ROLE OF PHYSIOTHERAPY FOR ADULT PATIENTS WITH CRITICAL ILLNESS

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Abstracts

Long-term complications of critical illness include intensive care unit (ICU)-acquired weakness and neuropsychiatric disease. Immobilisation secondary to sedation might potentiate these problems. Critical illness can last from hours to months, depending on the underlying pathophysiology and response to treatment. It carries high morbidity and mortality rates, and the associated care is a major determinant of healthcare costs. The evolution of intensive care medicine and integrated team management has greatly improved the survival of critically ill patients. In view of the high costs associated with ICU, every attempt should continue to be made to prevent complications and appropriately treat the primary underlying pathophysiology to minimize length of stay in ICU. There are common complications particularly associated with a prolonged ICU stay, including deconditioning, muscle weakness, dyspnoea, depression and anxiety, and reduced health-related quality of life. Chronic critical illness is associated with prolonged immobility and intensive care unit (ICU) stay and accounts for 5–10% of ICU stays, a proportion that appears to beincreasing . Because of these detrimental sequelae of long-term bed rest, there is a need for rehabilitation throughout the critical illness and thereafter , to address these effects. The amount of rehabilitation rehabilitation performed in ICUs is often inadequate,

A strategy for whole-body rehabilitation-consisting of interruption of sedation and physical and occupational therapy in the earliest days of critical illness-was safe and well tolerated, and resulted in better functional outcomes at hospital discharge, a shorter duration of delirium, and more ventilator-free days compared with standard care.

References

1. GOSSELINK, R., et al. *Physiotherapy for adult patients with critical illness*: Recommendations of the European Respiratory Society and European Society of Intensive Care Medicine Task Force on Physiotherapy for Critically Ill Patients Intensive Care Med (2008) 34:1188–1199.
2. SCHWEICKERT, W.D., et al. E*arly physical and occupational therapy in mechanically ventilated, critically ill patients: a randomised controlled trial.* Lancet. 2009 May 30;373(9678):1874-82. Epub 2009 May 14.

ENTERAL NUTRITION IN SEVERE ACUTE PANCREATITIS. QUESTIONS LOOKING FOR ANSWERS

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The nutritional policy in acute severe pancreatitis changed dramatically in the last two decades. After years of recommendations for *nihil per os* combined with total parenteral nutrition, the policy moved to enteral nutrition delivered by jejunostomy combined with total parenteral nutrition, than changed to only (if possible) enteral nutrition. Than enteral nutrition by nasojejunal tube was advocated. Lately intragastric nutrition in acute severe pancreatitis is under scrutiny.

The "classical" recommendation *nihil per os* aimed the pancreatic rest. Meanwhile the caloric needs should be delivered by total parenteral nutrition. Fasting has a lot of deleterious consequences: villous atrophy, decreased splanhnic blood flow, loss of epithelial tight junctions, decreased secretion of bile salts and IgA, decreased *gut associated lymphoid tissue(GALT)*, bacterial overgrowth and bacterial translocation. The impaired GALT alters the macrophage priming, promotes the release of cytokines, free oxygen species and arahidonic acid metabolits, all of them resulting in enhanced inflammatory reaction and systemic inflammatory response syndrome (1,4,6).

By contrast, enteral nutrition preserves the integrity and the barrier function of intestinal epithelium, enhances blood flow, avoids bacterial overgrowth and bacterial epithelial adhesion and prevents bacterial translocation. It is important to stress that infection of pancreatic necrosis is due to enteral bacteria (1,4,6). Thus, enteral nutrition is not only a support intervention, but a therapeutic one because it may directly influence the evolution of the disease by preventing infection of necrotic tissue. It also promotes bowel movements, shortening the duration of paralitic ileus and decreasing intra-abdominal pressure.

Taking into account that 65% of total immune tissues and 80% of immunoglobulin producing tissues belong to the digestive tract (*gut-associated lymphoid tissue* –GALT and *mucosal-associated lymphoid tissue* –MALT), enteral nutrition has an important

effect upon the local and systemic immune response during acute severe pancreatitis. In conclusion, fasting promotes inflammation and enteral nutrition promotes appropriate immune function.

Several studies and meta-analysis investigated different parameters (infection rate, morbidity, mortality, rate of surgery, ICU- and hospital length-of-stay, costs) in patients with acute pancreatitis comparing enteral versus parenteral nutrition (1,4,8,10). The last (2008) Cochrane meta-analysis showed that rates of mortality, hospital length-of-stay, local or systemic infections and of other complications favor enteral nutrition (19). Parameters of immune response also favor enteral nutrition, which results in lower rates of SIRS, sepsis, multiple organ dysfunction syndrome and mortality.

All these evidences translated into guideline recommendations of the European Society of Enteral and Parenteral Nutrition (7). Whenever possible in acute severe pancreatitis enteral nutrition should be used (grade A). If needed, parenteral nutrition may be associated to support energy and nutrients requirements. Whenever possible, the oral route should be used, if not, the alternative is endoscopically placed naso-jejunal feeding. Feeding jejunostomy may be performed during surgery. Standard enteral formula are well tolerated.

Today a consensus has been reached considering early enteral nutrition as *standard of care* in acute severe pancreatitis due to promotion of appropriate immune response, shortening and improvement of disease evolution, decreased infection and surgery rates and diminished costs. However, despite knowledge and commitment, the application of these principles into the daily practice is not an easy task. Enteral nutrition in acute severe pancreatitis is still associated with many questions, which are looking for answers.

Who needs nutritional support?

In mild/moderately severe acute pancreatitis the is no need for nutritional support due to the rapid resolution of the disease. Fating is still recommended for about 48 hours. By contrast, in acute severe pancreatitis early enteral nutrition is crucial for the course of the disease. But early recognition of the severe form is sometimes difficult and clinical, laboratory parameters or scores predict with variable accuracy the severity of the disease.

Which is the best route for enteral nutrition?

The degree of pancreatic stimulation depends upon the route of feeding delivery. Oral feeding is associated with the highest pancreatic secretion due to the cephalic phase (smell, taste) – resulting in the fasting recommendation. The lowest is associated with deep jejunal feeding, resulting in jejunostomy feeding. But the tendency to avoid surgery in acute severe pancreatitis precluded jejunostomy and promoted the endoscopically placed nas-jejunal tube as standard route. Double lumen tubes are recommended (the jejunal lumen for feeding and the gastric one for decompression). In fact, a lot of difficulties are connected with the use of the naso-jejunal tube (frequent duodenal obstruction due to enlargement of the pancreatic head, frequent displacement into stomach, need for a very experienced endoscopist). Lately naso-gastric tube feeding was investigated in acute severe pancreatitis (17,20,21). The results are encouraging demonstrating not only that disease evolution is not worse, but even shorten. But also this route is associated with difficulties (delayed gastric emptying due to duodenal obstruction and paralitic ileus, risk of regurgitation and aspiration).

How early is early?

All advantages of enteral nutrition may be achived with early, but not with late enteral feeding. But how early is early in patients with delayed gastric emptying and ileus? Some favor the introduction of enteral nutrition in the first 72 hours (16), others in the first 6 hours (17). Guidelines recommend as early as possible, in the moment it is tolerated. The tolerance should be individually evaluated due to the large interpersonal variability.

How to deal with digestive intolerance? How to achieve the targeted amount?

Introduction of enteral feeding should be gradual. The starting rate is 15-20ml/hour and it is increased progressively every 24-48 hours until the total amount of 25kcal/kg/day is achieved. Every 3-4 hours a check of residual volume should be performed, because it may signal the retrograde (stomach) tube displacement. The actual recommendations promote continuous delivery 24 hours/day.

What formula should be used?

Nutrients stimulate the exocrine pancreatic secretion to different degrees, lipids most and carbohydrates least. ESPEN recommendations include semielemental (small peptides) or elemental (aminoacids) with low content in long-chain fatty acids (grade A). Standard formulas may be used as well. Carbohydrates administration should be accompanied by insulin in order to achieve normoglycemia, a difficult goal in acute severe pancreatitis.

Is there a role for TPN?

In mild/moderate acute pancreatitis parenteral nutrition is contraindicated because it may worsen the evolution. In acute severe pancreatitis parenteral nutrition may be used when access to/amount of enteral feeding is impossible/inappropiate. Early (the first 48-72 hours) parenteral nutrition is contraindicated because it may aggravate the evolution. It may be introduced after 5 days, when failure of access or proper amount of enteral nutrition was documented.

Is enteral nutrition supportive or therapeutical intervention?

The patient with acute severe pancreatitis has intense hypercatabolism. Nutritional support doesn't aim to convert catabolism to anabolism, but to prevent energetic exhaustion and to preserve the visceral mass. The multiple advantages of enteral nutrition

displace parenteral nutrition, which dominated for decades the scene of nutrition in acute severe pancreatitis. The capacity of enteral nutrition to influence the acute phase response, the systemic inflammatory reaction and the infection rate recommend it as a therapeutical intervention promoting enhanced recovery.

In conclusion, enteral nutrition in severe acute pancreatitis is not an easy task and needs a lot of commitment and dedication. But, despite difficulties its early and "obstinate" application may favorably influence the evolution of the disease.

Bibliography:

1. MCCLAVE SA et al: Nutritional support în acute pancreatitis: a systematic review of the literature, *JPEN*, 2006, 30(2):143-156.
2. TIENGOU LE et al: Semi-elemental formula: is there a better chice for enteral nutrition în acute pancreatitis? Randomized comparative study, *JPEN*, 2006, 30(1):1-5.
3. WHITCOMB DC: Acute Pancreatitis, *N Engl J Med*, 2006, 354:2142-50.
4. MARIK PE, ZALOGA GP: Meta-analysis of parenteral nutrition versus enteral nutrition în patients with acute pancreatitis, *BMJ*, 2004, 328:1407-0.
5. KALE-PRADHAN PB et al: Enteral Nutrition în Patients with Pancreatitis, *Pharmacotherapy*, 1999, 19(9):1036-1041.
6. MEIER R et al: ESPEN guidelines on nutrition în acute pancreatitis, *Clinical Nutrition*, 2002, 21(2):173-183.
7. R MEIER et al: ESPEN guidelines on enteral nutrition: pancres; *Clinical Nutrition* 25,275-284, 2006.
8. CANO NJM: Nutrition in acute pancreatitis, *Crit Care & Shock,* 2004, 7(2):69-76.
9. ZHAO G et al: Clinical study on nutritional support în patients with severe acute pancreatitis, *World J Gastroenterol*, 2003, 9(9):2105-2108.
10. WINDSOR AC et al: Compared with parenteral nutrition, enteral feeding attenuates the acute phase response and improves disease severity în acute pancreatitis, *Gut*, 1998, 42:431-435.
11. GREENWOOD JK et al: Enteral nutrition in acute pancreatitis: A survey of practices în Canadian Intensive Care Units, *Nutrition in Clinical Practice*, 2004, 19(1):31-36.
12. CHANDRASEGARAM MD et al: The impact of parenteral nutrition on body composition of patients with acute pancreatitis, *Journal of Parenteral and Enteral Nutrition*, 2005, 29(2):65-73.
13. POWELL JJ et al: Randomized controlled trial of the effect of early enteral nutrition on markers of the inflammatory response in predicted severe acute pancreatitis; *Br J Surg*, 2000, 87:1375-1381.
14. RAIMONDO M, SCOLAPIO JS: What route to feed patients with severe acute pancreatitis: vein, jejunum, stomach?, *Am J Gastroenterol*, 2005, 100(2): 440-441.
15. ALSOLAIMAN MM et al: Should enteral feeding be the standard of care for acute pancreatitis?, *Am J Gastroenterol*, 2003, 98(11):2565.
16. ABOU-ASSI S et al: Hypocaloric jejunal feeding is better than total parenteral nutrition in acute pancreatitis: results of a randomized comparative study, *Am J Gastroenterol*, 2002, 97:2255-62.
17. EATOCK D et al: A Randomized Study of Early Nasogastric versus Nasojejunal Feeding in Acute Severe Pancreatitis, *Am J Gastroenterol*, 2005, 100:432-439.
18. GUPTA R et al : A randomised clinical trial to assess the effect of total enteral and total parenteral nutritional support on metabolic, inflammatory and oxidative markers in patients with predicted severe acute pancreatitis (APACHE II > or =6), *Pancreatology,*2003, 3:668.
19. AL-OMRAN M et al: Enteral versus parenteral nutrition for acute pancreatitis (review), http://www.thecochranelibrary.com, 2008, issue 3.
20. KUMAR A et al: Early Enteral Nutrition in Severe Acute Pancreatitis: A Prospective Randomized Controlled Trial Comparing Nasojejunal and Nasogastric Routes, *J Clin Gastroenterol,* 2006, 40(5):135.
21. GRIGORAS I et al: Is intragastric administration of enteral nutrition safe in severe acute pancreatitis?*, Crit Care,* 2009, 13(suppl 1):P140.

TRANSFUSION POLICY IN CRITICALLY ILL PATIENTS

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Anemia is a common and early condition in critically ill patients. 29% patients have Hb < 10g% at ICU admission (1). 95% patients have anemia in the 3rd ICU day (2). We studied the incidence of anemia in 132 critically ill patients with long ICU stay (>7 days) and found it presence in 110 (83.33%) patients at ICU admission and in 126 (95.45%) patients at ICU discharge (3).

Causes of acquired anemia in intensive care patients are blood loss due to surgery, trauma, gastrointestinal bleeding or medical procedures (mainly phlebotomy), hemodilution, decreased red blood cell production or increased distruction and others (4). Phlebothomy may result in 30-70ml blood withdrawal/day (1,2). In critically ill patients an increase of 3,5ml/day withdrawn blood doubles the risk of transfusion (5). The amount of blood withdrawn by phlebotomy decreases over time during ICU stay, mainly due to decreased number of ABG (5).

The critical illness is frequently associated with inflammatory status. The proinflammatory cytokines (TNFα, IL-1, IL-6) result in failure of circulating EPO to appropriately increase in response to Hb reduction (relative EPO deficiency), inhibition of erythrocyte production (relative EPO resistance) and decreased iron availability (6,7,8). Corwin terms this status as *anemia of critical illness*.

Iron metabolism is severely altered in during critical illness. The main features are decreased iron availability for erythropoesis and increased iron storage (9). Markers of iron metabolism alterations during inflammation are decreased serum Fe, decreased