Impact of Stress Ulcer in Morbidity and Mortality in Critically Ill Patients

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By

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Contents

List of abbreviations

CAP: community acquired pneumonia

CDAD: Clostridium difficile–associated diarrhea

C.difficile:clostridium difficile

CIB: clinically important bleeding

CO: cardiac output

COX-2: cyclooxygenase-2

CRF: corticotropin-releasing factor

CYP: cytochrome P450

ECL cells: enterochromaffin-like cells

EGF: epidermal growth factor

FDA: Food and Drug Administration

GI: gastrointestinal

GIP:gastric inhibitory peptide

H2RA: histamine-2 receptor antagonist

ICU: intensive care unit

iNOS: inducible NO synthase

IR: Immediate-release

IVAC: infection-related ventilator-associated complications

LODT: lansoprazole oral disintegrating tablet

MAPKs: mitogen-activated protein kinases

MODS: Multiple Organ Dysfunction Score

MRSA:methicillin resistant staphylococcus

NO: nitric oxide

NSAIDS:non steroidal anti inflamatory drugs

PEEP:positive end expiratory pressure

PG:prostaglandins

PPIs: proton pump inhibitors

ROS: reactive oxygen species

SRMD: Stress-related mucosal disease

SUP:stress ulcer prpphylaxis

UGI: upper gastrointestinal

VAP:ventilator associated pneumonia

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List of figures

List of tables

Introduction

Sterss-induced gastritis, also referred to as stress-related erosive syndrome, stress ulcer syndrome, and stress-related mucosal disease can cause mucosal erosions and superficial hemorrhages in patients who are critically ill or in those who are under extreme physiological stress restuling in minimal to severe gastrointestinal blood loss and leading to blood transfusion if not addressed. Patients who may have an increased risk of stress gastritis are those with massive burn injury, head injury associated with raised intracranial pressure, sepsis with positive blood culture results, severe trauma and multiple system organ failure. **(Reveiz et al ., 2009).**

Two entites are thought to normally play a role in the breakdown of the mucosal barrier: gastric acid secretion and defense mechanisms. With stress gastritis, gastric acid secretion is invariably either normal or decreased. Thus, acid hypersecretion is not a significant etiological factor; instead, the breakdown of the mucosal defense mechanism is the primary cause. The defense mechanisms particularly the mucous secretion tend to have a decrease in bicarbonate concentration

and therefore are unable to buffer the proton in the stomach. **(Yardley, 2001).**

Stress causes decreased blood flow to the mucosa leading to ischemia with subsequent destruction of the mucosal lining. **.(Herzig, et al , 2011).**

Of patients who are critically ill, 6% have overt bleeding, while fewer than 2-3% have clinically significant hemorrhage. Endoscopy has revealed evidence of intraepithelial hemorrhage in 52-100% of patients in the ICU within 24 hours of the onset of the stressor. **(Irwin , et al.,2001).**

Mortality/morbidity figures are high in older patients because of several factors including atherosclerosis that leads to reduced blood supply and impaired host defenses. The severity of the injury leads to a further reduction in blood flow to the gastrointestinal tract thereby resulting in further compromise of the mucosal barrier and an increased risk of gastritis. The presence of Helicobacter pylori may also contribute to the mucosal barrier breakdown and lead to stress gastritis. **(Constantin, et al., 2009).**

The mortality rate for critically ill patients with or without stress ulcer bleeding varies in large part with the type and severity of the underlying disease. Although mortality in ICU patients with stress ulcer bleeding to mortality is unclear. An earlier natural history study from a medical ICU that did not use prophylaxis found that the overall mortality rate for patients who bleed was 90% whereas it was only 13% for non-bleeders. **(Mulla, et al., 2001).**

Aim of the work

The aim of this work is to discuss normal defense mechanim against ulceration, pathophysiology, diagnosis, management of stress ulcer and its impact in morbidity and mortality in critically ill patients.

Normal defense mechanism against ulceration

Anatomical sonsideration:

Stomach and abdominal esophagus:

The stomach is the widest part of the alimentary tract and lies between the esophagus and the duodenum. It is situated in the upper abdomen, extending from the left upper quadrant downwards, forwards and to the right, lying in the left hypochondrium, epigastric and umbilical areas. It occupies a recess beneath the diaphragm and anterior abdominal wall bounded by the upper abdominal viscera on either side **(Harold, 2005).**

Its mean capacity increases from 30 ml at birth, to 1000 ml at puberty, to 1500 ml in adults. The peritoneal surface of the stomach is interrupted by the attachments of the greater and lesser omenta, which define the greater and lesser curvatures separating two surfaces **(Romanes, 2002).**

Parts of the stomach:

The stomach is divided for descriptive purposes into the fundus, body, pyloric antrum and pylorus, by arbitrary lines drawn on its external surface. The internal appearance and

microstructure of these regions varies to some degree **(Harold, 2005).**

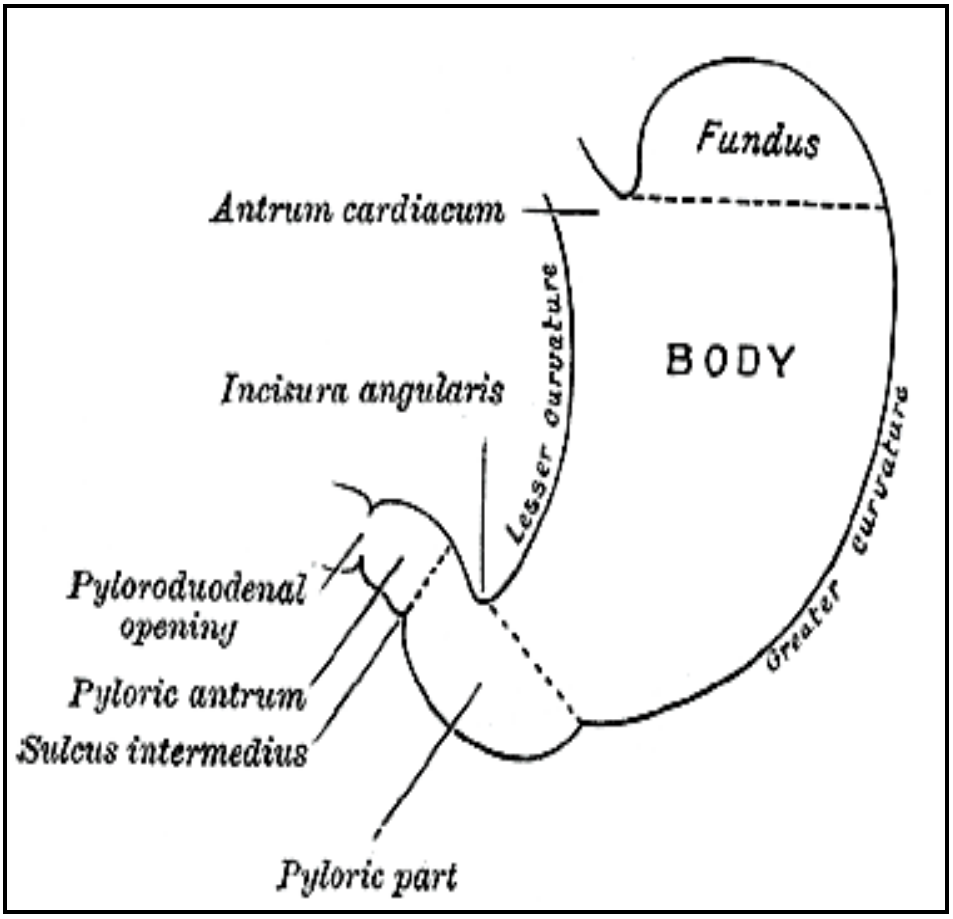

Figure (1): Anatomy of the Stomach (Harold, 2005)**.**

Functional anatomy:

The stomach can be divided into a proximal and a distal segment:

Proximal Stomach (fundus and body):

Mechanically, the proximal stomach serves as a reservoir and enlarges with food intake through a process controlled by the vagus nerve called receptive relaxation. The tonic contraction of the fundus in the postprandial period plays an important role in enhancing gastric emptying, especially of liquids **(Despopoulos and Stefan, 2003).**

Distal stomach (antrum and pylorus):

The gastric antrum and pylorus serve as a coordinated unit to triturate the gastric contents into liquefied chyme. Peristaltic waves begin at the gastric pacemaker along the greater curvature of the stomach and increase in strength as they sweep through the antrum toward the pylorus. The food and chyme is thereby trapped in a high-pressure area at the distal end of the stomach. Most of the contents are squirted back into the stomach while a small amount of chyme; containing particles smaller then 1 mm in diameter, pass through the pylorus and into the duodenum. In this way food is mechanically liquefied and mixed with biochemically important gastric juice **(Despopoulos and Stefan, 2003).**

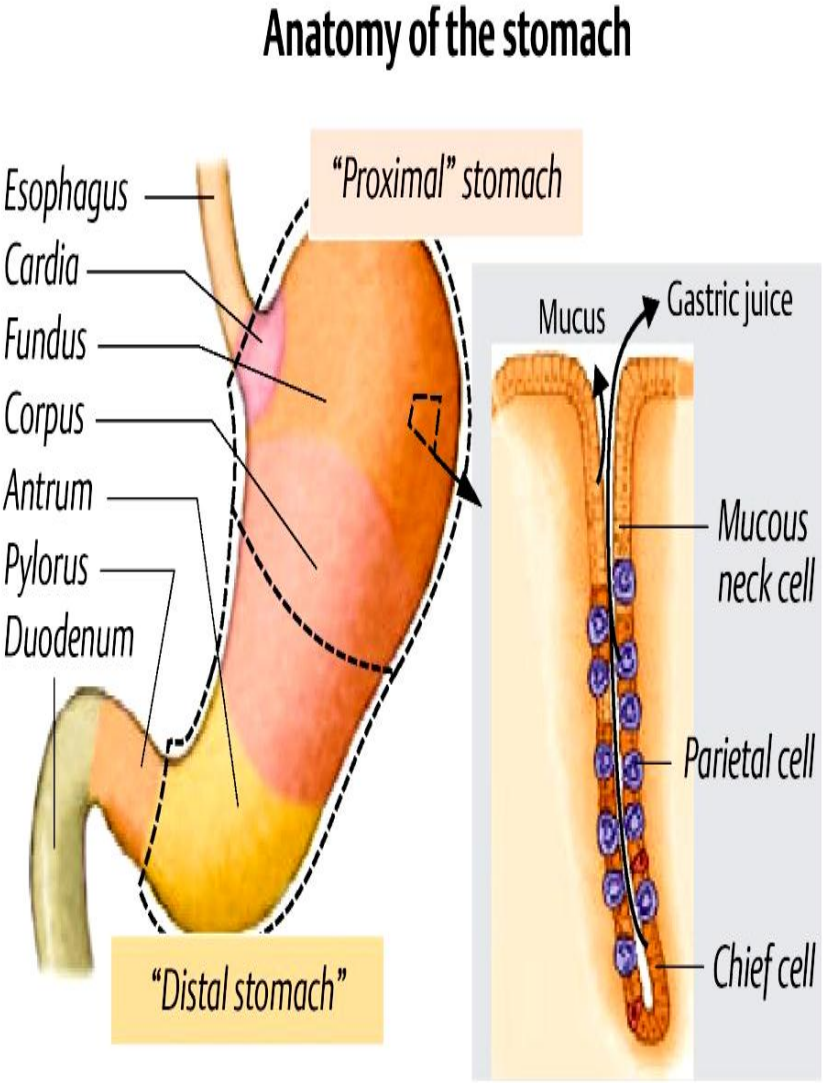

Figure (2): Functional Anatomy of the Stomach (Despopoulos and Stefan, 2003)**.**