Clinical manifestations of acute pontine infarction: a narrative review

Abstract. Background. A pontine stroke presents the complexity of symptoms. Little narrative reviews are published about the clinical manifestations of acute pontine infarction. We aimed to provide a narrative review of the clinical manifestations of acute pontine infarction. Materials and methods. A comprehensive electronic literature search was performed on Scopus, Web of Science, MEDLINE, SciELO, PubMed, The Cochrane Library, Embase, Global Health, CyberLeninka, RINC databases, and databases of government scientific libraries of Ukraine, European Union, United Kingdom, United States, etc. It was done to identify publications that discussed the clinical manifestations of acute pontine infarction. Results. A narrative review of the clinical manifestations of acute pontine infarctions is presented and discussed. Conclusions. We provided a comprehensive narrative review of the clinical manifestations of acute pontine infarctions.

Keywords: posterior stroke; manifestations; pons; pontine infarction; syndrome; review

Introduction
Pontine infarctions (PI) are the most common among ischemic strokes of the brainstem. Clinical presentation of PI ranges from the classical crossed syndrome, the less common pure motor or pure sensory stroke to respiratory and cardiac dysfunction and the rare condition known as locked-in syndrome [1–4]. Early diagnosis is critical as PI is associated with high mortality and morbidity [5–7]. An adequate understanding of anatomy and clinical presentation is required for evaluating and managing the disease [8, 9].

The purpose was to provide a narrative review of the clinical manifestations of acute pontine infarction.

Materials and methods
A comprehensive electronic literature search was conducted on Scopus, Web of Science, MEDLINE, SciELO, PubMed, The Cochrane Library, Embase, Global Health, CyberLeninka, RINC databases, and databases of government scientific libraries of Ukraine, European Union, United Kingdom, United States, etc. to identify scientific publications that discussed the clinical manifestations of acute pontine infarction. The applicable articles are cited and referenced. No limit is placed on publication time or the language of the article. All relevant articles were identified and screened by three authors (M. Prokopiv, S. Yevtushenko, O. Fartushna), and disagreements were resolved by consensus. The results are summarized narratively.

Results and discussion
The pons Varolii belongs to the middle vascular and anatomical area of the posterior circulation [10]. Pontine infarctions may be isolated (with ischemic foci in the pons) and/or combined (manifested by simultaneous pontine and extra-pontine lesions of the cerebellum or other structures of the posterior circulation) [11]. The most common causes of isolated PI are lesions of the basal
artery (BA) branches, long and short circumferential arteries (43%); lesions of small paramedian arteries (34%), and less often — occlusion of BA (21%) [12].

**Alternating pontine syndromes**

Classical pontine syndromes first described by J. Dejerine in 1914 are as follows [13–15]:

— Millard–Gubler caudal ventral pontine syndrome is one of the classical brainstem alternating syndromes caused by a unilateral lesion in ventral pons, manifesting with ipsilateral peripheral paresis of facial muscles and heterolateral central hemiparesis [16];

— Foville caudal tegmental pontine syndrome is a peripheral paresis of the muscles innervated by the afferent and facial nerves on the affected side, with contralateral central hemiparesis [17];

— Raymond–Céstan rostral tegmental pontine syndrome starts with homolateral paresis of the eye in combination with hemihypesthesia and hemiataxia on the opposite side [18].

However, alternating syndromes in PI patients are rare.

**Main clinical forms of pontine syndromes**

C. Bassetti and co-authors [19] identified three main clinical forms of pontine syndromes in 36 patients with MRI-confirmed isolated PIs: ventral, tegmental, and bilateral ventral tegmental.

Ventral pontine infarctions (in 21 patients) clinically manifested by dysarthria, pyramidal hemiparesis with predominant lesions of the upper extremities or facial-brachial monoparesis, sensory dysfunction (violation of superficial and proprioceptive sensitivity), emotional disorders (manifestations of the pseudobulbar syndrome).

Ventral–tegmental pontine infarction is characterized by the presence of hemiparesis, horizontal gaze palsy, lesions of the abductor nerve, horizontal nystagmus, convergent strabismus, internuclear internal or external ophthalmoplegia. Ventral-tegmental pontine infarction can manifest with the one-and-a-half syndrome that occurs when the structures in the brainstem that coordinate eyeball movements are damaged. A combination of signs of supranuclear and internuclear ophthalmoplegia on the affected side is also found. Paramedian pontine infarctions are manifested by a horizontal one-and-a-half syndrome: the eyeball on the affected side is immobile, does not move in any direction (inward, outward), and the other eyeball can move only outward, i.e. only within half the normal range of motion. If we conditionally take the volume of movements of each eyeball as a unit, then one and a half units of the volume of eye movements fall out in total — hence the name of the syndrome. In vertical one-and-a-half syndrome associated with damage to the upper parts of the brainstem, the eyeballs remain immobile when looking down, and when looking up, only one eyeball moves.

Tegmental pontine infarctions are manifested by lesions of the ascending (spinothalamic tract and medial loop) and a part of the descending pathways, as well as gray matter — nuclei of the V, VI, and VII pairs of nerves located there. Clinically, this PI is characterized by dysarthria, nystagmus, recurrent syndrome, pontine pseudobulbar syndrome, similar to the hemisphere, contralateral hypoesthesia of vibrational and proprioceptive sensitivity. Alternating Millard-Gubler, Foville, and Raymond-Céstan syndromes can arise.

Bilateral ventral-tegmental pontine infarctions (in 4 patients) were accompanied by pseudobulbar syndrome, bilateral sensorimotor dysfunction. The development of ventral or tegmental pontine infarctions is associated, according to the authors, with stenosis of OA branches (44%), which led to the development of large ventral infarcts. Stenosis and occlusion of small paramedian or perforating arteries (25%) mostly caused the development of small ventral or tegmental lacunar infarctions. The lesion of the long circumferential arteries was accompanied by the foci of pontine and extrapontine infarctions.

S. Kataoka and co-authors [20] performed a correlation analysis of neurological clinical features using MRI in 49 patients with acute paramedian pontine infarctions. Given the location of the lesion, three variants of PIs were identified: ventral, ventral-tegmental, and tegmental.

Ventral pontine infarction is clinically manifested by dysarthria, pyramidal hemiparesis with predominant lesions of the upper extremities or facial-brachial monoparesis, sensory dysfunction (impaired superficial and proprioceptive sensitivity), emotional disorders (manifestations of the pseudobulbar syndrome).

Ventral–tegmental pontine infarction is characterized by the presence of hemiparesis, horizontal gaze palsy, lesions of the abductor nerve, horizontal nystagmus, convergent strabismus, internuclear internal or external ophthalmoplegia. Ventral-tegmental pontine infarction can manifest with the one-and-a-half syndrome that occurs when the structures in the brainstem that coordinate eyeball movements are damaged. A combination of signs of supranuclear and internuclear ophthalmoplegia on the affected side is also found. Paramedian pontine infarctions are manifested by a horizontal one-and-a-half syndrome: the eyeball on the affected side is immobile, does not move in any direction (inward, outward), and the other eyeball can move only outward, i.e. only within half the normal range of motion. If we conditionally take the volume of movements of each eyeball as a unit, then one and a half units of the volume of eye movements fall out in total — hence the name of the syndrome. In vertical one-and-a-half syndrome associated with damage to the upper parts of the brainstem, the eyeballs remain immobile when looking down, and when looking up, only one eyeball moves.

Tegmental pontine infarctions are manifested by lesions of the ascending (spinothalamic tract and medial loop) and a part of the descending pathways, as well as gray matter — nuclei of the V, VI, and VII pairs of nerves. Clinical features of these infarctions are described above.

**Other pontine syndromes** are also described in the scientific literature.

The lateral pontine infarction occurs in case of damage to the lateral parts of the pons at different levels, which are mainly vascularized by the anterior inferior cerebellar artery, superior cerebellar artery, and perforating branches. It is much less common than medial. Lateral inferior pontine infarction affects 3/5 of the lateral caudal pons, nuclei of the VI, VII pairs of cranial nerves, descending nucleus and root of the V nerve, inferior and middle cerebellar peduncles, spinothalamic tract, and descending sympathetic pathways. The clinical manifestation is characterized by acute onset, dizziness, vomiting, nystagmus, cerebellar ataxia, ipsilateral lesions of the VI, VII, and VIII pairs of cranial nerves, Bernard-Horner syndrome with impaired pain and temperature sensitivity on the opposite side.

Total (bilateral) pontine infarction occurs due to BA or bilateral vertebral artery thrombosis. Typical clinical signs: loss of consciousness, oculomotor disorders, recurrent horizontal syndrome, lesions of the VI, VII, VIII pairs of cranial nerves, trismus, tetraplegia, muscle tone disorders, convulsions, hypo- or atony of limb muscles, cerebellar disorders. BA obstruction is accompanied by damage not only to the
Pons but also to the midbrain, and cerebellum [21]. Bilateral lesions of the upper part of the pons may be accompanied by the locked-in syndrome, manifested by short-term loss of consciousness, bilateral facial palsy, anarthria, and loss of voluntary movements of the eyeballs [3].

E. Kumral and co-authors [22] in clinical and radiological analysis of 150 patients with acute pontine infarctions identified 5 main clinical syndromes that depended on the level of disturbances of the pontine blood supply:

1. the anterior medial pontine syndrome (58 %), manifested itself in motor deficits with dysarthria, ataxia, and in third of patients — moderate segmental symptoms;
2. anterolateral pontine syndrome (17 %), characterized by motor and sensory deficits in half of the subjects and the predominance (56 %) of segmental symptoms;
3. segmental pontine syndrome (10 %), manifested itself in moderate motor deficit and associated with sensory disturbances, oculomotor disorders, and symptoms of vestibular dysfunction (dizziness, ataxia);
4. bilateral pontine syndrome (11 %), characterized by transient loss of consciousness, tetraparesis, the development of acute pseudobulbar syndrome;
5. unilateral multiple pontine infarctions were observed rarely (in 4 % of cases) and were always characterized by severe sensorimotor deficiency and segmental symptoms.

According to the authors, there were no cases of infarction localized in the dorsal or lateral tegmental areas of the pons.

**Lacunar pontine syndromes**

Lacunar syndromes in PI are caused by lesions of the corticospinal, corticonuclear, corticopontine, and ponto-cerebellar tracts and are as follows [11, 23].

Pure motor pontine stroke with hemiparesis or hemiplegia accounts for 10.2 % of all primary ischemic strokes and is predominated among other lacunar pontine infarctions [24].

Pure sensory pontine strokes occur in case of ischemic lesions of the dorsal part of the pons. Because the medial loop and the spinothalamic tracts converge here, the lesions lead to a disturbance of superficial and proprioceptive sensitivity. Such patients often complain of dysesthesia.

Dysarthria-clumsy hand pontine syndrome occurs in the development of lacunar infarction 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 [25].

J. Kim and co-authors [26] studied the clinical manifestations of acute PIs and determined the correlation of clinical syndromes with neuroimaging results. Among 37 patients examined, 17 (46 %) had a clinical picture of pure motor hemiparesis or hemiplegia, 3 (8 %) — sensorimotor hemiparesis, 4 (11 %) — ataxic hemiparesis, 6 (16 %) — dysarthria-clumsy hand syndrome, in one patient (3 %), dysarthria-hemiataxia as a variant of ataxic hemiparesis was detected, in 2 (5 %) — quadraataxic paresis, and in 4 (11 %) — dysarthria-facial paresis syndrome as a variant of dysarthria-clumsy hand syndrome. Therefore, some symptoms of PI may be masked by ischemia in the other areas of the brainstem, which are supplied by the arteries of the posterior circulation.

**Symptoms duration**

Typically, if the PI was small and not bilateral, symptoms and recovery may last for 6 months. However, if the stroke was massive, the symptoms duration can be permanent or last for years [11, 27–30]. Paramedian pontine infarct may also have a fluctuating course [31]. Even in MRI-positive PI cases, stroke symptoms might be transient, lasting from seconds to 24 hours, causing so-called mini-stroke or transient ischemic attack [6, 32–34]. The physicians should consider the duration of the symptoms while diagnosing and managing PI, as 7–40 % of patients experience a transient ischemic attack before a stroke [35–37]. About 1/3 of people who have a transient ischemic attack might have a more severe stroke within one year [38, 39].

**Conclusions**

PI can cause a variety of serious symptoms, including balance issues, dizziness, double vision, loss of sensation and coordination, vertigo, nausea, swallowing difficulty, weakness in one half of the body, numbness, slurred speech, and so-called locked-in syndrome, etc. The clinical course of PI is extremely important in clinical practice when deciding about a patient’s diagnosis, management, and prognosis. Proper identification of pontine stroke symptoms by the patients themselves or by individuals witnessing the patient’s symptoms is vital for the timely initiation of emergency treatment. Stroke education influences the timely recognition of symptom onset and is critical in reducing the burden of a stroke worldwide. We provided a comprehensive narrative review of the clinical features of PI.

**References**


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**Клінічні прояви гострих інфарктів варолієвого мосту: огляд**

**Резюме. Вступ.** Мостові інсульти характеризуються складними симптомами та є важкими в діагностиці. Проте опубліковано мало наукових праць, у яких описано клінічні прояви гострих інфарктів варолієвого мосту. Ми прагнули надати ймовірний та короткий огляд наукової медичної літератури щодо клінічних проявів гострих мостових інсультів. 

**Матеріали та методи.** Проведено комплексний електронний пошук літератури в базах даних Scopus, Web of Science, MEDLINE, SciELo, PubMed, Cochrane Library, Embase, Global Health, CyberLeninka, RINC, а також у базах даних державних наукових бібліотек України, Європейського Союзу, Великобританії, США та інших країн з метою виявлення наукових публікацій, у яких обговорювалися клінічні прояви гострих ішемічних мостових інсультів. 

**Результати.** Наведено та обговорено огляд наукової медичної літератури щодо клінічних проявів гострих інфарктів варолієвого мосту. Набезпечене та обговорено клінічні прояви варолієвого мосту. 

**Висновки.** Ми надали дослідний огляд клінічних проявів гострих ішемічних інсультів варолієвого мосту. 

**Ключові слова:** інсульт вертебробазилярного басейну; клінічні прояви; мост; інсульт мосту; синдром; огляд