Perforation of a Duodenal Ulcer into a Non-Parasitic Liver Cyst: A Rare Case of a Penetrate Hole Blockaded with Conservative Medical Management

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Abstract

An 88-year-old Japanese woman was admitted to our hospital for abdominal pain with a raised inflammatory reaction. Esophagogastroduodenoscopy (EGD) and computed tomography (CT) showed a duodenal ulcer punching a liver cyst. Since neither ascites nor free air were detected on CT and her family did not wish for aggressive medical treatment, the patient received clinical observation and conservative management. Antibiotic and proton-pump inhibitor therapy was effective, and she exhibited an uneventful recovery. A reexamination of EGD and CT confirmed that the fistulous tract between the liver cyst and duodenum was blockaded.

Key words: duodenal ulcer, non-parasitic liver cyst, esophagogastroduodenoscopy (EGD), computed tomography (CT)

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Introduction

Simple or non-parasitic hepatic cysts are estimated to affect between 1% and 2.5% of the general population. These cysts occur at ages; however, they are observed more frequently in the elderly population (1). Although the cysts are generally solitary, they can occur simultaneously (“several solitary cysts”), even in the absence of polycystic liver disease (2). Most liver cysts are present as thin-walled bubbles in the liver and usually do not produce any signs or symptoms; such cysts are commonly diagnosed incidentally on ultrasound or computed tomography (CT) examinations. In cases of serious complications of non-parasitic liver cysts, the cysts sometimes rupture spontaneously, with (3) or without (4) hemorrhage, or become infected (3, 5), often inducing pain; therefore, special treatment must be considered (2). Direct perforation of a liver cyst into the upper gastrointestinal tract is a very unusual event that requires emergency surgery (6-8). A few cases of communication of a cyst with the duodenum (6, 8-12) and stomach (7, 13) have been reported (Table 1). Perforation of a duodenal ulcer into a non-parasitic liver cyst is an extremely rare complication. To the best of our knowledge, no cases of naturally cured perforation of duodenal ulcers into hepatic cysts following conservative medical management have been previously reported.

Case Report

An 88-year-old Japanese woman was admitted from a group home for senile elderly to a nearby hospital complaining of faintness, appetite loss, and occasional right upper quadrant abdominal pain. The patient denied a history of abdominal trauma. She complained of occasional epigastric pain starting 10 days prior to the admission and had been prescribed an H₂ blocker for a stomachache by her family physician. Despite receiving this prescription, she continued to complain of occasional appetite loss and right upper quadrant abdominal pain. She lost consciousness for five minutes after sitting down in her wheelchair and was taken to a nearby hospital by ambulance. Her white blood cell count and C-reactive protein (CRP) level were high at 22,500/μL and 16.1 mg/dL, respectively. She was subsequently transferred to our hospital for a further evaluation

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and treatment the day after being hospitalized.

Two months prior to the present admission, a CT scan was performed in our hospital under an order by the patient’s family physician. The patient was found to have two large cysts in the liver, although no air was detected in either cyst (Fig. 1-i). At that time, CT demonstrated no free air in the abdomen; however, a cyst observed in subsegment 4 (S4) of the left liver lobe contained air bubbles, and a fistulous tract was noted between the cyst and duodenum (Fig. 1-ii, 2). A subhepatic extension of the cyst was in close proximity to the duodenum. The content of the cyst was draining into the duodenum, while the fistula allowed gas in the duodenum to penetrate into the cyst. Since neither ascites nor free air were detected on CT and her family did not wish for aggressive medical treatment, the patient received remedial fasting, clinical observation and conservative treatment. Esophagogastroduodenoscopy (EGD) confirmed the diagnosis following the visualization of a fistulous orifice of the anterior aspect of the duodenum with protrusion of the hepatic cyst membrane (Fig. 3).

The patient’s post-medication course was uneventful, and the infusion of antibiotics, including sulbactam/cefoperazone (SBT/CPZ; 1 g i.v. twice daily), for 10 days and the administration of a proton-pump inhibitor, omeprazole (20 mg i.v. twice daily), for three weeks were effective (Table 2). Twenty-five days later, repeat EGD was performed, which revealed the blockade of the penetrated hole (Fig. 4). On day 29, CT confirmed that the penetrated cyst had shrunk

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**Table 1. Previous Reports of Direct Perforation of Liver Cyst into the Upper Gastrointestinal Tract**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
<th>Age</th>
<th>Year/Sex</th>
<th>Presenting symptoms</th>
<th>Rupture site</th>
<th>Previous treatment</th>
<th>Procedure</th>
<th>Complications</th>
<th>Follow-up/Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>1990</td>
<td>83</td>
<td>M</td>
<td>severe abdominal pain, shock status</td>
<td>duodenum</td>
<td>Nil</td>
<td>emergency operation</td>
<td>N.S.</td>
<td>life-saved</td>
</tr>
<tr>
<td>8</td>
<td>2003</td>
<td>60</td>
<td>M</td>
<td>abdominal pain</td>
<td>duodenum</td>
<td>Nil</td>
<td>emergency operation</td>
<td>N.S.</td>
<td>life-saved</td>
</tr>
<tr>
<td>9,12</td>
<td>2003</td>
<td>82</td>
<td>F</td>
<td>abdominal pain</td>
<td>duodenum</td>
<td>Nil</td>
<td>scheduled operation</td>
<td>N.S.</td>
<td>1 month, symptom free</td>
</tr>
<tr>
<td>10</td>
<td>2011</td>
<td>80</td>
<td>F</td>
<td>fever, abdominal pain</td>
<td>duodenum</td>
<td>steroids for RA</td>
<td>scheduled operation</td>
<td>N.S.</td>
<td>7 days, symptom free</td>
</tr>
<tr>
<td>11</td>
<td>2013</td>
<td>69</td>
<td>M</td>
<td>nausea, appetite loss</td>
<td>duodenum</td>
<td>Nil</td>
<td>Nil</td>
<td>N.S.</td>
<td>27 days, symptom free</td>
</tr>
<tr>
<td>7</td>
<td>2007</td>
<td>78</td>
<td>M</td>
<td>abdominal pain</td>
<td>stomach</td>
<td>Nil</td>
<td>emergency operation</td>
<td>N.S.</td>
<td>6 months, symptom free</td>
</tr>
<tr>
<td>13</td>
<td>2013</td>
<td>68</td>
<td>F</td>
<td>fever</td>
<td>stomach</td>
<td>HD for renal failure</td>
<td>percutaneous drainage</td>
<td>N.S.</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

All published series of patients undergoing medical treatment for direct perforation of a liver cyst into the upper gastrointestinal tract are listed in positional and chronological order.

Nil: nothing, N.S.: not specified, RA: rheumatoid arthritis, HD: hemodialysis

**Figure 1.** (i) A CT scan performed two months prior to admission. There were two large cysts in the left liver lobe; however, no air was detected in either cyst (arrow). (ii) CT section at the level of the left subhepatic space. A large cystic mass (arrow) is present in the left liver lobe with air bubbles extending to the subhepatic space on admission. (iii) CT section of the hepatic cyst (arrow) on day 29 that had decreased in size and naturally drained. (iv) CT section of the hepatic cyst (arrow) on day 79 that had further decreased in size and been resorbed.
The clinical data showed that the C-reactive protein level quickly decreased following treatment with antibiotics. In addition, the anemia improved due to the blockade of the penetrated duodenal hole induced by the proton-pump inhibitor. Antibiotics, including sulbactam/ceperazone (SBT/CPZ; 1 g i.v. twice daily), were infused for 10 days.

WBC: white blood cell, CRP: C-reactive protein, SAA: serum amyloid A, RBC: red blood cell, Hb: hemoglobin, Hct: hematocrit

CT examination on Day 9

![CT examination](image1)

Figure 2. A CT section showed air bubbles in the left liver lobe with a fistulous tract between the cyst and duodenum.

Esophagastroduodenoscopy (EGD) results on Day 9

![Esophagastroduodenoscopy](image2)

Figure 3. EGD confirmed the diagnosis by visualizing a fistulous orifice (arrow) in the anterior aspect of the first duodenum with protrusion of the hepatic cyst membrane.

### Table 2. Clinical Data

<table>
<thead>
<tr>
<th>Unit</th>
<th>Day 1</th>
<th>Day 3</th>
<th>Day 8</th>
<th>Day 15</th>
<th>Day 26</th>
<th>Day 79</th>
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</thead>
<tbody>
<tr>
<td>WBC (/µL)</td>
<td>15,700</td>
<td>6,800</td>
<td>10,800</td>
<td>5,600</td>
<td>4,600</td>
<td>6,600</td>
</tr>
<tr>
<td>CRP (mg/dL)</td>
<td>25.96</td>
<td>7.56(5+)</td>
<td>0.96(1+)</td>
<td>0.10(-)</td>
<td>0.03(-)</td>
<td>0.02(-)</td>
</tr>
<tr>
<td>SAA (µg/dL)</td>
<td>---</td>
<td>208.8</td>
<td>58.1</td>
<td>2.9</td>
<td>3.8</td>
<td>5.5</td>
</tr>
<tr>
<td>RBC (/µL)</td>
<td>287</td>
<td>327</td>
<td>330</td>
<td>343</td>
<td>332</td>
<td>452</td>
</tr>
<tr>
<td>Hb (g/dL)</td>
<td>7.3</td>
<td>8.7</td>
<td>8.5</td>
<td>9.5</td>
<td>9.7</td>
<td>14.2</td>
</tr>
<tr>
<td>Hct (%)</td>
<td>24.8</td>
<td>28.1</td>
<td>29</td>
<td>31.9</td>
<td>32.2</td>
<td>44.2</td>
</tr>
</tbody>
</table>

SBT/CPZ 2g/day

The clinical data showed that the C-reactive protein level quickly decreased following treatment with antibiotics. In addition, the anemia improved due to the blockade of the penetrated duodenal hole induced by the proton-pump inhibitor. Antibiotics, including sulbactam/ceperazone (SBT/CPZ; 1 g i.v. twice daily), were infused for 10 days.

WBC: white blood cell, CRP: C-reactive protein, SAA: serum amyloid A, RBC: red blood cell, Hb: hemoglobin, Hct: hematocrit

and the amount of internal air had decreased (Fig. 1-iii, 4). The patient was discharged in excellent general condition 30 days after admission (Table 2) and returned to her group home. Eleven weeks later, she was reexamined with CT at our hospital under an order by her family physician, which demonstrated that the penetrated cyst had was shrunk in size and been resorbed (Fig. 1-iv).

### Discussion

Most non-parasitic hepatic cysts are congenital in origin, solitary, asymptomatic and not life-threatening. Some cysts require no treatment, as they can regress spontaneously. Hepatic cysts can occur at any age; however, the majority are found in patients 40 to 60 years of age (14).
Esophago-gastro-duodenoscopy (EGD) results on Day 25 and CT examination on Day 29

Normally, perforation of a cyst into the upper gastrointestinal tract requires surgical treatment after diagnosis (6-10, 12). However, the patient in this case did not undergo surgery due to her family’s request. She was fortunate because neither ascites nor free air were present in the abdomen, and treatment with remedial fasting, an infusion of antibiotics (SBT/CPZ) and the administration of a proton-pump inhibitor (omeprazole) was effective. It is also noteworthy that the cyst perforated the upper gastrointestinal tract. The treatment of choice for infected cysts is a combination of percutaneous drainage and antimicrobial therapy. Percutaneous transhepatic drainage is easily performed under ultrasonic guidance. However, the fluid usually refills the cyst, as secretions from the epithelial cell lining of hepatic cysts inhibit cyst obliteration (4). Recurrence of simple hepatic cysts after drainage alone has been reported (5). To prevent such recurrence, after removing the fluid, the physician may inject a substance called a sclerosant into the cyst to help stick the walls together. Therefore, following drainage, a sclerosant [such as alcohol (15)] may be injected into the cyst in an attempt to “stick the walls together” and prevent the cyst from being refilled with fluid. Yoshida et al. recommended that symptomatic simple hepatic cysts be treated with percutaneous transhepatic drainage following the insertion of an 8- or 9-French catheter into the cyst, followed by the injection of 200 mg of minocycline hydrochloride (a sclerosant) in 10 mL of saline and clamping for 30 minutes for seven to eight days, after which the catheter is removed. They concluded that the use of multiple injections of minocycline hydrochloride is a safe, definitive treatment for symptomatic solitary hepatic cysts (16). In the present case, the liver cyst was connected to the duodenum. Therefore, the roof of the liver cyst was removed (so called “unroofing” (17)) and the inside content was naturally drained, after which gastric juice as a sclerosant destroying the epithelial cell lining of the hepatic cyst poured into the lesion.

In conclusion, we herein described the case of a patient who exhibited perforation of a duodenal ulcer into a non-parasitic liver cyst. The perforation of the duodenal ulcer into the hepatic cyst was naturally cured with conservative medical management. To our knowledge, this is the first report to describe a case of naturally cured perforation of a duodenal ulcer into a hepatic cyst following conservative medical management.

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

References


