

# **Laparoscopic Repair of Perforated Duodenal Ulcer**

**Essay**

*Submitted for partial fulfillment of the requirements of  
Master Degree in General Surgery*

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**2014**

# بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا  
عَلَّمْتَنَا إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

صدق الله العظيم

سورة البقرة آية

(32)

# *Acknowledgement*

*First, I always like to thank **ALLAH**, the great and the merciful, who helps us to know and learn.*

*I would like to express my profound gratitude and appreciation to Prof. Dr. **Waleed Atef Elian**, professor of general surgery, faculty of medicine, Ain Shams University, for his kind supervision and continuous support that helped too much to accomplish his work.*

*I am very grateful to Prof Dr. **Amr Abdel Raouf Abdel Nasser**, professor of general surgery, faculty of medicine, Ain Shams University, for his continuous support and significant supervision that helped too much in finishing this work and bringing it to light.*

*My sincere thanks and appreciation truly go to Dr. **Mostafa Abdo Mohammed**, lecturer of general surgery faculty of medicine, Ain Shams University, who provided me his time effort and Knowledge to build up this work.*

*Lastly and not the least, I would like to thank my **family and friends**, who had suffered during accomplishing this essay, and everybody encouraged it.*

***Ayman Gehad Hussen Maady***

## **INTRODUCTION**

Duodenal perforation is a common complication of duodenal ulcer. Perforated duodenal ulcer is mainly a disease of young men, but because of increasing smoking in women and use of NSAID in all the age group, nowadays it is common in all adult population. (*Lunevicius & Morkevicius, 2005*)

More than 95 % of duodenal ulcers occur in the first part of duodenum. Perforation occurs in approximately 5-10 % of patients with duodenal ulcer. (*Malik & Mian, 2005*)

In most cases of perforation, gastric and duodenal content leaks into the peritoneum. This content includes gastric and duodenal secretions, bile, ingested food, and swallowed bacteria. The leakage results in peritonitis, with an increased risk of infection and abscess formation. There are three clinical phases in the process of perforation of duodenal ulcer can be distinguished:

- \* Phase 1: Chemical peritonitis/contamination.
- \* Phase 2: Intermediate stage. After 6–12 h many patients obtain some relief of pain.

\* Phase 3: After 12–24 h intra-abdominal infection appears. In more severe cases, shock may develop. (*Saber, 2012*)

The disease is life-threatening and early diagnosis and treatment is extremely important. Nowadays due to presence of really effective medications against peptic ulcer for decreasing the amount of acid and also eradication of *H.pylori* the incidence of that disease is less than before. Usually the only surgical procedure that is necessary is simple closure with or without omental patch. (*Zaji, 2007*)

In 1990, the first laparoscopic repair of perforated duodenal ulcer was reported, (*Lunevicius & Morkevicius, 2005*)

Laparoscopic repair of duodenal perforation is a useful method for reducing hospital stay, complications and return to normal activity, it can be performed in 85% of cases, making it possible to avoid a median laparotomy which can lead to wound infection and late incisional hernia. With better training in minimal access surgery now available, the time has arrived for it to replace the conventional open surgery. (*Lunevicius & Morkevicius, 2005*)

# **AIM OF THE WORK**

The aim of the study is to discuss the role of laparoscope in repair of perforated duodenal ulcer and its benefits over open surgery.

## **Objectives of the study**

### **Primary objectives**

Spotlighting laparoscopic method for repair of perforated duodenal ulcer.

### **Secondary objectives**

Comparing laparoscopic method with conventional open surgery for management of perforated duodenal ulcer.

# **ANATOMY OF THE DUODENUM**

The duodenum curves in a C around the head of the pancreas and is (10 inches (25cm)) long. At its origin from the pylorus it is completely covered with peritoneum for about (1 inch (2.5 cm)), but then becomes a retroperitoneal organ, only partially covered by serous membrane. (*Ellis, 2006*)

## **Relations**

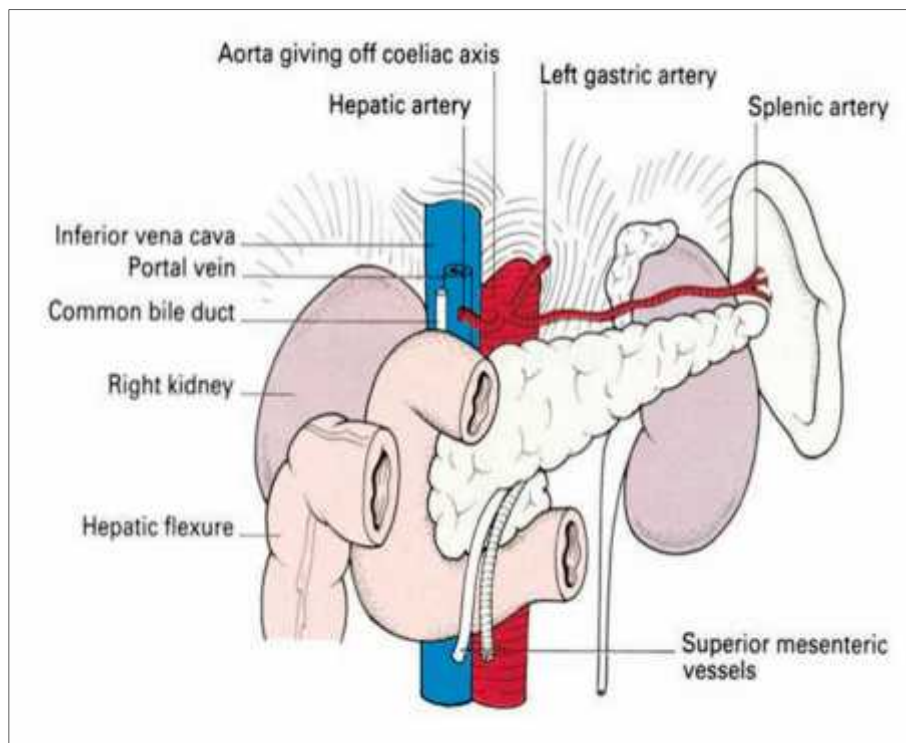


Fig. (1): The relations of the duodenum (*Ellis 2006*)

For descriptive purposes, the duodenum is divided into four sections (fig.1):

**The first part** (2 inches (5 cm)) ascends from the gastroduodenal junction, overlapped by the liver and gall-bladder. Immediately posterior to it lie the portal vein, common bile duct and gastroduodenal artery which separate it from the inferior vena cava. (*Ellis, 2006*)

The first part of the duodenum is overlapped by the liver and gallbladder, either of which may become adherent to, or even ulcerated by, a duodenal ulcer. Moreover, a gallstone may ulcerate from the fundus of the gall-bladder into the duodenum. The gallstone may then impact in the lower ileum as it traverses the gut to produce intestinal obstruction (gallstone ileus). (*Ellis, 2006*)

Extensive dissection of a duodenum, scarred by severe ulceration, may damage the common bile duct which passes behind the first part of the duodenum about 1 in (2.5 cm) from the pylorus. (*Ellis, 2006*)

**The second part** (3 inches (7.5 cm)) descends in a curve around the head of the pancreas. It is crossed by the transverse colon and lies on the right kidney and ureter. Half-way along, its posteromedial aspect enters the



common opening of the bile duct and main pancreatic duct (of Wirsung) on to an eminence called the duodenal papilla, (see fig. 2). This common opening is guarded by the *sphincter of Oddi*. The subsidiary pancreatic duct (of Santorini) opens into the duodenum a little above the papilla. (*Ellis, 2006*)

The hepatic flexure of the colon crosses the second part of the duodenum and the latter may be damaged during the right hemicolectomy. Similarly, the right kidney lies directly behind this part of the duodenum, which may be injured in performing a right nephrectomy. (*Ellis, 2006*)

Mobilization of the duodenum, together with the head of the pancreas and termination of the common bile duct, is performed by incising the peritoneum lateral to the second part of the duodenum and developing the avascular plane between these structures and the posterior abdominal wall-Kocher's maneuver. (*Ellis, 2006*)

The pancreas, as the duodenum's most intimate relation, is readily invaded by a posterior duodenal ulcer. This should be suspected if the patient's pain radiates into the dorsolumbar region. (*Ellis, 2006*)

**The third part** (4 inches (10 cm)) runs transversely to the left, crossing the inferior vena cava, the aorta and the third lumbar vertebra. It is itself crossed anteriorly by the root of the mesentery and the superior mesenteric vessels. Its upper border hugs the pancreatic head. (*Ellis, 2006*)

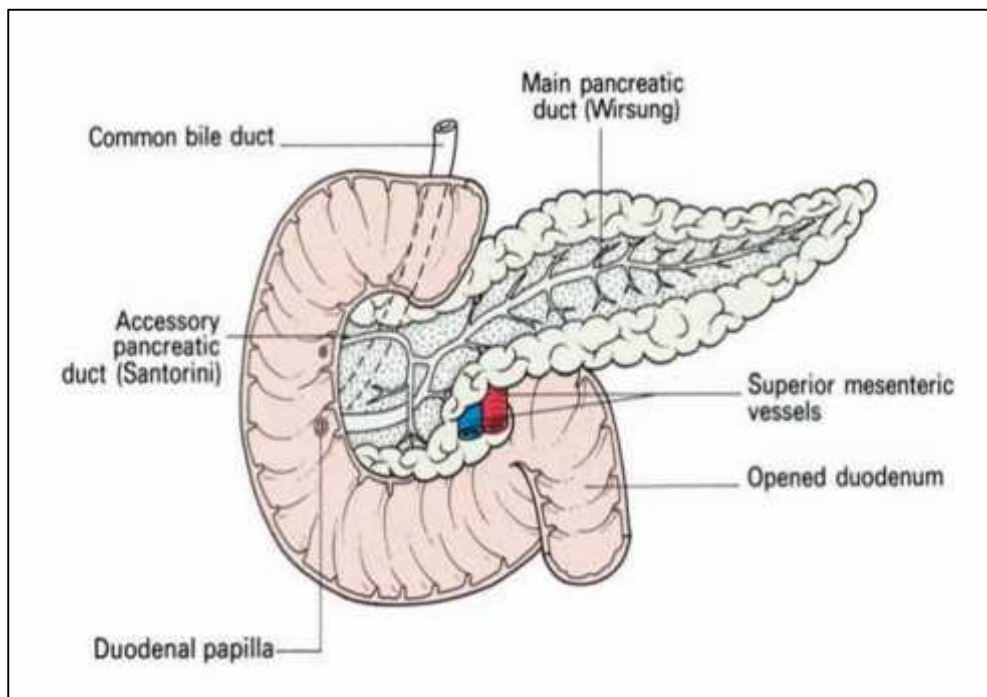


Fig. (2): The duodenum and pancreas dissected to show the pancreatic ducts and their orifices. (*Ellis 2006*)

**The fourth part** (1 inch (2.5cm)) ascends upwards and to the left to end at the duodenojejunal junction. The identity of the duodenal termination is confirmed by the presence of the *suspensory ligament of Treitz*, which is a

well-marked peritoneal fold descending from the right crus of the diaphragm to the duodenal termination, and by visualizing the inferior mesenteric vein which descends from behind the pancreas immediately to the left of the duodenojejunal junction. (*Ellis, 2006*)

## **Blood supply of the duodenum**

The superior pancreaticoduodenal artery arises from the gastroduodenal artery; the inferior pancreaticoduodenal artery originates as the first branch of the superior mesenteric artery. These vessels both lie in the curve between the duodenum and the head of the pancreas, supplying both structures. Interestingly, their anastomosis represents the site of junction of the fore-gut (supplied by the coeliac artery), and the mid-gut (supplied by the superior mesenteric artery), at the level of the duodenal papilla. (*Ellis, 2006*)

Erosion of the gastroduodenal artery by an ulcer results in severe haemorrhage. (*Ellis, 2006*)

# **EPIDEMIOLOGY OF** **PERFORATED DUODENAL ULCER**

Although there is a decreasing incidence, perforated duodenal ulcer remains a serious condition which generally requires surgical intervention, and is associated with a high mortality rate especially among the elderly. (*Saber, 2011*)

## **Incidence of duodenal ulcer**

The prevalence of duodenal ulcers is estimated to be 6-15% in the general population. (*Lau et al., 2011*)

In USA, a systematic review of the literature carried out in 2010, showed an incidence of *duodenal* ulcer disease of 6.1 per 10.000 individuals in USA. (*Knutsson & Buggild, 2010*)

A cohort study in Taiwan, targeting total 403,567 patients with peptic ulcer diseases, showed that incidences of duodenal ulcer decreased from 46.4 to 13.6 per 100,000 in both genders, in all age groups, and at all hospital levels. Significant increases in *Helicobacter pylori* eradication therapy (202% increase) and proton pump inhibitor use (1071% increase) might have contributed to these decreases. (*Wu et al., 2009*)

A Dutch study by *Groenen et al.* in 2009 led to the result that overall, 20,006 upper gastrointestinal endoscopies were performed, duodenal ulcers were diagnosed in 3.5% cases over time; incidence of duodenal ulcer disease in the Dutch population is steadily decreasing over time. Test and treatment regimens for H pylori have possibly contributed to this decline. (*Groenen et al., 2009*)

### **Incidence of perforated duodenal ulcer**

Each year disease peptic ulcer disease (PUD) affects 4 million people around the world. Perforation occurs in 2-10% of patients with peptic ulcer disease including patients taking Antiulcer drugs (Whether complete or incomplete course). (*Zelickson et al., 2011*) (*Ali et al., 2012*)

Duodenal perforation accounts for 60% of perforated peptic ulcers with incidence of 7-10 cases/ 100.000 adults per year. (*Imhof et al., 2008*) Duodenal ulcer is the predominant lesion of the western population. The perforation site usually involves the anterior wall of the duodenum. (*Bertleff & Lange, 2010*)

Perforated peptic ulcer used to be a disorder mainly of younger patients (predominantly males), but

recently the age is increasing (predominantly females). Current peak age is 40-60 years. (*Imhof et al., 2008*)

The incidence of perforated duodenal ulcer (PDU) disease increases with advanced age and this increase has been attributed to the high frequency of risk factors for peptic ulcer disease among elderly patients, e.g., *Helicobacter pylori* colonization or use of non-steroidal anti-inflammatory drugs (NSAIDs). (*Saber, 2011*)

## **Proton pump inhibitors and the incidence of PDU**

The introduction of Proton pump inhibitors (PPIs) was available in Sweden in 1988, a study done by *Hermansson et al.*, in 2009 including all cases of PDUs diagnosed in Sweden from 1974 to 2002 investigating the incidence of this disease before and after the introduction of the PPIs (i.e. the year 1988) in order to detect whether the introduction of this ulcer healing drug has influenced the incidence of this diseases, results were (Fig. 3):

**In Males**, the incidence of duodenal perforation varied from 2.3 to 6.4 per100.000 inhabitants per year. From 1974 to 1988 there was a rise in incidence ( $p < 0.002$ ) and from 1988 to 2002, a fall in incidence ( $p < 0.001$ ).

**While in Females,** the incidence varied from 1.5 to 4.0 per 100.000 inhabitants per year. Between 1974 and 1988 there was an increase in incidence ( $p < 0.001$ ) and from 1988–2002 a fall in incidence ( $p < 0.001$ ).

In the whole material mean age rose from 59.0 years to 67.9 years from 1974 to 2002. The mean age in males with duodenal complications rose from 52.8 years in 1974 to 62.1 years in 2002. In females the corresponding figures were 61.1 and 72.6. (*Hermansson et al. 2009*)

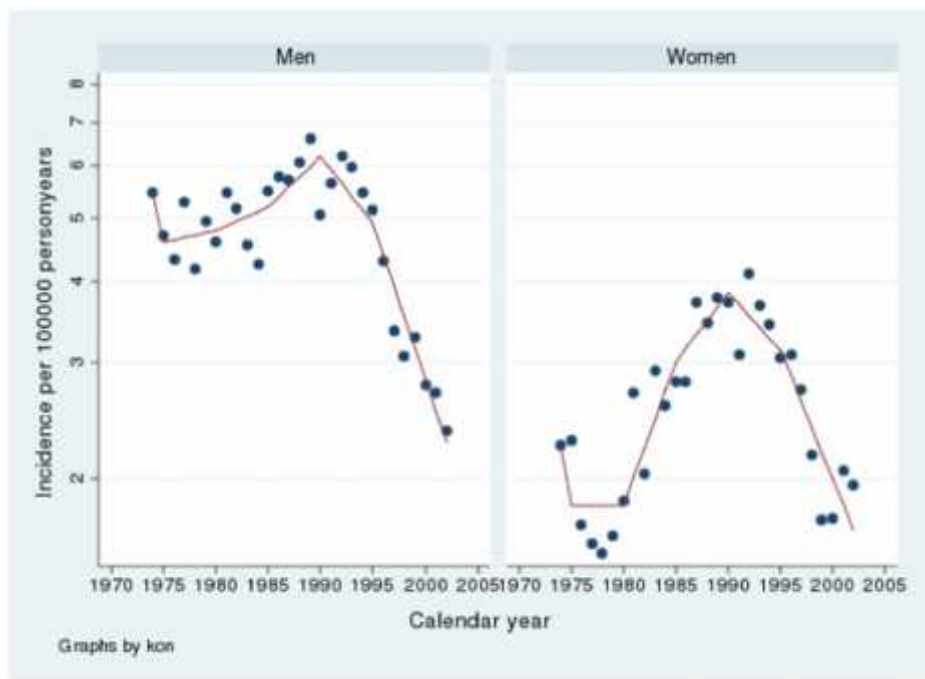


Fig. (3): Incidence of perforated duodenal ulcers in Sweden from 1974 to 2002. (*Hermansson et al., 2009*)