INTRODUCTION

Generalized peritonitis is inflammation of serosal membrane of peritoneal cavity. It may be primary or secondary. Primary peritonitis is due to bacterial origin such as TB or chemical due to bile leakage or liver or renal failure. Secondary peritonitis may be due to perforated viscus, Pelvic Inflammatory Diseases (PID) or complicated appendicitis. Secondary peritonitis due to perforate viscus is one of the most common fatal surgical emergencies. In the past, it was treated medically, so mortality was very high and may reach 90%.***(Hiyama and Bennion, 2001)***

In 1926, Krishner proved that mortality can be reduced by using appropriate surgical intervention to remove and eliminate the source of sepsis and so mortality rate decreased to below 50%.***(Malik et al., 2010)*** With the advance in antimicrobial therapy, surgical techniques and availability of intensive care, the mortality rate decreased to about 10% in well-equipped places to 20% in less equipped places which is still high.***(Strobel et al., 2011)***

The outcome of secondary peritonitis doesn't depend on the surgeon's skills and equipment of the place mainly.***(Batra et al., 2013)*** The rapid intervention and the ability of eliminating the source of infection showed to be the determinant of the outcome.***(Doklestić et al., 2014)***

Scoring systems were developed to identify high-risk patients who will need rapid intervention, who may need intensive care post-operatively, who may need damage control surgery instead of definitive surgery and who may need re-exploration.***(Malik et al., 2010)***

Identifying high-risk patients proved to help in reducing the mortality. It also provides objective method to compare the care provided by health care centers.***(Jain et al., 2015)***

Multiple scoring systems were developed to score the severity of the cases such as Acute Physiology And Chronic Health Evaluation (APACHE) II score, Physiological and Operative Severity Score for the enUmeration of Mortality and morbidity (POSSUM), Simplified Acute Physiology Score (SAPS), Sepsis Severity Score (SSS), Ranson score and Imrite score.***(Bosscha et al., 1997)***

Although they were developed to assess the general condition, they showed benefit in assessing cases of secondary peritonitis.***(Barriere and Lowry, 1995)***

APACHE II score was widely used to assess the prognosis in severely ill patients and used mainly for ICU patients as needs data collected on admission and 24 h after ICU admission and needs many investigations. It also doesn't rely on extension or the cause of peritonitis.***(Knaus et al., 1991)*** POSSUM has been used to compare surgical patients and assess the severity of surgical patients but it also not specific for patients of secondary peritonitis.***(Yii and Ng, 2002)***

Mannheim Peritonitis Index (MPI) is a simple scoring system specialized for cases of secondary peritonitis. It was developed in 1987 by Wacha et al., by analysis of 20 risk factors in 1256 patients presented with secondary peritonitis. Eight factors only showed to be reliable to predict prognosis which are age, sex, presence of organ failure, presence of malignancy, if the source of sepsis not colonic, extension of peritonitis and type of soiling.***(Wacha et al., 1987)***

MPI has the advantage of being simple, not needing sophisticated investigations and based on data that can be collected on admission and after laparotomy which are found on routine registers.***(Batra et al., 2013)*** It shows high specificity and sensitivity in predicting the prognosis although it has the disadvantage of ignoring the patient co-morbidities and the inability of eliminating the source of sepsis in some patients and it can't be calculated preoperatively.***(Malik et al., 2010)***

AIM OF THE WORK

This is a prospective study to detect the mortality of patients with peritonitis secondary to perforated viscus in the surgical department and Emergency rooms in Ainshams University Hospitals by using Mannheim Peritonitis Index (MPI) score for comparing our results with previous published studies' results that used the same score to evaluate the efficiency of our management protocols for those patients and if we need to update them.

ANATOMY OF ABDOMEN

The abdomen is the cavity extending from the chest to pelvic floor. It contains the digestive tract, liver and spleen. The abdominal digestive tract consists of abdominal esophagus, stomach, deudenum, small and large bowel and gall bladder.

Abdominal esophagus:

Esophagus is a muscular tube consistis of inner circular and outer longtudinal muscles measuring about 25 cm. It connects the pharynx to the stomach. It's consists of three parts: pharyngeal, thoracic and abdominal.***(Beasley, 1997)***

The abdominal esophagus is the part of esophagus after diaphragmatic crus. It pirces the right crus and extends 4-5 cm and ends at the cardia of the stomach.***(Postma et al., 2009)***

It's covered by the periton of greater omentum on anetrior and left surface and covered by periton of lesser omentum on right surface.***(Gray, 2008)***

Esophageal perforations may be due to

- Boerhaave's syndrome due to sever vomiting that cause longtudinal esophageal tear
- Penetrating malignant mass
- External trauma either penetrating or not penetrating
- Iatrogenic during endoscopy

Esophageal perforation if occurred in thoracic part, will cause mediastinitis. And if occurred in abdominal part, will cause peritonitis.

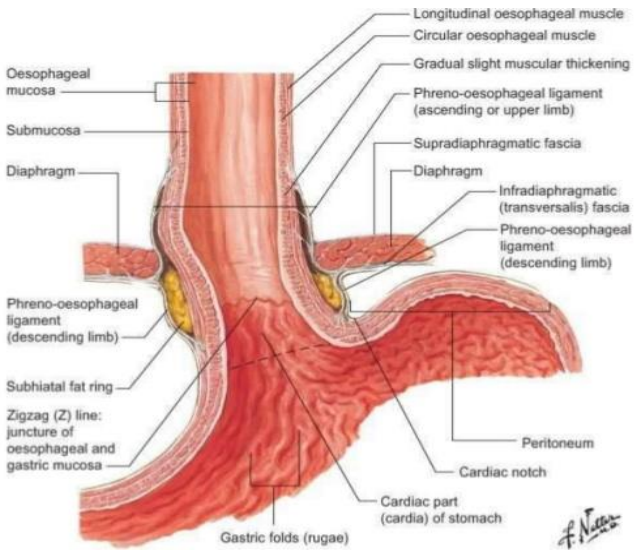

Figure (1): Anatomy of lower esophagus *(F.Netter, 1989)*

The Stomach:

The stomach is a highly vascular muscular structure. It's bag like and distendable according to the content. It's located at left hypochondorial area. It connects esophagus to deudenum. ***(Gray and Lewis, 2000)***

It's the first organ of digestion. It acts to digest and store foods by secreated acids and proteolytic enzymes. The food is crunched and transformed to semi-digested paticles 'chyme' by action of protease enzyme and muscular contractions.***(Krehbiel and Matthews, 2003)***

It consists of three parts:

- Fundus: above the level of the cardia
- Body: the part between fundus and antrum

- Antrum: the terminal part till the pylorus

It's totally covered by peritoneum which becomes thickened at greater and lesser cervatures forming greater and lesser omentum which are double folds of visceral peritoneum. It has two shincters: gastro esophageal sphicter which prevents reflux of food into esophagus & pyloric shincter which controls passage of food into deudenum after digestion. It has three muscle layers: inner oblique for crunching action, middle circular which forms pyloric sphincer and outer longtudinal layer.***(Romanes, 1986)***

Gastric mucosa is protected from secreated acids and protease enzyme by different mechanisms:***(Forssell, 1988)***

- The enzyme is secreated on non-active form which is activated inside stomach cavity by low PH to the acive form Pepsin.
- The mucosa is protected from acidic content by mucus layer maintained by prostaglandin E.
- Gastric cells secret bicarbonate to protect itself against acidity.
- The rapid turn over of mucosal cells after any injury.

Causes of gastric perforations include:

- Gastric ulcers
- Penetrating malignant ulcers
- Iatrogenic during UGI endoscope
- Penetrating trauma

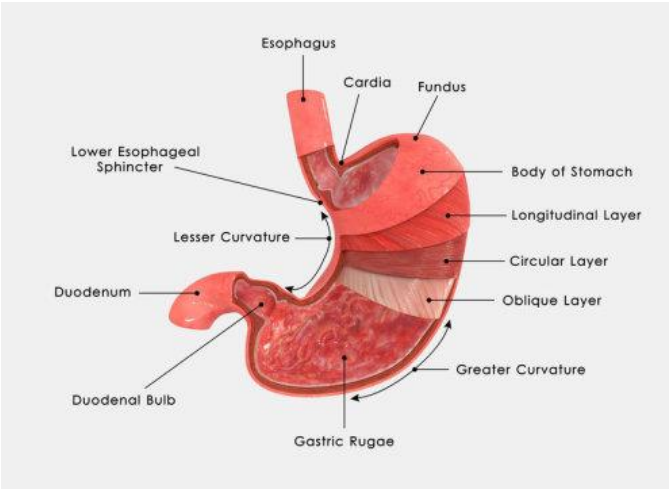

Figure (2): Anatomy of the stomach ***(Krehbiel and Matthews, 2003)***

The Deudenum:

It's the 1st part of the small intestine measuring about 25 cm. It's the widest and non- mobile part of small intestine. It starts after pyloric sphincter of the stomach and ends at deudenojeujenal junction.***(Agur et al., 2013)***

It's formed of four parts:***(Gray and Lewis, 2000)***

- 1st part: It's the superior part and lies at level of T1. It mesures about 5 cm. It's mobile and connected to liver by hepaticodeudenal ligament from the lesser sac.
- 2nd part: It's the vertical part and descends to the level of T3. It mesures about 10cm. It's retroperitoneal and non mobile. The (CBD) & the pancreatic duct open into this part.
- 3rd part: It's the horizontal part at the level of T3. It mesures about 7.5 cm. It's retroperitoneal and non mobile.

- 4th part: It's the ascending part and ascends to the level of T2. It measures about 2.5 cm. It's retroperitoneal and non mobile. It ends at deudenojeujenal junction.

The deudenojeujenal junction is the angle between 4th part of deudenum and jeujenum and lies at the level of T2. It's attached to the posterior abdominal wall by the ligament of Treitz.***(Agur et al., 2013)***

It's protected from gastric acidity by the following:***(Forssell, 1988)***

- Bile secreated by gall bladder which contains bicarbonate to neutralize the gastric secretions.
- Mucus layer secreated and maintained by prostaglandin E.
- The rapid turn over of mucosal cells after any injury.

Deudenal injury may occur due to:

- Perforated ulcer.
- Iatrogenic during UGI endoscope.
- Penetrating trauma or sever blunt trauma.

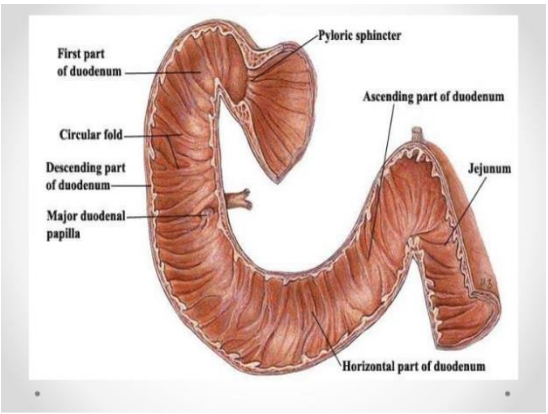

Figure (3): Anatomy of the deudenum ***(Agur et al., 2013)***

The Jeujenum and ileum:

The small intestine is formed of deudenum, jeujenum and ileum. Jeujenum and ileium measure about 5.5 m. They start from deudenojeujenal junction till the ileocecal junction. The iliocecal valve prevents reflux of the remenant of digested food from cecum to ileum. They are covered by mesentry which is a double fold of visceral peritoneum.

Jeujenum measures about 3 m and ileum measures about 2.5 m. They are similar to each other and no definitive point of demarcation between them. Jeujenum is the proximal part which is located mainly at left hypochondorial region and it has longer vasa recta and less arcades. Ileum is the distal part which is located mainly at right iliac fossa and has shorter vasa recta and more arcades. ***(Gray and Lewis, 2000)***

They are responsible for digestion and absorption of most of food content. Digestion is aided by pancreatic enzymes, bile secreted from gall bladder and bicarbonate secreted from deudenal glands to neutralize the gastric acids.

Meckel's diverticulm is an embryologic remenant of vitellin duct which carries blood supply to mid-gut. Normally it involutes and disappears completely, but some times a part of proximal part fails to involute and remains. It may become obstructed causing diverticulitis which may cause diverticular perforation.***(Sagar et al., 2006)***

Causes of intestinal perforation includes:

- Ischemic bowel due to mesentric artery or vein occlusion.
- Strangulated hernia.
- Chron's disease.
- Closed bowel by adhesions which may perforate if neglected.
- Meckel's diverticulum inflammation & perforation.
- Perforated malignant ulcer.
- Typhoid or T.B ulcers.
- Penetrating trauma.

The large intestine:

The large intestine is the last part of the GIT. It starts with cecum after ileocecal valve and ends at rectum at ano-rectal junction. It concists of cecum and appendix, ascending colon, transverse colon, descending colon, sigmoid colon and rectum.***(Drake et al., 2010)***

It measures about 1.5 m. It contains tinea coli which are smooth muscle bands shorter than the colon causing hustrations. It also contains appendices epiploicae which are sacs of peritoneum containing fat.***(Hounnou et al., 2002)***

It's responsible for absorbtion of water and electrolytes from digested food to perform last step in food digestion. Also it harbours bacterial flora which are responsible for food fermentation, vitamine formation as vitamine K and fighting bacteria.***(Canny and McCormick, 2008)***

The cecum is the first part of large intestine. It begins with iliocecal valve which prevents backflow of digested food to ilium. Appendix arise from cecum which is a blind ended structure which acts as lymphoid structure. Cecum is a retroperitoneal structure while appendix is covered with peritoneum called meso appendix.***(Agur et al., 2013)***

The ascending colon begins at the cecum and ends at hepatic flexure. It's a retroperitoneal structure. The transverse colon begins at hepatic flexure and ends at spleenic flexure. It's covered with peritoneum called mesentry.***(Gray and Lewis, 2000)***

The descending colon begins at the spleenic flexure and ends at the sigmoid colon. It's a retroperitoneal structure. The sigmoid colon is S-shaped structure begins after the descending colon and ends at rectum. It's completely covered with peritoneum called sigmoid mesocolon. ***(Agur et al., 2013)***

The rectum is the last part of large intestine which begins after sigmoid colon and ends at ano-rectal junction. The 1st part is covered by peritoneum from front and lateral sides only. The 2nd part is covered by peritoneum anteriorly only "retroperitoneal" and 3rd part is infra peritoneal.***(Drake et al., 2010)***

Causes of lagre intestine peforations include:

- Ischemic bowel due to mesentric artery or vein occlusion.
- Sigmoid volvolus.
- Perforated diverticulitis.
- Perforated appendicitis.
- Perforated malignat ulcer.
- Crohn's or ulcerative colitis.

- Penetrating trauma.
- Iatrogenic during colonoscopy.

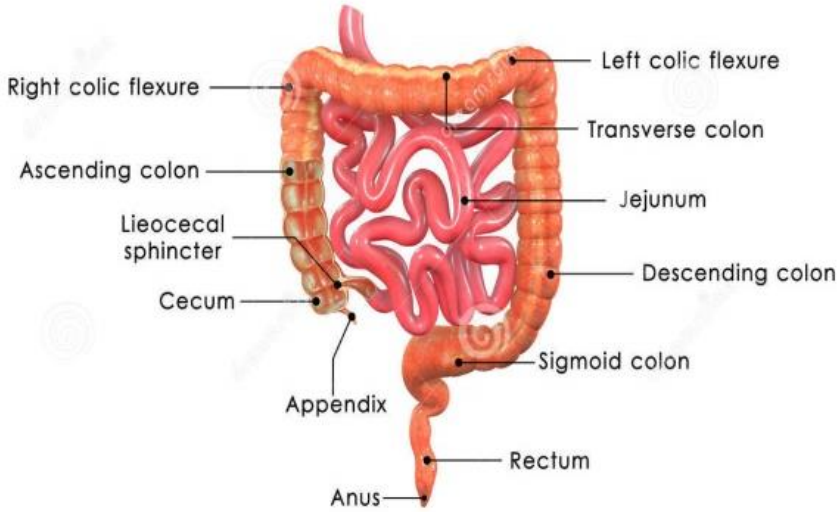

Figure (4): Anatomy of small and large intestine

The gallbladder:

The gallbladder is a pear-shaped stucture located between the right lobe and caudate lobes of liver . Its function is to store and concenterate bile and release it to help in fat digestion.

It's composed of fundus 'which is the part projecting from under the surface of liver and covered with periton' , body 'which is the part laying under the surface of liver' and neck 'which is the narrow terminal part'.***(Gray and Lewis, 2000)***

The cystic duct arises from the the end of the neck 'which is called the Hartmann's pouch' to join the common hepatic duct to

form the common bile duct. The common bile duct opens into the 2nd part of the deudenum at the ampulla of vater.***(Nagral, 2005)***

Causes of gallbladder or biliary tree perforation include:

- Acute gangenous cholecystitis
- Impacted stone at the neck or in the biliary tree
- Iatrogenic during ERCP.
- Penetrating trauma.

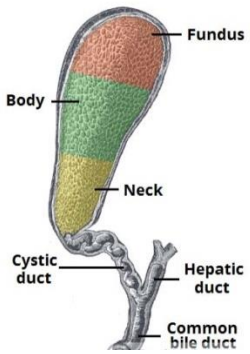

Figure (5): Anatomy of the gall bladder

The peritoneum:

It's a serous membrane of mesothelial origin covering the abdominal organs and lining the abdominal walls. It's the largest serous membrane in the body measuring about 1.7 m^2. The abdominal cavity is devided into preperitoneal space "which is located between abdominal wall and peritoneum" and intraperitoneal space "which is enclosed in peritoneal sac"***(Snell, 2011)***

The abdominal structures are devided 'as regard to their relation to peritoneal membrane' into:***(Drake et al., 2010)***

- Intraperitoneal structures: Liver and gall bladder, spleen, stomach, jeujenum, ileum, cecum and appendix, transverse colon, sigmoid colon, upper 1/3 of rectum and uterus and fallopian tubes.
- Retroperitoneal structures: Pancrease, kidneys, deudenum, ascending and descending colon and middle 1/3 of rectum.
- Infraperitoneal structures: Urinary bladder and lower 1/3 of rectum.

The peritoneum is composed of two layers:

- Visceral peritoneum which is in direct contact to organs. It's blood supply is derived fom visceral blood supply, so it's supplied by celiac and mesenteric vessels. Venous drainage is drained into portal circulation. It's sensetive to stretch and chemical irretations only and not sensetive to injury.***(Skandalakis et al., 2004)***
- Parietal peritoneum: which is lining abdominal wall. It's blood supply comes from abdominal wall blood vessels