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DIFFERENTIAL DIAGNOSIS OF HEMORRHAGIC AND ISCHEMIC STROKE

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Abstract:

Despite the enormous efforts of the world community aimed at combating stroke and its consequences, as well as the existing significant achievements in this area, a number of issues still remain controversial and unclear and require further study.So, it is not known for certain which ones, in what sequence , at what stage of the disease, in what combination and quantity, with what frequency and duration, numerous non-drug methods and medications should be considered the most effective for the rehabilitation of patients who have suffered a stroke.

Keywords: Stroke, neuroprotectors, cytokines, reperfusion.

The pathogenesis of focal cerebral ischemia (in the basin of individual arteries due to thrombosis or embolism) is described by the classical Virchow triad: a decrease in blood flow velocity, damage to the vascular wall and an increase in blood coagulability. Acute focal cerebral ischemia causes a certain sequence of molecular-biochemical changes in the brain substance, which can lead to tissue disorders resulting in cell death (cerebral infarction). The nature of the changes depends on the magnitude of the decrease in cerebral blood flow, the duration of this decrease, and also on the sensitivity of the brain substance to ischemia. Normally, the volumetric cerebral blood flow is 50 - 55 ml of blood per 100 g of brain substance per minute. A moderate decrease in blood flow (≈ 40 ml / 100 g / min) is accompanied by selective gene expression and changes in protein synthesis processes. A more pronounced decrease in blood flow (up to 30 ml per 100 g / min) is accompanied by the activation of anaerobic glycolysis and the development of lactic acidosis. With a decrease in the volumetric cerebral blood flow to 20 ml per 100 g / min, glutamate excitotoxicity develops and the content of intracellular calcium increases, which triggers the mechanisms of structural damage to membranes and other intracellular formations. With significant ischemia (up to 10 ml per 100 g / min), anoxic depolarization of membranes occurs, and cell death usually occurs within 6 - 8 minutes [4]. With focal cerebral ischemia, the degree of decrease in the cerebral blood flow velocity is different (in the center of the focus - complete cessation, towards the periphery, the velocity increases to normal). The area of the brain substance in which blood flow has decreased to less than 10 - 12 ml / 100 g per minute is called the "necrosis" / "nuclear heart attack" zone. Changes in the brain tissue are accompanied by a pronounced energy deficit, loss of ionic gradient, membrane depolarization with irreversible damage to cells (in the form of destruction of the cell membrane, mitochondria and DNA destruction). In most cases, necrosis in the nuclear zone of the infarction is formed within 5 minutes after occlusion of the artery [1, 10]. The substance of the brain located around the nuclear zone of the infarction, where the MC value ranges from 15 ml / 100 g per minute to 20 ml / 100 g per minute, is called the "penumbra" zone (ischemic penumbra - Latin paene- "almost" and umbra "shadow"). In the "penumbra" zone, the brain tissue is functionally inactive (there is not enough energy substrate for adequate functioning), but viable (the structural integrity of neurons and glial cells is preserved). The ischemic penumbra includes areas that recover on their own ("benign oligemia") and areas that, without successful reperfusion therapy, turn into cerebral infarction. The key role in the transformation of the "penumbra" zone into a necrosis focus is played by the duration of ischemia and the degree of decrease in MC. The rate of irreversible damage to the brain tissue is also determined by indicators of the oxygen-transporting properties of blood (anemia, impaired oxygenation of hemoglobin and its chemical properties), electrolyte balance, osmotic pressure, temperature, glucose content and the state of collateral blood flow [5,6]. The principle "time - brain" (English Time is brain) reflects the rapid rate of death of brain tissue cells under the influence of adverse factors. The concept of ischemic penumbra substantiates the need for emergency medical care for patients with acute cerebrovascular accidents [11,]. With a large ischemic zone that occupies at least 50% of the MCA blood supply basin, the disease may be accompanied by severe mass-effect edema and intracranial hypertension, displacement of brain structures with the development of temporotentorial involvement. Most of these strokes are caused by cardioembolic or thrombotic occlusion of the internal carotid artery or M1 MCA. Such a complicated course of stroke is malignant and occurs in 36–78% of patients with massive hemispheric ischemic stroke [8,9]. Cerebral edema with a pronounced mass effect and neurological deterioration can develop both within 24 - 36

hours after the onset of a stroke, or more gradually (over several days - a week) [4, 10]. A cascade of local inflammatory reactions involving immune cells and proinflammatory cytokines leads to significant brain damage, disruption of the blood-brain barrier and determines the triggering of the vasogenic component of cerebral edema. In 25% of patients with malignant stroke, cerebral edema begins on the first day of the disease [2]. In patients with malignant stroke, mortality reaches 78% (with only drug treatment) [4]. Venous stroke Thrombosis of the cerebral veins and sinuses of the dura mater is one of the rare causes of stroke, which often remains unrecognized [3]. The incidence of venous stroke ranges from 0.22 to 1.57 per 100,000 (the ratio of the incidence in women and men is 3/1 [12]. Gender differences may be due to an increased risk of cerebral vein and dural sinus thrombosis associated with pregnancy, the postpartum period, as well as with the use of oral contraceptives [13] Thrombosis of the cerebral veins or sinuses of the dura mater prevents the outflow of blood from the brain tissue, which leads to an increase in venous and capillary pressure with subsequent disruption of the blood-brain barrier, vasogenic edema and passage of interstitial plasma Further growth of ICP can lead to venous hemorrhage due to rupture of venules and capillaries.8 The most common risk factors for thrombosis of cerebral veins and sinuses of the dura mater are: prothrombotic conditions (both genetic and acquired); oral contraceptives; pregnancy and postpartum period; the presence of malignant neoplasms; infection; head injury [14]. Clinical manifestations in violation of blood flow through the internal carotid artery are largely determined by the development of collateral circulation and the level of occlusion. The defeat of the extracranial part of the internal carotid artery, as a rule, is characterized by moderate neurological symptoms and often manifests itself in the form of TIA or minor strokes. In this case, a mosaic nature of clinical manifestations may be noted. With occlusion in the area of origin of the ophthalmic artery or proximal to this area, the development of Denny-Brown ophthalmoplegic syndrome is characteristic, which is characterized by blindness on the side of the focus (due to ischemia of the retina and optic nerve) and central hemiplegia or hemiparesis, sometimes in combination with hemihypesthesia on the opposite side. With occlusion of the intracranial part of the internal carotid artery, pronounced neurological symptoms are often noted with the development of contralateral hemiparesis, hemihypesthesia, and impairment of higher mental functions.

Stroke is divided into hemorrhagic and ischemic (cerebral infarction). A minor stroke is distinguished, in which the impaired functions are fully restored during the first 3 weeks of the disease. However, such relatively mild cases occur in only 10-15% of stroke patients. Transient cerebrovascular accidents (PTCA) are characterized by the sudden onset of focal neurological symptoms that develop in a patient with vascular disease (arterial hypertension, ischemic heart disease (IHD), rheumatism, etc.) and last for several minutes, less often for hours, but no more than a day and end with a complete restoration of the impaired functions. Transient neurological disorders with focal symptoms that develop as a result of short-term local cerebral ischemia are also referred to as transient ischemic attacks (TIA). A special form of PNMC is acute hypertensive encephalopathy. More often, acute hypertensive encephalopathy develops in patients with malignant arterial hypertension and is clinically manifested by a sharp headache, nausea, vomiting, impaired consciousness, convulsive syndrome, in some cases accompanied by focal neurological symptoms.

Despite the fact that primary prevention is of decisive importance in reducing mortality and disability due to stroke, a significant effect in this regard is provided by the optimization of the system of care for ACVA patients, the introduction of therapeutic and diagnostic standards for them, including rehabilitation measures and the prevention of recurrent strokes.

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