

The use of Peptamen[®] HN to maximise absorption in chronic pancreatitis.

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Synopsis.

This case study discusses the management of a male patient with chronic pancreatitis and persistent *C. difficile* infection, requiring full enteral tube feeding for the majority of his admission, due to an unsafe swallow. As malabsorption and osmotic diarrhoea were problematic, a change in enteral formula was indicated.

Introduction to Chronic Pancreatitis:

Chronic pancreatitis (CP) is a progressive inflammatory condition often resulting in impaired digestion and thus malabsorption of nutrients. Its incidence is highest among middle-aged men (aged 45- 54 years), with approximately 45% of its cases linked to chronic alcohol misuse¹. Smoking, gallstones and autoimmune disorders are among the less common causes.

The pathogenesis of CP is heterogeneous, but tends to involve necrosis and inflammation, characterised by the progressive fibrotic destruction of the secretory cells of the pancreas. Ultimately, the exocrine and endocrine functions of the pancreas are likely to become impaired. Reduced endocrine function is likely to affect blood sugar control, and often results in diabetes².

Reduced pancreatic exocrine function impacts on intestinal enzymatic digestion, resulting in malabsorption and often malnutrition. The CP patient may therefore experience weight loss and micronutrient depletion, and classical malabsorption symptoms include diarrhoea and steatorrhea. Due to this profound impact on the gastrointestinal (GI) tract, dietetic management can play a very important role in the medical treatment of a CP patient.

GI symptoms (which can also include nausea and vomiting) often affect a patient's appetite and may mean they require additional nutrition support in order to prevent weight loss. Dietetic input with the CP patient may also include making suggestions to the prescriber with regard to appropriate dosages of pancreatic enzyme replacement therapy (PERT), which many CP patients require to reduce symptoms of malabsorption. With the pancreas playing such an integral role in fat digestion in particular, the dietitian may also consider the type (as well as the quantity) of fat in the patient's diet.

Medium-chain triglycerides (MCTs) do not require pancreatic digestion and are therefore likely to be better absorbed and utilised by a CP patient than longer chain fatty acids. Oral nutritional supplements and enteral tube feeds that contain MCTs may therefore be used in CP patients^{3,4}.

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Patient Background.

Demographic Information:

- 65 year old male
- White British
- Unemployed

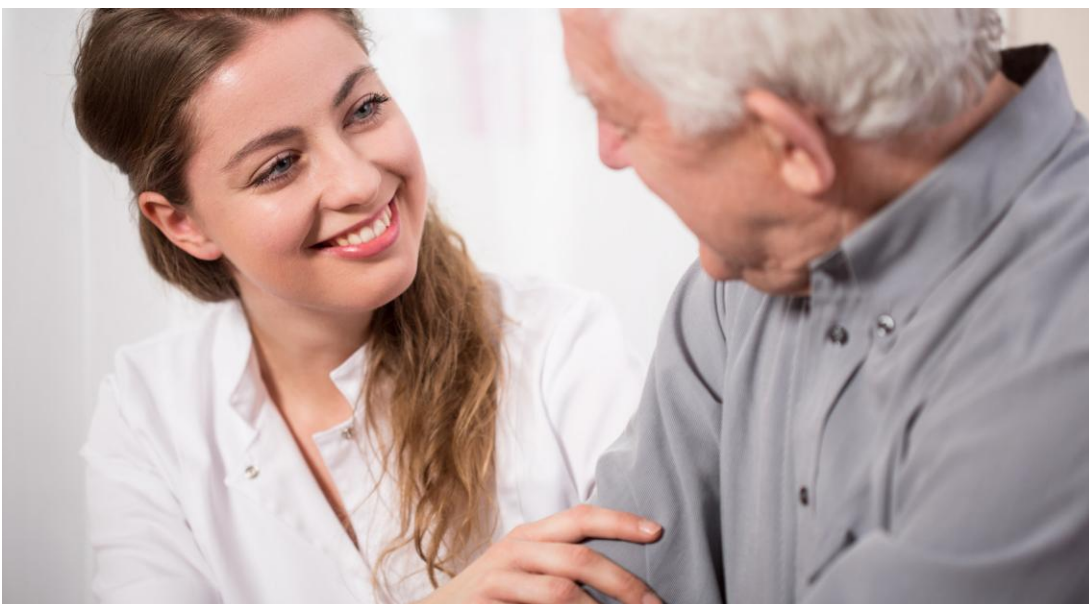
Presenting Complaint:

- Nausea and vomiting

Past Medical History:

- Previous cerebrovascular accident, with some residual left sided weakness
- Chronic pancreatitis
- Depression
- Alcoholic liver disease (Child-Pugh A cirrhotic)

The Child-Pugh scoring system is used to assess the severity and prognosis of chronic liver disease. It is graded from A-C, with C the most severe and thus worst prognosis. It is based on a combination of symptomatic and biochemical factors.



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Initial Nutritional Assessment:

Anthropometry - On Admission (October 2015)

- Weight 66.7kg
- Height 1.72m
- BMI 22.5kg/m² (healthy range)

Patient referred to dietetics one week later after unintentional weight loss of 2.1kg and new NBM status. Weight 64.6kg, BMI 21.8kg/m².

Relevant Biochemistry

(previous day's figures in brackets)

- Sodium 130 (131)
- Potassium 3.3 (3.5)
- CRP 186 (172)
- Urea, other electrolytes and liver function tests all in normal ranges

Clinical Observations

The patient's primary reason for admission had been nausea and vomiting (which were the likely cause of his slightly low electrolytes). At the time of initial dietetic assessment, one week later, symptom relief had still not been achieved, despite trialling anti-emetics. He was also experiencing significant epigastric pain.

He was under the gastroenterology team as an inpatient, and the working diagnosis was acute on chronic pancreatitis, with alcohol excess likely the key aetiological factor. He had a history of depression, and seemed particularly low in mood in the early stages of his admission. From the information provided by him, it appeared that this may have contributed to increased alcohol consumption in the period prior to admission.

He was also known to have a cirrhotic liver (Child-Pugh A) but was not showing any classical symptoms of decompensation. He was afebrile (although it was noted that he was on a high dose of paracetamol for analgesia), with fairly stable observations, a good urine output (approximately 50-100 ml / hour) and loose, pale, oily stools (bowels opening typically 2-4 times per day).

He was already on PERT in the community, but this had not yet been restarted in hospital due to his reduced dietary intake.

He had a chesty, productive cough, and he had been started on antibiotics 2 days prior to my review, due to possible aspiration pneumonia. He had experienced dysphagia when he suffered from a stroke 5 years ago, but his swallow function was thought to have been sufficient to tolerate a normal diet and fluids in the community. He had passed a bedside swallow assessment while on medical HDU, prior to his transfer to the gastroenterology ward, but on the day of the dietetic referral, his cough had worsened and his CRP was rising. The medical team therefore made him NBM as a precaution, and requested a formal swallow assessment from speech and language therapy. This was yet to take place at the time of my initial assessment.

Dietary Intake / Nutritional History

The patient did not think that he had previously been seen by a dietitian, either as an inpatient or in the community. As such, his understanding of the nutritional implications of his medical conditions (and medications) was limited. Despite his low mood prior to admission, he was adamant that he had maintained a good appetite and dietary intake until the last 3-4 days before admission, at which stage, his epigastric pain, nausea and vomiting meant that he was only managing small meals. He did not feel that he had lost any weight or muscle mass prior to admission.

During his week of admission, he had continued to eat, albeit smaller portions than at home, so I did not feel he was at risk of refeeding syndrome⁵. Only in the last 24 hours had his nutritional intake been negligible, due to his NBM status.

The speech and language therapist for the ward then diagnosed the patient with severe dysphagia and advised me that he would likely be NBM for days if not weeks. It was therefore clear that he would require artificial feeding.

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Dietetic Care Plan:

I asked the nursing staff to insert a nasogastric (NG) tube as soon as possible. The patient had initially been resistant to this idea, but became far more receptive when I fully explained the reasoning, and the tube was inserted later that afternoon.

Calculating Nutritional Requirements:

I used the Henry equation⁶ to calculate the patient's basal metabolic rate, using his age (65 years) and weight on referral to me (64.6kg):

$$13.0 \times 64.6 \text{ (kg)} + 567 = 1407 \text{ kcals}$$

I then applied a stress factor of 25% on account of his likely chest infection (as evidenced by rising CRP and chesty cough), and an activity factor of 15%, as he was mainly bed-bound due to pain at the time of my initial assessment⁷.

$$1407 \text{ kcals} \times 1.4 = 1970 \text{ kcals}$$

1970 kcals was therefore used as his initial daily energy requirement.

For protein, I used a range of 0.17-0.20g N/kg body weight⁷, again, on account of his likely infection:

$$0.17 \times 64.6 = 10.982 \text{ g N}^2 \\ (= 68.6 \text{ g protein / day})$$

$$0.20 \times 64.6 = 12.92 \text{ g N}^2 \\ (80.75 \text{ g protein / day})$$

For fluid, I used an initial figure of 30ml/kg body weight per day, as he was 65 years old⁷, giving 1938 ml. However, I was conscious of his increased gastrointestinal fluid losses in his stools, and would continue to monitor his hydration status and increase his fluid provision via his NG tube as appropriate.

Indeed, all of these nutritional requirements were revisited and revised throughout his stay, depending on his anthropometric and clinical status at any one time.

Formula Selection:

Due to his loose stools, I had initially considered using a standard polymeric non-fibre feed such as Osmolite®. However, on account of the fact that he was completely NBM, he would not have been able to safely take his PERT capsules orally, and there seems to be a lack of consensus regarding the best method of administering this medication via enteral feeding tubes. I therefore decided to use an enteral feed containing medium chain triglycerides (MCTs), in order to reduce the need for pancreatic fat digestion, with a view to avoiding using PERT altogether if tolerated.

I commenced feeding at a continuous rate (over 20 hours) with Vital® 1.5 (a concentrated, peptide-based feed designed for improved tolerance). Sadly this feed was not well tolerated, even after several days at a low rate, and his stools remained loose and frequent (bowels were opening 5-6 times each day, generally type 6 or 7).

However, the stools were no longer oily or difficult to flush, according to the patient, so I suggested that rather than being steatorrhea resulting from pancreatic insufficiency, this may have been osmotic diarrhoea, since this tube feed has a fairly high osmolality (630mOsm/kg, osmolarity 487mOsm/L).

I therefore decided to switch the feed to Peptamen® HN, a feed with which I had previously had great success in patients with poor absorption (and those with pancreatic insufficiency). Peptamen® HN is also peptide-based and with a high MCT content (70%), but crucially has a much lower osmolality than Vital® 1.5 (450mOsm/kg, osmolarity 350mOsm/L).

I also started this feed at a fairly low rate and ran it continuously for 20 hours. The patient very quickly showed signs of improved tolerance of this feed, with stools still not oily or difficult to flush, and soon becoming less frequent and firmer (bowels opening 2-3 times per day, type 4-5).

After an additional two weeks, and despite regular speech and language therapy involvement, there was no sign of improvement in his swallow, and MDT discussions soon turned to long term feeding plans. A plan was agreed with the patient for him to have a percutaneous endoscopic gastrostomy (PEG) feeding tube placed.

I had already begun to plan for the longer term, by shortening his hours on the feed,

condensing to 3 sessions of 3 hours (roughly around mealtimes), with 500ml Peptamen® HN administered at 167ml/hr. The patient was tolerating these fast rates without any issues.

Unfortunately, shortly after the date for his PEG was booked, he developed loose, frequent stools again. I temporarily switched back to a 20 hour continuous feed at a lower rate in case the higher feed rates had been contributing to his symptoms. Stool samples were sent, and it was found that he had acquired a *C. difficile* infection.

After experimentation it seemed that feed rate did not make any significant difference. I therefore returned to his 3 x 500ml feeds at 167ml/hr even during his active infection.

He would not be able to have his PEG tube placed until he was clear of infection. He was therefore commenced on the protocol antibiotic therapy for *C. difficile*, but without success. The infectious diseases team became involved, and recommended a faecal transplant for this man's refractory infection.

The initial faecal transplant was unsuccessful, but another attempt, one month later, proved to be successful, and his symptoms quickly improved. His PEG was then placed, and he was discharged home, successfully administering his own feeds in the community.

However, despite the hugely delayed discharge and refractory infection issues (as well as his multiple co-morbidities), the patient's nutritional status was kept remarkably stable throughout his long admission. I feel that much of this is owed to good MDT communication and patient compliance, but also to the type of enteral feed used for the majority of his hospital stay.

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Critical Evaluation:

Outcomes achieved included:

- Improved patient satisfaction
- Improved stool frequency and type (rapid progression from approximately 5-6 type 7 stools daily to 2-3 type 4/5 stools)
- Maintenance of nutritional status
- Engagement with physiotherapy and mobilising

I have previously found Peptamen® HN to be extremely effective in those with pancreatic insufficiency and/or poor GI tolerance, most likely due to the high percentage of MCT, as well as the partially hydrolysed nature of the whey protein. However, in this case study, I feel that Peptamen® HN was particularly useful in comparison to Vital® 1.5, due to its considerably lower osmolality.

This helped to control symptoms in the short-term at the start of the admission, but crucially also helped to maintain the patient's nutritional status throughout his long admission.

It is widely accepted that MCT based products can be appropriate in order to help reduce steatorrhea resulting from chronic pancreatitis^{3,4}.

Moreover, there is substantial evidence to support the use of semi-elemental whey protein in patients experiencing malabsorption⁸. Previous studies suggest improved clinical outcome as well as reduction in length of stay and economic cost of nutrition support using Peptamen® compared with parenteral or standard enteral formulas⁹⁻¹¹.

Conclusion:

This patient provided multiple, ongoing challenges for the MDT as a whole, and particularly for me as his inpatient dietitian. He had multiple co-morbidities, an extremely long admission, and refractory infection resulting in disturbed GI function and further delays to his treatment and discharge.

However, despite all the above, his nutritional status was well maintained, based on the parameters available. To me this emphasised the importance of choosing the most appropriate type of enteral feed. I had previously had great success with Peptamen® HN as a specialist feed for those with pancreatic insufficiency and/or malabsorption, but this case study in particular reinforced the clinical and therapeutic benefit it can bring to some complex cases.

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