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Short Communication

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Nutritional Support of Patients With the Abdominal Compartment Syndrome During Severe Acute Pancreatitis

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There is growing evidence in the literature that development of Abdominal Compartment Syndrome (ACS) in patients with Severe Acute Pancreatitis (SAP) has a strong impact on the course of disease. Incidence of ACS in patients with SAP is around 20%. The mortality rate in patients who developed ACS during SAP is 49%, while it is 11% in patients without this complication. The development of organ failure in SAP is in correlation with the presence of intra-abdominal hypertension which can deteriorate already compromised pancreatic perfusion and perfusion of gut in early stages of SAP. The latter leads to the alteration of gut functioning with consequent reduced possibility for enteral feeding. Enteral Nutrition (EN) facilitates gut motility and alleviates bacterial translocation, but in patients suffering from ACS during course of SAP could aggravate bowel ischemia. Parenteral nutrition is required as nutritional support in ACS, but it may increase bacterial translocation and deteriorate gut functioning. Since in the literature data there still have not had recommendations regarding nutritional support of patients with ACS during course of SAP, including optimal time for initiation, duration and amount of specific nutritional regiment, in this short review we have tried to give insight into problems in nutritional support in those patients. This should fortify the interest of physicians to make additional research in order to support further strategies for the more optimal nutritional support of patients with this lethal complication.

The abdominal compartment syndrome (ACS) is well described entity which importance in various clinical conditions has been recognized in the last two decades. It is defined as a state of serious organ dysfunction resulting from sustained increase in Intra-Abdominal Pressure (IAP).[1] There is growing evidence in the literature data that the development of ACS in patients with severe form of acute pancreatitis (SAP) has strong influence on the course of disease.[2-5] The incidence of Intra-Abdominal Hypertension (IAH) in patients suffering from SAP is approximately 70%, while ACS can be found in up to 27% of patients with this form of AP.[3,4,6,7] When we add to this a mortality rate of 49% in patients with SAP and ACS,[5] it is clear that IAH and ACS have become an issue of concern in patients with AP. In addition, it was recently mentioned that the number of patients with AP and this complication has increased, but still there have not had standard recommendations for interventional treatment of patients who develop ACS during SAP.[8] The step-up approach for conservative treatment of ACS was proposed several years ago.[9] However, the appropriate interventional procedure, including surgical technique, and optimal time for reacting in the treatment of AP patients suffering from this serious condition is still debated.

From a metabolic point of view, SAP is characterized by nitrogen waste and protein catabolism with negative nitrogen balance and secondary malnutrition.[10] Similarly with septic patients, the AP patients have an impaired capacity for net protein synthesis and are less sensitive to protein sparing of glucose infusion.[11] Also, in patients suffering from AP energy expenditure is increased 1.49 (1.08 to 1.78) of the predicting resting energy expenditure, 58% of patients with SAP have increase in energy expenditure, approximate net nitrogen loses are 20-40 grams per day, and proteolysis can be increased by 80%.[12] Therefore, the nutrition therapy is necessary in patients with SAP.

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Hypovolemia is common in AP, especially in the severe form of the disease, and is a result of a massive fluid loss into the retroperitoneal space and interstitial space overall. However, an early substantial fluid loss in patients with SAP occurs in retroperitoneal space and interstitial space of gut. Cytokines activation in the early phase of AP results in increased capillary permeability, vasoconstriction and transendothelial migration of leukocytes. This event is associated with significant increase of leukocytes infiltration with histological changes and decreasing in intestinal and pancreatic perfusion and mucosal ischemia of the gut.[13-16] In early stages of SAP the profound fluid losses in a "third space" associated with inflammation of the pancreas may induce splanchnic vasoconstriction. Hypovolemia also leads in decreasing in splanchnic perfusion with consequent cellular hypoxia especially in intestinal mucosa.[17,18]

It is certain that the gastrointestinal system and liver functions are the most vulnerable to the high Intra-Abdominal Pressure (IAP). Mainly two functions are altered: (1) the mucosal barrier function (influencing both intermucosal nutrient flow and bacterial translocation) and (2) the gastrointestinal motility. The reduction of splanchnic blood perfusion occurs at the level of IAP of 10 mmHg, with the exception of the adrenal glands.[19,20] The metabolic changes in the gut, such as acidosis and decreased intestinal oxygenation, are evident at the IAP level of 15 mmHg.[21] It was shown that IAP from 20-25 mmHg in the duration of 60 minutes leads to the bacterial translocation from gut.[22] In our recent study we found a highly significant correlation between IAP and procalcitonin (PCT) in patients with AP suggesting bacterial translocation.[7] The impact of elevated IAP on the gut is essential due to circumstantial evidences of relationship between bacterial translocation and multi-organ dysfunction syndrome.[23-25]

Present recommendations regarding nutritional support in patients with SAP favour enteral nutrition (EN) over parenteral nutrition (PN) due to several reasons. Firstly, nasoenteric tube feeding as compared with total PN reduced the rate of infection and mortality among patients with SAP throughout stimulating intestinal motility – thus reducing bacterial overgrowth, and increasing splanchnic blood flow which helps to preserve the integrity of the gut mucosa. Second, total PN lacks the trophic effect of EN and is associated with central venous catheter related infections as well as metabolic complications. Also, in all patients in whom the clinician decides that some form of nutritional support is indicated, should provide it by enteral route. Only in patients whose gut has failed or administration of EN is impossible for other reasons (prolonged ileus, complex pancreatic fistulae and ACS), total PN is indicated.[26-30]

However, in the literature data there still have not had recommendations regarding nutritional support in patients with ACS. This includes an optimal time for initiation and duration of specific nutritional regiment. Also, there has not had randomized control trials regarding nutritional support of patients suffering from AP and IAH. In a pilot study by Sun et al,[31] which compared the incidence of IAH in 60 patients with early or delayed administration of EN, IAH was more prevalent in patients with delayed EN administration. They were also argued that higher IAP may correlate with intolerance to enteral feeding.

Indeed, there are several papers, case reports and retrospective studies, in which were reported non-occlusive bowel ischemia and bowel necrosis after EN.[32-35] In most reports were suggested that EN may play a central role in bowel ischemia (Table 1).[32-40] The pathogenesis of ischemic changes of gut secondary to EN is multifactorial including intraluminal factors, such as increased energy demands in metabolically stressed enterocytes, intestinal bacterial overgrowth and increased bowel's intraluminal pressure with the subsequent reduction in gut perfusion. In AP patients who have hypovolemia, increased capillary permeability, splanchnic hypo perfusion and possible reperfusion injury after initial treatment, they surly may have additional mucosal damage after enteral feeding. In addition to this, patients with IAH and ACS already have significant decreased in splanchnic perfusion associated with mucosal ischemia, thus could have more pronounced mucosal damage of gut and serious gut dysfunction. It has been reported a gut barrier dysfunction in patients with ACS during AP by higher serum concentrations of antiendotoxin core antibodies and PCT in those patients, suggesting increased bacterial translocation from gut.[23]

Early results of our prospective observational study conducted among patients with SAP and ACS showed that majority of patients suffering from ACS during course of SAP had better tolerance for total PN than for EN, suggesting gut dysfunction. Regarding certain nutritional support in patients with ACS during course of SAP, we found that combined usage of EN and total PN was better tolerated than EN or total PN alone. Moreover, in patients who received total PN serum values of PCT were higher than in those who received EN or combined total PN and EN. However, in patients who received EN alone it was found a higher serum values of PCT noted at the time of IAH/ACS occurrence than in those who received combined EN and total PN. This fact point to the favorable effects of EN on gut functioning in patients with SAP, but without effects, even deterioration in gut functioning, in patients suffering from ACS during course of the SAP.[41]

It is certain that EN should be the first line of nutritional support in almost all patients with AP, but in those with IAH/ACS this route of feeding should be carefully monitored. The occurrence of further abdominal distension, elevation of IAP and high nasogastric output should result in immediate discontinuation of tube feeding rather than repeated attempts to alter the formula. In all patients with this serious condition the balanced usage of EN and total PN should be considered, even in those with intractable IAH. What should be the critical value of IAP which may indicate the adverse effects of EN remains to be examined. However, because of the complexity of intensive care together with the heterogeneity of patients with AP as unpredictable disease, optimal nutrition support remains a difficult topic to study. As a result, nutrition support practices among providers

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	Article type	Number of patients	Route	EN only	Part of changed bowel	Pathological changes	Outcome
De Brabandere K, et al[32]	Case report	1	ileum	Yes	Ileum and left colon	Congestion in ileum / colon necrosis	Survived
Melis M, et al[33]	Case report	1	Jejunum	Yes	Small bowel	Necrosis	Died
Gwon JG, et al[34]	Case report	2	Jejunum	Yes	Part of small bowel and colon / colon	Necrosis	Died / Survived
Marvin R, et al[35]	Retrospective study	13	11 in jejunum / 2 in duodenum	Yes in 12 patients	Small bowel and colon in 1 / jejunum and or ileum in 7 / ileum and right colon in 3 / right colon in 2 patients	From bowel inflammation to bowel necrosis with multiple perforations	6 died
Schunn CG, Daly JM[36]	Retrospective study	4	Jejunum	Yes	Small bowel in 4 and colon in 1	Necrosis	2 died
Brenner DW, Schellhammer PF[37]	Case report	1	Jejunum	Yes	Small bowel	Inflammation and ulcerations with necrosis	died
Munshi IA, et al[38]	Case report	1	Jejunum	Yes	Jejunum	Necrosis	died
Jorba R, et al[39]	Case report	1	Jejunum	Yes	Jejunum	Necrosis	died
Lawlor DK, et al[40]	Retrospective study	3	Jejunum	Yes	Jejunum and ileum	Necrosis	survived

Table 1: Non-occlusive bowel ischemia after enteral nutriotion.

are extremely variable. Well designed experimental and randomized clinical studies should be expected to shed more light of the most appropriate nutritional support in patients with this lethal condition.

CONFLICTS OF INTEREST: None.

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