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Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) Use as Cause Of Giant Ulcer In Gastric Antrum

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Abstract: ***Female, 61 years old, Balinese, came with chief complaint of hematemesis and melena. History of taking NSAIDs for a couple of years. Her esophagogastroduodenoscopy show giant ulcer with white base on gastric antrum. She treated with esomeprazole, antacids and sucralfate for 6 days in ward. The patient show good response and continue medication outpatient.***

Keyword: NSAIDs, Giant Ulcer

1. Background

Non-Steroidal anti-inflammatory drugs (NSAIDs) is widely used for treat and reduce pain on patient. NSAIDs uses and H. Pylori infection is two common cause of peptic ulcer. NSAIDs can initiate gastroduodenal ulceration and promote complication such as bleeding and perforation by interfering with the ability of the proximal gastrointestinal tract to maintain its defensive capabilities. Almost 5 to 20 percent of patients who use NSAIDs over long periods develop peptic ulcer disease [1, 2].

2. Case Presentation

Female, 61 years old, Balinese, came with chief complaint of hematemesis in the last 3 hours prior to admission. These were also accompanied by pain in the center of stomach or after meal and sometimes at night. The patient has melena for 2 weeks ago. There was history of taking NSAIDs almost every day because patient have osteoarthritis since a few years ago. There was no history of decreased body weight. There was no history of taking alcohol and smoking. Previous illness such as diabetes mellitus, cardiovascular disease and kidney disease not reported.

On Physical examination, it was found that her blood pressure was 110/70 mmHg, pulse rate 78 times/minute, respiration rate 20 times/minute. Anemic conjunctiva, tenderness on palpation of epigastrium. Laboratory result showed blood count: Hemoglobin: 8.5 gr/dl, WBC: 11.73 x 10^3, Platelet: 217 x 10^3, MCV: 95.3, MCH: 30.8. Liver function test and kidney function test was normal. The patient diagnosed with hematemesis melena causa suspect peptic ulcer and mild anemia. The patient was then admitted to ward and giving infusion normal saline, proton pump inhibitor esomeprazole 40 mg bolus intravenous two times daily, antacid 15ml three times daily, sucralfate 15 ml three times daily and planning for packed red cells transfusion with hemoglobin target above 10 gr/dl. The patient was planned for esophagogastroduodenoscopy to evaluate the source of hematemesis and melena.

Esophagogastroduodenoscopy result: esophagus was normal, there was giant ulcers with white base at the antrum region, duodenum was normal. The conclusion of esophagogastroduodenoscopy was giant ulcer (Forrest III) on antrum region.

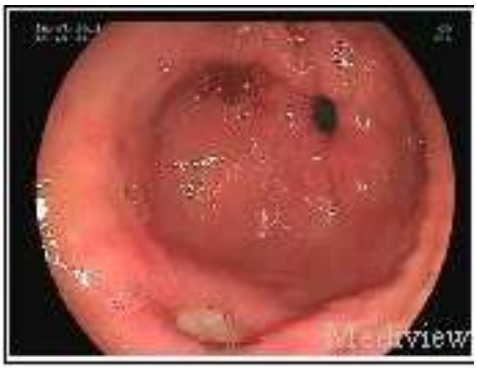

Figure 1: giant ulcers with white base at the antrum region

Biopsy done on the antrum area. The specimen evaluated by Pathology Anatomy department. Biopsy result shown: Conventional morphological features for superficial chronic gastritis and negative impression of H. pylori bacteria

3. Discussion

Peptic ulcer is a problem of the gastrointestinal tract that characterized by mucosal damage secondary to pepsin and gastric acid secretion. It usually occurs in the stomach and proximal duodenum; less commonly in the lower esophagus, the distal duodenum or the jejunum. Epigastric pain is the most common symptom [1].

Peptic ulcer affects approximately 4.5 million people every year in US. Approximately 10% of the US population has evidence of a duodenal ulcer at some time. Of those infected with *H pylori*, the lifetime prevalence is approximately 20% [3].

A peptic ulcer is a defect in the upper gastrointestinal mucosa that extends through the muscularis mucosa into deeper layers of the gut wall. There are two major risk factors for peptic ulcer disease – *Helicobacter pylori* and non-steroidal anti-inflammatory drugs (NSAIDs) [2].

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) are some of the most commonly used drugs. NSAIDs work mediated by their inhibition of prostanoid biosynthesis. Prostanoid

derivates arise from conversion of arachidonic acid by cyclo-oxygenase (COX) isoenzymes following cell injury [2].

NSAIDs are valuable agents in the treatment of arthritis and many other musculoskeletal disorders, and as analgesics in a wide variety of clinical scenarios. However, as stated above, their use has been limited mainly by their association with mucosal injury to the upper gastrointestinal tract, including the development of peptic ulcer disease and its complications, most notably upper gastrointestinal hemorrhage and perforation [4].

There are two distinct isoforms of COX. COX-1 is present in majority of cell including endothelial cell, gastrointestinal epithelium and platelets, and function continuously. COX-2 only present in few tissues and induced by inflammation. The therapeutic anti-inflammatory and analgesia effect by inhibiting COX-2. Majority NSAIDs work by inhibiting COX-1 and COX-2 (nonselective) [2,5].

Peptic ulcer was one of NSAIDs using complication. Inhibition of COX-1 in the gastrointestinal tract lead to reduction of prostaglandin secretion and its cytoprotectively effects in gastric mucosa, therefore increases the susceptibility to mucosal injury [2].

The pathophysiological features of toxic effects on the gastrointestinal tract relate to a suppression of gastric prostaglandins. The reduction in prostaglandins leads to a decrease in epithelial mucus, bicarbonate secretion, mucosal perfusion, epithelial proliferation, and ultimately the mucosal resistance to injury. The risk of life-threatening ulcer complications in long-term NSAID use ranges from 1% to 4% [6].

Risk factors for gastroduodenal bleeding in patients on NSAID therapy are age, prior peptic ulcer and co-medication with anti-platelet agents, anticoagulants, glucocorticosteroids and selective serotonin-reuptake inhibitors (SSRI) [7].

Obtaining a medical history, especially for peptic ulcer disease, *H pylori* infection, ingestion of nonsteroidal anti-inflammatory drugs (NSAIDs), or smoking, is essential in making the correct diagnosis. Gastric and duodenal ulcers usually cannot be differentiated based on history alone, although some findings may be suggestive. Epigastric pain is the most common symptom of both gastric and duodenal ulcers. It is characterized by a gnawing or burning sensation and occurs after meals—classically, shortly after meals with gastric ulcer and 2-3 hours afterward with duodenal ulcer. Food or antacids relieve the pain of duodenal ulcers but provide minimal relief of gastric ulcer pain [3].

The history and physical examination are important to identify patients at risk of ulcer, perforation, bleeding, or malignancy. However, a systematic review of models using risk factors, history, and symptoms found that they did not reliably distinguish between functional dyspepsia and organic disease. There are alarm symptoms such as unexplained weight loss, progressive dysphagia, odynophagia, recurrent vomiting, family history of gastrointestinal cancer, overt gastrointestinal bleeding, abdominal mass, iron deficiency anemia, or jaundice). Endoscopy is recommended for patients who are 55 years or older, or who have alarm symptoms [8].

Develop serious lesions requiring clinical attention. Removal of the aggravating factors, like smoking, alcohol, and coffee consumption should be sought [9]. Patient that develops an ulcer while on *NSAIDs*, the relevant *NSAID(s)* should be stopped if at all possible and traditional ulcer therapy with a PPI or an H2 antagonist started. PPIs are generally preferred as they are associated with more rapid healing. As in all patients with peptic ulcers, the individual's *H. pylori* status should also be assessed; if positive, appropriate therapy should be instituted. For patients who must remain on *NSAID* therapy or on low-dose aspirin, randomized trials have shown that ulcer healing occurs more rapidly with a PPI rather than with an H2 antagonist misoprostol, or sucralfate [4].

Pre-endoscopic intravenous proton pump inhibitor (PPI) (e.g., 80 mg bolus followed by 8 mg / h infusion) may be considered to decrease the proportion of patients who have higher risk stigmata of hemorrhage at endoscopy and who receive endoscopic therapy. However, PPIs do not improve clinical outcomes such as further bleeding, surgery, or death [10].

Patients with Upper GI tract Bleeding should generally undergo endoscopy within 24 h of admission, following resuscitative efforts to optimize hemodynamic parameters and other medical problems. patients who are hemodynamically stable and without serious comorbidities endoscopy should be performed as soon as possible in a non-emergent setting to identify the substantial proportion of patients with low-risk endoscopic findings who can be safely discharged. In patients with higher risk clinical features (e.g., tachycardia, hypotension, bloody emesis or NG aspirate in hospital) endoscopy within 12 h may be considered to potentially improve clinical outcomes [10].

Stigmata of recent hemorrhage (SRH) should be recorded as they predict risk of further bleeding and guide management decisions. The stigmata, in descending risk of further bleeding, are active spurting, non-bleeding visible vessel, active oozing, adherent clot, flat pigmented spot, and clean base [10].

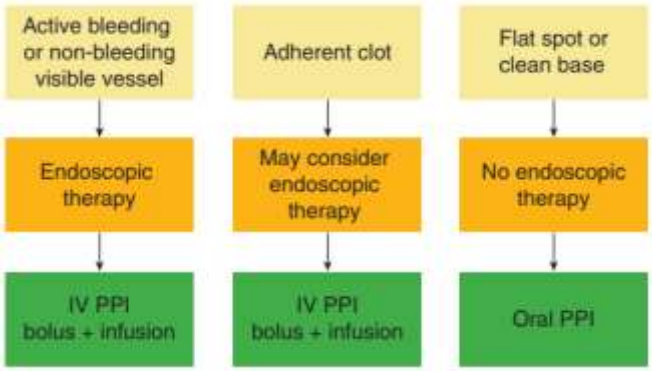

Figure 2: Recommended endoscopic and medical management based on stigmata of hemorrhage in ulcer base.

4. Conclusion

This case report discusses a 61 years old female with giant ulcer in gastric antrum caused by long term use oh NSAIDs. H pylori infection is negative. Patient treat by PPI and show good response. the use of NSAIDs must be carefully considered to see the advantages and disadvantages. in certain cases the use of PPI must be considered.

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