

## UNDIAGNOSED PERFORATION OF SILENT DUODENAL ULCER : A CASE REPORT WITH REVIEW

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### Abstract

This is a case report where one Malaysian Indian female in her early fifties died due to perforation of duodenal ulcer. She was apparently well before and had attended hospital for minor complaint of gastritis on the evening before her death. GIT perforation is an acute emergency, if diagnosis is missed then it adversely affect the life of the patient. The treatment of perforation and peritonitis is very time consuming and costly, and at times results in severe infection, peritonitis, adhesions, and shock. In some cases, even sudden death do occur in few hours after perforation. In this case, patient died within hour of acute symptoms due to shock. Fatality following peptic ulcer perforation usually takes variable time, which can range from few hours to many days or week.

Peptic ulcer disease is more aggressive in menopausal women. In peptic ulcer co-morbidity factors, mainly infection due to *Helicobacter pylori* and the regular intake of non-steroidal anti-inflammatory drugs have been identified as major risk factors. On postmortem examination, one fresh perforated duodenal ulcer was found in anterior part of duodenum. Peritoneal cavity was full with stomach and GIT contents. There were findings of chemical peritonitis and shock thereof. In this case, issue of negligence was raised. All related issues of missed diagnosis, negligence, or judgmental error are discussed. The raised medico-legal issues are addressed in question and answer form.

**Keywords:** *Peptic ulcer, perforation, sudden death, chronic duodenal ulcer, peritonitis, perforated duodenal ulcer.*

### Introduction

GIT perforation is an acute emergency, which necessitates admission and surgical intervention in such cases. Gut can perforate due to variety of reasons but in upper GIT, perforation occurs due to peptic ulcers that perforate in some cases. Peptic ulcer is a general term assigned to gastric ulcers as well as to duodenal ulcers.

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Ulcers are a breach in the mucosa of the alimentary tract that extends through the muscularis mucosa into the sub mucosa or deeper.<sup>1</sup> Peptic ulcers are chronic and they may occur in the upper part of alimentary tract (mainly stomach and first part of duodenum) or where any portion of the gastrointestinal tract get exposed to the aggressive action of acid/peptic juices<sup>1</sup>.

Chronic duodenal ulcer occurs at all age groups but peak incidence is in between 25 to 50 years. Duodenal ulceration is a remitting disease characterized by periods of activity and quiescence.<sup>4</sup> Exacerbations may be associated with stress, infection or dietary indiscretions and smoking. It is more acute on fasting and is relieved on eating so patients are usually obese due to frequent and indiscriminate eating. Chronic ulceration of the stomach is less common than in the duodenum, the size ratio of gastric to duodenal ulcer varies from 1 to 4 cm. Peptic ulcers are usually solitary lesions less than 4 cm diameter.

Classical ulcer is round to oval, sharply punched out at defect with relatively straight walls. Anterior wall of duodenum is more affected than posterior wall. The depth of these ulcers varies, from superficial lesions involving only the mucosa and muscularis mucosa or it can extend into muscularis propria. Majority of people have single ulcers. Chronic gastritis is virtually universal among patients with peptic ulcer disease, occurring in 85 to 100% of patients. Pylori infection is almost demonstrable in patients with gastritis. In recent study<sup>14</sup>, it was found that H. Pylori infection was not present in duodenal ulcer perforations.

Perforation occurs in 5% of cases and it accounts for two-thirds of ulcer deaths.<sup>4</sup> Peptic ulcer healing is very variable and at times takes decades<sup>3</sup>. Perforation may be due to weakening of site due to metaplasia of cells, thrombosis, damaged mucosa, inflammatory and immunogenic response, recent systemic or local infection.

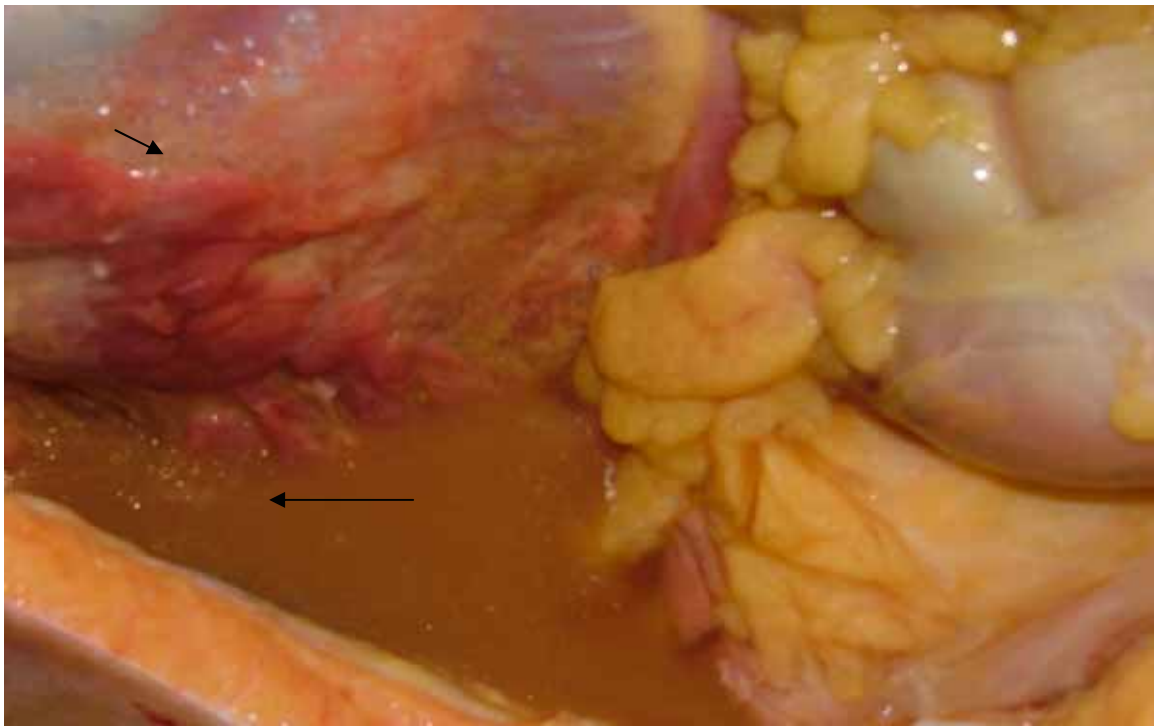
All complications result from the extension of ulceration, weakening of wall, and accompanying inflammation deeper into the wall of the gastro-duodenum.<sup>9</sup> In some rare instances tuberculosis<sup>10</sup>, sclerosis<sup>16</sup> also lead to perforations. When ulcer erodes through full thickness of gastro-duodenum, it may produce perforation or penetrate into adjacent structures. This occurs in 5-10% of patient with peptic ulcer.<sup>9</sup> Peptic ulcer hemorrhage or perforation occur in patients mostly with hitherto silent ulceration, particularly in the elderly or in those taking non-steroidal anti-inflammatory drugs<sup>15</sup>. The majority of patients dying from peptic ulceration have no symptoms of ulcer disease until presentation of their final, fatal illness.<sup>15</sup> Main complications of peptic ulcer disease are: hemorrhage, perforation, or obstruction.<sup>9,15</sup> Perforation is less common than bleeding and it occurs in about 5% of peptic ulcer patient<sup>1</sup>.

Peptic ulcers are usually located in the first part of duodenum in comparison to other sites like stomach, gastro-esophageal junction, jejunum, Meckel diverticulum in order of decreasing frequency.<sup>1</sup> Women are most affected after menopause<sup>1</sup>. Most duodenal ulcers occur within a few centimeters of the pyloric ring. The anterior wall of the duodenum is affected more often than the posterior wall. In this case, ulcer was seen in the anterior wall of first part of duodenum. The histological appearance varies from active necrosis to chronic inflammation and scarring, to healing. Perforated inflammatory condition spill gastric contents, bacterial flora in sterile peritoneum. In addition to all above this poses a serious electrolyte imbalance in the patient. In sudden natural deaths, death occurs immediately like within one hour or within 24 hours of the onset of terminal symptoms, which may be very different from the symptoms, which the patients were having so long<sup>2</sup>. Clinical history is often of value in determining the probable cause of death in such circumstances.<sup>3</sup>

## Case Report

One 51 years married female developed abdominal pain one day prior to her death. She was apparently well before and did not have any major signs and symptoms. As per the clinical notes and history given by relatives, she was diagnosed as a case of gastritis and was advised one plain erect X-ray of abdomen which was negative for pneumo-peritoneum. She had morning tea and water and then collapsed at the home in the

morning hours and was brought dead to casualty of this hospital. She died within one hour due to shock within one hour after perforation. There was no history of fever, vomiting or diarrhea. She was apparently well and working as cleaner, and was obese in built. There was no history of acute or chronic abdominal pain. She only complained of occasional vague and dull generalized ache in abdomen. There was no previous history of hypertension, diabetic mellitus arthritis, asthma, intake of steroid etc. She was not having coronary or Ischemic heart disease. There was no history of intake of aspirin on regular basis, off and on use of (panadol) pain killer was there.

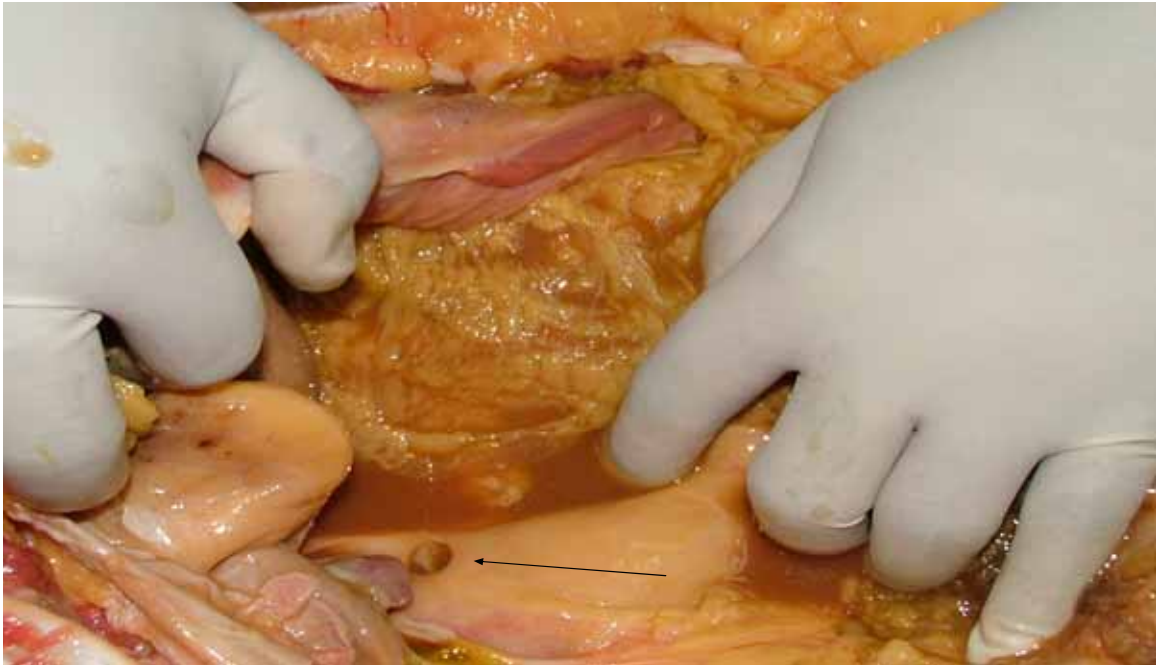


**Figure 1: Peritoneum cavity with GIT contents with inflamed peritoneum and leaked free fluid shown by arrows.**

The body was that of a well-nourished, medium sized, muscular adult Malaysian Indian female 158 cm in length and 68 kg in weight. The hands were normal and fingernails were cyanosed. Feet and toenails beds were bluish. There was 1500 ml of greenish grey to brownish colored fluid in the peritoneal cavity. There was no foul smell. Peritoneal wall was inflamed and red. There were no visible pus or fibrin tags found in the omentum or peritoneum. There was no fresh or altered blood in the stomach and intestines. We found an ulcer measuring 1.4 cm with perforation of anterior wall of duodenum. The ulcer had smooth edges and there was no eroding of blood vessels. It was not covered with omentum. Small bowel and large bowel were red in color. Appendix was a normal serosa of stomach, small bowel, ascending colon, transverse colon red and congested due to inflammation, omentum was red in color. There was no fibrin deposited on the omentum.

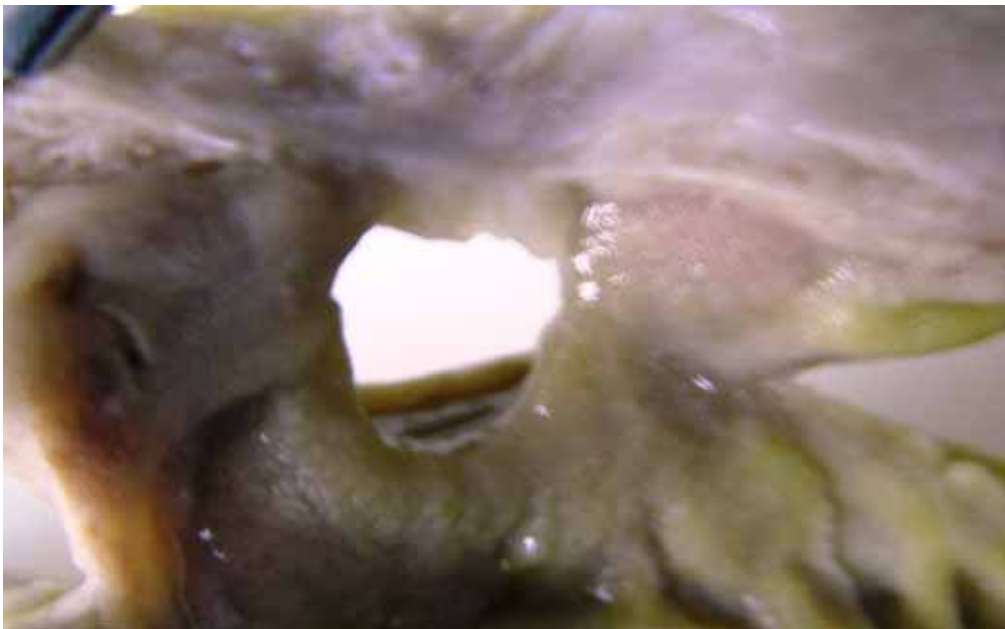
There was a perforated peptic ulcer in the anterior aspect of pylorus antrum. The opening was not sealed and still fluid was leaking from the perforated ulcer. It was round in shape and diameter about one cm.

Stomach lumen was opened through its greater curvature. Stomach mucosa was normal except at the pylorus antrum, as shown below:



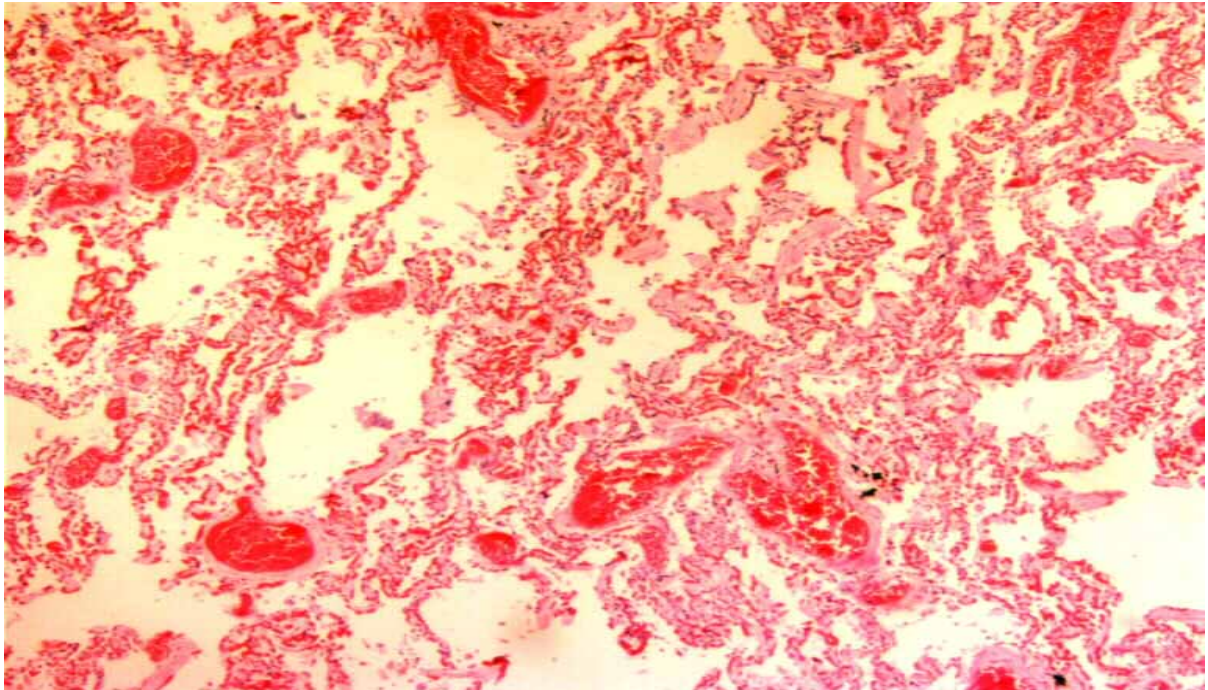
**Figure 2: Perforated duodenal ulcer with gastric contents spillage and inflamed peritoneum**

There was one perforated chronic duodenal ulcer in the wall of anterior surface of pylorus antrum. It was in round shape, the edges were smooth and there was no eroded blood vessel at the margin. There was one more ulcer with whitish base in the beginning of pylorus.



**Figure 3: Inner view of duodenal ulcer after preservation in formalin**

There were no polyps or diverticulitis in the intestine, and perforation or inflammation of appendix. There were no enlarged mesenteric, Para aortic lymph nodes. There was no blood in the lumen of small or large intestine. Rest of the systems were unremarkable. Microscopy did not show any remarkable finding except little gastritis.



**Figure 4: Shock Lung**

## Discussion

The pain of peptic ulcers is described as tender, burning, gnawing or aching. It is also correct that silent ulcers are also seen with no clinical symptoms.<sup>15</sup> Two duodenal ulcer perforations were silent and were only detected at autopsy as intestinal perforation in systemic sclerosis.<sup>16</sup> The majority of patients dying from peptic ulceration have no symptoms of ulcer disease until presentation of their final, fatal illness.<sup>15</sup>

In western industrialized nations, peptic ulcers develop in up to 10% of the general population at some point during life<sup>1</sup>. In one clinical study<sup>5</sup> here, a total number of 385 of upper GIT bleeding were examined. In this study, 196 patients were found to be bleeding from peptic ulcers. This group constituted 66 Malays, 116 Chinese, and 14 Indians who showed active bleeding ulcers on endoscopies. In this study majority, 85 % (167/196) had size less than 2 cm, and only 28 out of 196 had size more than 2 cm. In Malaysia, uncomplicated peptic ulcer is reported to be disproportionately high in the Chinese and Indians and low in Malay.<sup>5</sup>

Death is not very common in gastric perforation; it takes time ranging from immediate to few days to weeks depending on gut contents and infection, and only has 5 % fatality<sup>1</sup>. In another study<sup>6</sup>, the incidence of perforated peptic ulcer is one for every 160-hospital admissions. The incidence in the general worker population was one in 2633 persons per year. All perforations were found in males; Fifty five percent of the patients with perforated ulcers were between 40 and 60 years of age; 90 were between 30 and 60 years of age. Eighteen percent of the patients had no symptoms before the time of perforation. Perforation of the duodenum were twice as frequent as those of the stomach.<sup>6</sup>

In this case, age was 51years old, menopausal woman in contrast to above where all patients were male. In two case reports<sup>7</sup> where there was sudden death due to perforation of benign esophageal ulcers into thoracic aorta in

one case and in another it was due to perforation of left pulmonary vein.<sup>7</sup> In this case, sudden death was from shock without invading any vessel and associated bleeding.

In this case, relatives were upset about diagnosis and were adamant to lodge a case of negligence. Now main question of concern was whether there was any negligence on part of hospital or it was inadvertent error? Answers to the common medico-legal questions arising from such a case are discussed below as:

**Q1. Is the perforation of ulcers a known complication of peptic ulcer disease?**

A. It has three complications: hemorrhage, perforation or obstruction. Perforation of peptic ulcer is a known cause and can happen in chronic ulcerative disease in about 5 % of total cases<sup>1</sup>, and is possible in acute corrosive poisoning. In this, there was nothing suggestive of poisoning. Perforation occurs in 5% of cases out of total ulcer cases and it accounts for two-thirds of ulcer deaths, in total perforated cases.<sup>4</sup> In another study<sup>6</sup>, the incidence of perforated peptic ulcer is one for every 160-hospital admissions. It was a clear case of chronic duodenal ulcer, the margins of the ulcers were indurated, thickened, and rounded.

**Q2. Was she having perforation when she attended clinic on previous evening prior to her death.**

A. The perforation can easily be detected by presence of pneumoperitoneum on simple upright and left lateral decubitus position X-ray. There is usually a very tense abdomen on clinical examination. X-ray was negative of pneumo-peritoneum. The peritoneum showed only acute inflammatory changes, there was nothing indicative of old peritonitis. There was no fibrin, adhesions, fat necrosis, or pus pockets in the area. The peritoneum showed only acute inflammatory changes and it was not sealed with omentum which also indicated early incident.

**Q3. Patient was having truncal obesity so could it be missed on clinical evaluation.**

A. Yes, it can be missed but it was not in this case as all other measures were taken in addition to clinical examination. There is nothing to suggest on clinical examination as well as in investigation to sustain these charges.

**Q4. How much time will be sufficient to kill a person after untreated perforation?**

A. In such cases time limit varies and it depends on the type of perforation, gastric or GIT contents, and distress on respiratory system due to tense abdomen. Ulcer perforation is a serious condition characterized by severe pain, shock, collapse and intensely rigid abdomen. If perforations are presented beyond 24 hours then chances are very bleak due to widespread infection, fluid loss, pressure on lungs. In large perforation where bleeding is there or large volume of gas is present, the danger of fatality is immediate due to shock and respiratory distress. In this case, there was gap of 14 hours between being brought dead to hospital and her last attendance in the hospital. Now one genuine question can be whether, perforation occurred somewhere in the night. In this case, the lungs were partly collapsed and showed marked congestion and interstitial hemorrhages – a typical finding of shock lung on histo-pathological examination. There are more fair chances to conclude by all means that it could have happened few hours before death and patient went into shock as evident from lung findings.

**Q 5. Whether, it can be termed as Medical negligence? Was there an error of diagnosis? Alternatively, was it a case of medical negligence?**

Ans. No, error of diagnosis is not a medical negligence. If the action or omission is done in good faith but result is suboptimum, it is an error of judgment and not negligence.<sup>18</sup> Medical negligence is a gross lack of care and skills. Doctors should try to make diagnosis after proper examination and investigations, if needed.

A competent doctor is able to reach to a conclusion after excluding other possibilities of similar presentations on diagnosis or differential diagnosis. There is no evidence of peptic ulcer in the history and patient had nonspecific symptoms. She had a distended stomach and truncal obesity with lax abdominal wall.

Diagnosis is to identify one situation out of many probabilities. Every doctor is duty bound to make a diagnosis. In this case, diagnosis of gastritis was made and was treated accordingly. There were enough efforts to make a diagnosis with all reasonable means. There was a reasonable effort to deal with the case so it cannot be termed as Medical negligence. It also cannot be equated as missed diagnosis as attempt in clinical examination as well as in getting investigations were also tried by the treating doctor.

It would have been a case of misdiagnosis, if perforation had been present at the time of attending hospital on last evening. There is no evidence to prove that. If this perforation would have happened in the night then patient would have disturbed night and respiratory distress as abdomen becomes tense due to collection of many liters of free gases. Moreover, postoperative mortality is about 11 % as assessed in 263 operations.<sup>17</sup>

**Q6. What is the role of sub clinical infection in causing perforation of duodenal ulcers?**

A. H pylori are known to be a major cause of peptic ulcers. H pylori weaken the protective mucous coating of the stomach and duodenum, which allows acid to get through to the sensitive lining beneath. Both the acid and the bacteria irritate the lining and cause a sore, or ulcer, H pylori then produces a number of toxins and factors that in certain individuals cause inflammation and damage to the lining, leading to ulcer. It also alters certain immune factors. These infections weaken the wall and then help in perforation.

**Q7. Which ulcers do perforate early?**

. Acute ulcers due to infection and corrosives rupture more early in comparison to chronic ulcers due to weakened wall and erosion of layers. At times ulcers are diagnosed only after perforation<sup>1</sup>

**Conclusion**

Medical negligence is ‘easy’ to say but it is difficult to prove . Any premature opinion before considering all facts and review of literature can be disastrous for accused as well as to the person who is venturing such opinion. Here , medical negligence does come under per view of police as it happens during discharge of professional duties. Any complaint about negligence has to be made before professional body and it is purely a civil matter.

**References**

1. Kumar V, Abbas AK, Fuasto N .In: Robbins and Crotran.7<sup>th</sup> Ed, Elsevier Saunders, Philadelphia, 2005: 816-820.
2. Knight B. Simpson’s Forensic Medicine 11<sup>th</sup> Edition. Hodder Headline group. London. 1997: 105-12.
3. Sandler RS et al. The burden of selected digestive diseases in the United States. Gastroenterology 2002;122: 1500.
4. Alfred C, Robert JC, Steek A, Rahim M. In: Essential Surgical Practice, Vol1, Arnold, 4<sup>th</sup> edn. 2002: 282.
5. Kandaasami P, Harjit K, Hanafiah H. Clinical and endoscopic features of peptic ulcer bleeding in Malaysia. Med J Malaysia 2004; 59(5): 617-622.

6. Leo D. Nannini. An analysis of Acute perforated ulcers commentary by Paul Smith. In: <http://xnet.kp.org/permanentejournal/winter99pj/perspective.html> . Accessed on 15/2/06
7. Mo KM, Craig GM, Clark JV, Champ C. Sudden death from perforation of benign esophageal ulcer into a major blood vessel. *Postgrad Med J* 1988 Sept; 64:687-89.
8. Peptic Ulcer. In. <http://hopstechno.com/book35.htm> . Accessed on 15/2/2006
9. Schwartz SJ, Shies GT , Spencer C Frank et al. In: Principles of Surgery, Vol I , McGraw Hill , 7 edn.,1999:1194-1195.
10. Berney T , Badaour E, Totsch M , Mentha G , Morel P. Duodenal tunerculosis presenting as acute ulcer perforation. *Am J Gastroenterology* 1998; 93(10):1989
11. Arnaud JP, TuechJJ, RobertoB , Pessaux P , Nicolas R. Laparoscopic suture of perforated Duodenal Ulcer. *Surgical Laparoscopic, Endoscopy Percutaneous Techniques* 2002;12(3)145-147.
12. Chowdhary SK , Bhasin DK , Panigrahi D, Malik AK , Kataria RN , Behra A , Roy P , Singh K. Helicobacter pylori infection in patients with perforated duodenal ulcer. *Trop Gastroenterol* 1998;19(1):19-21.
13. Boey J ,Wong J. Perforated Duodenal Ulcers. *World J Sugery*1987;11(3):1432-2323.
14. Gupta S, Kaushik R, Sharma R, Attri A. The management of large perforations of duodenal ulcers. *BMC Surgery* 2005;5:15. Accessed on <http://www.biomedcentral.com/1471-2482/5/15> on 16/2/2006.
15. Pounder R. Silent peptic ulceration : deadly silence or golden silence? *Gastroenterology*1996; (2):626-31.
16. Ellen CE , Ruggiero FM , Seibold JR. Intestinal perforation( A common complication of scleroderma). *Digestive Diseases Sci* 1997;42(3):549-553.
17. Semenov VV , Sipratov, Zinov'sev SV. Results of the surgical treatment of perforated ulcer of the stomach and duodenum. *Khirurgiia (Mosk)* 1989;Oct;(10): 94-8.
18. Ganguly Mukherjee G, Soonawala R, Tank DK. In: *Medico-legal Aspects in Obstetrics and Gynaecology* , 1<sup>st</sup> ed. Jaypee Brothers ,New Delhi , 1997:24.