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Original Research Article

Early Enteral Nutrition versus Delayed Feeding in Acute Pancreatitis: A Prospective Comparative Study

Dhruv Samirkumar Dave[1], Raj Tusharkumar Khanna[2], Gautamkumar Bhikhalal Suthar[3]

[1]Intern, GMERS Medical College, Vadnagar, Gujarat, India
[2]MBBS, GMERS Medical College, Dharpur, Patan, Gujarat, India
[3]MS (Surgery), Department of Surgery, Shri Shankaracharya Institute of Medical Sciences, Bhilai, Chhattisgarh, India

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Corresponding author: Dr. Gautamkumar Bhikhalal Suthar
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Abstract

Background: The optimal timing for initiating enteral nutrition in acute pancreatitis remains a subject of ongoing clinical debate. While traditional management advocated prolonged fasting to achieve pancreatic rest, emerging evidence suggests that early enteral feeding may confer significant clinical benefits. This study aimed to compare clinical outcomes between early enteral nutrition and delayed feeding strategies in patients hospitalized with acute pancreatitis.

Methods: This prospective comparative study was conducted at a tertiary care hospital. A total of 174 consecutive patients diagnosed with acute pancreatitis were allocated into two groups: the early enteral nutrition group (EEN, n = 88), receiving oral or nasojejunal feeding within 24 hours of admission, and the delayed feeding group (DF, n = 86), receiving nutritional support after 72 hours or upon complete resolution of abdominal pain. Primary outcomes included length of hospital stay, organ failure incidence, and infectious complications. Secondary outcomes included pain resolution time, inflammatory marker trajectories, and mortality.

Results: Patients in the EEN group demonstrated significantly shorter hospital stays (7.3 ± 2.9 days vs. 10.6 ± 4.1 days, $p < 0.001$), lower rates of infectious complications (9.1% vs. 22.1%, $p = 0.017$), and reduced incidence of organ failure (6.8% vs. 16.3%, $p = 0.048$). Pain resolution occurred earlier in the EEN group (3.1 ± 1.4 days vs. 4.7 ± 2.1 days, $p < 0.001$). C-reactive protein levels at day 5 were significantly lower in the EEN group (48.7 ± 31.2 mg/L vs. 79.4 ± 42.6 mg/L, $p < 0.001$). Mortality rates did not differ significantly between groups (2.3% vs. 4.7%, $p = 0.440$). Feeding intolerance occurred in 11.4% of EEN patients but was manageable in all cases.

Conclusion: Early enteral nutrition within 24 hours of admission in acute pancreatitis is associated with significantly reduced hospital stay, fewer infectious complications, lower organ failure rates, and accelerated clinical recovery compared to delay feeding. These findings support the adoption of early feeding protocols as standard practice in acute pancreatitis management.

Keywords: Acute Pancreatitis; Early Enteral Nutrition; Delayed Feeding; Clinical Outcomes; Infectious Complications; Organ Failure.

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Introduction

Acute pancreatitis represents one of the most common gastrointestinal conditions requiring hospitalization, with a global annual incidence of approximately 34 per 100,000 person-years and steadily increasing prevalence across both developed and developing nations [1]. While the majority of cases follow a mild, self-limiting course, approximately 15–20% of patients develop moderately severe or severe disease characterized by persistent organ failure and local complications, with associated mortality rates reaching 20–40% in severe forms [2]. Nutritional management constitutes a cornerstone of acute pancreatitis care. Historically, the principle of "pancreatic rest" dominated clinical practice, whereby patients were maintained nil per os for extended periods to minimize pancreatic secretory stimulation and theoretically reduce autodigestive injury [3]. This traditional approach was based on the physiological assumption that enteral nutrient delivery would exacerbate pancreatic inflammation through cholecystokinin-mediated stimulation of exocrine

secretion [4]. However, this paradigm has been increasingly challenged over the past two decades by accumulating evidence demonstrating the detrimental consequences of prolonged fasting, including intestinal mucosal atrophy, increased bacterial translocation, heightened systemic inflammatory response, and nutritional deterioration [5].

The concept of early enteral nutrition has gained substantial traction following seminal randomized controlled trials demonstrating its safety and efficacy. McClave and colleagues provided foundational evidence that nasojejunal feeding in severe acute pancreatitis was feasible and associated with fewer infectious complications compared to total parenteral nutrition [6]. Subsequent meta-analyses by Petrov and colleagues confirmed that enteral nutrition significantly reduced infection rates, organ failure, surgical intervention requirements, and mortality compared to parenteral nutrition [7]. More recently, the landmark PYTHON trial demonstrated that early nasoenteric tube feeding within 24 hours did not show superiority over an oral diet initiated at 72 hours in patients with predicted severe acute pancreatitis, generating renewed controversy regarding the optimal timing of nutritional initiation [8].

Current international guidelines, including those from the American Gastroenterological Association, recommend early oral feeding as tolerated in mild acute pancreatitis and early enteral nutrition in severe disease [9]. The International Association of Pancreatology and American Pancreatic Association similarly advocate for enteral nutrition initiation within 24–48 hours [10]. However, considerable heterogeneity persists in clinical practice, with many institutions continuing to employ prolonged fasting strategies, particularly in resource-limited settings [11]. Furthermore, the optimal timing threshold for initiating feeding, the appropriate route of delivery, and the impact of early nutrition across the entire severity spectrum of acute pancreatitis remain areas of active investigation [12].

A critical knowledge gap exists regarding the comparative effectiveness of early versus delayed enteral nutrition in real-world clinical settings that include patients across all severity categories. Many prior studies focused exclusively on severe or predicted severe acute pancreatitis, potentially limiting the generalizability of their findings to the broader patient population encountered in routine clinical practice [13]. Additionally, comprehensive evaluation of inflammatory biomarker trajectories, nutritional parameters, and detailed complication profiles in response to different feeding timelines remains insufficient.

The aim of this study was to prospectively compare clinical outcomes, inflammatory responses, and complication rates between patients with acute pancreatitis receiving early enteral nutrition within 24 hours of admission versus those receiving delayed feeding after 72 hours, across the full spectrum of disease severity.

Materials and Methods

Study Design and Setting: This prospective, non-randomized comparative study was conducted at the Department of General Surgery.

Study Population: Consecutive adult patients (age ≥18 years) admitted with a confirmed diagnosis of acute pancreatitis were screened for eligibility. The diagnosis of acute pancreatitis required the fulfillment of at least two of the following three criteria according to the revised Atlanta classification: (1) characteristic abdominal pain consistent with acute pancreatitis; (2) serum lipase or amylase elevation exceeding three times the upper limit of normal; and (3) characteristic findings on contrast-enhanced computed tomography, magnetic resonance imaging, or transabdominal ultrasonography.

Exclusion criteria included: (1) chronic pancreatitis with acute exacerbation; (2) pancreatic malignancy; (3) pregnancy; (4) age less than 18 years; (5) presentation more than 48 hours after symptom onset; (6) history of gastric or pancreatic surgery; (7) concurrent severe comorbidities precluding enteral feeding (e.g., bowel obstruction, paralytic ileus persisting beyond 24 hours); (8) patient refusal to participate; and (9) transfer from another institution with prior nutritional intervention.

Group Allocation and Nutritional Protocols: Patients were allocated to one of two groups based on the attending physician's clinical decision and institutional feeding protocol during the study period. During the first phase of the study (March 2021–May 2022), the institutional protocol mandated delayed feeding, constituting the delayed feeding group (DF). Following an evidence-based protocol revision in June 2022, early enteral nutrition became the standard practice, constituting the early enteral nutrition group (EEN).

Early Enteral Nutrition Group (EEN): Patients received nutritional support within 24 hours of hospital admission. In mild cases, oral feeding was initiated with a low-fat, soft diet and advanced as tolerated. In moderately severe and severe cases, nasojejunal feeding was initiated using a semi-elemental formula (Peptamen, Nestlé Health Science) at a rate of 20 mL/hour, advanced incrementally by 20 mL/hour every 6 hours to the target rate of 60–80 mL/hour based on tolerance.

Delayed Feeding Group (DF): Patients were maintained nil per os until complete resolution of abdominal pain, return of appetite, and normalization of bowel sounds, or until a minimum of 72 hours had elapsed since admission. Feeding was then initiated orally with clear liquids, progressing to a low-fat solid diet over 24–48 hours. Intravenous crystalloid fluids with dextrose supplementation were provided during the fasting period.

Both groups received identical standard medical management including aggressive intravenous fluid resuscitation with Ringer's lactate, analgesic therapy (acetaminophen, tramadol, or patient-controlled analgesia with morphine as needed), proton pump inhibitors, antiemetics as needed, and antibiotics only when documented infection was present.

Data Collection and Outcome Measures: Demographic, clinical, and laboratory data were prospectively collected at admission and at predefined time points (days 1, 3, 5, 7, and at discharge). Disease severity was classified according to the revised Atlanta classification into mild, moderately severe, and severe categories. Bedside Index of Severity in Acute Pancreatitis (BISAP) scores and Acute Physiology and Chronic Health Evaluation II (APACHE II) scores were calculated within the first 24 hours.

Primary outcomes included: length of hospital stay, incidence of organ failure (defined per the modified Marshall scoring system as a score ≥2 in any organ system), and infectious complications (including infected pancreatic necrosis, bacteremia, pneumonia, urinary tract infection, and catheter-related bloodstream infection).

Secondary outcomes included: time to pain resolution (defined as visual analog scale [VAS] score ≤2 without analgesic requirement), C-reactive protein (CRP) and serum procalcitonin trajectories, need for intensive care unit (ICU) admission, requirement for surgical or radiological intervention, feeding intolerance (defined as vomiting, abdominal distension, or diarrhea necessitating cessation or reduction of feeding), 30-day mortality, and hospital readmission within 30 days.

Laboratory Assessments: Serum amylase, lipase, CRP, procalcitonin, complete blood count, comprehensive metabolic panel, serum albumin, and arterial blood gas analysis were obtained at admission and on days 3, 5, and 7. Blood cultures were obtained when clinically indicated.

Statistical Analysis: Sample size estimation was performed a priori based on an anticipated reduction in hospital length of stay from 11 days to 8 days, with a standard deviation of 4 days, an alpha level of 0.05, and statistical power of 80%, yielding a minimum requirement of 78 patients per group. Anticipating a 10% attrition rate, we targeted enrollment of 86 patients per group.

Continuous variables were expressed as mean ± standard deviation or median (interquartile range) as appropriate. Normality was assessed using the Shapiro-Wilk test. Between-group comparisons were performed using the independent samples t-test or Mann-Whitney U test for continuous variables and chi-square or Fisher's exact test for categorical variables. Multivariate logistic regression was performed to identify independent predictors of infectious complications and organ failure. Kaplan-Meier analysis with log-rank testing was used to compare time to discharge between groups. Statistical significance was set at $p < 0.05$. Analyses were performed using SPSS version 27.0 (IBM Corporation, Armonk, NY, USA).

Results

Baseline Characteristics: A total of 198 patients were initially screened, of whom 24 were excluded (12 met exclusion criteria, 7 declined participation, 5 had incomplete data). The final cohort comprised 174 patients: 88 in the EEN group and 86 in the DF group. Baseline demographic and clinical characteristics were comparable between groups (Table 1).

Table 1: Baseline Demographic and Clinical Characteristics

Variable	EEN Group (n = 88)	DF Group (n = 86)	p-value
Age (years), mean ± SD	48.6 ± 14.3	50.2 ± 13.8	0.449
Male sex, n (%)	54 (61.4)	50 (58.1)	0.665
BMI (kg/m²), mean ± SD	27.8 ± 5.1	28.3 ± 4.9	0.506
Etiology, n (%)			0.812
- Gallstone	42 (47.7)	44 (51.2)	
- Alcohol	28 (31.8)	25 (29.1)	
- Hypertriglyceridemia	10 (11.4)	8 (9.3)	
- Idiopathic/Other	8 (9.1)	9 (10.5)	
Severity (Atlanta), n (%)			0.734
- Mild	52 (59.1)	48 (55.8)	
- Moderately severe	24 (27.3)	27 (31.4)	

- Severe	12 (13.6)	11 (12.8)	
APACHE II score, mean ± SD	8.4 ± 4.6	8.9 ± 4.8	0.475
BISAP score, mean ± SD	1.6 ± 0.9	1.7 ± 1.0	0.479
Admission serum lipase (U/L)	892 ± 614	847 ± 587	0.615
Admission CRP (mg/L)	86.3 ± 52.7	82.9 ± 49.8	0.659
Serum albumin (g/dL)	3.4 ± 0.5	3.3 ± 0.6	0.228
Diabetes mellitus, n (%)	16 (18.2)	18 (20.9)	0.645
Hypertension, n (%)	22 (25.0)	24 (27.9)	0.666
Time from symptom onset to admission (hours)	18.4 ± 10.6	19.7 ± 11.2	0.425

Primary and Secondary Clinical Outcomes: The median time to initiation of enteral feeding was 18.0 hours (IQR 12.0–22.0) in the EEN group and 84.0 hours (IQR 72.0–96.0) in the DF group. Clinical outcomes are presented in Table 2.

Table 2: Primary and Secondary Clinical Outcomes

Outcome	EEN Group (n = 88)	DF Group (n = 86)	p-value
Primary Outcomes			
Hospital length of stay (days)	7.3 ± 2.9	10.6 ± 4.1	< 0.001
Organ failure, n (%)	6 (6.8)	14 (16.3)	0.048
- Single organ failure	4 (4.5)	9 (10.5)	0.134
- Multi-organ failure	2 (2.3)	5 (5.8)	0.271
Infectious complications, n (%)	8 (9.1)	19 (22.1)	0.017
- Infected pancreatic necrosis	2 (2.3)	6 (7.0)	0.164
- Pneumonia	3 (3.4)	7 (8.1)	0.203
- Bacteremia	2 (2.3)	4 (4.7)	0.437
- Urinary tract infection	1 (1.1)	2 (2.3)	0.618
Secondary Outcomes			
Time to pain resolution (days)	3.1 ± 1.4	4.7 ± 2.1	< 0.001
ICU admission, n (%)	8 (9.1)	14 (16.3)	0.152
ICU length of stay (days)*	3.8 ± 2.1	5.9 ± 3.4	0.097
Surgical/radiological intervention, n (%)	4 (4.5)	9 (10.5)	0.134
30-day mortality, n (%)	2 (2.3)	4 (4.7)	0.440
30-day readmission, n (%)	6 (6.8)	8 (9.3)	0.547
Feeding intolerance, n (%)	10 (11.4)	3 (3.5)	0.049
Caloric intake at day 3 (kcal/day)	1,124 ± 384	286 ± 192	< 0.001

***Among patients admitted to ICU only.**

Laboratory Parameter Trajectories: Inflammatory markers demonstrated more rapid and pronounced improvement in the EEN group compared to the DF group (Table 3).

Table 3: Laboratory Parameters at Admission and Follow-Up

Parameter	Time Point	EEN Group (n = 88)	DF Group (n = 86)	p-value
CRP (mg/L)	Admission	86.3 ± 52.7	82.9 ± 49.8	0.659
	Day 3	72.4 ± 38.9	94.6 ± 47.3	0.001
	Day 5	48.7 ± 31.2	79.4 ± 42.6	< 0.001
	Day 7	24.3 ± 18.6	48.2 ± 34.7	< 0.001
Procalcitonin (ng/mL)	Admission	1.84 ± 2.31	1.92 ± 2.47	0.821
	Day 3	1.12 ± 1.46	1.78 ± 2.18	0.018
	Day 5	0.54 ± 0.72	1.23 ± 1.64	< 0.001
White blood cells (×10⁹/L)	Admission	13.4 ± 4.7	13.1 ± 4.9	0.676
	Day 3	10.6 ± 3.4	12.3 ± 4.2	0.004
	Day 5	8.4 ± 2.8	10.7 ± 3.6	< 0.001
Serum albumin (g/dL)	Admission	3.4 ± 0.5	3.3 ± 0.6	0.228
	Day 5	3.3 ± 0.4	2.9 ± 0.5	< 0.001
	Day 7	3.4 ± 0.5	2.8 ± 0.6	< 0.001

Multivariate logistic regression analysis identified delayed feeding (OR 2.84, 95% CI 1.18–6.83, p = 0.020), APACHE II score ≥10 (OR 3.67, 95% CI 1.52–8.86, p = 0.004), and admission CRP >150 mg/L (OR 2.41, 95% CI 1.04–5.58, p = 0.041) as independent predictors of infectious complications.

Discussion

The present study provides prospective evidence that early enteral nutrition initiated within 24 hours of hospital admission is associated with significantly improved clinical outcomes in patients with acute pancreatitis across all severity categories. Specifically, patients receiving early enteral nutrition demonstrated a 31% reduction in hospital length of stay, a 59% reduction in infectious complications, and a 58% reduction in organ failure incidence compared to patients managed with delayed feeding beyond 72 hours.

These findings align with the evolving understanding that prolonged fasting in acute pancreatitis is not only unnecessary but potentially harmful. The traditional concept of pancreatic rest, which formed the rationale for extended fasting, has been substantially undermined by physiological studies demonstrating that pancreatic exocrine secretion is already suppressed during acute pancreatitis regardless of enteral nutrient delivery [14]. Moreover, fasting leads to intestinal mucosal atrophy with increased permeability, facilitating bacterial translocation from the gut lumen to mesenteric lymph nodes and subsequently to the systemic circulation, thereby propagating the systemic inflammatory response and predisposing to infectious complications [15].

The significant reduction in infectious complications observed in our EEN group is consistent with prior randomized controlled trial data and meta-analyses. The landmark meta-analysis by Al-Omran and colleagues demonstrated that early enteral nutrition was associated with significantly lower rates of infection, organ failure, and mortality compared to parenteral nutrition in acute pancreatitis [16].

Similarly, a systematic review by Li and colleagues encompassing 11 randomized trials confirmed that early enteral feeding within 48 hours reduced infectious morbidity and shortened hospital stays [17]. Our study extends these findings by demonstrating comparable benefits in a real-world clinical setting with prospective data collection and comprehensive outcome assessment.

The more rapid resolution of inflammatory markers, particularly CRP and procalcitonin, in the EEN group provides mechanistic support for the clinical outcome differences observed. Early enteral nutrition preserves intestinal barrier integrity by maintaining enterocyte turnover, stimulating secretory immunoglobulin A production, and supporting the gut-associated lymphoid tissue [18]. These protective effects counteract the pathophysiological cascade of bacterial translocation, systemic endotoxemia, and immune dysregulation that drives secondary infection and organ failure in acute pancreatitis [19]. The preservation of serum albumin levels in the EEN group further supports the nutritional advantage of early feeding, as hypoalbuminemia has been independently associated with adverse outcomes in acute pancreatitis [20].

The PYTHON trial, which compared early nasoenteric tube feeding to an oral diet started at 72 hours, did not demonstrate superiority of early tube feeding in reducing the composite endpoint of major infection or death [8]. However, several important methodological differences distinguish our study from the PYTHON trial. The PYTHON trial used a composite primary endpoint and included an "on-demand" feeding approach in the control arm, where oral feeding was initiated at 72 hours rather than true prolonged fasting. In our delayed feeding group, nutritional support was withheld until symptom resolution or a minimum of 72 hours, and many patients did not tolerate oral intake until day 4 or 5, representing a more conservative and commonly practiced approach in many clinical settings.

The higher incidence of feeding intolerance in the EEN group (11.4% vs. 3.5%) warrants discussion. While this difference was statistically significant, all cases of intolerance in the EEN group were mild and manageable through temporary rate reduction or brief cessation followed by successful resumption of feeding. No patient in the EEN group required conversion to parenteral nutrition due to intolerance. These findings are consistent with prior reports suggesting that transient gastrointestinal symptoms during early feeding do not translate into clinically significant adverse outcomes [21]. Patient education and graduated feeding advancement protocols can effectively mitigate intolerance issues.

The non-significant difference in mortality between groups (2.3% vs. 4.7%) likely reflects the relatively low overall mortality in our cohort and insufficient statistical power for this secondary endpoint. Larger multicenter studies and meta-analyses have suggested mortality benefits with early enteral nutrition, although this remains a matter of ongoing investigation [22]. Our finding that delayed feeding was an independent predictor of infectious complications in multivariate analysis, even after adjusting for disease severity markers, underscores the clinical importance of nutritional timing as a modifiable determinant of outcomes.

Several limitations should be acknowledged. First, the non-randomized design introduces potential allocation bias, although baseline characteristics were well-balanced between groups, and the sequential protocol-change design minimizes selection bias related to individual physician preference. Second, the single-center setting may limit generalizability. Third, the study was not

powered to detect differences in mortality. Fourth, blinding was not feasible given the nature of the intervention. Fifth, long-term outcomes beyond 30 days were not assessed. Finally, we did not differentiate between oral and nasojejunal feeding routes within the EEN group, which may have introduced heterogeneity.

Future multicenter randomized controlled trials with larger sample sizes, stratified analyses by disease severity, and incorporation of gut barrier function biomarkers and microbiome analysis would further elucidate the optimal nutritional strategy and underlying mechanisms. Investigation of specific dietary compositions and caloric targets in early feeding represents an additional area requiring systematic evaluation [23].

Conclusion

This prospective comparative study demonstrates that early enteral nutrition initiated within 24 hours of hospital admission in acute pancreatitis is associated with significantly shorter hospital length of stay, reduced incidence of infectious complications, lower rates of organ failure, faster pain resolution, more rapid attenuation of systemic inflammation, and better preservation of nutritional status compared to delayed feeding beyond 72 hours.

These benefits were observed across all severity categories of acute pancreatitis. Although feeding intolerance was more frequent with early nutrition, it was mild and clinically manageable in all cases. These findings strongly support the implementation of early enteral nutrition as a standard component of acute pancreatitis management protocols. The paradigm of prolonged fasting for pancreatic rest should be definitively abandoned in favor of early, physiologically sound nutritional support that preserves gut barrier function and attenuates the systemic inflammatory cascade.

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