Bacterial Translocation

Essay
Submitted for Partial Fulfillment of Master Degree in
Intensive Care Medicine

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2011

إعادة الإستيطان الباكتيرى

رسالة توطئة
للحصول على درجة الماجستير في الرعاية المركزة
مقدمة من

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Summary

Delayed sepsis, systemic inflammatory response syndrome (SIRS) and multiorgan failure remain major causes of morbidity and mortality in intensive care units. One factor thought to be important in the aetiology of SIRS is failure of the intestinal barrier resulting in bacterial translocation and subsequent sepsis.

There would seem to be little doubt that gut function in general, and intestinal barrier function in particular, are important determinants of outcome in critically ill patients. Methodological problems in confirming bacterial translocation, which is a direct measure of intestinal barrier function, has restricted investigations to patients undergoing laparotomy, and as such there is only limited data available relating to specific interventions that might preserve intestinal barrier function or limit bacterial translocation.

Based on the best currently available knowledge, glutamine supplementation, aggressive and targeted nutritional intervention, maintaining good splanchnic flow whilst limiting other inotropic support, the judicious use of antibiotics and directed SGD (Selctive gut decontamination) regimes hold some promise of limiting bacterial translocation. Future potential in decreasing

List of Contents

List of Abbreviations

BT	**:**	Bacterial translocation
AF	**:**	Ascetic fluid
Ang4	**:**	Angiogenin 4
ARDS	**:**	Acute respiratory distress syndrome
ARF	**:**	Acute renal failure
CRH	**:**	Corticotropin-releasing hormone
DSS	**:**	Dextran sulfate sodium
EN	**:**	Enteral nutrition
FBC	**:**	Full blood count
GALT	**:**	Gut-associated lymphoid tissue
GIT	**:**	Gastrointestinal tract
IBD	**:**	Inflammatory bowel disease
IBS	**:**	Irritable bowel syndrome
IgA	**:**	Immunoglobulin A
IGF-1	**:**	Insulin-like growth factor 1
IIR	**:**	Intestinal ischemia-reperfusion
IL	**:**	Interleukin
IO	**:**	Intestinal Obstruction
LBT	**:**	Lactulose breath test
LDH	**:**	Lactate dehydrogenase
LERS	**:**	Leukocyte esterase reagent strip
LPS	**:**	Lipopolysaccharides
MHE	**:**	Minimal hepatic encephalopathy

MLNs	**:**	Mesenteric lymph nodes
MMC	**:**	Migrating motor complex
MOF	**:**	Multiple organ failure
NASH	**:**	Nonalcoholic steatohepatitis
NO	**:**	Nitric oxide
NOD	**:**	Nucleotide-binding oligomerization domain
PCR	**:**	Polymerase chain reaction
PMN	**:**	Polymorphonuclear neutrophil
RCTs	**:**	Randomized control trials
SBP	**:**	Spontaneous bacterial peritonitis
SGD	**:**	Selective gut decontamination
SIBO	**:**	Small intestinal bacterial overgrowth
SIRS	**:**	Systemic inflammatory response syndrome
TLRs	**:**	Toll-like receptors
TPN	**:**	Total parenteral nutrition

List of Tables

List of Figures

Before all, Thanks to Allah

I would like to express my profound gratitude to **Prof. Dr. Mohammed Abd Al khalek Mohammed** Professor of Anesthesiology and Intensive Care Medicine, Ain Shams University for his most valuable advises and support all through the whole work and for dedicating much of his precious time to accomplish this work.

I am also grateful to **Dr. Ehab Hamed Abd El Salam** Assistant Professor of Anesthesiology and Intensive Care Medicine, Ain Shams University for his unique effort, considerable help, assistance and knowledge he offered me throughout the performance of this work.

My special thanks and deep obligation to **Dr. Sherif George Anis** Lecturer of Anesthesiology and Intensive Care Medicine, Faculty of Medicine, Ain Shams University for his continuous encouragement, supervision and kind care.

بسم الله الرحمن الرحيم

قَالُوا سُبْحَانَكَ لاَ عِلْمَ لَنَا إِلاَّ مَا عَلَّمْتَنَا
إِنَّكَ أَنتَ الْعَلِيمُ الْحَكِيمُ

صدق الله العظيم

البقرة الآية ٣٢

Introduction

Bacterial translocation (BT) is defined as the passage of viable and nonviable bacteria and bacterial products through the gut mucosal barrier to extra-intestinal sites such as the mesenteric lymph nodes (MLNs), blood, liver, and spleen. This process results in damage of the mucosal barrier, and a deficiency in the immune system of the host. After trauma, thermal injury, intestinal obstruction, hemorrhagic shock, intestinal transplantation, or abdominal irradiation, the gut mucosa cannot act as a strong barrier against the passage of intestinal bacteria and BT may occur. Under the pathophysiological conditions of critically ill patients, it stands to reason that the gut mucosal barrier and immunological functions may be impaired after the dissemination of pathogenic substances into the systemic circulation, which can trigger the pathophysiological changes causing sepsis and multiple organ failure **(Guarner et al., 2003).**

The conventional research methods for bacterial translocation and intestinal permeability are unfeasible or too complicated to be performed in clinical trial. The polymerase chain reaction (PCR) analysis has a higher sensitivity than blood and mesenteric lymph nodes (MLN) cultures for assessing BT from the intestine. It is a

promising tool in detecting BT in clinical trial **(Kane et al., 1998).**

Various treatment modalities have been proposed to prevent the occurrence of BT resulting from gut barrier damage. It is well known that immunonutrition supports both the immunological and metabolic functions of the gut, and reduces gut mucosal damage and bacterial translocation. Giving patients an enteral diet enriched with immunomodulatory components such as arginine, glutamine, and omega-3 fatty acids has been shown to reduce the incidence of septic complications and the hospital stay in critically ill patients **(Feryal et al., 2005).**

One such treatment involves probiotics, which are defined as food or drugs containing live microbes that have a beneficial physiological effect on the host. Previous studies suggest that lactic acid bacteria stimulate the immune system, exert a protective effect against invasive pathogens, and control intestinal bacterial overgrowth **(Chiva et al., 2002).**

Aim of the Work

The aim of the work is to highlight the pathogenesis of bacterial translocation and its complications and how to deal with.

Physiology of Intestinal Barrier

The intestinal surface epithelia have evolved several key functions that facilitate a peaceful coexistence with luminal microbial populations while maintaining immunological vigilance against invading microbes. These functions include down-regulation of pro-inflammatory signaling pathways, expression of key antimicrobial proteins that actively defend epithelial surfaces, and initiation of epithelial repair after mucosal injury. The resident bacteria play an important role in shaping these functions. These findings underscore the central role of commensal microbes in the development and maintenance of epithelial barrier integrity and suggest that disrupting these host-bacterial associations with broad-spectrum antibiotics may lead to disease **(Anisa et al., 2005).**

Cellular makeup of the intestinal epithelial barrier

The intestine's internal tissues are separated from the microbe filled lumen by a single epithelial layer that is only 20 μm thick. Far from being a homogeneous cell population, however, gut epithelial surfaces are composed of several distinct cell types, each of which contributes in a unique way to mucosal defense and the maintenance of barrier integrity.

The enterocyte is the most abundant cell type at both small and large intestinal epithelial surfaces (Fig. 1).

Enterocyte membranes, as well as the tight junctions that are formed between these cells, form an important physical barrier to microbial penetration. However, enterocytes also assume a more active role in defending epithelial surfaces by secreting a variety of antimicrobial proteins. These include two families of small, highly conserved antimicrobial peptides: α-defensins and cathelicidins. Members of both families exhibit broadspectrum antimicrobial activity against bacteria, fungi, and protozoa by disrupting microbial membranes **(Zasloff et al., 2002).**

Gut surfaces harbor other less-abundant epithelial cell lineages that also help to protect mucosal surfaces from bacterial encroachments. Goblet cells, found in both the small and large intestines, secrete large quantities of mucin, which is composed of highly glycosylated proteins that form a protective layer of gel-like mucus over the surface epithelium.

In the small intestine, Paneth cells are the key effectors of antimicrobial defense. These specialized epithelial cells are situated at the base of small intestinal crypts (Fig. 1) and harbor secretory granules containing several microbicidal proteins including α-defensins, lysozyme, and phospholipase A2. Paneth cells sense bacterial proximity by an unknown mechanism and react by discharging their microbicidal granule contents into the gut lumen **(Ayabe et al., 2000).**