# Nutrition therapy in acute and chronic pancreatitis

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## Abstract

Pancreatitis is an inflammatory disease associated with disorders of nutrient assimilation and, as a result, with significant changes in the nutritional status.

All patients with acute pancreatitis should be considered at nutritional risk and should be screened using validated screening methods. The optimal nutritional treatment for acute pancreatitis has been debated for decades. The traditional approach was "nothing in the mouth", only parenteral nutrition until the acute symptoms disappear and the level of serum pancreatic enzymes decreases. However, this tactic can contribute to various complications, starting with malnutrition and ending with sepsis due to damage of the intestinal mucosa. Clinical trials and meta-analyses have shown that patients with acute pancreatitis can tolerate oral nutrition and that oral / enteral nutrition is associated with a shorter hospital stay and a lower rate of complications compared to solely parenteral. Therefore, early oral nutrition with a low-fat "soft food" is recommended. In case of oral feeding intolerance, enteral nutrition is preferable, but not parenteral supply. A combination of enteral and parenteral nutrition may be recommended in patients who do not tolerate a sufficient amount of enteral nutrition.

Malnutrition in chronic pancreatitis cannot be detected using BMI alone, and a detailed nutritional assessment is required, including assessment of symptoms and organic functions, anthropometry, and biochemical tests. Nutritional therapy in chronic pancreatitis should be multifactorial and based on abstinence from alcohol and nicotine, and diet modification. International guidelines no longer recommend severe dietary fat restriction; on the contrary, a physiological diet is recommended, but with adequate replacement of pancreatic enzymes. In case of intolerance to physiological nutrition, a low-fat diet with oral nutritional supplements is recommended to replenish energy and nutrients.

This is a review of recent studies and guidelines on nutrition in pancreatitis for physicians and medical trainees.

**Keywords:** nutrition, acute pancreatitis, chronic pancreatitis, enteral nutrition, oral nutrition

## Introduction

Pancreatitis is an inflammatory disease associated with disorders of nutrient assimilation and, as a result, with significant changes in the nutritional status. The two major forms of inflammatory pancreatic disease, acute and chronic pancreatitis, are diseases where nutritional treatment is essential, absolutely necessary and important. But these forms require different approaches to nutrition management.

Acute pancreatitis (AP) in all cases and regardless of the severity of the disease requires adequate nutritional support. This support becomes extremely important in case of moderate and severe disease, when catabolic processes predominate, the possibilities of nutrient absorption are significantly reduced due to exocrine pancreatic insufficiency, but patients self-limit their diet due to pain and stool disorders.

Chronic pancreatitis (CP) is a disease of the pancreas in which recurrent inflammatory episodes result in the replacement of the functional pancreatic parenchyma with fibrotic tissue. This

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fibrotic reorganization leads to progressive exocrine and endocrine insufficiency [1]. In chronic pancreatitis, as in acute pancreatitis, the situation worsens due to sitophobia.

First fasting, and then strict dietary restrictions have been the basis of dietary advice over the years. However, recent studies have shown the irrationality of this approach and the need to change the nutritional therapy for pancreatitis.

## Acute pancreatitis

AP is a pathological condition that can cause nutritional insufficiency, moreover, about 30% of patients with AP are already malnourished at the time of the initial attack [2]. According to the recommendations of the European Society for Clinical Nutrition and Metabolism (ESPEN) 2020 Guide, patients with AP should be considered at moderate to high nutritional risk due to the catabolic nature of the disease and the negative impact of nutritional status on the course of the disease, and patients with severe AP should always be considered at high nutritional risk [3]. All patients with mild to moderate disease should be screened using validated screening methods such as "Nutritional Risk Screening - 2002" (NRS-2002); the nutritional risk assessment can be performed by using the NRS-2002 online at https://www.mdcalc.com/nutrition-risk-screening-2002-nrs-2002.

Body mass index can also be used to assess nutritional status and nutritional risk. A low body mass index, associated with malnutrition, is the common risk factor for severe AP. However, it is important to remember that obesity is also a known risk factor for severe AP, and therefore obese patients have an increased nutritional risk caused by the severity of the disease [4].

The optimal nutritional treatment for acute pancreatitis has been debated for decades. The traditional approach was "nothing in the mouth", only parenteral nutrition until the acute symptoms disappear and the level of serum pancreatic enzymes decreases. This approach was argued in theory - to allow the pancreas to rest. Most guides recommended this tactic despite the lack of clinical evidence. However, it can contribute to various complications, starting with malnutrition, the predominance of the catabolic process due to the restriction of energy intake at a time when energy needs are increased and ending with sepsis due to damage of intestinal mucosa. On the other hand, clinical trials and meta-analysis have shown that patients with AP can tolerate oral nutrition and that oral / enteral nutrition is associated with a shorter hospital stay and a lower rate of complications compared to parenteral nutrition [5-8].

The correct administration of fluids and food is a major medical task in patients with AP. Early oral nutrition with a "soft food" seems to be more beneficial in terms of caloric intake and equally tolerated compared to liquid diets. A recent meta-analysis, including 17 studies, identified that only 16.3% of patients with AP had intolerance to early oral feeding [6]. Thus, according to modern knowledge, oral nutrition is recommended as soon as it is clinically tolerated and independent of serum pancreatic enzyme levels in patients with mild AP. Oral nutrition can be done with the low-fat, soft usual "kitchen" products or with special pharmaceutical products for oral nutrition (eg Fresubin, Nutrison, Nutridrink, Nutricomp, etc.).

In case of oral feeding intolerance, enteral nutrition (EN) is preferable, but not parenteral supply [3,9]. Multiple randomized clinical trials and systemic meta-analyzes have shown that EN helps maintain the integrity of the intestinal mucosa, stimulates intestinal motility, prevents excessive growth of bacteria, increases splanchnic blood flow and, as a result, improves the evolution of AP. EN is safe and well tolerated, with significant decreases in complication rates, multi-organ failure, and mortality compared to parenteral nutrition (PN) [7,8]. EN should be started early, within 24-72 hours of hospitalization, in case of intolerance to oral feeding [8,10].

EN can be performed by gastric or duodenal tube (nasogastric, orogastric, nasoduodenal) or by surgical stoma (jejunostoma, gastrostoma, etc.). The nasogastric type is the most common. Administration through the stomach, which acts as a reservoir, may be intermittent (bolus or slow) or continuous, as opposed to intestinal administration, which should be continuous. However, about 15% of patients have an intolerance to this type of EN, mainly due to delayed gastric emptying and, in this case, feeding through the nasojejunal tube is required. Placement of the tube in the stomach is associated with a higher risk of pulmonary aspiration than placement in the intestine.

Common dietary foods or pharmaceutical products may be used for EN. Dietary foods should be ground and dissolved or suspended in water, homogenized so that it can be administered through a relatively thin tube. Nutritional foods may contain:

- proteins: milk, egg whites, minced lean meat, peas;
- lipids: olive oil, soybeans, sunflower, corn, egg yolk;
- carbohydrates: starch, sucrose, lactose, fructose.

The introduction of up to 400 ml of food is recommended for adults. Oral liquid medications are not recommended to be taken with meals to prevent excessive volume in the stomach at the same time. If medicine and food are to be given at the same time, the medicine must be given first.

Pharmaceutical products used for EN usually consist of polymeric or oligomeric formulations (elemental, semi-elemental) (Table I).

**Table I.** Characteristics of pharmaceutical products for enteral nutrition [11].

| | Polymeric formulations | Oligomeric formulations |
|---|---|---|
| Protein substrate | Whole protein (milk, whey, eggs, soy) | Peptides (semi-elemental formulas) or free aminoacids (elemental formulas) |
| Lipid subsrate | Long chain triglycerides | Medium or short chain triglycerides (does not require pancreatic enzymes or bile salts for digestion and absorption) |
| Carbohydrate substrate | Maltodextrin (usually)<br>Usually lactose and gluten free | Oligosaccharides |
| Other nutrients | Vitamins and microelements in daily doses | Variable |
| Other features | Often with a pleasant taste<br>Cheaper | More unpleasant taste<br>More expensive |
| Examples | Nutrizon®, Fresubin®, Ensure® | Peptamen®, Nutrien elementali® |

EN with polymeric formulations is effective and safety in most cases of AP [12]. EN formulations that contain fiber, especially insoluble, should be avoided, because insoluble fiber has an osmotic effect, retains water in the intestine, prolongs the emptying time of the stomach, can cause flatulence, bloating and diarrhea. Fruit-oligosaccharides may be recommended during recovery. They pass undigested through the small intestine and are metabolized in the colon by the intestinal microflora. In fact, they are prebiotics that serve as a source of energy for the normal intestinal microflora.

Parenteral nutrition should be given to patients with AP (including post-surgery conditions) who do not tolerate EN or who are unable to tolerate a sufficient amount of EN or if there are contraindications for EN [3]. A combination of EN and PN may be recommended in patients who do not tolerate a sufficient amount of EN.

## Chronic pancreatitis

The progressive nature of chronic pancreatitis (CP) with the replacement of functional tissue with fibrotic leads to the development of exocrine and endocrine insufficiency of the organ, which in turn leads to malabsorption and malnutrition. Malnutrition develops after 5-10 years in the case of alcoholic etiology and later in idiopathic CP [13]. The main causes of malnutrition in CP are pancreatic insufficiency with maldigestion on the one hand and citophobia with low food intake on the other hand. Alcohol abuse and smoking worsen the situation. Malnutrition has a serious negative impact on the outcome of the disease, it significantly reduces the quality of life and productivity of the patient. At the same time, malnutrition has a negative impact on the evolution of CP, accelerates the progression of the disease and aggravates exocrine insufficiency and, as a result, aggravates malnutrition. A vicious circle is created.

The classic clinical manifestation of malnutrition is low weight with low BMI. At the same time, half of patients with CP may be overweight or obese. But this increased BMI is associated with sarcopenia and nutrient deficiency [9,14]. Thus, malnutrition in CP cannot be detected using BMI alone, and a detailed nutritional assessment is required, including assessment of symptoms and organic functions, anthropometry, and biochemical tests. Clinical assessment should include: analysis of diet, appetite; presence of dyspeptic syndrome (ex, nausea, vomiting, early satiety) or symptoms of nutrient deficiency (macro- and microelements, vitamins, etc.) and organ and system disorders. The most useful tests for anthropometry, other than BMI, are hand-grip strength dynamometer, skinfold thickness, waist and mid arm muscle circumferences. A large number of biochemical tests can be informative: vitamins (A, D, E, K, B12), folic acid, ferritin, thyroid and parathyroid hormones, iron, Ca, trace elements (magnesium, selenium, zinc), etc. The ESPEN guide recommends screening for micro- and macronutrient deficiencies at least once every twelve months or more frequently in severe disease or uncontrolled malabsorption [3].

Good nutritional practice in CP includes screening to identify patients at nutritional risk, followed by a complete nutritional assessment and nutrition plan for patients at risk. Nutritional therapy should be multifactorial and based on abstinence from alcohol and nicotine, diet modification, and adequate pancreatic enzyme replacement therapy. Historically, patients with CP have been advised to follow a low-fat diet, even a diet without animal fats for severe steatorrhea. This recommendation was based on the fact that dyspepsia and steatorrhea are worse after fat intake. However, limiting fat intake most often leads to a restriction of the total caloric content of the diet, which exacerbates malnutrition, contributes to the insufficiency of macro- and microelements, vitamins, and, as a result, worsens the evolution and prognosis of CP. Despite the absence of large clinical trials, international guidelines no longer recommend severe dietary fat restriction; on the contrary, a physiological diet is recommended, but with adequate replacement with pancreatic enzymes [1,3,9].

For example, in the last ESPEN guideline experts with very high agreement, over 90%, voted for the following recommendations:

• patients with CP do not need to follow a restrictive diet;

• CP patients with a normal nutritional status should adhere to a well-balanced diet;

• malnourished patients with CP should be advised to consume high protein, high-energy food in five to six small meals per day;

• in patients with CP, there is no need for dietary fat restriction unless symptoms of steatorrhea cannot be controlled with adequate doses of pancreatic enzymes;

• in patients with CP, diets very high in fiber should be avoided [3].

The last recommendation is related to the fact that fibers can absorb pancreatic enzymes (including those administered for replacement) and can lead to inadequate substitution treatment.

Gastro-resistant enteric-coated microspheres or mini-microspheres of less than 2 mm in diameters are recommended for pancreatic exocrine insufficiency [1]. Micro-or mini-tablets of 2.2–2.5 mm in size may be also effective, although scientific evidence is more limited. The optimal dose of pancreatic enzymes is probably the main point of replacement therapy. Despite the fact that the guidelines recommend fairly high doses of enzymes (lipase dose of 40,000 - 50,000 PhU with main meals and half that dose with snacks), in clinical practice the doses taken are often much lower. Insufficient dose and inadequate pharmacological form of enzymes cannot stop steatorrhea and dyspeptic syndrome. This forces the patient to restrict the intake of fat, which in turn exacerbates malabsorption and completes a vicious circle.

Thus, in most cases of CP, a fractional physiological diet with proper enzyme replacement treatment is sufficient to maintain the required nutritional status. In some cases, adequate replacement therapy is not enough to normalize digestion and stop steatorrhea. In these cases, a low-fat diet and oral nutritional supplements are recommended, especially those containing medium chain triglycerides (MCTs). MCTs have an unpleasant taste and are associated with side effects such as abdominal cramps, nausea, and diarrhea. If MCTs are considered, their dose should be increased slowly, depending on patient tolerance.

And the last but not least point of nutritional therapy is the adequate intake of vitamins, macro- and microelements with food. Dietary restrictions and nutrient assimilation disorders in CP often lead to vitamin deficiencies, especially fat-soluble vitamins, mineral and micronutrient deficiencies. The serum level of these nutrients is recommended to be monitored and compensated if necessary.

## Conclusion

Acute and chronic pancreatitis are pathological conditions associated with nutritional deficiency, therefore, in all patients with AP and CP, the nutritional status should be monitored.

Early oral nutrition with a low-fat "soft food" is recommended in AP. In case of oral feeding intolerance, enteral nutrition is preferable, but not parenteral supply. A combination of EN and PN may be recommended in patients who do not tolerate a sufficient amount of EN.

Nutritional therapy in CP should be multifactorial and based on abstinence from alcohol and nicotine, and diet modification. International guidelines no longer recommend severe dietary fat restriction; on the contrary, a physiological diet is recommended, but with adequate replacement with pancreatic enzymes. In case of intolerance to physiological nutrition, a low-fat diet with oral nutritional supplements is recommended to replenish energy and nutrients.

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