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• BASIC RESEARCH •

Parenteral versus early intrajejunal nutrition: Effect on pancreatitic natural course, entero-hormones release and its efficacy on dogs with acute pancreatitis

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Abstract

AIM: To evaluate the effect of early intrajejunal nutrition (EIN) on the natural course, entero-hormone secretion and its efficacy on dogs with acute pancreatitis.

METHODS: An acute pancreatitis model was induced by injecting 1 ml/kg of combined solution (2.5 % sodium taurocholate and 8 000-10 000 BAEE units trypsin/ml) into the pancreas via pancreatic duct. Fifteen dogs were divided into parenteral nutrition (PN) group and EIN group. Two groups were isonitrogenous and isocaloric. EIN was used at postoperative 24 h. Serum glucose, calcium, amylase and lysosomal enzymes were determined before and 1, 4, 7 d after acute pancreatitis was induced. All the dogs were injected 50 uCi ^{125}I-BSA 4 h before sacrificed on the 7th day. The ^{125}I -BSA index of the pancreas/muscle, pancreas/blood, and pancreas pathology score (PPS) were determined. The peripheral plasma cholecystokinin (CCK), secretin (SEC) and gastrin were measured by ELISA and RIA, and was quantitative analysis of pancreatic juice and amylase, pancreatolipase and HCO_3^-, Cl^-, Na^+ and K^+ performed by an autochemical analyzer at 30, 60, 120 and 180 min after beginning PN or EIN on the first day.

RESULTS: There was no difference between two groups in the contents of serum calcium, amylase and lysosomal enzymes, ^{125}I-BSA index of pancreas/muscle and pancreas/blood and PPS. The contents of CCK and gastrin in EIN were higher than those in PN group at 60 and 120 min (P<0.05). The content of SEC post-infusion of nutrition solution was higher than that of pre-infusion of nutrition solution in both groups, and only at 60 min SEC in EIN group was higher than that in PN group. The content of gastrin in EIN was higher than that in PN group at 120 and 180 min (P<0.05). The changes of pancreatic juice, amylase, pancreatolipase and HCO_3^-, Cl^-, Na^+ and K^+ between two groups did not reach significantly statistical difference (P>0.05).

CONCLUSION: EIN does not stimulate entero-hormone and pancreatic juice secretion, and enzyme-protein synthesis and release. EIN has no effect on the natural course of acute pancreatitis.

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INTRODUCTION

Total parenteral nutrition (TPN) has been the standard practice for providing exogenous nutrients to patients with acute pancreatitis in order to improve their nutritional status and to avoid pancreatic stimulation. However, TPN is associated with certain disadvantages. In particular, there is an increased risk of central catheter infection, severe hyperglycaemia, and other metabolic and electrolyte disturbances and a possible exacerbation of metabolic disturbances. TPN may also result in gut barrier function alterations due to increasing intestinal permeability[1-10].

Benefits from the use of total enteral nutrition (TEN) have been noted in a number of other diseases, such as burns, trauma, and sepsis. In comparison with TPN, use of TEN reduces nosocomical infection, multiple organ failure (MOF), and length of hospitalization[3,4]. The use of early enteral feeding for nutritional support in patients with acute pancreatitis has not been evaluated systematically. The commonly encountered problems of gastric atony and outlet obstruction have limited the successful delivery of enteral formulas to patients with severe acute pancreatitis. In addition, many surgeons hold scrupulously that the EIN may exacerbate the clinical pathological features, and lead to recurrence of symptoms and delayed complications to be cured[11-18]. However, these problems could be overcome if enteral nutrition is delivered to the distal ileum far away from the Treitz' s ligament, to avoid stimulation of the cephalic and gastric phase, and those effects are not so pronounced as nutrients are delivered directly into jejunum[3,9-13]. Therefore, it is necessary to investigate the efficiency of early intrajejunal nutrition on pancreatitic clinicopathological changes, entero- hormone release and its efficacy on dogs with acute pancreatitis.

MATERIALS AND METHODS

Animal model

A total of 22 dogs weighing 18-22 kg, were allowed *ad libitum* intake of water. After fasted for 12-14 hours, all the dogs were induced anesthesia by intramuscular injection of ketamine 10 ml/kg, and intravenous injection of sodium pentobarbital 30 mg/kg. Under sterile conditions, a middle laparotomy and a duodenotomy were performed. The duodenum papilla was found and a silastic catheter was inserted into pancreatic tube and fixed for collecting pancreatic juice. Acute pancreatitis model was induced by injecting 1 mg/kg of combined solution of 2.5 % sodium taurocholate and 8 000-10 000 BAEF units trypin/ml into pancreas via pancreatic duct with a pressure of 30 cmH_2O, and the common biliary duct was clamped. After the model

was established, the duodenum and abdomen were closed. A catheter via jejunostomy was set at 30 cm away from the Treitz' s ligament. The neck region of the dogs was shaved and prepared in a sterile manner for catheterization. A silastic catheter (1.0 mm inner diameter and 1.5 mm outer diameter) was inserted through the external jugular vein to reach the superior vena cava. The catheter was fixed to connect the infusion solution. Fifteen dogs with acute pancreatitis survived over 7 days, and the death rate was 32 % (7/22). The trial was approved by the Institutional Animal Committee.

Experimental groups and preparation of nutritional solution

Fifteen dogs having survived over 7 days with acute pancreatitis were randomly divided into PN group (n=7) and EIN group (n=8). The two groups were isocaloric and isonitrogenous. The PN solutions were consisted of 7 % Vamin (SSPC, 9.4 g/1 000 ml) and 20 % Intralipid (SSPC) and 50 % glucose (GS). Non-protein calorie was 50 kC (209.2 kJ/kg) and nitrogen was 0.3 g/kg/d. The total volume of solution infused was 70 ml/kg/d. Energy index supported with glucose and fat emulsion was 1∶1. Multivitamins and electrolytes were also contained in TPN solutions. The normal saline was infused by 250 ml/kg during operation time and 8 h postoperation, and then infused with 125±25 ml/kg. The nutrient solution was infused at a constant infusion rate by a pump (100-120 ml/h).

The EIN solution was Nutrison (Nutricia). At the 24th h after acute pancreatitis was induced, the jejunum through jejunostomy catheter was infused 250 ml Nutrison and 500 ml NS. At the 48th h after acute pancreatitis, 500 ml Nutrison and 250 ml NS were infused for 5 days. The infusion rate was controlled by a microcomputer-pump (Nutricia). During EIN supporting, the content with insufficient calories and nitrogen were supplemented by partial parenteral nutrition[3,16,18].

Laboratory tests, ^{125}I-BSA index and pancreatic pathology

Serum glucose, calcium, amylase and lysosomal enzymes (according to Kit' s indication) were determined before and 1, 4, 7 d after the occurrence of acute pancreatitis. All the dogs were injected 50 uCi ^{125}I-BSA 4 h before sacrificed on the seventh day. The ^{125}I-BSA volume in the pancreas (/g), muscle of the right leg (/g) and blood (/ml) were tested by a r-accounter radioimmunology analyzer. The ^{125}I -BSA index of the pancreas/muscle and pancreas/blood was measured. Fixed tissues of the pancreatic head, body, tail and the total pancreas were sectioned and the histological change was observed. Pancreatitic pathological scores (PPS) were taken by the extent of pancreas tissue edema, inflammation, hemorrhage and necrosis according to scores 1, 2, 3 and 4, and PPS of the different parts of the pancreas was determined.

Entero-hormone determination

Twenty-four hours after the occurrence of acute pancreatitis, serum was used to determine the CCK and SEC (Peninsula Laboration, Inc.USA), and gastrin (Beijing Furui Bioengineer Co.) at the same time before and 30, 60, 120 and 180 min after PN or EIN. The former two samples were measured by competitive ELISA. The serum gastrin was measured by competitive binding RIA. The serum amylase, pancreaolipase and pancreatic juice, electrolytes (HCO_3^-, Cl^-, Na^+ and K^+) were determined by a 1 600 full-automatic biochemical analyser.

Statistical analysis

These data were expressed as means ± SEM, and comparison between two groups was made using χ^2 analysis of variance. A P value less than 0.05 was considered statistically significant.

RESULTS

Changes of serum glucose, calcium, amylase and lysosomal enzymes

After the acute pancreatitis model was prepared, serum glucose level in PN group was higher as compared with that in EIN group during the whole experimental period. Serum calcium was markedly decreased after acute pancreatitis was induced, and there was no difference in the changes of serum calcium in the latter days between two groups. Serum amylase and lysosomal enzymes (LE) increased and gradually decreased later during the experimental period, there was no difference between two groups (P>0.05) (Table 1).

Table 1 Effect of different nutrition support methods on serum glucose, calcium amylase and lysosomal enzymes

Groups	Time	Glu (umol/L)	Ca (mmol/L)	Amylase (SU)	LE(U)
PN group	Pre-AP	4.0±0.5	2.50±0.10	328±96	22±10
	30 min	8.3±0.8[a]	2.42±0.11	636±100[a]	53±11
	1 d	9.7±0.7[a]	2.35±0.09	1 689±298[a]	68±17
	4 d	10.3±1.0[a]	2.36±0.13	1 150±416[a]	45±14
	7 d	9.80±1.1[a]	2.20±0.11	1 060±260[a]	47±13
EIN group	Pre-AP	4.7±0.8	2.34±0.16	400±110	26±9
	30 min	8.4±1.0[a]	2.39±0.11	734±164	64±17
	1 d	8.9±0.8[a]	2.31±0.09	1 289±439[b]	71±13
	4 d	6.7±1.0	2.34±0.15	1 215±416	50±19
	7 d	6.6±0.7	2.30±0.09	1 169±362	52±20

[a]P<0.05, *vs* pre-SAP, [b]P<0.05, *vs* PN group, Pre-AP: before acute pancreatitis.

^{125}I-BSA index and pancreatitic pathological scores

^{125}I-BSA index of pancreas/muscle and pancreas/blood in EIN group (4.22±0.18 cpm/g, 0.22±0.03 cpm/ml) and PN group (3.69±0.26 cpm/g, 0.17±0.02 cpm/ml) did not reach statistical difference (P>0.05). Pancreatitic pathological scores (PPS) of different parts including head, body, tail and total pancreas in EIN group (1.40±0.24, 2.3±0.20, 2.1±0.20, 1.9±0.23) and PN group (1.30±0.38, 2.4±0.22, 2.2±0.22, 1.9±0.06) also did not reach statistical difference (P>0.05).

Entero-hormone, pancreatic juice and their components analysis

CCK The content of CCK in EIN group was higher than that in PN group at 30 and 60 min (P<0.05). There was no difference before and after nutrition liquid infusion in PN group (Table 2).

Table 2 Changes of serum CCK at different times (ng/ml)

Group	0 min	30 min	60 min	120 min	180 min
PN group	0.24±0.02	0.17±0.01	0.23±0.01	0.23±0.02	0.33±0.03
EIN group	0.22±0.02	0.29±0.06[a]	0.33±0.05[a]	0.29±0.03[a]	0.31±0.05

[a]P<0.05 *vs* PN group.

Table 3 Changes of serum SEC at different times (ng/ml)

Group	0 min	30 min	60 min	120 min	180 min
PN group	0.33±0.03	0.65±0.14	0.74±0.17	0.61±0.20	0.56±0.23
EIN group	0.35±0.06	0.61±0.17	0.88±0.25[a]	0.64±0.13	0.61±0.25

[a]P<0.05 *vs* PN group.

SEC SEC after infusion of nutrition liquid was higher than that before infusion of nutrition liquid ($P<0.05$), but it did not reach statistical difference between two groups at 30, 120 and 180 min ($P>0.05$). SEC only at 60 min in EIN group was higher than that in PN group (Table 3).

Gastrin The changes of gastrin in the PN group did no reach statistical difference before and after nutrition liquid infusion. Gastrin in EIN group gradually increased, and was higher than that in PN group at 120 and 180 min ($P<0.05$) (Table 4).

Table 4 Changes of serum gastrin at different times (pg / ml)

Group	0 min	30 min	60 min	120 min	180 min
PN group	12.5±3.7	13.9±4.1	20.8±5.1	16.4±5.2	17.6±6.3
EIN group	12.2±3.2	14.4±4.6	20.7±5.5	24.2±6.3[a]	25.3±6.5[a]

[a]$P<0.05$ *vs* PN group.

Pancreatic secretion and its component analysis

The changes of pancreatic juice, amylase, pancreatolipase, HCO_3^-, Cl^-, Na^+ and K^+ did not reach statistical difference between two groups (Table 5).

Table 5 Changes of pancreatic juice, amylase, pancreatolipase, HCO_3^-, Cl^-, Na^+ and K^+

Component	PN group	EIN group
Pancreatic juice (ml)	6±1	9±3
Amylase (U / L)	6 717±540	7 121±670
pancreatolipase (U / L)	629±78	661±101
HCO_3^- (mmol / L)	17±3	22±4
Cl^-(mmol / L)	117±11	126±9
Na^+(mmol / L)	133±21	147±17
K^+(mmol / L)	4.1±1.0	5.7±1.4

DISCUSSION

Autodigestion of the pancreas is the main mechanism of acute pancreatitis. The conception of pancreatic rest stems from the belief that stimulation of pancreatic exocrine function in patients with acute pancreatitis releases large quantities of proteolytic enzymes that result in autodigestion of the inflammatory pancreas and peripancreatic tissues, causing a deterioration in the patient' s condition. The presence of food in the stomach and duodenum elicits gastropancreatic and duodenopancreatic reflexs that result in stimulation of pancreatic exocrine secretions. Therefore, traditionally, enteral nutrition could be adopted after parenteral nutrition support for over 2-3 weeks. That means to keep the pancreas in rest and rehabilitation for a long time. However, these effects are not so pronounced when nutrients are delivered directly into the jejunum[11-18].

Heidenhain in 1 875 first demonstrated the effect of vagal and entero- hormone stimulation on pancreatic secretion, in which hormones played a more important role than vagal stimulation. There were three classic phases, namely cephalic, gastric and intestinal phases of digestion that describe the response of the pancreas to a meal. The hormones served a major function in mediating pancreatic exocrine secretion[16,19]. Normally, during the cephalic and gastric phases, oral nutrients can stimulate the release of gastric acid, duodenum juice and pancreatic enzyme, and activation of proteins and peptide in the nutrients commences after the peptidase enters the duodenum, where mucosal enterokinase cleaves trypsinogen to trypsin, leaving trypsin to further activate the other peptidases, and then stimulates entero-hormones secretion such as cholecystokinin (CCK), secretin (SEC) and gastrin to increase pancreatic secretion. It is known that CCK and SEC are synthesized in the mucosal I and S cells of the crypts of Liberkuhn in the proximal small intestine and released in the presence of luminal acid and bile. The gastic G cell product, gastrin, which serves a major function to promote gastric acid release also serves as a weak stimulator of pancreatic enzyme secretion. CCK is one of the most important entro-hormones known to stimulate pancreatic enzyme secretion. Some authors also found that avoidance of cephalic, gastric and duodenual stimuli by jejunal tube feeding did not result in pancreatic stimulation. They concluded that bypassing the stomach, and minimizing acid secretion, played an important role in keeping the pancreas at rest[16,21,22].

Some authors[20-25] described an experience of early enteral nutrition in severe acute pancreatitis using nasoenteral feeding. No patients developed relapse, hypertri-glyceridaemia or abnormalities of liver function, indicating that jejunal feeding can be used safely in acute pancreatitis without reactivation of the inflammatory process[26-32]. Our experimental results showed that the changes of serum glucose, calcium, and amylase did not reach statistical difference between two groups. The serum lysosomal enzymes is believed to be the gold standard for reflecting the extent of pancreatic tissue necrosis and inflammation and more attention has been paid to them in international medicine. Once the pancreatic tissue necrosis stoped, the volume of systemic lysosomal enzymes discharging from pancreatitis tissue would be attenuated. Our results indicated that serum lysosomal enzymes was markedly increased after acute pancreatitis was induced, but did not reach statistical difference between two groups. In addition, the ^{125}I-BSA index of pancreas/muscle and pancreas/blood reflected the permeability of the pancreas microcirculation. If this ^{125}I-BSA index decreased, microvessal permeability would be improved. Once the ^{125}I- BSA index in pancreatic tissue increased, the microvessal permeability elevated and deteriorated pancreatitis. Our study showed that administration of EIN did not increase the content of serum lysosomal enzymes, and deteriorate the course of acute pancreatitis. As to the PPS in different parts of the pancreas, there was no difference between EIN group and PN group. Kalfarentzos *et al*[17], reported that EIN was well tolerated following acute pancreatitis, and was of comparable efficacy to PN. In fact, EIN did not deteriorate pancreatitic pathology, and might be safely adopted in dogs with acute pancreatitis[26-32].

Normally, it is known that secretion of CCK, SEC and gastrin is mainly located in the duodenum and jejunum. The number of CCK-produced cells is 11-30 per square millimeter both in duodenum and in proximal jejunum, and their amount is 52.5±8 pmol/g, and 26±5 pmol/g respectively; The number of pancreatic SEC-produced cells is >31 in duodenum, 1-10 in proximal jejunum per square millimeter respectively, and their amount is 73±7 pmol/g and 32±4 pmol/g. The number of gastrin CCK-produced cells is >31 in gastric antrum, 11-30 in duodenum and 1-10 in proximal jejunum per square millimeter respectively, and their amount is 2 342±14 pmol/g, 1 397±192 pmol/g, 190±24 pmol/g, respectively. Therefore, the secretory locations of entero-hormones are mostly in gastric antrum, duodenum and proximal jejunum, and less in the distal jejunum. Theoretically, if the nutrients were infused from proximal jejunum to distal jejunum, it would decrease stimulatory activation of pancreatic secretion to a minimum degree[16].

To further study the possibility of EIN stimulated entero-hormones and pancreatic juice release, we observed the effects of different nutrition support methods on the CCK, SEC, gastrin and pancreatic secretion and their components. Our results suggested that the serum CCK increased at 60 min after nutrient was infused in EIN group as compared with PN group.

It was interesting that SEC was elevated after nutrition infusion in both groups, but it was only higher at 60 min in EIN group than in PN group. The serum gastrin was gradually increased in EIN group at 120 and 180 min as compared with PN group. Based on pancreatic juice and its component analysis, our results suggested that the amount of pancreatic juice was higher in EIN than in PN group. But the changes of the amylase, pancreatolipase and electrolytes were not significant. The study suggested that EIN indeed stimulated entero- hormones secretion at some degrees, but did not increase enzyme-proetin and pancreatic juice secretion. The reason was not clear, maybe due to the fact that the pancreatic acinar cells swelling, hemorrhage and necrosis decreased the physiological efficiency of entero-hormones by altering the membrane receptor number and activity.

In recent years, effect of human EIN or oral feeding on the natural course and entero-hormones secretion was seldom reporte. It was well tolerable, feasible and desirable as TPN in the management of acute pancreatitis, but it failed to reveal any detrimental effect on the clinical pathologic features of AP, and increase pancreatic secretion. Therefore, EIN can contribute to the study of pancreatic natural course, and may play an important role in keeping the pancreas at rest, bypassing the stomach, and minimizing acid secretion.

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