

Chronic fibro inflammatory injury in chronic pancreatitis results in progressive loss of exocrine acinar cells

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Introduction

Chronic Pancreatitis (CP) is a chronic inflammatory disease of the pancreas with progressive connective tissue deposition and progressive loss of exocrine and endocrine function. CP is characterized clinically by abdominal pain and symptoms associated with pancreatic exocrine dysfunction steatorrhea, weight loss, bloating and endocrine dysfunction diabetes-related symptoms. Pain characteristics range from episodes of acute pancreatitis to persistent abdominal pain. There are several possible causes of pain. Pain significantly reduces the patient's quality of life, leading to decreased appetite, restricted food intake, weight loss, and malnutrition. Chronic fibro inflammatory injury in CP results in progressive loss of exocrine acinar cells, resulting in decreased pancreatic enzyme and bicarbonate production and malabsorption of fat, carbohydrate, and protein increase.

Description

Fat malabsorption is usually most prominent, with a loss of more than 90% of glandular function leading to overt steatorrhea. Carbohydrate malabsorption can lead to bloating, abdominal pain, and bloating from undigested carbohydrate fermentation in the colon. As with other chronic inflammatory diseases, the nutritional status of patients with CP is receiving increasing attention. Early diagnosis and adequate management of malnutrition in CP, which is often unrecognized and greatly underestimated, is clinically relevant, with both factors contributing to hospitalization rates, disease complications, and overall costs, and mortality. A number of laboratory tests measurements of albumin, prealbumin, transferrin, retinol-binding protein, and C-reactive protein have been proposed to assess malnutrition, but these tests alone are not sufficient to assess

malnutrition in CP patients. This lack of a consistent definition of malnutrition has led to considerable heterogeneity among studies on malnutrition, and it is difficult to compare studies to draw conclusions. It's getting harder to pull out.

Physical causes of malnutrition include reduced food intake due to pain and dyspepsia due to decrease both endocrine and exocrine function of the pancreas. However, when the ingested food overcomes the digestive function of the pancreas, symptoms of PEI become apparent. Patients tend to adjust their eating habits to avoid or minimize symptoms, which explain why PEI patients are often asymptomatic. Endocrine deterioration is also a major cause of malnutrition, as it reduces carbohydrate absorption and utilization, leading to caloric breakdown. Overt diabetes is usually a late-stage feature of the disease, whereas PEI is an early-stage disease tends to appear. Indigestion and malabsorption of carbohydrates, proteins and fats can occur. In particular, fat malabsorption is more common and clinically evident, and can lead to malabsorption of fat-soluble vitamins (vitamins A, D, E, and K).

Conclusion

Deficiencies of water-soluble vitamins and trace elements (zinc, magnesium, calcium, thiamine, folic acid) have also been observed in CP patients. Additionally, the risk of thiamine deficiency as a result of concomitant alcoholism should be considered. Consequently, patients with chronic pancreatitis, especially those with advanced disease and steatorrhea, have demonstrated in recent studies that these patients have reduced serum levels of vitamin D metabolites and low bone mass. As you can see, there is a risk of developing significant bone loss. A deficiency in vitamin A can increase susceptibility to serious infections, and a deficiency in vitamins D and E is associated with an increased risk of cardiovascular disease. Although treated for endoscopic/surgical treatment of chronic complications, the nutritional status of CP patients is often ignored unless they are severely underweight.

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Conflict of Interest

The author has nothing to disclose and also state no conflict of interest in the submission of this manuscript.

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