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Editorial

Regarding Surviving Sepsis Campaign Guideline Recommendation 1 and 2 – Something that the Guideline doesn't Tell You

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Recently, fourth Surviving Sepsis Campaign (SSC) guideline was released [1]. It is welcome because the up-to-date knowledge was translated into clinical guidance. But few things were needed to be noted.

Ambiguous Term

In Sepsis-3, sepsis and septic shock are defined as below. "Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. Organ dysfunction can be identified as an acute change in total SOFA score ≥2 points consequent to the infection." "Septic shock is a subset of sepsis in which underlying circulatory and cellular/metabolic abnormalities are profound enough to substantially increase mortality. Patients with septic shock can be identified with a clinical construct of sepsis with persisting hypotension requiring vasopressors to maintain MAP ≥65 mm Hg and having a serum lactate level >2 mmol/L (18 mg/dL) despite adequate volume resuscitation. With these criteria, hospital mortality is in excess of 40%." [2]. In the most recent Survival Sepsis Campaign (SSC) guideline, first and second recommendations are as below [1]. "1. Sepsis and septic shock are medical emergencies, and we recommend that treatment and resuscitation begin immediately (Best practice statement, BPS)." "2. We recommend that, in the resuscitation from sepsis-induced hypoperfusion, at least 30 mL/kg of IV crystalloid fluid be given within the first 3 hours (strong recommendation, low quality of evidence)." Therefore, oddly, we encounter three distinctly used terms in the SSC guideline - sepsis, septic shock, and sepsis-induced hypoperfusion. In the same SSC guideline, sepsis –induce hypoperfusionis further described as below. "Sepsis-induced hypoperfusion may be manifested by acute organ dysfunction and/or ± decreased blood pressure and increased serum lactate."

Even there is no further detailed explanation, acute organ dysfunction is thought to indicate sepsis condition, and thus it will be related to acute SOFA score change of 2 or more. And, the phrase of 'decreased blood pressure and increased serum lactate' is almost identical with that of the septic shock identification criteria. Then, sepsis-induced hypoperfusion is clinically regarded as acute SOFA score change of 2 or more and/or ± MAP ≥65 mm Hg and serum lactate level >2 mmol/L. That means sepsis-induced hypoperfusion is a sort of time-point identifiable criteria. Among sepsis-induced hypoperfusion patients, if adequate volume resuscitation fails, the patients will require vasopressors support and the diagnosis of septic shock will be established. Thus, it seems that substantial overlap exists among septic shock and sepsis-induced hypoperfusion, and further clear demarcation would be necessary between these two terms.

Fluid Resuscitation

First and second recommendation of the SSC guideline means that emergent treatment for sepsis-induced hypoperfusion is a fluid resuscitation. It implied that sepsis-induced hypoperfusion is accompanied with a considerable degree of hypovolemia. Although sepsis is regarded as a distributive shock, not hypovolemic shock in traditional classification, it makes sense that intravascular volume depletion occurs in sepsis and septic shock. According to a theme evolved for sepsis over the past 30 years, local vasodilation and a vascular leak is the initial process of the sepsis pathophysiology [3]. Subsequently, intravascular fluid depletion follows and this alone can result in substantial hypoperfusion to body organs in sepsis and septic shock. Additionally, sepsis patients were likely to eat water and food less generally. Also, some sepsis patients would lose in-body fluid through diarrhea, rhinorrhea, productive cough, diaphoresis, or even tachypnea. Therefore, it is deemed that intravascular volume depletion owing to not only distributive type as well as absolute loss exists among sepsis patients. Thus, emergent fluid resuscitation is rational and needed empirically. The next point is that how to use fluid.

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Recommended volume is 30 ml/kg of crystalloid within the first 3 hours. Recent interventional studies have described this as usual practice in the early stages of resuscitation, and observational evidence supports the practice [4,5]. The average volume of fluid pre-randomization given in the PROCESS and ARISE trials was approximately 30 mL/kg, and approximately 2 liters in the PROMISE trial [6-8]. Thus, it seems that 30 ml/kg would be enough, but the volume is not all to consider.

It is because that the administration rate can be subdivided. 30 ml/kg within the first 3 hours is same as 10 ml/kg within 1 hour + 10 ml/kg within next 1 hour + 10 ml/kg within next 1 hour [30 ml/kg/3hr = 10 ml/kg/1hr + 10 ml/kg/1hr + 10 ml/kg/1hr]. However, even using 22G 25 mm IV cannular, the rate of flow with gravity is approximately 30 ml/min, it means that 1.8 L/hour [9]. So, recommended flow rate is easily achievable in real practice. If fluid resuscitation is an emergent treatment, the faster rate should be considered if available, i.e., faster than 10 ml/kg/hr within first 30 minutes or 1 hour, and then followed by slower rate. For example, 10 ml/kg within 30 minutes + 20 ml/kg within next 2.5 hours [30 ml/kg/3hr = 10 ml/kg/0.5hr + 20 ml/kg/2.5 hr]. Or 20 ml/kg within 1 hour + 10 ml/kg within next 2 hours can be considered [20 ml/kg/1hr + 10 ml/kg/2hr]. This dichotomized (or more) approach for administration rate enables more easily the usage of two crystalloid fluids - normal saline and balanced crystalloid. It is well known that chloride rich solution (which is normal saline) is harmful and avoidance of that solution is recommended as available. However, the electrolyte status, particularly serum potassium concentration, are unknown in sepsis patients at the presentation time, many of physicians hesitate the use of balanced crystalloid until the laboratory test reveals no hyperkalemia. Actually, intracellular potassium concentration would be decreased after balanced crystalloid infusion unless the intravascular potassium concentration is not bigger than that of the balanced crystalloid. But a concern still exists that the total potassium in the body will be increased under acute kidney injury because there may be inconsistence between input and output of the potassium. For this reason, normal saline 30 ml/kg within 3 hours are frequently chosen to be delivered to substantial numbers of sepsis patients. However, in the practice of subdivision of the flow rate, infusion volume of normal saline would be easily decreased, i.e., normal saline 10 ml/kg within 30 minutes + potassium concentration check urgently (such as using point-of-care test) + balanced crystalloid 20 ml/kg within next 2.5 hours. Even there is no data to support the clinical benefit of suggested practice, nearly 20% reduction of chloride infusion is expected when compared to normal saline 30 ml/kg within 3 hours.

The clinicians need to be familiar to the new SSC guideline, and also need to be beyond the literature of the guideline. The authors expect that our opinion would be one of the beyond things in clinical practice for the sepsis and septic shock.

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