

Abscess of the Round Ligament of the Liver Associated with Acute Obstructive Cholangitis and Septic Thrombosis

Norikazu Arakura¹, Yayoi Ozaki¹, Sachie Yamazaki², Kazuhiko Ueda², Masafumi Maruyama¹, Yoshimi Chou¹, Ryo Kodama¹, Mari Takayama¹, Hideaki Hamano¹, Eiji Tanaka¹ and Shigeyuki Kawa³

Abstract

A man with abscess of the round ligament of the liver associated with acute obstructive suppurative cholangitis and portal thrombosis is reported. A 63-year-old man was admitted with epigastralgia and high fever. Blood tests showed elevation of hepato-biliary enzymes and coagulopathy consistent with acute obstructive suppurative cholangitis and disseminated intravascular coagulation. Computed tomography revealed a small abscess of the round ligament of the liver and left portal thrombosis. After endoscopic biliary stenting, antibiotics and thrombolytic therapy, the high fever, disseminated intravascular coagulation and portal thrombosis rapidly improved, and the round ligament abscess was also later resolved.

Key words: abscess of the round ligament of the liver, portal thrombosis, acute obstructive suppurative cholangitis

(*Inter Med* 48: 1885-1888, 2009)

(DOI: 10.2169/internalmedicine.48.2396)

Introduction

The round ligament of the liver is a remnant of the fetal umbilical vein. After birth, the fetal umbilical vein is completely obliterated and replaced by fibrous connective tissue. In patients with portal hypertension, as in liver cirrhosis, the round ligament of the liver can become patent. Abscess of the round ligament of the liver can present with the clinical picture of an acute abdomen, but only a few cases have been reported in the English language literature (1-6). The infectious etiology has reportedly originated from acute cholangitis (6), acute cholecystitis (3), and acute pancreatitis (6); in some cases the origin is unknown. In all reported cases, surgical treatment with resection of the ligament was performed. Here, we report the case of a patient with abscess of the round ligament of the liver that was potentially a result of acute obstructive suppurative cholangitis (AOSC) and portal thrombosis, and resolved with conservative management.

Case Report

A 63-year-old man with a history of cholecystolithiasis was admitted to our hospital on December 2008 with epigastralgia, high fever, and shaking chills. Physical examination showed epigastric tenderness but no infection around the umbilicus. Laboratory evaluation revealed elevation of hepato-biliary enzymes and coagulopathy consistent with disseminated intravascular coagulation (DIC) (Table 1). Computed tomography (CT) showed a small cystic lesion accompanied with a finger of inflammatory change around the cyst consistent with abscess of the round ligament of the liver, cholecystolithiasis, and left portal thrombosis, but no dilatation of intrahepatic and common bile ducts (Fig. 1). Though serum amylase was elevated, imaging showed no findings suggestive of acute pancreatitis. Because the DIC was thought to be a sequela of acute obstructive suppurative cholangitis, emergent endoscopic retrograde cholangiopancreatography (ERCP) was performed. ERCP showed no

¹Department of Gastroenterology, Shinshu University School of Medicine, Matsumoto, ²Department of Radiology, Shinshu University School of Medicine, Matsumoto and ³Center for Health, Safety and Environmental Management, Shinshu University, Matsumoto

Received for publication April 22, 2009; Accepted for publication July 13, 2009

Correspondence to Dr. Norikazu Arakura, arakuran@shinshu-u.ac.jp

Table 1. Laboratory Data

Hematology		pAMY	1890 mg/dL
WBC	4,940 /mm ³	BUN	27 mg/dL
Neut.	93.7 %	Cr	0.98 mg/dL
RBC	390 × 10 ⁴ /mm ³	Na	140 mEq/L
Hb	12.7 g/dL	K	3.0 mEq/L
Ht	37.0 %	Cl	107mg/dL
Plt	6.4 × 10 ⁴ /mm ³	Ca	8.7 mg/dL
Blood Chemistry		FPG	117 mg/dL
TP	6.4 g/dL	Serological examination	
Alb	3.5 g/dL	CRP	3.09 mg/dL
T.Bil	3.48 mg/dL	Hemostatic examination	
D.Bil	2.19 mg/dL	PT%	64.8 %
AST	222 IU/L	AT-III	88.9 %
ALT	113 IU/L	FDP-DD	20.6 µg/mL
ALP	498 IU/L		
γ -GTP	221IU/L		
AMY	2044 mg/dL		



Figure 1. Late phase of contrast-enhanced abdominal CT scan on admission. Thrombosis of the left portal vein (arrow) was detected.



Figure 2. Early phase of contrast-enhanced abdominal CT scan performed 16 days after admission. The size of the round ligament abscess (arrow) was increased compared with that seen on admission, but clear encapsulation and internal liquefaction were shown.

cholangiolithiasis, but did identify suppurative bile fluid and narrowing of the distal bile duct. Gram-positive cocci identified as *Streptococcus anginosus* were cultured from the bile fluid and blood. Biopsy of the narrowed lower bile duct was negative for malignant cells, and was consistent with papillitis secondary to spontaneous excretion of bile stone(s). Following endoscopic biliary stenting and conservative treatment with antibiotics (meropenem trihydrate), the patient's high fever and DIC were rapidly improved and epigastric tenderness disappeared. With thrombolytic therapy, the portal thrombosis showed near-complete resolution, which was confirmed by CT on hospital day 16. The size of the round ligament abscess slightly increased with a capsule and liquid in the capsule had been clearly formed (Fig. 2); treatment

continued with oral levofloxacin monotherapy due to his improving clinical and laboratory evaluation. CT performed on hospital day 35 showed apparent improvement in the round ligament abscess.

Discussion

The round ligament of the liver, which is also referred to as the "ligamentum teres hepatis" and the "falciform ligament", represents the remnant of the fetal umbilical vein, and is a degenerative ligament of approximately 17 cm in length that extends from umbilicus to the umbilical portion

Table 2. Case Reports of Abscess or Necrosis of the Ligamentum Teres Hepatis, Falciform Ligament, and Round Ligament of the Liver

Authors (Year)	Age (years)	Sex (M or W)	Presentation	Concomitant disease	Microbiology	Therapy
Charuzi 1) 1976	75	W	Abdominal pain High fever	None	(-)	Surgical resection
Watson 2) 1988	84	W	Abdominal pain vomiting	Cholecystolithiasis	(-)	Surgical resection
Migliaccio 3) 1988	Not reported	Not reported	Right upper and epigastric rebound tenderness	Acute cholecystitis	Not reported	Surgical resection
Losanoff 4) 2002	18	M	Abdominal pain vomiting	None	Escherichia coli (peritoneal pus)	Surgical resection
Martin 5) 2004	52	W	Severe epigastralgia	Cholecystolithiasis	Not reported	Surgical resection
Tsukada 6) 2008	70	W	Abdominal pain High fever	Acute cholangitis Cholangiolithiasis Acute pancreatitis	Staphylococcus epidermidis	Eodoscopic papillotomy → Surgical resection
Current case 2009	63	M	Epigastralgia High fever	Acute cholangitis Cholangiolithiasis Portal thrombosis	Streptococcus anginosus (blood)	Eodoscopic papillotomy+ Endoscopic biliary drainage

of the portal vein through the falciform ligament of the liver (7). Missalek reported that the round ligament of the liver could be detected by ultrasound in 87% of healthy subjects (8).

Including the present case, only seven reports of abscess of the round ligament of the liver were identified in the English language literature (1-6), indicating the rarity of this condition (Table 2). A review of these cases shows a preponderance of elderly women, with the most common initial presentation being abdominal pain, fever, and vomiting. The origin of infection was apparent in only 3 cases: 1 with acute cholecystitis and 2 with acute cholangitis (1 case accompanied by acute pancreatitis). Two cases were complicated by cholecystolithiasis without apparent infection. All reported cases underwent surgical treatment including early resection of the inflammatory lesions in 5 patients with the symptoms of the peritonitis (1-5), and late resection of an abscess resistant to conservative treatment in one patient (6). In general, surgical treatment should be performed regardless of the size of the cyst immediately after the diagnosis of abscess complicated with peritonitis is reached, due to the risk of extensive spread and severe peritonitis (4). In the chronic phase, the abscess that does not ameliorate by conservative treatment should be removed regardless of the size of the cyst, because the abscess is easily affected by external force on the epigastrium. Recurrence would be unlikely, if the cavity of the abscess disappears after conservative therapy. With improvements in CT and US examinations, demonstration of the round ligament of the liver and prompt di-

agnosis of an abscess is possible. Based on the symptoms of acute abdomen, hematological studies and CT or US imaging, it should be possible to identify cases warranting emergent surgery (4).

Only a few reports have suggested that isolated AOSC can result in an abscess of the round ligament; thus it is possible that in the present case portal thrombosis of the left branch played a major role in the formation of abscess. Because portal thrombosis (septic thrombosis) of the left branch and round ligament abscess were identified on CT at the time of the hospitalization, it is likely that an infectious course probably progressed from AOSC to a septic state, ultimately resulting in the formation of a septic thrombosis and stasis of the left branch of portal vein, leading to round ligament abscess. The major causes of portal vein thrombosis are reported to be liver cirrhosis, neoplasm, infection, inflammatory disease (notably pancreatitis), and myeloproliferative disorders. In patients without cirrhosis or malignant disease, approximately 10% - 25% of cases with portal vein thrombosis are associated with sepsis (9-12). Other infectious causes of portal vein thrombosis might include portal pyemia secondary to suppurative appendicitis, biliary tract infection, post-abdominal surgery sepsis, amoebic colitis, acute necrotizing pancreatitis, and diverticulitis (9). In the current case, transient portal thrombosis may have been due to AOSC and sepsis, because the thrombosis disappeared immediately following treatment of AOSC and thrombolytic therapy. However, it cannot be denied that portal thrombosis was secondary to round ligament abscess contiguous with

the left portal vein.

The present patient had a history of cholecystolithiasis and cholangiolithiasis, which may have been the cause of AOSC, although this was not confirmed on ERCP. Narrowing of the lower bile duct corresponded to papillitis induced by recurrent cholangiolithiasis, and no malignant cells identified in the biopsy specimens. The biliary stent was removed after improvement of AOSC, and serum hepatobiliary enzyme levels remained within normal limits.

Here, we report an informative case of abscess of the round ligament of the liver that was possibly caused by

AOSC and septic thrombosis at left portal vein, and successfully treated conservatively. All of the reported cases of abscess of the round ligament of the liver have been treated surgically. Because the present case was accompanied by AOSC and portal thrombosis, conservative management with endoscopic biliary drainage and administration of antibiotics and a thrombolytic agent was performed. This management strategy was successful, although it took approximately 30 days to reach apparent improvement in the round ligament abscess.

References

1. Charuzi I, Freund H. Gangrene of the hepatic round ligament causing diffuse peritonitis: a case report. *Am Surg* **42**: 925-926, 1976.
2. Watson SD, McComas B, Rannick GA, Stanton PA Jr. Gangrenous ligamentum teres hepatis causing acute abdominal symptoms. *South Med J* **81**: 267-269, 1988.
3. Migliaccio AV. Disease of round ligament of the liver simulating acute gall bladder. Cases presenting as acute surgical emergencies not previously reported. *R I Med J* **71**: 239-242, 1988.
4. Losanoff JE, Kjossev KT. Isolated gangrene of the round and falciform liver ligaments: a rare cause of peritonitis. Case report and review of the world literature. *Am Surg* **68**: 751-755, 2002.
5. Martin TG. Video laparoscopic treatment for isolated necrosis and abscess of the round ligament of the liver. *Surg Endosc* **18**: 1395, 2004.
6. Tsukuda K, Furutani S, Nakahara S, et al. Abscess formation of the round ligament of the liver: report of a case. *Acta Med Okayama* **62**: 411-413, 2008.
7. Ying DJ, Ho GT, Cai JX. Anatomic bases of the vascularized hepatic teres ligament flap. *Surg Radiol Anat* **19**: 293-294, 1997.
8. Missalek W. Identification of the ligamentum teres hepatis by ultrasound and the clinical importance of its thickening. *Trop Geogr Med* **45**: 131-134, 1993.
9. Cohen J, Edelman RR, Chopra S. Portal vein thrombosis: a review. *Am J Med* **92**: 173-182, 1992.
10. Castillo M, Murphy B. Septic portal vein thrombophlebitis: computed tomography appearance. Case report. *Comput Radiol* **10**: 289-292, 1986.
11. Lim GM, Jeffrey RB, Ralls PW, Marn CS. Septic thrombosis of the portal vein: CT and clinical observations. *J Comput Assist Tomogr* **13**: 656-658, 1989.
12. Wakisaka M, Mori H, Kiyosue H, Kamegawa T, Uragami S. Septic thrombosis of the portal vein due to peripancreatic ligamental abscess. *Eur Radiol* **9**: 90-92, 1999.