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Mini Review

Hemodynamic Coherence and the Microcirculation

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Received: July 02, 2019; **Published:** October 21, 2019

Abstract

Microcirculation regulates the blood flow distribution within organs to balance oxygen delivery and demands. Microvascular alterations play a crucial role in its pathophysiology and results in organ dysfunction. Hemodynamic coherence must be maintained to expect the restoration of microcirculation through systemic driven resuscitation during shock.

Keywords: *Microcirculation; Hemodynamic Coherence; Hemorrhagic Shock; Microvascular Resuscitation*

Introduction

Microcirculation is the functional system that can promptly respond to the tissue changes and metabolic demand by regulating the blood flow to tissues, ensuring adequate oxygen delivery to meet the oxygen demand of the cells. It is composed of microvascular network of arterioles, capillaries, venules and microlymphatics. Alterations in this network affects all components of the microcirculation which are represented as targets of proinflammatory activity of cytokines leading to microcirculatory dysfunction [1-3].

Hemodynamic coherence also referred as macro-microvascular coupling describes a physiological condition in which macro hemodynamics values e.g. mean arterial pressure (MAP), cardiac output, systemic haemoglobin and/or oxygen delivery reflects microvascular perfusion [4]. In conditions of hemodynamic instability like sepsis, shock, cardiac failure, systemic inflammation or post cardiotomy syndrome following cardiac surgery this coupling may be disturbed, resulting in deteriorated microvascular perfusion. This concept of hemodynamic coherence implies that the manipulation of systemic hemodynamics through administration of volume, vasoactive agents and erythrocytes to achieve targeted hemodynamic end points, results in improved microvascular blood flow and thus in the correction of oxygen delivery and consumption mismatch within different organs and their cells [5]. This correlation between hemodynamic coherence and microcirculation must be maintained to expect the restoration of microcirculation through systemic hemodynamic driven resuscitation and might be influenced by not only the underlying pathophysiology, but also by the respective treatment applied.

Severity of microcirculatory abnormalities and their persistence over time are associated with organ dysfunction and increased mortality so that early recognition and therapeutic strategies for its improvement are becoming part of the complex treatment in such conditions.

Microcirculation and hemorrhagic shock

The local regulation of the arteriolar tone is a crucial factor in the microvascular matching of oxygen supply to oxygen demand. The key components of microcirculation regulating the microvascular blood flow are the endothelium and its luminal cover of glycosaminoglycan-containing layer called glycocalyx, which modulates the vasomotor tone. It also balance the microvascular fibrinolysis and thrombosis, promoting leukocyte migration and adhesion. The endothelial cells acts as metabolic sensors and conduct signals along the endothelium through "cell to cell" communication. During haemorrhagic shock and sepsis, there is macrovascular redistribution of the arterial blood flow at the expense of non vital organs and simultaneously blood flow is redistributed within the capillary networks of each organ by arteriolar tone and oxygen demand. This microvascular heterogeneity of blood flow is an essential property of normal microcirculation.

In haemorrahagic shock and hypoxic stimulus , the endothelial cells respond by promoting local release of nitric oxide (NO) through endothelial NO synthetase (eNOS) and prostacyclin (PGI_2) through prostaglandin endoperoxide H_2 synthetase-1 (PGHS-1), subsequently increasing cyclic guanosine 3′,5′-monophosphate (cGMP) and cyclic adenosine 3′,5′monophosphate (cAMP) respectively, in the arteriolar smooth muscle cells. This mechanism induces smooth muscle relaxation, microvascular dilatation and maintenance of an adequate microvascular blood flow. An additional feedback mechanism is provided by erythrocytes and haemoglobin molecule. Erythrocytes behaves as mobile oxygen sensors and under hypoxic conditions, haemoglobin undergoes structural changes and erythrocytes promotes release of adenosine triphosphate (ATP) and S-nitrosothiol (NO donor). The latter is converted to NO by deoxyhemoglobin thus causing vasodilation. Erythrocytes while passing through a hypoxic zone are able to induce vasodilation in a retrograde fashion, resulting in increase in microvascular blood flow [1-2,6].

Hemodynamic incoherence

The overwhelming inflammation induces functional and structural changes in the endothelium, glycocalyx, vascular smooth muscle cells and blood cells thus leading to release of endogenous factors by the activated immune cells or release from necrotic cells contributing to microvascular dysfunction. Severity of glycocalyx degradation and elevated plasma circulating levels of heparin sulfate and hyaluronic acid, correlates with mortality in septic shock. The alterations that are implicated in hemodynamic incoherence are the opening of pathological shunts , leading to increased systemic blood flow from arterioles to venules and shunting of blood vessels without passing through the microcirculation. Thus, microcirculatory PO_2 becomes lower than venous PO_2. Secondly, reduction of perfused vessel density leads to an increase in blood flow in the capillaries that are already perfused and thirdly, flow diversion of blood flow occurs from smaller to larger vessels in which normal flow is still present. These microvascular alterations explains the development of tissue hypoxia occurring despite of adequate resuscitation of fluid and maintained systemic hemodynamic parameters [7].

Implications for resuscitation

It is postulated that early phase of shock and sepsis is accompanied by an intact hemodynamic coherence between macro and micro circulation and infact fluid resuscitation within the first 24 hours of sepsis effectively improves microcirculation. On the other hand, it is well known that even if macrocirculation is optimized, organ function is severely compromised. Therefore, surrogates of organ perfusion such as lactate, peripheral temperature and capillary refill time, venous to arterial carbon dioxide difference ($Pv\text{-}PaCO_2$) have been incorporated in recommended treatment algorithms but they still do not suggest microvascular hypoperfusion. The highly sensitive and specific bedside test to evaluate low capillary density and impaired flow is microvascular flow index (MFI) and total vascular density. Recently new technology contrast enhanced ultrasound (CEUS) has been validated to assess microcirculation in several organs.

The catecholamine currently recommended for the treatment of distributive shock is norepinephrine but accumulating evidence showed that high doses are associated with negative patient outcome in shock. Therefore use of potent vasoconstrictors, non-adrenergic treatment strategies such as vasopressin and terlipressin is increasing rapidly. Although vasopressin seems to be more superior than terlipressin, detrimental effects of vasopressin have been described if administered as bolus or high doses in hypovolemic patients. Regardless of the type of vasoconstrictors, it has been reported that increase in mean arterial pressure (MAP), does not improve preexisting microvascular dysfunction with use of these agents [8].

Similarly use of β adrenergic agents, dopamine and dobutamine have failed to demonstrate improvement in microcirculation. Stronger vasodilatory compounds like levosimendan is more effective than dobutamine in improving oxygen delivery at microcirculation level [10]. Furthermore, simultaneous administration with norepinephrine may counteract the reduced density in the microvascular zone in which norepinephrine causes excessive vasoconstriction [9,10].

Conclusion

Hemodynamic coherence must be maintained for restoration of microcirculation through systemic hemodynamic driven resuscitation. Microcirculatory dysfunction are mainly related to factors like volume status, timing, hemodynamics and progress of the disease and leads to loss of hemodynamic coherence. Occult microvascular hypoperfusion should be diagnosed early on, to improve outcomes in these patients.

Bibliography

1. Miranda M., *et al*. "Microcirculatory dysfunction in sepsis: pathophysiology, clinical monitoring, and potential therapies". *American Journal of Physiology-Heart and Circulatory Physiology* 311.1 (2016): H24-H35.
2. Ellis G., *et al*. "The microcirculation as a functional system". *Critical Care* 9.4 (2005): 3-8.
3. Aird C. "Endothelium as an organ system". *Critical Care Medicine* 32.5 (2004): 271-279.
4. Ince C. "Hemodynamic coherence and the rationale for monitoring the microcirculation". *Critical Care* 19.3 (2015): S8.
5. Dellinger P., *et al*. "Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock 2012". *Intensive Care Medicine* 39.2 (2013): 165-228.
6. Cepinskas G., *et al*. "Inflammatory response in microvascular endothelium in sepsis: role of oxidants". *Journal of Clinical Biochemistry and Nutrition* 42.3 (2008): 175-184.
7. Nelson A., *et al*. "Circulating glycosaminoglycan species in septic shock". *Acta Anaesthesiologica Scandinavica* 58.1 (2014): 36-43.
8. Torgersen C., *et al*. "Comparing two different arginine vasopressin doses in advanced vasodilatory shock: a randomized, controlled, open-label trial". *Intensive Care Medicine* 36.1 (2010): 57-65.
9. Hernandez G., *et al*. "Effects of dobutamine on systemic, regional and microcirculatory perfusion parameters in septic shock: a randomized, placebo-controlled, double-blind, crossover study". *Intensive Care Medicine* 39.8 (2013): 1435-1443.
10. Morelli A., *et al*. "Levosimendan for resuscitating the microcirculation in patients with septic shock: a randomized controlled study". *Critical Care* 14.6 (2010): R232.

Volume 5 Issue 11 November 2019