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Case Report

Low-fat Elemental Enteral Nutrition in the Management of Pediatric Acute Pancreatitis

Hiroki MAKI*[1)], Emi SAWANOBORI[2)], Kouki AOYAMA[2)], Naomi KURATA[3)] and Akihito HOSODA[1)]

Abstract: Acute pancreatitis (AP) in childhood is rare, but is starting to be recognized more often. However, optimal enteral nutrition formulas for children with AP have not been determined. This report describes the successful management of AP, with low-fat elemental enteral nutrition, in a 5-year-old boy. The patient had been diagnosed with mild AP and started oral feeding after about 3 days of fasting, but his pancreatic enzyme levels subsequently became elevated. Therefore, to allow the pancreas to rest, total parenteral nutrition (TPN) was started. After starting TPN, there was no improvement in his pancreatic enzyme levels but, because his clinical symptoms were stable, a small amount of elemental diet was initiated. Elemental diet therapy was safely performed without return of any clinical symptoms and the patient's pancreatic enzyme levels slowly improved. Elemental diet was both safe and beneficial, providing clinical remission and improvement in quality of life. Early elemental diet therapy may therefore be a useful treatment strategy for pediatric patients with AP.

Key words: pediatric, acute pancreatitis, elemental diet, enteral nutrition

Introduction

Nutritional support is important in the management of acute pancreatitis (AP), and is comparable to medical therapies such as proteolytic enzyme agents, particularly in children. As reported in guidelines on nutrition in AP[1–4)], sufficient energy and nutrients should be provided after fasting in the acute disease phase, via an optimal route that depends on the pathological severity of the disease. It has been reported that 70% of patients receive oral feeding at admission, 20% receive total parental nutrition (TPN), and only 3% receive enteral feeding. Infants and toddlers are much more likely to receive TPN than older children (64% vs 17%)[5)]. Nutritional intervention is required in patients with malnutrition or inadequate oral ingestion. Moreover, some recent reports suggest that early oral or enteral feeding should be started within a few days if patients with AP achieve pain relief and improved gastrointestinal function[1)]. Nevertheless, the optimal nutritional approach for children is unclear.

[1)] *Department of Pharmacy, Kofu Municipal Hospital,* 366 Masutsubochou, Kofu City, Yamanashi 400-0832, Japan.
[2)] *Department of Pediatrics, Kofu Municipal Hospital.*
[3)] *Department of Healthcare and Regulatory Sciences, Division of Social Pharmacy, Showa University School of Pharmacy.*
* To whom corresponding should be addressed.

We used a low-fat elemental diet (ED) as nutritional intervention in a 5-year-old boy with mild AP. We administered the low-fat ED orally, without using tube devices, and this maintained clinical remission and improved our patient's quality of life.

Case Report

A 5-year-old Japanese boy was admitted to hospital 3 days after onset of illness. He had developed several digestive symptoms: mild abdominal pain, vomiting a small amount of yellow liquid, a fever of 38.9°C, headache, dehydration, weight loss, and fatigue. On examination, delayed growth was observed (his height was 2.5 standard deviations below the mean height for age and weight was 1.0 standard deviation below the mean weight for age) but no developmental retardation or anomalies were observed. He had previously visited a pediatric clinic for investigation of short stature, but no causes were found. His characteristics on admission are shown in Table 1.

The patient was diagnosed with mild AP according to the INSPIRE criteria, as shown in Table 2[6]. Diagnostic imaging showed no abnormalities, so the case was classified as idiopathic AP. The patient was administered gabexate mesilate, famotidine and cefotiam hydrochloride while fasting. When oral re-feeding was started after 3 days of fasting (day 7), the patient's pancreatic enzyme levels became elevated (Fig. 1 and Table 3). His maximal daily energy and

Table 1. Patient characteristics on admission to hospital

Clinical parameters	
Age	5 years
Gender	Male
Body weight	15.6 kg
Body height	98 cm
Body mass index	16.2 kg / m^2
Hematological parameters	
White blood cell count	8,100 / μl
Red blood cell count	4,810 × 10^3 / μl
Hemoglobin	13.5 g / dl
Hematocrit	40.1%
Platelet	230 × 10^3 / μl
pH	7.272
PaO_2	40.4 mmHg
$PaCO_2$	40.3 mmHg
HCO_3^-	17.3 mEq / l
Base excess	–8.2 mEq / l
SaO_2	68.8%

Biochemical parameters	
Total protein	6.6 g / dl
Albumin	4.4 g / dl
Aspartate aminotransferase	58 IU / l
Alanine aminotransferase	28 IU / l
Lactate dehydrogenase	296 mg / dl
Alkaline phosphatase	380 mg / dl
Total bilirubin	0.7 mg / dl
Urea nitrogen	17 mg / dl
Creatinine	0.36 mg / dl
Sodium	131.7 mEq / l
Potassium	4.3 mEq / l
Chloride	95 mEq / l
Calcium	9 mg / dl
Creatinine kinase	80 IU / l
Total cholesterol	150 mg / dl
Triglyceride	73 mg / dl
Glucose	68 mg / dl
C-reactive protein	1.4 mg / dl
p-Amylase	582 IU / l
Lipase	438 IU / l
Trypsin	4,500 ng / ml

Table 2. Application of INSPIRE criteria for diagnosis of acute pancreatitis in our patient

Acute pancreatitis criteria (at least 2 out of 3 required for diagnosis)	Criterion met in our patient
Abdominal pain suggestive of, or compatible with AP (ie, abdominal pain of acute onset, especially in the epigastric region)	Yes
Serum amylase and / or lipase activity at least 3 times greater than the upper limit of normal (IU / l)	Yes
Imaging findings characteristic of, or compatible with AP*	No

*For example, using transabdominal ultrasonography, contrast-enhanced computed tomography, endoscopic ultrasonography, magnetic resonance imaging or magnetic resonance cholangiopancreatography.

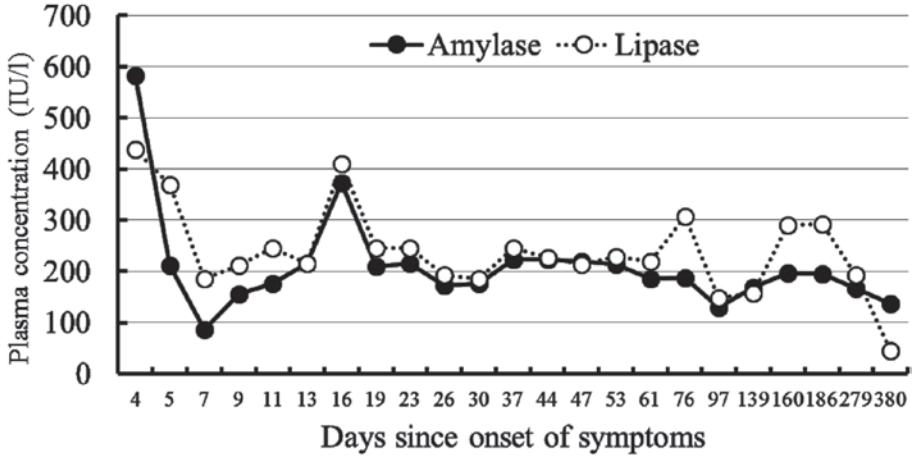

Fig. 1. Changes in plasma amylase and lipase levels during hospitalization and after discharge

Table 3. Clinical and biochemical parameters for our patient during hospital admission and after discharge

		Days after onset of symptoms									After discharge	
		4	7	11	16	19	26	37	44	47	186	380
p-Amylase	(IU / l)	582	86	177	373	210	172	224	224	220	195	136
Lipase	(IU / l)	438	185	245	410	246	193	246	227	214	293	45
CRP	(mg / dl)	1.4	-	-	0.1	0.1	N.D.	N.D.	N.D.	N.D.	N.D	N.D
Albumin	(g / dl)	4.4	-	-	4.3	4.0	4.2	4.3	4.2	4.5	4.6	4.8
Total Cholesterol	(mg / dl)	140	150	-	148	131	89	197	139	123	156	173
Body weight	(kg)	15.6	15.5	15.1	14.1	14.4	14.8	15.4	15.5	-	16	17.5
Body height	(cm)	98.0										103.9

N.D., not detected.

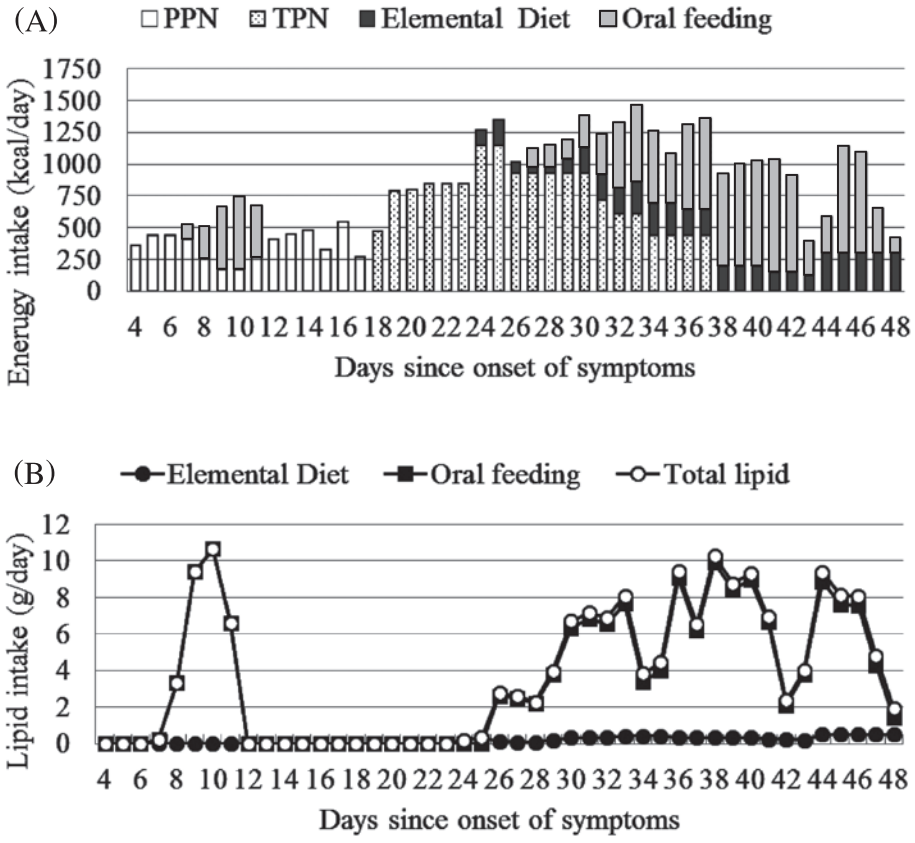

Fig. 2. Energy intake and lipid intake
(A) Nutrition management and energy intake during hospitalization.
(B) Daily fat intake from meals during hospitalization – from ED, from oral feeding and total.
PPN, peripheral parenteral nutrition ; TPN, total parenteral nutrition.

fat intake were 739 kcal and 10.7 g, respectively (Fig. 2). Feeding was stopped again on day 18. The patient was given TPN to allow the pancreas to rest, because post-pyloric tube feeding can be distressing for pediatric patients. His clinical symptoms stabilized, so nutritional management with oral ED was initiated, and followed by additional oral diet (day 24), to enable withdrawal and discontinuation of TPN. The ED (Elental; EA Pharma Co., Ltd. Tokyo, Japan) contained amino acids (17.6%), dextrin (79.3%), lipids (0.6%), minerals (2.0%), and vitamins (0.5%), and provided 1 kcal/ml, 0.05 g / ml protein, and 1.7 mg / ml lipids (Table 4). It did not contain fiber, carnitine or selenium.

After starting the ED combined with an oral diet, the patient's total daily energy intake was increased to 1,033 kcal / day and his fat intake was 8.5 g / day. The ED provided approximately 300 kcal / day without exacerbating pancreatitis-related symptoms or increasing pancreatic enzyme levels (Fig. 1 and Table 3). The ED therapy made it possible to provide sufficient energy while restricting fat intake. We were able to withdraw the patient from TPN on day 38 and discharge him from hospital on day 48. After discharge, he continued ED therapy at home. He has reported no further clinical symptoms for more than 1 year, and his clinical laboratory

Table 4. Composition of the elemental diet used to manage acute pancreatitis in our patient

Total	80 g (300 kcal)	Amino acids	13.14 g
Amino acids	13.14 g	L-Isoleucine	642 mg
Carbohydrate (dextrin)	63.41 g	L-Leucine	899 mg
Lipid (soybean oil)	0.51 g	L-Lysine · HCl	888 mg
Vitamin A	223.2 μg (648 IU)	L-Methionine	648 mg
Vitamin D	1.3 μg (51.2 IU)	L-Phenylalanine	871 mg
Vitamin B1	152 μg	L-Threonine	523 mg
Vitamin B2	244 μg	L-Tryptophan	151 mg
Vitamin B6	220 μg	L-Valine	701 mg
Vitamin B12	0.72 μg	L-Histidine · HCl · H2O	501 mg
Niacin	2.20 mg	L-Arginine · HCl	1,125 mg
Pantothenic acid	1.10 mg	L-Alanine	899 mg
Folic acid	44 μg	Mg · K · L-Aspartate	1,036 mg
Vitamin C	7.80 mg	Na · L-Aspartate · H2O	867 mg
Vitamin K	9 μg	L-Glutamine	1,932 mg
Vitamin E	3.3 mg (3.3 IU)	Glycine	505 mg
Biotin	39 μg	L-Proline	630 mg
Choline	8.56 mg	L-Serine	1,159 mg
Na	260.0 mg (11.3 mEq)	L-Tyrosine	110 mg
K	217.6 mg (5.6 mEq)		
Cl	516.8 mg (14.6 mEq)		
Mg	40.0 mg (3.3 mEq)		
Ca	157.6 mg (7.9 mEq)		
P	121.6 mg (3.9 mmol)		
Fe	1.8 mg (32.2 μmol)		
I	15.2 μg (0.1 μmol)		
Mn	0.3 mg (5.5 μmol)		
Cu	0.2 mg (3.2 μmol)		
Zn	1.8 mg (27.5 μmol)		

The diet was prepared by dissolving 80 g of the elemental diet in 300 ml of cold or lukewarm drinking water (1 kcal / ml). The solution was administered orally once daily or divided into several portions per day. In cases of tube feeding, the solution is administered into the duodenum or jejunum continuously over 24 hr via a nasogastric tube or a gastric or intestinal fistula (at an injection rate of 75-100 ml / hr).

data have not been exacerbated. He has maintained good nutritional status according to his serum albumin and total cholesterol levels (Table 3). He has grown in keeping with his height standard deviation (Fig. 3).

Discussion

Several reports have indicated that nutritional management is an efficacious and cost-effective strategy for the treatment of AP[7, 8]. The issues that should be considered during the nutritional management of AP are: optimal timing of the nutritional therapy, optimal feeding route (oral, gastric, jejunal, or parenteral), and optimal nutrient formulation (elemental, semi-elemental,

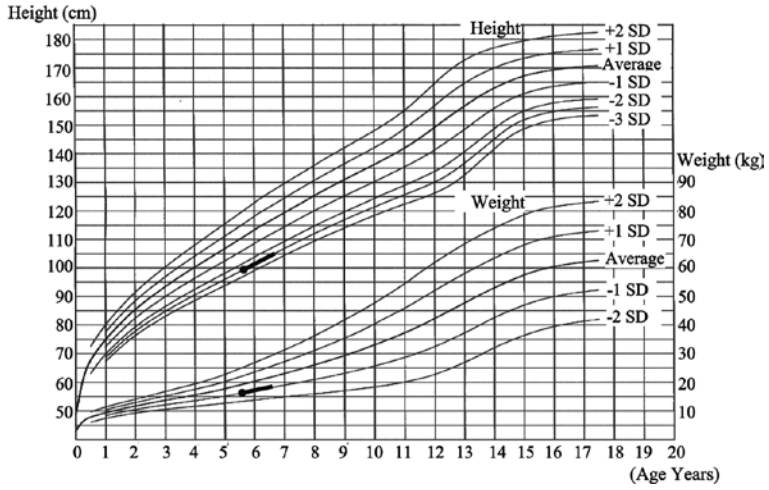

Fig. 3. Height-for-age and weight-for-age percentiles for boys aged 0–18 years, showing the growth curve for our patient (indicated by thick lines beginning with a filled circle).

polymetric, immune-enhancing, and prebiotics and probiotics)[1, 9, 10]. Few reports and guidelines on nutritional management of pediatric patients with AP have been published[4]. Because children require high energy and nutrition for growth, children with AP are at risk of acute malnutrition because of the energy and nutrition deficiencies caused by the catabolic state of the disease and treatment using oral food restriction[11]. It is extremely important to supply sufficient energy and nutrients according to the condition of the patient.

The causes of AP differ between pediatric and adult patients[12]. The cause of the disease in our patient was not identified from his anamnesis, medical history, diagnostic imaging, or biochemical analysis. Therefore, he was diagnosed with idiopathic disease. Nutritional management was necessary to improve his nutritional status and achieve normal growth.

According to the European Society for Clinical Nutrition and Metabolism guidelines, parenteral nutrition is required only when the gut has failed or enteral nutrition is impossible for other reasons (eg, because hypoalimentation will cause protein catabolism and may worsen prognosis). Nutritional support therapy is not always required in cases of mild-to-moderate AP, but it is required when an unexpected complication develops or when advancing to an oral diet within 7 days is not possible. In patients stabilized on parenteral nutrition, repeated efforts should be periodically conducted to initiate enteral nutrition[1]. Kumar and Gariepy have reported that oral nutrition is well tolerated in patients with mild disease and that such treatment strategies may be beneficial in children with AP, on a case-by-case basis[13].

In our patient's case, it was possible to switch to oral feeding using a low-fat diet after fasting for about 3 days because his symptoms were not severe and clinical improvements were observed during fasting. However, clinical laboratory data indicated that his condition was exacerbated after starting ingestion, so we needed to stop oral feeding. We first performed peripheral parenteral nutrition (PPN) under fasting conditions. However, the energy

requirements of the patient were approximately 1,300 kcal / day, and the energy provided by PPN was insufficient. The patient was malnourished, so temporary TPN was selected to avoid nutritional depletion, instead of post-pyloric tube feeding (to avoid distress). The exacerbation observed after the oral re-feeding with a low-fat diet may have been because the re-feeding was initiated too soon or because the diet contained an inappropriate amount of fat. Therefore, we stopped the feeding and started oral administration of a low-fat ED containing amino acids after the temporary TPN therapy. We progressively increased nutritional support to the patient and added a low-fat diet.

Other authors have reported that initiating oral feeding at an early stage may stimulate pancreatic secretion, and cause recurrence of pain[14]. Teich *et al* reported that normalization of serum lipase levels was not required for enteral nutrition in patients with mild AP[15].

Meng *et al* indicated that a non-liquid (soft) diet did not increase pain recurrence after re-feeding[16]. Elemental formulas (containing individual amino acids and almost no fat) cause less stimulation of the pancreas than standard formulas with intact proteins and long-chain fatty acids, because an ED stimulates only low levels of cholecystokinin secretion[17]. However, long-term fat restriction with nutritional management strategies such as ED or TPN without lipid emulsion may lead to essential fatty acid deficiency – after 2 weeks in pediatric patients, and after 4 weeks in adult patients[18]. Thus, when AP needs to be managed over a long period, essential fatty acid supplementation, such as lipid emulsion infusion, should be considered. The ED that we used to treat our patient (Elental, Table 4) is a suitable formulation for patients with AP[15]. The ED is a transintestinal high-calorie diet that can be absorbed easily with little residue or fat. Therefore, oral ED is a physiological method of providing more calories during the early phase of mild pediatric AP. Although there are no clear recommendations regarding the daily fat intake or fat restriction targets in pediatric AP, the fat intake in the current patient was 8.5 g / day during oral ED therapy (total 1,033 kcal / day). This was adequately low to maintain his pancreatic enzyme levels and provide sufficient total energy.

Conclusion

Nutritional management combining ED with oral feeding in our patient (including ED therapy at home after discharge from hospital) was safe, maintained clinical remission, and improved our patient's quality of life. Further prospective clinical studies are necessary to better define optimal nutritional management of pediatric AP.

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Conflict of interest

We declare no conflict of interest.

Patient consent

Obtained.

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