Updates on Enteral versus Parenteral Nutrition in Pancreatitis

Essay

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By

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Amr Mohammed Sharaf El-Din

List of Contents

List of Abbreviations

Abbrev.	*Full-term*
ABW	: Actual body weight
AIP	:Autoimmune pancreatitis
AjBW	: Adjusted body weight
ALT	: Alanine aminotransferase
AP	: Acute pancreatitis
APACHE	: Acute Physiology and Chronic Health Evaluation
ARDS	: Acute respiratory distress syndrome
AST	: Aspartate aminotransferase
CARS	: Compensatory anti inflammatory response syndrome
CBC	: Complete blood count
CMV	:Cytomegalovirus
CPGs	: Clinical practice guidelines
CRP	: C-reactive protein
CT	: Computed tomography
EBV	: Epstein-Barr virus
EEN	: Early enteral nutrition
ERCP	: Endoscopic retrograde cholangiopancreatography
EUS	: Endoscopic ultrasonography
GALT	: Gut-associated lymphoid tissue
GI	: Gastrointestinal
HPA	: Hypo thalamic- pituitary-adrenal

IBW : Ideal body weight

ICU : Intensive care unit

IL : Interleukin

IV : Intravenous

KUB : Kidneys-ureters-bladder

LDH : Lactic dehydrogenase

MCP-1 : Monocyte chemotactic protein 1

MRCP : Magnetic resonance cholangio-pancreatograply

NALT : Nasal-associated lymphoid tissue

NLR : Neutrophil-lymphocyte ratio

NSAID : Non-steroidal anti-inflammatory drug

PCM : Protein-Calorie Malnutrition

PMN : Polymorphonuclear

RCTs : Recent controlled trials

REE : Required energy expenditure

ROS : Reactive oxygen species

SIRS : Systemic inflammatory response syndrome

TAP : Trypsinogen activation peptide

TNF-α : Tumor necrosis factor-alpha

VZV : Varicella-zoster virus

List of Tables

List of Figures

Introduction

Acute pancreatitis is one of the most common diseases of the digestive system leading to large physical and economic burdens ***(Peery et al., 2012).***In the majority of patients, acute pancreatitis runs a mild clinical course. However, in patients who develop necrotizing pancreatitis, mortality rate increases ***(van Santvoort et al., 2011)***.

Pancreatitis is a potentially fatal disease that requires nutritional support which is considered a primary issue in the therapy of the disease ***(Chang et al., 2013)***.

Optimal nutritional support in acute pancreatitis has been a subject of debate for decades. Initially, the concept of pancreatic rest by fasting was thought to improve outcome because enteral nutrition was believed to aggravate inflammation through pancreatic stimulation. Subsequently, parenteral nutritional support was believed to avoid pancreatic stimulation and provide the needed nutritional components.***(Lodewijkx,et al.,2016)***

Recently, many trials on enteral nutrition were performed showing a benefit of enteral nutrition, which became superior to parenteral nutrition, although several limitations should be taken into account ***(Tenner, et al., 2013)***.

The optimal route of enteral nutrition remains unclear, but normal or nasogastric tube feeding seems safe when tolerated. When nasogastric nutrition is not tolerated, or when the caloric need is not reached, nasojejunal feeding tube located beyond Treitz' ligament is recommended***(Gerritsen,et al.,2015).***

The clinical course of acute pancreatitis varies from a mild transitory form to a severe necrotising disease. Most episodes of acute pancreatitis (80%) are mild and self limiting, subsiding spontaneously within 3–5 days. Patients with mild pancreatitis respond well to medical treatment, requiring little more than intravenous fluid resuscitation and analgesia. In contrast, severe pancreatitis is defined as pancreatitis associated with organ failure and/or local complications such as necrosis, abscess formation, or pseudocysts***(Yokoe, et al., 2015).***

The management of acute pancreatitis has been controversial over the past decades, varying between a conservative medical approach on the one hand and an aggressive surgical approach on the other. There has been great improvement in knowledge of the natural course and pathophysiology of acute pancreatitis over the past decade ***(Carter, et al., 2017).***

Aim of the Essay

The aim of the essay is to highlight the role of enteral nutrition in comparison with parenteral nutrition in pancreatitis.

Chapter (1):
Diagnosis of Pancreatitis

Acute pancreatitis (AP) is one of the most common diseases of the gastrointestinal tract, leading to tremendous emotional, physical, and financial human burden. An increase in the annual incidence for AP has been observed in most recent studies ***(Peery et al., 2012).***

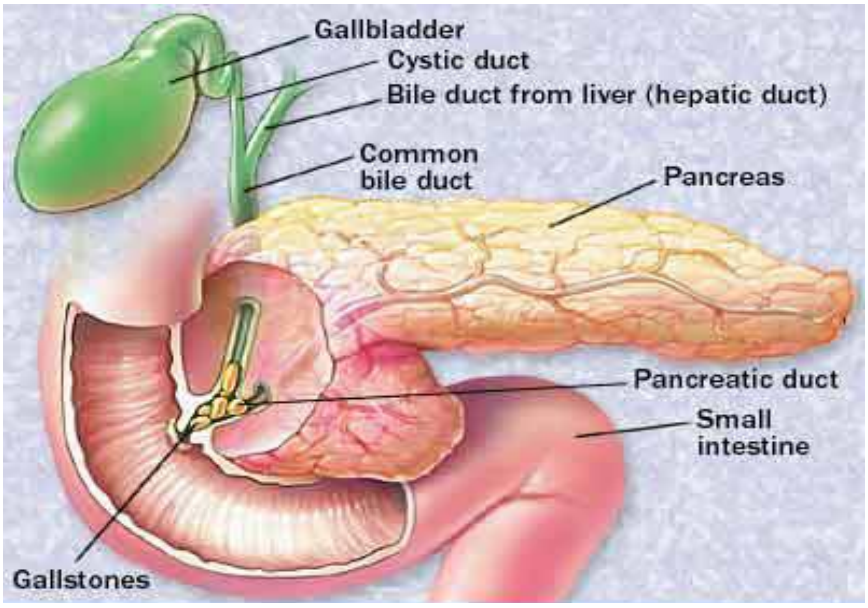

Figure (1): Position and structure of pancreas***(Paulet al.,2015)***

- **Function of the pancreas**

Classically, the pancreas has two roles,an "endocrine" role, relating to the secretion of insulin and other substances within pancreatic islets helping control blood sugar levels and metabolism within the body, and an "exocrine" role,

relating to the secretion of enzymes involved in digesting substances from outside of the body***(Paulet al.,2015).***

Sugar control and metabolism are controlled by Approximately 3 million cell clusters called pancreatic islets which are present in the pancreas, within these islets are four types of cells which are involved in the regulation of blood glucose levels. Each type of cell secretes a different type of hormone:(1) α alpha cells secrete glucagon (increase glucose in blood),(2) β beta cells secrete insulin (decrease glucose in blood),(3) δ delta cells secrete somatostatin (regulates/stops α and β cells) and (4) PP cells, or γ (gamma) cells, secrete pancreatic polypeptide (affect satiety). These act to control blood glucose through secreting glucagon to increase the levels of glucose, and insulin to decrease it***(Paul et al.,2015).***

Also the pancreas plays a vital role in the digestive system. It secretes the pancreaticfluid that contains digestive enzymes into the duodenum. These enzymes help to break down carbohydrates (usually starch), proteins and lipids. Cells are arranged in clusters called acini. Secretions into the middle of the acinus accumulate in intralobular ducts that drain to the main pancreatic duct, which drains directly into the duodenum. The cells are filled with granules containing the digestive enzymes. These are secreted in an inactive form called (zymogens or proenzymes). When released into the

duodenum, they are activated by the enzyme enteropeptidase present in the lining of the duodenum. The proenzymes are cleaved, creating a cascade of activating enzymes: enteropeptidase activates the proenzymetrypsinogen by cleaving it to form trypsin. The free trypsin then cleaves the rest of the trypsinogen, as well as chymotrypsinogen to its active form chymotrypsin ***(Hall&John2011).***

The pancreatic enzymes include Proteases, for the digestion of proteins, (trypsinogen and chymotrypsinogen). Lipase fotthe digestion of fats, andAmylase, for the digestion carbohydrates. The pancreas also secretes phospholipase A2, lysophospholipase, and cholesterol esterase. Secretion of these proenzymes is via the hormones gastrin, cholecystokinin and secretin, which are secreted by cells in the stomach and duodenum in response to distension and/or food***(Hall&John 2011).***

- **Pathophysiology of AP**

Pathophysiology of AP encompasses complex cascaded events of acinar cell inflammation, involvement of immune system, and systemic pathological outcomes ***(Shamoon,2016).***

Premature activation of intra-acinar digestive zymogens is one of the early hallmarks of AP. The resultant autodigestion of pancreasleads to release of pro-inflammatory mediators such

as tumor necrosis factor-α, interleukin (IL)-1β, IL-6, which intermingle with microcirculation, causing increased vascular permeability, edema, hemorrhage, and necrosis of pancreas ***(Ramnath,et al.,2010).***

Profound acinar cell injury and amplified inflammatory responses give rise to systemic inflammatory response syndrome (SIRS) and multiple organ dysfunction syndrome (MODS), ultimately responsible for AP-associated mortality. The immune system is thought to play an important role in the disease patho-genesis of AP. Complex immunological events underlie progres-sion of AP ***(Zheng,2013).***

Dysregulated immune responses during AP include increased leukocyte counts, migration and activation of pro-inflammatory innate immune cells (neutrophils and mac-rophages) as well as depletion of T-lymphocytes and raised levels of plasma pro-inflammatory cytokines ***(Shamoon,2016).***

- ## Etiology of acute pancreatitis

Many causes of acute pancreatitis have been discovered. The main causes are gallstone migration and alcohol abuse. Other causes are uncommon, situational, controversial, or have not yet been completely elucidated***(Bruno,2008).***

1- Obstruction of the common bile duct by stones (38%)