



Hypertriglyceridemia With Total Parenteral Nutrition Versus Enteral Nutrition In Patients With Acute Pancreatitis

Thesis

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To God, who gave me everything I have in my life

To my Father whose prayers led me to my position today

To my wife who helped me complete this work

To the one whose precious warm soul shines all my days

and who helped encouraged and always supported me

My beloved Mother

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قالوا

سبحانك لا علم لنا
إلا ما علمتنا إنك أنت
العليم العظيم

صدق الله العظيم

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ABSTRACT

Background: Acute pancreatitis (AP) is an inflammatory condition of the pancreas that can cause local injury, systemic inflammatory response syndrome, and organ failure. Worldwide, AP is a common gastrointestinal condition and there is evidence that the incidence has been rising in recent years. AP is associated with substantial suffering, morbidity, and cost to the health care system.

Objective: Our study aims to compare between the effect of enteral (EN) and parenteral nutrition on serum triglycerides in patient with acute pancreatitis in intensive care unit of Ain Shams University (ICU). We found that enteral route is more safe than parenteral nutrition regarding serum triglycerides.

Patients and Methods: An informed written consent was obtained from patients and /or relatives who were included in this study. All patients were adults, more than 18 years old, admitted to ICU of Ain Shams University Hospital, either have acute pancreatitis on admission or acquired acute pancreatitis in ICU due to different causes in period of six months of approval of the protocol. Patients were divided into two groups: 30 patients started total parenteral nutrition (TPN) one day after admission consisting of 70% carbohydrate (in the form of dextrose 25%) and 30% fat (smoflipid 250 ml, 2 gm per ml). The other 30 patients started enteral nutrition rich in carbohydrates with low fat content one day after admission (peptamen) 6 scoops in 200 ml water every 2 hours, stopped from 12 am to 8 am. Serum triglyceride level was measured at first day of admission, then 3 days after admission, then one week after admission.

Results: Our study showed that TPN was associated with more increase of serum triglycerides level from first day of admission to 3 days after admission when compared with enteral nutrition, there is also increase in serum triglycerides from 3days to one week after admission, comparing the three periods together, there is increase in serum triglycerides with TPN group more than in enteral group.

Conclusion: The study showed that TPN was associated with higher mortality rate than EN, also there was an increase in serum triglycerides with TPN more than EN; suggesting that, enteral nutrition is a safer mode of nutrition in patients with acute pancreatitis.

KEYWORDS: ACUTE PANCREATITIS (AP), ENTERAL NUTRITION (EN).

INTRODUCTION

Acute pancreatitis (AP) is one of the most common inflammatory diseases of the gastrointestinal tract, with an increasing incidence in recent years. Though most patients with AP have a benign clinical course, approximately 10%–20% of patients develop severe AP with significant mortality of at least 30%. Disturbance of pancreatic blood flow, pancreatic ischemia and activated coagulation plays an important role in the development of AP as well as in the progression of the disease to severe necrotizing pancreatitis (*Hong et al., 2017*).

Acute pancreatitis (AP) is morphologically classified as either interstitial oedematous or necrotizing pancreatitis according to the revised Atlanta classification. Interstitial edematous pancreatitis is the most frequent type, whereas necrotizing pancreatitis represents the severe form of the disease. Necrotizing pancreatitis can be complicated by organ failure or secondary infection, both with a negative impact on patient outcome (*Verdonka et al., 2018*).

Due to pancreatitis the body suffers a hyper catabolic state where nutritional requirements increase at a time when intake is reduced, leading to an overall deficit. Thus, nutritional support is a crucial component of disease management. Historically, total parenteral nutrition (TPN) was provided to all patients with severe pancreatitis to meet their increased

requirements and to provide for pancreatic ‘rest’. More recently, researches demonstrated maladaptive changes in intestinal morphology and function, where TPN is the sole source of nutrition, and shown the protective role of enteral feeding in preserving intestinal mucosal integrity (*Deirdre et al., 2014*).

Parenteral nutrition can be used in acute pancreatitis as second-line therapy if nasojejunal tube feeding is not tolerated and nutritional support is required or if the nutritional goals cannot be reached with oral or enteral feeding (*Mirtallo et al., 2012*).

Most of the controversies in the field of parenteral nutrition in AP are related to the use of lipids and in particular long-chain triglycerides, because it is still unclear whether hyperlipidemia is a cause or a consequence of acute pancreatitis or a combination of both; however it is proven that the use of intravenous lipids in pancreatitis is safe if hypertriglyceridemia is avoided by keeping the serum levels within normal ranges (*Gianotti et al., 2009*).

In this study we aim to demonstrate the effect of enteral nutrition versus parenteral nutrition on the level of triglycerides in patients with acute pancreatitis.

AIM OF WORK

In this study we aimed to demonstrate the effect of enteral nutrition versus parenteral nutrition on the level of triglycerides in patients with acute pancreatitis.

Chapter 1

PATHOPHYSIOLOGY

Definition:

Acute pancreatitis is an inflammatory disorder of the pancreas causing mild to severe life threatening conditions. It is one of the most frequent gastrointestinal causes of hospital admission (*Ismail and Bhayana, 2017*).

Epidemiology:

Acute pancreatitis (AP) is the third most common ‘principal’ hospital discharge diagnosis for gastrointestinal (GI), liver, and pancreatic disease, following GI hemorrhage and cholecystitis (*Singh et al., 2017*).

Worldwide, the incidence of acute pancreatitis ranges between 5 and 80 per 100,000 populations, with the highest incidence recorded in the United States and Finland. Nearly 80% of mild to moderate pancreatitis recover spontaneously. However, overall 5%-20% patients progress to severe acute pancreatitis (SAP), which has a high risk of mortality (*Janisch and Gardnara, 2015*).

Classification:

There is a wide range of classifications for acute pancreatitis. The Revised Atlanta Classification 2012 classified acute pancreatitis according to the severity of the disease, morphology and temporal relation (*Working Group IAP/APA, 2013*).

A. Classification according to the severity of pancreatitis:

Acute pancreatitis is classified into three forms based on the severity (*Forsmark et al., 2016*).

1. **Mild acute pancreatitis:** which is characterized by the absence of organ failure and local or systemic complications.
2. **Moderately severe acute pancreatitis:** which is characterized by transient organ failure (resolves within 48 hours and without persistent organ failure >48 hours) and/or local or systemic complications.
3. **Severe acute pancreatitis:** which is characterized by persistent organ failure that may involve one or multiple organs.
4. **Critical acute pancreatitis:** this stage is identified by determinant based classification. It is characterized by the presence of infected peri-pancreatic necrosis and persistent organ failure.

The following table shows Revised Atlanta Classification in comparison to the old one and the Determinant based classification:

Table (1): Classification of pancreatitis according to severity
(Lankisch et al., 2015)

	Atlanta classification 1992 ⁴⁵	Revised Atlanta classification 2012 ⁴⁶	Determinant-based classification 2012 ⁴⁷
Mild	No organ failure and no local complications	No organ failure and no local or systemic complications	No (peri)pancreatic necrosis and organ failure
Moderately severe	..	Transient organ failure (<48 h) and/or local or systemic complications without persistent organ failure (>48 h)	Sterile (peri)pancreatic necrosis and/or transient organ failure (<48 h)
Severe	Local complications and/or organ failure: PaO ₂ ≤60% or creatinine ≥152.6 μmol/L or shock (systolic blood pressure ≤60 mm Hg) or gastrointestinal bleeding (>500 mL/24 h)	Persistent organ failure (>48 h):* single organ failure or multiple organ failure	Infected (peri)pancreatic necroses or persistent organ failure (>48 h)
Critical	Infected (peri)pancreatic necroses and persistent organ failure

Neither Atlanta classifications have a fourth critical group; this group is solely in the determinant-based classification. *Persistent organ failure is now defined by a modified Marshall score (appendix).⁴⁸

B. Classification according to the phases of pancreatitis:

Temporally, there are three types of acute pancreatitis as follows

(i) Early-first week:

Only clinical parameters are important for treatment planning, and is characterized by a complex inflammatory reaction. The course of AP starts with a systemic proinflammatory phase systemic inflammatory response syndrome (SIRS), followed by a mixed inflammatory response syndrome mixed antagonist response syndrome (MARS) and finally leads to a phase with a suppressed inflammatory response compensatory anti-inflammatory response syndrome (CARS).

(ii) Late-after the first week:

Following the early phase, AP moves towards the second phase, including SIRS, sepsis, local and systemic complications, persistent organ failure, and possibly death (Phillip et al., 2014).

C. Classification according to the morphology of pancreatitis:

Morphologically, there are three types of acute pancreatitis as follows:

- (i) Acute oedematous (interstitial) pancreatitis.
- (ii) Acute necrotizing pancreatitis.
- (iii) Haemorrhagic pancreatitis.