CASE REPORT

Gastric Outlet Obstruction Induced by a Gastric Wall Abscess after Cholecystitis

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Abstract

In December 2011, an 84-year-old Japanese man was admitted with severe cholecystitis. We treated the cholecystitis using antibiotics, and his symptoms improved. In March 2012, he was readmitted after experiencing a sudden bloating sensation and vomiting. We determined that a gastric wall abscess had protruded into the antrum. The gastric wall was adjacent to the gallbladder, and the gastric abscess was related to a known case of cholecystitis. Upper gastrointestinal endoscopy indicated that the abscess was under the mucous membrane of the gastric antrum. Pus, exuding from the abscess, was aspirated with an endoscope.

Key words: cholecystitis, gastric outlet obstruction, gastric wall abscess


Introduction

Gastric wall abscess (GWA) formation is a rare condition that is primarily caused by bacterial infection of the gastric wall (1, 2) and often has a poor prognosis. Recently, the early diagnosis of GWA and aggressive treatment with antibiotic therapy has become more common, improving patient outcomes without surgery (3). We herein report a rare case of acute GWA, with marked stomach distension due to gastric antrum stricture.

Case Report

An 84-year-old Japanese man was admitted in December 2011 with severe cholecystitis and a pericholecystic abscess. Abdominal computed tomography (CT) showed that the cholecystitis had spread to the gastric antrum (Fig. 1). He refused surgical cholecystectomy and was treated with antibiotics. The pus cultured from the gallbladder revealed Enterococcus faecium infection. Intravenous antibiotics were administered, including doripenem (0.75 g/day) for four days, followed by pazufloxacin (1,000 mg/day) for an additional 14 days. After the antibiotic treatment, the patient’s symptoms improved, and he was discharged in January 2012. In March 2012, he was readmitted after experiencing sudden bloating, vomiting and a high-grade fever. His body temperature was 38.5°C; however, he was hemodynamically stable. Initial laboratory tests were conducted to determine the white blood cell count (12,400/μL), hemoglobin level (13.9 g/dL), platelet count (24.6×10⁴/μL), C-reactive protein level (8.48 mg/dL) and total bilirubin level (0.4 mg/dL).

Based on the findings of ultrasonography (Fig. 2a), abdominal CT (Fig. 2b) and magnetic resonance imaging (Fig. 2c), we determined that the GWA had protruded into the antrum. The gastric wall was adjacent to the gallbladder, and the GWA was related to the patient’s known cholecystitis. Upper gastrointestinal endoscopy was performed on the day after admission, the results of which indicated that the abscess was under the mucous membrane of the gastric antrum and had disintegrated naturally (Fig. 3a). Pus from the abscess was aspirated endoscopically (Fig. 3b). After this treatment, the patient’s symptoms disappeared, and his GWA improved with antibiotic therapy. He has not experienced any episodes of recurrence in the three years since discharge (Fig. 4).

Discussion

GWA formation, first described by Cruveilhier (4), is a

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rare pathological condition of the stomach. A GWA is defined as a site of inflammation—primarily of the submucosa—with thickening of the gastric wall submucosa due to infiltration by neutrophils and plasma cells. Konjetzny described three causes of GWA: primary, secondary and idiopathic (5). The pathogenesis of GWA most likely involves bacterial infection, either through direct invasion of the gastric mucosa (primary) or hematogenous/lymphogenous spread from another site of infection (secondary) (6). Idiopathic GWAs are defined as abscesses with an uncertain cause. Based on the extent of the disease, GWAs may appear as either diffuse, suppurative gastritis (diffuse GWA) (7, 8) or a focal, intramural abscess (localized GWA) (6, 9), accounting for 5-15% of cases (10).

Until recently, GWAs were rarely diagnosed before surgery because they are seldom considered in the differential diagnosis of acute abdomen (11). Patients usually present with acute abdomen and septicemia; other symptoms include nausea, vomiting, hematemesis, hiccups, prostration and fever (2). Two specific, but seldom present clinical signs, are Deininger’s sign (decreased pain when changing from the supine to the sitting position) (12) and vomiting of frank pus (13). In our patient, the GWA formed two months after the cholecystitis improved, suggesting that the potential for GWA formation should be considered after treatment in serious cases of cholecystitis. We concluded that the present GWA developed directly from the gallbladder as a result of the potential spread of infection during cholecystitis.

Endoscopy, endoscopic ultrasound (EUS) and CT are useful for diagnosing GWA (14-16). The details of this case indicate that early radiological and ultrasonographic examinations are also useful for diagnosing GWA and decreasing the patient’s risk of mortality. Furthermore, we noted that sudden bloating and vomiting may be indicative of GWA formation. GWAs have a sonographic appearance that is inhomogeneous, with predominant hypoechoic lesions located primarily in the muscularis propria or the submucosal layer. Recently, EUS has been described as being convenient for visualizing GWAs and, as first reported by Hu et al., being useful for making the initial GWA diagnosis (15). EUS findings indicative of diffuse type GWA include thickening of the gastric wall, primarily in the submucosa, with a blurred interface between the submucosa and muscularis propria (15, 17). In cases of localized GWA, EUS demonstrates the presence of hypoechoic lesions within the submucosa (10, 18).

Before the advent of routine antibiotic therapy, the mortality associated with GWAs was very high (>90%). Even with the use of antibiotics, the mortality rate remains greater than 60%, likely because the condition is often misdiagnosed and therapy is initiated late in the patient’s clinical course (9, 19). Endoscopic intervention, as well as antibiotic therapy, is efficacious for treating these lesions. We summarized previous reports of endoscopic interventions for GWA in Table. These papers indicate that the use of aggressive

![Image 1](https://via.placeholder.com/150)

Figure 1. Initial severe cholecystitis in December 2011. An abdominal computed tomography scan revealed that the cholecystitis had spread to the gastric antrum.

![Image 2](https://via.placeholder.com/150)

Figure 2. Images of the gastric outlet obstruction-induced gastric wall abscess (arrows) obtained on the day of hospitalization. (a) Ultrasonogram: the lesion is located primarily in an area of high signal echo intensity. (b) Abdominal computed tomography: the lesion is located in an area of high density. (c) T2-weighted magnetic resonance imaging: the lesion is a high-signal intensity lesion.
endoscopic intervention, in conjunction with antibiotic therapy, is especially effective for treating localized GWAs. The endoscopic interventions employed in these studies were varied and included the use of biopsy forceps, needle knife incision, mucosal resection, and EUS-guided drainage. Our method of pus aspiration provides another option for GWA

Table. Summary of Published Cases of Endoscopic Treatment of Gastric Wall Abscesses

<table>
<thead>
<tr>
<th>Ref. No.</th>
<th>Published Year</th>
<th>Age (years)/Sex</th>
<th>Abscess Formation</th>
<th>Endoscopic Treatment Procedure</th>
<th>Use of Antibiotics</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
<td>1989</td>
<td>74/F</td>
<td>Localized</td>
<td>Biopsy forceps</td>
<td>+</td>
</tr>
<tr>
<td>8</td>
<td>1996</td>
<td>51/M</td>
<td>Diffuse</td>
<td>Biopsy forceps</td>
<td>+</td>
</tr>
<tr>
<td>26</td>
<td>1998</td>
<td>27/F</td>
<td>Localized</td>
<td>Biopsy needle</td>
<td>+</td>
</tr>
<tr>
<td>27</td>
<td>1998</td>
<td>81/?</td>
<td>Localized</td>
<td>Needle knife</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>2001</td>
<td>63/M</td>
<td>Localized</td>
<td>Forceps</td>
<td>-</td>
</tr>
<tr>
<td>28</td>
<td>2004</td>
<td>44/M</td>
<td>Localized</td>
<td>Biopsy</td>
<td>+</td>
</tr>
<tr>
<td>17</td>
<td>2007</td>
<td>36/M</td>
<td>Diffuse</td>
<td>Snare mucosa resection</td>
<td>+</td>
</tr>
<tr>
<td>29</td>
<td>2007</td>
<td>62/M</td>
<td>Localized</td>
<td>Needle knife</td>
<td>?</td>
</tr>
<tr>
<td>30</td>
<td>2008</td>
<td>28/F</td>
<td>Localized</td>
<td>Cystotome/stent</td>
<td>+</td>
</tr>
<tr>
<td>31</td>
<td>2010</td>
<td>63/F</td>
<td>Localized</td>
<td>Insulated-tip knife</td>
<td>+</td>
</tr>
<tr>
<td>32</td>
<td>2010</td>
<td>48/M</td>
<td>Localized</td>
<td>Needle knife/catheter for drainage</td>
<td>+</td>
</tr>
<tr>
<td>21</td>
<td>2012</td>
<td>50/M</td>
<td>Diffuse</td>
<td>Needle knife</td>
<td>+</td>
</tr>
<tr>
<td>33</td>
<td>2013</td>
<td>65/M</td>
<td>Localized</td>
<td>EUS-guided drainage</td>
<td>+</td>
</tr>
<tr>
<td>34</td>
<td>2014</td>
<td>63/F</td>
<td>Localized</td>
<td>Endoscopic transgastric drainage</td>
<td>+</td>
</tr>
<tr>
<td>Our case</td>
<td></td>
<td>83/M</td>
<td>Localized</td>
<td>Aspirated pus with an endoscope</td>
<td>+</td>
</tr>
</tbody>
</table>

Figure 3. Upper gastrointestinal endoscopy performed on the day after admission. (a) Upper gastrointestinal endoscopy showed that the abscess under the mucous membrane of the gastric antrum had disintegrated naturally (arrow). (b) Pus exuded from the abscess and was aspirated with an endoscope. After this procedure, the abscess reduced in size (arrow).

Figure 4. Follow-up abdominal computed tomography (CT) performed after treatment of the gastric wall abscess. On follow-up abdominal CT for the cholecystitis and gastric wall abscess, the gallbladder was found to adhere to the gastric antrum. Gastric outlet obstruction was not observed on this CT scan. Fig. 4a shows the findings noted eight months later, and Fig. 4b shows the findings noted 14 months after treatment of the gastric wall abscess.
treatment, if the pus can be disintegrated.

EUS is useful as both a diagnostic tool and treatment method for GWA. Munroe and Chen provided the first report of EUS being used as a therapeutic intervention in a patient with GWA (20), whereas Yang and Yen suggested that EUS examinations are useful for the early diagnosis and treatment of GWAs (21). In the same report, the authors also noted that the associated pus may be too viscous for aspiration with a small-gauge needle; a large-gauge needle should be used to puncture a GWA. On the other hand, EUS intervention carries the risk of creating a GWA, as reported by Itonaga et al. (22), who described the case of a patient with GWA (20), whereas Yang and Yen suggested that treatment with prophylactic antibiotics may be necessary (22). In the present case, the GWA was not treated surgically; rather, the abscess disintegrated naturally, and the pus was removed endoscopically. Finally, the treatment also involved systemic antibiotic administration, thus allowing the patient to recover without surgical intervention.

Gastric outlet obstruction (GOO) includes obstruction in the antropyloric area and/or bulb or post-bulbar duodenal segments. Although malignancies are common causes of GOO (23, 24), a significant number of patients have benign causes. GWAs, also causes of GOO, are rare and potentially fatal conditions that require early diagnosis and appropriate management. Endoscopy, CT, ultrasound and pathogen cultures can be helpful in diagnosing these lesions.

The authors state that they have no Conflict of Interest (COI).

References