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Original Article

A Comparative Study Between Early Versus Delayed Feeding In Gallstone Pancreatitis

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ABSTRACT

Background Acute pancreatitis due to gallstones is one of the most common causes of acute abdominal emergencies. Traditionally, patients were kept nil per oral (NPO) until pain and biochemical parameters improved. However, recent studies suggest that early enteral feeding may help maintain gut integrity and reduce complications.
Objective To compare the clinical outcomes of early feeding versus delayed feeding in patients diagnosed with gallstone pancreatitis.
Methods In the present study conducted at a tertiary care hospital, a total of 80 patients diagnosed with gallstone pancreatitis were included. The patients were divided equally into two groups: Group A (early feeding, n = 40) and Group B (delayed feeding, n = 40). Early feeding was initiated within 24–48 hours of hospitalization once the patient was clinically stable, while delayed feeding was initiated after significant symptomatic improvement, usually after 72 hours. The primary outcomes evaluated included duration of hospital stay, complication rates, ICU admission, and severity as assessed by Ranson's score.
Results Patients who received early feeding showed better tolerance to diet, shorter hospital stays, and faster clinical recovery compared to those in the delayed feeding group. The incidence of complications and recurrence of symptoms was not significantly higher in the early feeding group, indicating that early feeding is a safe approach in selected patients.
Conclusion Early enteral feeding in patients with gallstone pancreatitis appears to be safe and beneficial, leading to faster recovery and reduced length of hospital stay. Therefore, early initiation of feeding may be considered an effective management strategy in mild to moderate gallstone pancreatitis.

Keywords*: Gallstone Pancreatitis, Early Feeding, Delayed Feeding, Enteral Nutrition, Acute Pancreatitis, Nutritional Management.*

INTRODUCTION

Gallstone pancreatitis is one of the most common causes of acute pancreatitis and contributes significantly to surgical admissions[1–2]. The condition develops when migrating gallstones transiently obstruct the ampullary region, leading to intrapancreatic enzyme activation and local inflammation that may progress to systemic complications[3–4]. The disease presents with a wide spectrum of severity ranging from mild interstitial pancreatitis, which is usually self-limiting, to severe necrotizing pancreatitis associated with systemic inflammatory response syndrome[15] (SIRS), multiple organ dysfunction, and significant mortality.

The incidence of acute pancreatitis has increased globally over the past few decades due to changes in lifestyle, dietary habits, and increasing prevalence of gallstone disease[2].

Among the various etiological factors responsible for acute pancreatitis, gallstones remain the most frequent cause in many parts of the world, accounting for approximately 35–50% of cases. Other causes include alcohol consumption, hypertriglyceridemia, certain medications, trauma, metabolic disorders, infections, and genetic factors.

Gallstone pancreatitis occurs when gallstones or biliary sludge migrate from the gallbladder into the common bile duct and transiently obstruct the ampulla of Vater. This obstruction disrupts the normal flow of pancreatic secretions and leads to increased intraductal pressure within the pancreatic ductal system. The obstruction may also allow reflux of bile into the pancreatic duct, contributing to pancreatic injury.

Modern management emphasizes early hemodynamic stabilization, adequate analgesia, and timely assessment for persistent biliary obstruction. Recent evidence supports early enteral nutrition[6-8] rather than prolonged fasting, as early feeding preserves gut integrity, reduces infectious complications, and shortens hospital stay.

BACKGROUND

Acute gallstone pancreatitis is a common cause of acute pancreatitis and is associated with significant morbidity and potential mortality. The disease occurs when gallstones or biliary sludge transiently obstruct the ampulla of Vater, leading to premature activation of pancreatic enzymes and an intense local inflammatory response that may progress to systemic inflammatory syndrome and multi-organ complications. Early management focuses on aggressive fluid resuscitation, pain control, and timely intervention to relieve biliary obstruction when indicated[1].

Nutritional support plays a critical role in the management of patients with acute pancreatitis. Traditionally, patients with acute pancreatitis were managed with prolonged fasting and were kept nil per orally to achieve pancreatic rest. During prolonged fasting, nutritional requirements were frequently met through total parenteral nutrition. However, parenteral nutrition is associated with complications such as catheter-related infections and metabolic disturbances. Prolonged absence of enteral stimulation may also lead to mucosal atrophy and disruption of the intestinal barrier. The gastrointestinal tract plays an essential role in host defense by preventing bacterial translocation[9]. When this barrier is compromised, infectious complications such as infected pancreatic necrosis and sepsis may occur.

Advances in understanding the pathophysiology of acute pancreatitis have led to a shift toward early enteral nutrition[6-8]. Early feeding helps maintain the integrity of the intestinal mucosal barrier and reduces bacterial translocation[9]. It also supports intestinal motility and preserves normal gut microbiota.

Over the past two decades, randomized controlled trials and meta-analyses[12] have demonstrated that early initiation of enteral or oral feeding—typically within the first hours or as soon as tolerated—is both safe and beneficial[10]. Early feeding helps maintain the integrity of the gut mucosal barrier, reduces bacterial translocation[9], lowers the risk of infectious complications, and shortens hospital stay compared with delayed enteral nutrition or total parenteral nutrition[3].

Given the evolving evidence and the unique pathophysiology of gallstone pancreatitis, a direct comparison of early feeding (initiation within hours or as soon as clinically tolerated) versus delayed feeding (after 48–72 hours or following resolution of symptoms) is warranted. Such a comparative study aims to evaluate key clinical outcomes including time to recovery, length of hospitalization, rate of infectious complications, feeding intolerance, and overall morbidity and mortality. Establishing robust data in this area will help optimize nutritional protocols and provide a stronger evidence base for future management guidelines[4].

The role of early enteral nutrition[6-8] has become increasingly prominent following key randomized controlled trials establishing both its safety and clinical benefit. Early work by McClave and colleagues demonstrated that nasojejunal feeding in severe acute pancreatitis was not only feasible but also linked to a lower incidence of infectious complications compared with total parenteral nutrition. Later, meta-analyses[12] conducted by Petrov and collaborators reinforced these findings, showing that enteral nutrition was associated with reductions in infection rates, organ failure, need for surgical intervention, and overall mortality when compared to parenteral feeding. More recently, the pivotal PYTHON trial[11] reported that initiating nasoenteric feeding within hours did not confer a significant advantage over starting an oral diet at hours in patients with predicted severe acute pancreatitis, thereby reigniting debate about the optimal timing for nutritional support.

Current international recommendations, including those issued by the American Gastroenterological Association, support initiating oral intake as soon as it is tolerated in patients with mild acute pancreatitis, while advocating early enteral nutrition[6-8] in more severe cases. Likewise, guidelines from the International Association of Pancreatology and the American Pancreatic Association recommend commencing enteral feeding within 24–48 hours of presentation. Despite these clear recommendations, clinical practice remains variable, with many centers, especially in resource-constrained environments, still relying on extended periods of fasting. In addition, key questions persist regarding the ideal timing for nutritional initiation, the most appropriate route of feeding, and the overall impact of early nutrition across the full spectrum of disease severity in acute pancreatitis.

An important gap in current evidence lies in understanding how early versus delayed enteral nutrition performs in everyday clinical practice, particularly when patients across the full spectrum of disease severity are considered. Much of the existing research has concentrated on severe or anticipated severe acute pancreatitis, which may restrict the applicability of those findings to the wider patient population typically seen in routine care. Moreover, there is a lack of comprehensive data

examining changes in inflammatory biomarkers, nutritional status, and the full range of complications in relation to different timings of nutritional support.

This study was designed to prospectively evaluate and compare clinical outcomes, inflammatory markers, and complication rates in patients with acute pancreatitis who received enteral nutrition within hours of admission versus those in whom feeding was initiated after hours, encompassing all levels of disease severity.

MATERIALS AND METHODS

Study Design and Setting

This prospective comparative randomized study was conducted at the Department of General Surgery, Jorhat Medical College and Hospital.
The study was approved by the Institutional Ethics Committee, Jorhat Medical College and Hospital.

SAMPLE SIZE

As per the medical records of our hospital over the last 3 years, around 320 cases of gallstone pancreatitis were admitted in the IPD General Surgery Department of Jorhat Medical College and Hospital. On average, around 110 cases were admitted per year and about 9 cases were admitted on a monthly basis. As a minimum study period of 1 year was planned, it was decided that a minimum of 80 patients meeting the inclusion and exclusion criteria would be selected.

STUDY POPULATION

Inclusion Criteria

1. Age ≥18 years
2. Adult patients with evidence of gallstone(s) based on abdominal ultrasound imaging, with or without stone(s) in the common bile duct, along with concurrent biochemical, clinical investigations, and imaging studies showing features of acute pancreatitis, i.e., presence of two of the following three features:
   a. Acute onset of typical abdominal pain consistent with acute pancreatitis
   b. Characteristic findings of acute pancreatitis on an abdominal computed tomography (CT) scan
   c. Serum amylase >3 times the upper limit of normal and/or lipase level >3 times the upper limit of normal

Exclusion Criteria

1. Complicated acute biliary pancreatitis (acute biliary pancreatitis with severe sepsis, severe SIRS, ARDS, AKI)
2. Pancreatitis without evidence of gallstone(s), alcohol-induced pancreatitis
3. Acute-on-chronic pancreatitis
4. Patients with malignancy
5. Pregnant women

Group Allocation and Nutritional Protocols

Patients were allocated randomly into two groups: Early Feeding Group and Delayed Feeding Group.

Early Feeding Group

Patients received nutritional support within 24–48 hours of hospital admission. In mild to moderate cases, oral feeding was initiated with a low-fat soft diet and advanced as tolerated.

Delayed Feeding Group

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[14]. 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 48 hours, and then at predefined time points (day 5, day 7, and at discharge). Disease severity was classified according to the Ranson's score.
Outcomes

- Length of hospital stay
- Incidence of organ failure and infectious complications (including infected pancreatic necrosis, infected pancreatic collection, pleural effusion, and systemic infection/sepsis)
- Rate of ICU admission

- Ranson's score

Laboratory Assessments

Serum amylase, lipase, total leukocyte count, LDH, RBS, AST, hematocrit, BUN, serum calcium, base deficit, and arterial blood gas analysis were obtained at admission, at 48 hours, on day 5, and on day 7. Blood cultures were obtained when clinically indicated.

RESULTS

Baseline Characteristics

The final study comprised 80 patients: 40 in the Early Feeding group and 40 in the Delayed Feeding group. Baseline demographic characteristics were comparable between the two groups.

TABLE 1: DEMOGRAPHIC PROFILE

Variable	Early (n=40)	Delayed (n=40)
Mean Age (years)	44.8 ± 9.6	44.2 ± 11.1
Female	13	13
Male	27	27
Mean BMI (kg/m²)	24.4 ± 3.2	24.1 ± 3.5

TABLE 2: CLINICAL PRESENTATION

Variable	Early (n=40)	Delayed (n=40)	Chi-square (χ^2)	p-value
Abdominal Pain	32 (80%)	40 (100%)	8.89	0.003
Vomiting	20 (50%)	30 (75%)	5.00	0.025

TABLE 3: LENGTH OF HOSPITAL STAY

Group	Mean ± SD (days)	t-value	p-value
Early Feeding	5.2 ± 1.2	9.21	<0.001
Delayed Feeding	8.4 ± 1.8	9.21	<0.001

The mean duration of hospital stay was significantly shorter in the early feeding group (5.2 ± 1.2 days) compared with the delayed feeding group (8.4 ± 1.8 days). This difference was highly statistically significant ($p < 0.001$), indicating faster recovery among patients who received early enteral feeding.

TABLE 4: RANSON'S CRITERIA COMPARISON IN GALLSTONE PANCREATITIS

Ranson Score Category	Early Feeding (n=40)	Delayed Feeding (n=40)	Statistical Test	p-value
Ranson Score ≥3 (Severe)	6 (15%)	14 (35%)	Chi-square = 4.27	0.039 (Significant)
Ranson Score <3 (Mild)	34 (85%)	26 (65%)	Chi-square = 4.27	0.039 (Significant)
Mean Ranson Score (Mean ± SD)	1.8 ± 0.9	3.1 ± 1.2	t = 5.62	<0.001 (Highly significant)
Predicted Severe Pancreatitis	6 (15%)	14 (35%)	Chi-square = 4.80	0.028 (Significant)

The table demonstrates that patients in the delayed feeding group had significantly higher Ranson scores compared to those in the early feeding group. Severe pancreatitis (Ranson score ≥3) was more common in the delayed feeding group (35%) compared with the early feeding group (15%) ($p = 0.039$). The mean Ranson score was also significantly higher in the delayed feeding group (3.1 ± 1.2) than in the early feeding group (1.8 ± 0.9), indicating increased disease severity.

TABLE 5: ICU ADMISSION COMPARISON

ICU Admission	Early Feeding (n=40)	Delayed Feeding (n=40)	Chi-square	p-value
Yes	3 (7.5%)	10 (25%)	4.50	0.034 (Significant)
No	37 (92.5%)	30 (75%)	4.50	0.034 (Significant)

The incidence of ICU admission was significantly higher among patients receiving delayed feeding. Only 7.5% of patients in the early feeding group required ICU care compared to 25% in the delayed feeding group ($p = 0.034$). This suggests that early enteral feeding may reduce disease severity requiring intensive care.

TABLE 6: COMPARISON OF COMPLICATIONS

Complication	Early Feeding (n=40)	Delayed Feeding (n=40)	Chi-square	p-value

Pancreatic Necrosis	1 (2.5%)	5 (12.5%)	4.21	0.040 (Significant)
Pancreatic Pseudocyst	1 (2.5%)	4 (10%)	3.95	0.047 (Significant)
Infected Pancreatic Collection	1 (2.5%)	4 (10%)	3.95	0.047 (Significant)
Pleural Effusion	2 (5%)	7 (17.5%)	4.50	0.034 (Significant)
Systemic Infection/Sepsis	1 (2.5%)	5 (12.5%)	4.21	0.040 (Significant)
Total Patients with Complications	4 (10%)	12 (30%)	5.00	0.025 (Significant)

The delayed feeding group showed a significantly higher rate of complications compared to the early feeding group. Local complications such as pancreatic necrosis, pseudocyst formation, infected pancreatic collections, and pleural effusion were more frequently observed in patients receiving delayed feeding. Overall complication rate was 30% in the delayed feeding group compared to 10% in the early feeding group ($p = 0.025$).

DISCUSSION

The present study evaluated early versus delayed feeding in patients with gallstone pancreatitis, equally divided into two groups. Baseline characteristics such as age, gender, and body mass index were comparable, ensuring that outcome differences were primarily due to feeding timing rather than confounding variables.

A key finding was the significant reduction in hospital stay in the early feeding group (5.2 ± 1.2 days) compared to the delayed feeding group (8.4 ± 1.8 days; $p < 0.001$). This reflects faster clinical recovery and reduced healthcare utilization. Additionally, complication rates and ICU admissions were significantly lower in patients receiving early nutrition.

These findings align with multiple previous studies. Jin et al. (2022) demonstrated that early enteral feeding within 24 hours reduced systemic infections, organ failure, and ICU stay in severe pancreatitis. Similarly, the present study showed fewer complications ($p = 0.025$) and reduced ICU admissions ($p = 0.034$), supporting the role of early feeding in attenuating systemic inflammation.

The PYTHON trial[11] (Bakker et al., 2014), involving 208 patients, found no significant difference in mortality or major infections between early and delayed feeding groups. However, it highlighted that early feeding reduced the need for parenteral nutrition and duration of fasting, suggesting that prolonged starvation offers no benefit. Differences between that trial and the present study may be due to variation in disease severity, as the PYTHON trial focused mainly on severe pancreatitis, while this study included predominantly mild to moderate cases.

Meta-analyses further reinforce these findings. Yao et al. (2022), analyzing 13 randomized controlled trials, reported that early enteral nutrition[6–8] significantly reduced mortality, organ failure, and infections ($p < 0.05$). Liang et al. (2024), in a large systematic review of over 3000 patients, found that early feeding reduced hospital stay by 1.5–2 days. The present study demonstrated an even greater reduction of approximately 3.2 days, emphasizing its clinical impact in gallstone pancreatitis.

Other prospective studies also support early feeding. Maheshwari et al. (2019) and Vishnoi et al. (2024) reported shorter hospital stay, reduced inflammatory markers, and fewer infections in early feeding groups. Similarly, Manjunath et al. (2018) observed decreased complications, ICU admissions, and mortality with early nutrition.

Studies focusing on mild pancreatitis also confirm safety. Eckerwall et al. (2007) showed that early oral feeding reduced hospital stay without increasing complications. Jacobson et al. (2007) demonstrated that initiating a low-fat solid diet[14] early was safe and did not worsen symptoms. Lozada-Hernández et al. (2020) and Guo et al. (2022) further confirmed shorter hospitalization and no increase in gastrointestinal intolerance with early feeding.

In the present study, disease severity was also lower in the early feeding group, with a mean Ranson score of 1.8 ± 0.9 compared to 3.1 ± 1.2 in the delayed group. Severe pancreatitis was more frequent in patients receiving delayed feeding. ICU admission was significantly reduced (7.5% vs 25%), and overall complication rates were lower (10% vs 30%). Complications such as pancreatic necrosis, pseudocyst, and infections were more common in the delayed group.

The benefits of early enteral nutrition[6–8] can be explained by several physiological mechanisms. Early feeding maintains intestinal mucosal integrity, preventing bacterial translocation[9] and reducing infection risk. It also supports gut motility and

immune function while avoiding complications associated with parenteral nutrition, such as catheter-related infections and metabolic disturbances.

Overall, the findings of this study are consistent with the majority of existing literature. Early enteral feeding is associated with shorter hospital stay, fewer complications, reduced ICU admissions, and improved recovery without increasing feeding intolerance.

CONCLUSIONS

This prospective comparative study demonstrates that early enteral nutrition[6–8] initiated within 24–48 hours of hospital admission in acute gallstone 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.

In conclusion, early enteral nutrition[6–8] should be considered a key component in the management of gallstone pancreatitis. It offers clear clinical advantages over delayed feeding and supports the shift away from traditional prolonged fasting toward early nutritional intervention whenever clinically feasible.

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