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INTENSIVE IN THERAPY VASOPRESSORS WHEN USED BLOOD IN THE VEINS MORPHOLOGICAL CHANGES

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Annotation: Last in years sepsis severe arterial hypotension , cardiogenic and distributive shock in cases vasopressors (noradrenaline, vasopressin , dopamine , phenylephrine) and others) life save stay for first in line is being used . Long term and high in doses vasopressor infusion endothelial in cells , smooth muscle on the floor and vein of the wall in the structure reversible and irreversible morphological to changes take arrival This is defined . in the thesis vasopressors vein to the wall impact mechanisms , histological and ultrastructure changes , as well as this of changes clinical consequences analysis will be done .

Keywords : vasopressors , noradrenaline, arterial hypotension , endothelial dysfunction , vein wall remodeling , smooth muscle cells apoptosis , intensive therapy , sepsis shock

RELEVANCE

By world every 30 million per year with excess sepsis illness record I am dying . level 25–50 % organization will reach .

Surviving Sepsis Campaign 2021 Noradrenaline is the first in the instructions . choice vasopressor as recommendation done although , its far short-term (from 24–72 hours) more than) and high Use in doses (>0.5–1 μg /kg/min) vein on the wall irreversible fibrosis , endothelial denudation and microcirculator stream to the violation reason to be possible .

Current on the day vasopressor-induced vein injury according to randomized research number very few , animals and in vitro models taken information and clinical to practice complete extrapolation as It won't be . This because of of the subject scientific and clinical relevance high .

PURPOSE

Intensive therapy under the circumstances different vasopressor blood of drugs (noradrenaline , vasopressin , dopamine , phenylephrine) veins in the wall (arteries , arterioles , venules) releasing morphological and ultrastructure changes analysis to do , their dose and duration with dependence determination and clinical consequences assessment .

To further specify and deepen the main purpose above, the following can be highlighted. Elucidating Mechanisms - To investigate the direct and indirect mechanisms of action of vasopressors on the structural components of the vascular wall (endothelium, smooth muscle, basal membrane), such as oxidative stress, induction of apoptosis, and loss of mitochondrial function.

Studying Dose-Time Dependency - To demonstrate, based on experimental and clinical data, the clear relationship between the onset and severity of morphological changes (reversible vs. irreversible) and the parameters of vasopressor infusion—specifically dose (maximum dose, average dose) and total duration (24 hours, 72 hours, 7 days).

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Comparing Drug Types - To identify differences in the vascular effects of various vasopressors (e.g., pure alpha-agonist phenylephrine vs. catecholamines vs. vasopressin) and compare their risk profiles.
Linking to Clinical Significance - To logically substantiate how these vascular wall alterations impact microcirculatory dysfunction, loss of responsiveness to vasopressors (refractory shock), the development of multiple organ dysfunction syndrome, and even long-term patient outcomes.
Establishing a Basis for Protective Strategies - Based on the findings, to scientifically justify potential strategies for protecting the vascular wall from the adverse effects of vasopressors (vasoprotection), such as early adjunctive therapy, rapid dose de-escalation, and the search for novel protective agents.

CONCLUSION

Far term and high in doses vasopressors blood veins on the wall noticeable morphological changes (endothelial denudation , smooth muscle apoptosis , fibrosis) it comes , this and organ perfusion far term violation and many members shortage in development important role to play possible . Vasopressor dose possible as much as possible fast reduction , second row early administration of drugs (vasopressin , angiotensin II) add and vein the wall protection to do strategies working exit current is a task . The severity of vascular injury demonstrates a clear dose- and time-dependent relationship with vasopressor exposure, particularly during continuous infusions exceeding 72 hours or at doses above recommended thresholds. Such changes may partially explain the development of refractory shock states, loss of vasopressor responsiveness, and progression to multiple organ dysfunction syndrome.
These findings emphasize the importance of rational vasopressor use in intensive therapy, including early achievement of target mean arterial pressure, prompt dose de-escalation when clinically feasible, and avoidance of unnecessarily prolonged high-dose infusions. The early introduction of adjunctive vasoactive agents (such as vasopressin or angiotensin II) may reduce catecholamine burden and mitigate vascular wall injury.

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