



## ASPEN Case Study - Annette Prinsloo RD(SA)

### Background information

The patient, a 47 yr old male, was admitted to hospital because of epigastric pain (of 6 months duration), haematemesis and maelena stools. The diagnosis of chronic pancreatitis as well as of a pseudoaneurysm in the pancreatico-duodenal artery was made.

### Patient's course

On admission the patient had an angiogram, a gastroscopy, and blood was transfused according to standard protocols of the hospital. The pseudoaneurysm was also embolised.

### Diagnosis

Chronic pancreatitis

### Anthropometry

Weight : 48 kg  
 Height : 163 cm  
 BMI : 18 kg/m<sup>2</sup>  
 MUAC: 20.5 cm (< 5th percentile)  
 AMA: < 5th percentile  
 AFA: < 5th percentile

### Medication

Tramil 100 mg qid  
 Panado 500 mg tds  
 Tazocin 4.5g tds  
 Vitamin K 1 mg stat

### Biochemistry

	31/05/2009	1/6/2009	2/6/2009	3/6/2009	5/6/2009
Sodium	130	132	135	133	135
Potassium	5.5	4.2	1.9*	4.5	4.9
Chloride	97	100	100	99	99
Urea	2.8	2.8	2.5	2.6	2.6
Creatinine	39	36	31	35	37
Albumin	26	34*	26	24	27
Tot protein	73	59*	65	65	72
T Billirubin	11	11	10	11	8
D Billirubin	<1	5	8	3	3
S-ALP	69	62	68	62	65
S-GGT	240	202	216	209	231
S-ALT	22	12	11	12	14
S-AST	93	16	17	20	20
S-CRP	67.3	54	46.9	40	#

\* Blood values as extracted from patient's file  
 # Blood value not available

### Nutritional management and discussion

This malnourished (grade I; currently 80% of his ideal body weight, anthropometric measurements below the fifth percentile; assumed BMI <18.5) patient was referred to the dietitian for nutritional management .

The patient's nutritional requirements were calculated using the ESPEN recommendations<sup>1</sup> on the basis of his ideal body weight of 59.8 kg and based on a BMI of 22.5.

Total energy: 1734 kCal/d @ 25k Cal/kg

Protein: 59 g/d @ 1 g/kg/d

Chronic pancreatitis is characterised by the loss of endocrine and exocrine function due to the destruction of acinar and islet cells and the replacement of gland by fibrous tissue.<sup>2</sup> The loss of exocrine function leads to malabsorption, maldigestion, steatorrhoea, weight loss and undernutrition,<sup>2</sup> as was seen in this patient. In exocrine pancreatic insufficiency, carbohydrate digestion is usually maintained for a long time by salivary amylase and brush-border oligosaccharidases.<sup>3</sup> The loss of endocrine function, however, leads to glucose intolerance. Glucose intolerance is reported to occur in 40–90% of all cases with severe chronic pancreatitis.<sup>3</sup> In 20–30% of all patients insulin-dependant diabetes can develop and is associated with impaired glucagon release. Glucose intolerance due to loss of endocrine function was not seen.<sup>3</sup>

The severe epigastric pain that the patient presented with is a common symptom of chronic pancreatitis and is often debilitating. Such patients often present with weight loss and malabsorption of macronutrients, especially in the later course of the disease. The primary nutritional concern is the possibility of further deterioration in the patient's nutritional status because of increased energy requirements (30 – 50% of patients with chronic pancreatitis have increased energy expenditure)<sup>3</sup> as well as the likely decreased nutrient intake during this hospital admission and investigative procedures. The goal of nutritional therapy was therefore to prevent further weight loss. The patient received 1.5 liters of a semi-elemental feed high in dipeptides to drink orally. The feed was in a powder form and was reconstituted according to the instructions from the manufacturer. The approach and implementation of the administration of enteral feeding was done according to the 2009 ASPEN enteral nutrition practice recommendations.<sup>4</sup>

The feed was supplemented with a carbohydrate monomer to meet the patient's carbohydrate requirements.<sup>1</sup> Although the majority (± 80%) of patients can be treated adequately with normal food

supplemented by pancreatic enzymes, a small population of patients ( $\pm 10\%$ ) might not tolerate this strategy and might require oral nutrition supplementation.<sup>1,3,5</sup> Oral nutrition supplements containing peptides are more efficient than whole protein supplements.<sup>1</sup> However compliance on these supplements may not be good due to palatability. A protein intake of 1.0–1.5 g/kg has been recommended and is usually well tolerated.<sup>1</sup> Thirty percent of energy should initially be given as fat, and in the presence of persistent steatorrhoea a portion of the fat requirements should be administered in the form of medium chain triglycerides (MCTs).<sup>1</sup>

MCTs however have a lower energy density, are not very palatable and may induce side-effects such as abdominal pain, nausea and diarrhoea. The semi-elemental feed used in this case contained 70% of the fat in the form of MCTs. Fat soluble vitamins (A, D, E and K), vitamin B12 and other micronutrients should be supplemented if serum levels are below the recommended lower limit of the normal range. In general, a low fibre diet is recommended, because fibre may adsorb enzymes and delay the absorption of nutrients.<sup>1,3</sup> With regard to the use of MCTs, a recent study from India compared the efficacy of MCT-enriched-commercial dietary supplements with dietary counselling for homemade food in the management of malnutrition in patients with chronic pancreatitis.<sup>6</sup> BMI increased in both the counselling group and the supplementation group ( $17.2 \pm 1.7$  vs  $18.1 \pm 1.8$  kg/m<sup>2</sup>,  $P = .001$ ;  $16.7 \pm 1.6$  vs  $18.2 \pm 1.6$  kg/m<sup>2</sup>,  $P = .001$ ). and there were similar improvements in triceps skinfold thickness, dietary intake, faecal fat, and pain score during a period of 3 months in both groups. The study concluded that dietary counselling for a balanced homemade diet was as good as commercial food supplements in improving malnutrition in patients with chronic pancreatitis.

Avoidance of alcohol, dietary modification and pancreatic enzyme supplements are the corner stone of nutritional management in patients with chronic pancreatitis. In addition, an adequate energy intake should be secured because of the reported increase in resting energy expenditure.<sup>3</sup> Frequent meals (4–5 times a day) should be given. The diet should be rich in carbohydrates and proteins. The carbohydrate intake should be limited in the presence of overt diabetes mellitus. An adequate quantity of exogenous pancreatic enzymes is necessary to correct protein and lipid maldigestion. Steatorrhoea is usually more difficult to correct than azotorrhea.<sup>3</sup> Therefore, a suitable amount of lipase per meal is necessary to provide adequate lipolysis. There is no defined dose for these enzyme supplements and patients will need to take 2–6 pills with each meal.<sup>3</sup> Practical goals for this therapy include: weight control, symptomatic relief of steatorrhoea, or a decrease in 72-hour faecal fat excretion. It is important to give the enzymes before the meal to ensure adequate mixing. If the enzyme treatment response is not

satisfactory, the addition of an acid inhibitor (proton pump inhibitor) can be tried.<sup>1,3</sup> Decreasing the duodenal acid load can prevent the inactivation of lipase in the small bowel. In 10–15% of patients oral supplements can help to attenuate weight loss and delay the use of enteral tube feeding.<sup>3</sup>

Currently, there is a paucity of consistent and conclusive evidence to document a preferential route of nutrient supplementation, i.e. Enteral vs Parenteral, in patients with chronic pancreatitis with attendant severe maldigestion and malnutrition. Enteral nutrition can be useful, when dietary recommendations are not met, or before, and after, pancreatic surgery. Semi elemental formulae fed jejunally are usually well tolerated. Enteral and parenteral recommendations for patients with chronic pancreatitis are based on observation, due to the lack of prospective trials in this field.

### References:

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