

Nutritional Management of Pancreatic Disease Virtual Study Day

Nutritional Management of Chronic Pancreatitis

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Declaration of Interest: Honoria received for presenting from Viatrix

Overview

- What is Chronic pancreatitis?
- Drivers of Malnutrition in Chronic Pancreatitis
- Nutritional Assessment and Management
- Diagnosing Pancreatic Exocrine Insufficiency – the factors to consider
- Pancreatic **E**nzyme **R**eplacement **T**herapy

Abbreviations

- ALD** = Alcoholic Liver Disease
- BMI** = Body Mass index
- BO** = Bowel opening
- CF** = Cystic Fibrosis
- CP** = Chronic Pancreatitis
- EN** = Enteral Nutrition
- ESPEN** = European Society for Enteral and Parenteral Nutrition
- FE-1** = Faecal Elastase
- GI** = Gastrointestinal
- GORD** = Gastro Oesophageal Reflux Disease
- HOP** = Head of Pancreas
- NBM** = Nil by Mouth
- NJ** = Naso-jejunal
- ONS** = Oral Nutrition Supplements
- PEI** = Pancreatic Exocrine Insufficiency
- PERT** = Pancreatic Exocrine Replacement Therapy
- PN** = Parenteral Nutrition
- PPI** = Proton Pump Inhibitor

ESPEN Guidelines

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ESPEN Guideline

ESPEN guideline on clinical nutrition in acute and chronic pancreatitis

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Consensus for the management of pancreatic exocrine insufficiency: UK practical guidelines

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ABSTRACT

Introduction Pancreatic exocrine insufficiency is a finding in many conditions, predominantly affecting those with chronic pancreatitis, pancreatic cancer and acute necrotising pancreatitis. Patients with pancreatic exocrine insufficiency can experience gastrointestinal symptoms, maldigestion, malnutrition and adverse effects on quality of life and even survival.


There is a need for readily accessible, pragmatic advice for healthcare professionals on the management of pancreatic exocrine insufficiency.

Methods and analysis A review of the literature was conducted by a multidisciplinary panel of experts in pancreatology, and recommendations for clinical practice were produced and the strength of the evidence graded. Consensus voting by 48 pancreatic specialists from across the UK took place at the 2019 Annual Meeting of the Pancreatic Society of Great Britain and Ireland annual scientific meeting.

Results Recommendations for clinical practice in the diagnosis, initial management, patient education and long term follow up were developed. All recommendations achieved over 85% consensus and are included within these comprehensive guidelines.

with improved survival and quality of life (QoL) in patients with PEI.^{8–10}

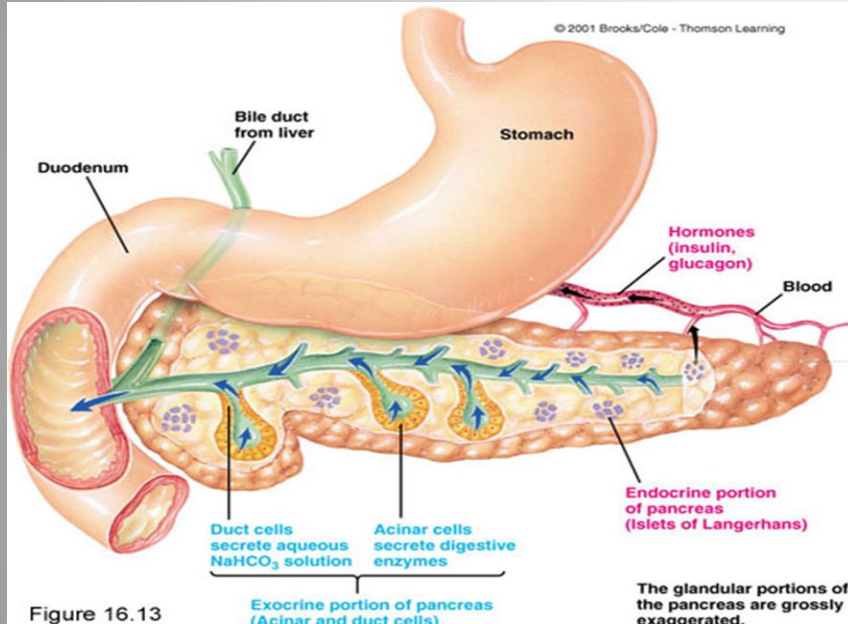
PEI may be underdiagnosed and undertreated in the UK, as demonstrated in other European countries.¹¹ Patient support groups report management of PEI as the most common concern raised on their patient helpline (Pancreatic Cancer UK, 2015), and ‘difficulty in managing GI problems, diet and digestion’ are documented as the primary unmet need in patients with pancreatic cancer (PC).¹² In addition, patients with chronic pancreatitis (CP) feel unsupported by healthcare professionals (HCPs) in the management of PEI (Pancreatitis Supporters Network, 2015). Consequently, there is a need for readily accessible, pragmatic advice for both specialist and non-specialist HCPs. The aim of this article is to provide evidence-based guidance on the diagnosis and management of PEI, including differential diagnosis and follow-up. This article does not make detailed recommendations regarding the management of cystic fibrosis (CF) as this is



Pancreatic Exocrine Insufficiency and Diabetes Masterclass
May 2019

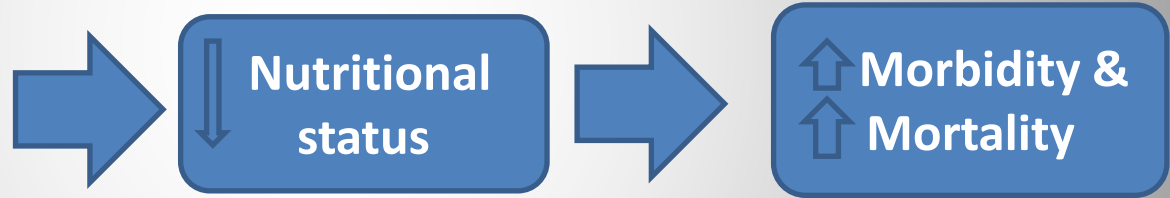
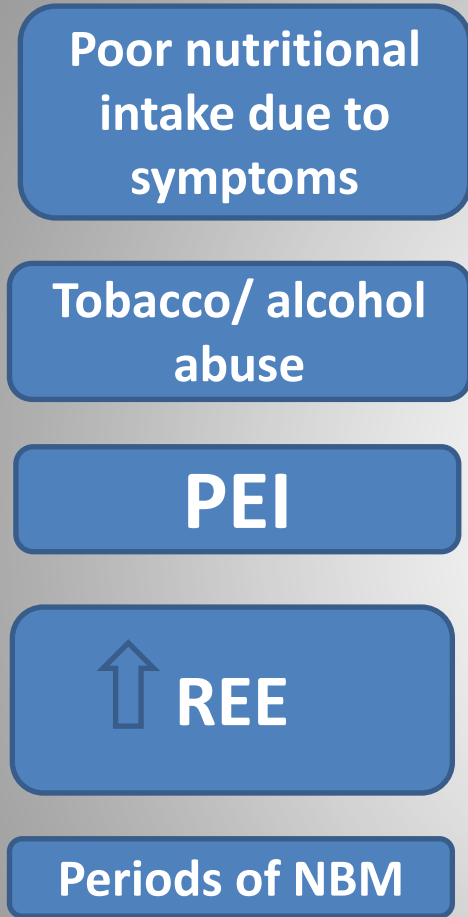
Professor J. Enrique Domínguez-Muñoz
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Chronic Pancreatitis (CP)



Chronic pancreatitis is a disease with **progressive** and **irreversible inflammatory changes** in the **pancreas** that result in **permanent structural damage** with **fibrosis**, which can lead to **impairment of exocrine (PEI)** and often **endocrine function**.

Drivers of malnutrition in Chronic Pancreatitis



Malnutrition is often a late but important manifestation in the course of CP and depends on the intensity and duration of the underlying disease

Malnutrition in CP

Both pain and loss of pancreatic function can lead to malnutrition

Lohr 2017

26% of patients underweight

Olesen 2017

Sarcopenia present in 17%. Sarcopenia associated with an increase in hospitalisation and reduced survival.

Olesen 2017

>50% overweight/obese however had lower muscle stores & reduced hand grip strength

Duggan et al 2014

Dietetic aims

The main goals of nutritional therapy are to:

- Prevent undernutrition/ improve nutritional status
- Reduce symptoms of maldigestion +/- malabsorption
- Prevent micronutrient deficiencies

Most patients with CP admitted to hospital will require alternative feeding i.e. enteral feeding or TPN?

True or False?

Nutritional Management of CP

- Effective management of pain and **abstinence from alcohol** can improve nutritional status on their own
- **80%** of patients can be managed with **normal food supplemented by PERT** Meier 2006
- **10-15%** will require **ONS**
- **5%** of patients will require **EN** Gianotti et al 2009
- **<1%** of patients will require **PN** Gianotti et al 2009

Food first

- **Dietary counselling as effective as ONS** at improving CP patient's nutritional status
Singh et al 2008
- **No need for dietary fat restriction** unless steatorrhoea cannot be controlled
ESPEN 2020
- CP patients with **normal nutritional status** (usually early stage) should adhere to a **well balanced diet**
ESPEN 2020
- **Malnourished** patients should be advised to consume high protein, high energy food in **five to six small meals per day**
ESPEN 2020, PEN Canada 2022
- **High fibre diets are not recommended** as they may inhibit PERT, and result in **malabsorption**
Dutta 1985, PEN Canada 2022

ONS and CP

- **Oral** nutrition support with dietary counselling is usually **sufficient** to improve nutritional status

Singh et al 2008



- **ONS** should be prescribed to undernourished patients **only if oral nutrition is insufficient for reaching the calorie and protein goals**

ESPEN 2020

- **MCT** would seem theoretically to have **potential advantage over LCT**. MCTs are less dependent on Lipase for their absorption

Caliari 1993, ESPEN 2020



- MCTs have an unpleasant taste and are associated with adverse effects like cramps, nausea and diarrhoea

Caliari 1993, 1996, ESPEN 2020

Enteral nutrition (EN)

- EN is indicated if patients with malnutrition are **not responding to oral nutrition support** ESPEN 2020
- **NJ** should be used in patients with pain, delayed gastric emptying, persistent nausea or vomiting and gastric outlet syndrome ESPEN 2020
- Semi- elemental formulas with **MCT** can be used **if standard formulas are not tolerated** ESPEN 2020
- If **no improvement with MCT** formulae, **PERT** should be introduced alongside Ferrie 2011
- Where needed, **enteral feeds should be peptide** and medium-chain triglyceride-based (grade 2C; 100% agreement) Phillips 2021
- **Patients with PEI receiving enteral feeds usually tolerate semi-elemental (peptide) preparations.** However, where malabsorption symptoms persist, enzymes can be flushed via the feeding tube every 2 hours or added to the feed itself (93% agreement) Phillips 2021
- **Jejunal feeding may require PERT** more so than gastric feeding alongside peptide feed as less stimulation of the pancreas Phillips 2021

Parenteral nutrition (PN)

- PN may be indicated in patients with
 - Gastric outlet obstruction
 - A need for gastric decompression
 - No enteral access to the jejunum
 - Complex fistulating disease
 - Intolerance to EN or full needs cannot be met enterally

The preferred route is central venous access

Nutritional requirements in CP

	Requirements
Energy	25kcal/ kg BMI 18.5-30kg/m ² and <65 years (Dickerson et al, 1991)
Protein	1-1.5g/kg/day (Imrie et al, 2010)
Fat	No need for fat restriction unless symptoms of steatorrhea cannot be controlled (ESPEN 2020) 30% of total calories can be given as fat (Duggan et al, 2010) Supplementation with MCT fat source may be useful (Giger et al, 2004, Duggan et al, 2010) with gradual introduction and monitoring
Carbohydrate (CHO)	Diets rich in CHO advised unless CP concurrent with Diabetes (Duggan et al 2010)
Fibre	Very high fibre diets (>25g/day) are not recommended as they may absorb enzymes and delay nutrient absorption (Dutta 1985)
Fat soluble vitamins (A,D,E &K) vitamin B12, folate & thiamine, iron, ferritin, selenium, zinc, copper	Micronutrient deficiencies are prevalent in patients with CP (Sikkens et al, 2013; Duggan et al, 2014) Insufficient evidence to suggest routine/ blind supplementation of micronutrients in absence of malabsorption Should be assessed and monitored with supplementation, if deficiencies are present (Duggan et al, 2010, Lohr et al, 2017 ESPEN 2020) In community setting may be difficult to obtain micronutrient screen.
Alcohol	Life long abstinence recommended

PEI

Globally agreed definition across pancreatic societies:

PEI is defined as a reduction of pancreatic exocrine activity in the intestine at a level that prevents normal digestion

Hoffmeister 2015, Toouli 2010, Phillips 2021

The most common cause of PEI is CP due to loss of pancreatic parenchyma and reduced secretion

Phillips et al 2021

22-94% of prevalence rates for PEI – due to wide range of PEI markers used in studies

ESPEN 2020

PEI may go undetected because the signs and symptoms are **similar to** those of other **GI conditions**

The degree of PEI correlates with disease severity

Dumasy et al 2004

Steatorrhea may not become apparent until greater than 90% of pancreatic function is lost

DiMagno et al, 1973

Pancreatic Exocrine Insufficiency Diagnosis in Clinical Practice



Symptoms of
malabsorption

Nutritional
markers

PANCREATIC EXOCRINE
INSUFFICIENCY

Faecal
elastase

Diagnosing PEI in CP

A number of factors need to be considered to reliably diagnose PEI:

- GI symptoms
- Anthropometry
- Nutritional Markers
- Faecal Elastase
- Stage of CP
- CT Findings

GI / Clinical Symptoms of PEI

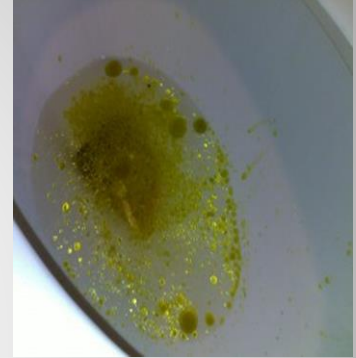
- Large volume stool
- Undigested food in the stool
- Post-prandial abdominal pain
- Offensive smelling stools
- Nausea / colicky abdo pain
- GORD
- Bloating/flatulence
- Weight loss despite good oral intake
- Hypoglycaemia in patients with Diabetes

O'Keefe et al 2001, Genova Diagnostics 2008, Freiss & Michalski 2009

GI Symptoms of PEI - Steatorrhoea

- Loose watery yellow/orange stool
- Floats / difficult to flush away
- Oily / visible food particles

- VERY LATE SYMPTOM



However... these symptoms can often be masked by other medications and self imposed fat restrictions

Anthropometry in Diagnosing PEI

- There is no agreed diagnostic % weight loss for PEI
- A single BMI measurement is of limited diagnostic use on its own and can be normal in patients with PEI
- BMI should not be used solely because it does not take sarcopenia into account in the obese patients with CP
- Evidence for recording specific anthropometric measurements is poor or absent for patients with PEI with limited data in CP

Phillips et al 2021

- I feel % weight loss is the most useful anthropometric calculation to aid with diagnosing PEI

Nutritional Deficiencies in Predicting PEI

Most Sensitive Nutritional Markers:

- Low Magnesium
- Low Vitamin E
- Low Retinol Binding Protein / Vitamin A

Phillips 2021

“The higher the number of nutritional deficiencies the higher the chance of PEI”

Professor Dominguez-Munoz 2019

Nutritional Deficiencies in Predicting PEI in CP

Low levels of vitamin E reported in CP patients with PEI or steatorrhoea

Kalvaria 1986, Marotta 1994, Nakamura 1996

Retinol-binding protein (a marker of vitamin A status) may be lower in patients with alcohol induced CP and steatorrhoea vs patients with CP alone.

Marotta 1994

Magnesium and zinc deficiency have been reported in CP, but not specifically in patients with PEI

Lindkvist 2012, Vujasinovic 2019

Vitamin D deficiency has been shown to have a high incidence in CP (53%–66%) but no significant difference when PEI is present.

Duggan 2014, Marotta 1994, Sikkens 2013, Dutta 1982

Given the evidence available, no finding is specific enough to recommend using serum micronutrients alone as a diagnostic marker for PEI.

CT Findings in diagnosing PEI

Morphological changes of CP including calcification and main pancreatic duct dilatation can be identified on CT scanning

Pancreatic calcification is a late/severe feature of CP with PEI present in 50% of patients with substantial calcification

Scuro 1990, Lankisch 1986

Only 47% of patients with severe PEI & CP were shown to have significant **atrophy** and **ductal dilatation** on CT

Malfertheiner 1986,1989

Ductal dilatation diagnosed via ERCP has a stronger association with PEI than calcification

Dominguez-Munoz 1995

Therefore, although radiological evidence of pancreatic morphological abnormalities is supportive of a diagnosis of PEI, further evidence is required.

Stage of CP in diagnosing PEI

The latency between onset of first symptoms and signs of CP, including pain and malabsorption / malnutrition is between 5-10 years in alcoholic, but delayed in non-alcoholic CP

Hao 2018

PEI is reported in 94% of patients within 10 years of CP onset

Dumasy 2004

In CP, progressive destruction of the pancreatic tissue results in PEI

Keller 2005

Basically the longer the patient has CP, the higher the likelihood of PEI given its progressive nature

Faecal Elastase (FE-1)

- Several tests for PEI exist – eg Coefficient of fat absorption, indirect C-labelled mixed triglyceride breath test
- FE-1 is used in clinical practice - less invasive & readily available
- Only small sample of faeces required
- Stable at room temperature for 3 days
- The lower the FE-1 concentration, the higher the probability of PEI.

Faecal Elastase

- Analysed in RVH Paediatric Biochemistry Lab
(Ph 9063 2148/3064)
- Ideally should be **a formed motion** to suit sample technique!!
- Type 7 BO can be analysed but likely result will be “Not Reportable”
- **Watery sample can cause a false positive result!** Consider repeat!

Community Faecal Elastase Testing

- Recently some GPs refusing to organise FE-1 unless a labelled request form received from Referring Consultant
- Not all patients requiring FE-1 have a Consultant!
- Dietitian can interpret results!

Faecal Elastase Test

Sensitivity

- 82-100% in severe pancreatic disease
- 33-100% in moderate pancreatic disease
- 25-65% in mild pancreatic disease

Specificity

- 55-100% in mild pancreatic disease

Detection

- < 200ug/g stool – moderate pancreatic disease
- < 100ug/g stool – severe pancreatic disease



Potential for Screening for Pancreatic Exocrine Insufficiency Using the Fecal Elastase-1 Test

J. Enrique Domínguez-Muñoz¹ · Philip D. Hardt² · Markus M. Lerch³ · Matthias J. Löhr⁴

“Physicians should be aware that an exact cut-off of FE-1 levels for PEI in different clinical scenarios cannot be established, and that FE-1 levels should be considered together with an appropriate evaluation of symptoms, signs, and nutritional status”.

“FE-1 level of 200-250 is likely not normal. 500 is normal”

Dominguez-Munoz et al 2017

Considered alone FE-1 shows a problem with enzyme secretion only

Dominguez-Munoz – Masterclass 2019

All patients with Chronic Pancreatitis should have a faecal Elastase Test to diagnose PEI?

Yes or No?

When is FE-1 required/ not required?

Required

- **Patients with GI symptoms of maldigestion with or without known associated conditions**
- **Maldigestion** symptoms: steatorrhoea, wt loss, diarrhoea, abdo pain or bloating
- **Associated conditions:** patients with coeliac disease, IBS, HIV, Type 1 DM and acute severe pancreatitis after initial presentation

My opinion:

In early/mild CP consider FE-1 if symptoms non specific

Not Required

- **Steatorrhoea / malabsorption symptoms in pts with CP with dilated pancreatic duct or severe pancreatic calcification**
- Severe necrotising Pancreatitis
- Post-op Total Pancreatectomy
- HOP Cancer

Phillips et al 2021

PEI reported in 94% of patients within 10 years of CP onset so FE-1 not then required

Dumasy 2004

Pancreatic Exocrine Insufficiency Diagnosis in Clinical Practice



Symptoms of
maldigestion

Nutritional
markers

**PANCREATIC EXOCRINE
INSUFFICIENCY**

Faecal
elastase

Combined Factors for PEI

Consider all factors in pie chart in patients with any condition or disease potentially causing PEI +/- changes in anthropometry + **poor Glycaemic Control in patients with DM.**

- Abnormal FE-1 alone = **Not PEI**. Check other factors
- Abnormal FE-1 and Symptoms of Maldigestion – **most likely PEI**
- Normal FE-1 and Symptoms of Maldigestion = **Not PEI** (? Bacterial overgrowth)
- Abnormal FE-1 and Abnormal Nutritional markers = **Most likely PEI**
- Normal FE-1, Symptoms of Maldigestion and Abnormal Nutritional markers = **Most likely PEI** as FE-1 can be falsely negative
- Abnormal FE-1, Symptoms of Maldigestion, and Abnormal Nutritional markers = **Definite PEI**

Case Study

- 34 year old with CP x 3-4 years and alcoholic decompensated ALD (no ascites)
 - Poor oral dietary intake pre-admission to hospital
 - Exceeding energy needs orally since admission
 - BMI 22kg/m². 3.1% weight loss in 10 days since admission
 - Reports BO x 3-4 / day (brown in colour and semi-formed) since admission
 - On laxatives as per GI Dr to prevent encephalopathy
 - Denies wind / bloating / abdo discomfort on eating
 - Pancreatic ductal dilatation in the body and tail on CT scan
-
- Q. Is a Faecal Elastase required?

Case Study Discussion

- Q. Is a Faecal Elastase required in this case?
- Factors considered in decision making:
 - ? Malabsorbing as losing weight despite exceeding full energy needs orally
 - ? Frequent Semi – formed BO secondary to PEI or laxatives
 - ? Abdo discomfort masked by analgesia
 - Pancreatic ductal dilatation in the body and tail on CT scan – strong association with PEI
 - Only has had CP x 3-4 years so not advanced disease
 - Pill Burden of PERT
- Yes a Faecal Elastase is required to prevent ? unnecessary pill burden
- FE-1 Result: 189 = moderate PEI. PERT introduced.
- I would not have requested FE-1 if no laxatives involved in this case as so many of the above factors suggesting PEI. I would have introduced Creon as first line treatment.

PERT

- **PERT (Pancreatic Enzyme Replacement Therapy) is the mainstay treatment of PEI**
- All products derived from pigs! Non-porcine products failed in Phase III clinical trial. "Organisations representing Jewish and Muslim communities have said that these treatments are acceptable to use" – PCUK
- **Creon (Pancreatin) generally used first – line.**
- **Start PERT at 44,000-50,000 with meals and snacks.**
- **Remember PERT also with ONS eg 50,000 with sip feed.**
- **Fat free ONS may also need PERT due to CHO malabsorption**

PERT

- Like CP, exocrine insufficiency is progressive and doses escalate with time
- Some patients need really high doses (> 150, 000 units with a meal = > 25 capsules / day = 9-10 x 100 cap tubs per month)
- There is **no maximum dose** proposed for adults or children with non-CF related PEI
- Significant pill burden
- Treat like insulin – different doses for different patients for different meals

Phillips 2021

Presentation by Phillips

What if symptoms do not improve?

- **? Adequate dose** - minimum dose 44 000 - 50 000 with meals and 25 000 per snack. Consider increasing dose.
- **? Correct timings** **? Divided doses** - spreading PERT throughout a meal rather than consuming before or after a meal
- **? Able to swallow** - if capsules unable to be swallowed, they should be opened, placed on an acidic puree (eg fruit puree/ fruit yogurt/ juice ONS) and swallowed at intervals. Granules should not be chewed or crushed, as this removes the enteric coating, resulting in premature activation of the enzymes. The mouth should be rinsed with cool water to prevent ulceration

What if symptoms do not improve?

? **Compliance with advice** – should be swallowed with a cold drink. Ensure capsules are stored at $<25^{\circ}\text{C}$

? **Taking too much fibre** - very high fibre diets ($>25\text{g} / \text{day}$) may affect enzymes and delay nutrient absorption so not recommended

? **Alternative PERT product needed** – consider alternative eg Nutrizym, Pancrex V, Pancrease if the PERT dose is $>10,000$ units lipase/kg/day or $100,000$ units lipase per meal or no improvement with increasing doses

Phillips 2021

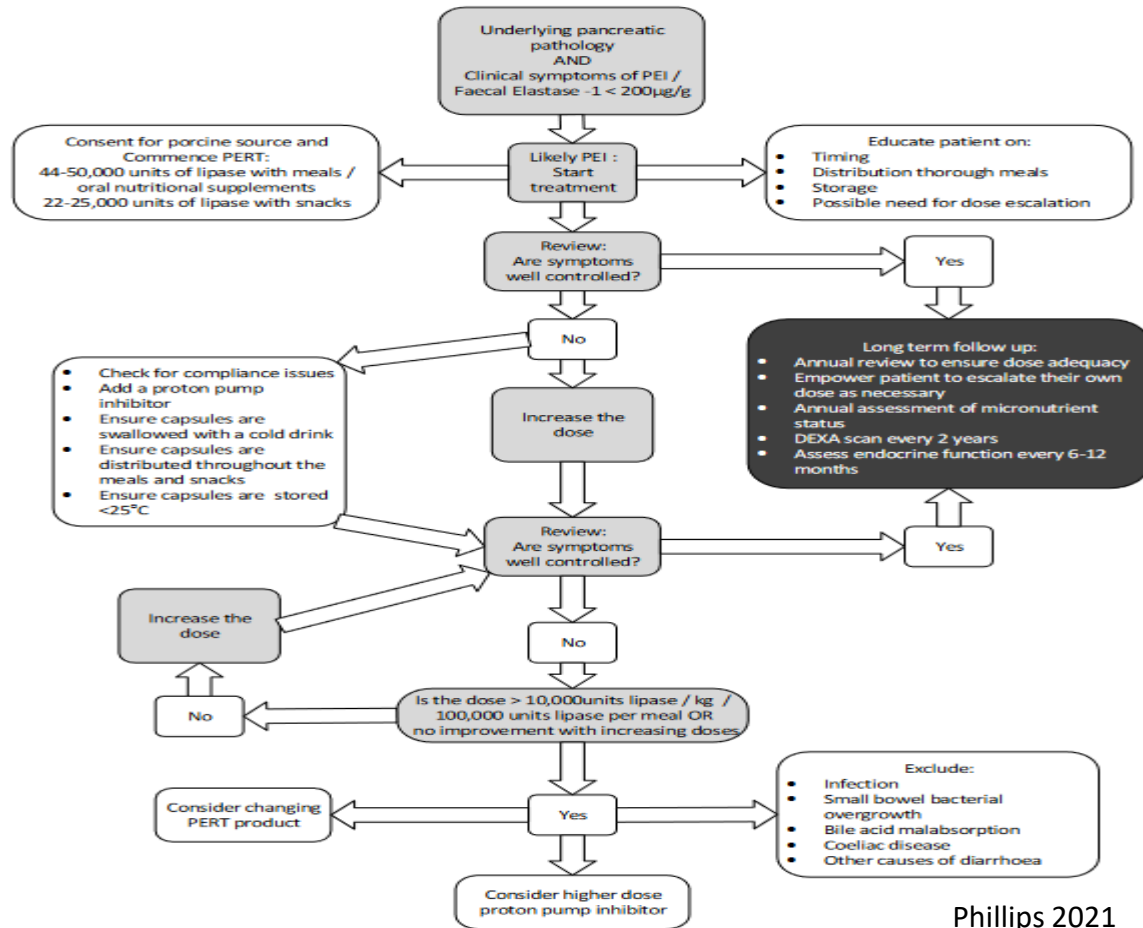
? **Exclude other causes if exceeding above stated doses** – such as infection / SBBO / BAM / Coeliac Disease / other causes of diarrhoea

? a PPI is required

The addition of PPI

- The addition of PPI can be beneficial if PERT is not effective, as PERT may be inhibited by gastric acid
- However data is not consistent
- Given conflicting data and potential for long-term complications of PPI use eg C-diff, low Mg, nausea and diarrhoea, the addition of **PPI is a second-line treatment**
- **Introduce PPI once daily if not responding to PERT alone. May require PPI twice daily** if the PERT dose is >10,000 units lipase/kg/day or 100,000 units lipase per meal or no improvement with increasing doses or no improvement with trial of alternative PERT product
 - Dominguez-Munoz recommends **Omeprazole 20mg twice daily** before breakfast and evening meal if twice daily is needed

Phillips 2021



PERT in CP and Survival

PERT has been shown to improve fat and nitrogen absorption, nutritional parameters and GI symptoms

Ramesh 2013

PEI has been shown to be an independent factor related to **mortality** in patients with CP

de la Iglesia - Garcia 2018

An absence of PERT on discharge was an independent risk factor for **survival** in those undergoing surgery for CP

Winny 2014

Bone Health & CP

- The prevalence of osteoporosis is 1 in 4 and 2 out of 3 can experience osteopathy Duggan et al 2014
- Offer a DEXA every 2 years Phillips et al 2021
- Basic preventative measures:
 - Optimise Calcium and Vitamin D sources
 - Correct dosing of PERT
 - Regular weight bearing exercise
 - Smoking/ alcohol cessation Lohr et al 2017

Routine Follow-up of patients with PEI

- Annual review to ensure PERT dose adequacy
 - Empower patient to escalate their own PERT dose as necessary
 - Annual assessment of micronutrient screen
 - DEXA scan every 2 years as high prevalence of Osteoporosis and Osteopenia with PEI
 - Assess endocrine function every 6-12 months
-
- Screening may need to occur more frequently in those with severe disease or uncontrolled malabsorption

Phillips 2021

ESPEN 2020

Table 5

Nutritional assessment in the patient with chronic pancreatitis.

Anthropometric assessment	Biochemical assessment	Symptom assessment	Body composition
<ul style="list-style-type: none">• Change in body weight• Functional assessment: Hand-grip strength dynamometry/6-min walk tests/sit to stand tests.• Skin fold thickness, waist circumference and mid arm muscle circumference.• Presence of ascites/edema	<ul style="list-style-type: none">• Fat soluble vitamins (A, D, E, K)• Bone health (Parathyroid hormone)• Trace elements (magnesium, selenium, zinc)• Anemia screen (iron studies, B12, folate, ferritin and CRP)• Glycemic control: HbA1c and random glucose	<ul style="list-style-type: none">• Change in dietary intake• Appetite• Presence of symptoms that impact on oral intake (nausea/pain/indigestion/early satiety)• Presence of exocrine/endocrine dysfunction	<ul style="list-style-type: none">• CT/US imaging of muscle stores (muscle mass)• DXA scanning (bone mineral density)

CRP = C-reactive protein, HbA1c = hemoglobin A1c, CT = computed tomography, US = ultrasound, DXA = dual-energy X-ray absorptiometry.

Take Home Messages

- Chronic pancreatitis is a disease with progressive and irreversible inflammatory changes
- Malnutrition is often late in the course of CP & depends on the intensity & duration of the underlying disease
- The most common cause of PEI is CP
- A number of factors need to be considered to reliably diagnose PEI
- Faecal Elastase is not always required to diagnose PEI
- Permission should be given to patients to dose escalate PERT
- Annual assessment of micronutrient screen
- **Appropriate therapy improves outcomes.**

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