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Vasoplegic Syndrome

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Syndrome

Cardiopulmonary Bypass

Vasoplegic syndrome has been associated with a combination of endothelial injury, arginine-vasopressin system dysfunction, release of other vasodilatory mediators, and a muscle vasodilatory response (Sharkey, 2014).

This increases capillary permeability causes interstitial edema and reduced intravascular volume.

All leading to decreased volume and increasing NO levels further dilating arterial smooth muscle.

An efflux of potassium through ATP sensitive channels results in hyperpolarization of the cell causing inactivation of voltage gated calcium channels which cause further vasodilation and vascular dysfunction.

NO then causes dephosphorylation of the myosin light chain by increasing production of cyclic GMP which prevents muscle contraction by limiting actin and myosin interaction (Omar, Zedan, & Nugent, 2015).

Significance of Vasoplegia

Vasoplegic patients are frequently seen in patients undergoing cardiothoracic bypass (CPB) or in long OR cases.

Vasoplegic syndrome is a loss of vasomotor tone and a medical emergency (Sheaff, et al, 2018).

It causes severe hypotension and hypoperfusion to vital organs (Abou-Arab, et al, 2018).

Generally seen in post cardiac surgery patients or during shock (Sheaff, et al, 2018).

Requires very high dose pressers and inotropic support.

May be non-response to medications or fluids and require further escalation of care.

Why does it matter?

As a CVICU nurse vasoplegia is frequently seen in patients undergoing cardiothoracic bypass (CPB) or in long OR cases.

Vasoplegia is difficult to manage and requires excellent nursing and physician knowledge and attention.

Vasoplegia has a high mortality rate and is not well known.

It is common in most shock states in intensive care patients.

CRNAs will experience vasoplegia and need to be familiar with it both in post-op patients and acute shock states emergencies.

Underlying Pathophysiology

Contact with the CPB circuit immediately absorbs plasma proteins into the bioremediators and directly activates the kinins, complement, and clotting pathways.

The kinin pathway produces bradykinin and kallikrein which lead to neutrophil activation.

Both the intrinsic and extrinsic clotting cascades produce thrombin which results in fibrin deposits at the catheterization site.

Thrombin goes on to activate platelets which adhere to other platelets, neutrophils, and exposed basement membranes.

Vasoplegic

The complement pathway leads to formation of C5a which further activates neutrophils.

These neutrophils when activated release enzymes and reactive oxygen species (ROS) that adhere to membrane surfaces and to endothelial surfaces.

Multiple factors, including thrombin, C5a, and cytokines, activate endothelial cells that produce reactive substances, including nitric oxide (NO) and prostacyclin, and express surface receptors (Omar, Zedan, & Nugent, 2015).

Following the acute response to CPB the inflammatory response is exacerbated by reinfusion of the blood lost during surgery.

The reinfused blood contains hemolysed erythrocytes and macroaggregates such as denatured proteins, fat globules and platelet and leukocyte aggregates.

These fragments clog small capillaries further stimulating inflammation.

Additionally, reproduction syndrome of the heart and lungs causes neutrophil adherence and further ROS release which causes direct protein, lipid, and nucleic acid damage.

This increases capillary permeability causes interstitial edema and reduced intravascular volume.

All leading to decreased volume and increasing NO levels further dilating arterial smooth muscle.

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Implication for Nursing Care

Understanding the risk factors along with the signs and symptoms of vasoplegic syndrome is essential importance to APNs.

Nurses play a vital role in identifying hypotension unresponsive to treatment.

Severe hypotension is a medical emergency and nurses at the bedside should be prepared to take further interventions to maintain tissue oxygenation such as fluids, oxygen, ventilatory support, and vasoactive medications.

Identification of possible high risk patients by CRNAs in post-op and post-shock should have close observation and constant blood pressure monitoring.

Nursing should be prepared for end of life discussions and care, should interventions not succeed.

Conclusion

Understanding factors that lead to vasoplegia can help prevent and treat it.

Multiple factors play a role in loss of vasomotor tone all of which lead to low end organ perfusion.

Refractive to normal hypotensive treatments such as fluid and catecholamines.

Goals of therapy include: restoring MAP, maintaining adequate cardiac output, and restoring tissue perfusion (Sharkey, 2014).

Prevention, assessment, and early treatment are all of great importance to decrease mortality and morbidity.

References


