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Review

Rare Causes of Acute Pancreatitis: Drugs, Eosinophilia, and Autoimmunity

Mauro Turrin *

Former Department of Internal Medicine, Ospedali Riuniti Padova Sud "Madre Teresa di Calcutta", Monselice (Padova), Italy; E-Mail: m.turrin@libero.it

* **Correspondence:** Mauro Turrin; E-Mail: m.turrin@libero.it

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Abstract

***Background*:** Among the rarest causes of acute pancreatitis, in addition to the drugs (DIAP), there are eosinophilic pancreatitis (EP) and autoimmune pancreatitis (AIP).

***Methods*:** We surveyed on PUBMED the descriptions of clinical cases of eosinophilic pancreatitis appeared from 1990 to June 2019 and those related to new drugs responsible for acute pancreatitis.

***Results*:** We found forty reports on eosinophilic pancreatitis associated or not with hypereosinophilia and gastro-intestinal manifestations. There are several reports on drugs implicated in acute pancreatitis.

***Conclusions*:** Here we discuss the importance of hypereosinophilia in EP and IgG–4 increase in Type 1 AIP. Differential diagnosis with pancreatic neoplasms and therapy schedules are also discussed as well.

Keywords

Acute pancreatitis; drug-induced pancreatitis; eosinophilic pancreatitis; autoimmune pancreatitis; immunoglobulin G4-related disease

1. Introduction

Eosinophilic pancreatitis (EP) is a very rare disease. In the list of rare diseases compiled by Orphanet register [1], this term does not appear. The list includes autoimmune type 1 pancreatitis at no. 280302 (Synonym: lymphoplasmocytic sclerosing pancreatitis or IgG4-related pancreatitis) and type 2 at no. 280315 (Synonym: duct-centric pancreatitis). However, their prevalence is not defined and at no. 103919, undefined autoimmune pancreatitis (AIP) has been placed.

EP is a benign form, it is accompanied in 80% of cases with peripheral eosinophilia and can be associated with idiopathic hypereosinophilic syndrome (HIES), which is defined as a non-clonal lymphoproliferative multisystem disease characterized by persistent eosinophilia and associated with infiltration and damage to the eosinophilia-associated organ.

It is characterized by a persistent eosinophil count >1,500/µL for more than six months (normal: <500/µL), the absence of etiology identifiable by eosinophilia, and the presence of signs and other symptoms associated with the organs such as heart, lungs, gastrointestinal tract, skin, and nervous system. It is often associated, in up to 85% of patients, with eosinophilic gastroenteritis (EoG) [102].

In the Orphanet register [1] the "hypereosinophilic syndrome with undetermined significance" (HEUS) is classified at the number 3260 (phenotype MIM number: 607685). Its prevalence is unknown and the age of onset is between 20 and 50 years with male/female ratio = 4–9:1.

The forms of EP described in the literature [2-11, 13-22, 24-28] do not present a particular clinical picture, and in most cases, they were treated surgically because they were detected as pseudocystic tumor like forms [2-6, 8, 9, 13-15, 29, 106]. Eosinophilic pancreatitis often occurs in an acute form.

In the frequent cases associated with hypereosinophilia, the diagnosis is suggestive but must be confirmed by biopsy and also to exclude autoimmune pancreatitis.

Autoimmune pancreatitis (AIP) is a fibro-inflammatory disease that accounts for 4.6–5% of chronic pancreatitis. It presents a widespread or focal involvement of the pancreas, progressive in nature, which is manifested by jaundice, associated or not to pancreatic mass. It is currently included in the hyper-IgG4 pathology group.

Acute pancreatitis can depend on several causes (**Table 1**).

Table 1 Etiology of acute pancreatitis. Modified from earlier studies [49, 50].

Mechanics
Gallstones (choledocholithiasis)
Microlithiasis
Biliary sludge
Ascariasis
Periampullary diverticulum
Pancreatic or periampullary tumor
Ampullary stenosis
Duodenal stricture or obstruction

Toxic
Ethanol
Methanol
Scorpion venom
Organophosphate poisoning
Horsetail (66)
Methomyl (insecticide) [67]
Metabolic
Hyperlipidemia of type I, IV, V [103]
Hypercalcemia: excessive dosage of vitamin D, hyperparathyroidism,
total parenteral nutrition
Iatrogenic
Didanosine
Azathioprine/6-mercaptopurine
Pentamidine
Stibogluconate
Tetracyclines: doxycycline [69], tigecycline [61]
Amoxicillin/clavulanic acid [62]
Metronidazole [63]
Methimazole [64]
Antihypertensives: furosemide, thiazide, α-methyldopa
ACE inhibitors: captopril, benazepril, enalapril [30-34], lisinopril [35-37], ramipril [38], quinapril
ARBs: irbesartan [39, 40], valsartan [41], and losartan [40, 42-44]
Amiodarone [65]
Sulfasalazine
5-Aminosalicylic acid (mesalazine)
Statins: simvastatin, pravastatin, rosuvastatin [45-47]
DPP-4 inhibitors (dipeptidyl peptidase-4 inhibitors: incretin): sitagliptin, saxagliptin, alogliptin, and vildagliptin [48, 51]
GLP–1 (glucagon -like-peptide–1) receptors agonists: albiglutide, exenatide, and liraglutide [52, 53]
SGLT2 (sodium-glucose cotransporter 2) inhibitors: canagliflozin [54, 55]
L-asparaginase
Valproic acid
Sulindac
Salicylates
Calcium
Estrogen
Trenbolone (anabolic steroid)
Tamoxifen (56)
Antipsychotics: olanzapine, clozapine, quetiapine, and mirtazapine [57]
Paracetamol

Codeine
Propofol
Vedolizumab [58]
Receptor tyrosine kinase inhibitor (TKI): pazopanib [59], and lenvatinib [60]
Riluzole [68]
Isotretinoin [69]
Paclitaxel [70]
Illicit drugs: cannabis [71, 72], cocaine
Infectious [105]
Viruses: Mumps, Coxsackie, HBV, CMV, Varicella-zoster, HSV, and HIV
Bacteria: Mycoplasma, Legionella, Leptospira, Salmonella
Mushrooms: Aspergillus
Parasites: Toxoplasma, Cryptosporidium, Ascaris, Fasciola, and Hydatid disease
Traumatic
Closed abdominal trauma
Penetrating trauma
Iatrogenic damage during surgery (sphincterotomy)
Congenital
Type V choledochocele (Caroli's disease)
Pancreas divisum
Annular pancreas
Vascular
Ischemia
Atheroembolism
Vasculitis (polyarteritis nodosa, systemic lupus erythematosus)
Genetics
Mutations of the CFTR gene (cystic fibrosis transmembrane conductance regulator)
Other genetic mutations: PRSS–1 (serine protease 1: this gene encodes a cationic trypsinogen), SPINK- 1 (serine protease inhibitor, Kazal type 1), CTRC (chymotrypsin C)
From other causes
Endoscopic retrograde cholangiopancreatography (ERCP)
Pregnancy
Renal transplantation
Alpha-1-antitrypsin deficiency
Celiac disease
Autoimmune pancreatitis

Eosinophilic pancreatitis is not reported in this table.

About 2% cases of acute pancreatitis are caused by drugs. In 85–95% of these cases, the resolution is spontaneous and occurs in 3–7 days. The drugs with the highest incidence of pancreatitis are:

- didanosine (analog of inosina, used in HIV–1 therapy): 23%;

- azathioprine, 6-mercaptopurine and mesalazine: 3–5%;
- ACE-inhibitors and sartans: 1–2%.

Regarding the intake of losartan (associated or not with hydrochlorothiazide), there are only three reports in PubMed related to six cases of acute pancreatitis induced by this drug [40, 42-44].

The most recent drugs included in the list of those associated with acute pancreatitis are statins, the latest atypical antipsychotics, dipeptidyl peptidase-4 inhibitors (DPP–4, incretin) [48, 51], GLP–1 Agonists [52, 53], SGLT2 inhibitors [54, 55], and vedolizumab [58].

Among antibiotics, in addition to tetracyclines and tigecycline [61], at least four cases of acute pancreatitis from amoxicillin + ac clavulanate have been reported in the literature [62].

Regarding oral metronidazole, used for the eradication of *Helicobacter pylori*, in a recent Swedish large population-based case-control study [62], it was associated with an increased risk of acute pancreatitis within 30 days of exposure to both single and combined regimens (with amoxicillin).

As for statins, the association had already been detected years ago in several studies and clinical cases [45, 47] and was confirmed by a 2015 epidemiological study [46]. The latter study found that the use of HMG-CoA reductase is associated with an increased rate of incidence of acute pancreatitis, especially during the first year of use.

Most of the reports related to the ARBs and ACE inhibitors indicated in **Table 1** confirmed the injurious action of the drug for rechallenge. Regarding DPP–4 inhibitors (incretin), the incidence of acute pancreatitis was found to significantly increased in diabetic patients treated with gliptins compared to control groups with a difference in absolute risk of 0.13%. This increase translates into one or two additional cases of acute pancreatitis per 1,000 patients treated for two years [51]. The third common cause of acute pancreatitis is hypertriglyceridemia [73], accounting for up to 10% of all cases (hypertriglyceridemia-induced acute pancreatitis: HTGP). Among the drugs implicated in acute pancreatitis, thiazides, estrogen (clomiphene), tipranavir/ritonavir, tamoxifen [56], olanzapine, mirtazapine [57], isotretinoin [104] mainly act through the hypertriglyceridemia they cause.

Chronic familial pancreatitis (Orpha code 676) is a very rare form of chronic infantile pancreatitis, with a prevalence of 1–9/1,000,000, with autosomal dominant transmission, and its age of onset varies from infancy to adolescence.

Hereditary pancreatitis (or autosomal dominant hereditary pancreatitis) has a high risk of pancreatic cancer.

2. Differential Diagnosis

In the case of marked persistent hypereosinophilia, non-intense pain, rapid resolution, normal pancreas on CT scan and the exclusion of other associated pathologies, it could be acute eosinophilic pancreatitis in the normal pancreatic form.

There are eight diagnostic criteria for this disease **(Box 1)**.

1- Symptomatology and an increase of pancreatic enzymes. Clinical and biohumoral criteria for the diagnosis of eosinophilic pancreatitis [74]
2- Persistent eosinophilia (> 1.5 × 10^9/L for more than six months)
3- Association (not mandatory) with eosinophilic gastroenteritis

4- Increase in IgE (not obligatory) with normal IgG values
5- History of allergic diseases (not mandatory)
Exclusion criteria
6- Negativity of any fecal parasite, negativity in the tests for rheumatic, autoimmune and leukemia diseases
7- Exclusion of autoimmune pancreatitis by the normal levels of IgG4
8- Exclusion of pancreatic neoplasia

For the detection of eosinophilia, eosinophilic pancreatitis is considered for the differential diagnosis with some other pathologies, as indicated in **Table 2**.

Table 2 Pathologies with which eosinophilic pancreatitis is considered for the differential diagnosis.

• **Familial eosinophilia**
• **Acquired eosinophilia**
○ **Secondary**
- Parasitic diseases
- Allergic diseases
- Neoplasms
- Eosinophilic pneumonia: Loeffler syndrome
- Churg Strauss syndrome, allergic pulmonary aspergillosis
- Connective diseases: scleroderma, panarteritis nodosa
- Dermatitis herpetiformis
- Inflammatory bowel disease (IBD)
- Sarcoidosis
- Addison's disease
- Hyper-Ig E syndrome
- Drugs
- DRESS syndrome (reaction to a drug with eosinophilia and systemic symptoms)
- Dermatosis
○ **Clonal**
- Acute leukemia
- Chronic myeloid leukemia
○ **Idiopathic**
- Chronic eosinophilic leukemia
• **Eosinophilia without organ infiltration**

In most cases of EP, there is the simultaneous involvement of the gastrointestinal tract in the prevalent form (up to 85% of patients) of eosinophilic gastroenteritis (EoG), an association that can also appear later during the disease [2, 17].

There are only two reports in the literature documenting a prolonged time gap between previous eosinophilic gastroenteritis and acute eosinophilic pancreatitis [18, 26].

Eosinophilic fibrosis of the pancreas has been described in the context of eosinophil multi-visceral fibrosis [14, 75]. The EP should be differentiated from chronic pancreatitis associated with idiopathic hypereosinophilic syndrome [HIES].

A condition of hypereosinophilia associated with chronic pancreatitis has been described in two articles, reporting a total of 51 cases [12, 23]. An Indian study, performed in 2018 on a population of 114 cases of chronic pancreatitis, found the presence of peripheral eosinophilia in 24.5% of patients, predominantly women [79].

EP is considered by some authors to be a variant of autoimmune pancreatitis [8,10] in the context of IgG4-related systemic diseases **(Table 3)** [80].

Table 3 Spectrum of IgG4-related systemic diseases. Modified from Stone et al. [80].

PANCREAS	● Type 1 (IgG4-related AIP) autoimmune pancreatitis
BILE DUCTS	● IgG4-related sclerosing cholangitis
SALIVARY GLANDS	● Mikulicz disease (IgG4-related dacryoadenitis and sialadenitis)
	● Sclerosing sialadenitis (Küttner tumor, IgG4-related submandibular gland disease)
ORBIT	● Inflammatory orbital pseudotumor (IgG4-related orbital inflammation orbital inflammatory pseudotumor) ● Chronic sclerosing dacryoadenitis (lacrimal gland enlargement, IgG4-related dacryoadenitis)
RETROPERITONEAL SPACE	● A subset of patients with "idiopathic" retroperitoneal fibrosis (Ormond disease) and related disorders (IgG4-related retroperitoneal fibrosis, IgG4-related mesenteritis)
AORTA	● Chronic sclerosing aortitis and periaortitis (IgG4-related aortitis or periaortitis)
THYROID	● Riedel's thyroiditis (IgG4-related thyroid disease)
CHEST	● IgG4-related interstitial pneumonitis and pulmonary inflammatory pseudotumors (IgG4-related respiratory disease)
KIDNEY	● IgG4-related kidney disease (including tubulointerstitial hypocomplementemic nephritis [TIN] and membranous glomerulonephritis [GN] secondary to IgG4-RD)
BRAIN	● IgG4-related hypophysitis ● IgG4-related pachymeningitis ● IgG4-related midline destructive disease (palate, nasal septum, head and neck)
LYMPH NODES	●Asymptomatic IgG4-related lymphadenopathy

In EP, pancreatic CT scan may be normal (Balthazar score = 0) [21, 25, 26], may present pancreatic duct dilatation, or may frequently manifest a pseudocyst or pseudotumoral form [3-7, 9, 13-15, 29].

The histological features of eosinophilic pancreatitis have two distinct forms:

- widespread eosinophilic infiltration in pancreatic ducts, in the acini and interstitium with phlebitis and eosinophilic arteritis, and with the minimal presence of lymphocytes and plasma cells;

- intense and localized eosinophilic infiltrate with pseudocyst formation [8, 16].

A systematic search of PubMed for original and review articles in English published during 1990 to June 2019 was performed using keywords and text related to EP and AIP, which included "eosinophilic pancreatitis", "autoimmune pancreatitis", "diagnosis", "case reports", "IgG4-related disease", "rare causes", and "recurrent acute pancreatitis".

There were total 40 reports of EP appearing from the following countries: USA [7, 8, 14, 15, 18, 25, 28, 78, 100], Turkey [3, 17], China [16, 22], Korea [13, 21], France [9, 11, 77, 101], India [6, 27], Japan [20, 24, 106], Denmark [18], UK [5], Brazil [29], Italy [26], and Tunisia [76].

If IgG4 is elevated, more than twice of the upper limit of the normal value (usually IgG4 is around 5–6%, with normal values up to 135 mg/dL), it could be autoimmune pancreatitis.

The diagnostic criteria for the AIP are indicated in **Box 2**.

The criteria for the diagnosis of autoimmune pancreatitis (HISORt criteria [81, 82]):

H	Histology suggestive of autoimmune pancreatitis
I	Pancreatic imaging suggestive of autoimmune pancreatitis (CT and/or cholangiopancreatography MRI)
S	Serology IgG4 ≥2 times of the upper limit
O	Other organ involvement (biliary stenosis, parotid/lacrimal gland involvement, mediastinal lymphadenopathy, retroperitoneal fibrosis)
Rt	Response to steroid treatment and resolution or a marked improvement in pancreatic and extrapancreatic manifestations

Autoimmune pancreatitis is classified into type 1, type 2, and type NOS (not otherwise specified). AIP type 1 is a more severe condition than the other subtypes [89].

The clinical manifestations and epidemiological and histological features of autoimmune pancreatitis are described in **Tables 4** and **5**.

Table 4 Clinical manifestations of autoimmune pancreatitis (AIP).

Pancreatic	- A pancreatic mass that can be confused with pancreatic carcinoma or with lymphoma
	- Mild to moderate abdominal pain with or without attacks of acute pancreatitis or chronic pancreatitis
	- Recurrent pancreatitis is common and appears more frequently in patients with focal pancreatitis than the diffuse form. Although the recurrence is common, AIP is not a frequent cause of recurrent pancreatitis

	in the Western countries
	- Stenosis of the pancreatic duct
	- Peripancreatic vascular complications (rare)
Of the biliary tract	- Obstructive jaundice (IgG4-associated cholangitis)
Other events	-Sjögren syndrome
	- Pulmonary nodules
	- Autoimmune thyroiditis
	- Interstitial nephritis
	- Retroperitoneal fibrosis

Table 5 Epidemiological and histological features of the two forms of autoimmune pancreatitis (AIP), type 1 and type 2. Modified from Shimosegawa et al. [83].

Autoimmune Pancreatitis	Type 1 AIP	Type 2 AIP
Average age	6th decade	4th decade
Sex	Predominantly Male	Both equally
Histological pattern	LSP (LymphoPlasmacytic Sclero sing Pancreatitis) or Lobulocentric Pancreatitis	ICDP (Idiopathic Duct-Centric Pancreatitis)
Histological features	Infiltrated lymphoplasmacellular	Infiltrated lymphoplasmacellular
	periductal	GEL (Granulocytic Epithelial Lesion)
	Storiform fibrosis	Ductal obstruction
	Obliterative vasculitis	
Plasma cells IgG4+ (immunohistochemistry)	+++	-/+
Serum IgG4 (> 135 mg/dL)	Increased (50–70%)	Normal
Muti-organ involvement	Sclerosing sialadenitis	Chronic inflammatory diseases of the intestine (IBD)
	Sclerosing cholangitis	
	Retroperitoneal fibrosis	
	Interstitial nephritis	

Treatment outcome	Excellent response to steroid but recurrence is common	Excellent response to steroid and recurrence is rare

The most common morphological aspect, detected in 85% of patients, is given by a pancreatic mass or by the enlargement of the entire pancreas [84]. Therefore, the confirmatory diagnosis is made by pancreatic biopsy.

Some recent studies from Japan have shown that with the minimally invasive technique of the needle aspiration under echo-endoscopic guidance [29, 85], it is possible to formulate very accurate histopathological diagnosis.

In the differential diagnosis, with non-invasive technique, between autoimmune pancreatitis and pancreatic cancer, the use of contrast-enhanced harmonic endoscopic ultrasound (CEH-EUS) was found to be useful [86].

If it is important to make the differential diagnosis with pancreatic neoplasia [81], the possible association of AIP, in the presence or during the remission phase, with pancreatic cancer [87, 88] should be considered.

Furthermore, the detection of certain antibodies may help in the diagnosis of AIP:

- anti-plasminogen-binding protein-peptide antibodies (anti-PBP),
- anti-carbonic anhydrase II antibodies (anti-CA-II),
- anti-carbonic anhydrase IV antibodies (anti-CA-IV), and
- anti-lactoferrin antibodies (anti-LF).

Anti-PBP antibodies were found positive in 94% of cases AIP in comparison to the absence in the control patients [90].

The pathologic differential diagnosis of Immunoglobulin G4-related disease is indicated in **Table 6**.

Table 6 Conditions that can mimic IgG4-related disease histopathologically. Adapted from Bledsoe et al. [91].

▪ **Infections**	Bacterial
	Mycobacterial
	Viral
	Spirochetal, e.g., syphilis
	Infections involving specific sites, such as aortitis, otitis media, mastoiditis, etc.
▪ **Tumors**	Inflammatory myofibroblastic tumor
	Inflammatory infiltrate in the background of tumors
▪ **Lymphoproliferative disorders**	MALT lymphoma with plasmacytic differentiation
	Plasma cell neoplasia

▪ **Eosinophilic disorders**	Eosinophilic angiocentric fibrosis
	Kimura's disease
	Angiolymphoid hyperplasia with eosinophilia (ALHE)
▪ **Inflammatory/Autoimmune disorders**	Inflammatory pseudotumor
▪ **Systemic disease**	Multicentric Castleman disease
▪ **Rosai–Dorfman disease (Sinus Histiocytosis with Massive Lymphadenopathy: SHML)**	Sarcoidosis
	ANCA-associated vasculitis
	Granulomatosis with polyangiitis (Wegener's dieses)
	Eosinophilic granulomatosis with polyangiitis (Churg-Strauss)
▪ **Pancreatobiliary tract**	Primary sclerosing cholangitis
	Type 2 AIP
	Follicular cholangitis
▪ **Orbit/Salivary glands**	Sjögren syndrome
	Chronic sialadenitis, not otherwise specified

3. Therapy

The therapy of autoimmune pancreatitis involves the use of glucocorticoids.

In the literature, different dosages and durations of the therapies have been empolyed:

A. Prednisolone: 30–40 mg/day for 2–4 weeks with doses of 5 mg/day every 2–4 weeks up to 5 mg/day; maintenance dose: 2.5–5 mg/day [92];

B. Prednisolone: 0.5 mg/kg body weight/day for two weeks [93];

C. Prednisone: 40 mg/day for 4–6 weeks with 5 mg weekly doses per week for a total of 11 weeks (initial treatment in IgG4-associated cholangitis) [94].

It is also possible to use immunomodulatory drugs, such as azathioprine, in case of Ig4-associated cholangitis (2 mg/kg/day for 1–3 years). In the event of steroid failure or relapse or AIP without IgG4-cholangitis, the monoclonal anti-CD20 antibody, rituximab, can be administered [95]. Recently, a clarification on the treatment of autoimmune type 1 and type 2 pancreatitis has been published by Okazaki et al. in January 2017 [96].

For the treatment of IgG4-related disease, an international consensus was published in 2015 that analyzed various pharmacological options reported so far [97]. In the study, in addition to cortisone therapy, the role of conventional steroid-sparing drugs was defined (azathioprine, mycophenolate mofetil, 6-mercaptopurine, methotrexate, tacrolimus, and cyclophosphamide) and the use of rituximab was suggested as a depletor of B cells.

A study from Italy in 2015 [98] reported methotrexate as a promising, safe and low-cost agent to maintain glucocorticoid-induced remission of IgG4-related disease with systemic involvement.

Regarding the IgG-4 sclerosing cholangitis, the Japanese guidelines [99] were published in February 2019, which defined, among other things, the classification of the same in four subtypes, the relationship with the inflammatory hepatic pseudotumors, the development of cholangiocarcinoma, algorithms for diagnosis and treatment, and the differential diagnosis with primary sclerosing cholangitis (PSC).

The most common therapy of eosinophilic pancreatitis involves the use of prednisolone 40 mg/day [17, 19] for 2–3 weeks. Caglar et al. described a scheme that suggests the use of prednisone 40 mg/day for six weeks with ketotifen 500 mg/day, with a dose of 4 mg every two weeks to a maintenance dose of 16 mg/day [17].

Maeshima et al. instead described a scheme with prednisolone 30 mg/day + montelukast 10 mg/day for one month [20]. However, there are currently no reliable data to establish the duration of therapy. It is also possible to make the use of alternatives of steroids, such as sodium cromoglycate [11, 17], ketotifen [11], montelukast [11, 20], azathioprine, and hydroxyurea.

4. Conclusions

- The number of drugs involved in acute pancreatitis is constantly increasing.
- Eosinophilic pancreatitis is a very rare but benign pathology as only about forty cases have been described in the literature. It is often associated with hypereosinophilia and eosinophilic gastroenteritis.
- It is considered a variant of autoimmune pancreatitis. The diagnosis requires an instrumental technique (ultrasound, CT, MRCP, EUS, and also ERCP) up to biopsy, considering the frequent initial pseudo-diagnosis of pancreatic neoplasia and the consequent lack of preoperative suspicion. The therapy involves the use of corticosteroids.
- Autoimmune pancreatitis is a rare disease that is classified into three forms: type 1, type 2, and type NOS. Type 1 is associated with the involvement of multiple organs in the IgG4-related disease. It may occur in a tumor-like form or may also be associated with pancreatic neoplasia. A dosage of IgG4 is helpful in the diagnosis that in any case will need instrumental analysis up to the pancreatic biopsy.

Therapeutic options include cortisone, steroid-sparing drugs, immunomodulatory agents, and the monoclonal antibody, rituximab.

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Glossary Abbreviations

CT: Computed tomography
EUS: Endoscopic ultrasound
CEH-EUS: Contrast-enhanced harmonic endoscopic ultrasound
EUS-FNA: Endoscopic ultrasound-guided fine needle aspiration
ERCP: Endoscopic retrograde cholangiopancreatography
MRI: Magnetic Resonance Imaging
MRCP: Magnetic Resonance Cholangiopancreatography
EGID: Eosinophilic Gastrointestinal Disordes
EoG: Eosinophilic Gastroenteritis
EoE: Eosinophilic Esophagitis
EoC: Eosinophilic Colitis
PCE: Primary Colonic Eosinophilia
EP: Eosinophilic Pancreatitis
AIP: Autoimmune Pancreatitis
DIP: Drug Induced Pancreatitis
HE: Hypereosinophilia
HES: Hypereosinophilic Syndrome
HEUS: Hypereosinophilic Syndrome of Unknown Significance
HEFA: Hereditary (familial) HE
HER: Secondary (reactive) HE
IHES: Idiopathic Hypereosinophilic Syndrome
HTGP: Hypertriglyceridemia-induced acute pancreatitis

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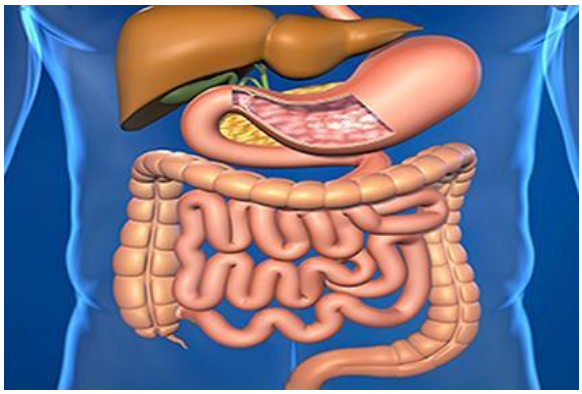

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