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Pneumoperitoneum Secondary to Pyloric Ulcer Perforation Following Diagnostic Esophagogastroduodenoscopy (OGDS)- From Scope to Operative Table

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Abstract

Background: Esophagogastroduodenoscopy (OGDS) is the primary diagnostic modality for upper gastrointestinal bleeding [1]. While generally safe [2], iatrogenic perforation risk increases in patients with compromised mucosa, where routine baropneumatic insufflation can easily rupture a fragile ulcer bed, demanding emergent exploratory laparotomy [3-5].

Case Presentation: A 71-year-old female presented with epigastric pain, vomiting, and melena. She reported long-term NSAID and corticosteroid use without proton pump inhibitor gastroprotection [6]. Following resuscitation for severe blood loss anemia, a diagnostic OGDS revealed a prominent duodenal (D1) diverticulum but no active bleeding or obvious ulceration [7]. Two hours post-procedure, she developed acute dyspnea, progressive abdominal distension, and rapid hemodynamic collapse requiring emergency intubation and vasopressor support [8]. An emergent abdominal computed tomography (CT) scan demonstrated massive tension pneumoperitoneum with a suspected pyloroduodenal perforation [9]. Emergency exploratory laparotomy confirmed a 1.5 cm anterior prepyloric perforation, which was successfully managed with omental patch repair [10].

Conclusion: Diagnostic OGDS carries a rare but lethal risk of baropneumatic perforation in patients with mucosal injury from chronic NSAID and corticosteroid use [4]. Furthermore, anatomical variants like duodenal diverticula can mask coexisting ulcers during endoscopy [7]. Clinicians must maintain high suspicion for tension pneumoperitoneum if acute cardiopulmonary distress develops post-endoscopy, as early recognition and prompt surgical intervention are vital for survival [5].

Keywords: Esophagogastroduodenoscopy (OGDS), Gastric Perforation, Duodenal Diverticulum, Upper Gastrointestinal Bleeding, Peptic Ulcer Disease, Tension Pneumoperitoneum

Introduction

Esophagogastroduodenoscopy (OGDS)—also referred to as upper gastrointestinal endoscopy—stands as the definitive diagnostic and therapeutic modality for evaluating suspected upper gastrointestinal bleeding (UGIB), dyspepsia, and mucosal pathology [1]. Widely recognized for its high diagnostic accuracy and minimally invasive nature, routine diagnostic OGDS is generally regarded as an exceptionally safe procedure, boasting an overall complication rate of less than 0.01% in standard clinical practice [2]. Among possible adverse events, iatrogenic perforation represents one of the most feared and potentially lethal complications [3]. While esophageal and duodenal perforations are more frequently reported, perforation of the gastric wall or pyloroduodenal junction during a purely diagnostic examination is an exceedingly rare phenomenon [1, 2].

When iatrogenic perforation does occur during diagnostic OGDS, it is rarely the result of direct mechanical perforation by the endoscope tip alone; rather, it typically arises from a complex interaction between baropneumatic insufflation and underlying mucosal vulnerability [4]. In a healthy gastrointestinal tract, the muscularis propria and serosa can easily accommodate routine air or carbon dioxide insufflation [4]. However, in the presence of severe peptic ulcer disease (PUD) or chronic mucosal inflammation, the structural integrity of the gastrointestinal wall is profoundly compromised [11]. Chronic co-prescription of non-steroidal anti-inflammatory drugs (NSAIDs) and systemic corticosteroids—particularly in the absence of proton pump inhibitor (PPI) gastroprotection—is a well-established driver of synergistic mucosal injury, microvascular ischemia, and subsequent ulceration [6]. Within these friable ulcer beds, wall thickness is significantly attenuated, dramatically lowering its

tensile threshold [4]. Consequently, the elevation of intraluminal pressure during routine endoscopic air insufflation can easily exceed the mechanical limits of the weakened pyloroduodenal wall, precipitating acute baropneumatic rupture and rapid-onset pneumoperitoneum [4].

The diagnostic detection of such perforations or underlying ulcerations during the index endoscopy can be further complicated by anatomical distortion or concurrent pathology [7]. For instance, the presence of benign luminal outpouchings, such as duodenal diverticula, can alter normal luminal architecture, trap insufflated air, and create visual blind spots that obscure a coexisting ulcer or micro-perforation at the pyloroduodenal junction [7]. When a perforation goes unrecognized during the procedure, the continued leakage of luminal air and gastric contents into the peritoneal cavity rapidly leads to tension pneumoperitoneum, systemic inflammatory response syndrome (SIRS), and severe hemodynamic collapse [8].

Herein, we report the instructive case of a 71-year-old female with chronic NSAID- and corticosteroid-induced peptic ulcer disease who developed massive tension pneumoperitoneum secondary to a pyloroduodenal ulcer perforation following a routine diagnostic OGDS. This case underscores the critical physiological hazards of baropneumatic pressure in structurally compromised mucosa [3], highlights how anatomical variations like duodenal diverticula can mask pathology during endoscopy [7], and emphasizes the paramount importance of early clinical recognition and immediate surgical intervention in averting fatal outcomes [5, 7].

Case Presentation

A 71-year-old Malay female with a significant past medical history of bronchial asthma and chronic sciatica presented to the emergency department reporting a two-day history of worsening epigastric pain, progressive lethargy, and repeated episodes of non-bilious vomiting. To manage her debilitating sciatic pain, she had been self-medicating with a prolonged course of systemic corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDs) without concurrent proton pump inhibitor (PPI) gastroprotection [6].

Upon initial clinical assessment, she was hemodynamically unstable, requiring an intravenous infusion of noradrenaline to maintain mean arterial pressure [8]. Physical examination revealed marked conjunctival and cutaneous pallor; however, peripheral pulses were well-perfused with warm extremities. Abdominal palpation revealed a soft, non-tender abdomen without peritoneal signs or guarding. Digital rectal examination (DRE) demonstrated stale melenic stool, confirming upper gastrointestinal bleeding [1].

Laboratory investigations confirmed severe acute-on-chronic blood loss anemia, with a baseline hemoglobin level of 6.3 g/dL. Remarkably, initial organ function panels were otherwise preserved: serum creatinine and urea were within normal limits, liver transaminases were unelevated, and her coagulation profile was normal. Immediate fluid resuscitation and transfusion of packed red blood cells were initiated. Following adequate volume resuscitation, the patient stabilized hemodynamically, allowing for the successful weaning and cessation of the noradrenaline infusion.

With stable vital signs, she was transferred to the endoscopy suite for a diagnostic esophagogastroduodenoscopy (OGDS) [2]. Endoscopic evaluation of the esophagus, stomach, and duodenum revealed no active bleeding, stigmata of recent hemorrhage, or obvious peptic ulceration [2]. However, a prominent benign diverticulum was identified in the first part of the duodenum (D1) [7].

Two hours post-procedure while recovering in the general ward, the patient complained of rapidly worsening abdominal distension and acute-onset dyspnea [8]. Given her underlying history of bronchial asthma, initial bedside management was directed toward treating a suspected acute bronchospasm; however, her respiratory distress escalated simultaneously with progressive, tense abdominal distension [8]. Her clinical condition deteriorated rapidly into hemodynamic collapse, necessitating emergency endotracheal intubation, mechanical ventilation, and the re-initiation of intravenous noradrenaline support [8].

Once stabilized, an urgent contrast-enhanced computed tomography (CT) scan of the abdomen and pelvis was performed [9]. Imaging revealed massive, diffuse pneumoperitoneum with a distinct focal air locule adjacent to the pyloroduodenal junction, highly suspicious for an iatrogenic perforation (Fig 1) [9]. Significant reactive free fluid was noted in the perihepatic and perisplenic spaces, the right paracolic gutter, and the pelvis [9].

The patient was immediately transferred to the operating theater for an emergency exploratory laparotomy [10]. Intraoperative exploration confirmed a 1.5 cm prepyloric perforation on the anterior wall with localized biliary and gastric contamination (Fig 2) [10]. A primary surgical repair of the perforation with an omental patch (Graham patch repair) and thorough peritoneal lavage were successfully performed [10]. The patient was transferred to the Intensive Care Unit (ICU) post-operatively for invasive hemodynamic monitoring and ventilator weaning, where she made a gradual but steady clinical recovery [10].

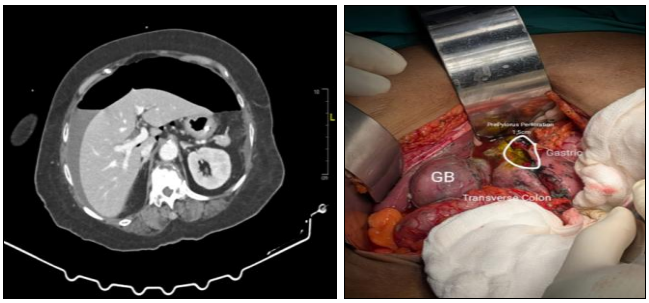

Fig 1 and 2: CT images showing massive pneumoperitoneum and intraoperative finding of pre pyloric perforation

Discussion

Iatrogenic perforation following diagnostic esophagogastroduodenoscopy (OGDS) is an exceptionally rare clinical event, occurring in roughly 0.01% of standard diagnostic procedures [2, 12]. Unlike therapeutic endoscopies—where mucosal tears or transmural defects typically result from direct mechanical shearing during dilation, mucosectomy, or hemostatic clip placement—perforations during purely diagnostic examinations are predominantly driven by physiological baropneumatic insufflation acting upon a structurally vulnerable gastrointestinal wall [3, 4]. When diagnostic perforation does occur, the prepyloric and duodenal regions represent common anatomical sites of rupture due to their luminal tapering and acute angular anatomy [13]. This case illustrates

a critical clinical convergence of chronic pharmacological mucosal injury, anatomical concealment of pathology during endoscopy, and the life-threatening physiological consequences of post-procedural tension pneumoperitoneum [6, 7, 8].

The biomechanical mechanism of diagnostic perforation in this patient is best governed by Laplace's Law, which states that wall tension is directly proportional to intraluminal pressure and radius, and inversely proportional to wall thickness ($T=2wP \cdot r$, where T represents wall tension, P is intraluminal pressure, r is luminal radius, and w is wall thickness) [4]. In a healthy stomach or duodenum, a robust muscularis propria and serosa easily accommodate routine air or carbon dioxide insufflation [11]. However, chronic peptic ulcer disease severely attenuates the mucosal and submucosal layers, significantly reducing local wall thickness (w) at the prepyloric junction [11, 14]. During diagnostic evaluation, routine endoscopic air insufflation exponentially elevates intraluminal pressure (P) [4]. Within the thinned, friable ulcer bed, the mechanical tension rapidly exceeds the tensile threshold of the diseased tissue, precipitating acute baropneumatic rupture rather than direct mechanical puncture by the gastroscope tip [3, 4].

This profound mucosal vulnerability was directly tied to the patient's unbuffered, chronic co-prescription of non-steroidal anti-inflammatory drugs (NSAIDs) and systemic corticosteroids [6]. Long-term NSAID therapy inhibits cyclooxygenase-1 (COX-1) and COX-2 enzymes, leading to systemic depletion of gastroprotective prostaglandins, reduced mucosal blood flow, decreased epithelial mucus and bicarbonate secretion, and impaired epithelial cellular proliferation [6, 15]. Concurrent systemic corticosteroid therapy synergistically exacerbates this injury by suppressing tissue repair, inhibiting angiogenesis, and delaying ulcer healing [16]. Crucially, this pharmacological combination created a clinical diagnostic trap upon presentation [16]. Despite presenting with severe blood loss anemia (6.3 g/dL) and melena secondary to an active prepyloric ulcer, the patient's abdomen was clinically soft and non-tender on initial physical examination. Systemic corticosteroids are notorious for blunting systemic inflammatory responses and masking the classic peritoneal signs of ulcer erosion, micro-perforation, or localized peritonitis, thereby conveying a deceptive baseline of tissue integrity prior to endoscopy [16].

A pivotal learning point in this case is the negative endoscopic finding for peptic ulceration during the index procedure, despite the subsequent intraoperative discovery of a 1.5 cm prepyloric perforation [7, 10]. The presence of a prominent benign diverticulum in the first part of the duodenum (D1) played a major role in obscuring the visual field [7]. Duodenal diverticula can significantly distort normal luminal architecture, induce eccentric mucosal folding, and cause localized mucosal prolapse or luminal angulation [7, 17]. During endoscopy, insufflated air can become trapped within the diverticular pouch or surrounding redundant folds, creating visual blind spots that easily conceal adjacent prepyloric or pyloroduodenal ulcers [7, 18]. Furthermore, if the ulcer bed had already suffered a sealed micro-perforation covered by omentum prior to the procedure, standard diagnostic air insufflation would inevitably disrupt this fragile fibrinous seal without presenting obvious active luminal hemorrhage during scope withdrawal [2, 12].

The patient's rapid post-procedural deterioration highlights a dangerous clinical pitfall: misattributing the systemic consequences of tension pneumoperitoneum to primary pulmonary pathology [8]. Two hours post-OGDS, the patient developed acute dyspnea that was initially managed as an acute asthma exacerbation [8]. In reality, the continuous escape of luminal air through the 1.5 cm prepyloric defect into the closed peritoneal cavity resulted in tension pneumoperitoneum—a life-threatening surgical emergency [8]. Tension pneumoperitoneum exerts profound cardiopulmonary pathophysiological effects: massive abdominal distension elevates the diaphragms bilaterally, severely compressing baseline lung volumes, reducing functional residual capacity (FRC), and inducing basal atelectasis and acute ventilation-perfusion (V/Q) mismatch that mimics acute bronchospasm [8, 19]. Simultaneously, the rapid rise in intra-abdominal pressure directly compresses the inferior vena cava and mesenteric veins, drastically reducing venous return (preload) to the right heart [19]. This acute drop in preload causes severe distributive and hypovolemic shock, explaining the patient's rapid hemodynamic collapse that required emergency endotracheal intubation, vasopressor resuscitation, and emergent exploratory laparotomy [8, 10, 19]. Ultimately, this case reinforces that acute-onset dyspnea accompanied by progressive abdominal distension following any endoscopic procedure must be treated as iatrogenic perforation and tension pneumoperitoneum until proven otherwise [8]. When diagnostic OGDS is performed on elderly patients with chronic NSAID and corticosteroid exposure, endoscopists must exercise extreme caution with air insufflation, remain vigilant for anatomical masking by diverticula, and maintain a low threshold for early post-procedural cross-sectional imaging when clinical deterioration occurs [3, 6, 7].

Conclusion

While diagnostic esophagogastroduodenoscopy (OGDS) remains an overwhelmingly safe modality, this case underscores the life-threatening potential of baropneumatic perforation in patients with severely compromised gastroduodenal mucosa. Chronic co-administration of NSAIDs and systemic corticosteroids not only diminishes the tensile strength of the gastric wall—predisposing it to rupture under routine air insufflation—but also masks the classic peritoneal signs of impending perforation. Furthermore, endoscopists must exercise heightened vigilance when anatomical variants, such as duodenal diverticula, are present, as these can easily distort luminal architecture and conceal coexisting ulcerations. Crucially, clinicians must maintain a high index of suspicion for tension pneumoperitoneum in any patient developing acute cardiopulmonary distress post-endoscopy. As demonstrated in this case, the profound decreased venous return caused by massive free intra-abdominal air can easily masquerade as a primary respiratory event, such as acute bronchospasm. Prompt recognition, early cross-sectional imaging, and emergent surgical intervention remain imperative to averting fatal outcomes in these complex scenarios.

Administrative Declarations

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the signed consent form is available for review upon request.

Ethical Approval

Ethical approval was not required for this case report, as per the institutional guidelines of our hospital, provided that written informed consent was obtained from the patient.

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Competing Interests

The authors have declared that no competing interests exist. No funding was involved in this study.

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