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Review Article

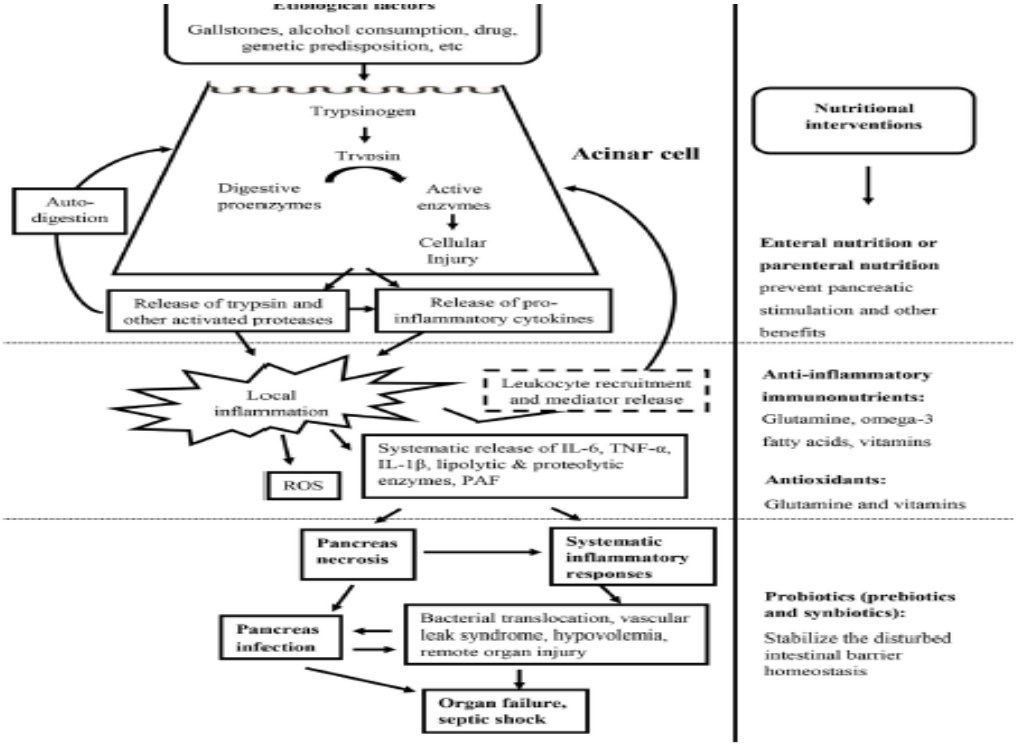
Nutrition in acute pancreatitis

Sahar Khairy, Professor of Pediatrics
Clinical Nutrition Consultant, National Nutrition Institute

Definitions

Early Phase Acute Pancreatitis (AP): (within 1 week) Characterized by the systemic inflammatory response syndrome (SIRS) and / or organ failure;
Late Phase AP: (>1 week) Characterized by local complications.
Mild AP: Lacks both organ failure and local or systemic complications
Moderately Severe AP: Transient organ failure (organ failure of <2 days), local complications, and/or exacerbation of coexistent disease
Severe AP: Presence of persistent organ failure (organ failure that persists for >/2 days)

Targeted nutritional interventions during the whole episode of acute pancreatitis



Pan et al. Nutritional Interventions in Clinical AP Frontiers in Immunology
www.frontiersin.org June 2017 | Volume 8

Atlanta Classification for Grading Severity of Acute Pancreatitis

Grade of Severity	Criteria for Classification
Mild acute pancreatitis	No organ failure No local or systemic complications
Moderate-severe acute pancreatitis	Organ failure that resolves (Early) within 48 hours (transient organ failure) Local or systemic(Late, >1 week) complications without persistent organ failure
Severe acute pancreatitis (Highly catabolic Nutrient consuming)	Persistent organ failure (>48 hours): Single organ failure Multiorgan failure

Therapeutic goals in acute pancreatitis

Overall goal

Initiating an oral diet as soon as possible for patients with acute, severe acute pancreatitis

Therapeutic goals

To counteract catabolism

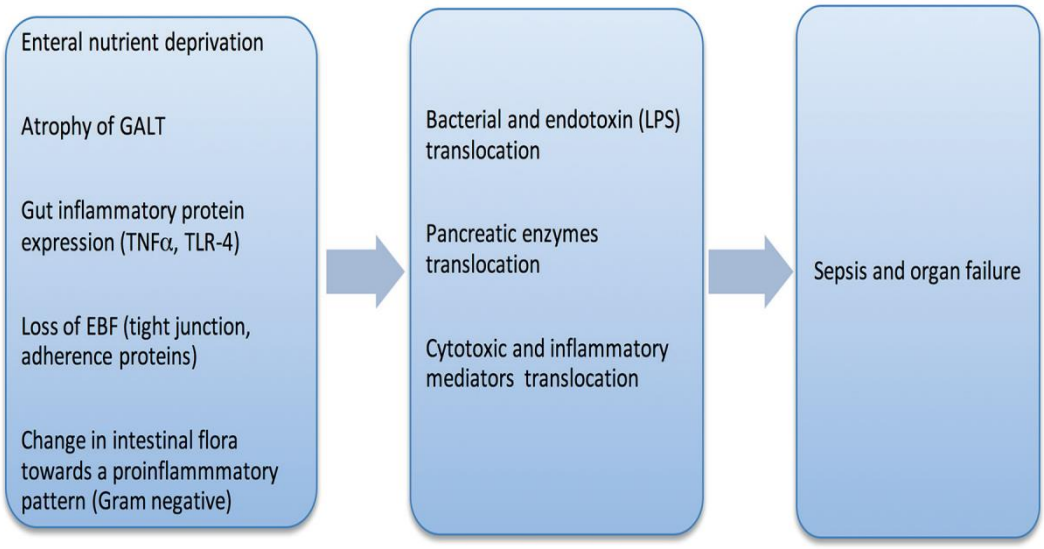
To abate pancreatic inflammation by decreasing exocrine stimulation and

To manage metabolic disturbances that may be present

Metabolic changes in acute pancreatitis

Increased	Decreased
Energy expenditure	Insulin response
Gluconeogenesis	Glutathione
Proteolysis	Vitamins A, C and E
Urea turnover	Selenium
Lipolysis	Methionine
BCAA oxidation	Glutamin
Glutamine oxidation	

Harmful consequences of PN and enteral starvation



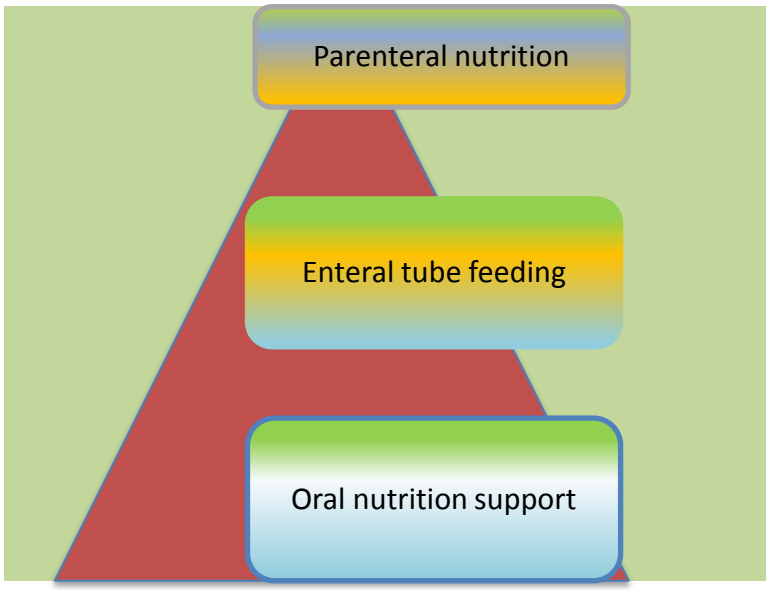
Abbreviations: GALT: Gut Associated Lymphoid Tissue; TNF-a: Tumor Necrosis Factor-a; TLR-4: Toll Like Receptor-4; EBF: Epithelial Barrier Function; LPS: Lipopolysaccharide

Timing of Initiation of Feeding

Initiation of early feeding within 24 hours of presentation regardless of predicted severity

- ❑ Decreased length of stay
- ❑ Decreasedcomplication rate
- ❑ cost-effectiveness
- ❑ Better prognosis
- ❑ Decreasedmortality

Feeding rout



Composition of oral Feeds

For patients who tolerate oral intake, research clinical trials have shown

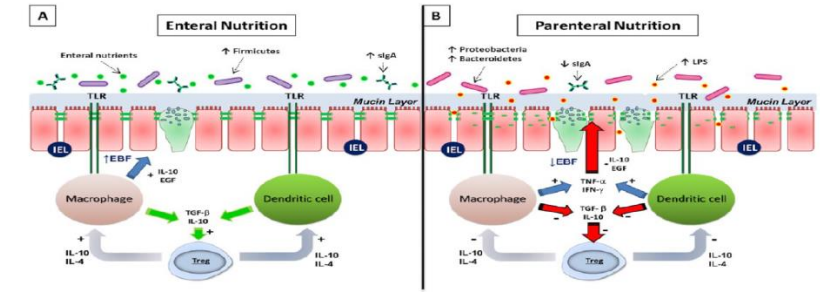
- No difference between starting patients on clear liquids with plans to advance the diet and starting with a solid diet

Enteral Nutrition Versus ParenteralNutrition

Parenteral nutrition was historically recommended for patients with acute pancreatitis

- for a longer resting period for the pancreas
- limiting the stimulation of exocrine pancreatic secretion
- minimizing enzyme-driven inflammation
- Providing patients with nutrition.
- providing exogenous nutrients to maintain lean body mass and avoid dynamic ileus

Enteral Nutrition Versus ParenteralNutrition



A) In the enterally fed state,intestinal epithelial barrier function is maintained by the interaction between *Firmicutes-dominated intestinal microbiota and the host* immune system. (B) With dependence on parenteral nutrition, the lack of enteral nutrients leads to an altered microbiome, characterizedby increased *Proteobacteria, which in turn drives a proinflammatory state in the intestinal mucosa, resulting in epithelial barrier*dysfunction. EBF, epithelial barrier function; EGF, epidermal growth factor; IEL, intraepithelial lymphocyte; IFN, interferon; IL,interleukin; LPS, lipopolysaccharide; sIgA, secretory immunoglobulin A; TGF, transforming growth factor; TLR, Toll-like receptor;TNF, tumor necrosis factor; Treg, T-regulatory cell.

(Farokh R. Demehri; Meredith Barrett; and Daniel H. Teitelbaum Changes to the Intestinal Microbiome With Parenteral Nutrition: Review of a Murine Model and Potential Clinical Implications. Nutrition in Clinical Practice 30(6). December 2015 798–806)

Why EN is safe during an attack of acute pancreatitis ?

In healthy subjects, both oral and enteral feeding stimulates amylase, lipase, and trypsin secretion, as well as gastrin and cholecystokinin

An elemental enteral formula may reduce enzyme secretion by 50%

On the other side, in acute pancreatitis, both an animal model and a prospective study on patients showed that pancreatic exocrine secretion is suppressed during AP

These mechanisms may explain why EN is safe during an attack of AP

Why EN is safe during an attack of acute pancreatitis ?

Conversely, parenteral nutrition impairs metabolic response, increasing plasmatic insulin and glucose

Precipitate metabolic response to stress leading to a heavy state of protein catabolism and insulin-resistance.

PN “puts at rest” the bowels, impairing its absorption and barrier function to infection and sepsis

Enteral vs Parenteral

Current guidelines

Suggest avoiding use of PN

Except in situations

- ❑ Enteral feeding is not feasible
- ❑ Minimum caloric requirements are not met

Gastric vsJejunal

Jejunal feeding offers the benefit of by passing any element of gastroparesis, pancreatic edema, or pseudocysts encroaching on the stomach or duodenum.

However placement requires endoscopy, often under fluoroscopic guidance may require a bridle, suture, or mechanical clip placement to secure its positioning.

The nasogastric tube has demonstrated to be safe and useful as well as the nasojejunal tube

Nutritional Formulae for Enteral Nutrition

Selection of EN formulae

	POLYMERIC	SEMI-ELEMENTAL	ELEMENTAL
Nitrogen (casein, lactalb., soy)	whole proteins	small peptides	amino-acids
Carbohydrates	complex carbohydrates	simple sugar, glucose polymers or starch	simple sugar
Fats	long chain triglycerides (LCTs).	medium chain triglycerides (MCTs).	very low fats but variable amounts of lipids, usually MCTs and/or essential fatty acids
Osmolarity	300	300 – 450	300 - 600
Indications	multiple	allergy, malabsorption	multiple allergies, severe malabs.
Advantages	palatable, cheap	hypoallergenic rapid absorption	non-allergenic immunomodulatory
Disadvantages	intact GIT	bitter, expensive	expensive, bad taste, hyperosmolar

Nutritional Formula

All international guidelines recommend a small peptide and medium chain triglyceride (MCT) oil based formulation (grade B recommendation)

ESPEN guidelines recommend peptide-based formulas (grade A recommendation)

even if they acknowledge that a standard formula can be tried if tolerated (grade C recommendation)

The use of glutamine supplementation, immune-nutrition, prebiotics or probiotics is not supported by large-scale studies

Conversely, glutamine-supplements are effective in reducing mortality, complications, and length of stay if given in total, when such approach is inevitable.

All international guidelines recommend a small peptide and medium chain triglyceride (MCT) oil based formulation (grade B recommendation)

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even if they acknowledge that a standard formula can be tried if tolerated (grade C recommendation)

Formula delivery techniques

Current guidelines recommend continuous feeds as the preferred approach over cyclic or bolus feeds

Energy requirements in severe acute pancreatitis, according to ESPENguidelines

Substrate	Quantity	Notes
Proteins	1.2- 1.5 g/kg/day	If not present renal failure or severe hepatic failure
Carbohydrates	3- 6 g/kg/day	Plasma glucose should be ≤ 10 mmol/l (180 mg/dl)
Triglycerides	Up to 2 g/kg/day	Plasma triglycerides should be ≤ 3 mmol/l (266 mg/dl)

Summary of Nutrition Recommendations for Severe Acute Pancreatitis

Management of Severe Acute Pancreatitis	Recommendation
Enteral vs parenteral feeds	Enteral feeding preferred
Timing of feeding	Early feeding within48 hours is preferred
Gastric vs jejunal route for tube feeding	Gastric or jejunal acceptable
Composition of feeds	Full solid oral diet as tolerated; no benefit of elemental formula for tube feeding
Pancreatic exocrine insufficiency	Replacement benefit limited to severe or necrotizing pancreatitis
Prebiotics and probiotics	Insufficient data to support standard use

References

Carol Rees Parrish.

Pragmatic Management of Nutrition in Severe Acute Pancreatitis. NUTRITION ISSUES IN GASTROENTEROLOGY, SERIES #179 . PRACTICAL GASTROENTEROLOGY, SEPTEMBER,2018

E. RINNINELLA, M.G. ANNETTA, M.L. SERRICCHIO, A.A. DAL LAGO,G.A.D. MIGGIANO, M.C. MELE.

Nutritional support in acute pancreatitis:5from physiopathology to practice. An evidence-based approach. European Review for Medical and Pharmacological Sciences. 2017; 21: 421-432

M.K Maheshwari, AviralTyagi, B.K Gupta.

Role of early enteral feeding in acute pancreatitis'' IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 18, Issue 4 Ser. 2 (April. 2019), PP 06-10 www.iosrjournals.org

Meera Ramanathan; and Abdul Aziz Aadam.

Nutrition Management in Acute Pancreatitis. American Society for Parenteral and Enteral Nutrition. Nutrition in Clinical Practice Volume 34 Supplement 1 October 2019 S7–S12. DOI: 10.1002/ncp.10386

OHSU HEALTH SYSTEM.

OFFICE OF CLINICAL INTEGRATION AND EVIDENCE-BASED PRACTICE .GUIDELINE FOR THE MANAGEMENT OF ACUTE PANCREATITIS 2019

Pan L-L, Li J, Shamoon M, Bhatia M and Sun J (2017)

Recent Advances on Nutrition in Treatment of Acute Pancreatitis. Front. Immunol. 8:762. doi: 10.3389/fimmu.2017.00762

Simone Gärtner, Antje Steveling, and Peter Simon.

Timing of oral refeeding after acute pancreatitis. American Pancreatic Association. Version 1.0, August 19, 2016 [DOI: 10.3998/panc.2016.23]

Farokh R. Demehri; Meredith Barrett; and Daniel H.

Teitelbaum Changes to the Intestinal Microbiome With Parenteral Nutrition: Review of a Murine Model and Potential Clinical Implications. Nutrition in Clinical Practice 30(6). December 2015 798–806