Fluid and Nutritional Management Strategy for Severe Acute Pancreatitis

Eun Taek Park, M.D., Ph.D.
Division of Hepatobiliary and Pancreas, Department of Internal Medicine, Gospel Hospital, University of Kosin College of Medicine, Busan, Korea

Introduction

Severe acute pancreatitis (SAP) is associated with high morbidity and mortality due to the development of pancreatic and extra-pancreatic necrosis, their subsequent infection and multisystem organ failure (MOF). SAP develops in two phases. During the first 1-2 wk, a pro-inflammatory response occurs, which results in systemic inflammatory response syndrome (SIRS), a sterile response in which sepsis or infection rarely occurs. After the first 1-2 wk, a transition from a pro-inflammatory to an anti-inflammatory response occurs. During this compensatory anti-inflammatory response syndrome (CARS), the patient is at risk for the translocation of intestinal flora due to intestinal barrier failure, which is followed by the development of secondary infection. Mortality occurs in two peaks. Early mortality is the result of severe SIRS with MOF. Late mortality is the consequence of infection resulting in sepsis. Meanwhile, changes in gene expression detected early after injury precede the occurrence of subsequent complications with a typical clinical picture. Rapid depletion of energy resources leads to immunosuppression and persistent inflammation and immune suppression catabolism syndrome (PICS). Early diagnosis of immune disorders and appropriate nutritional therapy can significantly reduce the incidence of complications, length of hospital stay, and mortality.

What strategy for fluid and nutrition management for severe acute pancreatitis?

The goal of intravenous fluid resuscitation in acute pancreatitis is to adequately perfuse the pancreatic microcirculation so that pancreatic necrosis and its subsequent complications can be minimized or even prevented. Vigorous intravenous hydration leads to hemodilution and relief of hemoconcentration. The role of HCT in determining severity is related to hemoconcentration. SAP is often accompanied with pathophysiological coexistence state of hypovolemic shock and distributive shock, the release of numerous inflammatory factors, the increase of systemic capillary permeability and the disorder of capillary leak and oxygen metabolism. All these symptoms will seriously affect the function of each organ, even leading to multiple organ failure. To correct the early hemodynamic abnormality is the first priority for the treatment of SAP. Fluid insufficient will increase the cyclic hypoxia of the organ and tissue which could lead to organ dysfunction and increasing mortality. There are significant correlations between early fluid resuscitation and the duration of SIRS, organ function failure rate...
and hospital mortality decline.

Parenteral nutrition (PN) used to be a modality of choice for the treatment of acute pancreatitis, it placed the pancreas in a condition of “rest.” The goal was to inhibit gastric secretion, indirectly influencing pancreatic secretion, or to inhibit pancreatic secretion directly. However, PN results in intestinal atrophy and mucosal dysfunction, leading to abnormal intestinal mucosal permeability that facilitates bacterial translocation. Advantage and safety of early EN in acute pancreatitis have been confirmed with the decrease in the overall complication rates, surgical intervention, and mortality and morbidity rates, which encourages the rapid return of normal gut function and reduces the cytokine-generated stress response that occurs during an acute episode of pancreatitis.

Conclusion

Despite limitations to our current knowledge on the appropriate rate and monitoring of hydration, our results support other studies showing the importance of early aggressive intravenous hydration in the management of patients with acute pancreatitis. Current evidence confirms that the administration of enteral nutrition is beneficial for the treatment of SAP. Enteral feeding reduces mortality, infectious complications and MOF. As far as the route of enteral feeding is concerned, nasogastric tube feeding is likely to be equally as effective as naso-jejunal feeding in SAP.

References