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Perforated Duodenal Ulcer in High Risk Patients

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1. Introduction

Peptic ulcer disease remains one of the most prevalent disease of the gastrointestinal tract with annual incidence ranging from 0.1% to 0.3% in western countries. There are well-known two major precipitating factors: Helicobacter pylori infection and the use of non-steroidal anti-inflammatory drugs (NSAIDs) and the ulcer incidence increases with age for both duodenal and gastric ulcers.

Peptic ulcer disease (PUD) is considered as a mucosal functional derangements due to intraluminal aggressive factors and defects in endogenous defense mechanisms affecting the mucosa and extend through the muscularis mucosa. Some of these functional defects may be caused by the presence of H pylori colonization of the antral mucosa and antral mucosal metaplasia of the proximal duodenum. In vivo and in vitro data support this concept, particularly with reference to the mechanisms of Helicobacter pylori-induced aberrations in gastric and duodenal mucosal function. Standard medical therapy for peptic ulcer disease includes antisecretory medications as well as antibiotics designed to eradicate H pylori colonization.

Complications of peptic ulcer disease are bleeding, perforation and obstruction. These complications can occur in patients with peptic ulcers of any etiology. Perforation occurs in about 5% to 10% of patients with active ulcer disease. Duodenal, antral and gastric body ulcers account for 60%, 20% and 20% of perforations, respectively, of peptic ulcers. Open and laparoscopic abdominal exploration are always indicated in gastroduodenal perforation. Hemodynamic instability, signs of peritonitis and free extravasation of contrast material on upper gastrointestinal tract contrast studies make the decision for operation more urgent and imperative. But, the advent of proton pump inhibitors and Helicobacter pylori eradication in the management of chronic peptic ulcer disease has reduced the operative treatment of this condition to its complications. Perforated duodenal ulcer remains a major life threatening complication of chronic peptic ulcer disease.

The incidence of peptic ulcer disease in normal populations has declined over the past few years following a more streamlined pharmacological intervention. This can be attributed to the efficiency of histamine 2 (H2) blockers and proton pump inhibitors. Additionally, the diagnosis and eradication of Helicobacter pylori infection, now known to be a major factor in the pathogenesis of peptic ulcer disease, has almost eliminated the role of surgery in the

elective management of peptic ulcer disease. However, the incidence of perforated duodenal ulcers has either remained the same or has been increasing with the resultant increase in the incidence of emergency surgery. Although the use of potent H2 blockers and proton pump inhibitors has caused a marked decline in the incidence of peptic ulcer perforation, no such decline has been seen in the eradication of H. pylori infection.

Patients with perforated duodenal ulcers include those with acute ulcers, such as patients on nonsteroidal anti-inflammatory drugs (NSAIDs) and those with chronic ulcer disease who are refractory to or noncompliant with medical treatment. Another contributing factor to the increased incidence of perforation of duodenal ulcer is the decrease in elective anti-ulcer surgery. Patients presenting with an acute abdomen suggestive of a perforated duodenal ulcer are generally between 40 and 60 years of age although the number of patients over the age of 60 has been gradually increasing. Approximately 50% to 60% of these patients have a history of peptic ulcer disease, while a smaller number have a history of use of NSAIDs. Now, It's settled that H. pylori infection and NSAID use are two independent risk factors associated with perforated duodenal ulcers, and the lack of duodenitis in NSAID users as compared with those with H. pylori infection suggests a differing pathogenesis.

The frequency of perforated peptic ulcer is decreasing among the overall population but it is becoming more frequent among old people. The higher mortality rate in the old population, justifies the search of prognostic factors specific for the elderly.

2. High risk elderly patients

"High risk" surgical patients
Age > 60
Congestive cardiac failure
Ischaemic heart disease
Cardiac arrhythmia
Hypertension
COPD
Pulmonary embolus
Chronic renal insufficiency
Diabetes mellitus with end-organ damage
Long term steroid therapy
Chronic liver disease
Cerebrovascular disease
Peripheral vascular disease

Table 1. Showed patients with high risk of post operative death.

Risk is a term that is understood differently by different individuals depending on expectation and previous experience. The term "high risk surgical patient" is poorly defined. The term should refer to the group of patients, who were considered to be at high risk of post operative death, and were included in studies of pre-operative "optimization" to a pre-determined oxygen delivery [table 1]. From a practical point of view 'high risk' can probably be defined in two different ways: the first is relevant to an individual and suggests that the risk to an individual is higher than for a population; the second compares the risk of the procedure in question with the risk of surgical procedures as a whole. Furthermore, many investigators suggest that surgical patients for whom the probable mortality is greater than 20% should be considered 'extremely high-risk' patients. There are two main components in identification of high risk for surgery. The first relates to the type of surgery and the second to the cardiopulmonary functional capacity of the patient. There are methods that can be used to assess risk in various patient groups and in the author's opinion , the two most useful scoring systems in surgical risk assessment remain the American Society of Anesthesiologists (ASA) score and the patients' clinical criteria. Both of these assessments are simple to use and do not require additional resources. Surgical risk, in turn, has two components: the extent and the duration of the procedure both can cause an increase in postoperative oxygen demand and an increase in cardiac output or an increase in oxygen extraction. The classification of surgical interference is done in accordance with the extension and/or complexity of the procedure, with one or several of the mentioned characteristics:

S1. Minor Surgery: minimal extension, local anesthesia, ambulatory.

S2. Major Simple Surgery: performed on one organ or system, without any other added procedure

S3. Major Complex Surgery: performed on one organ or system, with other procedure or procedures related with the scheduled one, potential important bleeding, perhaps with some surgical problem that can be solved.

S4. Major Multiple Surgery: on several organs or systems, important bleeding, potential perioperative complications, it needs special preparation

S5. “Rescue” surgery, danger of death

The second item is the functional capacity of the patient that determines his ability to support the postoperative demand of increased oxygen consumption and therefore of cardiac output. Myocardial ischemia only becomes part of this equation if the ischemia limits ventricular function and cardiac output.

The definition of “elderly” is controversial and the traditional demographic definitions include those patients exceeding 65 years of age as the functional deterioration is more frequently apparent beyond the age of 70 years. For the elderly, one should categorize age-related pre-existing chronic illness; age related functional physical decline, or preoperative risk status. The most important surgeon responsibility is to decide whether to operate or not when the patient is of high surgical risk. The decision-making process is complex in elderly surgical candidates. Among the currently available risk assessment tools, American Society of Anesthesiologists (ASA) scoring system despite does not measure operative risk, rather it assesses the degree of sickness or physical state prior to anesthesia and surgery. The assessment of cardiac risk is addressed by the Cardiac Risk Index (CRI) in noncardiac surgery and the risks of postoperative respiratory complications are age over 70; perioperative bronchodilator use; abnormal chest x-ray; and high ASA grade.

The Acute Physiological and Chronic Health Evaluation (APACHE) is the best known physiological scoring system. It is based on twelve physiological variables and is currently being used in general and surgical intensive care patients. Age is an independent risk factor built into above mentioned risk prediction tools; ASA, Cardiac Risk Index (CRI) and APACHE. Preoperative Assessment of medication use is highest in elderly persons who require multiple medications to treat their complex set of medical problems. Medications necessary for managing medical conditions can put elderly individuals at risk of medication-induced problems such as adverse drug effects, drug-drug interactions, or drug toxicities. The greater the number of medications taken, the greater the risk of a clinically serious drug-drug interaction and the adverse drug reactions experienced by elderly patients often tend to be more severe than those experienced by younger patients. The reduced organ reserve capacity of elderly persons contributes to this as every organ system loses reserve capacity with age.

3. Epidemiology of perforated PUD

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. The frequency of perforated peptic ulcer is decreasing among the overall population but it is becoming more frequent among old people. The higher mortality rate in the old population, justifies the search of prognostic factors specific for the elderly. The high mortality from perforated peptic ulcer underlines the importance of risk stratification. Over the past decades, important changes have occurred in the epidemiology of peptic ulcer disease. The discovery of Helicobacter pylori in the early 1980s as a major cause of peptic ulcer disease had a significant impact on the treatment of ulcer disease. The significance of the discovery led to the award of the 2005 Nobel Prize in Medicine to Robin Warren and Barry Marshall [figure 1]. H pylori eradication therapy was proven to cure patients with previous chronic, recurrent ulcer disease.

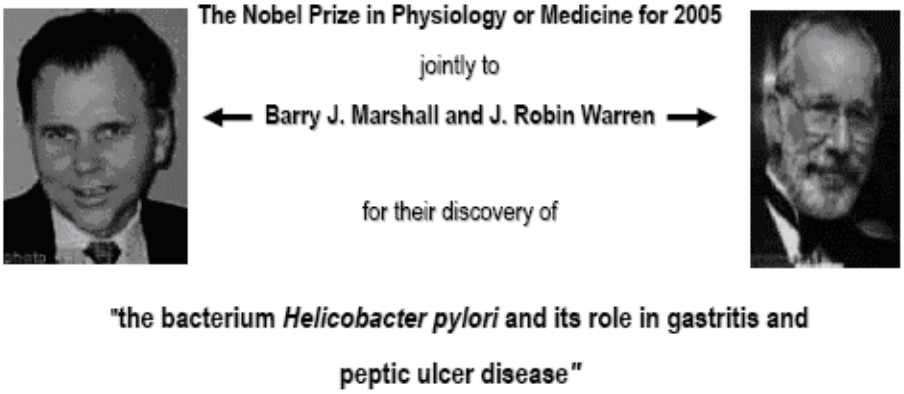

Fig. 1. Nobel Prize winners in Medicine and Physiology at the 2005

Peptic ulcer complications have a high mortality, especially in elderly patients and it is therefore important to understand the epidemiology of this disease in order to investigate if

complications can be prevented. Despite new efficient drugs to treat peptic ulcer disease and increasing knowledge about its aetiology, the incidence of peptic ulcer complications, i.e. perforation and bleeding, have been reported by several groups to be unchanged. Further research into the epidemiology of H pylori infection showed that the prevalence of this bacterium was decreasing over time in recent decades, presumably as a result of improvements in living conditions. The overall decline of peptic ulcer disease is likely to be due to a combination of factors including the introduction of acid suppressive medication, a decreasing prevalence of H pylori in subsequent birth cohorts and the development of eradication treatment for H pylori-positive ulcer patients, which prevents chronic relapsing ulcer disease. The introduction of newer NSAIDs and a tendency for the prescription of lower doses of acetylsalicylic acid for patients with cardiovascular disease may also have contributed to the changing epidemiology of ulcer disease.

With the decreased incidence of ulcer disease, the incidence of ulcer complications may have been affected as well. But most studies showed that the incidence of the most important complication, ulcer bleeding, remained stable notwithstanding the decreasing incidence of peptic ulcers.

The introduction of an endoscopy database allowed for closer investigation of the incidence and epidemiology of gastric and duodenal ulcers, complication rates and classifications. Mortality after perforated and bleeding peptic ulcer increases. An increased burden of comorbidity among elderly patients did not explain the association between advanced age and increased mortality, with the strongest association observed among patients with no history of hospital-diagnosed comorbidity.

Studies on the incidence of perforated duodenal ulcer are limited and data are largely based on findings observed over two decades ago. The epidemiological data on duodenal ulcer perforation was obtained mainly from medical records registry units all over the world for patients admitted with ulcer perforation. The incidence of perforated duodenal ulcer disease increases with advanced age and this increase has been attributed to the high frequency of risk factors for PUD among elderly patients, e.g., Helicobacter pylori colonization or use of non-steroidal anti-inflammatory drugs. Perforated peptic ulcer has an overall reported mortality of 5%–25%, rising to as high as 50% with age. Being closely related to advanced age, increased burden of comorbidity may partially explain the higher mortality among elderly patients.

Several studies support the notion that NSAID is a risk factor not only in uncomplicated peptic ulcer disease, but also in regard to perforated ulcers. The higher risk was maintained during treatment and disappeared after treatment termination. The added risk is dose-dependent and also includes low-dose acetyl salicylic acid. According to a study by Sorensen et al from 2000 the risk increase further when low-dose ASA is combined with NSAID. Proton pump inhibitors (PPI) have become one of the most sold drugs in the world and are nowadays also available over the counter in Sweden. It is a well known fact that PPI protect against peptic ulcer complications in NSAID users. Interestingly the introduction of PPI was almost simultaneous with the beginning of a falling incidence in peptic ulcer complications. H pylori is an important pathogenic factor in peptic ulcer disease, although studies that investigate the connection between H pylori and peptic ulcer complications are somewhat divergent. Helicobacter Pylori is, without a doubt, connected to peptic ulcer disease and its complications , perforation and bleeding, however, other factors such as NSAID and smoking are of great importance as well [figure 2]. Smoking is a another important risk factor for peptic ulcer perforation. smoking more than fifteen cigarettes daily

increased the risk of peptic ulcer perforation 3,5 times. The prevalence of smoking has declined during the last twenty years in most western countries, especially in men, which could perhaps account for fewer ulcer complications.

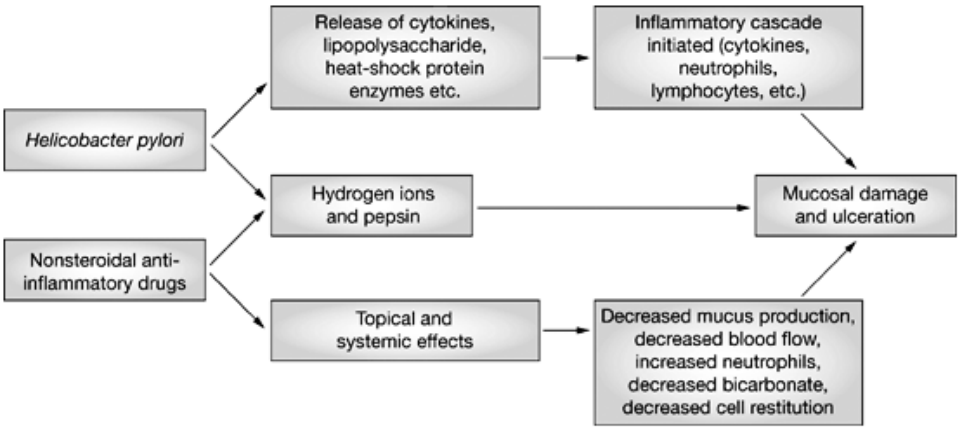

Fig. 2. The synergistic and independent effects of Helicobacter pylori and nonsteroidal anti-inflammatory drugs on gastric mucosal damage.

4. Pathophysiology of perforated peptic ulcer

The duodenal mucosa resists damage from the effect of aggressive factors, such as gastric acid and the proteolytic enzyme pepsin, with the help of several protective factors, such as a mucous layer, bicarbonate secretion, and protective prostaglandins. The epithelial cells of the stomach and duodenum secrete mucus in response to irritation of the epithelial lining and as a result of cholinergic stimulation. A portion of the gastric and duodenal mucus exists in the form of a gel layer, which is impermeable to acid and pepsin. Other gastric and duodenal cells secrete bicarbonate, which aids in buffering acid that lies near the mucosa. Prostaglandins E (PGE) have an important protective role by increasing the production of both bicarbonate and the mucous layer. When an alteration occurs in the aggressive and/or protective factors, a duodenal ulcer occurs such that the balance is in favor of gastric acid and pepsin. Any process that increases gastric acidity (eg, individuals with increased maximal and basal acid output), decreases prostaglandin production (eg, NSAIDs), or interferes with the mucous layer (eg, H pylori infection) can cause such an imbalance and lead to peptic ulcer disease.

Full understanding of the pathophysiology and pathogenesis of duodenal ulcers requires a brief discussion of the two major etiologies: NSAID use and H pylori infection. NSAIDs are pathogenic through their inhibition of the cyclooxygenase-1 (COX-1) pathway, which normally produces protective prostaglandins. These prostaglandins are protective because they augment both bicarbonate and mucous production, as mentioned above. However, perhaps more important, prostaglandins augment mucosal blood flow, and their inhibition leads to impairment of blood flow, leaving the mucosa vulnerable to damage. Infection with H pylori is likely pathogenic by means of a variety of indirect mechanisms as the organism does not generally colonize the duodenum. H pylori infection leads to an inflammatory state

in which high levels of tumor necrosis factor-alpha (TNF-alpha) and other cytokines are produced and in turn stimulate gastric acid production directly by increasing gastrin release from G cells and inhibit somatostatin production by antral D cells. This leads to a net increase in gastric acid secretion, which leads to an increased acid load in the duodenum, overwhelming the mucosal defense. Duodenal acid exposure can lead to gastric metaplasia, whereby the duodenal mucosa can take on characteristics of gastric mucosa. H pylori can then colonize the duodenal mucosa and adhere to cells. This adherence leads to a variety of second-messenger signals, which invoke an immunologic response against those cells causing mucosal damage by host neutrophils and other inflammatory cells. H pylori also affects the gastric and duodenal mucous layer, because this organism produces proteases that degrade the protective mucous layer. Moreover, H pylori infection decreases the production of epidermal growth factor, which normally promotes healing of gastric and duodenal mucosa. H pylori produces proteins that may serve as chemotactic factors for neutrophils and monocytes, which act as proinflammatory cells. H pylori also affects the gastric and duodenal mucous layer, because these organisms produce proteases that degrade the protective mucous layer. H pylori does not lead to the development of gastric and duodenal ulcers through alteration of the bacterial flora. 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 PPU can be distinguished:

Phase 1: Chemical peritonitis/contamination. The perforation causes a chemical peritonitis. Acid sterilizes the gastroduodenal content; it is only when gastric acid is reduced by treatment or disease (gastric cancer) that bacteria and fungi are present in the stomach and duodenum.

Phase 2: Intermediate stage. After 6–12 h many patients obtain some relief of pain. This is probably due to the dilution of the irritating gastroduodenal contents by ensuing peritoneal exudates.

Phase 3: Intra-abdominal infection. After 12–24 h intra-abdominal infection supervenes

Subsequent third-spacing of fluid in the peritoneal cavity due to perforation and peritonitis leads to inadequate circulatory volume, hypotension, and decreased urine output. In more severe cases, shock may develop. Abdominal distension as a result of peritonitis and subsequent ileus may interfere with diaphragmatic movement, impairing expansion of the lung bases. Eventually, atelectasis develops, which may compromise oxygenation of the blood, particularly in patients with coexisting lung disease.

5. Prognostic factors

The continuing problem with perforated duodenal ulcer stands in contrast to the fall in admissions for uncomplicated duodenal ulcers noted since the 1970's and largely attributed to the introduction of H 2 antagonists. The high incidence of complications necessitates the identification of factors associated with the morbidity and mortality of patients undergoing surgery for perforated peptic ulcer. The patient population with perforation tends to be elderly ; mean age 60–70, chronically ill and those patients often taking ulcerogenic medication. Mortality rate after surgery for perforated duodenal ulcer is much more higher in the elderly that reach up to 50%. This can be explained by the occurrence of concomitant

medical diseases but also by difficulties in making the right diagnosis resulting in a delay of >24 hours. The longstanding perforation more than 24 hours together with major medical illness and preoperative shock collectively predicted the outcome in patients with perforated duodenal ulcer as [table 2].

RISK FACTORS	SCORE
1.Numbers of hrs since ulcer perforation	
24hrs or less	0
More than 24 hrs	1
2.Pre operative systolic BP (mm of Hg)	
100 or more	0
Less than 100	1
3.Any one or more systemic illness	
Absent	0
Present	1

Table 2. Boey score-risk factor to predict mortality

The mortality rate increased progressively with increasing numbers of risk factors: 0%, 10%, 45.5%, and 100% in patients with none, one, two, and all three risk factors of Boey, respectively. Definitive surgery can be done safely in good-risk patients. Simple closure is preferable in those patients with uncomplicated perforations if any risk factor is present. Truncal vagotomy and drainage may be required if there is coexisting bleeding or stenosis. Nonoperative treatment deserves re-evaluation in patients with all three risk factors because of their uniformly dismal outcome after operation.

6. Clinical course of PDU in elderly

Studies have shown that nearly half of patients presenting with complicated peptic ulcer disease (PUD), have no history of the disease. On endoscopy, unsuspected ulcers have been found in people who were taking nonsteroidal anti-inflammatory drugs (NSAIDs). Two courses of the disease were observed: the first is defined by acute disease of less than 24 hours' duration preceding surgery. Classic patients with perforated peptic ulcer disease usually present with a sudden onset of severe sharp abdominal pain that may be generalized pain or epigastric urging these patients assuming a fetal position. Abdominal examination findings are usually consistent with generalized tenderness, rebound tenderness, guarding, and rigidity. However, the degree of peritoneal findings is strongly influenced by a number of factors, including the size of perforation, amount of bacterial and gastric contents contaminating the abdominal cavity, time between perforation and presentation, and spontaneous sealing of perforation. Accordingly, the second course of perforation is of longer duration, starting with various abdominal complaints and presenting more severely only after the first 24 hours. Patients belonging to the second course may also demonstrate signs and symptoms of septic shock, such as tachycardia, hypotension, and anuria, but these indicators of shock may be absent in elderly patients or

in those with other systemic illness. In recent years, patients presenting with perforated duodenal ulcers have tended to be elderly and chronically ill and taking one or more ulcerogenic drugs. Several studies have shown the mean age of such patients to be more than 60 years. In elderly patients, signs and symptoms may be minimal. In patients over age 60 with perforated ulcer, more than 80 % had only mild abdominal pain. Other reported symptoms were dyspepsia, anorexia, nausea, and vomiting. Severe abdominal pain was present in only less than 20 % of patients. Duration of symptoms is usually protracted and delayed. Although minority of those patients had no abdominal findings, most had abdominal tenderness, with up to two thirds having classic signs of peritonitis.. There is a changing scene with perforated peptic ulcer. The older age of presentation, the increased association with non-steroidal anti-inflammatory drugs, associated increased debility, and resulting higher mortality in the elderly, are causing a rethink in management protocols.

7. Management protocol

There are two main accepted regimen of treatment of perforated duodenal ulcer; non-operative and surgical treatment. Non-operative treatment should be rendered in perforated peptic ulcers only when the patient shows definite signs of improvement both symptomatically and clinically, and there is a definite "walling off" of the ulceration, or when the patient's condition is too poor to permit operation. With good operative risk, patients should be prepared for surgery of ulcer-definitive procedure of the surgeon's choice; for example vagotomy and pyloroplasty or antrectomy. Purulent peritonitis would dictate only secure closure of the perforation [table 2].

7.1 Nonoperative treatment

The introduction of novel peptic ulcer drugs, such as H2 receptor blockers and proton pump inhibitors, caused a prompt decline in elective operations for peptic ulcer disease in recent times. On the other hand, surgery for peptic ulcer complications, such as perforations has not changed. Effective medical management of peptic ulcer disease has reduced the incidence of gastric outlet obstruction as a complication, but perforation especially in the elderly remains unchanged and is, in fact, on the increase. There is a changing trend in emergency surgery for perforated duodenal ulcer from definitive anti ulcer surgery to simple closure followed by Helicobacter pylori eradication. Surgical emergency due to a perforated peptic ulcer – whether treated laparoscopically or by open repair – is associated with a significant postoperative morbidity and mortality. Therefore, risk-stratification of these subjects provides surgeons with an important tool to plan the management. The dominant treatment of perforated duodenal ulcer in the first half of the 20th century was surgical closure. In most perforated duodenal ulcers that were successfully surgically closed, the perforation was a harbinger of subsequent major morbidity from peptic ulceration. This was in the form of re-perforation, hemorrhage, obstruction, or intractability. The major concern against simple closure is the possible risk of future serious complications of relapse. Some authors claim that prognosis is not related to the surgical procedure itself and the current policy is not to perform definitive ulcer surgery in cases of PPU. A simple procedure should be the one of choice for an emergency operation and extensive procedures should be reserved only in selected patients despite good results are obtained with simple procedures.

Several facts support an alternative to the currently accepted therapy of the perforated duodenal ulcer, that is, immediate surgical closure of the perforation with or without an ulcer-definitive procedure. The following facts are included:

1. Most ulcers are associated with infection with H pylori, including ulcers that perforate.
2. Almost all ulcers associated with H pylori can be healed with combined medical therapy; ie, antibiotics and proton-pump inhibitors or H2 blockers. The rate of relapse is very low and re-infection is rare.
3. The administration of H2 blockers and proton-pump inhibitors and elimination of NSAIDs are now essential components of medical therapy. Such therapy has favorably affected the natural history of duodenal ulcers, including those that perforate.
4. Approximately half of duodenal ulcers that perforate will have self-sealed when first seen by the physician.
5. The perforation of a duodenal ulcer that has sealed spontaneously can be treated nonoperatively with low morbidity, including releakage and abdominal abscess.
6. Death due to peritonitis reflects protracted leakage and secondary bacterial contamination.
7. Major associated disease is a significant risk factor for death following perforation of a duodenal ulcer.

7.1.1 Principles of conservative treatment

Principles of conservative treatment include nasogastric suction, pain control, antiulcer medication, and antibiotics. Nonsurgical treatment has been recognized for a long time. The first major series was published by Taylor nearly 50 years ago; it reported a mortality rate of 11% in the nonsurgical treatment group, compared to 20% in the surgical group. Since then, because of improvements in operative and postoperative care, the mortality rate with surgical treatment of perforated peptic ulcer has decreased to about 5%. Failure of conservative treatment is generally defined as development of septic shock, multiple organ failure or intra-abdominal abscess. Conservative treatment failure exposes patients to the risk of delayed surgical closure with mortality rates between up to 50% , depending on the timing of secondary surgery. While conservative treatment was first proposed to patients not eligible for surgery, some few investigators have tried this approach in rather fit patients but in fact these studies have reported high mortality compared to the results achieved by surgical repair in elderly or medically frail patients. The systematic introduction of PPI use and HP eradication seems to have favorably influenced the results of conservative therapy through reduction of mortality.

7.1.2 Failure of conservative treatment

Definition of prognostic factors for conservative treatment has been a concern for all investigators. The presence of shock at admission is a major criterion for conservative treatment failure and implies that, even in a moribund patient. The presence of haemodynamic instability militates in favor of prompt surgery. The presence of shock being one of the Boey criteria, has a strong correlation of mortality.

7.2 Surgical Therapy

Surgery is recommended in patients who present with hemodynamic instability, signs of peritonitis and free extravasation of contrast on upper gastrointestinal contrast studies. If

contamination of the upper abdomen is minimal and the patient is stable, a definitive ulcer procedure can be performed. For a perforated duodenal ulcer, this may include a highly selective vagotomy, a truncal vagotomy and pyloroplasty, or vagotomy and antrectomy.

7.2.1 Preoperative preparation

Fluid resuscitation should be initiated as soon as the diagnosis is made. Essential steps include insertion of a nasogastric tube to decompress the stomach and a Foley catheter to monitor urine output. Intravenous infusion of fluids is begun, and broad-spectrum antibiotics are administered. In select cases, insertion of a central venous line for accurate fluid resuscitation and monitoring. As soon as the patient has been adequately resuscitated, emergent exploratory laparotomy should be performed.

7.2.2 Surgical procedures

a. Laparoscopic Surgery

The traditional management of a perforated duodenal ulcer is closure with omental patch and a thorough abdominal lavage. More recently this has been shown to be able to performed using a laparoscope. The only proven advantage of the laparoscopic technique appears to be decreased postoperative pain. Operating times are longer compared to open techniques and hospital time appears to be similar to conventional treatment.

b. Immediate Definitive Surgery

Attempts have been made to improve upon the results of simple closure and lavage in response to the large number of patients more than 25% continue to have symptoms attributable to their ulcer diathesis after surgery. Since the 1940's the concept of immediate definitive ulcer surgery has been raised but debated amongst surgeons. There is good evidence that, in the emergency situation, highly selective vagotomy combined with simple omental patch closure of the perforation, in patients without the risk factors, is just as effective as that performed in the elective setting with less mortality and ulcer recurrence rate. Truncal vagotomy with drainage has its advocates as an expedient operation familiar to most surgeons. Immediate definitive ulcer surgery has not gained widespread popularity as it is associated with a higher mortality than simple closure in patients at risk of suffering from complications of surgery. Many agree that an appropriate approach is to select only those with a chronic history of ulcer disease for more than 3 months and without preoperative risk factors for immediate definitive surgery. A major difficulty is defining preoperatively the patients with chronic ulcer history as many ulcers showed silent history, many patients are too unwell to give a reliable history of their disease and finally, perforations occurs as the first manifestation of the ulcer diathesis.

7.3 Percutaneous peritoneal drainage

The higher mortality rate in the old population, justifies the search of prognostic factors specific for the elderly in whom the difficult management was attributed to their concomitant diseases. The criteria of Taylor's method in selected cases were diagnosis of perforation in less than 12 hours, with stable hemodynamic condition and age not exceeding 70 years. Emergency abdominal operations are commonly performed and carry high morbidity and mortality risk, particularly in elderly patients due to presence of coexisting

cardiopulmonary disease, late admission and presence of peritonitis. So, in high risk elderly patients with perforated duodenal ulcer and established peritonitis, pus should be drained with the least invasive maneuver. Transnasogastric placement of a drainage catheter through the perforated ulcer was said to be as successful as definitive therapy. High-risk peptic ulcer perforation patients can be managed by putting in an intra-abdominal drain supported by conservative treatment with reduced death rate and patients improvements.

7.3.1 Operative technique

In conjunction with conservative measures, percutaneous peritoneal drainage was performed under local anaesthesia through a 3- cm long skin incision at the level of right anterior superior iliac spine and the lateral edge of the rectus muscle. The incision spitted the external oblique aponeurosis, internal oblique and transversus abdominus along the direction of their fibers. Upon interning the peritoneal cavity, the index finger was swiped in all direction to allow protection and good drainage. A wide bored percutaneous intra-abdominal drain.

8. Aims and concerns

Type of surgery	**Morbidity**	**Total no. (%)**
Simple closure (*n* = 29)	a. Pulmonary embolism (1)	8 (27.5)
	b. Septicemia (4)	
	c. Respiratory failure (1)	
	d. Wound infection (1)	
Closure + Acid reduction procedure (*n* = 8)	a. Stomal obstruction (1)	3 (37.5)
	b. Post operative anastamotic leak with septicemia (1)	
	c. Gastric fistula (1)	
Gastric resection (*n* = 17)	a. Wound infection (1)	6 (35.2)
	b. Duodenal blow out (1)	
	c. Respiratory failure (4)	

Table 3. Morbidity related to type of surgery.

Perforated peptic ulcer disease continues to inflict high morbidity and mortality. Although patients can be stratified according to their surgical risk, optimal management has yet to be described. The accepted therapeutic options in patients with perforated peptic ulcer are simple closure or immediate definitive operation. The non-operative management of perforated peptic ulcer has previously been shown to be both safe and effective although it remains controversial. Taylor's conservative treatment, originally proposed for the treatment of choice in perforated acute peptic ulcer in 1951. Today it is reserved for patients considered to be too ill to stand the stress of surgery or in situations where immediate

surgery is unavailable. Minimal surgical intervention (percutaneous peritoneal drainage) can significantly lower the mortality rate among a selected group of critically ill, poor risk patients with perforated peptic ulcer disease.

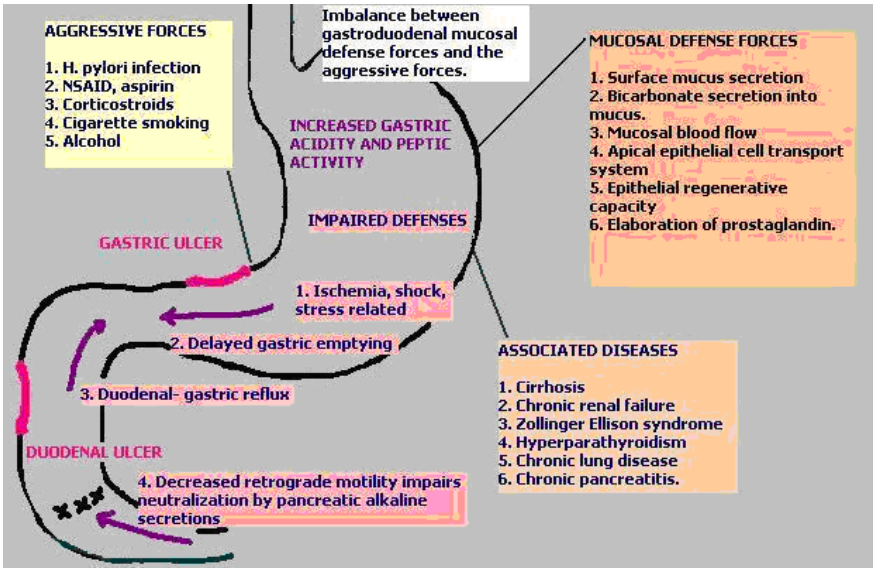

Fig. 3. Shows the imbalance between aggressive factors and protective factors in peptic ulcer disease.

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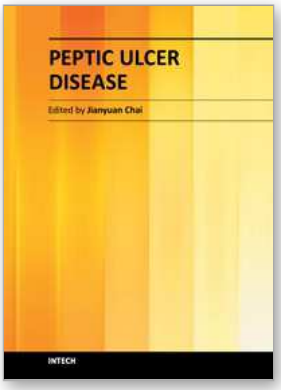

Peptic Ulcer Disease

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Peptic ulcer disease is one of the most common chronic infections in human population. Despite centuries of study, it still troubles a lot of people, especially in the third world countries, and it can lead to other more serious complications such as cancers or even to death sometimes. This book is a snapshot of the current view of peptic ulcer disease. It includes 5 sections and 25 chapters contributed by researchers from 15 countries spread out in Africa, Asia, Europe, North America and South America. It covers the causes of the disease, epidemiology, pathophysiology, molecular-cellular mechanisms, clinical care, and alternative medicine. Each chapter provides a unique view. The book is not only for professionals, but also suitable for regular readers at all levels.

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