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Research Article

DIAGNOSTICS OF MICROCIRCULATION DISTURBANCES IN CRITICAL CONDITIONS IN PATIENTS WITH ACUTE CEREMONIAL CIRCULATION DISORDER AND SEVERE CRANIO-BRAIN INJURY

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ABSTRACT

A new emergency method is proposed for determining the degree of microcirculation disorders in critical conditions in patients with acute cerebrovascular accident and severe traumatic brain injury, based on the capillary-venous difference in blood hemoglobin. The technical method is simple and convenient for any clinical laboratory and allows you to control the dynamics of the effectiveness of ongoing intensive care.

KEYWORDS

Capillary, venous hemoglobin, brain, trauma.

INTRODUCTION

Traumatic brain injury (TBI) is the leading cause of death and morbidity among people under 45 worldwide and remains one of the most common types of injury to this day. In the general structure of injuries, it accounts for about 40-50% of all types of injuries, and

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the number of patients with TBI in recent decades has only been growing [5,11,17,23].

Among the causes of TBI, domestic and road traffic injuries are leading. TBI is a huge global health problem. The cost of neurorehabilitation after TBI is more than $35 billion a year. The total loss to society from disability and the cost of providing medical care to the victims is about $100 billion a year. The overall mortality in severe TBI reaches 65–70%, and 50% of victims who have had TBI lose their ability to work to some extent [9].

The study of neuronal damage and the mechanisms of their protection in critical conditions is an urgent problem in resuscitation. In patients with TBI, factors contributing to the development of secondary brain damage include: arterial hypotension, hypoxia, excessive formation of reactive oxygen species, anemia, endotoxemia, hyper- and hypocapnia, disorders of water-electrolyte and energy metabolism [1,16,17].

Modern ideas about the pathogenesis of TBI are based on the identification of primary and secondary factors of brain damage [11,18].

The action of the primary traumatic agent triggers the development of biochemical and immunological reactions that lead to destructive processes. Oxidative phosphorylation in mitochondria is disturbed, the concentration of intracellular calcium increases, free oxygen radicals and vasoactive metabolites of arachidonic acid are released, and the mechanisms of the complement cascade and lipid peroxidation are activated [11].

The action of factors of secondary brain damage leads to a disruption in the delivery of oxygen and nutrients to brain cells and causes their insufficient utilization. There are disorders of cerebral microcirculation, oxygenation and metabolism of neurons, brain edema and its ischemia develop [12]. Secondary ischemic brain damage, according to different authors, develops in 36.0–42.6% of patients with TBI, the severity of which corresponds to an average degree, and in 81.0–86.4% of patients with severe TBI [4]. A number of modern studies consider microcirculatory dysfunction as the central mechanism for the formation of multiple organ failure in critical conditions [24,28].

Among the extracranial factors of secondary brain damage that contribute to the development and maintenance of intracranial hypertension, there are: arterial hypotension, hypoxemia, hypo- and hypercapnia, hyperthermia, disturbances in electrolyte homeostasis and water-energy metabolism. All these factors trigger a chain of pathological reactions that lead to impaired oxygenation and metabolism of nerve cells and the development of cerebral ischemia. Increasing ischemia causes cerebral edema with an increase in its intracranial volume, which, in turn, leads to a further increase in ICP. Due to intracranial hypertension, cerebral blood flow decreases and brain perfusion is disturbed [28, 30].

Cerebral hemodynamic disorders leading to insufficient brain perfusion and the development of its ischemia are considered to be the main factors causing secondary brain damage in TBI [29].

The death of neurons in TBI also occurs due to the initiation of apoptosis processes, which can be triggered both directly by the action of a traumatic agent on the cell genome, and due to the damaging effect of inflammatory mediators. At the moment of injury, a sharp inhibition of the autonomic system of cerebral blood flow regulation develops, which leads to a significant decrease in volumetric cerebral blood flow in the area of injury and adjacent brain tissue [21].

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When autoregulation of cerebral blood flow is disturbed in the acute period of TBI, the ability of cerebral capillaries to compensatory changes in tone in response to changes in blood pressure and carbon dioxide levels in arterial blood is impaired, which leads to an increase in the sensitivity of the brain to ischemic damage. Under conditions of reduced cerebral blood flow, even a slight decrease in cerebral perfusion pressure can lead to the development of secondary ischemic brain damage. This position is also confirmed by the fact that arterial hypotension in the acute period of TBI, leading to a decrease in cerebral perfusion, becomes a statistically significant prognostic factor for the development of an unfavorable outcome [25].

In addition, there is evidence that with an increase in the duration of episodes of arterial hypotension, the risk of developing such outcomes increases by more than 2.5 times [8].

One of the earliest extracranial factors of secondary brain damage, which begins to act immediately after injury, is arterial hypotension (a decrease in systolic blood pressure less than 90 mm Hg). As a result of a decrease in blood pressure, brain perfusion decreases and its ischemia develops. The most common causes of arterial hypotension in patients with severe TBI include hypovolemia and violations of the central regulation of hemodynamics. Hypovolemia - a discrepancy between the volume of circulating blood (BCV) and the capacity of the vascular bed - may be due to shock and blood loss in patients with TBI and severe extracranial injuries (fractures of large bones of the skeleton, damage to internal organs, extensive wounds of the skin), as well as insufficient fluid intake from - for violations of the level of wakefulness, fluid loss during hyperthermia, diarrhea, vomiting, polyuria. Sometimes a number of patients have a sufficient fluid content in the body, but a decrease in BCV is associated with vasodilation or increased capillary permeability. Violations of the central regulation of hemodynamics occur as a result of the spread of the dislocation syndrome to the level of the medulla oblongata with involvement of the vasomotor center in the pathological process or with primary contusion of the brainstem [13,26,27].

The significance of the problem of acute cerebrovascular accident (ACV) is determined by its prevalence, high mortality and disability. Vasoconstriction in ischemic stroke leads to the development of circulatory hypoxia. In the early stages of ischemic stroke, there is a decrease in the oxygen delivery index due to the development of a hypodynamic type of blood circulation. In the future, against this background, pulmonary complications and microcirculation disorders join. One of the reasons for the development of hemodynamic disorders, and consequently the oxygen transport system, is a dysfunction of the stem structures as a result of their damage, which is confirmed by clinical, neurophysiological, radiological and pathoanatomical data [2,3,10].

The features of microcirculation disorders established in ischemic stroke make it possible to recommend active use of drugs that improve arterial blood flow and relieve vascular spasm to patients with ischemic stroke both in the early and late recovery periods. Detection of microcirculatory disorders in patients with ischemic stroke and their timely correction at the early stages of neurorehabilitation will improve the quality of life and optimize early rehabilitation in this category of patients [6].

Against the background of acute focal cerebral ischemia, a microcirculatory-cellular cascade of reactions is realized. Medical science has advanced in the systematization of microcirculatory and cellular reactions that occur in IS [2,22], but their pathogenetic

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mechanisms and clinical significance have not been studied enough.

An urgent problem is the search for technologies for studying microcirculation not only in patients with acute ischemic stroke, but also with chronic disorders of cerebral circulation.

Laboratory signs of hypoxemia include a decrease in the partial pressure of oxygen in arterial blood (PaO_2) less than 60 mm Hg. and a decrease in arterial oxygen saturation (SaO_2) of less than 90%.

Direct assessment of microcirculatory blood flow using instrumental research methods has a number of advantages over indirect methods. Thanks to modern technical advances, it has become possible to promote modern methods for studying microcirculation in clinical practice [14,19].

Despite the existing limitations, a set of facts indicates the effectiveness of microcirculation monitoring and may be the rationale for choosing therapies. There is a proven relationship between low cardiac output, inadequate oxygen delivery, reduced venous hemoglobin oxygen saturation (SvO_2), central venous hemoglobin oxygen saturation ($ScvO_2$), and poor outcome in critically ill patients. The use of these variables as endpoints for intensive care was associated with favorable disease outcomes. It was assumed that this leads to an improvement in tissue perfusion and saturation with oxygen, a decrease in the level of complications and organ dysfunction [7,20].

At the same time, as some authors note, there is no objective and reliable method of dynamic control of microcirculatory blood flow in critical conditions in the practice of intensive care units.

The development of secondary brain damage significantly aggravates the condition of patients with TBI, impairs the recovery of mental and motor activity, and increases the risk of developing an unfavorable outcome. In this regard, prevention and timely correction of factors of secondary brain damage remain the most important task in the treatment of patients with severe TBI and ischemic stroke.

Thus, by preventing and limiting the action of secondary ischemic factors of brain damage, it is possible to significantly improve the prognosis of ischemic stroke in severe TBI.

The purpose of the study: to develop a method for diagnosing microcirculation disorders and to evaluate its information content in patients with acute cerebrovascular accidents and severe TBI.

Material and methods. 37 patients aged 32 to 65 years (mean age 56.3 ± 3 years) were examined in the neuro-reanimation department of the Bukhara regional branch of the Republican Scientific Center for Emergency Medical Care. Hemoglobin in capillary blood and from the subclavian vein was studied in 11 patients with acute cerebrovascular accident and in 14 patients with severe TBI.

Based on the opposite effect of Fareus Lindqvist, that in pathological conditions with a decrease in the diameter of the vessel, blood viscosity increases slightly, and therefore the red blood indices (Hb and Ht) in the central and peripheral vessels should be different, we proposed a simple method for determining microcirculation disorders based on the difference in capillary - venous hemoglobin (Hbcapillary-Hbvenous) blood. As is known, in healthy people, in the study of hemoglobin, the values of capillary and venous blood do not have a significantly

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significant difference. The control group consisted of 12 patients.

Method for emergency diagnosis of disorders of blood microcirculation in patients is carried out as follows. Upon admission to the clinic, before the start of infusion therapy, the patient is simultaneously taking capillary blood from the finger and venous blood from the subclavian vein. Blood for hemoglobin content is examined with an MKMF-1 microcalorimeter. At the same time, saturation is measured on the finger with a pulse oximeter. The values of capillary and venous hemoglobin obtained from the general blood test are compared. According to the ratio of the difference in capillary-venous hemoglobin (HbcapillaryHbvenous, g/l) to the blood saturation index (SpO2,%), from the proposed formula:

KDDBM = (Hbcap-Hbvein) / SpO2

determine the coefficient of the degree of disturbance of blood microcirculation (KDDBM, conventional units). The directly proportional dependence of the coefficient of the degree of disturbance of blood microcirculation on the comparative value of the obtained difference in capillary-venous hemoglobin and the inversely proportional dependence on the index of blood saturation, shows that the greater the difference in capillary-venous hemoglobin of the blood, the more disturbed microcirculation and the lower the value of oxygen saturation of arterial blood. With an increase in the coefficient of the degree of disturbance of blood microcirculation from 0.1 and more conventional units. the degree of disturbance of blood microcirculation is diagnosed. Normally, this ratio is 0.01-0.09 conventional units. units, (Patent UZ FAP No. 02139).

RESULTS AND DISCUSSION

Our studies have shown a significant difference in capillary-venous hemoglobin (from 7 to 12 g/l. P<0.05) in patients with severe TBI and acute cerebrovascular accident, while in patients in the control group this difference was 2-3 g/l. l., (see table 1).

The difference in capillary-venous hemoglobin (Hb) in patients control and main groups (g/l). Table 1.

Patients	Number of patients	Hb capillary	Hb venous	Difference ($Hb_{capillary}$ - Hb_{venous})	SpO_2	KDDBM, conventional units	P
Control gr.	12	114±2,4	112±1,6	2 ±1,2	96±1,4	0,02	P>001
Severe TBI	14	119±2,3	107±2,1	12±2,2	79±3,2	0,15	P<005
Strokes	11	122±1,2	115±1,1	7±1,3	82±1,3	0,1	P<005

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Total	37	-	-	-	-	-	-

At the same time, it was found that the largest difference in capillary-venous hemoglobin was in patients with severe TBI. Loss of central regulation of vascular tone and hypovolemia due to blood loss in severe TBI seem to exacerbate changes in the rheological properties of blood, which leads to severe microcirculation disorders.

Thus, the results of emergency diagnostics obtained by the proposed method indicate that the more severe the patient's condition, the higher the index of the degree of disturbance of blood microcirculation (from 0.1 and more standard units), which means that microcirculation is disturbed more and more deeply, and the level of saturation of arterial blood decreases accordingly. The method for emergency diagnosis of blood microcirculation disorders in these patients is convenient, simple and accessible to all emergency clinical laboratories. EFFECT: method makes it possible to provide a fast reliable result of the analysis of blood microcirculation disorders, control the effectiveness and timely correction of the ongoing infusion therapy, reduce the time of research and reduce their cost.

CONCLUSION

The proposed method for emergency diagnosis of blood microcirculation disorders in patients with acute cerebrovascular accidents and severe TBI allows you to quickly determine microcirculation disorders, select the composition of infusion therapy and ensure effective monitoring of the results obtained from it in dynamics.

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