

COMMENTARY

A Commentary on Role of Fibre in Nutritional Management of Pancreatic Diseases

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DESCRIPTION

The pancreas plays a crucial role in digestion and absorption of nutrients. In both acute and chronic pancreatitis, an exocrine and endocrine pancreatic insufficiency can develop, which impairs the digestive and absorptive processes [1]. The main clinical consequence of Exocrine Insufficiency (PEI) is malnutrition, consequent to maldigestion-driven malabsorption of macro- and micronutrients, like fat-soluble vitamins, minerals, and trace elements [2]. Consequently, the nutritional management of Pancreatic Disease (PD) regarding timing of refeeding, diet composition in terms of macro and micronutrient and nutritional supplement, should be tailored considering specific case-by-case.

In Acute Pancreatitis (AP), ESPEN guideline recommends oral feeding as soon as clinically tolerated and independent of serum lipase concentrations [3]. Low-fat, soft oral diet shall be used when reinitiating oral feeding in patients with mild AP and, in those patients with AP and inability to feed orally, EN shall be preferred to Parenteral Nutrition (PN). EN should be started early, within 24-72 h of admission, in case of intolerance to oral feeding [3]. In AP, an adequate diet is important to restore energy balance and to preserve gut barrier function. A central actor in the "pancreas-intestinal barrier axis" is ruled up by Gut Microbiota (GM) linked to a range of physiological, immunological, and pathological activities [4]. In this context, several works have focused on the role of probiotics in AP but inconsistent and sometimes conflicting results, especially regarding safety, adverse effects, and reduction of infection and mortality rates [5,6]. More promising results in AP have been obtained by modulating the GM with prebiotics. A prebiotic

enriched diet is associated with low rates of pancreatic necrosis infection, hospital stay, systemic inflammatory response syndrome and multiorgan failure, which are all complications of AP. The protective effect seems to relate to the ability of Soluble Dietary Fibres (SDFs), to stabilize the disturbed intestinal barrier homeostasis and to reduce the infection rate [7-10]. We would underline that, if PEI developed, a high content of DF may be associated with an increased wet fecal weight and fecal fat excretion because of inhibition of pancreatic enzymes by the fibre. Precisely, the pancreatic enzymes could be absorbed on the fibre surface or entrapped in pectin, a gel-like substance, and likely inactivated by "anti-nutrient" compounds present in some foods (i.e., saponins, phytate, trypsin inhibitors) [10], which in turn may exacerbate malabsorption in PEI [11]. On the other hand, these substances can be considered as "non-nutritive compounds with positive effects on health", reducing blood sugar levels and insulin responses to starchy foods, decreasing cholesterol plasma levels and triglycerides levels. Their negative or positive effects depend on the specific clinical context [10].

Nutritional plan needs to be tailored also in Chronic Pancreatitis (CP), in which malabsorption of micro- and macronutrients connected to the insufficient level and/or activity of pancreatic enzymes, is the major cause of progressive nutritional and metabolic impairment [12]. Thus, in CP should be advised to consume high protein, high-energy food in five to six small meals per day [3]. Fat restriction is no longer recommended; studies on endogenous and exogenous enzymes show that survival of enzyme activity is enhanced by the presence of their respective substrates i.e., dietary triglycerides [13]. Deciding the fibres amount in a dietetic plan for CP deserves attention, since fibres may inhibit pancreatic enzyme therapy or reduce their activity, which in turn results in malabsorption of fat and/or carbohydrate maldigestion [14]. Furthermore, the action of Dietary Fibre (DF) on pancreatic enzymes increases in the upper small intestine where the impairment of bicarbonate secretion as consequence of PEI led to an acidic environment [15]. Thus, undigested, and unabsorbed nutrients can enhance intestinal gas production, abdominal pain, and steatorrhea [16]. We desire to be precise that these disturbances may occur in normal healthy subjects when the fibre content

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of their diet is suddenly increased or in patients with Intestinal Bowel Syndrome (IBS). Therefore, clinicians should understand whether they are consequent to a reduction in enzymatic pool or secondary to coexisting functional symptoms.

Certainly, several factors should be kept in mind in nutritional management of PD [16]. Severity of disease, impairing digestive and absorptive processes, need of enzyme replacement therapy, presence of abdominal symptoms should be taken into consideration when elaborate a dietary plan in PD with regards to macro, micronutrients, and fibres content.

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