EDITORIAL

Volemic replacement during spinal cord injury patients.

María Bermúdez¹

¹ *Servicio Anestesiología, Reanimación y Terapéutica del Dolor. Hospital Universitario Lucus Augusti. Lugo. 27003. España.*

Correspondence to:

María Bermúdez López, Servicio Anestesiología, Reanimación y Terapéutica del Dolor.
Hospital Universitario Lucus Augusti.
E-mail: majobl79@gmail.com

Conflicts of interests

Nothing to declare

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Acute traumatic spinal cord injury (SCI) is frequently associated with systemic hypotension, which may be attributable to associated traumatic injuries with hypovolemia, direct severe spinal cord trauma itself, or a combination [1][2]. At the same time, the traumatic neurogenic shock is a rare but serious complication of SCI. It is life-threatening and associates bradycardia and hypotension caused by a medullary trauma. Cardiovascular instability is a frequent complication of SCI, especially when the upper thoracic and cervical cord is involved. Sympathetic denervation results in arteriolar dilation and the interruption of cardiac sympathetic innervations (T1-T4) promotes bradycardia and reduces myocardial contractility [3]. Neurogenic

shock generally lasts from 1 to 3 weeks [3].

The principal clinical concern is to avoid secondary (irreversible) cord damage [3]. Ischemia of the spinal cord is thought to be one of the most important contributors to neuronal injury and neurological deficit after acute SCI. For that reason, treatment of hypotension and resuscitation to maintain the mean arterial pressure at high-normal levels, 85 to 90 mm hg, for the first 7 days following an SCI is highly recommended [2][4][5]. Therefore, not only the surgical management, by relieving or preventing compression of neural structures; but also, the medical optimization of cord perfusion and oxygenation, may be associated with better neurological results after SCI [3][4].

In this context it is essential the ICU monitoring which allows the early detection of

hemodynamic instability, cardiac disturbances, pulmonary dysfunction, and hypoxemia [2]. Management of SCI is directed toward limiting secondary injury and maximizing neurologic recovery [3].

The hemodynamic monitoring and consequently fluid therapy in order to maintenance of the mean arterial pressure above 85 mmHg reduces secondary complications [6]. Normally, a combination of fluid therapy with vasopressors is needed. The blood volume of patients with SCI is reduced by approximately 20% [7]. However, congestive heart failure may occur, particularly if fluids are administered injudiciously [3][8]. Therefore, the question still without answer: how much and which fluids? There is lack of studies comparing different managements.

A reasonable strategy is one in which 1)hypotension is rigorously avoided and 2)cord perfusion is optimized with volume expansion and blood pressure augmentation, so long as these interventions do not impact unfavorably on other organ systems [5]. At this point it is essential that the fluids are guided by invasive hemodynamic monitoring to avoid congestive heart failure, fluids should be titrated with particular attention to incremental changes in cardiac output and filling pressures [3]. In fact, Lehmann et al reported that episodic hypotension unrelated to hypovolemia was identified in 68% of severe cervical injured patients, requiring the use of intravenous pressors in half [9]. Therefore, the fluid management in this patients is challenging and it is mandatory the use of cardiac, hemodynamic, and respiratory monitoring devices to detect cardiovascular dysfunction in patients following acute spinal cord injury [2]. As a result, the goal-directed fluid therapy is essential in this clinical setting. Besides this, pulmonary artery catheterization or transesophageal echocardiography should be considered in the presence of severe hemodynamic compromise[2][5].

In the other hand, SCI is associated with a high prevalence of hyponatremia, which can cause various clinical symptoms and increase mortality. Various factors including traumatic brain injury with an abbreviated injury scale score ≥ 3 and higher American Spinal Injury Association impairment scale, bradycardia, vasopressors, and nosocomial pneumonia. may be associated with hyponatremia in patients with acute SCI. These factors can help clinicians identify and monitor SCI patients with a higher hyponatremia risk, and potentially help patients to avoid severe complications [10]. Consequently, hypoosmolar and glucose solutions should be avoided in the SCI.

Finally, the volemic replacement during SCI has an important impact on the neurological

outcome of these patients and deserves further study. Recommendations about hemodynamic optimization (PAM> 85 mmHg) have a potential benefit but the most appropriate threshold level of MAP and how to get it (farmacological strategy and fluid therapy) it is still unclear.

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