We found the *classic alternating Wallenberg syndrome* in 9 (60%) patients with LMI, ie this syndrome was pathognomonic for this clinical form of cerebral infarction. In another 6 (40%) patients, the neurological status corresponded to the *incomplete Wallenberg syndrome*, that the most often manifested with systemic dizziness, nystagmus, cerebellar ataxia, and bulbar disorders. Such neurological symptoms occurred when the paramedian branches of the lower BA, medial or lateral branches of PICA were occluded with the following development of the small lesions (4-6 mm³) in the medulla oblongata.

IMAGING FEATURES OF LATERAL MEDULLARY INFARCTION

In study patients, MRI of the brain, performed after 12-24 hours from the beginning of the stroke, revealed isolated

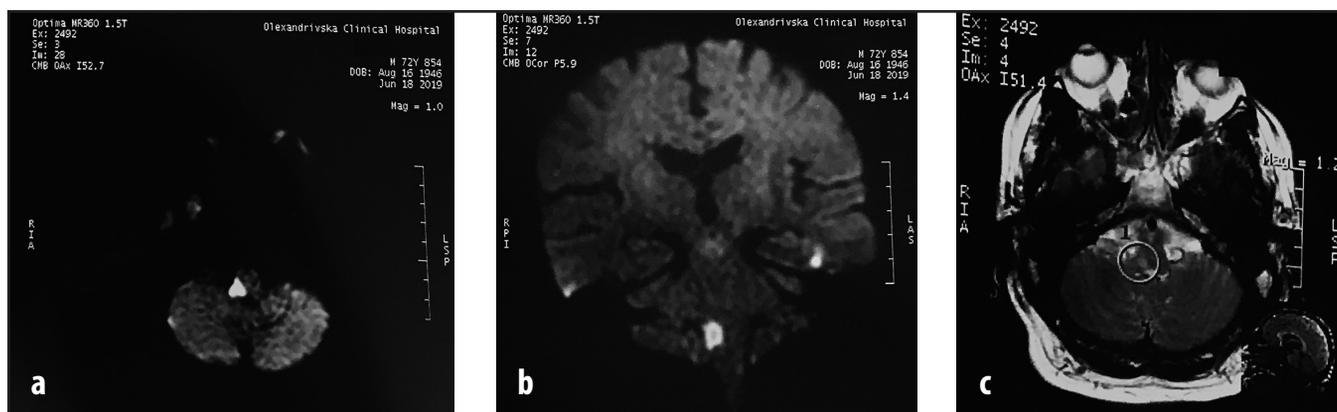


Fig. 1a,b,c. Magnetic resonance imaging of the brain of patient P., 72 years old, with acute lateral ischemic infarction of the medulla oblongata, in diffusion-weighted mode (axial projection). The MRI was done within 8 hours after the symptom's onset. The right posterolateral region of the medulla oblongata revealed hyper-intensive acute ischemia with a reduced diffusion coefficient.

lesions of the medulla oblongata in 3 patients, combined lesions of the medulla oblongata and cerebellum - in 6 patients. Most lateral medullary round infarcts were localized in the middle part of the medulla oblongata (75%), less often - in the upper and lower (25%). Their average size was 45.6 mm³. In 6 patients, the focus of ischemia was not visualized, although they had a typical neurological clinical picture of medullary lateral infarction. False-negative results, in this case, are explained by the presence of small infarct foci that are not visualized in the spatial image [22].

CLINICAL CASE PRESENTATION

To illustrate the features of a clinical course LMI, we present our clinical case observation. Patient P., 72 years old, was hospitalized in the neurology department of the University Hospital (Oleksandriivska Clinical Hospital, Kyiv, Ukraine) in June 2019, with complaints of severe systemic dizziness, nausea, repeated vomiting, impaired coordination of movements (inability to walk and stand with a tendency to fall to the right). All symptoms developed suddenly during the elevation of the blood pressure to 170/120 mm Hg. Since 2003, pt monitored for hypertension. However, antihypertensive prescriptions have not been taken systematically.

Findings: pulse - 72 beats/min, rhythmic, blood pressure - 160/80 mm Hg. Bernard-Horner syndrome on the right, horizontal nystagmus, segmental anesthesia of pain and temperature sensitivity on the right part of the face, pronounced dysphagia, dysphonia, dysarthria, stuck out tongue not deviating, decreased pharyngeal reflex. Paresis of the extremities was not detected, tendon reflexes on the hands - symmetrical, knee reflexes are S>D, Achilles reflexes are D = S, a positive symptom of Strumpel bilateral. Decreased muscle tone in the right extremities. Left-sided hemihypesthesia of pain and temperature sensitivity by the conductive type, intentional tremor when performing finger, heel-knee tests. The volume of the neurological deficit on the NIHSS scale is 9 points. The average severity of impairment on the scale of B. Hoffenberth and co-authors is 22 points (severe impairment).

Ultrasound examination of the main vessels of the head and transcranial Doppler revealed intracranial stenosis of the lumen of the right VA up to 86%, stenosis of PICA - 72%. MRI of the brain is presented in Figure 1. The neurological deficit in this clinical observation is due to the lesions of laterally localized structures of the medulla oblongata.

Diagnosis: lateral cerebral infarction with alternating Wallenberg syndrome in a patient with subcortical hypertensive and arteriosclerotic encephalopathy of III stages.

DISCUSSION

LMI causes a range of symptoms due to ischemia in the lateral part of the medulla oblongata in the brainstem. The most prevalent etiological factor is an occlusion of the intracranial portion of the vertebral artery (VA) followed by the posterior inferior cerebellar artery (PICA) and its branches [23-25]. Cerebral embolism, hypoplastic vertebral artery, or vertebral artery dissection is the commonest cause of LMI in young patients.

ANATOMICAL FEATURES OF LATERAL MEDULLARY INFARCTION

According to the anatomical topography and features of its blood supply, the medulla oblongata is referred to the proximal intracranial area of the brain stem [9]. The blood supply of this area is carried out by arterial branches, which successively depart from the intracranial VA. These branches are anterior and posterior spinal arteries, as well as PICA, and its numerous branches. The small paramedian arterial branches of VA and PICA supply blood to the lateral part of the medulla oblongata. There are lateral, medial (paramedian), and dorsal branches of the posterior circulation arteries and the corresponding areas of the medulla oblongata, which correspond to the location of the infarct in the case of acute cerebrovascular accident [26]. Accordingly, there are lateral, medial infarctions of the medulla oblongata, as well as combinations of lateral and medial infarctions [27-29].

J. S. Kim and co-authors (1999) [30] in a clinical study reported that by the localization, 75% of medulla oblongata strokes are lateral and only 25% are medial. According to other authors [31], the frequency of lateral and medial infarctions is 78 and 19%, respectively, ie, their ratio is 4:1. Dorsal medulla oblongata stroke is quite rare [2, 9].

LMI occurs in the area of its lateral parts due to occlusive lesions of small paramedian arteries and lateral branches, which depart from VA, PICA, lower divisions of the basal artery, rarely anterior inferior cerebellar artery [9]. Therefore, such concepts as the "lateral medullary syndrome" and "dorsolateral cerebral infarction" are still used as synonyms for LMI.

CLINICAL FEATURES OF LATERAL MEDULLARY INFARCTION

The clinical manifestation of LMI was first described in 1895 by the famous German neurologist Adolf Wallenberg (1862-1949), who initially identified the development of cerebral infarction of the medulla oblongata with embolic occlusion PICA [32].

C. M. Fisher and co-authors (1961) [26], based on the analysis of the arterial system of sixteen patients with LMI and 26 observations of other authors, concluded that lateral cerebral infarction in approximately 75% of cases is caused by VA's occlusion. JS. Kim and co-authors (1995) [33] during the examination of 34 patients diagnosed occlusive lesions in the area of PICA (23.5%), VA (38.2%), and simultaneous lesions of VA and PICA (26.5%). However, occlusive lesions of the arteries were not detected in 11.8% of cases.

Therefore, LMI can occur in the case of lesions of the intracranial HA, PICA, rarely BA, and PICA. Therefore, the clinical course of LMI should not be identified with the previously described PICA syndrome and its variants.

The clinical presentation of LMI is closely related to the topography of the lesion of the medulla oblongata (upper, middle, lower). This topography can be determined on sections of T2-weighted MRI, using the following criteria: the upper part of the medulla oblongata corresponds to dorsolateral protrusion at the level of the lower leg of the cerebellum; the middle part of the medulla oblongata corresponds to the ventrolateral protrusion of the lower nucleus of olive; the lower part is a rounded shape of the medulla oblongata.

W. Kameda and co-authors (2004) [31] analyzed the clinical and MRI parameters of 214 patients and showed that the most often LMIs were localized in the middle part of the medulla oblongata (35% of cases), in the upper part - in 29% of cases, and the least - in the lower part (15%).

Summarizing the data of the neurological clinical features of lateral medullary infarcts, given in publications, the representative symptoms that are characteristic for the alternating Wallenberg syndrome, known as lateral medullary syndrome and posterior inferior cerebellar artery syndrome, can be identified as follows:

- impaired sensitivity on the face (89%) by the segmental dissociated type in the areas of Zelder that is located

on the stroke side. It developed due to damage to the descending tract and the spinal nuclei of V pair of cranial nerves;

- hypoesthesia of pain and temperature sensitivity by the Hemi type on the opposite to the stroke side. It occurs due to dysfunction of the spinal-thalamic pathway;
- dysarthria, dysphonia (75%) due to paresis of the muscles of the vocal cords and soft palate, caused by the damage to the motor double nucleus (Nucl. ambiguus);
- dizziness (73%) - dysfunction of the vestibular nuclei;
- Bernard-Horner syndrome (72%) on the side of the lesion (dysfunction of the descending sympathetic fibers);
- cerebellar ataxia on the side of ischemia due to lesions of the anterior and posterior spinocerebellar pathways;
- horizontal nystagmus (57%) due to damage to the vestibular nuclei and their connections;
- dysphagia (57%) as a result of damage to the nuclei of the IX and X pairs of nerves;
- headache (47%).

In the clinical situation, described above, the pathological process (thrombosis - in 60% of cases, embolism - in 35%) is localized in HA, and not PICA, as thought previously [34].

If the LMI extends to the lateral surface of the bridge, then its typical features are accompanied by signs of muscle paresis innervated by VI and VII cranial nerves.

IMAGING FEATURES OF LATERAL MEDULLARY INFARCTION

DWI MRI is the best diagnostic test to confirm LMI. The infarcted area has a high DWI signal. LMI was identified by the MRI of the brain, performed within 24-48 hours after the onset of the stroke. On MRI LMI foci has the shape of a triangle (7-10 mm long in the rostrocaudal direction), facing the apex to the bottom of the IV ventricle, and the base to the lateral surface of the medulla oblongata. Ventrally, the infarct area was limited by olive, medially - by the roots of the XII pair of cranial nerves, dorsally reaching the gray matter of the bottom of the fourth ventricle, rostrally - to the pons.

CONCLUSIONS

LMI results from a vascular event in VA or PICA and characteristically has varied neurologic manifestations. LMI often affects descending spinal tract, descending sympathetic tract fibers, inferior cerebellar peduncle and dorsolateral medulla, the nucleus of the trigeminal nerve, nuclei, and fibers of the vagus nerves and glossopharyngeal, spinothalamic tract, and vestibular nuclei. As a result, it presents as complete (classic) or incomplete lateral medullary syndrome, also called Wallenberg syndrome or posterior inferior cerebellar artery syndrome. The first case of LMI was reported and described by Jewish neurologist and neuroanatomist who practiced in Germany, Adolf Wallenberg (1862-1949). Knowledge of the features of the neurological clinical course and im-

aging features of LMI is important for the medical doctor, helping to diagnose this type of posterior circulation stroke 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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