Summer 2018

Vasoplegic Syndrome

Monica Arce
arce1@otterbein.edu

Vasoplegic Syndrome

Monica Arcé BSN-RN, CCRN
Otterbein University, Westerville, Ohio

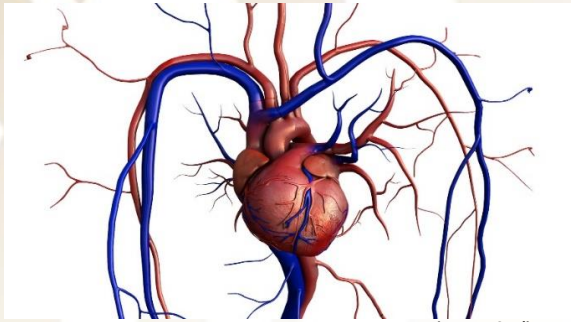
Introduction

What is Vasoplegic Syndrome?

- Vasoplegic syndrome is a loss of vasomotor tone and a medical emergency (Shaefi, 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 (Shaefi, et al., 2018)
- Requires very high dose pressors 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 cardiopulmonary 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 some shock state emergencies



Picture 1. Cardiovascular system
(Consultants, 2017)

Presentation of Process

Risk Factors

- Cardiopulmonary Bypass
- Blood transfusion
- Organ transplantation
- Sepsis
- Shock States
- Extended OR cases

(Liu, Yu, Yang, & Green, 2017)

Signs and Symptoms

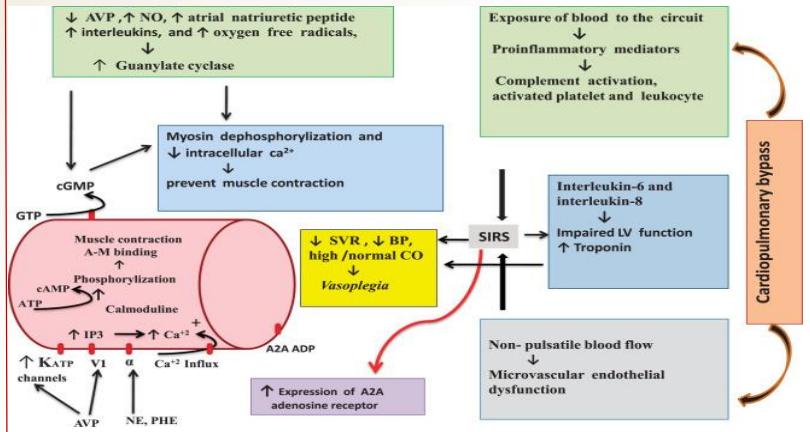
- Significant arterial hypotension
  - MAP <50
  - Without clear identifiable cause
  - Refractory to traditional treatment
- Normal or high cardiac output
- Low systemic vascular resistance
- Unresponsive to fluid therapy
- Unresponsive to catecholamine treatment

(Liu, Yu, Yang, & Green, 2017)

Underlying Pathophysiology

- Contact with the CPB circuit immediately absorbs plasma proteins into the biomembranes and directly activates the kinin, complement, and clotting pathways
- The kinin pathway produces bradykinin and kallikren which lead to neutrophil activation
- Both the intrinsic and extrinsic clotting cascades produce thrombin which results in fibrin deposits
- Thrombin goes on to activate platelets which adhere to other platelets, neutrophils, and exposed basement membranes
- 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 vasoactive substances, including nitric oxide (NO) and prostacyclin, and express surface receptors." (Omar, Zedan, & Nugent, 2015)
- Following the acute responses to CPB the inflammatory response is exacerbated by reinfusion of the blood lost during surgery
- The reinfused blood contains hemolyzed erythrocytes and macroaggregates such as denatured proteins, fat globules and platelet and leukocyte aggregates
- These fragments clog small capillaries further stimulating inflammation
- Additionally, reprofusion 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
- An efflux of potassium through ATP sensitive channels results in hyperpolarization of the cell causing inactivation of voltage gated calcium channels which causes further vasodilation and vascular disfunction
- 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)



Picture 2. Visual representation of the pathophysiology involved in vasoplegic syndrome
(Omar, Zedan, & Nugent, 2015)

Significance of Pathophysiology

- Severe, persistent, refractory hypotension has a high morbidity and mortality (Lambden, Creagh-Brown, Hunt, Summers, & Forni, 2018)
- Multifactorial and difficult to manage
- "Vasoplegic syndrome has been attributed to a combination of endothelial injury, arginine-vasopressin system dysfunction, release of other vasodilatory inflammatory mediators, and a muscle hyperpolarization." (Sharawy, 2014)
- Without appropriate systemic vascular resistance even a high cardiac output has no where to go
- Leads to hypoperfusion of organ and organ systems which in turn leads to end organ failures (Abou-Arab, et al., 2018)
- Understanding the pathophysiology of vasoplegia is of vital importance to promptly treat the cause and maintain adequate oxygenation to tissues

Treatments

- Vasopressors (high dose)
  - Norepinephrine
  - Vasopressin
  - Phenylephrine
- Methylene Blue
- β_1 blockade
- α_2 agonist
- Glucocorticoids
- Blood Products
- Isotonic Fluids

(Levy, et al., 2018)

(Shaefi, et al., 2018)

Picture 3. Methane Blue used to inhibit NO synthesis
("Singin' the blues: What can you do with methylene blue?")

Implication for Nursing Care

- Understanding the risk factors along with the signs and symptoms of vasoplegic syndrome is of upmost importance to APNs
- Nurses play a vital role in identifying hypotension unresponsive to interventions
- 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 CRNA's in pre-op and post-op 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 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 (Sharawy, 2014)
- Prevention, assessment, and early treatment are all of great importance to decrease morbidity and mortality

References

Abou-Arab, O., Martineau, L., Bar, S., Huette, P., Amar, A. B., Caus, T., ... & Lorne, E. (2018). Postoperative Vasoplegic Syndrome Is Associated With Impaired Endothelial Vasomotor Response in Cardiac Surgery: A Prospective, Observational Study. Journal of cardiothoracic and vascular anesthesia.

Consultants, C. (2017, June 23). Transcatheter Aortic Valve Replacement (TAVR) Surgery | Cardiovascular Consultants, Ltd. Retrieved from http://cvcheart.com/transcatheter-aortic-valve-replacement/

Lambden, S., Creagh-Brown, B. C., Hunt, J., Summers, C., & Forni, L. G. (2018). Definitions and pathophysiology of vasoplegic shock. Critical Care, 22(1). doi:10.1186/s13054-018-2102-1

Levy, B., Fritz, C., Tahon, E., Jacquot, A., Auchet, T., & Kimmoun, A. (2018). Vasoplegia treatments: the past, the present, and the future. Critical Care, 22(1), 52.

Liu, H., Yu, L., Yang, L., & Green, M. S. (2017). Vasoplegic syndrome: An update on perioperative considerations. Journal of clinical anesthesia, 40, 63-71.

Omar, S., Zedan, A., & Nugent, K. (2015). Cardiac vasoplegia syndrome: pathophysiology, risk factors and treatment. The American journal of the medical sciences, 349(1), 80-88.

Shaefi, S., Mittel, A., Klick, J., Evans, A., Ivascu, N. S., Gutsche, J., & Augoustides, J. G. (2017). Vasoplegia following Cardiovascular Procedures–Pathophysiology and Targeted Therapy. Journal of cardiothoracic and vascular anesthesia

Sharawy, N. (2014). Vasoplegia in septic shock: Do we really fight the right enemy? Journal of Critical Care, 29(1), 83-87. doi:10.1016/j.jcrc.2013.08.021

Singin' the blues: What can you do with methylene blue? (n.d.). Retrieved from http://sinaiem.org/singin-the-blues-what-can-you-do-with-methylene-blue/

Sterling SA, Puskarich MA, Shapiro NI, Trzeciak S, Kline JA, Summers RL, Jones AE. Characteristics and outcomes of patients with vasoplegic versus tissue dysoxic septic shock. Shock. 2013;40(1):11.

OTTERBEIN UNIVERSITY