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The Syndrome of External Secretory Function Insufficiency is a Common Complication of Chronic Pancreatitis

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Annotation: Chronic pancreatitis (CP) is a long-term inflammatory disease of the pancreas manifested by irreversible morphological changes that cause pain and/or persistent decrease in function. In CP, morphological changes in the pancreas persist after cessation of the etiological factor.

Keywords: pancreas, immunomorphological factor, Functional tests, diagnostics, oxidative stress, fibrosis and stone formation in the pancreas.

The diagnosis is established on the basis of a characteristic pain syndrome, signs of insufficiency of external secretory function of the PG in a patient who regularly takes alcohol. Unlike acute pancreatitis, in chronic pancreatitis, there is rarely an increase in the level of enzymes in the blood or urine, so if this happens, you can suspect the formation of a pseudocyst or pancreatic ascites Persistently elevated levels of amylase in the blood suggests macroamylasemia (in which amylase forms large complexes with plasma proteins that are not filtered by the kidneys, and the urine has normal amylase activity) or extrapancreatic sources of hyperamylasemia.

Chronic pancreatitis is a syndrome of destructive, inflammatory conditions arising from long-standing pancreatic injury. Patients complain of chronic abdominal pain and may develop malabsorption from pancreatic insufficiency. In the most severe cases, diabetes mellitus results. Although etiological factors are many, alcohol is the most common cause in the United States population. Less common causes include chronic biliary tract disease, hereditary pancreatitis, cystic fibrosis, hyperlipidemia, hyperparathyroidism, and pancreas divisum. Regardless of cause, the disease process leads to a final common pathway of irregular fibrosis, acinar loss, islet cell loss, and inflammatory infiltrates. Thus, chronic pancreatitis may be defined as a continuing inflammatory disease, characterized by irreversible morphologic changes, that typically causes pain and/or permanent loss of function.

Chronic pancreatitis (CP) is a syndrome characterized by chronic progressive pancreatic inflammation, fibrosis, and scarring, resulting in damage to and loss of exocrine (acinar),

endocrine (islet cells), and ductal cells [1]. The syndrome is commonly associated with clinical features of abdominal pain, exocrine and endocrine insufficiency, secondary pancreatic cancer, and other complications. It is accepted that inflammation-led fibrosis culminates in CP [2]. Although acute pancreatitis (AP) and CP were believed to be distinct entities [3], a wealth of data support that AP, recurrent AP (RAP), and CP represent a disease continuum [4, 5]. The etiology of CP has traditionally been classified as alcohol, hereditary, obstructive, hyperlipidemia, and idiopathic. Recent evidence supports the notion that, in most patients, more than one "etiology" is present. The TIGAR-O classification system is grouped by risk modifiers, not etiologies, that may interact to produce pancreatic disease: toxic-metabolic, idiopathic, genetic, autoimmune, recurrent and severe AP-associated CP, and obstructive etiologic factors [6]. The development of this classification system was based on the principle that an individual's risk of developing CP is decided by one or more risk factors [7]. A two-hit hypothesis model can be used to outline the pathogenesis of CP [8]: in the setting of pre-existing AP risk factors (genetic, metabolic, and environmental), an initial first episode of AP (first hit) initiates or activates the immune system, followed by complete recovery, or pathologically by progression towards CP. This cascade of steps toward CP is triggered, provided that there is ongoing damage to the pancreas via oxidative stress or repeated episodes of acute inflammation, which may or may not be clinically apparent [4]. Collectively, this sequence has been coined the sentinel AP event (SAPE) hypothesis [9, 10]. Overall, approximately 20% of patients with AP have a recurrence and 36% of RAP patients go on to develop CP [4]. Here we will review further the myriad risk factors that contribute to the progression to end-stage CP and also cover the current treatment modalities for CP.

Extrapancreatic sources of hyperamylasemia and hyperamylasuria (by W. W. Salt II, S. Schtnkor)

Renal insufficiency

Salivary diseases:

epidemic parotitis

nodule

radiation sialadenitis

Complications of maxillofacial surgery

- ✓ lung cancer -oesophageal cancer
- ✓ ovarian cancer -Macroamylasemia
- ✓ Burns -Diabetic ketoacidosis
- ✓ Pregnancy -Kidney transplantation
- ✓ Trauma to the brain

Medication:

Morphine

Diseases of abdominal organs:

Biliary tract diseases (cholecystitis, choledocholithiasis)

Complications of peptic ulcer disease - perforation or penetration of ulcers

bowel obstruction or infarction

ectopic pregnancy

peritonitis

aortic aneurysm

Postoperative hyperamylasemia

Methods of imaging in the diagnosis of chronic pancreatitis. The diagnosis of CP is usually made by cross-sectional imaging, typically CT or MRI. The diagnosis in those with advanced CP is usually obvious on these studies, with pancreatic calcification, atrophy, and a dilated or irregular pancreatic duct. The addition of MRCP allows more accurate identification of pancreatic ductal abnormalities than does CT or MRI alone, particularly if the hormone secretin is administered during MRCP [37]. The diagnosis of CP in less-advanced disease is more challenging, and a combination of endoscopic ultrasonography (EUS) and direct pancreatic function testing is utilized. Despite these techniques, early diagnosis remains difficult and often inaccurate. Interested readers are referred to a recent review of diagnostic approaches

- ✓ Radiography of the PG area.
- ✓ Transabdominal ultrasound (ductal dilation, pseudocysts, calcification, dilation of the common bile duct, portal, splenic vein, ascites).
- ✓ Endoscopic ultrasound.
- ✓ ERCPG (changes in the structure of the ducts, pseudocysts).
- ✓ Computed tomography (with intravenous contrast).
- ✓ Scintigraphy with administration of granulocytes labelled with 99mTc or 111Ip.

Functional tests can be divided into three groups:

- ✓ Direct tests of PG secretion. The collection and examination of PG juice or duodenal contents after stimulation of PG secretion by exogenous hormones or hormone-like peptides (secretin-cholecystokinin test);
- ✓ indirect tests - examination of duodenal contents after food stimulation (Lund's test);
- ✓ oral tests - performed without cannulation of the duct or probe insertion (N-benzoyl-L-tyrosyl-para-aminobenzoic acid test).

The etiology of CP has traditionally been classified as alcohol, hereditary, obstructive, hyperlipidemia, and idiopathic. Data indicate that AP progresses to RAP then to CP in a disease continuum. However, not all AP becomes recurrent, and not all RAP progresses to CP. Whether AP proceeds to RAP and to CP is determined by a multitude of risk factors, including exposure to alcohol, smoking, hereditary mutations, ductal obstruction, and autoimmune factors. Increased knowledge regarding these etiologies has enhanced our understanding of the disease and changed our approach to the diagnosis and management of this elusive disease. Current management of CP involves patient education, counseling regarding alcohol and tobacco abstinence, a multidisciplinary team approach to pain management, medical treatment of PEI, addressing malnutrition and osteoporosis, and adjustment of PERT and diabetic agents. In a carefully selected subset of patients, endoscopic and surgical intervention may be appropriate.

CONCLUSION: The study provides a theoretical generalisation and a novel solution the actual scientific problem on definition of markers of progression of various clinical and morphological forms of chronic pancreatitis and increase the effectiveness of the treatment of patients on the basis of studying the role of immunomorphological factors, treatment of patients on the basis of studying the role of immunomorphological factors, oxidative stress in the progression of fibrosis and stone formation in the pancreas.

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