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HEMORRHAGIC STROKE: CURRENT STATE OF THE PROBLEM

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At the present stage, the problem of cerebrovascular diseases (CVDs) and stroke continues to occupy one of the leading positions in clinical neurology. The incidence of circulatory system diseases remains consistently high, and stroke continues to be one of the leading causes of mortality, placing a significant burden on the national economy due to the high costs of treatment, medical rehabilitation, and losses in productivity (Gusev E.I., Skvortsova V.I., 2007; Stakhovskaya L.V., 2014).

In Central Asia, the stroke incidence standardized to the European population was 3.28 cases per 1,000 population in 2010. The 28-day mortality rate from stroke onset was 0.91 cases per 1,000 population, while overall case fatality reached 25.3% (21.8% in men and 28.3% in women) (Stakhovskaya L.V., Klochikhina O.A. et al., 2013).

Currently, due to state health policies aimed at preventing circulatory system diseases and modernizing medical care for acute cerebrovascular accidents (ACVA), mortality has decreased and clinical outcomes have improved. In particular, over the past 11 years, mortality from ACVA has decreased by 52.3% and amounted to 123 per 100,000 population by 2016. Disability resulting from CVDs decreased by 73.8% (Gusev E.I. et al., 2017).

In the overall structure of ACVA, hemorrhagic stroke (HS) accounts for 8–30% of cases in the population. Mortality in the acute phase of the disease ranges from 35% to 50%. Among survivors, most patients experience persistent and severe neurological deficits, leading to disability and social maladaptation. Only 20% of patients are able to perform self-care independently six months after intracerebral hemorrhage (Gusev V.I., 2013; Dashyan V.G., 2009; Starodubtseva O.S., 2012). Large-scale epidemiological studies have revealed a tendency toward an increasing incidence of HS from western to eastern regions (Lutsky M.A., 2016).

Hemorrhagic stroke should be regarded as a syndrome of intracranial hemorrhage resulting from acquired changes and/or congenital anatomical defects associated with impaired vasculogenesis. These include destructive changes in small- and medium-caliber lenticulostriate arteries in arterial hypertension; cerebral vascular aneurysms, most commonly saccular aneurysms; arteriovenous malformations (AVMs) and cerebral angiomas; dural arteriovenous fistulas; cerebral amyloid angiopathy with amyloid protein deposition in cerebral vessels; mycotic aneurysms and arteritis as complications of infectious processes with septicemia; carotid–cavernous fistulas; and Moyamoya disease (Gusev E.I., 2013).

Rupture of a blood vessel inevitably leads to disruption of white matter tracts and irreversible damage to neurons in the basal ganglia or cerebral cortex. Direct mechanical compression of brain tissue surrounding the hematoma and the vasoconstrictive effects of blood breakdown products result in decreased local cerebral blood flow, formation of an ischemic zone (penumbra), and activation of ischemic pathobiochemical cascades. The volume of the ischemic zone may exceed the volume of the hematoma severalfold.

The clinical course of HS differs from that of ischemic stroke (IS) in several aspects: cerebral edema persists longer; recurrent hemorrhages are more frequent; acute obstructive hydrocephalus may develop; there is a high risk of brainstem displacement due to rapidly progressing cerebral edema; and secondary ischemia may develop around the intracerebral hematoma, which is often resistant to treatment.

In addition, the course and outcome of the acute phase of HS are aggravated by numerous somatic complications resulting from prolonged immobilization and decreased immune reactivity. The most frequent and dangerous complications include superficial and deep vein thrombosis of the lower extremities with possible progression to pulmonary embolism,

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disseminated intravascular coagulation syndrome, hypostatic nosocomial pneumonia, and acute stress-related ulcers, predominantly of the upper gastrointestinal tract (Stakhovskaya L.V., 2014).

The “gold standard” for diagnosing HS in the hyperacute phase remains non-contrast computed tomography (CT). Magnetic resonance imaging (MRI) is more informative in the subacute and chronic phases. In addition, CT angiography and MR angiography have been introduced into clinical practice, allowing the identification of various vascular abnormalities underlying the etiology of HS (Gaidar B.V., 2007; Suslina Z.A. et al., 2016; Shmyrev V.A. et al., 2012).

Despite the lack of definitive evidence demonstrating the superiority of surgical treatment over conservative therapy, intensive efforts have been made in recent years to develop and implement innovative surgical approaches for HS (Dashyan V.G., 2009). Worldwide, the number of neurosurgical interventions for non-traumatic intracranial hematomas has increased to 13,820 cases (Gusev E.I., 2017). Given the extremely limited regenerative capacity of neurons in acute cerebrovascular catastrophes, neuroprotection—aimed at protecting neurons from the damaging effects of neurochemical cascades—remains one of the key challenges in modern neurology.

Prognostically unfavorable factors in HS include impaired consciousness at disease onset, large hematoma volume and medial localization, intraventricular hemorrhage, high systolic blood pressure in the early hours of stroke, early development of papilledema, history of previous stroke or myocardial infarction, and vital function disturbances. The prognosis of HS largely depends on the development of complications, the most severe of which include intraventricular hemorrhage, brain displacement, development of acute obstructive hydrocephalus, and secondary hemorrhages into the brainstem. In favorable cases, autolysis of blood within the hemorrhagic focus occurs within 1–2 weeks; after erythrocyte breakdown and resorption of blood degradation products, a smooth-walled cavity (cyst) filled with yellowish fluid remains at the site of the hematoma.

Extracerebral complications, rather than neurological deficits, often determine outcomes in severe strokes, usually occurring 4–10 days after onset. Stroke represents a powerful stressor for the organism and almost invariably leads to exacerbation or decompensation of chronic diseases, which are common in individuals over 50–60 years of age. Among extracerebral complications, pulmonary embolism plays a leading role and is responsible for death in every fourth patient. Less frequent causes of death, in descending order, include acute cardiovascular failure, pneumonia, gastrointestinal bleeding, acute renal failure, and myocardial infarction. The most severe manifestation of extracerebral pathology is multiple organ failure syndrome, the prevention of which should be given particular attention.

The persistent upward trend in the incidence of hemorrhagic stroke, along with high mortality and disability rates, necessitates an in-depth investigation of the etiological factors and pathogenetic mechanisms underlying this form of cerebrovascular pathology, thereby confirming the relevance and importance of further research in this field.

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