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NUTRITIONAL SUPPORT IN ACUTE PANCREATITIS: A COMPARATIVE STUDY OF TOTAL PARENTERAL NUTRITION AND ENTERAL NUTRITION

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ABSTRACT

Background: The purpose of this study is to investigate the effects of various nutritional support techniques, notably EEN and TPN, on the clinical progression of AP. Through the process of contrasting different approaches during the key phases of the disease, our objective is to determine which way is the most efficient in terms of enhancing patient outcomes while simultaneously reducing the likelihood of complications. **Methods:** This observational study analyzed 440 patients who were diagnosed with acute pancreatitis (AP). Clinical criteria were used to determine whether TPN or EEN should be administered, including the severity of AP, the presence of dynamic intestinal obstruction, and the risk of infection complications. The nutritional support was enhanced by standard AP management, which involved providing pain relief, administering fluids, prescribing antibiotics, and closely monitoring pancreatic enzymes. **Results:** Early administration of EEN increased pancreatic secretion and impaired AP course. Use of TPN is recommended for 5-14 days after AP onset since it has been shown to improve PA and illness progression. EEN is crucial during the purulent-necrotic phase of AP, promoting recovery when provided at the right moment. Reduced postoperative mortality to 4.2% with the proposed nutritional and surgical strategies. Overall mortality decreased to 3.7% in AP patients. **Conclusion:**The study emphasized the significance of a precisely timed nutritional support strategy. In the early stages, the use of EEN worsened AP, whereas TPN proved to be effective when administered for 5-14 days. Enteral nutrition has demonstrated its value during the purulent-necrotic phase. By implementing a careful balance of nutritional support and utilizing less invasive surgical techniques, a notable decrease in mortality rates was observed.

KEYWORDS

Acute pancreatitis, protein-energy provision, nutritional support, enteral nutrition, parenteral nutrition, intestinal lavage, pancreatic secretion.

INTRODUCTION

Acute pancreatitis (AP) is a potentially life-threatening condition characterized by the sudden inflammation of the pancreas. The mortality rate in AP can exhibit significant variation, with mild cases showing a range of 4-10% and severe, destructive forms reaching as high as 25-80% [1]. Severe acute pancreatitis (SAP) is associated with a significant mortality rate, primarily resulting from sepsis, necrosis, and multiple organ failure triggered by infection in the pancreas or peripancreatic area [2]. Around 80% of deaths resulting from SAP are associated with sepsis, necrosis, and multiple organ failure [3].

The management of AP often requires the suppression of pancreatic secretion to prevent the worsening of the condition. The role of nutritional support is crucial in this process, as the method and timing of nutritional intervention have a significant impact on patient outcomes. Efficient management necessitates ensuring proper rest for the pancreas, which relies on sufficient provision of protein-energy (PEP) [4,5]. According to certain researchers, administering total parenteral nutrition (TPN) during treatment may enhance the nutritional status of patients without triggering pancreatic juice secretion, thus allowing the pancreas to fully rest [6]. On the other hand, additional studies [7] have highlighted the potential drawbacks of parenteral nutrition support, such as the risk of infection, as well as the occurrence of hyperglycemia and hyper-electrolyte imbalance syndrome. Several studies [8-10] have demonstrated the numerous benefits of enteral nutrition (EEN) in terms of lowering infection rates, reducing mortality, and decreasing the need for surgical intervention.

The purpose of this study is to investigate the effects of various nutritional support techniques, notably EEN and TPN, on the clinical progression of AP. Through the process of contrasting different approaches during the key phases of the disease, our objective is to determine which way is the most efficient in terms of enhancing patient outcomes while simultaneously reducing the likelihood of complications.

MATERIALS AND METHODS

STUDY DESIGN AND PARTICIPANTS

This observational study was carried out from January 2022 to December 2023 year at Parkar hospital, Ratnagiri, India, following approval from the institutional review board. Our study included 440 patients who were diagnosed with acute pancreatitis (AP) and had a mean age of 58.7±5.8 years.

Patients were divided into two groups based on the type of protein-energy provision (PEP) during the enzyme toxaemia phase and the intermediate phase of AP:

Group I comprised 405 individuals who were administered total parenteral nutrition (TPN).

Group II comprised 35 individuals who were administered with early enteral tube feeding (EEN) beginning on the second day of the disease. Clinical criteria were used to determine whether TPN or EEN should be administered, including the severity of AP, the presence of dynamic intestinal obstruction, and the risk of infection complications. The nutritional support was enhanced by standard AP management, which involved providing pain relief, administering fluids, prescribing antibiotics, and closely monitoring pancreatic enzymes.

Intervention and Procedures

Acute pancreatitis was regarded as the initial manifestation of pancreatic inflammation. Exclusions were made in the analysis for cases of AP recurrences, as they typically exhibit a more positive trajectory. This can be attributed to the decline in pancreatic functional activity, which occurs as a result of tissue mass loss, intra-organ fibrosis, and the formation of connective tissue around the organ. These factors impede the escape of enzymes. The clinic adhered to a cautious treatment approach for patients with AP until the emergence of purulent-necrotic lesions, which were deemed the sole indication for surgical intervention.

The comprehensive treatment plan was based on the severity of AP and included various interventions such as pain management, correction of hemodynamic and circulation disorders, detoxification therapy, suppression of pancreatic and gastric secretion, oxygen therapy, antibacterial therapy, and nutritional support.

The developed strategy involved performing intestinal lavage using a naso-jejunal catheter that was endoscopically placed upon admission. The rapid emptying of the bowel from chyme was facilitated, while the function of APUD cells located in the intestinal wall and pancreatic secretion was suppressed. The catheter was placed 10-20 cm below the duodenojejunal junction. The catheter's placement was confirmed using radiological imaging. The intestinal lavage involves the use of an electrolyte isotonic solution that is cooled to a temperature of 16–18°C. The solution is administered at a rate of 100-10 ml per

minute, with a total volume of up to 800–1000 ml. The effectiveness criterion was determined by the presence of a significant amount of liquid stool. Starting the next day, warm saline or 5% glucose solution was administered into the intestine, providing the patient with up to 1000 ml of fluid to meet their hydration needs, but not providing significant energy.

The severity of AP prognosis was evaluated using the Ranson criteria. In cases where complete information was not available, the prognosis relied on the clinic's system, which showed a correlation with the results obtained from Ranson's criteria. When choosing the energy substrate for TPN, it was based on the knowledge that high levels of triglycerides in the blood can increase the risk of AP. According to Dudrick, parenteral nutrition with glucose can sustain metabolic processes for up to 2 weeks without causing fat deficiency. In addition, fats used for parenteral nutrition are typically given with heparin. However, this can be problematic for patients with acute pancreatitis as it increases the risk of erosive bleeding. Thus, during the initial two stages of AP, the energy substrate employed consisted solely of concentrated (15-20%) glucose solutions, devoid of any fat emulsions.

Outcome Measures

An evaluation was conducted to determine the effects of the nutritional support method on pancreatic secretion and the activity of the inflammatory process. This assessment was based on various clinical and laboratory indicators. Several factors can be assessed to determine the presence and severity of a medical condition. These include pain, body temperature, blood leukocyte count, left shift of neutrophils, dynamic intestinal obstruction, blood amylase levels, the presence of inflammatory infiltrate in the abdominal cavity, ultrasound (US) and CT signs of AP, and levels of gastrointestinal peptides (secretin, cholecystokinin, gastrin, somatostatin).

A model involving complete external pancreatic fistulas was utilized to evaluate exocrine pancreatic secretion in a group of 18 patients. Nutritional support was administered through a high protein diet, EEN with a well-balanced oligopeptide mixture, or TPN. 9 patients experienced pancreatic fistula formation due to trauma to the pancreas, 7 patients who developed it as a result of necrotic complications of acute pancreatitis and 2 patients who had pancreaticoduodenal resections for pancreatic cancer. The evaluation of pancreatic secretion was based on the daily volume of juice secreted through the fistula. Oral feeding resulted in the largest amount of released juice, which was 100%.

RESULTS

DEMOGRAPHICS AND PATIENT CHARACTERISTICS

Our study included 440 patients who were diagnosed with acute pancreatitis (AP) and had a mean age of 58.7±5.8 years. The study included 280 male participants (63.63%) and 160 female participants (36.36%). The primary causes of AP were gallstone disease (18.9%), alcohol consumption (43.2%), or a combination of both (37.9%).

IMPACT OF NUTRITIONAL SUPPORT ON CLINICAL MANIFESTATIONS

Patients in the TPN group attained faster pain relief, typically within 1–2 days, compared to those in the EEN group, where pain persisted for 4–6 days or more. The TPN group experienced resolution of hyperthermia within 4 days, whereas the EEN group had persistent hyperthermia for 14 days or longer. There appears to be a longer period of elevated body temperature in the EEN group, indicating a prolonged inflammatory response when early enteral feeding is used. The TPN group experienced normalization of serum amylase levels within 7–10 days. In contrast, patients in the EEN group showed elevated amylase levels for over 10 days, suggesting that TPN might be more successful in suppressing pancreatic activity. TPN was found to be linked to a decrease in the occurrence of dynamic intestinal obstruction, while EEN was found to contribute to the advancement of this condition. It is indicated that TPN may be beneficial in reducing the occurrence of serious gastrointestinal complications in patients with AP. In patients with moderate AP receiving TPN, gastrostasis was either absent or resolved within 3 days. Nevertheless, the observed characteristic persisted for a duration exceeding 3 days in the EEN group, which brings attention to a potential drawback of early enteral feeding in this particular patient population. No inflammatory infiltrates were found in the TPN group. Nevertheless, it is worth noting that a significant portion of patients (32%) in the EEN group experienced the development of inflammatory infiltrates, despite having mild acute pancreatitis. These results suggest that early enteral feeding could potentially worsen local inflammation, especially in patients with less severe forms of AP. Table 1 summarizes the comparative effects of total parenteral nutrition (TPN) and early enteral nutrition (EEN) on the clinical manifestations of acute pancreatitis (AP) when initiated during the enzymatic toxaemia phase.

Comparative Analysis of Nutritional Support on Pancreatic Secretion

TPN plays a crucial role in supporting basal pancreatic secretion. This secretion, when subjected to pharmacological suppression, decreased significantly to only 5-7% of the levels observed with oral feeding. Basal pancreatic secretion levels were not achieved until day 5 in the absence of intestinal lavage. Nevertheless, by implementing initial intestinal lavage alongside H2-blockers and octreotide administration, the desired outcome of basal secretion was successfully attained within just one day. Irrespective of the mixture's composition, the volume of secretion through the pancreatic fistula was significantly increased by EEN, accounting for 80-90% of the volume observed with oral feeding. There were no notable distinctions observed in the impact of balanced and oligopeptide mixtures on the progression of AP and pancreatic secretory activity in patients with pancreatic fistulas. The influence of TPN and EEN on pancreatic secretion in patients with pancreatic fistulas is summarized in Table 2.

PROTEIN-ENERGY DEFICIENCY AND NITROGEN LOSSES

The study also examined nitrogen losses and the potential for protein-energy deficiency across various levels of AP severity.

MILD AP: Nitrogen losses were within expected levels. The administration of 12–14 g of nitrogen and 1600–1800 kcal provided an adequate amount of nitrogen and calories to maintain a positive nitrogen balance during days 3-5 of the disease.

Moderate AP: Between days 3–5, nitrogen losses increased 1.5 times, and between days 7–10, they returned to normal. The TPN supplied 14–16 g of nitrogen and 2000–2400 kcal, resulting in a positive nitrogen balance by days 8–10.

Severe AP: Multiple organ failure (MOF) was facilitated by maximum nitrogen losses that were three times or greater than normal values. Even with sufficient purulent cavity drainage, it was almost impossible to achieve a positive nitrogen balance in less than two to three weeks.

Before the clinical manifestation of purulent necrotic lesions, certain indicators of protein-energy deficiency were noted in patients with severe AP. These indicators included body weight loss, decreased skin fold thickness, reduced arm muscle circumference, hypoalbuminemia, hypoproteinemia, and lymphopenia.

MORTALITY AND SURGICAL OUTCOMES

The study revealed notable variations in mortality rates depending on the type of nutritional support and the timing of surgical intervention. In the early stages of enzymatic toxemia, the mortality rate was significantly higher at 48%. However, surgical intervention during the purulent phase resulted in a notable reduction in mortality to 19.3% with an overall mortality rate of 14%. The proposed nutritional and surgical strategies, which involved TPN and minimally invasive surgical methods, resulted in a decrease in postoperative mortality to 4.2%. The overall mortality rate among AP patients who received TPN was 3.7%. The significance of precise timing in the implementation of nutritional support and the choice of suitable surgical approaches to enhance patient outcomes in acute pancreatitis is highlighted by these findings.

DISCUSSION

This study emphasizes the critical importance of nutritional support in the management of acute pancreatitis (AP), highlighting the need to customize the approach based on the patient's disease phase and severity. Based on our research, it appears that implementing total parenteral nutrition (TPN) within the first 5-14 days of acute pancreatitis (AP) is more effective than early enteral nutrition (EEN) in controlling pancreatic secretion, reducing the inflammatory response, and minimizing complications such as dynamic intestinal obstruction and purulent complications.

To facilitate the healing process of the pancreas, conventional therapy methods for AP often involve a prolonged period of fasting, which helps to decrease pancreatic secretion and allow the pancreas to rest.

Based on the previous studies conducted, it has been determined that the recommended duration of therapeutic fasting and artificial nutrition varies depending on the severity of AP for patients with mild AP, a fasting period of 5-7 days is advised. Those with moderate AP should undergo fasting for 10-12 days. As for individuals with severe AP, it is recommended to continue fasting for 15 days or longer, until the pancreatic ductal system is completely sealed [11].

During the initial stages of AP, specifically in the enzymatic toxemia phase, our findings suggest that TPN provides a more effective method by reducing pancreatic stimulation. The ability of TPN to sustain metabolic processes without the addition of fats, which could worsen the condition, further reinforces its application during this crucial period [12]. The TPN group demonstrated a notable decrease in the occurrence of dynamic intestinal obstruction and a quicker return to normal serum amylase levels, highlighting the efficacy of TPN in managing the acute phase of the disease.

Although early introduction of EEN has been linked to various negative outcomes, it seems to have a positive impact during the purulent-necrotic phase. During this phase, which usually occurs after 10-12 days of the illness, there is a greater need for increased nutritional support to assist in the process of recovery. It proved to be an effective supplementary source of energy and nitrogen. The EEN was started with a well-balanced mixture, carefully calculated at 0.5 kcal/ml, up to a maximum of 1000 kcal. This was done alongside the infusion of protein-energy support, gradually increasing the volume and concentration of the product over a period of 2-3 days, until reaching the desired energy amount calculated at 35-40 kcal/kg of the patient's ideal body weight. Patients with purulent complications achieved a positive nitrogen balance only through radical necrosectomy or minimally invasive interventions such as puncture drainage or drainage through a mini-access. When primary radical operations were carried out for extensive purulent lesions in the presence of a prolonged negative nitrogen balance, the mortality rate was found to be high. Nevertheless, the initial decompression of purulent cavities through puncture or mini-access has been shown to alleviate intoxication, prevent multiple organ failure (MOF), address protein-energy deficiency, facilitate necrosis rejection, and lower mortality rates. An evaluation of the trophic status revealed that there were indicators of a protein-energy deficit even before the development of purulent necrotic lesions. These indicators included a daily loss of 0.4–0.8 kg of body weight, a reduction in skinfold thickness and arm muscle circumference, and increases in hypoalbuminemia, hypoproteinaemia, and lymphopenia, which were indicative of a deterioration in the T-cell immune system's functional state [13, 14]. EEN was discontinued only when a radical operation provided normal digestion with sufficient physical activity, adequate sleep, normal temperature, appetite, and haemoglobin and total protein levels at the lower limit of normal.

Our study highlights the importance of closely examining nitrogen losses and the potential for protein-energy deficiency in individuals with different severities of AP. In cases of mild AP, patients were able to maintain a positive nitrogen balance with relatively modest nutritional support. However, for patients with moderate to severe AP, more intensive intervention was necessary to achieve similar outcomes. The results emphasize the difficulties in managing severe acute pancreatitis, where significant nitrogen losses can greatly hinder recovery and potentially result in multiple organ failure if not properly addressed. Calculating the necessary compensation for nitrogen losses in severe AP would involve administering 11 Liters of solutions per day, which is highly impractical, along with a caloric intake that exceeds the body's ability to absorb [15].

The use of EEN was stopped once a radical procedure allowed for regular digestion along with enough physical activity, proper sleep, normal temperature, appetite, and levels of hemoglobin and total protein within the normal range. The timing and method of nutritional support are therefore pivotal. While TPN is recommended during the initial phases of AP, enteral nutrition should be cautiously introduced only during the purulent-necrotic phase, where it can support recovery without exacerbating the disease. Patients with moderate AP experienced gastrostasis, which resulted in the development of post-necrotic cavities and purulent complications in certain instances [16]. An integrated approach to treatment proved crucial in reducing postoperative mortality, as evidenced by the significant decrease observed with the early use of TPN, especially when combined with minimally invasive surgical strategies. On the other hand, the increased death rates seen with early surgical intervention during the enzymatic toxemia phase highlight the importance of caution and timing in both nutritional and surgical treatment.

The findings have significant clinical implications, indicating that TPN should be the preferred method of nutritional support in the early stages of AP, while EEN should be saved for the later purulent-necrotic phase. This approach not only enhances patient outcomes but also minimizes the risk of worsening the disease. Future research should prioritize the fine-tuning of timing and composition of nutritional interventions. It is crucial to investigate the potential advantages of combining TPN with selective early introduction of EEN in a controlled manner.

LIMITATIONS

It is required to note the limitations of this study. There is a possibility that the findings cannot be generalized to a larger population due to the observational aspect of the study and the relatively small sample size of the EEN group. In addition, although the study offers significant insights, it is necessary to conduct randomized controlled trials to validate these findings and create definite guidelines for the dietary management of AP.

CONCLUSION

This study emphasizes the importance of implementing a strategic approach to nutritional support in cases of acute pancreatitis. During the initial 5-14 days of AP, the effectiveness of Total Parenteral Nutrition (TPN) varies depending on the severity of the disease. Enteral tube feeding (EEN) is generally not advised in the early stages, but it may prove advantageous during the purulent-necrotic phase as a means of transitioning to oral feeding. Intestinal lavage plays a crucial role in inhibiting pancreatic secretion during the initial phases of acute pancreatitis. Various interventions, such as TPN, surgical procedures, and conservative management, have been shown to have a substantial effect in decreasing mortality rates. Implementing these strategies can lead to improved outcomes for patients with acute pancreatitis.

Table 1: Comparative Effects of TPN and EEN on Clinical Manifestations of AP

Comparative Criteria	Group I (TPN)	Group II (EEN)
Pain Relief	1–2 days	4–6 days or more
Hyperthermia Duration	Up to 4 days	Up to 14 days
Amylase Normalization	7–10 days	>10 days
Dynamic Intestinal Obstruction	Reduced	Progresses
Gastrostasis in Moderate AP	Absent or (< 3days)	Present (>3 days)
Inflammatory Infiltrate	Absent	32% of mild AP cases

Table 2: Comparative Analysis of Nutritional Support on Pancreatic Secretion in Patients with Pancreatic Fistulas

Nutritional Method	Relative Volume of Pancreatic Juice (%)	Direction of Secretion Suppression
Oral Nutrition	100%	
EEN with Balanced Mixtures	85%	
TPN with Glucose Solutions	5–7%	

REFERENCES

1. Dellinger R. P., Levy M. M., Carlet J. M. et al. Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008. Crit. Care Med. 2008; 36 (1): 296—327.
2. Jha RK, Ma Q, Sha H, Palikhe M. Acute pancreatitis: a literature review. Med Sci Monit. 2009;15(7):RA147–56. [PubMed] [Google Scholar] [Ref list]
3. Whitcomb DC. Clinical practice. Acute pancreatitis. N Engl J Med. 2006;354(20):2142–50. [PubMed] [Google Scholar] [Ref list]
4. McClave S. A., Chang W. K., Dhaliwal R., Heyland D. K. Nutrition Support in Acute Pancreatitis: A Systematic Review of the Literature. J. Parenter. Enteral Nutr. 2006; 30 (2): 143—156.
5. Singh N., Sharma B., Sharma M., Sachdev V., Bhardwaj P., Mani K., Joshi Y.K., Saraya A. Evaluation of early enteral feeding through nasogastric and nasojejunal tube in severe acute pancreatitis: a noninferiority randomized controlled trial. Pancreas. 2012; 41 (1): 153–159. DOI: 10.1097/MPA.0b013e318221c4a8
6. Lodewijkx PJ, Besselink MG, Witteman BJ, et al. Nutrition in acute pancreatitis: a critical review. Expert Rev Gastroenterol Hepatol 2016;10:571-80. [Crossref] [PubMed]
7. Oláh A, Romics L Jr. Enteral nutrition in acute pancreatitis: a review of the current evidence. World J Gastroenterol 2014;20:16123-31. [Crossref] [PubMed]

8. Stimac D, Poropat G, Hauser G, et al. Early nasojejunal tube feeding versus nil-by-mouth in acute pancreatitis: A randomized clinical trial. Pancreatology 2016;16:523-8. [Crossref] [PubMed]
9. Petrov MS, Kukosh MV, Emelyanov NV. A randomized controlled trial of enteral versus parenteral feeding in patients with predicted severe acute pancreatitis shows a significant reduction in mortality and in infected pancreatic complications with total enteral nutrition. Dig Surg 2006;23:336-44; discussion 344-5. [Crossref] [PubMed]
10. Gupta R, Patel K, Calder PC, et al. 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. [Crossref] [PubMed]
11. 4. Petrov M.S., van Santvoort H.C., Besselink M.G., van der Heijden G.J., Windsor J.A., Gooszen H.G. Enteral nutrition and the risk of mortality and infectious complications in patients with severe acute pancreatitis: a meta-analysis of randomized trials. Arch Surg. 2008; 143 (11): 1111–1117.
12. Dudrick SJ, Wilmore DW, Vars HM, Rhoads JE. Long-term total parenteral nutrition with growth, development, and positive nitrogen balance. Surgery. 1968;64(1):134-142.
13. 5. Huang H.H., Chang S.J., Hsu C.W., Chang T.M., Kang S.P., Liu M.Y. Severity of illness influences the efficacy of enteral feeding route on clinical outcomes in patients with critical illness. J Acad Nutr Diet. 2012; 112 (8): 1138–1146. DOI: 10.1016/j.jand.2012.04.013
14. Tenner S, Baillie J, DeWitt J, Vege SS American College of Gastroenterology. American College of Gastroenterology guideline: management of acute pancreatitis. Am J Gastroenterol. 2013;108:1400–1415; 1416.
15. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, Tsiotos GG, Vege SS Acute Pancreatitis Classification Working Group. Classification of acute pancreatitis--2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013;62:102–111.
16. Lugli AK, Carli F, Wykes L. The importance of nutrition status assessment: the case of severe acute pancreatitis. Nutr Rev. 2007;65:329–334.