Evidence-based solutions to clinical problems

Nutritional management of chronic pancreatitis

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Tallaght Hospital
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• Drivers of malnutrition in chronic pancreatitis
• Defining malnutrition
• Micronutrient deficiency
• Bone health in chronic pancreatitis
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• Endocrine dysfunction
• Exocrine dysfunction and type 3c diabetes
• Algorithm for nutritional assessment
Background

**Definition**
CP is defined as a continuing inflammatory disease of the pancreas characterised by irreversible morphological changes that typically cause abdominal pain and/or permanent loss of pancreatic function.

**Pathology**
- Progressive exocrine atrophy, with replacement of the normal pancreatic tissue with fibrous tissue leading to gland enlargement.
- Main duct dilated, tortuous and strictured.
- Gland becomes fibrotic and shrinks.
- Secretions may calcify.
- Islets initially preserved, but later blood supply compromised and they atrophy.

**Cause**
- Excess alcohol consumption
- Pancreatic duct obstruction
  - Pancreas divisum
  - Cystic fibrosis
- Hypercalcaemia
- Autoimmunity
- Gene mutations
- Hypertriglyceridaemia
- Idiopathic

**Incidence**
- Est. 3.5-10 per 100,000 population per year.
- More common in males, particularly alcoholic CP.
- Usually begins in adulthood.
Drivers of malnutrition in CP

Maldigestion
- Insufficient secretion of pancreatic lipase
- Insufficient micelle formation due to inadequate bicarbonate production
- Precipitation of bile acids due to acidic SI

Malabsorption
- Resting energy expenditure may be higher by 30-50%

Malnutrition
- Pain
- Alcoholism
- Poor dietary intake
- Diabetes
- Other symptoms
Our data: symptoms

### Symptoms and Dietary Intake

<table>
<thead>
<tr>
<th>Symptom</th>
<th>N</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intermittent/ occasional pain</td>
<td>43</td>
<td>69.4</td>
</tr>
<tr>
<td>Malabsorption</td>
<td>37</td>
<td>61.7</td>
</tr>
<tr>
<td>Anorexia/ satiety</td>
<td>27</td>
<td>45.0</td>
</tr>
<tr>
<td>Vomiting</td>
<td>22</td>
<td>36.7</td>
</tr>
<tr>
<td>Daily/ constant pain</td>
<td>19</td>
<td>30.6</td>
</tr>
<tr>
<td>Fatigue</td>
<td>13</td>
<td>21.0</td>
</tr>
<tr>
<td>Taste disturbances</td>
<td>10</td>
<td>16.7</td>
</tr>
<tr>
<td>Constipation</td>
<td>9</td>
<td>14.5</td>
</tr>
<tr>
<td>Bloating</td>
<td>8</td>
<td>12.9</td>
</tr>
<tr>
<td>Acid reflux</td>
<td>4</td>
<td>6.4</td>
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<table>
<thead>
<tr>
<th></th>
<th>Malabsorption</th>
<th>Vomiting</th>
<th>Poor appetite</th>
<th>Taste Disturbances</th>
<th>Fatigue</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
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<tr>
<td>Intermittent (N)</td>
<td>21</td>
<td>22</td>
<td>11</td>
<td>32</td>
<td>15</td>
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<tr>
<td>Constant (N)</td>
<td>16</td>
<td>3</td>
<td>11</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>P-value</td>
<td>0.006*</td>
<td>0.015*</td>
<td>0.19</td>
<td>0.16</td>
<td>0.49</td>
</tr>
</tbody>
</table>

Symptoms will affect dietary intake
The impact of alcohol on nutrition

• Major causal factor

• Excess alcohol intake an independent risk factor for malnutrition
  – Displacement of food
  – Effects on appetite

• Malabsorption
  – Impaired/ altered biliary or pancreatic function
  – Direct effects of alcohol on the GIT
Defining malnutrition in CP

- Malnutrition
  - Underweight
  - Overweight

- Muscle mass
- Functional capacity
- Micronutrient status
Our data: BMI in CP

- Overweight and obesity 50% in CP
- Underweight <10%
CP patients: obese, but compared to controls

Less obesity
Lower BMI
Lower fat stores
Lower muscle stores
Lower strength

<table>
<thead>
<tr>
<th></th>
<th>Male Patient</th>
<th>Male Control</th>
<th>P-value</th>
<th>Female Patient</th>
<th>Female Control</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI, mean</td>
<td>25.9 (4.9)</td>
<td>28.5 (4.2)</td>
<td>0.007*</td>
<td>25.5</td>
<td>26.7 (3.5)</td>
<td>0.422</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(5.4)</td>
<td>(3.5)</td>
<td></td>
</tr>
<tr>
<td>Obese, n (%)</td>
<td>24 (53.3)</td>
<td>39 (81.3)</td>
<td>0.038**</td>
<td>7 (41.2)</td>
<td>12 (66.7)</td>
<td>n/a^2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(41.2)</td>
<td>(66.7)</td>
<td></td>
</tr>
<tr>
<td>TSF</td>
<td>7.7 (5.3)</td>
<td>12.5 (6.6)</td>
<td>0.000*</td>
<td>11.3</td>
<td>21.4 (6.7)</td>
<td>0.001*</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(7.6)</td>
<td>(6.7)</td>
<td></td>
</tr>
<tr>
<td>MUAC</td>
<td>29.0 (3.4)</td>
<td>32.4 (2.9)</td>
<td>0.000*</td>
<td>27.4</td>
<td>30.3 (3.4)</td>
<td>0.046*</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(4.1)</td>
<td>(3.4)</td>
<td></td>
</tr>
<tr>
<td>MAMC</td>
<td>26.6 (2.9)</td>
<td>28.5 (2.3)</td>
<td>0.001*</td>
<td>23.9</td>
<td>23.6 (1.7)</td>
<td>0.763</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(3.1)</td>
<td>(1.7)</td>
<td></td>
</tr>
<tr>
<td>Handgrip strength</td>
<td>39.9 (7.7)</td>
<td>43.3 (7.7)</td>
<td>0.048*</td>
<td>25.9</td>
<td>26.3 (5.7)</td>
<td>0.865</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(7.3)</td>
<td>(5.7)</td>
<td></td>
</tr>
</tbody>
</table>
Nutrient deficiency

- Even in patients with apparently good nutritional status

Mechanisms
- Poor dietary intake
- Increases losses
- Increased requirements
- Impaired binding of nutrients
- Antioxidant activity
- Malabsorption

Vitamin E deficiency 75%
Case report: neurological manifestations
More prevalent in presence of steatorrhoea, malnutrition and alcoholism
More prevalent than deficiencies of vitamins A, D and K

Vitamin A deficiency 16%
Case report: Alcoholic CP, DM: corneal ulceration, steatorrhoea, cachexia and anaemia

2. Yokota et al, J Neurol, 1990
# Nutrient deficiency

<table>
<thead>
<tr>
<th><strong>Vitamin D deficiency</strong></th>
<th><strong>Zinc and copper</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower vitamin D levels in CP vs. controls, correlated with faecal elastase(^1)</td>
<td>A zinc-binding compound of pancreatic origin facilitates intestinal absorption</td>
</tr>
<tr>
<td><strong>Vitamin B12 deficiency</strong></td>
<td>Deficiency possible, especially with DM(^3)</td>
</tr>
<tr>
<td>Due to inadequate protease secretion by the pancreas, which is required to release B12 for absorption in the T ileum(^2)</td>
<td>Normal zinc in studies(^3)</td>
</tr>
<tr>
<td>Appears to be relatively uncommon</td>
<td>May be increased copper excretion in CP/ DM(^3)</td>
</tr>
<tr>
<td></td>
<td>However high levels found in alcoholic CP(^4)</td>
</tr>
</tbody>
</table>

3. Quillot et al, Pancreas. 2001
Our data: vitamin status

<table>
<thead>
<tr>
<th>Vitamin A µmol/L</th>
<th>Patients (n=62) n (%)</th>
<th>Controls (n=66) n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1.6</td>
<td>Deficiency</td>
<td>8 (12.9)</td>
</tr>
<tr>
<td>1.6 - 3.7</td>
<td>Normal</td>
<td>42 (67.7)</td>
</tr>
<tr>
<td>&gt;3.7</td>
<td>Toxicity</td>
<td>12 (19.3)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vitamin E µmol/L</th>
<th>Patients (n=62) n (%)</th>
<th>Controls (n=66) n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;21.3</td>
<td>Deficiency</td>
<td>6 (9.7)</td>
</tr>
<tr>
<td>21.3 - 43.8</td>
<td>Normal</td>
<td>48 (77.4)</td>
</tr>
<tr>
<td>&gt;43.8</td>
<td>Toxicity</td>
<td>8 (12.9)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vitamin E:Lipid ratio µmol/L</th>
<th>Patients (n=62) n (%)</th>
<th>Controls (n=66) n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;3.8</td>
<td>Deficiency</td>
<td>14 (22.6)</td>
</tr>
<tr>
<td>3.8 - 6.3</td>
<td>Normal</td>
<td>46 (74.2)</td>
</tr>
<tr>
<td>&gt;6.3</td>
<td>Toxicity</td>
<td>2 (3.2)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vitamin D nmol/L</th>
<th>Patients (n=62) n (%)</th>
<th>Controls (n=66) n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;25mmol/L</td>
<td>Deficiency</td>
<td>7 (11.2)</td>
</tr>
<tr>
<td>&lt;50mmol/L</td>
<td></td>
<td>36 (58.1)</td>
</tr>
<tr>
<td>&lt;80mmol/L</td>
<td>Insufficiency</td>
<td>58 (93.5)</td>
</tr>
</tbody>
</table>

Vitamin A: deficiency and excess
Guidelines for replacement?

Italian consensus guidelines for chronic pancreatitis
Frulloni et al. *Dig Dis Sci.* 2010

Our practice:
- ADEKs fat soluble vitamin supplement if deficient and recheck
- Borderline? Multivitamin
- Separate vitamin D supplement
- Vitamin B12

ESPEN GUIDELINES

ESPEN Guidelines on Enteral Nutrition: Pancreas


Nausea and diarrhea. The diet should be low in fibre, since fibres absorb enzymes and lead to a reduced intake of nutrients. Fat-soluble vitamins (vitamin A, D, E, K) as well as other micronutrients should be supplemented if clinical deficit is apparent.

A lot of enzyme supplements are available that differ in enzyme content and pharmacological activity.
Bone health in CP

- Increased risk of low bone density
  - Malabsorption, poor diet, vitamin D deficiency, immobility, smoking, alcoholism
  - Risk greater or equal than other comparable diseases
    - IBD, coeliac disease, CF, post-gastrectomy
    - Recommend routine DXA, supplementation of calcium, vitamin D
    - Guidelines lacking for CP
Patients With Chronic Pancreatitis Are at Increased Risk for Osteoporosis

Sinead N. Duggan, BSc, Dipl. RD, * Marra O’Sullivan, PhD, † Samuel Hamilton, MD, ‡ Sinead M. Feehan, BSc, Dipl. RD, § Paul F. Ridgway, MD, * and Kevin C. Condon, MD *


### Table

<table>
<thead>
<tr>
<th></th>
<th>Patients</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean age</strong></td>
<td>48.7 (11.8)</td>
<td>48.05 (10.7)</td>
</tr>
<tr>
<td><strong>Upper tertile for age</strong></td>
<td>57.0</td>
<td>56.0</td>
</tr>
<tr>
<td><strong>% Male</strong></td>
<td>75.5</td>
<td>72.4</td>
</tr>
<tr>
<td><strong>% who finished third level education or higher</strong></td>
<td>21</td>
<td>24</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>%</th>
<th>N</th>
<th>%</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Osteoporosis</strong></td>
<td>18</td>
<td>34</td>
<td>6</td>
<td>10.2</td>
<td></td>
</tr>
<tr>
<td><strong>Osteopenia</strong></td>
<td>14</td>
<td>26.4</td>
<td>14</td>
<td>23.7</td>
<td>0.004**</td>
</tr>
<tr>
<td><strong>Normal bone density</strong></td>
<td>21</td>
<td>39.6</td>
<td>39</td>
<td>66.1</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Mean (SD)</th>
<th>P-value</th>
<th>n</th>
<th>Mean (SD)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean T-score</strong></td>
<td>53</td>
<td>-1.77 (1.47)</td>
<td><strong>0.001</strong>*</td>
<td>58</td>
<td>-0.89 (1.06)</td>
<td></td>
</tr>
<tr>
<td><strong>Male T-score</strong></td>
<td>40</td>
<td>-1.76 (1.08)</td>
<td></td>
<td>42</td>
<td>-0.74 (0.98)</td>
<td></td>
</tr>
<tr>
<td><strong>Female T-score</strong></td>
<td>13</td>
<td>-1.81 (2.36)</td>
<td>0.945</td>
<td>16</td>
<td>-1.28 (1.19)</td>
<td>0.117</td>
</tr>
</tbody>
</table>

**Figure 1A**

Box plot showing smoking tertiles (pack year history) vs lowest T-score for patients.

**Figure 5**

Box plot showing lowest T-scores for patients across age tertiles.

ANOVA P = 0.004
Systematic review & meta-analysis: Osteoporosis in chronic pancreatitis

Osteoporosis pooled prevalence

Overall osteopathy pooled prevalence
Nutritional requirements and diet

• More than 80% of patients may be treated with diet and enzymes (ESPEN (B))
• High calorie intake – 35 kcal/Kg
  – Some energy (10%) may be salvaged by colonic bacterial metabolism
• Moderate fat: 30% of calories
  – Mostly vegetable fat? No studies to support
  – Severe fat restriction not appropriate
  – MCT fat
• High carbohydrate
  – Limit in diabetes
  – High fiber may absorb enzymes
• High protein 1-1.5 g/Kg
Oral nutritional supplements

• In most cases normal food will be sufficient to maintain nutritional status, ONS required in 10-15% of cases (ESPEN (C))
  – Regular assessment should be done to ensure adequacy of diet
  – Where low dietary intake persists, ONS may be tried

• Whole protein ➔ peptide-based, MCT

  – Dietitian advice vs. MCT supplements alone; 3 months
  – Improvements in BMI, MAC, TSF in both groups
  – Increased intake of calories, fat, carbohydrate, protein in both groups
Nutrition support:  
When should we feed?

**Enteral nutrition**

1. When patient cannot ingest sufficient calories because of pain or obstruction
2. When weight loss continues despite apparently sufficient intake
3. In presence of acute complications (e.g. AP)
4. Prior to surgery

**Parenteral nutrition**

1. When enteral feeding is not possible
2. Indications:
   - Gastric outlet obstruction
   - Complex pancreatic fistulae
   - Severe malnutrition prior to surgery (EN not possible)

<1% of cases  

5% of cases

ESPEN Guidelines

ESPEN Guidelines on Enteral Nutrition: Pancreas

Endocrine dysfunction: Pancreatogenic diabetes

- Diabetes complicates CP in 30-50% of cases
- Due to loss of islet cell function, possibly due to micro-ischaemia
- Type 3c DM, poorly characterised
- Dietary management complicated by
  - Malabsorption, alcoholism, erratic diet
  - Hypoglycaemia a particular concern
- Dietary restriction less stringent due to risk of hypo and malnutrition
## Pancreatogenic Diabetes: Special Considerations for Management

YunFeng Cui\textsuperscript{a,b}, Dana K. Andersen\textsuperscript{a}

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Type 1 IDDM juvenile onset</th>
<th>Type 2 NIDDM adult onset</th>
<th>Type 3c pancreatogenic postop. onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ketoacidosis</td>
<td>common</td>
<td>rare</td>
<td>rare</td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>severe</td>
<td>usually mild</td>
<td>mild</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>common</td>
<td>rare</td>
<td>common</td>
</tr>
<tr>
<td>Peripheral insulin sensitivity</td>
<td>normal or increased</td>
<td>decreased</td>
<td>increased</td>
</tr>
<tr>
<td>Hepatic insulin sensitivity</td>
<td>normal</td>
<td>normal or decreased</td>
<td>decreased</td>
</tr>
<tr>
<td>Insulin levels</td>
<td>low</td>
<td>high</td>
<td>low</td>
</tr>
<tr>
<td>Glucagon levels</td>
<td>normal or high</td>
<td>normal or high</td>
<td>normal or high</td>
</tr>
<tr>
<td>PP levels</td>
<td>normal or low (late)</td>
<td>high</td>
<td>low</td>
</tr>
<tr>
<td>GIP levels</td>
<td>normal or low</td>
<td>normal or high</td>
<td>low</td>
</tr>
<tr>
<td>GLP-1 levels</td>
<td>normal</td>
<td>normal or high</td>
<td>normal or high</td>
</tr>
<tr>
<td>Typical age of onset</td>
<td>childhood or adolescence</td>
<td>adulthood</td>
<td>any</td>
</tr>
</tbody>
</table>

IDDM = Insulin-dependent diabetes mellitus; NIDDM = non-insulin-dependent diabetes mellitus. Modified from Slezak and Andersen [13], with permission.
Antioxidants

• Promising data initially for the treatment of pain, improvement of QOL, reduction in working days lost in chronic pancreatitis

• ANTICIPATE study, published June 2012

• Double-blind RCT\(^1\): Antox vs. placebo, n=70 CP patients

• ‘Antioxidant therapy did not reduce pain or improve quality of life, despite causing a sustained increase in blood levels of antioxidants’.

1. Siriwarden et al. Gastroenterology. 2012 (Jun 5) Epub
Pancreatic enzyme therapy

A reduction in steatorrhoea and an adequate energy intake are the most important principles of nutrition therapy in chronic pancreatitis

• Greater BMI loss in those with untreated malabsorption
  – Weight loss can occur even in mild malabsorption
• Malabsorption of protein and carbohydrate, as well as fat
  – Higher amt of amylase and trypsin maintained in the ileum
• Fat malabsorption
  – Stools tend to be bulky and formed
  – Visible oil virtually pathogenic of CP (fat loss of 30-40 g/day)
  – Clinical inspection of stool unreliable, except in gross malabsorption
• Other signs or symptoms
  – Weight loss, bloating, flatulence, pain, cramping
• How to assess exocrine dysfunction?

Tests
- 24-hr Faecal fat
- Secretin test
- Chymotrypsin
- BT-PABA
- Sudan III stain test
- Faecal Elastase-1
Faecal Elastase-1

- Widely used
- Cheap, non-invasive, widely available
- Pancreatic enzyme that is not degraded during digestion and may be measured in the stool
- Not affected by enzyme use
- Does not require timed stool collection
- Does not require special diet
- But – sensitivity limited in mild pancreatic insufficiency
How to administer PERT
**Expert commentary: how we do it**

C. W. Imrie*, G. Connet†, R. I. Hall‡ & R. M. Charnley**

*Lister Department of Surgery, Glasgow Royal Infirmary, Glasgow, UK.
†Southampton University Hospitals NHS Trust, Southampton, UK.
‡Royal Derby Hospital, Derby, UK.
**Freeman Hospital, Newcastle upon Tyne, UK.

<table>
<thead>
<tr>
<th>Trade name</th>
<th>Lipase (U)</th>
<th>Amylase (U)</th>
<th>Protease (U)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-enteric coated</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pancrex V powder</td>
<td>25 000</td>
<td>30 000</td>
<td>1400</td>
</tr>
<tr>
<td>Pancrex granules</td>
<td>5000</td>
<td>4000</td>
<td>300</td>
</tr>
<tr>
<td>Pancrex V capsules</td>
<td>8000</td>
<td>9000</td>
<td>430</td>
</tr>
<tr>
<td>Pancrex V tablets</td>
<td>1900</td>
<td>1700</td>
<td>110</td>
</tr>
<tr>
<td>Enteric-coated</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creon 10</td>
<td>10 000</td>
<td>8000</td>
<td>600</td>
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<tr>
<td>Creon 25</td>
<td>25 000</td>
<td>18 000</td>
<td>1000</td>
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<tr>
<td>Creon 40</td>
<td>40 000</td>
<td>25 000</td>
<td>1600</td>
</tr>
<tr>
<td>Creon Micro</td>
<td>5000</td>
<td>3600</td>
<td>200</td>
</tr>
<tr>
<td>Nutrizym 10</td>
<td>10 000</td>
<td>9000</td>
<td>500</td>
</tr>
<tr>
<td>Nutrizym 22</td>
<td>22 000</td>
<td>19 800</td>
<td>1100</td>
</tr>
<tr>
<td>Pancrease HL</td>
<td>25 000</td>
<td>22 500</td>
<td>1200</td>
</tr>
<tr>
<td>Pancrex V Forte tablets</td>
<td>56 000</td>
<td>5000</td>
<td>330</td>
</tr>
</tbody>
</table>

- Lipase irreversibly denatured by pH<4
- Enteric-coated preparations developed
- Coating only dissolves when pH is >5.5
Dose and administration

• Min dose of **25,000-50,000 per meal** to reduce steatorrhoea to <15g/ day to compensate for pancreatic insufficiency\(^1\)

• Dietary assessment vital – check diet regularly and move to protein supplementation early\(^2\)

• Dose should be gradually increased until symptoms are controlled\(^2\)

• Try a PPI or H2 blocker

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When treatment fails...

Pancreatic enzyme replacement therapy in chronic pancreatitis

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Practice points

- Every patient with EPI should be treated with enzyme replacement therapy (PERT), independent of the degree of steatorrhea and maldigestion.
- The recommended dosage of PES ranges from 25,000 to 75,000 units of lipase for a main meal and 10,000 to 25,000 units for snacks, depending on the fat content of the meal.
- Half of the dose should be swallowed at the start of the meal and the remainder should be taken halfway the meal.
- Referral to a dietician is an essential part of the treatment.

Practice points

- When treatment fails the first step is to evaluate compliance and to increase the dose of pancreatic enzymes up to a maximum of 10,000 IU lipase/kg/day.
- The next step is inhibition of gastric secretion by administration of a proton pump inhibitor.
- Other causes of steatorrhea such as coeliac disease and bacterial overgrowth should be ruled out.
- A last resort may be the restriction of fat while compensating caloric losses with an energy enriched diet.
Dietary assessment

• Type of food eaten (fat content)
  • Meals, snacks, liquids, supplements
  • Method of cooking
  • Volume of food at each meal
  • Timing of meals

• When enzymes are being taken
  • How much taken at each time
  • How are enzymes taken (crushed, sprinkled, whole)

• PPI/ H2 Blockers

• Symptoms post-prandially; malabsorption, constipation

• Weight, weight history, muscle mass

Individualised patient education vital so they can alter enzymes with changing circumstances
Patient information booklet on the use of pancreatic enzymes
Produced by the Nutrition Interest Group of the Pancreatic Society of Great Britain and Ireland, in conjunction with Abbott Nutrition, updated January 2013

Summary
- Lifelong pancreatic enzyme supplementation is important for your nutrition and long-term health
- Dose and frequency varies between individuals
- Your dietitian and doctor are there to help with any questions or concerns

For more information contact your dietitian, doctor or clinical nurse specialist
In summary..

• Many factors to consider when dealing with nutrition in chronic pancreatitis
• Malabsorption, pain, poor diet, symptoms
  – N&V, anorexia, fatigue, constipation, bloating, acid reflux
• Patient may be overweight or obese, but likely to have low muscle stores, strength
• Nutrient deficiencies common, but excess may also feature
• Smoking, alcohol may affect nutritional status
• Diabetes may be a feature
• Bone health neglected problem
• Some will require ONS, enteral feeding and parenteral feeding, most manageable with dietary counseling
• PERT mainstay of treatment
Tallaght Hospital algorithm for the nutritional management of patients with chronic pancreatitis

2013 meeting of the Pancreatic Society of Great Britain & Ireland

- 6th Annual Nutrition Symposium

BT convention Centre, Liverpool
27-29th November