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CASE REPORT

Gastrointestinal perforation: relation to corticosteroid use and COPD – a case report

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Gastrointestinal perforations are a complication of 2–10% of duodenal ulcers. There are a variety of etiologies associated with duodenal ulcer formation and its complications. Corticosteroid use is associated with an increased risk of duodenal ulcer perforation, with the first documented case in 1950. Other important medications associated with perforation include NSAIDS and opioids. Beyond medication, one of the most common disease processes, chronic obstructive pulmonary disease (COPD), has been found to be associated with peptic ulcer disease. Up to 30% of COPD patients have been found to have peptic ulcers, and COPD frequency in peptic ulcer disease is 2–3 times the general population. We herein present a case of an acute duodenal ulcer perforation in a patient receiving corticosteroid treatment for an acute COPD exacerbation.

Keywords: *intestinal perforation*; *steroids*; *COPD exacerbation*; *peptic ulcer*; *acute*

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A 79-year-old man with a medical history significant for COPD and alcohol abuse presented to our facility with a 3-week history of worsening dyspnea, cough, and clear sputum production. A week prior to presentation, he presented to his primary care provider who prescribed a 7-day course of levofloxacin. Although his cough and sputum production had improved, his dyspnea persisted which prompted him to present to our emergency department. The patient had no additional medical history. His social history was notable for alcohol abuse; the patient reported drinking approximately five to six drinks a day. On examination, the patient was afebrile, with a blood pressure of 162/77 mmHg, heart rate of 86 bpm, respiratory rate of 18/min, and oxygen saturation of 93% on a 2-L nasal cannula. Lung examination revealed mild expiratory wheezes bilaterally and decreased air movement in the right lung field. The patient's all other examinations were unremarkable.

Laboratory analysis demonstrated hyponatremia, elevated total bilirubin, and normocytic anemia. The remainder of the patient's complete blood count and metabolic panel were unremarkable. A chest radiograph illustrated right middle lobe atelectasis. A chest CT, without contrast, demonstrated complete atelectasis of the right middle lobe with multiple fluid-filled bronchi in the right lower lobe. Debris was also noted in the trachea.

The patient was treated with methylprednisolone 125 mg IV once then 60 mg IV daily. Due to concern for possible aspiration pneumonia, ceftriaxone and metronidazole treatment was initiated. The patient was not placed on proton pump therapy. The patient's dyspnea and sputum production improved steadily. On day 3 of the patient's hospitalization, the patient developed a sharp epigastric pain that came upon with no inciting event. The patient also developed increased white sputum production and an inability to swallow his secretions. Abdominal examination revealed mild tenderness with no rebound or guarding.

An obstruction series was obtained which demonstrated free air under the diaphragm, although it was unclear whether this was truly pneumoperitoneum or dilated bowel loops. An emergent abdominal CT confirmed pneumoperitoneum, most consistent with perforated viscus. The patient was sent for an emergent exploratory laparotomy, and intraoperative examination revealed an acute duodenal ulcer perforation. The perforation was repaired with open primary repair with omental patch. Pathology results demonstrated smooth muscle and fibrous tissue with focal

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acute inflammation and necrosis. The sample was not tested for *Helicobacter pylori*.

Discussion

The case described illustrates the correlation between COPD and peptic ulcer disease. In a patient receiving corticosteroids for an acute exacerbation of COPD, clinicians need to consider the complications of peptic ulcer disease, paying close attention to subtle changes in abdominal examination (1). There have been reports that corticosteroid use and high cortisol levels could mask the peritoneal signs in a gastrointestinal perforation (2–6). Our patient presented without typical clinical signs of an acute abdomen, with only mild tenderness to palpation with no rebound or guarding that would classically be seen. Theoretically, steroids, with their immunosuppressive effects, could turn down the systemic response to an acute perforation. Further studies are needed to confirm this effect. Numerous case control studies have found an association of perforated diverticular disease with corticosteroid use (7–9). The largest study, involving 889 cases of diverticular perforation and 8,980 population controls from 1990 to 2005, found a threefold increase in diverticular perforation risk with corticosteroid use (9).

Although this patient did not have a formal diagnosis of peptic ulcer disease, his history of COPD and treatment for a COPD exacerbation placed him at risk for perforation. As mentioned previously, COPD frequency in peptic ulcer disease is increased 2–3 times (4). While the duodenal sample was not tested for *H. pylori*, there is a high likelihood that the patient would have tested positive. In patients with endoscopically confirmed duodenal ulcers, *H. pylori* has been found to be seropositive in 73–94% of patients (10, 11). There is an increase in positive *H. pylori* serology in patients with COPD compared to patients without the disease (12). It also appears that the more severe the COPD, the higher the likelihood a patient is positive for *H. pylori* (13).

Beyond being a risk factor, COPD has been associated with the complications of peptic ulcers. COPD has been found to be an independent risk factor for peptic ulcer bleeding regardless of NSAID use or other medical illnesses (14). In patients who have perforation or bleeding from their peptic ulcers, having COPD was found to increase the 30-day mortality dramatically (15).

Peptic ulcer disease places significant economic strain on the healthcare system despite our knowledge of the disease (16). Understanding the contributory and aggravating factors to peptic ulcer disease is central to lowering costs. Additional studies are needed to determine if the severity of COPD correlates with increased risk of perforation.

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