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MORPHOLOGICAL BASIS OF VASCULAR DYSFUNCTION ASSOCIATED WITH VASOPRESSOR USE IN INTENSIVE CARE

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Abstract

Vasopressors (norepinephrine, vasopressin, phenylephrine, epinephrine) remain the cornerstone of maintaining perfusion of vital organs in septic, cardiogenic, and hypovolemic shock. However, prolonged or high-dose vasopressor therapy is frequently associated with the development of refractory vasoplegia, ischemia–reperfusion organ injury, and multiple organ dysfunction.

The obtained data substantiate the necessity for early monitoring of endothelial function, timely reduction of vasopressor doses upon achieving target perfusion parameters, and the search for adjuvant strategies aimed at protecting the vascular wall, including low-dose corticosteroids, inducible nitric oxide synthase (iNOS) inhibitors, and agents that restore the endothelial glycocalyx.

Keywords

Vasopressors, norepinephrine, vasopressin, endothelial dysfunction, glycocalyx, refractory shock, sepsis, intensive care, vascular morphology, endothelial apoptosis, nitrosative stress.

Relevance

Vasopressor therapy remains the primary and often the only means of maintaining adequate organ perfusion in refractory shock of various etiologies, including septic, cardiogenic, hemorrhagic, and neurogenic shock. According to international registries (2023–2025), more than 60% of patients with septic shock and up to 90% of patients with stage III–IV cardiogenic shock receive vasopressors within the first 48 hours of admission to the intensive care unit.

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At the same time, the average duration of vasopressor support has increased from 2–3 days in 2010 to 5–9 days at present, while cumulative doses of norepinephrine often exceed 1–2 µg/kg/min for several consecutive days.

Aim of the Study

To systematically investigate morphological and ultrastructural changes of the vascular wall (endothelium, glycocalyx, smooth muscle layer, and adventitia) in arterioles and muscular- and elastic-type arteries in patients and experimental models during prolonged vasopressor therapy under critical conditions, and to establish a causal relationship between the severity of these changes and the development of refractory vasoplegia, multiple organ failure, and mortality.

Conclusion

The conducted comprehensive experimental and clinical study made it possible, for the first time, to demonstrate that prolonged (more than 72–96 hours) and/or high-dose use of vasopressors (norepinephrine, vasopressin, and their combinations) leads to direct, morphologically verified, and largely irreversible damage to all layers of the vascular wall, including the endothelium, glycocalyx, smooth muscle layer, and adventitia.

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