



Stress Ulcer Perforation in the Intensive Care Unit Patient Following Cardiac Surgery

Yoğun Bakım Hastasında Kardiak Cerrahi Takiben Gelişen Stres Ülser Perforasyonu

Stress Ulcer Perforation

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Öz

Gastrointestinal komplikasyonlar yoğun bakım hastalarında yaygın olarak tanımlanmıştır. Bunlardan stres-ilişkili mukozal hastalıkla bağlantılı ülser, kanama ve perforasyon hastanede kalışı ve mortaliteyi arttırabilmektedir. Bu yazıda akut miyokard enfarktüsü sonrası yapılan koroner anjiyografi esnasında oluşan, iatrojenik koroner damar perforasyonu neticesi gelişen perikardiyal tamponat nedeniyle ameliyat edilen yoğun bakım hastasında, stres ülseri perforasyonu ile ilişkili sıradışı bir mortalite olgusunu sunuyoruz.

Anahtar Kelimeler

Ülser; Ülser Perforasyonu; Yoğun Bakım

Abstract

Gastrointestinal complications are common in intensive care unit patients. Some complications, including ulceration, bleeding, and perforation related to stress-related mucosal disease can prolong hospital stays and increase the risk of death. Herein we report on an unusual case of an intensive care unit patient who died following perforation of a stress ulcer. The patient had undergone surgery for pericardial tamponade, which occurred after iatrogenic coronary vessel perforation during coronary angiography for acute myocardial infarction.

Keywords

Ulcer; Ulcer Perforation; Intensive Care

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Introduction

Since Selye's description of the "alarm reaction," an immediate reaction to a stressor, it has been realized that the gastrointestinal (GI) tract is a major target of the stress response that leads to acute gastric and duodenal erosions. Stress ulcer syndrome refers to gastroduodenal erosions or ulcers that occur as acute responses to major physiological stressors. Clinically, these conditions may manifest as upper GI perforation [1].

Case Report

A 62-year-old man with no prior cardiac history presented at an emergency department one night because of chest pain at rest. The electrocardiogram showed an ascending ST segment elevation of 1 mm, with the J point on the isoelectric line, and a Q wave in leads II and III. His creatine kinase level was elevated (470 U/L; normal 0–171 U/L). The patient was diagnosed with acute inferior wall myocardial infarction and was transferred to our institution for primary percutaneous coronary intervention. Coronary angiography performed within one hour of admission to our hospital revealed thrombotic obstruction of the middle right coronary artery, which indicated angioplasty. However, a coronary rupture (depicted by significant leakage of the contrast agent into the pericardium) was observed as the guide-wire was being advanced during the angiographic procedure. The echocardiogram revealed moderate to extensive circumferential pericardial effusion and evidence of cardiac tamponade. The patient's condition began to deteriorate and emergency surgical repair was performed. He was then transferred to a cardiac intensive care unit (ICU), where he was treated for approximately 30 days. Despite prophylactic administration of a proton pump inhibitor to prevent ulcer formation, as well as other therapeutic measures (energetic respiratory and circulatory support), the patient's condition deteriorated and the cardiac surgeon consulted with a general surgeon to discuss the patient's situation. During the initial consultation, he was found to be dehydrated and was experiencing cold sweats. His temperature was 38°C, his blood pressure was 97/54 mmHg, and his pulse rate was 101 beats/min. Abdominal examination revealed no abdominal movement and marked distension. His laboratory findings were as follows: white blood cell count, 15,700/mm³ (neutrophils 91.5%); hematocrit, 31.1%; hemoglobin, 10.3 g/dL; platelet count, 346,000/mm³; alanine transaminase, 2 U/L; aspartate transaminase, 28 U/L; urea, 300 mg/dL; and creatinine, 3.97 mg/dL. A rectal examination revealed an empty rectum. Computed tomography was not performed because of dependence on the mechanical ventilator and severe acute renal impairment. Gastroscopy subsequently performed in the ICU revealed a perforation in the first part of the duodenum (Figure 1) and the patient underwent emergency surgery. Laparotomy revealed a perforated 1 cm ulcer on the anterior aspect of the proximal duodenal bulb and 200 mL of gastric contents mixed with biliary fluid in the peritoneal cavity. After peritoneal washing, the duodenal ulcer was repaired with three silk sutures and an omental patch. The remainder of the peritoneal cavity was explored and found to be normal. Postoperatively, the patient was transferred to our surgical ICU. Despite optimal management in the ICU, the patient's inflammatory markers, creatinine, and alanine transaminase continued to increase, and his urinary

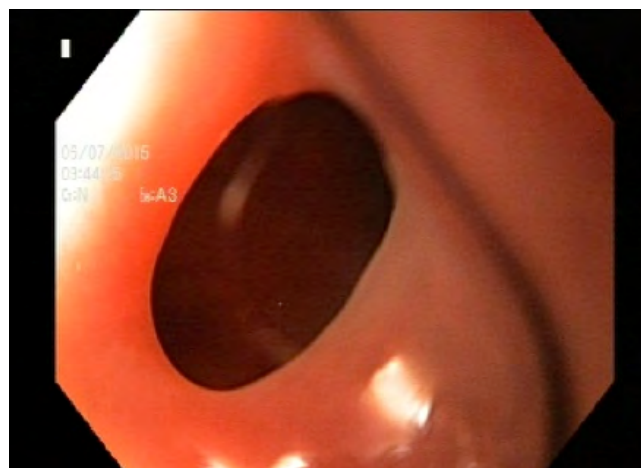


Figure 1. Endoscopic image of the perforation located in the proximal duodenal bulb.

output remained low. The patient had a difficult postoperative course that was complicated by wound infection and septicemia. The patient did not recover from septic shock. He was kept on a ventilator and inotropic drugs with antibiotics. The patient died from cardiopulmonary failure and shock four days following the second operation.

Discussion

Stress-related mucosal damage (SRMD) of the GI tract was first described in 1971 by Lucas et al., who used the term stress-related erosive syndrome [2]. Since then, many terms have been used to describe SRMD in critically ill patients, including stress ulcer, stress erosion, stress gastritis, hemorrhagic gastritis, erosive gastritis, and stress-related mucosal disease. The incidence of SRMD has been reported to range from 6% to 100% in critically ill patients [3]. Stress ulcers develop within hours of major trauma or serious illness, most often in the proximal regions of the stomach. Endoscopy performed within 72 h of a major burn or cranial trauma revealed acute mucosal abnormalities in >75% of patients [4]. Stress ulcers are common complications in many clinical conditions, but they rarely perforate. They generally occur in the fundus or body of the stomach, but sometimes occur in the antrum, duodenum, or distal esophagus. Stress ulcers tend to be shallow and cause oozing of blood from superficial capillary beds. Deeper lesions may also occur, which can erode into the submucosa and cause massive hemorrhage or perforation [5]. Although not completely understood, the pathophysiology of stress ulcers is likely to be multifactorial. Inadequate systemic perfusion, mucosal blood flow, and cellular oxygenation appear to play important roles in the development of stress ulcers, as do decreased gastric pH, increased mucosal permeability, and alterations in normal protective mechanisms [6,7]. Stress ulcers are an important clinical disorder. Two major complications, upper GI perforation and bleeding, are significant concerns in critically ill patients and are associated with a high risk of death. GI complications are particularly common in critically ill patients and also carry a high risk of death. Many authors have reported the prevalence of GI complications and predictors of GI complications in specific patient populations. These data allow practitioners to evaluate not only the risk of complication but also the risk of death in individual patients. Chan et al. investigated the GI complications in neurosurgical

patients [8]. Their retrospective report of 526 patients revealed that 36 (6.8%) had endoscopically or surgically confirmed GI complications. All of these patients had GI bleeding, and two had evidence of ulcer perforation. Eleven patients died as a direct result of the GI complication. In a prospective study of 11,508 cardiac surgery patients, D'Ancona et al. identified 147 GI complications in 129 patients (1.2%) [9]. The complications included gastroesophagitis (12.2%), upper GI hemorrhage (28.6%), perforated peptic ulcer (4.7%), cholecystitis (6.8%), pancreatitis (8.8%), intestinal ischemia (11.5%), colitis (12.2%), diverticulitis (3.4%), intestinal occlusion (1.1%), and lower GI hemorrhage (0.7%). In a study by Tsiotos et al., perforation occurred after stress ulceration in <1% of patients in a cardiac surgery ICU [10]. The potential causes of stress ulcers are numerous and are related to patient characteristics such as age and underlying disease processes, as well as to interventions such as mechanical ventilation. The primary cause of stress in our patient was likely to be severe illness. In addition, the patient had had endotracheal intubation for an extended period of time. Most critically ill patients are unable to provide a history or description of their symptoms, which limits and/or delays the timely diagnosis of GI disorders. Therefore, it is essential that critical care specialists who suspect GI complications initiate preventive strategies and carefully monitor the patients for evidence of such complications. The optimal agent for acid suppression is unknown, and there is controversy and substantial variation in clinical practices regarding the choice of agent for acid suppression in critically ill patients. Based on the results of randomized trials and the recommendations of the American Society of Health System Pharmacists, all critically ill patients who are at high risk of GI complications such as bleeding and perforation should receive anti-ulcer prophylaxis [11]. Because of his poor clinical condition, our patient did not display the classic signs of peritonitis such as generalized tenderness and rigidity. Therefore, the diagnosis of bowel perforation was not initially apparent and could only be made after exploratory laparotomy in the ICU. Surgical exploration also revealed perforation of the first part of the duodenum, which was intractable despite intensive anti-ulcer therapy. There has been a significant decrease in the rates of elective surgery for treating uncomplicated peptic ulcers; however, complications such as perforation and obstruction persist and require urgent surgical management. The first report of a series of patients presenting with perforation of a duodenal ulcer was made in 1817 by Travers. Although the earliest description of surgical treatment was made by Mikulicz in 1884, the first successful operation for a perforated duodenal ulcer occurred in 1894 [12]. Mortality related to a perforated duodenal ulcer is dependent on the presence or absence of several risk factors. Individual risk can also be assessed using the Acute Physiology and Chronic Health Evaluation II system. Although prompt closure of the perforation offers the best chance for survival, it also creates additional stress exacerbating the patient's clinical state. Therefore, surgical repair must be done as quickly as possible. The overall mortality rate is approximately 10% in most studies. In the developing world the high morbidity and mortality rates of patients with a perforated duodenal ulcer are probably due to delayed presentation. In particular, patients in whom the

diagnosis is overlooked almost always die. Risk factors affecting the prognosis of perforated ulcer included delayed treatment (> 24 h), preoperative shock (systolic blood pressure < 100 mmHg), and serious comorbidities. The mortality rate increases to 100% in patients with all three factors. This is consistent with our experience in the present case. Potential adverse effects of pharmacologic agents that suppress gastric acid secretion are particularly relevant to critically ill patients, and include nosocomial pneumonia and clostridium difficile infection. Other adverse effects, such as intolerance, drug interactions, and thrombocytopenia are rare. Despite these risks, prophylactic anti-ulcer therapy is essential to reduce the risk of stress ulcer-related complications. Perforated stress ulcers are potentially fatal complications in ICU patients. Such ulcers may be missed if practitioners are not alert to their likelihood in critically ill patients [13-15].

In conclusion, based on our limited experience with this patient, we wish to make the following recommendations: First, all ICU patients should receive prophylactic anti-ulcer therapy and possibly cimetidine; second, ICU specialists should be vigilant for complications of SRMD; and third, surgical closure of a perforation must be simple and performed quickly.

Competing interests

The authors declare that they have no competing interests.

References

1. Selye H. The general adaptation syndrome and the diseases of adaptation. *J Clin Endocrinol Metab* 1946;6:117-230.
2. Lucas CE, Sugawa C, Riddle J, Rector F, Rosenberg B, Walt AJ. Natural history and surgical dilemma of "stress" gastric bleeding. *Arch Surg* 1971;102(4):266-73.
3. Choung RS, Talley NJ. Epidemiology and clinical presentation of stress-related peptic damage and chronic peptic ulcer. *Curr Mol Med* 2008;8(4):253-7.
4. DePriest JL. Stress ulcer prophylaxis. Do critically ill patients need it? *Postgrad Med* 1995;98(4):159-8.
5. Cook DJ. Stress ulcer prophylaxis: gastrointestinal bleeding and nosocomial pneumonia. Best evidence synthesis. *Scand J Gastroenterol Suppl* 1995;210:48-52.
6. American Society of Health-System Pharmacists. ASHP Therapeutic Guidelines on Stress Ulcer Prophylaxis. ASHP Commission on Therapeutics and approved by the ASHP Board of Directors on November 14, 1998. *Am J HealthSyst Pharm* 1999;56(4):347-79.
7. Cook D, Heyland D, Griffith L, Cook R, Marshall J, Pagliarello J. Risk factors for clinically important upper gastrointestinal bleeding in patients requiring mechanical ventilation. *Crit Care Med* 1999;27(12):2821-7.
8. Chan KH, Mann KS, Lai EC, Ngan J, Tuen H, Yue CP. Factors influencing the development of gastrointestinal complications after neurosurgery: results of multivariate analysis. *Neurosurgery* 1989;25(3):378-82.
9. D'Ancona G, Baillot R, Poirier B, Dagenais F, de Ibarra JL, Bauset R, et al. Determinants of gastrointestinal complications in cardiac surgery. *Texas Heart J* 2003;30(4):280-5.
10. Tsiotos GG, Mullany CJ, Zietlow S, van Heerden JA. Abdominal complications following cardiac surgery. *Am J Surg* 1994;167(6):553-7.
11. Spirt MJ, Stanley S. Update on stress ulcer prophylaxis in critically ill patients. *Crit Care Nurse* 2006;26(1):18-9.
12. Andrew G Hill. Management of perforated duodenal ulcer. In: Holzheimer RG, Mannick JA, editors. *Surgical Treatment: Evidence-Based and Problem-Oriented*. Zuckschwerdt Publishers 2001.
13. Marik PE, Vasu T, Hirani A, Pachinburavan M. Stress ulcer prophylaxis in the new millennium: a systematic review and meta-analysis. *Crit Care Med* 2010;38(11):2222.
14. Houssa MA, Atmani N, Nya F, Abdou A, Moutakiallah Y, Bamou M, et al. Stress gastric ulcer after cardiac surgery: Pathogenesis risk factors and medical management. *World Journal of Cardiovascular Diseases* 2013;3(3):312-6.
15. Bateman BT, Bykov K, Choudhry NK, Schneeweiss S, Gagne JJ, Polinski JM, et al. Type of stress ulcer prophylaxis and risk of nosocomial pneumonia in cardiac surgical patients: cohort study. *BMJ* 2013;347:f5416.

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