What is the best way to feed patients with pancreatitis?
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Purpose of review
Patients with acute pancreatitis have traditionally been treated with 'bowel rest'. Recent data, however, suggest that this approach may be associated with increased morbidity and mortality. This paper reviews evolving concepts in the nutritional management of patients with acute pancreatitis.

Recent findings
Both experimental and clinical data strongly support the concept that enteral nutrition started within 24 h of admission to hospital reduces complications, length of hospital stay and mortality in patients with acute pancreatitis. Clinical trials suggest that both gastric and jejunal tube feeding is well tolerated in patients with severe pancreatitis. Although there is limited data for the optimal type of enteral feed, a semielemental formula with omega-3 fatty acids is recommended. On the basis of current evidence, immune modulating formulas with added arginine and probiotics are not recommended.

Summary
Nutritional support should be viewed as an active therapeutic intervention that improves the outcome of patients with acute pancreatitis. Enteral nutrition should begin within 24 h after admission and following the initial period of volume resuscitation and control of nausea and pain. Patients with mild acute pancreatitis should be started on a low-fat oral diet. In patients with severe acute pancreatitis, enteral nutrition may be provided by the gastric or jejunal route.

Keywords
enteral nutrition, gastric feeding, jejunal feeding, nutrition support, omega-3 fatty acids, pancreatitis, parenteral nutrition

Introduction

Acute pancreatitis is one of the most common pancreatic diseases, with a reported incidence rate of between 4.9 and 80/1000 per year [1•]. Approximately 70–80% of patients have mild pancreatitis; these patients are usually treated with a short period of bowel rest (no enteral intake), intravenous hydration and analgesia [1•]. Severe acute pancreatitis (SAP) results in a hypermetabolic, hyperdynamic, systemic inflammatory response that creates a highly catabolic stress state [1•,2]. SAP is associated with significant morbidity and mortality and a prolonged hospital stay. Patients with SAP have traditionally been treated with bowel rest and parenteral nutrition [3]. Clinical studies performed in the last decade have demonstrated that the traditional approach to the management of acute pancreatitis is associated with increased morbidity, a longer duration of hospital stay with an increased risk of dying. This paper will review current concepts in the nutritional management of patients with acute pancreatitis, with an emphasis on randomized controlled clinical trials (RCTs) (Table 1 [4–14,15•,16–21,22••]) with a summary (meta-analysis) of these trials (Table 2).

Patients with pancreatitis are classified as having either mild or severe (SAP) disease. This classification is based on a constellation of signs, symptoms and computed tomography findings present both on admission and 48 h later [23–25]. This distinction is important in the management of patients with acute pancreatitis, as the course of the disease differs markedly between these two groups of patients. SAP is associated with pancreatic necrosis and infectious complications. Infected pancreatic necrosis occurs in approximately 25% of patients with SAP after 1 week and in 75% after 3 weeks [26,27]. The mortality rate is 5–10 times higher if the necrotic tissue becomes infected. The risk of pancreatic infection is related to the extent of pancreatic necrosis and therefore the severity of the disease. The finding that the microorganisms causing pancreatic infection are common enteric pathogens implies that bacterial translocation from the intestinal tract to pancreas may play a role in the pathogenesis of pancreatitis-induced sepsis. Although prophylactic antibiotic therapy showed some promise and has been widely used, recent studies and meta-analyses [28,29•,30] have demonstrated that this approach does not reduce the risk of infections. Similarly,

Table 1 Current concepts in the nutritional management of patients with acute pancreatitis

Reference	n	Inclusion criteria	Time to feed (h)	Type of feed	Enteral route
Enteral vs. parenteral nutrition (n = 9)					
Kalfarentzos et al. [4]	38	APACHE ≥ 8 and/or Glasgow ≥ 3	<48	Semielemental	NJ
McClave et al. [5]	32	Ranson ≥ 3	<48	Semielemental	NJ
Windsor et al. [6]	13	Glasgow ≥ 3	<24	Polymeric	NJ
	21	Glasgow < 3	<24	Polymeric	p.o.
Abou-Assi et al. [7]	53	Ranson ≥ 3 (unable to take p.o. >48 h)	>48	Elemental	NJ
Olah et al. [8]	17	Glasgow ≥ 3, CRP > 150	<24	Elemental	NJ
	72	Glasgow ≥ 3, CRP > 150	<24	Elemental	NJ
Gupta et al. [9]	17	APACHE > 5	<48	Polymeric	NJ
Louie et al. [10]	28	Ranson ≥ 3	>96	Semielemental	NJ
Eckerwall et al. [11]	22	APACHE ≥ 8 and/or CRP > 150	<24	Polymeric	NG
	26	APACHE < 8 and CRP < 150	<24	Polymeric	NG
Petrov et al. [12]	69	APACHE ≥ 8 and/or CRP > 150	<24	Semielemental	NJ
Gastric/p.o. vs. jejunal/fasting (n = 4)					
Eatock et al. [13]	49	Glasgow ≥ 3, APACHE ≥ 8 and/or CRP > 150	24−72	Semielemental	NG vs. NJ
Kumar et al. [14]	30	APACHE ≥ 8	>48	Semielemental	NG vs. NJ
Pandey et al. [16]	28	Clinical and laboratory diagnoses of AP	>48	Polymeric	p.o. vs. NJ
Eckerwall et al. [15•]	59	APACHE < 8 and CRP < 150	<48	Polymeric	p.o. vs. fasting
Standard vs. immune-modulating formula (n = 3)					
Hallay et al. [17][a]	16	Clinical and laboratory diagnoses of AP	<24	Stressen[b] vs. control	NJ
Lasztity et al. [19]	28	Clinical and laboratory diagnoses of AP	<24	Polymeric + 3.3 g omega-3 FFA	NJ
Pearce et al. [18]	31	APACHE > 7	<72	I-Complete[b] vs. control	NJ
Prebiotic/probiotic (n = 3)					
Olah et al. [21]	45	Clinical and laboratory diagnoses of AP	<48	Semielemental + Lactobacillus plantarum	NJ
Karakan et al. [20]	30	Clinical and laboratory diagnoses of AP	>48	Semielemental + prebiotic fiber	NJ
Besselink et al. [22••]	296	Glasgow ≥ 3, APACHE ≥ 8 and/or CRP > 150	<72	Semielemental + probiotic[c]	NJ

AP, acute pancreatitis; FFA, free fatty acid; NG, nasogastric; p.o., oral(ly); NJ, nasojejunal.
[a] Unclear if randomized.
[b] Added glutamine, arginine and omega-3 FFA.
[c] Multispecies probiotic.

proinflammatory cytokine inhibitors have failed to make a significant impact on the outcome [31].

Until recently, nutritional support in critically ill patients, and those with pancreatitis in particular, was considered an afterthought and merely a means of providing protein and calories. However, recent and emerging data suggest that the route, timing, quantity and composition of the nutrients have important disease-modifying properties. Furthermore, the traditional approach to the nutritional management of both mild and SAP may be severely flawed, with recent evidence suggesting that nutritional support may be the most important intervention in the management of patients with acute pancreatitis.

Enteral vs. parenteral nutrition

Strict starvation and parenteral nutrition have been considered as a fundamental intervention in the management of acute pancreatitis. Enteral nutrition and oral food intake were considered contraindicated and only introduced once the patient was pain free and passing flatus. Recent data, however, suggest that these assumptions are incorrect. Furthermore, it is now generally appreciated that parenteral nutrition (as compared with enteral nutrition) is associated with significant complications, related to the parenteral nutrition itself as well as the gastrointestinal (GUT) starvation that is inevitably associated with parenteral nutrition [32]. These effects are particularly important in the patient with pancreatitis. Many

Table 2 Overall results of meta-analysis (odds ratio, 95% confidence interval; fixed effects model)

	Mortality	Infections	MOF	Hospital LOS
Enteral vs. parenteral nutrition	0.5 (0.26−0.97)[*]	0.33 (0.2−0.54)[¶]	0.32 (0.18−0.56)[¶]	−5.5 (−7.9 to −2.61)[¶]
Standard vs. IMD	0.36 (0.09−1.49)	0.34 (0.08−1.44)	0.24 (0.03−1.75)	−3 (−7.7 to 1.7)
Gastric/p.o. vs. jejunal	0.69 (0.26−1.88)	−	−	4.26 (−3.82 to 12.34)
Probiotic	1.85 (0.95−3.61)[#]	0.91 (0.58−1.44)	1.39 (0.69−2.79)	−3.15 (−6.4 to 0.16)

IMD, immunomodulating diet; LOS, length of stay; MOF, multiorgan failure; p.o., oral(ly).
[*] $P = 0.04$.
[#] $P = 0.07$.
[¶] $P < 0.0001$.

studies report that parenteral nutrition impairs humoral and cell-mediated immunity, increases the vigor of the proinflammatory response, increases bacterial translocation and increases infection rates in experimental models and critically ill patients [32]. Clinical and experimental studies have demonstrated higher levels of both local and systemic proinflammatory mediators with parenteral nutrition as compared with enteral nutrition.

Lack of enteral feeding results in gastrointestinal mucosal atrophy, bacterial overgrowth, increased intestinal permeability and translocation of bacteria, bacterial products or both into the circulation [33–35]. Total parenteral nutrition (TPN) may therefore promote bacterial translocation in patients with pancreatitis. Enteral nutrition prevents atrophy and maintains the integrity of the gut mucosa and gastrointestinal-associated lymphoid tissue (GALT). Enteral nutrition maintains the commensal bacterial flora, which together with the effect on the gut mucosa may limit bacterial translocation and infection. In an experimental pancreatitis model, enteral nutrition as compared with parenteral nutrition reduced systemic plasma endotoxin, bacterial translocation to the portal and systemic blood and bacterial colony counts in the mesenteric lymph nodes, pancreas and lung [36].
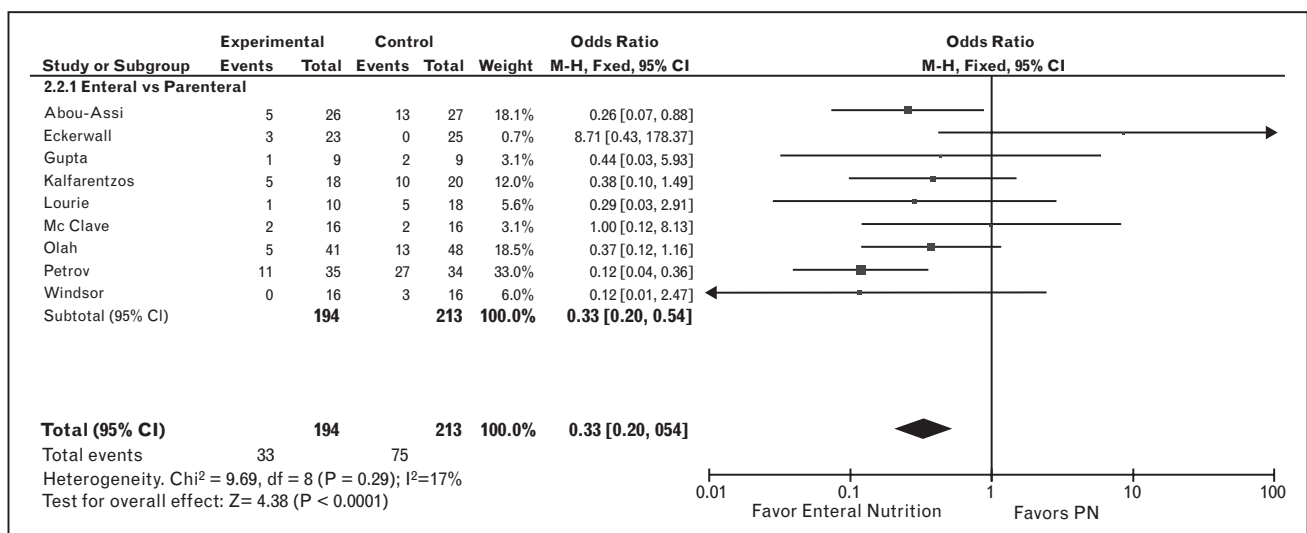
The benefits of enteral nutrition (as opposed to parenteral nutrition) may be particularly important in patients with pancreatitis, a proinflammatory disease complicated by secondary infections. Parenteral nutrition would be expected to worsen the degree of inflammation as well as the risk of pancreatic infection. In an experimental study [37] comparing early oral feeding with parenteral nutrition in a murine model of acute pancreatitis, the histopathological changes in pancreatic tissue were less pronounced in the group of rats that were fed orally.

Oral feeding, enteral nutrition or both have been considered to be harmful in acute pancreatitis as it is thought to stimulate exocrine pancreatic secretion and consequently the autodigestive process. In patients with acute pancreatitis, it was postulated that the premature activation of proteolytic enzymes within acinar cells following enteral feeding would lead to autodigestion and therefore exacerbate the tissue injury. However, in both experimental models and patients with acute pancreatitis, it has been demonstrated that the secretion of pancreatic enzymes is markedly reduced [38,39], making enteral nutrition feasible.

Nine RCTs have been reported to date, which have compared parenteral nutrition with enteral nutrition in patients with acute pancreatitis [4–12]. Although the inclusion criteria (and severity of illness), time to feeding and formula used differ somewhat between studies (Table 1), a summary (meta-analysis) of these studies (see Table 2) demonstrates a significant reduction in mortality, infectious complications, multiorgan failure (MOF) and hospital length of stay (LOS) with enteral nutrition. The study by Eckerwall et al. [11] was the only RCT in which enteral nutrition did not appear to improve patient outcome as compared with parenteral nutrition. It should, however, be noted that a serious imbalance in randomization appears to have occurred in this study. In the parenteral nutrition group, 32% of patients were defined as having SAP as compared with 61% in the

Figure 1 Effect of route of nutritional support (enteral vs. parenteral nutrition) on the acquisition of new infections

Study or Subgroup	Experimental Events	Total	Control Events	Total	Weight	Odds Ratio M-H, Fixed, 95% CI
2.2.1 Enteral vs Parenteral						
Abou-Assi	5	26	13	27	18.1%	0.26 [0.07, 0.88]
Eckerwall	3	23	0	25	0.7%	8.71 [0.43, 178.37]
Gupta	1	9	2	9	3.1%	0.44 [0.03, 5.93]
Kalfarentzos	5	18	10	20	12.0%	0.38 [0.10, 1.49]
Lourie	1	10	5	18	5.6%	0.29 [0.03, 2.91]
Mc Clave	2	16	2	16	3.1%	1.00 [0.12, 8.13]
Olah	5	41	13	48	18.5%	0.37 [0.12, 1.16]
Petrov	11	35	27	34	33.0%	0.12 [0.04, 0.36]
Windsor	0	16	3	16	6.0%	0.12 [0.01, 2.47]
Subtotal (95% CI)		194		213	100.0%	0.33 [0.20, 0.54]
Total (95% CI)		**194**		**213**	**100.0%**	**0.33 [0.20, 054]**
Total events	33		75			

Heterogeneity. Chi² = 9.69, df = 8 (P = 0.29); I²=17%
Test for overall effect: Z= 4.38 (P < 0.0001)

Weight is the relative contribution of each study to the overall effect (odds ratio and 95% confidence interval) on a log scale assuming a fixed effects model.

enterally fed patients. This imbalance is further evidenced by the higher IL-8 levels (22 vs. 80 pg/ml) in the enterally fed patients. This imbalance likely accounts for the findings of this study.

The risk of infectious complications is significantly ($P < 0.0001$) reduced with enteral nutrition (Fig. 1). This finding may largely explain the benefit of enteral nutrition in patients with acute pancreatitis. Infection of the pancreatic tissue is a dreaded complication, which increases the risk of death. As discussed above, multiple mechanisms may explain the reduction of infections with enteral nutrition as apposed to parenteral nutrition. Eckerwall et al. [11] assessed intestinal permeability by excretion of orally administered polyethylene glycol (PEG) in the urine of patients with acute pancreatitis. In this study, there was no significant difference in PEG excretion, change in antiendotoxin antibodies, C-reactive protein (CRP) and IL-6 between the two groups of patients. However, as discussed above, there were serious randomization differences in this study that precludes definite conclusions being made.

In addition to reducing the risk of infection in acute pancreatitis, enteral nutrition may reduce the degree of inflammation and the systemic inflammatory response as compared with parenteral nutrition. In the study by Windsor et al. [6], CRP, a marker of systemic inflammation, fell significantly in the enterally fed patients, whereas it remained unchanged in the parenteral nutrition group. Similarly, Louie et al. [10] demonstrated a more rapid decline in CRP levels in enterally as opposed to parenterally fed patients. Not all studies, however, have reproduced these findings [9,12]. In the Windsor et al. [6] study, serum antiendotoxin antibodies (Endocab IgM) increased in the parenteral nutrition group, whereas the levels remained unchanged in the enterally fed patients.

As enteral nutrition appears to modulate the inflammatory response the timing of this intervention may be important. In experimental models of acute pancreatitis, bacterial colonization and infection occurs within hours of the induction of pancreatitis [40]. Although subgroup analysis is fraught with many difficulties, we noted a further reduction in the risk of death [odds ratio (OR) 0.32 (0.13–0.76), $P = 0.01$, $I^2 = 9\%$] when excluding those studies that initiated feeding after 48 h.

Both experimental and clinical data, therefore, strongly support the concept that enteral nutrition reduces the complications and mortality in acute pancreatitis when compared with parenteral nutrition. It is likely the enteral nutrition positively influences the disease process and should be initiated as early as possible (within 24 h of admission). Parenteral nutrition should be avoided at all costs, and is likely to increase mortality [32].

The optimal enteral route and formulation is reviewed below.

Nasogastric vs. nasojejunal feeding

Enteral nutrition is preferred over parenteral nutrition for improving the outcome of patients with SAP and has largely replaced parenteral nutrition. It has, however, been assumed that patients should be fed with a nasojejunal tube, which is placed beyond the ligament of Treitz to prevent (limit) stimulation of the exocrine pancreas. However, as discussed above, experimental and clinical studies [38,39] have shown that in acute pancreatitis exocrine secretion in response to cholecystokinin and other secretagogues is markedly suppressed. Furthermore, delivery of enteral feed distal to the ligament of Trietz does not preclude duodenal exposure to nutrients, as a degree of reflux is inevitable.

The placement of a nasojejunal tube has historically required endoscopy or radiographic screening, with the inherent risks of intrahospital transfer, costs and the delayed introduction of feeding. Consequently, the role of nasogastric feeding has been explored. Eatock et al. [13] randomized 49 patients with SAP to receive a semi-elemental diet via a nasogastric or nasojejunal tube. In this study, there was no difference between groups in clinical outcome as well as changes in the CRP, pain as measured by a visual analogue score and analgesic requirements (Tables 1 and 2). Kumar et al. [14] demonstrated similar findings in a study of 30 patients with SAP. Despite limited data (two studies), nasogastric feeding appears well tolerated and offers similar benefits to nasojejunal feeding. Recently, a novel method (electromagnetic guidance system) of nasojejunal placement at the bedside has been introduced (Corpak, VIASYS Medical Systems, Wheeling, Illinois, USA; Fig. 2) [41]. In our hands, we have a 95% success rate at postpyloric placement with an average procedure time (nose to distal duodenum) of 5 min (unpublished data).

Patients with 'mild' pancreatitis are usually managed with fluids, analgesics and 'pancreatic rest'. All oral feeds are usually stopped until abdominal pain subsides, ileus improves, the patient passes flatus and he/she no longer requires narcotic analgesics. Patients are then usually refed small meals that are rich in carbohydrates and poor in proteins and fat. The caloric intake is then gradually increased over a period of 3–6 weeks. Eckerwall et al. [15•] randomized 59 patients with mild acute pancreatitis to immediate oral feeding or fasting until resolution of pain and resumption of bowel activity. Patients in the oral feeding group were immediately allowed to drink and eat freely as tolerated. There was no significant difference between the groups in clinical symptoms or biochemical markers of systemic infection. The patients in the oral

Figure 2 Corpak electromagnetic guidance system for nasojejunal tube placement
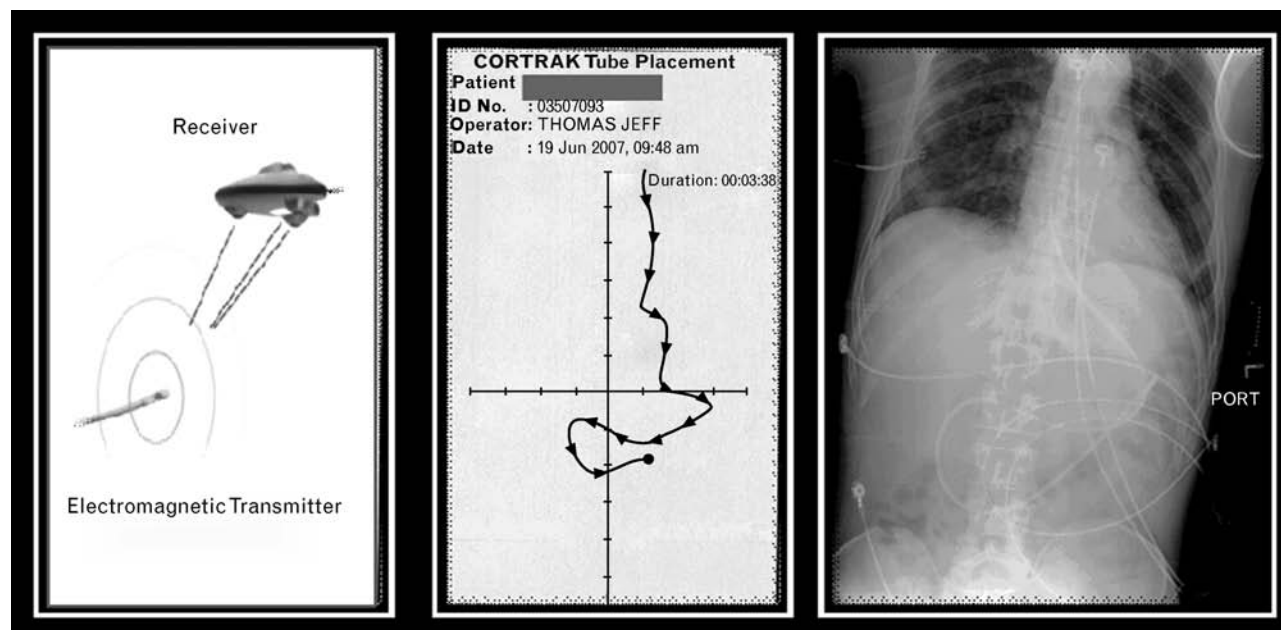
feeding group began solid foods earlier (3 vs. 5 days) and had a shorter length of hospital stay (4 vs. 6 days). Pandey *et al.* [16] randomized patients with pancreatitis severe enough to stop oral feeding for 48 h to receive either oral or jejunal tube feeds. Four patients (26%) in the oral group and none in the enteral tube group had a relapse of pain. Pain relapse increased length of hospital stay. The difference between these two studies in the clinical course of the patients who were fed orally may be related to the timing of the initiation of oral feeding.

Immunomodulating diets

Immunomodulating diets (IMDs) are balanced nutritional formulations (i.e. contain protein, carbohydrate, lipids, minerals, trace elements and vitamins) that are supplemented with increased quantities of nutrients that have been demonstrated to improve immune cell function and modulate inflammation. Immunonutrients that have been added to IMDs include arginine, glutamine, omega-3 polyunsaturated long-chain fatty acids and antioxidants (such as ascorbic acid and selenium). The use of IMDs in critically ill patients is a controversial and evolving topic. As IMDs may modulate the systemic inflammatory response in patients with pancreatitis it has been suggested that these formulations may be beneficial in this disease.

In a quasi-randomized study, Hallay *et al.* [17] studied the changes in immunological and nutritional parameters and outcome in patients with acute pancreatitis who received an IMD. Nine patients received stressen multifiber

(added arginine, glutamine and fish oil), whereas seven patients received a standard polymeric diet. In this study, serum IgG, IgM, retinol-binding protein and prealbumin increased (recovered) more rapidly and the CD4 : CD8 ratio was maintained in those patients receiving the immunomodulating diet. Pearce *et al.* [18] randomized 31 patients with SAP to an IMD with added arginine, glutamine and omega-3 fatty acids (I-Complete, Fresenius, Homberg, Germany) or an isonitrogenous, isocaloric control formula. The primary endpoint of this study was a reduction of the CRP of 40 mg/l after 3 days of feeding. This endpoint occurred in two out of 15 (13%) patients in the IMD group and six out of 16 (38%) in the control group. The mean CRP increased in the IMD group, whereas it trended down in the control group. Similarly, the serum albumin decreased in the IMD group, whereas it increased in the control group. There was, however, no significant difference between the two groups in the Sequential Organ Failure Assessment (SOFA) score, complication rate, LOS and cytokine levels.

Lasztity *et al.* [19] randomized 28 patients with SAP to jejunal enteral feed supplemented with omega-3 fatty acids (3.3 g/day) or a control enteral feed. Supplementation with omega-3 fatty acids resulted in a significant decrease in length of hospitalization. There was, however, no difference in acute phase reactants or complications between the two groups. Although the data are limited, the results of these studies tend to mirror our findings on the use of IMD in general ICU patients; that is, the IMDs containing arginine may exacerbate the inflammatory process with no obvious clinical benefit,

whereas IMDs supplemented with fish oil (alone) decrease markers of inflammation and are associated with significantly fewer recurrent infections, a shorter LOS and a reduced mortality [42[•]].

Semielemental or polymeric formula

Both semielemental/elemental and polymeric formula have been used in patients with acute pancreatitis and both have been demonstrated to be superior to parenteral nutrition. Semielemental formulas contain small peptides and medium-chain triglycerides, whereas polymeric formulas are comprised of nonhydrolyzed proteins and long-chain triglycerides. Semielemental formula has several theoretical advantages over polymeric formula. Exocrine pancreatic function is decreased in pancreatitis and absorption of a semielemental formula, which does not require the presence of pancreatic enzymes, should be better than that of a polymeric formula [43]. Semielemental formula stimulates pancreatic secretions to a lesser degree than polymeric formula and may therefore decrease the risks of acute pain episodes after nutrition [44]. Furthermore, in animal models, a semielemental formula is more effective in maintaining the integrity of the intestinal mucosa [45] and the prevention of septic complications due to translocation of gastrointestinal bacteria [40].

One RCT has been published to date that has compared a semielemental with a polymeric enteral formulation in patients with acute pancreatitis. Tiengou et al. [46] randomized 30 patients with severe pancreatitis (Balthazar Score ≤ B) to a semielemental diet (Peptamen; Nestlé Clinical Nutrition, Noisiel, France) or an isocaloric, isonitrogenous, isovolemic formula after resolution of ileus and resumption of flatus. Although all patients had a favorable outcome, the elemental diet was associated with slightly but statistically significant less weight loss and hospital stay. There was no difference in tolerance of the formula (pain, bloating, etc.) or any other clinical parameter between the two feeds. This study, however, has a number of factors, which limit the interpretation of the results. Peripheral parenteral nutrition was used in half the patients prior to insertion of the nasojejunal tube and the duration of fasting before insertion of the naso-jejunal tube was 7.5 days in the elemental group and 8.6 days in the polymeric group.

Probiotics and fiber

It has been observed in experimental pancreatitis that anaerobic bacteria and lactobacilli are significantly reduced within 6–12 h both in the distal small bowel and in the colon. These alterations lead to significant overgrowth with potentially pathogenic microorganisms such as Escherichia coli, dramatic increases in mucosal barrier permeability and in endothelial permeability, which is associated with increased pathogenic microbial colonization, translocation, microbial growth in mesenteric lymph nodes and finally pancreatic tissue [47,48]. It is postulated that a similar mechanism occurs in patients with SAP.

Ingestion of specific fiber-fermenting lactic acid bacteria (probiotics) and fermentable fiber (prebiotics) is known to reduce intestinal colonization with potentially pathogenic Gram-negative bacteria, to reduce bacterial translocation, to reduce proinflammatory cytokine induction and upregulate immune function [49,50]. It has been postulated that pre/probiotics may reduce the rate of infection, limit the extent of tissue necrosis and improve the outcome in patients with acute pancreatitis [49]. Karakan et al. [20] randomized 30 patients with SAP who were receiving nasojejunal feeding to prebiotic fiber supplementation or control enteral feeds. Inflammatory markers normalized more rapidly in the prebiotic group that had a significantly shorter hospital stay.

Olah et al. [21] randomized patients with acute pancreatitis to nasojejunal enteral nutrition supplemented with Lactobacillus plantarum and oat fiber and a control group that received heat-inactivated L. plantarum. Pancreatic infection and length of hospital stay was significantly less in the group of patients who received the active probiotic. The Dutch Acute Pancreatitis Study was a multicenter randomized, double-blind, placebo controlled trial in which 298 patients with SAP were randomly assigned within 72 h of the onset of symptoms to receive a probiotic preparation (containing multiple species of Lactobacilli and Bifidobacterium) or placebo administered enterally twice daily for 28 days [22[••]]. There was no difference in the rate of infectious complications between groups; however, the group of patients receiving the probiotic had a significantly higher incidence of MOF and a higher mortality (16 vs. 6%, $P = 0.01$). Nine patients in the probiotic group developed nonocclusive mesenteric ischemia, whereas none of the patients in the placebo group developed this complication. The development of bowel ischemia largely explained the difference in MOF and mortality between the two groups. The cause of the increased occurrence of bowel ischemia in the probiotic group is unclear.

Conclusion

Current evidence suggests that the 'traditional' approach to the management of patients with pancreatitis is without scientific foundation and likely to increase complications, length of hospital stay and mortality. In patients with both mild and SAP, early onset enteral feeding (within 24 h of admission) helps to maintain gut function, allows improved tolerance with fewer problems with ileus, abrogates the inflammatory process, results in less infective complications and reduces mortality.

Nutritional support should be viewed as an active therapeutic intervention that improves the outcome of patients with acute pancreatitis. Enteral nutrition should begin within 24 h after admission and following the initial period of volume resuscitation and control of nausea and pain. Patients with mild acute pancreatitis should be started on a low-fat oral diet. In patients with SAP, enteral nutrition may be provided by the gastric or jejunal route. We prefer the nasojejunal route, as we have a high success rate with bedside placement and this approach is well tolerated by our patients. In those institutions that rely on endoscopic or fluoroscopic placement of nasojejunal tubes, we recommend a trial of gastric feeding prior to placement of a nasojejunal tube. Although the data are limited, we prefer a semielemental diet with medium-chain triglycerides and omega-3 fatty acids as apposed to a polymeric formula. At this time, IMDs with added arginine and probiotics should not be given to patients with acute pancreatitis. The role of additional omega-3 fatty acid supplementation remains to be determined. Parenteral nutrition should be avoided in patients with acute pancreatitis.

Acknowledgement

References and recommended reading

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- of special interest
- of outstanding interest
Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 175).

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