

Short Communication

*Corresponding author

Mihailo Bezmarevic, MD

Clinic for General Surgery
Military Medical Academy
Crnotravska 17

11000 Belgrade, Serbia

Tel. +381-641-994288

Fax: +381-113-608550

E-mail: bezmarevicm@gmail.com

Volume 1 : Issue 1

Article Ref. #: 1000POJ1105

Article History

Received: November 25th, 2015

Accepted: January 4th, 2016

Published: January 4th, 2016

Citation

Bezmarevic M, Panisic-Sekeljic M. Nutritional support of patients with the abdominal compartment syndrome during severe acute pancreatitis. *Pancreas Open J.* 2016; 1(1): 14-18. doi: [10.17140/POJ-1-105](https://doi.org/10.17140/POJ-1-105)

Copyright

©2016 Bezmarevic M. This is an open access article distributed under the Creative Commons Attribution 4.0 International License (CC BY 4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

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

Mihailo Bezmarevic, MD^{*}; Marina Panisic-Sekeljic, MD

Clinic for General Surgery, Military Medical Academy, Crnotravska 17, 11000 Belgrade, Serbia

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.

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

	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 nutrition.

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.

REFERENCES

- Kirkpatrick AW, Roberts DJ, De Waele J, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. *Intensive Care Med.* 2013; 39(7): 1190-1206. doi: [10.1007/s00134-013-2906-z](https://doi.org/10.1007/s00134-013-2906-z)
- Gecelter G, Fahoum B, Gardezi S, Schein M. Abdominal compartment syndrome in severe acute pancreatitis: an indication for a decompressing laparotomy? *Dig Surg.* 2002; 19: 402-404. doi:[10.1159/000065820](https://doi.org/10.1159/000065820)
- Chen H, Li F, Sun JB, Jia JG. Abdominal compartment syndrome in patients with severe acute pancreatitis in early stage. *World J Gastroenterol.* 2008; 14: 3541-3548. doi: [10.3748/wjg.14.3541](https://doi.org/10.3748/wjg.14.3541)
- Dambrauskas Z, Parseliunas A, Gulbinas A, Pundzius J, Barauskas G. Early recognition of abdominal compartment syndrome in patients with acute pancreatitis. *World J Gastroenterol.* 2009; 15: 717-721. doi: [10.3748/wjg.15.717](https://doi.org/10.3748/wjg.15.717)

- van Brunshot S, Schut AJ, Bouwense SA, et al. Abdominal compartment syndrome in acute pancreatitis: a systematic review. *Pancreas.* 2014; 43(5): 665-674. doi: [10.1097/MPA.000000000000108](https://doi.org/10.1097/MPA.000000000000108)
- De Waele JJ, Leppäniemi AK. Intra-abdominal hypertension in acute pancreatitis. *World J Surg.* 2009; 33(6): 1128-1133. doi: [10.1007/s00268-009-9994-5](https://doi.org/10.1007/s00268-009-9994-5)
- Bezmarevic M, Mirkovic D, Soldatovic I, et al. Correlation between procalcitonin and intra-abdominal pressure and their role in prediction of the severity of acute pancreatitis. *Pancreatology.* 2012; 12(4):337-343. doi: [10.1016/j.pan.2012.05.007](https://doi.org/10.1016/j.pan.2012.05.007)
- Trikudanathan G, Vege SS. Current concepts of the role of abdominal compartment syndrome in acute pancreatitis - an opportunity or merely an epiphenomenon. *Pancreatology.* 2014; 14(4): 238-243. doi: [10.1016/j.pan.2014.06.002](https://doi.org/10.1016/j.pan.2014.06.002)
- Cheatham ML. Nonoperative management of intraabdominal hypertension and abdominal compartment syndrome. *World J Surg.* 2009; 33(6): 1116-1122. doi: [10.1007/s00268-009-0003-9](https://doi.org/10.1007/s00268-009-0003-9)
- Bouffard YN, Delafosse BX, Annat GJ, Viale JP, Bertrand DM, Motin JP. Energy expenditure during severe acute pancreatitis. *J Parenter Enteral Nutr.* 1989; 13(1): 26-29. doi: [10.1177/014860718901300126](https://doi.org/10.1177/014860718901300126)
- Variyam EP, Fuller RK, Brown FM, Quallich LG. Effect of parenteral amino acids on human pancreatic secretion. *Dig Dis Sci.* 1985; 30: 541-546.

12. Cano N. Nutrition in acute pancreatitis. *Crit Care & Shock*. 2004; 7: 69-76.
13. Ahlborg G, Weitzberg E, Lundberg JM. Circulating endothelin-1 reduces splanchnic and renal blood flow and splanchnic glucose production in humans. *J Appl Physiol*. 1995; 79(1): 141-145.
14. Weitzberg E, Ahlborg G, Lundberg JM. Long-lasting vasoconstriction and efficient regional extraction of endothelin-1 in human splanchnic and renal tissues. *Biochem Biophys Res Commun*. 1991; 180:1298-1303. doi: [10.1016/S0006-291X\(05\)81336-1](https://doi.org/10.1016/S0006-291X(05)81336-1)
15. Kaufmann P, Tilz GP, Smolle KH, Demel U, Krejs GJ. Increased plasma concentrations of circulating intercellular adhesion molecule-1 (cICAM-1) in patients with necrotizing pancreatitis. *Immunobiology*. 1996; 195: 209-219. doi: [10.1016/S0171-2985\(96\)80040-4](https://doi.org/10.1016/S0171-2985(96)80040-4)
16. Werner J, Z'graggen K, Fernandez-del Castillo C, Lewandowski KB, Compton CC, Warshaw AL. Specific therapy for local and systemic complications of acute pancreatitis with monoclonal antibodies against ICAM-1. *Ann Surg*. 1999; 229: 834-840; discussion 841-842.
17. Ammori BJ. Role of the Gut in the Course of Severe Acute Pancreatitis. *Pancreas*. 2003; 26(2): 122-129.
18. Milev B, Mirkovic D, Bezmarevic M, Misović S, Mitrović M, Jovanović M, et al. Intra-abdominal hypertension and abdominal compartment syndrome. *Vojnosanit Pregl*. 2010; 67(8): 674-680. doi: [10.1053/j.ajkd.2010.08.034](https://doi.org/10.1053/j.ajkd.2010.08.034)
19. Diebel LN, Dulchavsky SA, Wilson RF. Effect of increased intraabdominal pressure on mesenteric arterial and intestinal mucosal blood flow. *J Trauma* 1992; 33: 45.
20. Friedlander MH, Simon RJ, Ivatury R, DiRaimo R, Machiedo GW. Effect of hemorrhage on superior mesenteric artery flow during increased intra-abdominal pressures. *J Trauma*. 1998; 45(3): 433-89.
21. Cheatham ML. Abdominal compartment syndrome: pathophysiology and definitions. *Scand J Trauma Resusc Emerg Med*. 2009; 17: 10. doi: [10.1186/1757-7241-17-10](https://doi.org/10.1186/1757-7241-17-10)
22. Rutherford EJ, Skeete DA, Brasel KJ. Management of the patient with an open abdomen: techniques in temporary and definitive closure. *Curr Probl Surg*. 2004, 41(10): 815-876. doi: [10.1067/j.cpsurg.2004.08.002](https://doi.org/10.1067/j.cpsurg.2004.08.002)
23. Al-Bahrani A, Darwish A, Hamza N, et al. Gut Barrier dysfunction in critically ill surgical patients with abdominal compartment syndrome. *Pancreas*. 2010; 39: 1064-1069. doi: [10.1097/MPA.0b013e3181da8d51](https://doi.org/10.1097/MPA.0b013e3181da8d51)
24. Grootjans J, Lenaerts K, Derikx JP, et al. Human intestinal ischemia-reperfusion induced inflammation characterized: experiences from a new translational model. *Am J Pathol*. 2010; 176(5): 2283-2291. doi: [10.2353/ajpath.2010.091069](https://doi.org/10.2353/ajpath.2010.091069)
25. Kanwar S, Windsor AC, Welsh F, Barclay GR, Guillou PJ, Reynolds JV. Lack of correlation between failure of gut barrier function and septic complications after major upper gastrointestinal surgery. *Ann Surg*. 2000; 231: 88-95.
26. Gianotti L, Meier R, Lobo D, et al. ESPEN Guidelines on Parenteral Nutrition: Pancreas. *Clinical Nutrition*. 2009; 28: 428-435. doi: [10.1016/j.clnu.2009.04.003](https://doi.org/10.1016/j.clnu.2009.04.003)
27. Spanier BW, Bruno M, Mathus-Vliegen EM. Enteral Nutrition and Acute Pancreatitis: A Review. *Gastroenterology Research and Practice*. 2011; 9 pages. doi: [10.1155/2011/857949](https://doi.org/10.1155/2011/857949)
28. Oláh A, Romics Jr L. Enteral nutrition in acute pancreatitis: A review of the current evidence. *World J Gastroenterol*. 2014; 20(43): 16123-16131. doi: [10.3748/wjg.v20.i43.16123](https://doi.org/10.3748/wjg.v20.i43.16123)
29. Bakker OJ, van Brunschot S, van Santvoort HC, et al. Early versus On-Demand Nasoenteric Tube Feeding in Acute Pancreatitis. *N Engl J Med*. 2014; 371: 1983-1993. doi: [10.1056/NEJMoa1404393](https://doi.org/10.1056/NEJMoa1404393)
30. Mirtallo JM, Forbes A, McClave SA, et al. International consensus guidelines for nutrition therapy in pancreatitis. *JPEN J Parenter Enteral Nutr*. 2012; 36(3): 284-291. doi: [10.1177/0148607112440823](https://doi.org/10.1177/0148607112440823)
31. Sun JK, Li WQ, Ke L, et al. Early enteral nutrition prevents intra-abdominal hypertension and reduces the severity of severe acute pancreatitis compared with delayed enteral nutrition: a prospective pilot study. *World J Surg*. 2013; 37: 2053-2060. doi: [10.1007/s00268-013-2087-5](https://doi.org/10.1007/s00268-013-2087-5)
32. De Brabandere K, De Waele B, Delvaux G. Colonic ischemia and perforation associated with enteral feeding through an ileal tube. *Nutrition in Clinical Practice*. 2010; 25(3): 301-303. doi: [10.1177/0884533610368705](https://doi.org/10.1177/0884533610368705)
33. Melis M, Fichera A, Ferguson M. Bowel Necrosis Associated With Early Jejunal Tube Feeding. *Arch Surg*. 2006; 141:701-704. doi: [10.1001/archsurg.141.7.701](https://doi.org/10.1001/archsurg.141.7.701)
34. Gwon JG, Lee YJ, Kyoung KH, Kim YH, Hong SK. Enteral nutrition associated non occlusive bowel ischemia. *J Korean Surg Soc*. 2012; 83:171-174. doi: [10.4174/jkss.2012.83.3.171](https://doi.org/10.4174/jkss.2012.83.3.171)
35. Marvin R, McKinley B, McQuiggan M, Cocanour C, Moore F. Nonocclusive Bowel Necrosis Occurring in Critically Ill Trauma Patients Receiving Enteral Nutrition Manifests No Reliable Clinical Signs for Early Detection. *The American Journal of Surgery*. 2000; 179: 7-12. doi: [10.1016/S0002-9610\(99\)00261-5](https://doi.org/10.1016/S0002-9610(99)00261-5)

36. Schunn CDG, Daly JM. Small bowel necrosis associated with post-operative jejuna tube feeding. *J Am Coll Surg*. 1995; 180: 410-416.
37. Brenner DW, Schellhammer PF. Mortality associated with feeding catheter jejunostomy after radical cystectomy. *Urology*. 1987; 30: 337-340. doi: [10.1016/0090-4295\(87\)90296-2](https://doi.org/10.1016/0090-4295(87)90296-2)
38. Munshi IA, Steingrub JS, Wolpert L. Small bowel necrosis associated with early post-operative jejunal tube feeding in a trauma patient. *J Trauma*. 2000; 49: 163-165.
39. Jorba R, Fabregat J, Garcia Borobia F, Torras J, Poves I, Jaurrieta E. Small bowel necrosis in association with early post-operative enteral feeding after pancreatic resection. *Surgery*. 2000; 128: 111-112. doi: [10.1067/msy.2000.104119](https://doi.org/10.1067/msy.2000.104119)
40. Lawlor DK, Inculet RI, Malthaner RA. Small bowel necrosis associated with jejuna tube feeding. *Can J Surg*. 1998; 41:459-462.
41. Bezmarevic M, Panisic-Sekeljic M, Popadic A, Mirkovic D, Soldatovic I. Gut Dysfunction in Abdominal Compartment Syndrome during Severe Acute Pancreatitis and Dilemmas in Nutritional Support. *Clinical Nutrition*. 2015; 34(Suppl 1): S46.