New approach in managing patients with septic shock

Sepsis remains the most common cause of vasodilatory shock worldwide. The mainstay of haemodynamic treatment of septic shock is fluid resuscitation followed by vasopressors where fluids alone are insufficient to achieve target blood pressure.

This article, published in the journal Critical Care, proposes the concept of “broad spectrum vasopressors” as a new approach to the initial management of septic shock. Based on this strategy, patients with septic shock are started on multiple vasopressors with a different mechanism of action simultaneously while the vasopressor sensitivity is assessed.

Vasopressor sensitivity, the article says, could be assessed by sequential removal of vasopressors or developing a vasopressor sensitivity panel. Once the vasopressor sensitivities are assessed, then the vasopressors are de-escalated accordingly.

This concept, the article explains, is in keeping with current antimicrobial therapy paradigm wherein clinicians obtain cultures and start broad-spectrum antibiotics with the intention of de-escalating the antibiotics once the causative organism is identified.

Norepinephrine, a catecholamine, is the first-line vasopressor used worldwide but given that all routinely used catecholamines target the same adrenergic receptors, many clinicians may add a non-catecholamine vasopressor where refractory hypotension due to septic shock is present. Of note, the timing of this additional intervention is variable. This decision is based on three key factors: availability, familiarity, and safety profile.

Another factor must be considered, according to the article, and this is the potential vasopressor response – because following appropriate volume resuscitation, the response to different vasopressor classes is neither uniform nor predictable. Critically ill patients who are non-responders to high-dose catecholamines have a dismal outcome.

Similarly, patients have a variable response to non-catecholamine agents including vasopressin and angiotensin II: but where patients exhibit a blood pressure response the outcomes are improved over non-responders. The ATHOS-3 study, for instance, reported that a responder’s chance of survival is significantly better than that of patients who fail to respond to angiotensin II.

It follows that in patients with septic shock, the article points out, the choice of vasopressor should be governed by the patient’s likelihood of responding and the sensitivity to treatment.

However, a key hurdle to implementing the “broad spectrum vasopressors” strategy is that there is currently no bedside test that predicts the blood pressure response to catecholamines, vasopressin, or angiotensin II. Moreover, not all of these vasopressors are currently available worldwide due to either a lack of regulatory approval or cost considerations.

Despite these hurdles, the article suggests exploring this hypothesis: Does time to sensitive vasopressor response improve outcomes in septic shock? This is a question worth answering and may prove an essential approach in managing these critically ill individuals.

Source: Critical Care
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