

Management of Gastrointestinal Failure in ICU

An Essay

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Intensive Care

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List of Abbreviations

Ach:	acetylcholine
AP:	acute pancreatitis
APACHE II:	Acute Physiology and Chronic Health Examination II:
APCWG:	acute pancreatitis Classification Working Group
APFC:	Acute peripancreatic fluid collections
aPTT:	activated partial thromboplastin time
ARDS:	acute respiratory distress syndrome
ASBO:	acute small bowel obstruction
CCK:	cholecystokinin
CCK:	cholecystokinine
CECT:	contrast enhanced computerized tomography
CGRP:	calcitonin gene-regulated peptide
CT:	Computed topography
DDI:	2',3'-dideoxyinosine
EGF :	epidermal growth factor
ERCP:	Endoscopic retrograde cholangiopancreatography
EUS:	endoscopic ultrasound
FGF :	fibroblast growth factor
GABA:	gamma butyric acid
GRP:	gastrin releasing polypeptide
HGF :	hepatocyte growth factor
IBS:	irritable bowel syndrome
IGF-I/II :	growth factor I/II
INN:	lenomorelin
KGF :	keratinocyte growth factor
MMC:	migrating motor complexe
MRCP:	Magnetic retrograde cholangiopancreatography
MRI:	Magnetic resonance imaging
NK1:	neurokinin1
NO:	nitric oxide
NPO:	nothing by mouth
NPY:	neuropeptide Y
OR:	operating room
ORS:	Oral rehydration solutions
PACAP:	pituitary adenylate cyclase activating , polypeptide
PCD:	Percutaneous catheter drainage
PGE2:	prostaglandin E2
PHI:	peptide histidine isoleucine
PNPFC:	Post-necrotic pancreatic/peripancreatic fluid collections

POI:	post-operative ileus
PT:	prothrombin time
PYY:	peptide YY
SBFT:	Small Bowel Follow Through
SBO:	Small bowel obstruction
SBS:	short bowel syndrom
SIBO:	Small intestinal bacterial overgrowth
SIRS:	systemic inflammatory response syndrome
SK:	substance K
SP:	substance P
TGF :	transforming growth factor (alfa and beta)
TRH:	thyrotropin-releasing hormone
US:	ultrasonography
VIP:	vasoactive intestinal polypeptide.
WON:	Walled-off necrosis

Introduction

The gut is considered to play a significant role in the processes of systemic inflammation, sepsis and multiple organ dysfunction syndrome following hemorrhagic shock, trauma, burns, pancreatitis, major abdominal operations, and in critically ill patients in general (**Mittal and Coopersmith 2014**).

The concept of gut as a great player in critical illness dates back to the 1940s, when live enteric bacteria were found in the peritoneal washings of dogs after hemorrhagic shock. The phenomenon of bacterial passage through the intestinal wall termed as bacterial translocation. (**Sertaridou *et al.*, 2015**)

Delayed gastric emptying, noted in around 50% of mechanically ventilated ICU patients, leads to intolerance to nasogastric delivery of food, bacterial overgrowth in the upper GI tract, gastric colonization and an elevated risk for pulmonary aspiration and ICU-acquired infection (**Ritz *et al.*, 2000**)

Abnormal small bowel motility also causes abdominal distention with a risk of respiratory insufficiency, or osmotic diarrhea leading to hypovolemia, incomplete absorption and negative nitrogen balance. Furthermore, digestion and absorption may additionally be impaired by the small intestinal motor

dysfunction and the damaged mucosal structure. (**Ritz et al., 2000**)

It has been recognized that, apart from the intestinal ischemia-reperfusion injury, gut luminal contents, including the mucus gel layer, pancreatic proteases and gut flora, as well as the luminal response to splanchnic ischemia play also an important role in modulating gut injury (**Deitch and Ulloa, 2010**).

Luminal pancreatic proteases appear to be crucial for the development of gut-derived sepsis following hemorrhagic shock , while bile-derived tumor necrosis factor- α seems to act on the luminal side of the mucosa in the endotoxin-induced gut injury model, causing intestinal damage (**Sertaridoua et al., 2015**).

Multiple organ failure is a major cause of morbidity and mortality in critically ill patients (**Gregory et al 2010**). The influence of gastrointestinal failure development on mortality was tremendous .The risk of death showed a twenty-three fold increase. The patients with gastrointestinal failure have ICU mortality of 20.1% compared to 10.8% in patient without gastrointestinal failure. On admission only 20% of all gastrointestinal failure cases were diagnosed, while 82% of gastrointestinal failure cases were clinically manifested by the end of the first week in ICU. (**Annika, 2008**)

Gastrointestinal failure should be considered a relevant clinical predictor of increased mortality and prolonged ICU stay. Several risk factors for the development of gastrointestinal failure could be identified. **(Gideon P., 2008)**

Acute pancreatitis is one of the most common gastrointestinal disorders requiring acute hospitalization worldwide, with a reported annual incidence of 13 - 45 cases per 100,000 persons .**(IAP/APA guidelines, 2012)**. Aggressive resuscitation may prevent the onset of lethal multi system organ failure. Ranson criteria (table 5) are used to grade the severity of acute pancreatitis **(Gideon P., 2008)**.

Bowel obstruction are common among critically ill patients and may be the underlying reason for intensive care admission or may be develop as a part of another disease . Obstructions of the small bowel may be mechanical or paralytic. Recent abdominal surgery is a Principal cause of Adynamic ileus among critical care patients. Up two thirds of intensive care unit patients are affected predominantly with disorders gastric and colonic motor dysfunction. **(Gregory et al., 2010)**

Acute mechanical bowel obstruction constitutes a major cause of morbidity and financial expenditure in hospitals around the world and a significant cause of admissions to emergency surgical departments. **(Markogiannakis et al., 2007)**

Short bowel syndrome is a disabling malabsorptive condition associated with a high frequency of complications and high utilization of healthcare resources. (**Parrish et al., 2014**) The exact prevalence of short bowel syndrome in the United States is unknown but has been estimated at 10,000–15,000 patients. Many patients with short bowel syndrome require long-term parenteral nutrition and/or intravenous fluids. (**Matarese, 2012**)

Gastrointestinal motility disorders are common in critically ill patients, occurring mainly because of physiological responses to the severe disease itself, enteral nutrition intolerance, use of medications (particularly antibiotics and prokinetics), infection, and immunosuppression. Diarrhea is the most frequent complication among the changes observed in intensive care unit patients receiving enteral nutrition. Among other factors, diarrhea can be caused by alterations in the colonic response, microbial contamination of enteral nutrition formulas, low-fiber diet, hypoalbuminemia, disturbances of the intestinal flora, increased use of antibiotics and concurrent drug therapy, and *Clostridium difficile* infection. (**Lordani et al., 2014**)

Diarrhea is frequently observed in ICU patients, but the reported prevalence differs according to the definition and the setting, between 2 and 95%. Diarrhea is frequently reported in the ICU. Little is known about diarrhea incidence and the role of the different risk factors alone or in combination. In a study done by *Thibault et al., 2013* to determine the risk factors for diarrhea in ICU it was focused on contribution of feeding, antibiotics and antifungal drugs. (**Thibault et al., 2013**)

Aim of the essay

Is to discuss different causes of gastrointestinal failure management within ICU and how to prevent and diagnose as well as its proper management to improve ICU outcome

Chapter I

Physiology of the GIT

The gastrointestinal tract is a continuous tube that extends from the mouth to the anus. Its primary function is to serve as a portal whereby nutrients and water can be absorbed into the body. In fulfilling this function, the meal is mixed with a variety of secretions that arise from both the gastrointestinal tract itself and organs that drain into it, such as pancreas, gallbladder, salivary glands. Likewise, the intestine displays a several patterns of motility that cause mixing the meal with digestive secretions and move it along of the length of the gastrointestinal tract (Windmaier, 2004)

Splanchnic circulation

The blood flow to the stomach, intestines, pancreas, and liver is arranged in a series of parallel circuits, with all the blood from the intestine, pancreas, and spleen drains through portal vein to the liver and from the liver via hepatic veins to the inferior vena cava. The viscera and the liver have about 30% of the cardiac output via the celiac, superior mesenteric and inferior mesenteric arteries (Scott-Douglas et al., 2002)

Gut mucosal barrier (GMB)

Gut mucosal barrier is a multi-tiered defense against potentially lethal systemic invasion by intestinally contained microorganisms, which are potentially lethal to the host. There are three types

1st is Physical barrier function:

Mechanical barrier: in which the enterocyte's lipid membrane, bonded to adjacent cells by tight junctions, is a physical barrier to bacterial invasion. The orderly turnover of healthy cells is crucial to the maintenance of the barrier. Coordinated intestinal motility also common disturbed in ICU patient is not only essential to stimulate cellular turnover, but also maintains the flow of bacterial toxins the digestive tract

Chemical and enzymatic :A gastric pH <4.5 is antibacterial. Intestinally produced mucus and secreted humoral factors protect the host from bacterial translocation. Like bile salts mucin secreted through gastrointestinal tract non specifically bind bacteria and endotoxins.

2nd is immunological barrier :

Local; the intestinal mucosa and submucosa are richly inhabited by macrophages, lymphocytes, neutrophils, and mast

cells. IgA binds bowel bacteria , induce bacteriolysis , prevents bacterial adherence to the mucosal surface .

Systemic that the bacteria and toxins migrate across the Gut mucosal barrier enter the portal circulation, and filter through the Kupffer cell macrophage bed of the liver. After sampling the portal antigens, these cells are involved in the mediation of systemic immune responses

3rd is bacteriologic barrier:

The maintenance of normal gut microbial ecology is critical in the prevention of enteric autoinfection. The intestinal micro flora not only preserves barrier integrity and enhances mechanical function by stimulating and nourishing the mucosal cells but also acts to prevent over growth and colonization by potentially harmful organism.

(Evance, 2005).

Regulation of Gastrointestinal function

There are three main modalities for gastrointestinal regulation that operate in a complementary fashion to ensure that function is appropriate. First, endocrine regulation is mediated by the release of hormones by triggers associated with the meal. These hormones travel through the bloodstream to change the activity of distant segment of gastrointestinal tract, an organ

draining into it (e.g.: the pancreas), or both. Second some similar modulators are not sufficiently stable to persist in the bloodstream, but instead alter the function of cells in the local area where they are released, in a Paracrine fashion. Finally the intestinal system is endowed with extensive neural connections. These include the connections to central nervous system (extrinsic innervations, but also the activity of a largely autonomous (enteric nervous system) that comprises both sensory and secreto-motor neurons. The enteric nervous system integrates central input to the gut. But can also regulate gut function independently in response to changes in the luminal environment. In some cases, the same substance can mediate regulation by endocrine, Paracrine, and neurocrine pathways (e.g.: cholecystokinin) (**Hansen 2003**).

Hormones/paracrine:

Biologically active polypeptides that are secreted by nerve cells and gland cells in the mucosa act in a Paracrine fashion, but they also enter the circulation. Measurement of their concentration in blood after a meal has removed the shades on the roles these gastrointestinal hormones play in the regulation of gastrointestinal secretion and motility (**Nakazato, 2001**)

The hormonal influence and the interplay with enteric nervous system, takes place after and in between meals. The post prandial response includes release of insulin, neurotensin, cholecystokinin (CCK), gastrin glucagon like peptide (GLP-1 and