

MORPHOLOGICAL CHARACTERISTICS OF PATHOMORPHOLOGICAL CHANGES DEVELOPING IN CHILDREN FOLLOWING RESUSCITATION PROCEDURES

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ABSTRACT: This article examines the morphological characteristics of pathomorphological changes that may develop in children following resuscitation procedures. Although resuscitation is aimed at restoring vital physiological functions, the periods of hypoxia, ischemia, and subsequent reperfusion may cause secondary structural damage to tissues and organs. Particular attention is given to morphological changes in the central nervous system, heart, lungs, liver, and kidneys, which are highly sensitive to oxygen deprivation and circulatory disturbances. The development of cellular edema, degenerative and dystrophic changes, microcirculatory disorders, vascular congestion, and, in severe cases, necrotic processes is discussed. The severity of these changes depends on the duration and depth of hypoxia, the underlying clinical condition, the child's age, and the effectiveness and duration of resuscitation. Understanding the pathomorphological mechanisms of post-resuscitation injury is important for assessing the severity of organ damage, predicting possible complications, and improving approaches to post-resuscitation management and rehabilitation in pediatric patients.

Keywords: children, resuscitation, post-resuscitation period, pathomorphological changes, hypoxia, ischemia, reperfusion injury,

cellular damage, oxidative stress, microcirculation, tissue edema, necrosis, central nervous system, internal organs, morphological changes.

Resuscitation procedures in pediatric patients are aimed at restoring adequate respiratory and circulatory functions and preventing irreversible damage to vital organs. However, the restoration of spontaneous circulation and oxygen delivery does not always result in immediate normalization of tissue function. A period of oxygen deprivation followed by restoration of blood flow may initiate a complex cascade of cellular and vascular alterations. These processes form an important morphological basis of post-resuscitation syndrome and may affect several organs simultaneously. One of the principal mechanisms underlying post-resuscitation pathomorphological changes is hypoxic-ischemic injury. During cardiac or respiratory arrest, the reduction of tissue perfusion leads to inadequate oxygen and substrate delivery to cells. The central nervous system is particularly vulnerable because neuronal cells have a high metabolic demand and limited tolerance to oxygen deprivation. At the morphological level, hypoxic-ischemic injury may manifest as cellular swelling, cytoplasmic eosinophilia, nuclear alterations, neuronal degeneration, and disturbances of the microvascular bed. In severe and prolonged cases, irreversible neuronal injury and necrotic changes may develop. The brain is therefore considered one of the most important target organs during the post-resuscitation period. Cerebral edema, vascular congestion, perivascular edema, and disturbances of cerebral microcirculation may contribute to secondary neurological injury. Reperfusion of previously ischemic brain tissue can additionally increase oxidative stress and inflammatory responses. The

accumulation of reactive oxygen species may damage cellular membranes, mitochondria, and intracellular proteins, thereby aggravating neuronal dysfunction and structural injury. The cardiovascular system also undergoes significant changes after resuscitation. Temporary myocardial hypoxia and ischemia may impair the structural and functional integrity of cardiomyocytes. Morphologically, myocardial tissue may demonstrate cellular swelling, granular or vacuolar degeneration, interstitial edema, and microcirculatory disturbances. In more severe cases, prolonged ischemia and reperfusion may result in irreversible myocardial injury.

CONCLUSION

Post-resuscitation pathomorphological changes in children represent a complex process associated with hypoxia, ischemia-reperfusion injury, oxidative stress, microcirculatory disturbances, and cellular metabolic dysfunction. The central nervous system, heart, lungs, kidneys, and liver are among the organs most susceptible to structural damage following severe oxygen deprivation and circulatory failure. The morphological manifestations of post-resuscitation injury may include cellular and tissue edema, vascular congestion, degenerative and dystrophic changes, microcirculatory disorders, and, in severe cases, irreversible necrotic processes. The severity and distribution of these changes depend on the duration and depth of hypoxia, the underlying clinical condition, the child's age, and the effectiveness of resuscitation measures. A detailed understanding of the morphological mechanisms underlying post-resuscitation injury is important for assessing the extent of organ damage and predicting possible complications. Early recognition and prevention of secondary ischemic and reperfusion-associated injury may contribute to

improved outcomes in pediatric patients. Therefore, further clinicopathological and morphological research is necessary to clarify the mechanisms of post-resuscitation tissue damage and to develop more effective approaches to pediatric post-resuscitation care and rehabilitation.

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