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Role of Intensive Care Unit in The Management of Non -Traumatic Acute Abdomen

Essay

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قالوا

سببنا انك لا تعلم لنا
إلا ما علمتنا إنك أنت
العليم العظيم

صدق الله العظيم

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List of Abbreviations

AAA	: Abdominal aortic aneurysm
ABC	: Airway, breathing, circulation
ACS	: Acute coronary syndrome
AMAE	: Acute mesenteric arterial embolism
AMAT	: Acute mesenteric arterial thrombosis
AMI	: Acute mesenteric ischemia
APACHE	: Acute Physiology and Chronic Health Evaluation
aPTT	: Activated partial thromboplastin time
AST	: Aspartate amino transferase
B.i.d.	: Twice a day
β-OHB	: Beta hydroxy butyrate
BUN	: Blood urea nitrogen
°C	: Celsius
C. difficile	: Clostridium difficile
CABG	: Coronary artery bypass graft
CBC	: Complete blood count
CDI	: Clostridium difficile infection
CHF	: Congestive heart failure
CK	: Creatine kinase
COPD	: Chronic obstructive pulmonary disease
CRP	: C- reactive protein
CT	: Computed tomography
CTA	: Computed tomography angiography
cTN	: Cardiac troponin
CVP	: Central venous pressure
CXR	: Chest x. ray
DKA	: Diabetic ketoacidosis
2,3 DPG	: 2,3 diphosphoglycerate
DVT	: Deep venous thrombosis
ECG	: Electrocardiogram
ED	: Emergency department
ELISA	: Enzyme-linked immunoabsorbent assay

List of Abbreviations (Cont.)

ERCP	:	Endoscopic retrograde cholangio-pancreatography
EUS	:	Endoscopic ultrasonography
FFP	:	Fresh Frozen Plasma
Fr	:	French
GI	:	Gastrointestinal
GP	:	Glycoprotein
HCG	:	Human chorionic gonadotropin
HIV	:	Human immunodeficiency virus
HPF	:	High-power field
IBD	:	Inflammatory bowel disease
ICU	:	Intensive care unit
IM	:	Intramuscular
IMA	:	Inferior mesenteric artery
IU	:	International unit
IV	:	Intravenous
kPa	:	kilopascal (1 kPa \equiv 1000 Pa) = (unit of pressure)
LBBB	:	Left bundle branch block
LBO	:	Large-bowel obstruction
LDH	:	Lactate dehydrogenase
LDL	:	Low density lipoprotein
LFTs	:	Liver function tests
MI	:	Myocardial infarction
MOF	:	Multiple-organ failure
MRA	:	Magnetic Resonance Angiography
MRI	:	Magnetic Resonance Imaging
MVT	:	Mesenteric venous thrombosis
NG	:	Nasogastric
NOMI	:	Non-occlusive mesenteric ischemia
NPO	:	Nothing per os
NSAIDs	:	Non steroidal anti-inflammatory drug
P _a O ₂	:	Pressure of arterial oxygen

List of Abbreviations (Cont.)

PCI	: Percutaneous coronary intervention
PCR	: Polymerase chain reaction
PID	: Pelvic inflammatory disease
PO	: Per os
PT	: Prothrombin time
PUD	: Peptic ulcer disease
RAAA	: Ruptured abdominal aortic aneurysm
RBCs	: Red blood cells
RUQ	: The right upper quadrant
SBO	: Small-bowel obstruction
SBP	: Systolic blood pressure
SC	: Subcutaneous
SIRS	: Systemic inflammatory response syndrome
SLE	: Systemic lupus erythematosus
SMA	: Superior mesenteric artery
STEMI	: ST elevation myocardial infarction
TPN	: Total parenteral nutrition
UFH	: Unfractionated heparin
US	: Ultrasound
UTI	: Urinary tract infection
WBCs	: White blood cells

Introduction

Abdominal pain is one of common problems encountered by doctors, either in primary or secondary health care (specialists). It may be mild, but it may also be a life threatening sign. It has been estimated that almost 50 % adults have experienced abdominal pain and it accounts for 5-10 % of all emergency visits (**An and West, 2008**).

Acute abdominal pain generally refers to undiagnosed pain that arises suddenly and is of less than 48 hours duration. Abdominal pain poses diagnostic challenges for emergency clinicians. Causes include medical, surgical, intraabdominal and extraabdominal elements. Associated symptoms often lack specificity and atypical presentations of common diseases are frequent (**Birnbaue et al., 2012**).

Immediate life threatening causes include: abdominal aortic aneurysm, mesenteric ischemia, perforation of gastrointestinal tract (including peptic ulcer, bowel, esophagus or appendix), acute bowel obstruction, volvulus, ectopic pregnancy, placental abruption, myocardial infarction, splenic rupture (eg, secondary to Epstein Barr Virus, leukemia, trauma) and diabetic ketoacidosis. The combination of a careful history, including a precise characterization of the pain and physical examination can often distinguish between organic and non organic causes of abdominal pain and is crucial for creating a focused and appropriate differential diagnosis (**McQuaid, 2012**).

Among the difficulties complicating the diagnosis of intraabdominal disorders in older adults are pre existing illnesses that alter classical manifestations, inability to obtain accurate history and medications that can cause, confound or mask disease processes and alterations in laboratory baseline and physical findings (**Glaspy et al., 2004**).

All these factors contribute to a higher morbidity and mortality for acute abdominal disorders in older adults. For these reasons patients with an acute abdomen often need to be admitted to an Intensive Care Unit peri-operatively for monitoring and management of surgical and medical complications (**Kapadia, 2004**).

Aim of the Study

Is to discuss the pathophysiology, etiology, diagnosis and management of non-traumatic acute abdominal pain in Intensive Care Unit.

Chapter (I)

Pathophysiology of Acute Abdomen

Abdominal pain is an unpleasant experience commonly associated with tissue injury. The sensation of pain represents interplay of pathophysiologic and psychosocial factors. A pain-free acute abdomen is more likely in the elderly, in children and in the third trimester of pregnancy (**Kavanagh, 2004**).

Pain is warning signal, which clearly demonstrates that something goes wrong with that particular part of the body. Simply, pain is an emergency call (**Simunovic, 2013**).

The term acute abdomen generally refers to previously undiagnosed pain that arises suddenly and is of less than 7 days (usually less than 48 hours') duration. Abdominal pain that persists 6 hours or longer is usually caused by disorders of surgical importance. In general acute abdomen is usually indicative of infection, inflammation, ischemia or perforation of an abdominal structure (**Kapadia, 2004**).

Pathophysiology of acute abdominal pain

Pain receptors in the abdomen respond to mechanical and chemical stimuli. Stretch is the principal mechanical stimulus involved in visceral nociception, although distention, contraction, traction, compression and torsion are also perceived. Visceral receptors responsible for these sensations are located on serosal surfaces, within the mesentery and within the walls of hollow viscera, in which they exist between the muscularis mucosa and submucosa. Mucosal receptors respond primarily to chemical stimuli, in contrast to other visceral nociceptors that respond to chemical or mechanical stimuli. A variety of chemical stimuli are capable of triggering these receptors including substance P, bradykinin, serotonin,

histamine and prostaglandins, which are released in response to inflammation or ischemia (**Kavanagh, 2004**).

Types of Abdominal Pain:

Visceral Pain: Is poorly localized. It is frequently described as “deep” & “dull & aching” or “pressure-like”. Visceral pain is caused by over-distention or spastic contraction of a hollow viscus or by stretching of the capsule of a solid organ or a metabolic cause (Arterial injection of lactate, hypertonic, acidic or alkaline IV solutions, or bradykinin as well as black widow spider bites, porphyria or diabetic ketoacidosis all produce visceral abdominal pain) (**Silen, 2005**).

Severe episodes may be accompanied by sensitization to painful stimuli along with autonomic activation (i.e. sweating, nausea or vomiting, tachycardia or bradycardia followed later by deteriorating blood pressure, skin color and hyperesthesia) (**Silen, 2005**).

Visceral pain is transmitted by C nerve fibers that are commonly found in muscle, periosteum, mesentery, peritoneum and viscera (**Millham et al., 2010**).

Visceral pain is poorly localized due to lower number of nerve endings in visceral organ than other organs such as the skin and since the innervations of viscera is multisegmental. It is usually perceived to be in the epigastrium, periumbilical or hypogastrium. It occurs since the visceral organs in the abdomen transmit sensory afferent stimuli to both sides of the spinal cord (**Squires and Postier, 2012**).

Pain is perceived in the abdominal region corresponding to the diseased organ's embryonic origin. Visceral pain from structures that originated from the foregut (stomach, pancreas, liver and gallbladder and proximal duodenum) manifests in the

epigastrium; visceral pain from structures of the midgut (remainder of duodenum, small bowel, proximal large bowel) manifests in the periumbilical region; and visceral pain from structures of the hindgut (middle and distal large bowel, pelvic genitourinary organs) manifests in the suprapubic region. Ischemia, inflammation, or distention of hollow organs or capsular stretching of solid organs produces visceral type pain (McNamara and Dean, 2011).

Somatic Pain: is caused by inflammation of the parietal peritoneum following contact with blood, bile, gastric acid or inflammatory exudates. It is sharper & more discrete than visceral pain & is localized to the site of inflammation. The pain is often aggravated by movement or vibration. Also, it is exacerbated by pressure, both from the examining fingers & from within the inflamed viscus (e.g. "tenderness to palpation"). Involuntary muscular contraction over the area of inflammation ("involuntary guarding") & "rebound tenderness" (caused by any sudden movement of the inflamed peritoneum) are characteristic findings. For example, in acute appendicitis, the early pain is periumbilical visceral pain, which is followed by the localized somatoparietal pain at McBurney's point produced by inflammatory process of the parietal peritoneum (Squires and Postier, 2012).

Parietal pain: is conveyed by A- δ fibers, which are abundantly found in the skin and muscle.

Referred Pain: Pain originating in the viscera may sometimes be perceived as originating from a site distant from the affected organ **Table 1**. Referred pain is usually located in the cutaneous dermatomes sharing the same spinal cord level as the visceral inputs. As an example, nociceptive inputs from the gallbladder enter the spinal cord at T5 to T10. Thus, pain from an inflamed gallbladder may be perceived in the scapula (Squires and Postier, 2012).