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Giant Perforated Duodenal Ulcer with Complete Common Bile Duct Erosion in an Elderly Patient: A Rare, Fatal Complication of NSAID Use - First Reported Case Worldwide

Islam S[1,2], Dial S[1,2], Maughn A[1,2], Harnarayan P[1,2]

[1]Department of General Surgery, San Fernando General Hospital, San Fernando, Trinidad & Tobago; [2]Department of Clinical Surgical Science, University of the West Indies, St. Augustine, Trinidad & Tobago.

Corresponding Author:
Dr Shariful Islam
Email: sssl201198@yahoo.com

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Abstract

Background: The liberal use of non-steroidal anti-inflammatory drugs (NSAIDs) can be potentially fatal, especially in the elderly. NSAIDs are commonly associated with peptic ulcer diseases (PUD), may lead to bleeding and perforations. However, PUD perforation associated with complete erosion of common bile duct (CBD) has not been reported in the English literature before. We present the world's first documented case of complete erosion of CBD with duodenal ulcer (DU) perforation secondary to the use of NSAIDs in an elderly patient. *Case Report*: An 87-year-old female with one month history of right hip replacement presented to the emergency department with two-day history of epigastric pain. She had been using NSAIDS (diclofenac) for her hip pain. On examination she was found to be in septic shock with a peritonitis abdomen, and erect chest X-ray revealed a large pneumo-peritoneum. After resuscitation, patient consented for an emergency exploratory laparotomy. Intra-operatively, a giant ulcer involving 70-80% the circumferences of the first part of the duodenum with complete erosion of the common bile duct were discovered. Damage control surgery was done, but the patient became hemodynamically unstable post-operatively and passed away on day two of the surgery. *Conclusion*: While NSAIDs are widely used for pain management, careful consideration is necessary when prescribing them to elderly patients. This case highlights the potentially catastrophic complications of NSAID use in older adults.

Keywords: Duodenal Ulcer, Elderly, NSAIDs, Outcomes, Perforation.

Introduction

Peptic ulcers are defined as erosions in the gastric or duodenal mucosa that extends through the muscularis mucosa. Giant duodenal ulcers (GDUs) are a subset of peptic ulcers and are defined as a benign, full thickness ulcer at least 2 cm in diameter, usually involving a large portion of the duodenal bulb [1]. There are multiple risk factors to consider in the pathogenesis of peptic ulcer disease (PUD); tobacco use, alcohol abuse and steroids, non-steroidal anti-inflammatory drugs (NSAIDs), and *Helicobacter pylori* (*H. pylori*) infection [2]. A steady decrease in the prevalence of *H. pylori* infection has been observed in most populations in recent decades [3,4]. This change may represent a birth-cohort pattern [4]. Some factors contributing to this observation include improved sanitation, decreasing family size, changes in dietary habits and improvements in refrigeration equipment [3].

In 1897, aspirin, the first NSAID was made available by Felix Hoffman of the Bayer Company. Later, in the early 1970s, the mechanism of action was established and multiple other NSAIDs were made available for use [5]. Since its introduction, it has been noted that NSAIDs have increasingly become the leading causative factor in the development of PUD [2,6]. In our case study, we highlight a devastating complication of NSAIDs,

a giant perforated duodenal ulcer with complete erosion of the common bile duct- possibly the first to be described in the literature.

Case Report

A female patient in her late 80s presented to our accident and emergency department with two (2) day history of intermittent burning epigastric pain with no association with meals and was partially relieved by liquid antacid. The patient reported that her condition had worsened over the last 24 hours; the pain became continuous and generalised. The patient also gave history of nausea and vomiting several times over the last 24 hours and had not passed any flatus or any bowel action during this time. She had no co-morbid condition except severe osteoarthritis of right hip joint for which she underwent right hip replacement one month ago. Her post-operative recovery was uneventful except mild to moderate pain in her right hip. She was subsequently evaluated by a general practitioner and prescribed NSAIDs (diclofenac 75 mg SR, twice daily), which she has been taking for the past two weeks. On examination she was found to be ill looking, her mucous membranes were dry, mildly pale, and anicteric. She was tachycardic with a pulse of 120 beats per minute, hypotensive 90/60 mm Hg, respiratory rate of 26 beats per minute and SpO_2 of 94% on room air. Her abdomen was grossly distended, tense and tender with board like rigidity. There was no palpable mass or hernia noted and the bowel sounds were absent. Digital rectal examination was normal and there was no blood on gloves.

Blood investigations revealed an elevated total leukocyte count of 19×10^9/L with hemoglobin of 10.5 gm/dL, and a mildly deranged renal function with a serum creatinine of 2.4 mg/dL, but normal serum electrolytes. An erect chest X-ray PA view including both domes of diaphragm demonstrated huge pneumo-peritoneum under right dome of diaphragm **[Fig.1]** and the electrocardiogram (ECG) revealed sinus tachycardia. An arterial blood gas analysis revealed mild metabolic acidosis.

The patient was resuscitated, and broad-spectrum antibiotics were initiated. She was then evaluated by a cardiologist and the on-call anaesthesiologist. A portable echocardiogram showed no signs of vegetation, thrombus, or any other cardiac abnormalities, but revealed an ejection fraction (EF) of 28%. A multidisciplinary team meeting, involving the cardiologist and anaesthesiologist, was conducted. The patient and her family were thoroughly counselled regarding the diagnosis, the need for emergency lifesaving laparotomy, and the high risk involved. Consent for the surgery was obtained. Under general anaesthesia with endotracheal tube intubation entire abdomen was prepared and draped. A midline laparotomy was performed, revealing moderate bilious fluid on entering the abdomen **[Fig.2]**. Initial exploration revealed a giant posterior ulcer was noted involving 70-80% the circumferences of the first part of the duodenum **[Fig.3]**. Further exploration showed complete erosion of the common bile duct **[Fig.4]**

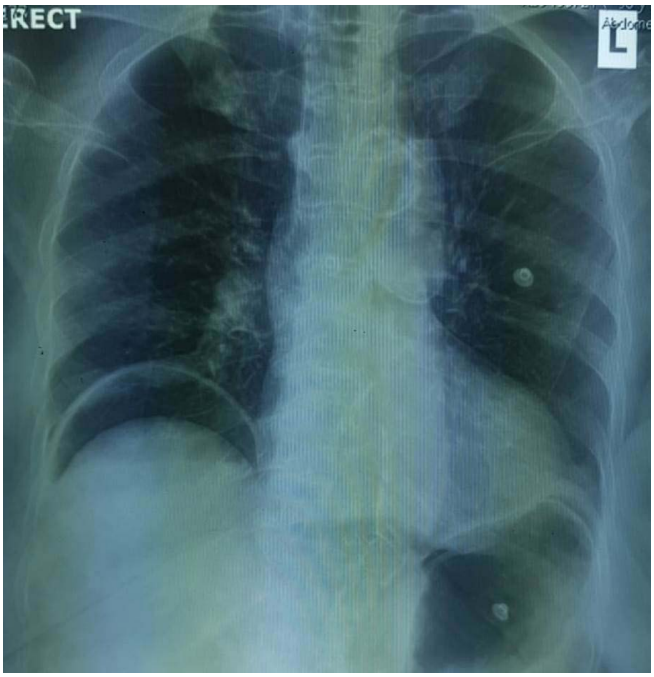

Fig.1: *Erect chest X-ray PA view showing free air under right dome of the diaphragm.*

exposing distal free lumen of the common hepatic duct **[Fig.5]**.

The patient remained hemodynamically unstable on the table requiring double inotropes; hence a damage control laparotomy consisting of antrectomy with Foleys catheter drainage of the biliary tree was performed. She was then admitted into the intensive care unit for stabilization and optimization followed by second look laparotomy plus minus GI and biliary reconstruction. However

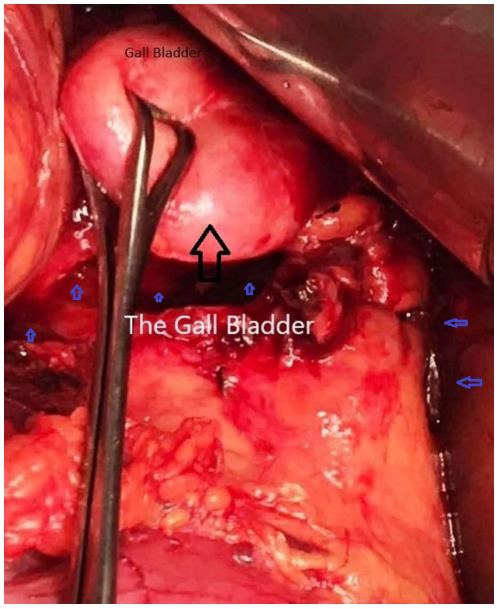

Fig.2: *Intra-operative picture demonstrating free bile in abdomen (blue arrows) and the gall bladder (black arrow).*

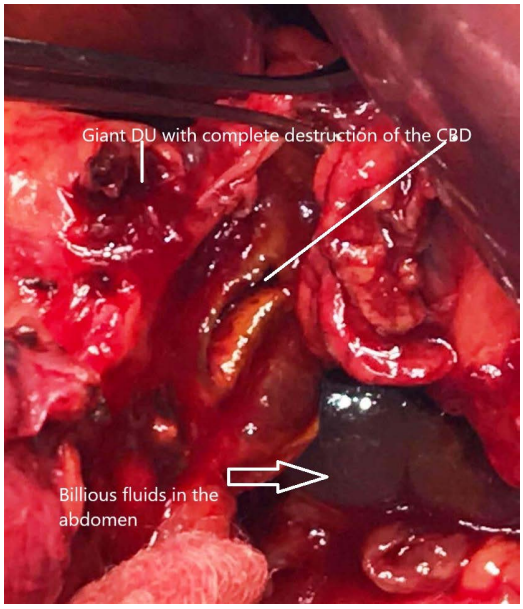

Fig.4: *Intra-operative image demonstrating giant duodenal ulcer perforation with complete destruction of the common bile duct (CBD) - white lines.*

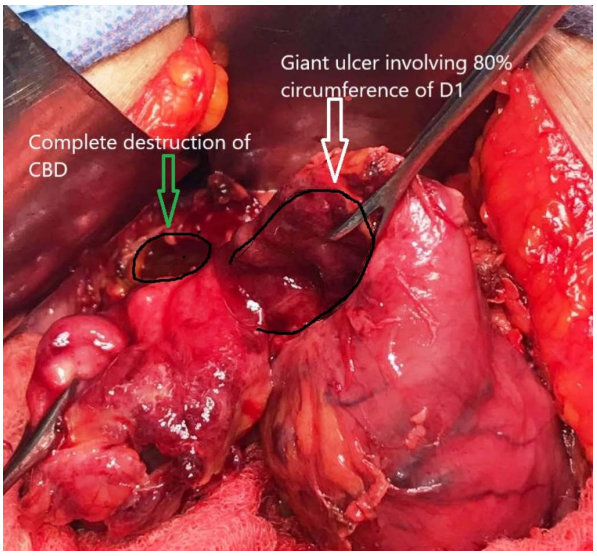

Fig.3: *Intra-operative image showing giant ulcer involving 80% circumference of the first part of the duodenum (D1- white arrow) and complete destruction of the common bile duct (CBD- green arrow).*

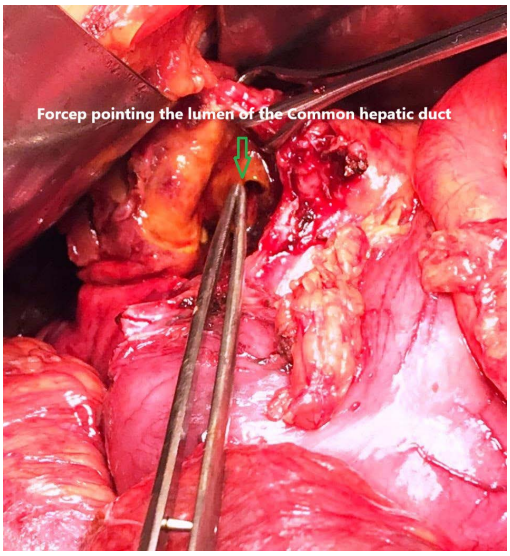

Fig.5: *Intra-operative photo showing free distal lumen of the common hepatic duct (green arrow).*

patient condition worsened post-operatively requiring multi-organ support. Unfortunately, she passed away on the 2nd post-operative day.

Discussion

NSAIDs have been in around for centuries, beginning with the use of Willow bark. There were two periods in history when the evolution of the pharmaceutical aspect of the drug occurred. Firstly in 1897 when aspirin was isolated and made available, then in the early 1970s when the mechanism of action was discovered and other NSAIDs were made available [5]. The mechanism of action is attributed to the inhibition of prostaglandin (PG) synthesis which is important in the inflammatory process. PG also plays a vital role in the natural protection of the gastric mucosa. It inhibits acid secretion, stimulates mucus and bicarbonate secretion and alters mucosal blood flow, all of which has been shown to protect the gastric mucosa [7]. With the use of non-selective NSAIDs, this gastric protective aspect of PG is lost, resulting in a mucosal lining which is susceptible to damage and ulceration [8].

GDUs are a subset of PUD, which can be described as "duodenal ulcers on steroids". GDUs are more extensive, may involve surrounding structures and cause more morbidity and mortality than the usually encountered duodenal ulcers. GDUs may present similarly to the usual peptic ulcers with perforation, bleeding or even gastric outlet obstruction. Most giant duodenal ulcers are situated posteriorly, destroying a large part of the wall of the bulb, or of the duodenum just distal to the bulb. They have also been found to penetrate deeply into the pancreas as described by K. Lumsden *et al*. [9].

In our review of the literature, we have not encountered any case similar to our case, with such extensive ulceration involving the common bile duct. It is possibly the first such case describing a duodenal ulcer with resultant choledochal-duodenal fistula. Review of data by E. B Newton *et al*. suggests that daily NSAID use plays a more prominent role in the formation of GDUs than in standard sized duodenal ulcers [10]. NSAIDs use have increased and is now frequently prescribed by physicians as well as sold over the counter, being used for pains and inflammation [8,11]. Despite its harmful side effects, NSAIDs have also been frequently used in the elderly populations [8]. Multiple studies have shown that some prescribing health care professionals and most of the using population are unaware of the possible side effects of NSAIDs [12-15].

Given our patient's history, it is quite possible that multiple factors lead to the development of her peptic ulcer however, most notably is the use of NSAIDs which preceded her abdominal symptoms. We acknowledge the importance of NSAIDs to treat pain and inflammation. There is no doubting its effectiveness. However, given the severity of possible side effects and the data supporting lack of awareness of such, it is our duty as health care providers to educate our patients. This case highlights and serves as a reminder that the use of NSAIDs should be judicious. In so doing we may be able to take precautions and possibly avoid catastrophic side effects.

Conclusion

NSAIDs have become a staple in pain management, but its liberal use can lead to a potentially fatal outcome, especially in the elderly. Due precautions need to be taken by the general practitioners while prescribing it to the vulnerable groups. Patient education on the use and side effects of NSAIDs is off utmost importance to avoid any undue delays in presentation to the emergency department to decrease possible catastrophic outcome. Our case highlights one of catastrophic side effects of NSAIDs in elderly with a fatal outcome and serves as a reminder that its use should be judicious.

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