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

Huan-Long Qin, Zhen-Dong Su, Lei-Guang Hu, Zai-Xian Ding, Qing-Tian Lin

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 uC

RESULTS: There was no difference between two groups in the contents of serum calcium, amylase and lysosomal enzymes. The 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 performed by an autochemical analyzer at 30, 60, 120 and 180 min after beginning PN or EIN on the first day.

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.

Qin HL, Su ZD, Hu LG, Ding ZX, Lin QT. Parenteral versus early intrajejunal nutrition: Effect on pancreatic natural course, entero-hormones release and its efficacy on dogs with acute pancreatitis. World J Gastroenterol 2003; 9(10): 2270-2273
http://www.wjgnet.com/1007-9327/9/2270.asp

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.

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 nosocomial infection, multiple organ failure (MOF), and length of hospitalization. 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 that the EIN may exacerbate the clinical pathological features, and lead to recurrence of symptoms and delayed complications to be cured. 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. Therefore, it is necessary to investigate the efficiency of early intrajejunal nutrition on pancreatic 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 BAEE units trypsin/ml into pancreas via pancreatic duct with a pressure of 30 cmH2O, 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 kcal (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 Nutrin (Nutricia). At the 24th h after acute pancreatitis was induced, the jejunum through jejunostomy catheter was infused 250 ml Nutrin and 500 ml NS. At the 48th h after acute pancreatitis, 500 ml Nutrin 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, 125I-BSA index and pancreatic pathology**

Serum glucose, calcium, amylase and lysosomal enzymes (according to Kt’s indication) were determined before and 1, 4, 7 d after the occurrence of acute pancreatitis. All the dogs were injected 50 uG 125I-BSA 4 h before sacrificed on the seventh day. The 125I-BSA volume in the pancreas (g), muscle of the right leg (g) and blood (ml) were tested by a r-counter radioimmunology analyzer. The 125I-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. Pancreatic 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, pancreolipase and pancreatic juice, electrolytes (HCO₃⁻, 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 χ² 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 increased and gradually decreased later during the experimental period, there was no difference between two groups (P>0.05) (Table 1).

| 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* | 2.42±0.11 | 636±100* | 53±11 |
| 1 d   | 9.7±0.7* | 2.35±0.09 | 1 689±298* | 68±17 |
| 4 d   | 10.3±0.1* | 2.36±0.13 | 1 150±16* | 45±14 |
| 7 d   | 9.80±1.1* | 2.20±0.11 | 1 060±260* | 47±13 |
| EIN group | Pre-AP | 4.7±0.8 | 2.34±0.16 | 400±110 | 26±9 |
| 30 min | 8.4±1.0* | 2.39±0.11 | 734±164* | 64±17 |
| 1 d   | 8.9±0.8* | 2.31±0.09 | 1 289±39* | 71±13 |
| 4 d   | 6.7±0.1 | 2.34±0.15 | 1 215±16 | 50±19 |
| 7 d   | 6.6±0.7 | 2.30±0.09 | 1 169±362 | 52±20 |

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

**125I-BSA index and pancreatic pathological scores**

125I-BSA index of pancreas/muscle and pancreas/blood in EIN group (4.22±0.18 cpm/g, 0.23±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). Pancreatic 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).

| 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* | 0.33±0.05* | 0.29±0.03* | 0.31±0.05 |

*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* | 0.64±0.13 | 0.61±0.25 |

*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 not 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)

|                | 0 min  | 30 min | 60 min | 120 min | 180 min |
|----------------|--------|--------|--------|---------|---------|
| PN group       | 12.5±3.7 | 13.9±1.1 | 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 | 25.3±6.5 |

$^{a}P<0.05$ 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 pancreatic tissues, causing a deterioration in the patient’s condition. The presence of food in the stomach and duodenum elicits gastropancreatic and duodenopancreatic reflexes 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.

Heidenhain in 1875 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.

Normally, during the cephalic and gastric phases, oral nutrients can stimulate the release of gastric acid, duodenal 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 gastric 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 entero-hormones known to stimulate pancreatic enzyme secretion. Some authors also found that avoidance of cephalic, gastric and duodenal 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.

Some authors described an experience of early enteral nutrition in severe acute pancreatitis using nasoenteral feeding. No patients developed relapse, hypertriglyceridaemia or abnormalities of liver function, indicating that jejunal feeding can be used safely in acute pancreatitis without reactivation of the inflammatory process. 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 stopped, 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., reported that EIN was well tolerated following acute pancreatitis, and was of comparable efficacy to PN. In fact, EIN did not deteriorate pancreatic pathiology, and might be safely adopted in dogs with acute pancreatitis.

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 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 3±7 pmol/g and 32±5 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 jejunal, and less in the distal jejunal. Theoretically, if the nutrients were infused from proximal jejunum to distal jejunum, it would decrease stimulatory activation of pancreatic secretion to a minimum degree.

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, pancratoilipase and electrolytes were not significant. The study suggested that EIN indeed stimulated entero- hormones secretion at some degrees, but did not increase enzyme-protein 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.

REFERENCES
1. Kalfarentzos FE, Karavias DD, Karatzas TM, Alevizatos BA, Andreoulakis JA. Total parenteral nutrition in severe acute pancreatitis. J Am Coll Nutr 1991; 10: 156-162
2. Buchman AL, Moukarzel AA, Bhutta S, Belle M, Ament ME, Eckert CD, Hollander D, Gornbein J, Kopple JD, Vijayaraghavan SR. Parenteral nutrition is associated with intestinal morphologic and functional changes in humans. JPN 1995; 19: 453-460
3. Qin HL, Su ZD, Hu LG, Deng ZX, Lin QT. Effect of early intrajejunal nutrition on pancreatic pathological features and gut barrier function in dogs with acute pancreatitis. Clin Nutr 2002; 21: 469-473
4. Mitchell RM, Byrne MF, Baillie J. Pancreatitis. Lancet 2003; 26: 1447-1455
5. Ammori BJ. Gut barrier dysfunction in patients with acute pancreatitis. J Hepatobil Pancreat Surg 2002; 9: 411-412
6. McClave SA, Greene LM, Snider HL, Makk LJ, Byrne MF, Baillie J. Pancreatitis. J Gastroenterol Hepatol 2003; 18: 469-473
7. Vison N, Hechketeweler P, Butel J, Bernier J. Effect of continuous jejunal perfusion of elemental and complex nutritional solutions on pancreatic enzyme secretion in human subjects. Gut 1978; 19: 194-198
8. Mallett P. Enteral nutrition by alimentation jejunoanastomosis in 11 cases of severe acute pancreatitis. In controversies in acute pancreatitis. Holllender LF (ed.). Berlin 1982: P293
9. Lobo DN, Memon MA, Allison SP, Rowlands BJ. Evolution of nutritional support in acute pancreatitis. Br J Surg 2000; 87: 695-707
10. Windsor AC, Kanwar S, Li AG, Barnes E, Guthrie JA, Spark JL, Welsh F, Guillou PJ, Reynolds JD. Compared with parenteral nutrition, enteral feeding attenuates the acid phase response and improves disease severity in acute pancreatitis. Gut 1998; 42: 431-435
11. Abou-Angsi S, Craig K, O’ Keeffe SJ. Hypocaloric jejunal feeding is better than total parenteral nutrition in acute pancreatitis: results of a randomized comparative study. Am J Gastroenterol 2002; 97: 2255-2262
12. Olah A, Pardavi G, Belajyi T, Nagy A, Issekutz A, Mohammeg GE. Early nasojejunal feeding in acute pancreatitis is associated with a lower complication rate. Nutrition 2002; 18: 259-262
13. Chen QP. Enteral nutrition and acute pancreatitis. WJG 2001; 7: 185-192
14. Hallay J. Kovacs G, Szatmari K, Szatmari K, Bako A, Szentkereszy Z, Lakkos G, Sipka S, Sapy P. Early jejunal nutrition and changes in the immunological parameters of patients with acute pancreatitis. Hepatogastroenterology 2001; 48: 1489-1492
15. Eatock FC, Brombacher GD, Steven A, Irrie CW, McKay CJ, Carter R. Nasogastric feeding in severe acute pancreatitis may be practical and safe. Int J Pancreatol 2000; 28: 23-29
16. Qin HL, Su ZD, Hu LG, Ding ZX, Lin QT. Effect of early intrajejunal nutrition on secretion of entero-hormone and its efficiency in acute pancreatic dogs. Chang Wai Chang Nei Ying Yang 2002; 9: 193-195
17. Kalfarentzos F, Kehagias J, Mead N, Kokkinis K, Gogos CA. Enteral nutrition is superior to parenteral nutrition in severe acute pancreatitis: results of a randomized prospective trial. Br J Surg 1997; 84: 1665-1669
18. Qin HL, Su ZD, Ding ZX, Lin QT. Effects of enteral nutrition on uptake of amino acid and enzyme-protein synthesis of pancreatic acinar cell in acute pancreatic dogs. Zhonghua Wai Ke Za Zhi 2003; 41: 146-149
19. Takacs T, Hajnal F, Nemeth J, Lononovics J, Pap A. Stimulated gastroduodenal hormone release and gallbladder contraction during continuous jejunal feeding in patients with pancreatic pseudocyst is inhibited by octreotide. Int J Pancreatol 2000; 28: 215-220
20. Macfie J. Enteral versus parenteral nutrition. Br J Surg 2000; 87: 1121-1122
21. Neoptolemos JP, Ratary M, Finch M, Suttorn R. Acute pancreatitis: the substantial human and financial costs. Gut 1996; 42: 886-891
22. Al-Omran M, Groof A, Wilke D. Enteral versus parenteral nutrition for acute pancreatitis. Cochrane Database Syst Rev 2003; 3: CD002837
23. Austrums E, Pupelis G, Snipek P. Postoperative enteral stimulation by gut feeding improve outcomes in severe acute pancreatitis. Nutrition 2003; 19: 487-491
24. Powell JJ, Murchison JT, Fearson KC, Ross JA, Sirtwardena AK. Randomized controlled trial of the effect of early enteral nutrition on markers of the inflammatory response in predicted severe acute pancreatitis. Br J Surg 2000; 87: 1375-1381
25. McGregor CS, Marshall JC. Enteral feeding in acute pancreatitis: just do it. Curr Opin Crit Care 2001; 7: 89-91
26. Sanabria A. Randomized controlled trial of the effect of early enteral nutrition on markers of the inflammatory response in preventing severe acute pancreatitis. Br J Surg 2001; 88: 728
27. Eckerwall G, Andersson R. Early enteral nutrition in severe acute pancreatitis: providing nutrients, gut barrier protection, immunomodul or all of them? Scand J Gastroenterol 2001; 36: 449-458
28. Pupelis G, Austrums E, Jansone A, Sprucs R, Wehbi H. Randomised trial of safety and efficacy of postoperative enteral feeding in patients with severe pancreatitis: preliminary report. Eur J Surg 2000; 166: 383-387
29. Sahin M, Ozer S, Vatansev C, Aksoy M, Vatansev H, Aksoy F, Dilisiz A, Yilmaz O, Karadeniz M, Akta M. The impact of oral feeding on the severity of acute pancreatitis. A m J Surg 1999; 178: 394-398
30. Hamvas J, Schwab R, Pap A. Jejunal feeding in necrotising acute pancreatitis- a retrospective study. A cta Chir Hung 1999; 38: 177-185
31. Tesinsky P. Nutritional care of pancreatitis and its complication. Curr Opin Clin Nutr M tab Care 2002; 5: 395-399
32. Duerksen DR, Becton S, Yaffe C, Parry DM. Does jejunal feeding with a polymeric immune-enhancing for increasing pancreatic exocrine output as compared with TPN: case report. Nutrition 2000; 16: 47-49

Edited by Zhang JZ and Wang XL