

Perforated Gastric Carcinoma Presenting as Septic Shock: A Case Report

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Abstract

Gastric malignancy perforation is rare and may present with nonspecific or extra-abdominal symptoms, delaying diagnosis. We present the case of a 64-year-old man who presented with dyspnea and undifferentiated shock with minimal abdominal signs but massive free abdominal fluid and air. Emergency laparotomy revealed diffuse purulent peritonitis and a 5 cm antral perforation due to a perforated gastric adenocarcinoma. In conclusion, perforated gastric cancer can present primarily with shock and few abdominal signs. Rapid ultrasound/CT diagnosis and a staged damage-control surgical strategy with secondary definitive resection improve chances of survival.

Keywords: Gastric Malignancy Perforation; Gastric Cancer; CT Diagnosis

Introduction

Perforation of a gastric malignancy is an uncommon but life-threatening complication that may present with a heterogeneous and often misleading clinical picture. Secondary peritonitis resulting from gastrointestinal perforation classically manifests with circulatory instability and systemic inflammatory signs, while the more familiar localized peritoneal signs may emerge only later in the clinical course. This diagnostic ambiguity is compounded when the initial presenting complaint is extra-abdominal, such as dyspnea, prompting consideration of cardiopulmonary etiologies before an abdominal source is recognized. Early and accurate diagnosis is critical, as delays in source control are associated with increased morbidity and mortality in perforative peritonitis. We report a case of a perforated gastric ulcer, later confirmed histologically as adenocarcinoma, that initially presented as undifferentiated shock with minimal abdominal symptomatology, illustrating both the diagnostic challenge and the staged surgical management strategy employed in critically ill patients.

Case Report

A 64-year-old man with his family presented to the emergency department, reporting dyspnea that had been present for three hours following a prolonged car journey. During triage, he started to complain of dizziness and required immediate recumbent positioning. History-taking was complicated by a language barrier.

The patient reported a smoking history of 40 cigarettes per day; no prior illnesses or regular medications were disclosed.

On examination, the patient was in a markedly reduced general condition, with tachypnea of 30 breaths/min and intercostal retractions. He was profoundly hypotensive with blood pressure unmeasurable and exhibited cyanotic extremities; heart rate was 60 beats/min. The abdomen was soft and compressible, with tenderness on deep palpation but without abdominal guarding.

Initial arterial blood gas analysis showed an oxygen saturation of 96% with no evidence of metabolic or respiratory derangement (pH 7.44; lactate 1.2 mmol/L [normal range 1.0-1.8]; bicarbonate 23.6 mmol/L [normal range 22-26]. Complete blood count was unremarkable, without leukocytosis. Laboratory findings notable for coagulopathy and organ dysfunction included a Quick value (prothrombin activity) of 58%, partial thromboplastin time of 50 seconds, sodium 129 mmol/L, urea 98 mg/dL (normal range 16-48), and creatinine 2.5 mg/dL (normal range 0.7-1.2). C-reactive protein was markedly elevated at 200 mg/L (normal <5).

While further diagnostic work-up was being arranged, the patient received fluid resuscitation with 2000 mL of balanced crystalloid solution supported by the inotrope/vasopressor combination cafedrine/theodrenaline (Akrinor), achieving hemodynamic stabilization. As abdominal complaints became more pronounced, point-of-care ultrasonography was performed. Cardiac assessment revealed no signs of acute right heart strain, pneumothorax, or pleural effusion. However, massive free intraperitoneal fluid was identified.

Further evaluation with contrast-enhanced computed tomography (CT) of the thorax and abdomen revealed no acute pulmonary pathology. In the abdomen, however, CT demonstrated massive free fluid together with free intraperitoneal air and gas bubbles surrounding the stomach, consistent with gastric perforation.

In summary, the clinical picture was consistent with septic shock and incipient acute kidney failure secondary to a CT-confirmed suspected gastric perforation. Emergency laparotomy was performed. On entry into the abdomen, diffuse purulent peritonitis with putrid ascites was evident in all four quadrants. Exploration revealed a 5 cm perforated ulcer in the gastric antrum, with extension through the mesocolon. A biopsy was obtained from the ulcer margin. After extensive peritoneal lavage, the perforation was closed by primary suture (Graham patch repair) and reinforced with an omental patch, and drains were placed.

The patient remained in the intensive care unit over the following days. Under broad-spectrum antibiotic therapy and continued fluid resuscitation, he progressively stabilized.

Histopathological examination of the biopsy specimen revealed an adenocarcinoma. Once inflammatory markers had normalized and hemodynamic status was stable, a staged Billroth II gastric resection with Roux-en-Y reconstruction was performed.

Discussion

The case illustrates a perforated gastric ulcer with secondary peritonitis and septic shock. Its presentation is consistent with previously described cases of perforative peritonitis [1]. The initial presentation with dyspnea, hypotension, and reduced general condition, alongside only discreet abdominal complaints, highlights the diagnostic challenge posed by perforation, as early symptoms are frequently nonspecific [2-4]. Smoking is among the most common predisposing factors for perforation, implicated in up to 88% of cases [5].

Secondary peritonitis may initially present predominantly with circulatory instability and systemic signs, while classic peritonism develops only later in the course [3]. In large cohorts of perforative peritonitis, diagnosis is established predominantly on clinical grounds and subsequently confirmed by imaging, with nonspecific symptoms and comorbidities contributing to diagnostic delay [1]. The scenario described here, with dyspnea occurring after a car journey, demonstrates that cardiopulmonary causes are typically considered first in the differential diagnosis, which can further obscure an underlying abdominal source [6].

When hollow viscus perforation is suspected, contrast-enhanced CT of the abdomen is regarded as the gold standard, given its high sensitivity for detecting pneumoperitoneum, free intraabdominal fluid, and extraluminal gas bubbles [6]. In cohort studies of perforative peritonitis, CT and ultrasonography are described as complementary modalities that together substantially improve diagnostic yield; ultrasonography in particular allows early detection of free fluid [1]. The sonographic detection of massive free fluid in this case, subsequently confirmed by CT demonstrating free air and gas bubbles around the stomach, is consistent with these recommendations [6].

Surgical management of gastric perforation depends on the size of the defect, local tissue condition, comorbidities, and hemodynamic stability [6]. For perforated ulcers, emergency laparotomy with primary closure and omental patch ("Graham patch") is frequently recommended as initial management, particularly in the setting of septic shock and severe peritonitis, in keeping with damage control principles [5]. Cohort studies of perforative peritonitis have shown that early surgical source control together with thorough lavage and drainage is decisive for outcome, with delays significantly increasing mortality and complication rates [1].

In severely septic patients, a staged approach is increasingly described, in which an initial, limited, life-saving procedure is performed for sepsis control with definitive reconstruction deferred to a period of hemodynamic stability [3]. The management described in this case with an initial primary closure with omental patch followed by delayed Billroth II resection with Roux-en-Y anastomosis after stabilization is consistent with this concept of staged management [5]. Studies of perforative peritonitis emphasize that this stepwise approach may particularly improve perioperative outcomes in high-risk surgical patients [1].

Secondary peritonitis with generalized purulent peritoneal contamination requires, in addition to surgical source control, aggressive critical care management including volume resuscitation, vasopressor support, adequate broad-spectrum antibiotic therapy, and close monitoring [3]. Larger series of perforative peritonitis report substantial rates of organ failure, particularly renal insufficiency and respiratory complications, which significantly determine prognosis [1]. The clinical course described here—initial renal impairment followed by stabilization under fluid resuscitation and antibiotic therapy—is consistent with outcomes reported in the literature when early and adequate source control is achieved [5].

Conclusion

This case underscores the importance of maintaining a high index of suspicion for hollow viscus perforation in patients presenting with unexplained shock, even when abdominal symptoms are minimal or overshadowed by extra-abdominal complaints such as dyspnea. Prompt use of focused ultrasonography and CT imaging is essential to avoid diagnostic delay in such atypical presentations. Furthermore, this case highlights the value of a staged surgical strategy in critically ill, septic patients: initial damage-control surgery to achieve source control, followed by definitive oncologic resection once hemodynamic and inflammatory parameters have normalized. Close interdisciplinary collaboration between emergency medicine, radiology, surgery, and critical care is essential to reduce the substantial morbidity and mortality associated with perforative peritonitis, particularly when an underlying malignancy, as in this case, is ultimately identified.

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