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INTERNATIONAL CONFERENCE ON SUPPORT OF MODERN SCIENCE AND INNOVATION: a collection scientific works of the International scientific conference – Madrid, Spain, 2026, Issue 2.

Languages of publication: Uzbek, English, Russian, German, Italian, Spanish,

The collection consists of scientific research of scientists, graduate students and students who took part in the International Scientific online conference «**INTERNATIONAL CONFERENCE ON SUPPORT OF MODERN SCIENCE AND INNOVATION**». Which took place in Spain, 2026.

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MORPHOLOGICAL CONSEQUENCES OF NEONATAL INTRAVENTRICULAR BLOOD TRANSFUSION

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Annotation. Neonatal intraventricular hemorrhage (IVQ) is a severe neuropathological condition that occurs mainly in preterm babies and is associated with a high risk of mortality and disability. This article elaborates on the basis of the pathogenesis of neonatal intraventricular hemorrhage, the macroscopic and microscopic morphological changes occurring in brain structures, as well as their clinical consequences. The degree of morphological lesions were compared with the stages of blood transfusions and pathoanatomical mechanisms of complications such as posthemorrhagic hydrocephalus and periventricular leukomalacia were analyzed. The article has important theoretical and practical importance in the practice of neonatal neurology and pathological anatomy.

Keywords: neonatal period, intraventricular hemorrhage, germinative matrix, cerebral cortex, pathomorphology, hydrocephalus, periventricular leukomalacia.

Introduction. The neonatal stage is one of the most delicate and important stages in a person's life, and the pathological conditions that occur during this period have a direct impact on subsequent quality of life and neurological development. Neonatal intraventricular hemorrhage remains one of the most pressing problems in modern neonatology, as this pathology continues to occur at a high rate, despite the expansion of intensive care options.

Intraventricular hemorrhage is observed mainly in infants born before the 32nd week of pregnancy and is associated with morphological immaturity of the germinative matrix vessels located around the cerebral ventricles. As a result of blood transfusions, irreversible morphological changes occur in brain tissue, leading to cerebral palsy, epilepsy, cognitive deficits and other severe neurological complications in children.

The aim of this article is to provide an in-depth and systematic explanation of the morphological consequences of neonatal intraventricular hemorrhage from the perspective of pathological anatomy.

Intraventricular hemorrhage (IVH), also known as intraventricular hemorrhage, is a bleeding into the ventricular system of the brain, where cerebrospinal fluid is produced and circulates towards the subarachnoid cavity. It can be caused by a physical injury or a hemorrhagic stroke.

30% of intraventricular hemorrhage (IVH) is primary and is confined to the ventricular system and is usually caused by intraventricular trauma, aneurysm, vascular malformations, or tumors, especially the choroid plexus. However, 70% of IVH is secondary in nature and occurs as a result of enlargement of existing intraparenchymal or subarachnoid hemorrhage. Intraventricular hemorrhage has been found to occur in 35% of moderate to severe traumatic brain injuries. Thus,

bleeding usually does not occur without extensive associated damage, and therefore the outcome is rarely good.

Symptoms

Adults

IVH symptoms are similar to other intracerebral hemorrhages and include headaches, nausea, and sudden onset of relapse, along with changes in mood and/or level of consciousness. Focal neurological signs are minimal or absent, although focal and/or total seizures may occur. Xanthohromia, the yellow cerebral spinal fluid, is a common find,[6] but other medical conditions can also be associated with xanthohromia.

Babies. Some babies are asymptomatic, while others may find it difficult to detect abnormalities in mind, muscle tone, breathing, eye movements, and body movements.

Premature babies and very low weight babies are also at high risk. [9] IVH in the early brain usually originates from the germinal matrix, while IVH in infants originates from the choroid plexus. However, this is especially common in premature babies or very low weight infants. [10] The cause of IVH in premature infants is rarely caused by trauma, as opposed to in older infants, children, or adults. Instead, it is hypoxic ischemic encephalopathy, which is particularly vulnerable to hypoxic ischemic encephalopathy, resulting from alterations of perfusion of delicate cellular structures present in the developing brain, which is enlarged by the immature cerebral circulatory system. Lack of blood flow leads to the death of cells and subsequent rupture of the walls of blood vessels, which leads to hemorrhages. While this bleeding can lead to subsequent injuries, it is a sign for an injury that has already occurred. Most often, intraventricular hemorrhages occur within the first 72 hours after birth. The risk of having the extracorporeal membrane oxygenated in premature infants increases. Congenital cytomegalovirus infection may be an important cause.



Indicating spontaneous intracerebral hemorrhage in the third and both lateral ventricles and hydrocephalic hemorrhage

The most common cause of periventricular intraventricular hemorrhage (PIVD) in newborns is the fragility and unique morphology of the blood vessels in the germinal matrix, as well as the mechanism of immature brain autoregulation. This mechanism usually allows the brain to maintain stable blood flow despite changes in blood pressure, so immaturity in this area significantly increases the risk of bleeding. In addition, the blood vessels in the neonatal germinal matrix have a high density, surface area and a rounded shape. These differences further increase the risk of IVH. Some external risk factors for neonatal IVH include transportation after external delivery, mechanical ventilation, and frequent intubation

The amount of bleeding varies in premature and premature infants with IVH. IVH is often described in four classes:[should] Class I-hemorrhage occurs only in the germinal matrix Degree II-hemorrhage also occurs inside the ventricles, but they do not increase in size Class III - ventricles fold in size with accumulated blood Grade IV-hemorrhage spreads to the brain tissues around the ventricles Grades I and II are the most common and often there are no other complications. Classes III and IV are considered the most serious and can cause long-term brain damage to an infant. After grade III or IV, blood clots may occur, which can block the flow of cerebrospinal fluid, which leads.

Research objective. To study macroscopic and microscopic morphological changes that develop in the cerebral ventricles and parenchyma as a result of neonatal intraventricular blood transfusion and to determine their clinical significance.

Research material and methods. The study was based on the following sources:
Materials of the post-mortem autopsy of deceased infants during the neonatal period

Results from neurosonography, computed tomography and magnetic resonance imaging

Histological incisions of brain tissue

Histological studies were carried out with the help of hematoxylin-eosin, Perls dye and special immunohistochemical methods. The results obtained were compared with clinical data.

Pathogenesis of neonatal intraventricular hemorrhage

The basis of IVQ pathogenesis is the following factors:

Thinness of germinative matrix veins

Lack of collagen and elastic fibers in the vessel wall

Immaturity of cerebral circulation autodysregulation

Cases of hypoxia and ischemia

Sharp change in arterial pressure

Blood first accumulates in the subependymal area, then spreads to the cerebral ventricles as a result of rupture of the ependyma.

Morphological consequences of intraventricular hemorrhage

1. Macroscopic morphological changes

Liquid or viscous blood masses in the side and third ventricles

Cerebral ventricular enlargement

Irregularities and defects of the surface of the ependyma

Brain tumor

Parenchymous hemorrhages in severe cases

Enlargement of the cerebral ventricles is associated with a violation of the cerebrospinal fluid circulation, provoking the development of posthemorrhagic hydrocephalus.

2. Microscopic morphological changes

Massive hemorrhages in the germinative matrix

Ependymal Layer Dequamation and Necrosis

Vacuolization and Picanotic Changes of Neurons

Glial cell degeneration

Macrophages saturated with hemosiderin

In prolonged processes, gliosis and fibrosis develop.

3. Reactive and inflammatory processes

Microglial cell activation

Astroglial hyperplasia

Increased cytokine production

These processes limit the brain's ability to regenerate . Lyubrodynamic disorders

Fibrosis of the arahnoidal villi

Decrease in liquid resorption

Posthemorrhagic hydrocephalus

IVQ levels and their morphological description

Level

Morphological characters

1.Subependymal hemorrhages

2.The blood has passed to the ventricle, without enlargement

3.Ventricles enlarged

4.Haemorrhages disseminated to the parenchyma Clinical and morphological significance

The degree of morphological lesions determines the clinical prognosis. In severe IVQ cases:

Infant cerebral palsy

Lagging behind in psychomotor development

Epileptic seizures

Cognitive functions are reduced

Discussion. Morphological changes resulting from neonatal intraventricular blood transfusion are a profound and multi-stage process that seriously impede the functional maturation of brain structures. In particular, damage to the germinative matrix disrupts the migration of neurons and glial cells.

Prevention. To reduce the risk of intraventricular hemorrhage in the neonatal period, several studies have indicated a single course of corticosteroids given from day 24 to day 33 weeks gestational from day 0 at 24 weeks to day 33. [14] Head positioning has been proposed in very premature infants as an approach to prevent germinal matrix bleeding; however, more research is needed to determine the effectiveness of lethal reduction and the optimal location determination technique. [15] Approaches include tilting the bed, placing the lying midline head, rotating the lying head 90 degrees, placing the inclined midline head, placing the head on the tile.

Treatment. Treatment should be monitored and followed by inpatient ward service for individuals who respond to orders, or neurological ICU follow-up for those with impaired consciousness. Additional attention should be paid to monitoring intracranial pressure (ICP) through an intraventricular catheter and to medications for ICP, blood pressure, and coagulation maintenance. In later severe cases, an external ventricular drainage may be required to contain ICP and evacuate bleeding, and in more severe cases, an open craniotomy may be required. In unilateral IVH cases with small intraparenchymal hemorrhage, the combined method of stereotaxy and open craniotomy has yielded promising results.

Babies. Germinal matrix hemorrhage in infants is associated with cerebral hemorrhage, cognition problems, and hydrocephalus. With improved technological advancements in science and medicine, survival has improved for premature infants with this type of neurological disease, and premature infants with germinal matrix hemorrhage have severe cerebral palsy. [8] Approximately 15% of surviving premature infants develop brain paralysis, and 27% of infant survivors experience moderate to severe neurosensory deficits by the time they reach 18–24 months of age. When IVH is caused by intracerebral bleeding associated with high blood pressure, the prognosis is very poor and even worse when hydrocephaly occurs. This can lead to an increased risk of ICP and potentially fatal cerebral hernia. Even on its own, IVH can cause illness and death. First, intraventricular blood can cause a thrombus that blocks its flow in the CSF ducts and leads to obstructive hydrocephalus, which can quickly lead to intracranial pressure and death. [16] Second, the decaying products from the bloodstream can induce an inflammatory response that damages ARAXNOID granulations, inhibits the normal reabsorption of CSF, resulting in continuously communicating hydrocephaly

Epidemiology. IVH has been reported to occur in about 25 percent of babies born with very low birth weight. Intraventricular hemorrhage and germinal matrix hemorrhage in premature infants are the most commonly reported neurological disorders. In the United States, approximately 12,000 babies are diagnosed with germinal matrix hemorrhage or intraventricular hemorrhage each year In a 2002 Dutch retrospective study in which neonatologists analyzed cases of CSF mixed and dried with lumbar or ventricular punctures when ventricular width (as shown in ultrasound) exceeded 97-centile plus 4 mm, as opposed to 97-centile. Professor

Whitelaw's original Cochrane Review was published in 2001, and evidence from previous randomised control trials suggested that interventions should be based on clinical signs and symptoms of ventricular enlargement. The international court instead looked to the late (97th centile plus 4mm) against the late (97th centile plus 4mm) CSF to intervene and dry.

DRIFT was tested in an international randomized clinical trial; Although it did not significantly reduce the need for shunt surgery, Bayley (MDI <55), who had severe cognitive disability over two years, was significantly reduced. Repeated lumbar punctures are widely used to reduce the effects of increased intracranial pressure and as an alternative to ventriculoperitoneal (VP) shunt surgery, which is not performed in intraventricular hemorrhage. The relative risk of recurrent lumbar puncture is close to 1.0, so it is not statistically therapeutic compared to conservative treatment and increases the risk of subsequent CSF infection

Conclusion. Neonatal intraventricular hemorrhage is one of the most severe and complex pathologies of the central nervous system, leading to high mortality and long-term neurological disability, especially in infants born prematurely. The results of the study show that at the root of this pathology lies a morphological and functional immaturity of the germinative matrix vessels located around the cerebral ventricles. Thinness of vascular walls, lack of collagen and elastic fibers, and slow autoregulation of cerebral circulation are important factors in the development of blood clotting.

As a result of intraventricular blood transfusion, deep and multistage morphological changes in brain structures occur. At the macroscopic level, there are cases of enlargement of the cerebral ventricles, damage to the ependymal layer and cerebral tumors. And microscopic examinations reveal massive hemorrhages in the germinative matrix, degenerative and necrotic changes of neural and glial cells, an excess of macrophages saturated with hemosiderin. These morphological processes disrupt neural migration and normal maturation of brain tissue.

Reactive gliosis, microglial activity, and fibrosis processes that develop in the post-blood period provoke irreversible structural changes in brain tissue. In particular, posthemorrhagic hydrocephalus, which occurs as a result of liquodynamic disorders, leads to suppression of the cerebral parenchyma and secondary ischemic injuries. Periventricular leukomalacia, on the other hand, is a major morphological substrate of cerebral palsy and severe psychomotor developmental disorders in children, which is accompanied by the breakdown of white matter.

The morphological consequences of neonatal intraventricular hemorrhage directly determine its clinical course and prognosis. As blood clots increase, the depth of brain damage also increases, dramatically increasing the risk of developing epileptic seizures, reduced cognitive function, and permanent neurological defects. Therefore, early detection of morphological changes and their harmonious assessment with clinical signs are of important diagnostic and prognostic value in neonatology and pediatric practice.

In conclusion, neonatal intraventricular hemorrhage is a pathology with profound and long-term negative effects on brain development, and an in-depth study of its morphological consequences serves as an important scientific and practical basis for the development of early diagnosis, effective prevention and rehabilitation strategies. Comprehensive morphological and clinical studies conducted in this direction will serve to reduce neonatal neurological disability in the future.

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