



Case Study of Perforated Duodenal Ulcer

Frouk Elrashid Mustafa Omer¹, Mohammed Al-rasheed Mostafa Abueissa²

¹. Department of Emergency, Cairo University, Elzahra private Hospitals KSA

² Cairo University, General physician

Abstract: Duodenal perforation is a rare but lethal condition. [1] The mortality rate ranges reported from 8% to 25% in the literature. [2][3][4]. In 1688, the perforated duodenal ulcer was described by Muralto and reported by Lenepneau. [5] Subsequently. In 1929, Cellan-Jones described a technique for repairing perforations by using an omental, and later in 1937, Graham has modified that technique. [7][5]. Typically, patients with duodenal ulcers have nocturnal abdominal pain or feel hungry. If perforation occurs, it usually can cause sudden onset of severe pain in the upper abdomen. [9]. Imaging has an essential role in diagnosis, and subsequently, for early resuscitation. Appropriate selection of therapeutic alternatives and risk-assessment can decrease the risk of morbidity and mortality. [10]

Keywords: Duodenal ulcer, duodenal perforation, Peptic ulcer

Patient consent was obtained.

I. CASE PRESENTATION

A 46-year-old Bangladeshi female patient was brought by an ambulance complaining of severe abdominal pain, shortness of breath (SOB) and repeated vomiting. She does not have in history of medical importance. The condition was started suddenly 2 days ago with abdominal pain epigastric area and refereed to tip of right scapula. According to the patient the pain is the first time in last 6 month ,sharping in nature ,continues ,8 grade in pain grade scale, no reliving factors and increase with hunger. The patient denied syncopal attack associated with her condition. Patient was not smoker and denied tacking neither elicits drugs nor alcohol. No past family and surgical histories of medical importance. On time of examination, her blood pressure was 100\67mmhg, pulse rate was 112 beat per min, respiratory rate 22 spo2 on room air 96 and her temperature was 37. There were no abnormal findings on respiratory and neurological examinations. Rather than abdominal examination revealed epigastric tenderness and whole abdomen was rigid and Lab findings were unremarkable rather than elevated WBCS (15000) in CBC. Abdomen erect and supine X-ray revealed air black rim under diaphragm. Abdominal U\S revealed free fluid in abdomen

II. Management and outcome

In resustation room, patient received pain killer (Perthidene), ant emetic (PREMPRAN injection), normal saline and Patient was shifted for emergency laparotomy with a diagnosis of peritonitis possibly due to perforated ulcer. On opening the abdomen, 450 mL of pus was drained; a perforation of about 8 mm diameter was located on the anterior wall of first part of duodenum. After that, whole of abdomen was irrigated with normal saline. Postoperatively patient was kept nil per orally and intravenous fluids were supplemented for initial two days of surgery. Then discharged with outpatient clinic follow up.

III. INCIDENCE

Mortality rate in perforation peptic ulcer (PPU) patients reported ranging from 1.3% to 20%. [11][12][13] Other studies have reported 30 days mortality rate reaching 20%. [14] The primary prognostic factor is the time interval between the duodenal perforation and treatment. The interval time greater than 24 hours increases mortality. [5] The American Society of Anesthesiologists (ASA) score and Boey score are the most common validated scoring systems to predict outcomes in duodenal perforation. [15]. Studies show the mortality rate is higher in patients older than 65-year-old compared to younger patients. [16]. The postoperative mortality rate in patients with PPU is estimated to be 6% to 10%. [16] Delays in treatment greater than 24 hours, age more than 60 years old, concomitant diseases, and systolic blood pressure less than 100 mmHg are the main risk factors that increase the mortality rate. [17]

Although the incidence of PUD has decreased in recent years, it is still the prominent cause of duodenal perforation. [11] Annually peptic ulcer disease (PUD) affects 4 million people worldwide, with an incidence rate of 1.5% to 3%. [18][19] Decreased rates of PUD are explainable by eradication treatment for *Helicobacter pylori* and the use of proton pump inhibitors (PPIs). However, the perforation of peptic ulcers is still concerning. The aging population, overuse of non-steroidal anti-inflammatory drugs (NSAIDs), NSAIDs interaction with selective serotonin reuptake inhibitors, and steroids are possible reasons which can explain the high incidence of perforation. [4][20] Studies show more ulcer perforation occurs in the morning, and the mechanism is explainable by circadian variation in acid-secretion. [10] Less than 2% of traumatic abdominal injuries involve the duodenum. [21]

Less than 1% of ingested foreign bodies can cause gastrointestinal perforations. [22]

Pathophysiology

The leading causes of perforated peptic ulcers remain NSAIDs use and *H. pylori*. [11] NSAIDs can reduce prostaglandin secretion by inhibiting COX-1 in the gastrointestinal tract, leading to a gastro duodenal mucosal injury. *H. pylori* infection is another contributory factor for duodenal ulcers. Possibly, *H. pylori*, by inducing gastric metaplasia, stimulating immune response and gastric acid secretion, and reduce mucosal defense, can cause duodenal injury and subsequently duodenal perforation. [23]

Presentation of the patient

The classic triad in PPU patients is tachycardia, sudden onset of abdominal pain, and abdominal rigidity. Abdominal tenderness with rigidity and tachycardia are common clinical signs. [9]

IV. COMPLICATION

BLEEDING

The incidence of gastro duodenal bleeding secondary to acid-peptic disease and hospital admissions for this complication have not significantly changed in the last 2 decades. [24]-[25] mortality rates following ulcer bleeding have remained at approximately 10%. [26]-[27]

Gastric Outlet Obstruction

(GOO) Benign GOO secondary to peptic ulcer disease remains prevalent and represents approximately 5% to 8% of ulcer related complications [28]

Sepsis is common and accounts for 40% to 50% [10]

Postoperative complications were reported in 30% of patients. [32]

Laboratory Tests

Leukocytosis and high C-reactive protein level indicate existing of inflammation or infection. [29].

Serum gastrin levels are useful in patients with a history of recurrent ulcers to establish the diagnosis of Zollinger-Ellison syndrome.[13]

CHEST XRY

In acute upper abdominal pain, an urgent upright chest radiograph (CXR) is a basic essential test for duodenal perforation. Upright CXR shows free air below the diaphragm in 75% of patients. However, it can be normal, especially in patients who present early after symptoms initiation.[31]

CT

Double-contrast computed tomography (CT) scan is the most valuable method for diagnosing duodenal perforation. It should be performed whenever there is a clinical suspicion and the patient does not need immediate surgery. CT features of perforation include discontinuity of the duodenal wall and the presence of extra luminal air or extravasated oral contrast.[30]

V. PROGNOSIS

Mortality rate in perforation peptic ulcer (PPU) patients reported ranging from 1.3% to 20%.[11][12][13] Other studies have reported 30 days mortality rate reaching 20%.[20] Significant risk factors that increase the mortality rate are co-morbidities, resection surgery, presence of shock at admission, female, elderly patients, metabolic acidosis, a delayed presentation of more than 24 hours, acute renal failure, hypoalbuminemia, smokers, and being underweight.[33][34]

VI. Management and treatment

Management of duodenal perforations needs immediate surgical procedure. The choice of surgical treatment depends on the size and localization of the perforation

VII. Conclusion

Duodenal perforation is a rare, but potentially life-threatening injury. Multiple etiologies are associated with duodenal perforations such as peptic ulcer disease and H.Pylori infection . Computed tomography with intravenous and oral contrast is the most valuable imaging technique to identify duodenal perforation. Urgent upright chest radiograph (CXR) is a basic essential test.Immediate surgery is required for patients presenting with peritonitis and/or intra-abdominal sepsis.

VIII. References

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