

Feeding the angry pancreas?

T Shah, G Suri, M Ghatage, V Gaikwad, R Kulkarni

Citation

T Shah, G Suri, M Ghatage, V Gaikwad, R Kulkarni. *Feeding the angry pancreas?*. The Internet Journal of Surgery. 2008 Volume 20 Number 1.

Abstract

AIMS & OBJECTIVES: To determine whether patients of acute pancreatitis benefit from early enteral feeding. **MATERIALS & METHODS:** A total of 30 patients diagnosed with acute pancreatitis were studied in the hospital, 16 patients had mild attacks and 14 had moderate attacks of pancreatitis. Both groups received early enteral feeding and were monitored for SIRS, intra-abdominal sepsis, multi-organ failure (MOF), stay in ICU and general well-being & cost. **DISCUSSION & RESULTS:** Classically, patients with severe acute pancreatitis were treated with gut rest with or without parenteral nutrition because enteral feeding was thought to stimulate the pancreas and worsen pancreatic injury. Recent data suggest that parenteral nutrition does not hasten pancreatic recovery and that enteral feeding is well tolerated. Potential benefits of enteral feeding include decreased permeability, prevention of bacterial translocation and reduction in secondary pancreatic infection. Enteral feeding is also less expensive and is associated with fewer cases of central line catheter-related sepsis. **CONCLUSION:** This study suggests that enteral feeding is both safe and may promote more rapid resolution of toxicity in patients with mild acute pancreatitis.

INTRODUCTION

The term “feeding the angry pancreas” here means feeding in cases of “acute” pancreatitis. Pancreatitis is an inflammatory process in which pancreatic enzymes autodigest the gland. The gland can sometimes heal without any impairment of function or any morphologic changes. This process is known as acute pancreatitis. It can recur intermittently, contributing to the functional and morphologic loss of the gland. Recurrent attacks are referred to as chronic pancreatitis.

Conventional management of acute pancreatitis was based on gut rest, with or without provision of total parenteral nutrition, i.e. keeping the patient “nil by mouth” (NBM) (1,2). This was based on the belief that enteral feeding will further stimulate the pancreas and exacerbate the inflammatory response by releasing more enzymes. So it was believed that feeding will lead to autodigestion of the pancreas.

But this belief has been replaced by new concepts now:

It has been well documented that in the early course of acute pancreatitis, enzyme synthesis is virtually abolished (1,3,14). Hence there is no danger in giving enteral feeding. Secondly, the gut mucosa plays a major role in the outcome which was not well understood before. New concepts support maintaining the normal gut mucosal barrier function and this

can be best managed by giving enteral nutrition rather than depriving the gut of its essential nutrients as happens in parenteral nutrition. It has also been seen in the study that mild attacks can resolve rapidly and spontaneously. A recent report has suggested that enteral nutrition is safe and may promote more rapid resolution of toxicity in patients with mild acute pancreatitis.

MATERIALS AND METHODS

Between 2004 and 2006, a clinical case series study of benefits of enteral nutrition in acute pancreatitis was undertaken in D.Y. Patil Hospital, Kolhapur. Patients with a serum amylase of greater than 400 IU and clinical evidence of acute pancreatitis were enrolled. Patients with evidence of severe pancreatitis, without clinical evidence of acute pancreatitis, patients with known chronic pancreatitis, and those presenting more than 48 hours after admission were excluded from the study.

USG of the abdomen was done in all patients and CT scan was done in few (n=9) patients where USG was inconclusive or there was suspicion of severe pancreatitis. Routine investigations, serum amylase and LFT were done in all patients.

The primary endpoint of the study was the incidence of systemic inflammatory response syndrome, with sepsis,

Feeding the angry pancreas?

organ failure, hospital stay, and mortality as secondary endpoints.

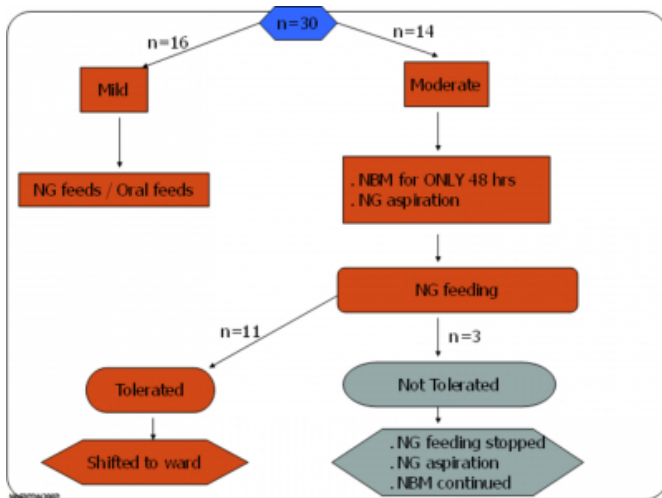
RESULTS

From May 2004 to May 2006, 30 consecutive patients with pancreatitis fulfilled the enrolment criteria. Based on RANSON'S criteria and APACHE II scoring, patients (n=30) were divided into a mild (n=16) and a moderate (n=14) category. Out of these 30 patients, 24 were males and 6 were females, with an average age of 50 years (range: 28-60).

The patients with mild attacks of pancreatitis (n=16) were started on enteral feeding on the day of admission itself, whereas patients with moderate attacks (n=14) were kept "nil by mouth" (NBM) for the first 48 hours and then given a trial of enteral feeding. Those who did not tolerate enteral feeding were resumed back to NBM status and nasogastric aspiration and waited for further 48 hours to allow rest to the gut. (Figure 1)

Figure 1

Figure 1: NG: naso-gastric, NBM: nil by mouth



All cases with mild attacks tolerated enteral feeding, but out of the moderate cases, 3 did not tolerate enteral feeding and they were resumed back to NBM for further 48 hours. (Figure 2)

Figure 2

Figure 2

	NG Feeding Tolerated	Not Tolerated	Percentage Tolerated
Mild n=16	16	0	100 %
Moderate n=14	11	3	78.57 %

All the cases were studied for the duration of ICU stay and complications, e.g. sepsis, abscess, necrosis, pseudo-pancreatic cyst, ileus and mortality. (Figure 3 & 4)

Figure 3

Figure 3

	Average ICU stay
Mild n=16	1 Day
Moderate n=14	3 Days

Figure 4

Figure 4

	Sepsis	Abscess	Necrosis	Pseudo-pancreatic cyst	Death	Ileus
Mild n=16						
Moderate n=14				1		2

DISCUSSION

Most morbidity and deaths from acute pancreatitis are consequences of the immuno-inflammatory response to pancreatic necrosis or infection. This response manifests as a spectrum of diseases, with clinical progression from systemic inflammatory response syndrome (SIRS) to sepsis, multiple organ failure (MOF), and death.^(1,3) The gastrointestinal tract is increasingly seen as a potential source of fuel for such an immuno-inflammatory response in

critically ill patients because of experimental data suggesting that the endogenous cytokines responsible for this response are stimulated by endotoxin and other bacterial products absorbed by a metabolically deprived gut. This concept of mucosal injury has also been invoked as a cause of the inflammatory response in experimental acute pancreatitis. In parallel with this concept, several studies have shown a reduction in septic morbidity following early enteral feeding in other immuno-inflammatory conditions associated with trauma, thermal injury, and major surgery. A proposed mechanism for these improved clinical outcomes suggests that feeding the gut maintains normal gut barrier function against the translocation of luminal bacteria and toxins. Moreover, in contrast to parenteral nutrition, enteral feeding seems to modulate the acute phase response and preserves visceral protein metabolism, suggesting down-regulation of the splanchnic cytokine response₍₁₀₎.

In our case series study, we got 100% tolerance of enteral feeding in cases of mild attacks of pancreatitis and 78.57% tolerance in cases of moderate attacks. These results almost matched with other studies and confirmed the role and benefits of enteral feeding in cases of mild & moderate cases of acute pancreatitis. Since these patients were started on early enteral feeding, their requirement of parenteral fluids was reduced. It was also not necessary to prescribe antibiotics for a prolonged period. Together this was a major determinant in reducing the hospital stay and thereby the cost of stay and medication to the patient. The average ICU stay in our study for patients with mild pancreatitis was 1 day and for moderate pancreatitis was 3 days. Two patients in the group of moderate pancreatitis developed prolonged ileus and had to be kept NBM for a longer period. We did not give parenteral nutrition to these patients but again attempted enteral nutrition once peristalsis was established. All the patients were followed on OPD basis regularly. Only one patient developed a pseudopancreatic cyst which resolved spontaneously.

CONCLUSION

From our study, we concluded that enteral feeding in cases of mild & moderate pancreatitis is tolerated easily. As compared to the old concept of “no enteral feeding”, there were no significant complications with enteral feeding. Additional economical benefits were also seen like short duration of ICU stay and benefits of using nasogastric tube feeding which was cheaper, simpler and easy to use as compared to parenteral nutrition.

References

1. Compared with parenteral nutrition, enteral feeding attenuates the acute phase response and improves disease severity in acute pancreatitis. Windsor ACJ, Kanwar S. *Gut* 1998; 42: 431-435.
2. Randomized controlled trial of the effect of early enteral nutrition on markers of the inflammatory response in predicted severe acute pancreatitis. Powell JJ et al. *Br J Surg* 2000; 87: 1375-1381.
3. Comparison of the safety of early enteral vs. parenteral nutrition in mild acute pancreatitis. [JPEN J Parenter Enteral Nutr 1997] PMID: 9002079
4. Early enteral feeding versus “nil by mouth” after gastrointestinal surgery: systematic review and meta-analysis of controlled trials. *BMJ* 2001; 323(7316): 773.
5. Nasogastric feeding in severe acute pancreatitis may be practical and safe. *Int J Pancreatol* 2000; 28: 23-9.
6. Early enteral nutrition in severe acute pancreatitis: a prospective randomized controlled trial comparing nasojejunal and nasogastric routes. *J Clin Gastroenterol* 2006; 40: 431-4.
7. Enteral nutrition is superior to parenteral nutrition in severe acute pancreatitis: results of a randomized prospective trial. Kalfarentzos F. *Br J Surg* 1997; 84: 1665-9.
8. A randomized study of early nasogastric versus nasojejunal feeding in severe acute pancreatitis. *Am J Gastroenterol* 2005; 100: 432-9.
9. Evidence-Based Treatment of Acute Pancreatitis - A Look at Established Paradigms. Stefan Heinrich, Markus Schäfer. *Ann Surg* 2006; 243: 154-168.
10. A randomised clinical trial to assess the effect of total enteral and total parenteral nutritional support on metabolic, inflammatory and oxidative markers in patients with predicted severe acute pancreatitis (APACHE II > or =6). *Pancreatology* 2003; 3: 406-13. Epub 2003 Sep 24.
11. Enteral versus parenteral nutrition for acute pancreatitis. Al-Omran M, Groof A. *Cochrane Database Syst Rev* 2003; (1): CD002837.
12. Recent Advances in Surgery, Vol. 27,103-111.
13. Recent Advances in Surgery, Vol. 29,149-160.
14. Nutrition support in acute pancreatitis: a systematic review of the literature. *JPEN J Parenter Enteral Nutr* 2006; 30: 143-56.

Author Information

Tapan A. Shah

Resident in Surgery

Gurmeet Singh Suri

Resident in Surgery

M.N. Ghatage

Associate Professor in Surgery

Vaishali Gaikwad

Associate Professor in Surgery

R.M. Kulkarni

Professor & HOD Surgery