

ORIGINAL RESEARCH

EARLY VERSUS DELAYED ENTERAL FEEDING IN ACUTE PANCREATITIS: A PROSPECTIVE OBSERVATIONAL STUDY**Dr Raju N¹, Dr Loganayaki D^{1*}, Dr Sugumar S¹**¹ Assistant Professor, Department of General Surgery, Government Medical College and Hospital, Tiruppur, Tamil Nadu, India

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ABSTRACT

Background: Acute pancreatitis produces a systemic inflammatory response that increases metabolic demands and may impair intestinal mucosal integrity. Early enteral feeding has been proposed to preserve gut barrier function and accelerate recovery, but its optimal timing among clinically stable patients remains uncertain in routine practice. **Objective:** To compare the clinical outcomes and post-feeding biochemical profiles of early versus delayed enteral feeding in patients with acute pancreatitis. **Methods:** This prospective observational study was conducted over 12 months in the Department of General Surgery at a tertiary care teaching hospital in Tamil Nadu. One hundred eligible patients with acute pancreatitis were enrolled by non-probability consecutive sampling. Fifty commenced enteral feeding within 24 hours of admission and 50 commenced feeding after 48 hours, according to decisions made by the treating surgical team. The groups were compared for refeeding pain, ICU admission and duration of hospitalization. Blood urea, serum creatinine, serum amylase and serum lipase were assessed at admission and after feeding initiation. Categorical variables were compared using the Pearson chi-square or Fisher exact test, biochemical variables using the independent-samples Student t-test, and hospital stay using the Mann-Whitney U test. **Results:** The groups were comparable in age, sex, aetiology and recorded disease-severity scores. Refeeding pain was less frequent in the early-feeding group than in the delayed-feeding group (16.0% versus 46.0%; $p = 0.001$). None of the early-fed patients required ICU care, compared with 48.0% of delayed-fed patients ($p < 0.001$). Median hospital stay was 3 days (IQR: 2–3) in the early-feeding group and 4 days (IQR: 4–5) in the delayed-feeding group ($p < 0.001$). Biochemical parameters did not differ significantly between the groups at admission or after feeding initiation. **Conclusion:** Early enteral feeding within 24 hours was well tolerated and was associated with less refeeding pain, lower ICU use and shorter hospitalization without biochemical deterioration in this selected cohort of patients with acute pancreatitis. Because feeding timing was not randomized, the findings are susceptible to confounding by indication and require confirmation in randomized studies.

Keywords: Acute pancreatitis; delayed enteral feeding; early enteral feeding; hospital stay; refeeding pain

INTRODUCTION

Acute pancreatitis is an acute inflammatory disorder of the pancreas with a clinical spectrum that ranges from a brief, self-limiting illness to a severe syndrome accompanied by persistent organ failure and local or systemic complications [1]. Diagnosis rests on the presence of at least two of three criteria: characteristic acute upper abdominal pain, elevation of serum amylase or lipase to more than three times the upper limit of normal, and imaging findings consistent with pancreatic inflammation [2]. While the majority of cases are mild or moderately severe and resolve without lasting sequelae, a smaller proportion follow a severe course associated with substantial morbidity, prolonged hospitalization and mortality. Gallstones and alcohol consumption are among the leading aetiological factors, although their relative contributions vary between populations and clinical settings [3].

Beyond the local inflammatory process, acute pancreatitis provokes a systemic metabolic response characterized by increased energy expenditure and accelerated protein catabolism. In more extensive disease, circulating inflammatory mediators, intestinal hypoperfusion and impaired gastrointestinal motility may disrupt mucosal integrity and increase intestinal permeability [4]. A compromised intestinal barrier can facilitate translocation of enteric bacteria and their products across the gut wall, potentially seeding infected pancreatic necrosis and other systemic complications. Nutritional

support is therefore an integral component of management: it meets the heightened metabolic demands of the acute illness, helps maintain mucosal integrity and supports recovery [5].

Historically, patients with acute pancreatitis were kept strictly nil by mouth until pain had subsided and enzyme levels had fallen, on the premise that any oral intake would stimulate pancreatic secretion and perpetuate inflammation [6,7]. Prolonged fasting, however, carries real costs: negative energy balance, intestinal mucosal atrophy, impaired immune function and delayed recovery. If parenteral nutrition is used to bridge the fasting period, it introduces additional risks, including catheter-related infection, metabolic disturbances and substantially higher treatment costs. These considerations have progressively undermined the traditional policy of mandatory fasting, which is no longer considered appropriate for clinically stable patients [8].

Over the past two decades, early oral or enteral feeding has been incorporated into mainstream management. In mild disease, oral intake can often be initiated as tolerated without waiting for complete resolution of pain or normalization of enzyme levels [9]. When oral feeding is not immediately feasible, enteral nutrition delivered through a nasogastric or nasojejunal tube is strongly preferred over parenteral nutrition [10]. Current guidelines from the American College of Gastroenterology and the European Society for Clinical Nutrition and Metabolism support the initiation of oral or enteral feeding within the first 24–48 hours in clinically stable patients, with the exact timing, route and composition individualized according to disease severity and tolerance [8,11].

Evidence from systematic reviews, meta-analyses and randomized trials suggests that early enteral nutrition is safe and improves several relevant outcomes, including feeding tolerance, infection rate and duration of hospitalization, compared with prolonged fasting or parenteral nutrition [12,13]. The comparison between very early and delayed enteral nutrition in patients without shock or necrotic complications at enrolment, however, remains less thoroughly evaluated in routine hospital settings, where patient characteristics and local practices vary considerably [14,15]. Further prospective data from this setting are therefore needed to determine whether earlier initiation of feeding produces meaningful gains in clinical recovery without biochemical deterioration.

Against this background, the present study compared the clinical outcomes and post-feeding biochemical profiles of early (within 24 hours) and delayed (after 48 hours) enteral feeding among 100 selected patients with acute pancreatitis managed at a tertiary care teaching hospital in Tamil Nadu. The aim was to compare clinical outcomes and post-feeding biochemical profiles of early versus delayed enteral feeding in patients with acute pancreatitis. The specific objectives were to compare the frequency of refeeding pain, requirement for ICU care and duration of hospital stay between the feeding groups, and to compare blood urea, serum creatinine, serum amylase and serum lipase at admission and after initiation of feeding.

MATERIALS AND METHODS

Study design and setting

This prospective observational study was conducted in the Department of General Surgery, Government Medical College and Hospital, Tiruppur, Tamil Nadu, a tertiary care teaching hospital. Data were collected over 12 months, from May 2025 to April 2026.

Ethical considerations

Approval was obtained from the Institutional Ethics Committee, Government Medical College and Hospital, Tiruppur (approval no. 7/31-551/ME/2025, dated 14 May 2025), before commencing the study. Written informed consent was obtained from each participant prior to enrolment. All data were recorded on coded proformas, and participant confidentiality was maintained throughout collection, analysis and reporting. The study was conducted in accordance with the Declaration of Helsinki and applicable national ethical guidelines [16].

Participants

Consecutive patients admitted under the Department of General Surgery with a confirmed diagnosis of acute pancreatitis who satisfied the eligibility criteria and provided written consent were enrolled.

Inclusion criteria: Patients younger than 70 years with a confirmed diagnosis of acute pancreatitis, systolic blood pressure above 90 mmHg, serum creatinine below 2 mg/dL, and no evidence of shock, pancreatic or peripancreatic necrosis, or peripancreatic abscess at enrolment were included. Study eligibility was determined from clinical, laboratory and imaging findings available at enrolment. The complete Ranson score was calculated at 48 hours and was used to characterize disease severity rather than to determine initial eligibility or feeding timing.

Exclusion criteria: Patients aged 70 years or older, those with systolic blood pressure of 90 mmHg or lower, serum creatinine of 2 mg/dL or higher, shock at presentation, pancreatic or peripancreatic necrosis, or peripancreatic abscess were excluded.

Sample size and group allocation

A total of 100 eligible patients were prospectively enrolled using non-probability consecutive sampling. The timing of enteral feeding was determined solely by the treating surgical team as part of routine clinical care, without intervention by the investigators. Participants were categorized according to the observed timing of feeding initiation: those who commenced feeding within 24 hours of admission formed the early-feeding group, whereas those who commenced feeding after 48 hours formed the delayed-feeding group. Patients who commenced feeding between 24 and 48 hours were not included in the comparative cohort. Recruitment continued until 50 eligible participants had been included in each group.

Feeding and clinical management

The study did not prescribe a standardized route, formula, caloric target, initial volume or rate, or schedule for progression of enteral feeding. These decisions, including whether feeding was given orally or through a feeding tube and whether it was continued, advanced, withheld or stopped, were made by the treating surgical team according to the patient's clinical condition and feeding tolerance. Feeding-route and formula details were not systematically captured. ICU admission and hospital discharge were likewise determined by the treating team according to routine institutional practice; no study-specific ICU-admission or discharge criteria were imposed by the investigators.

Clinical assessment and baseline investigations

After written consent, a structured history was obtained covering age, sex, characteristics and radiation of abdominal pain, alcohol consumption, gallstone history and previous pancreatitis episodes, followed by a complete physical examination. Baseline investigations included complete blood count, random blood glucose, serum lactate dehydrogenase, aspartate aminotransferase, packed cell volume, blood urea, serum creatinine, arterial blood gas analysis, serum amylase and serum lipase. Serum amylase or lipase above three times the upper limit of normal was regarded as supportive of the diagnosis [2]. At 48 hours, serum calcium, packed cell volume, blood urea, serum creatinine and arterial blood gas parameters were reassessed for Ranson scoring. All participants included in the comparative cohort had contrast-enhanced CT findings available during the index hospitalization, permitting calculation of the original Balthazar CT severity index [17]. The post-feeding biochemical values used in the comparison were the first routine measurements available after feeding initiation; the interval between feeding initiation and blood sampling was not standardized by the study protocol.

Operational definitions

Acute pancreatitis: Diagnosis required at least two of the following: characteristic acute upper abdominal pain, serum amylase or lipase more than three times the upper limit of normal, and imaging findings consistent with acute pancreatitis [2].

Feeding groups: Early enteral feeding was defined as initiation of enteral nutrition within 24 hours of hospital admission, and delayed enteral feeding as initiation more than 48 hours after admission. **Refeeding pain:** New-onset or worsening

abdominal pain after initiation of feeding; ICU requirement: Admission or transfer to t; e intensive care unit during the index hospitalization. Duration of hospital stay: The number of days from the date of admission to the date of discharge.

Statistical analysis

Data were entered into Microsoft Excel (Microsoft Corporation, Redmond, WA, USA) and analysed using IBM SPSS Statistics, version 26.0 (IBM Corp., Armonk, NY, USA). Categorical variables are presented as frequencies and percentages and were compared using the Pearson chi-square test or Fisher exact test when expected cell counts were below five; the Fisher-Freeman-Halton extension was used for tables larger than 2×2 . Biochemical variables are presented as mean $\pm$ standard deviation and were compared using the independent-samples Student t-test, with mean differences and 95% confidence intervals. Duration of hospital stay was presented as median and interquartile range and compared using the Mann-Whitney U test. All tests were two-sided, and $p < 0.05$ was considered statistically significant [18].

RESULTS

Sociodemographic characteristics

The early and delayed feeding groups were comparable in age and sex distribution (Table 1). Across the full cohort, 45.0% of participants were aged 25–34 years and 31.0% were aged 35–44 years, with progressively fewer in older bands; only 4.0% were aged 55–65 years. The age distribution did not differ significantly between groups ($p = 0.948$). Men constituted 69.0% of the cohort (70.0% in the delayed group, 68.0% in the early group), and sex distribution was likewise comparable ($p = 0.829$).

Table 1. Sociodemographic characteristics of the study participants (N = 100)

Characteristic	Delayed feeding (n = 50), n (%)	Early feeding (n = 50), n (%)	Total (N = 100), n (%)	p value
Age group (years)				
25–34	24 (48.0)	21 (42.0)	45 (45.0)	0.948
35–44	15 (30.0)	16 (32.0)	31 (31.0)	
45–54	9 (18.0)	11 (22.0)	20 (20.0)	
55–65	2 (4.0)	2 (4.0)	4 (4.0)	
Sex				
Male	35 (70.0)	34 (68.0)	69 (69.0)	0.829
Female	15 (30.0)	16 (32.0)	31 (31.0)	

p value for age group from the Fisher–Freeman–Halton exact test (expected cell counts below five); p value for sex from the Pearson chi-square test.

Aetiology and clinical presentation

Pain radiating to the back was present in all 100 participants. Alcohol was the aetiological factor in 69.0% of the cohort, with a similar proportion in each group (70.0% delayed, 68.0% early; $p = 0.829$). Gallstones were identified in 30.0% of delayed-fed and 32.0% of early-fed patients ($p = 0.829$). No significant between-group differences were observed for either aetiological factor (Table 2).

Table 2. Aetiology and clinical presentation of acute pancreatitis (N = 100)

Clinical characteristic	Delayed feeding (n = 50), n (%)	Early feeding (n = 50), n (%)	Total (N = 100), n (%)	p value
Pain radiating to the back	50 (100.0)	50 (100.0)	100 (100.0)	
Alcohol aetiology	35 (70.0)	34 (68.0)	69 (69.0)	0.829
Gallstone aetiology	15 (30.0)	16 (32.0)	31 (31.0)	0.829

p values are from the Pearson chi-square test. Percentages use each group as the denominator.

Disease severity assessment

The distributions of the recorded disease-severity scores were comparable between groups. The complete Ranson score, assessed at 48 hours, was 2 in 55 (55.0%) participants and 3 in 45 (45.0%) participants, with no significant between-group difference ($p = 0.841$). Contrast-enhanced CT findings were assessed using the original Balthazar computed tomography severity index (CTSI), which combines a morphological score (0–4 points) with a pancreatic-necrosis score (0–6 points). Because pancreatic necrosis was an exclusion criterion, all participants had a necrosis score of 0. CTSI scores of 1 and 2 therefore corresponded to Balthazar grades B and C, respectively. A CTSI score of 2 was observed in 66 (66.0%) participants and a score of 1 in 34 (34.0%), with identical distributions in both feeding groups ($p = 1.000$) (Table 3).

Table 3. Comparison of disease severity scores between the feeding groups (N = 100)

Severity score	Delayed feeding (n = 50), n (%)	Early feeding (n = 50), n (%)	Total (N = 100), n (%)	p value
Ranson score				0.841
Score 2	27 (54.0)	28 (56.0)	55 (55.0)	
Score 3	23 (46.0)	22 (44.0)	45 (45.0)	
Balthazar CT severity index				1.000
Score 1 (Balthazar grade B)	17 (34.0)	17 (34.0)	34 (34.0)	
Score 2 (Balthazar grade C)	33 (66.0)	33 (66.0)	66 (66.0)	

CTSI, computed tomography severity index. The original Balthazar CTSI was calculated by adding the morphological score (0–4 points) and the pancreatic necrosis score (0–6 points). Because pancreatic necrosis was an exclusion criterion, all participants had a necrosis score of 0. Accordingly, CTSI score 1 represented Balthazar grade B (focal or diffuse pancreatic enlargement), while CTSI score 2 represented grade C (intrinsic pancreatic abnormality with peripancreatic inflammatory changes). p values were obtained using the Pearson chi-square test.

Clinical outcomes

Refeeding pain occurred in 23 (46.0%) patients in the delayed-feeding group compared with 8 (16.0%) patients in the early-feeding group ($p = 0.001$). ICU admission was required by 24 (48.0%) patients in the delayed-feeding group, whereas none of the patients in the early-feeding group required ICU admission ($p < 0.001$). The median duration of hospitalization was 4 days (IQR: 4–5 days) in the delayed-feeding group and 3 days (IQR: 2–3 days) in the early-feeding group ($p < 0.001$) (Table 4).

Table 4. Comparison of clinical outcomes between the feeding groups (N = 100)

Clinical outcome	Delayed feeding (n = 50)	Early feeding (n = 50)	Total (N = 100)	p value
Refeeding pain, n (%)				0.001*
Present	23 (46.0)	8 (16.0)	31 (31.0)	—
Absent	27 (54.0)	42 (84.0)	69 (69.0)	—
ICU admission, n (%)				<0.001*
Required	24 (48.0)	0 (0.0)	24 (24.0)	—
Not required	26 (52.0)	50 (100.0)	76 (76.0)	—
Duration of hospital stay, median (IQR), days	4 (4–5)	3 (2–3)	3.5 (3–4)	<0.001*

The Pearson chi-square test was used for refeeding pain, Fisher's exact test for ICU admission because of the zero-cell frequency, and the Mann–Whitney U test for duration of hospital stay. *Statistically significant at $p < 0.05$.

Biochemical parameters

At admission, no statistically significant differences were observed between the delayed- and early-feeding groups in random blood glucose (185.60 ± 43.40 versus 184.30 ± 43.20 mg/dL; $p = 0.881$), blood urea (33.96 ± 3.92 versus 33.60 ± 3.88 mg/dL; $p = 0.645$), serum creatinine (0.90 ± 0.23 versus 0.90 ± 0.25 mg/dL; $p = 1.000$), serum amylase (678.60 ± 194.70 versus 695.90 ± 180.40 U/L; $p = 0.645$), or serum lipase (249.22 ± 153.10 versus 258.02 ± 151.90 U/L; $p = 0.774$). Following the initiation of feeding, the groups also did not differ significantly in blood urea (32.66 ± 3.85 versus 32.48 ± 3.61 mg/dL; $p = 0.810$), serum creatinine (0.96 ± 0.27 versus 0.96 ± 0.26 mg/dL; $p = 1.000$), serum amylase ($80.54 \pm$

13.30 versus 81.22 ± 13.70 U/L; $p = 0.803$), or serum lipase (82.04 ± 15.30 versus 84.54 ± 13.60 U/L; $p = 0.392$) (Table 5).

Table 5. Comparison of biochemical parameters at admission and after initiation of feeding (N = 100)

Parameter	Delayed feeding (n = 50), mean $\pm$ SD	Early feeding (n = 50), mean $\pm$ SD	Mean difference (95% CI)	p value
At admission				
Random blood glucose (mg/dL)	185.60 ± 43.40	184.30 ± 43.20	1.30 (–15.90 to 18.50)	0.881
Blood urea (mg/dL)	33.96 ± 3.92	33.60 ± 3.88	0.36 (–1.19 to 1.91)	0.645
Serum creatinine (mg/dL)	0.90 ± 0.23	0.90 ± 0.25	0.00 (–0.10 to 0.10)	1.000
Serum amylase (U/L)	678.60 ± 194.70	695.90 ± 180.40	–17.30 (–91.79 to 57.19)	0.645
Serum lipase (U/L)	249.22 ± 153.10	258.02 ± 151.90	–8.80 (–69.35 to 51.75)	0.774
After initiation of feeding				
Blood urea (mg/dL)	32.66 ± 3.85	32.48 ± 3.61	0.18 (–1.30 to 1.66)	0.810
Serum creatinine (mg/dL)	0.96 ± 0.27	0.96 ± 0.26	0.00 (–0.11 to 0.11)	1.000
Serum amylase (U/L)	80.54 ± 13.30	81.22 ± 13.70	–0.68 (–6.07 to 4.71)	0.803
Serum lipase (U/L)	82.04 ± 15.30	84.54 ± 13.60	–2.50 (–8.27 to 3.27)	0.392

Values are presented as mean $\pm$ standard deviation. Mean differences were calculated as delayed feeding minus early feeding. p values were obtained using the independent-samples Student's t-test. CI, confidence interval; SD, standard deviation.

DISCUSSION

This prospective observational study compared early (within 24 hours) and delayed (after 48 hours) enteral feeding in 100 selected patients with acute pancreatitis. Early feeding was associated with less refeeding pain, lower ICU use and shorter hospitalization, without evidence of biochemical deterioration after feeding initiation. Although the groups had similar demographic, aetiological and recorded severity-score distributions, residual differences in clinical condition and treatment selection could have influenced the observed associations.

The cohort was predominantly male (69.0%) and predominantly alcohol-related (69.0%). McClave and colleagues, in a study comparing early enteral with parenteral nutrition in mild acute pancreatitis, similarly observed a predominance of male participants and alcohol-related aetiology [19]. Manjunath and Abhishek reported comparable age, sex and comorbidity profiles between early and delayed feeding groups in their randomized trial of patients with severe acute pancreatitis [14]. Powell and colleagues and Wereszczynska-Siemiatkowska and colleagues likewise documented comparable baseline profiles between their early and delayed enteral nutrition cohorts [15,20].

The distributions of the recorded Ranson and CT severity scores were comparable between groups. McClave et al. reported similar severity characteristics across their feeding groups [19]. Manjunath and Abhishek found no significant difference in Ranson scores between early- and delayed-feeding groups [14], while Powell et al. reported comparable entry characteristics among patients predicted to develop severe disease [15]. Nevertheless, the complete Ranson score is determined only after 48 hours and cannot establish equivalence in clinical trajectory at the time feeding decisions are made.

Refeeding pain was significantly less frequent in the early-feeding group (16.0% versus 46.0%; $p = 0.001$). One possible explanation is that early reintroduction of luminal nutrients helps to preserve enteric motility and mucosal integrity and limits the physiological disruption associated with prolonged fasting; however, this mechanism was not examined in the present study, and the difference may also reflect confounding by indication. Vaughn and colleagues, in a systematic review of early versus delayed feeding in acute pancreatitis, concluded that available trials did not demonstrate an increased risk of recurrent abdominal pain with early feeding [12]. Levy and colleagues prospectively described recurrent pain in approximately one-fifth of patients during refeeding in a multicentre cohort, and identified more severe disease on CT and higher serum lipase concentrations before refeeding among the predictors of pain relapse [21]. Sathiaraj and colleagues found that beginning oral intake with a soft diet did not increase pain recurrence relative to a clear-liquid diet

[22], and Moraes and colleagues showed that starting with a full solid diet was safe, with pain recurrence rates similar to those of clear-liquid or soft initial diets [23].

None of the early-fed patients required ICU care, compared with 48.0% in the delayed-feeding group ($p < 0.001$). Feng and colleagues, in a PRISMA-compliant meta-analysis, reported that early enteral nutrition was associated with a significantly lower risk of multiple organ failure, although not of mortality, compared with delayed nutrition [13]. In predicted severe acute pancreatitis, Wereszczynska-Siemiatkowska and colleagues found that delayed enteral nutrition was associated with more infected necrosis, respiratory failure, ICU transfer and deaths than enteral nutrition started within 48 hours [20], and Manjunath and Abhishek likewise reported fewer ICU admissions with early feeding [14]. Wu and colleagues, in a retrospective series, found a lower organ-failure rate among patients commenced on enteral nutrition within 48 hours [24]. Taken together, these data suggest that maintaining enteral nutrition early may limit the systemic inflammatory amplification that follows prolonged gut barrier disruption, although most of this evidence derives from patients with predicted severe disease. The magnitude of the ICU difference in the present study (48% versus 0%) is notably large for an observational comparison in which group allocation was not randomized; this observation is discussed further in the Limitations.

Duration of hospitalization was shorter in the early-feeding group, with a median stay of 3 days (IQR: 2–3) compared with 4 days (IQR: 4–5) in the delayed-feeding group ($p < 0.001$). Vaughn et al. concluded that early feeding did not increase adverse events and may shorten hospital stay in mild acute pancreatitis [12]. Eckerwall et al. reported that immediate oral feeding accelerated recovery and shortened hospitalization relative to conventional fasting [9], while Sathiaraj et al. and Moraes et al. reported similar findings with earlier oral reintroduction [22,23]. The observed one-day difference in median stay may be clinically relevant; however, part of this difference may be structurally related to the feeding-time definitions and non-random clinical selection.

No significant between-group differences in blood urea, serum creatinine, serum amylase or serum lipase were observed at admission or after feeding initiation. McClave et al. reported that early enteral nutrition was well tolerated in mild acute pancreatitis without worsening the biochemical course [19]. Powell et al. found that early enteral nutrition did not exacerbate inflammatory markers in patients with predicted severe disease [15]. Abou-Assi et al. reported favourable metabolic and clinical outcomes with jejunal enteral feeding compared with total parenteral nutrition [25], and Gupta et al. found that total enteral nutrition did not adversely affect metabolic, inflammatory or oxidative markers in predicted severe acute pancreatitis [26]. The similar post-feeding values in the present study provide no evidence of biochemical worsening associated with earlier feeding; however, the absence of ICU admission among early-fed patients cannot independently establish safety because feeding timing was selected clinically. Bakker et al. found that routine nasoenteric feeding within 24 hours did not reduce major infection or death compared with an on-demand strategy in predicted severe acute pancreatitis [27], underscoring the importance of randomized allocation and patient selection.

Limitations

The principal limitation was the non-randomized observational design. Feeding timing was determined clinically, creating a substantial risk of selection bias and confounding by indication; clinicians may have initiated feeding earlier in patients who appeared more stable, while patients with slower recovery or evolving organ dysfunction may have been fed later and required ICU care. The complete separation in ICU admission and hospital-stay distributions should therefore not be interpreted causally. The temporal relationship between ICU admission and feeding initiation was not analysed; because some patients may have been admitted to the ICU before feeding began, reverse causation cannot be excluded. No multivariable adjustment for individual clinical trajectories was performed. A formal sample-size calculation was not undertaken, the sample was relatively small, and non-probability sampling at a single centre limits generalizability. The Ranson score can be completed only at 48 hours, and the revised Atlanta classification was not applied. Because delayed feeding was defined as initiation after 48 hours, part of the hospital-stay difference may be inherent to group definition. Patients fed between 24 and 48 hours were excluded, introducing an additional selection concern. The feeding route, formula, dose and progression were not standardized or systematically captured, and the timing of post-feeding biochemical sampling was not protocolized. ICU admission and discharge were based on routine clinical judgement rather

than study-specific criteria. Comorbidities, baseline nutritional status, prior medication use, infectious complications, mortality and costs were not systematically assessed, and follow-up ended at discharge.

CONCLUSION

Among selected patients with acute pancreatitis, enteral feeding initiated within 24 hours was associated with less refeeding pain, lower ICU use and a shorter median hospital stay than feeding initiated after 48 hours, without evidence of biochemical deterioration. These findings support the feasibility of early feeding in clinically stable patients but do not establish a causal treatment effect because allocation was not randomized. Multicentre randomized studies using standardized feeding protocols, revised Atlanta severity classification, predefined ICU and discharge criteria, and adjustment for relevant prognostic factors are required to confirm these observations.

Data availability

The data supporting the findings of this study are available from the corresponding author on reasonable request.

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No external funding was received for this study.

Conflicts of Interest

The authors declare no conflict of interest.

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