The Turkish Journal of ***Academic Gastroenterology*** • *2026; 25(2): 157-162*

Manuscript Received: *18.12.2025* • ***Accepted:*** *22.06.2026*

CASE REPORT

A rare cause of acute abdomen: Falciform ligament appendagitis

Nadir görülen bir akut batın sebebi: Falsiform ligaman apendajiti

Elif ALICI BIYIK[1], Mehmet YILDIZ[2], Engin ALTINKAYA[2], Hilmi ATASEVEN[2]

Departments of [1]Family Medicine and [2]Gastroenterology, Sivas Cumhuriyet University School of Medicine, Sivas, Türkiye

ABSTRACT • Diseases of the hepatic falciform ligament, which is anatomically located between the right and left lobes of the liver and separates these lobes, are extremely rare. Falciform ligament appendagitis may develop due to vascular compromise or extension of inflammation from adjacent organs. Patients who develop falciform ligament appendagitis require supportive treatment and antibiotics if needed. Surgical treatment may also be required in some cases. In this case report, we present a patient who was admitted to the hospital with abdominal pain, followed up with the diagnoses of acute pancreatitis and acute cholecystitis, and who subsequently developed an acute abdomen-like clinical presentation in the following period despite improvement in the follow-up and was diagnosed with hepatic falciform ligament appendagitis. Falciform ligament appendagitis should be considered in the differential diagnosis of patients with acute abdomen.

Keywords: Falciform ligament appendagitis, abdominal pain, acute abdomen, case report

ÖZET • Anatomik olarak karaciğerin sağ ve sol lobları arasında bulunan ve bu lobları ayıran hepatik falsiform ligamentin hastalıkları son derece nadir görülmektedir. Dolaşım bozukluğu ya da komşu organlardaki enflamasyonun yayılması ile falsiform ligaman apendajiti gelişebilir. Falsiform ligaman apendajiti gelişen hastalarda destekleyici tedavi ve ihtiyaç halinde antibiyotik kullanımı gereklidir. Bazı vakalarda cerrahi tedavi de gerekebilir. Bu olgu sunumunda; karın ağrısı şikâyeti ile hastaneye başvuran, akut pankreatit ve akut kolesistit tanıları ile takip edilen ve takibinde iyileşme göstermesine karşın ilerleyen dönemde akut batın benzeri klinik gelişen, hepatik falsiform ligaman apendajiti tanısı tespit edilen bir olguyu sunduk. Akut batın benzeri klinik bulguları olan hastalarda ayırıcı tanılar arasında falsiform ligaman apendajiti akla gelmelidir.

Anahtar kelimeler: Falsiform ligaman apendajiti, karın ağrısı, akut batın, olgu sunumu

INTRODUCTION

The hepatic falciform ligament, which morphologically divides the liver into two main lobes, right and left, is anatomically located between these two lobes (1). Diseases originating from the hepatic falciform ligament itself are very rare. It is usually secondary to inflammatory processes of the surrounding tissues (2).

In this article, we report a case who was hospitalised in our clinic with acute pancreatitis and acute cholecystitis, who was diagnosed with falciform ligament appendagitis (FLA) based on clinical, laboratory, and radiological findings, and who subsequently developed recanalization of the paraumbilical vein during follow-up. This case report has been prepared with written consent from the patient and without using any data that could identify the patient.

CASE REPORT

A 73-year-old male patient was admitted to the emergency department with complaints of epigastric pain radiating to the back. On physical examination, body temperature was 36.7 C°, pulse rate 83/min, respiratory rate 21/min, blood pressure

Correspondence: Elif ALICI BIYIK • Department of Family Medicine, Sivas Cumhuriyet University, Faculty of Medicine, Sivas, Türkiye • *E-mail: drelif.alici@gmail.com* • *A rare cause of acute abdomen: Falciform ligament appendagitis* • *The Turkish Journal of Academic Gastroenterology 2026;25:157-162.* ***Doi: 10.17941/agd.1981487***

160/80 mmHg and abdominal examination was unremarkable except tenderness in the epigastric region. Laboratory tests were as follows; white blood cell (WBC): 14.2 $\times 10^9$/L, C-reactive protein (CRP): 6.3 mg/L, Amylase: 2022 U/L, Lipase: 2725 U/L, alanine aminotransferase (ALT): 175 U/L, aspartate aminotransferase (AST): 318 U/L, gamma-glutamyl transferase (GGT): 726 U/L, alkalin posphatase (ALP): 168 U/L, lactate dehydrogenase (LDH): 570 U/L (Table 1). Hepatobiliary ultrasonography (USG) performed in the emergency department revealed a distended gallbladder with a transverse diameter of 43 mm and gallbladder wall thickness of 7 mm. A 27-mm gallstone was observed in the neck of the gallbladder. Acute pancreatitis and acute cholecystitis were considered with these clinical and laboratory findings. The patient was admitted to the gastroenterology service for follow-up and treatment.

The patient had a history of hypertension and had been hospitalised and treated twice before with a diagnosis of acute pancreatitis. His previous pancreatitis histories were 6 years and 1 year ago. The patient stated that he did not use medication for hypertension and did not attend follow-up visits. His family history was unremarkable. Patient's oral intake was stopped and appropriate fluid replacement was started. Metronidazole 3 x 500 mg IV, ciprofloxacin 2 x 400 mg IV, pantoprazole 1 x 40 mg IV were started.

In the follow-up of the patient, there was a significant improvement in abdominal pain although no painkillers were given. On the fourth day of hospitalisation, the abdominal pain started to intensify again. Because of these findings, physical examination revealed marked tenderness in the epigastric region and diffuse abdominal defence. A mass with a diameter of approximately 5 x 6 cm was palpated in the epigastric region. In laboratory tests, there was regression in liver function tests, amylase and lipase values, while acute phase reactants tended to increase. CRP in particular was significantly elevated. Although the progression to necrotising pancreatitis was also considered,

Table 1 Laboratory findings on patient's follow-up.

Laboratory Findings	1st	2nd	4th	6th	10th	17th	Referance Range	Units
WBC	14.2	10.84	12.18	7.96	7.81	7.72	4-10.5	10^9/L
BUN	20.4	13.4	14.9	11.2	18.8	15.5	8-23	mg/dL
Creatinine	1.39	1.66	1.39	1.29	1.39	1.22	0.7-1.2	mg/dL
ALT	175	171	118	45	40	41	0-41	U/L
AST	318	88	54	17	33	24	0-40	U/L
LDH	570	395	347	340	311	263	135-225	U/L
GGT	726	566	475	320	267	171	10-71	U/L
ALP	168	165	135	129	133	147	40-130	U/L
T. Bilirubine	1.31	1.94	1.31	0.87	0.52	0.71	0.1-1.2	mg/dL
D. Bilirubine	0.38	1.16	1.08	0.49	0.26	0.37	0.0-0.3	mg/dL
Amylase	2022	927	373	94	165	195	28-100	U/L
Lipase	2725	379	166	107	226	133	13-60	U/L
CRP	6.3	105.7	243.2	216.0	86.3	34.6	0-5	mg/L

WBC: White blood cell count; BUN: Blood urea nitrogen; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; LDH: Lactate dehydrogenase; GGT: Gamma-glutamyl transferase; ALP: Alkaline phosphatase; T. Bilirubine: Total Bilirubine; D. Bilirubine: Direct Bilirubin (Conjugated Bilirubin); CRP: C-reactive protein.

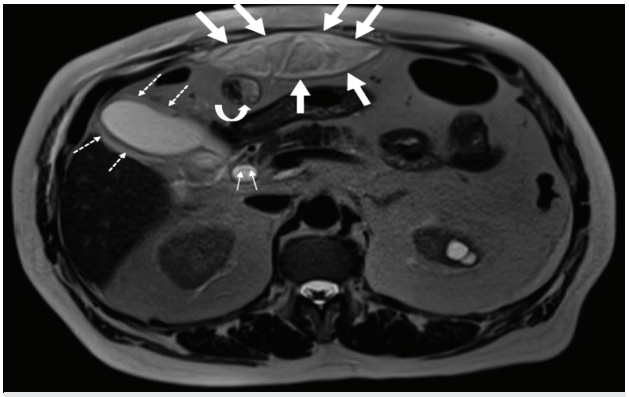

Figure 1 Axial T2 weighted imaging (T2WI) section shows hypointensity with leveling of the lumen of the choledochal duct (thin arrows), thickening and oedema of the gallbladder wall (dashed arrows), thickening and oedema of the ligamentum teres hepatis (oblique arrow) and fluid collection in the omentum adjacent to the anterior abdominal wall (thick arrows).

a clinical condition other than acute pancreatitis and acute cholecystitis was suspected with these data. After consultation with the infectious diseases department, antibiotherapy was changed to meropenem 3 x 1 gr IV. Magnetic resonance cholangiopancreatography performed to visualise the choledochal duct showed a common bile duct diameter of 13.5 mm. An appearance compatible with bile sludge was observed in the choledochal lumen. The transverse diameter of the gallbladder was 50 mm and the gallbladder wall was 7 mm. Free fluid was observed in fatty planes in the periportal and peripancreatic area. Fluid loculations measuring 106 x 40 x 90 mm were observed in the omentum adjacent to the anterior abdominal wall (Figure 1).

In the patient whose abdominal pain became severe again and acute abdomen was suspected, computed tomography (CT) imaging was also planned for differential diagnosis. Non-contrast abdominal CT was performed because of high creatinine levels. On CT, density increases secondary to intense inflammation in the falciform ligament and surrounding tissues, a well-demarcated, round-shaped hyperdense fatty mass lesion in the prehepatic fat planes in the ligamentum teres hepatis (round ligament) tracing, and fluid collection in the anterior abdominal wall neighbourhood were observed (Figure 2). Based on these findings, the patient was diagnosed as FLA. Nonsteroidal anti-inflammatory drugs were added to the treatment. On the sixth day of hospitalisation, abdominal pain and tenderness started to decrease and defence sign disappeared on abdominal examination. On the tenth day, pain and tenderness completely resolved. The patient was discharged on hospital day 17 with clinical and laboratory improvement (Table 1).

When the patient was discharged, creatinine value regressed and the gallbladder, sac wall, choledochal duct and choledochal lumen were normal

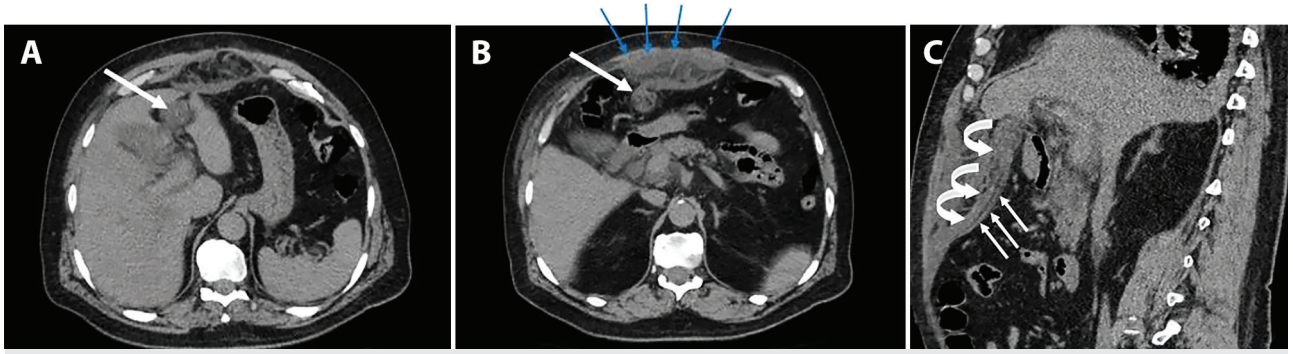

Figure 2 In axial contrast-negative CT images: **A:** Thickness of falciform ligament (arrow), **B:** Well circumscribed, round shaped hypodense fatty mass lesion in the ligamentum teres hepatis trace in the prehepatic fat planes (white arrow) and fluid collection in the neighbourhood of the anterior abdominal wall (blue arrows), **C:** Sagittal images showing the extension of the mass from the omentum to the anterior abdominal wall (oblique arrows) and the ligamentum teres hepatis (arrows).

on contrast-enhanced upper abdominal magnetic resonance imaging (MRI) performed for control. The pancreas was demonstrated homogeneous enhancement throughout and no appearance in favour of pancreatic necrosis was detected. Thickening of the falciform ligament, an oedematous, well circumscribed fatty mass lesion in the omental adipose tissue, measuring 25 mm in diameter at its widest point, and fluid collection in the neighbourhood of the anterior abdominal wall were persistent. Recanalization of the paraumbilical vein was also seen (Figure 3).

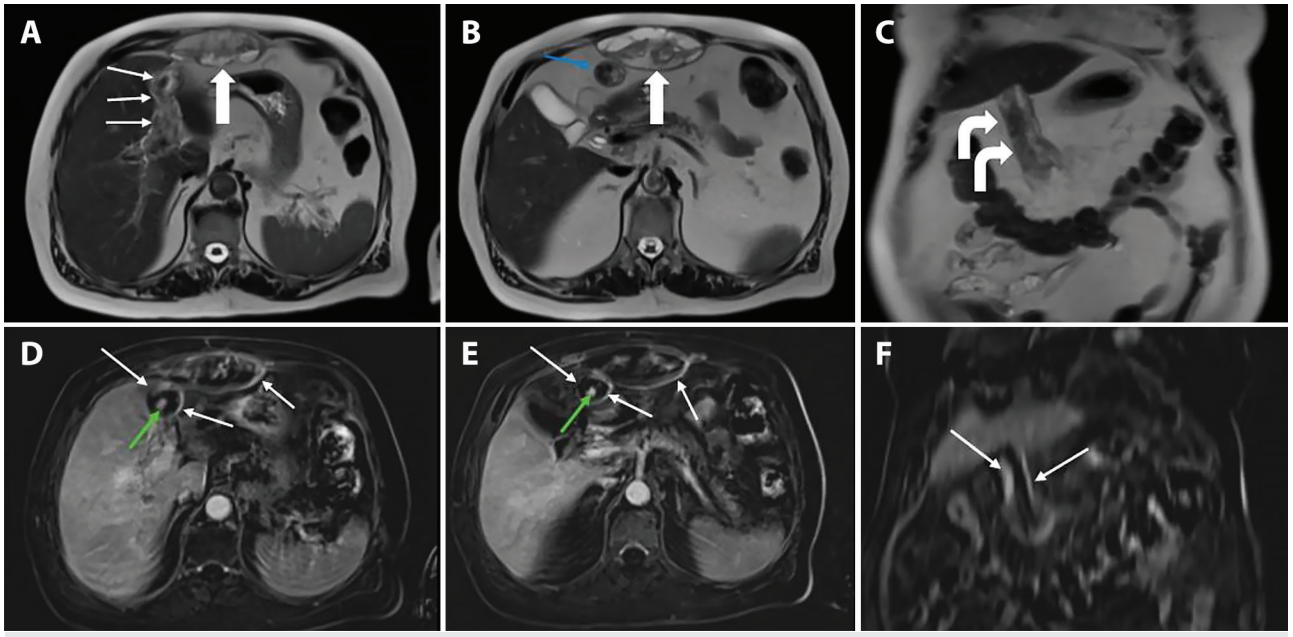

Figure 3 **A and B:** Axial T2WI images show thickening of the falciform ligament (thin white arrows), omental fatty tissue edematous, well circumscribed mass lesion (thin blue arrow), fluid collection in the neighbourhood of the anterior abdominal wall (thick white arrow). **C:** Coronal sections show extension of the oedematous mass to the anterior abdominal wall (oblique arrows). Axial **(D and E)** and coronal **(F)** contrast subtracted images show circumferential contrast enhancement around the described mass and fluid collection (white arrows) and recanalization of the paraumbilical vein (green arrows).

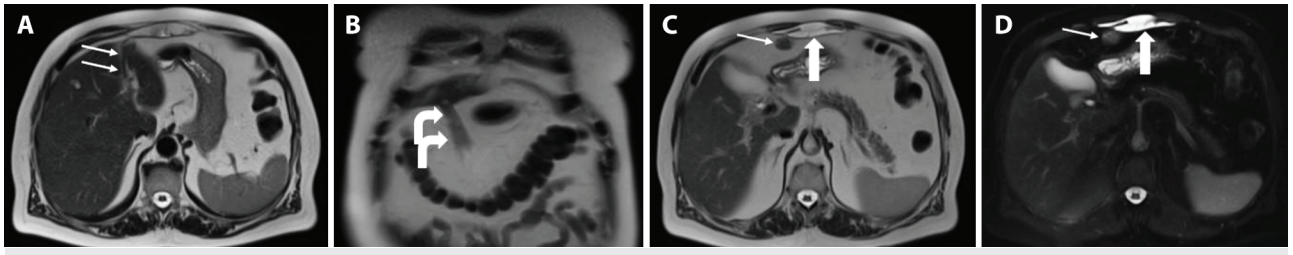

Figure 4 In non-contrast abdominal MRI performed 3 months after the start of treatment **A:** Axial T2WI images show normalisation of the thickening of the falciform ligament (white arrows). **B:** Coronal T2WI image shows a marked reduction in the thickness of the oedematous mass extending to the anterior abdominal wall (oblique arrows). Axial T2WI **(C)** and fat-suppressed axial T2WI **(D)** images show a marked reduction in the thickness of the mass in the omental fatty tissue (white thin arrow) and a marked reduction in the fluid loculation in the neighbourhood of the anterior abdominal wall (thick white arrow).

At the 3-month follow-up visit and laboratory tests were unremarkable. Non-contrast MRI of the upper abdomen showed that the thickening of the round ligament had normalised, the thickness of the inflamed omental fatty tissue extending to the anterior abdominal wall was significantly reduced and its diameter was 15 mm at the widest point (Figure 4).

DISCUSSION

The peritoneal segments covering the diaphragmatic surfaces of the liver lobes come together on the right side in the sagittal plane to form the hepatic falciform ligament and extend to the anterior wall of the abdomen and the lower surface of the diaphragm. When descending downwards, the ligamentum teres hepatis, which is the residue of the umbilical vein, and the vena paraumblicalis course between the two leaves of this ligament (3).

Diseases originating from the falciform ligament itself are very rare. It is usually seen clinically due to inflammation of the adjacent organs and tissues. Although the etiology of FLA formation is not known exactly, two mechanisms are prominent. The first one is FLA caused by circulatory disturbance due to torsion, infarction or direct compression, and the second one is FLA caused by the spread of inflammation in the surrounding tissues and organs to the liver and falciform ligament. In cases that develop secondary to circulatory disorder and lead to necrosis, surgical treatment may be required, but most of the time treatment is performed by non-surgical methods (4). In our case, since FLA developed after acute cholecystitis and acute pancreatitis, we think that the second mechanism was effective and the inflammation in the surrounding organs spread to this ligament.

All patients with FLA complain of abdominal pain. This pain is usually in the epigastric region. As expected, inflammatory markers may be elevated. Diagnosis can be made with abdominal USG and abdominal CT. However, it may not always be detected radiologically. Nonsteroidal anti-inflammatory drugs for pain and antibiotics may be used when clinically needed. However, there is no specific antibiotherapy for FLA. Surgical excision can be performed when necessary. Infarction and necrosis findings are observed histologically in the excised tissues (5). It is likely that the development of infarction and necrosis is due to impaired blood supply. The most important difference in our case from the cases in the literature was the recanalization of the umbilical vein. To the best of our knowledge, reopening of the umbilical vein in cases with FLA and re-closure after treatment has been observed in our case for the first time. We think that this is due to inflammation around the ligemantun teres hepatis. Because inflammatory response is known to cause pathological angiogenesis by various mechanisms.

Since FLA developed secondary to acute pancreatitis and acute cholecystitis in our case, clinical recovery was achieved with bed rest, analgesia, antibiotherapy and appropriate hydration during the period when oral nutrition was off. The patient's symptoms began to resolve six days after admission (the second day after FLA was considered), and the pain completely resolved on the tenth day. Laboratory findings also improved with the regression of clinical findings. On physical examination, although the mass finding in the epigastric region did not disappear, the abdomen was comfortable with palpation (6).

Although the diagnosis of FLA is difficult to be made clinically alone, radiological imaging is very helpful in the diagnosis. Conditions that may cause abdominal pain including acute gastritis, gastric ulcer, cholecystitis and pancreatitis are included in the differential diagnosis (7). As in our case, existing clinical conditions leading to abdominal pain may lead to confusion and delay the diagnosis of FLA.

Patients who present with abdominal pain, for whom another diagnosis is considered on initial examination but clinical improvement is not achieved in the expected time with appropriate treatment or who have recurrent abdominal pain after improvement or who develop an acute abdomen-like symptoms should be evaluated for FLA. Clinical and radiological awareness is important for differential diagnosis.

Conflict of Interest: *The authors declare that they have no conflicts of interest regarding the publication of this article.*

Data Availability Statement: *All data generated or analyzed during this study are included in this published article.*

Fundings: *This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.*

REFERENCES

1. Paulsen F, Waschke J. Sobotta: İnsan Anatomisi Atlası - İç Organlar. 23. Baskı. Münih: Urban & Fischer; 2011.
2. Tanrıkulu Y, Okuş A, Şen Tanrıkulu C, Yılmaz G. Akut Abdomenin Nadir Bir Nedeni: Falsiform Ligament Nekrozu. KTO Karatay Üniversitesi Sağlık Bilimleri Dergisi. 2020;1(1):1-3.
3. Arıncı K, Elhan A. Anatomi. 1. Cilt. Ankara: Güneş Kitabevi; 2020. s. 265-75.
4. Tison C, Regenet N, Frampas E, Masliah C, Le Borgne J. Gangrène isolée du ligament rond du foie [Gangrene of the ligamentum teres hepatis]. Gastroenterol Clin Biol. 2005;29(2):204-5. French. doi: 10.1016/s0399-8320(05)80739-0.
5. Hamal D, Fernandes A, Sagma J, et al. Falciform Ligament Infarction: A Case Report and Review of the Literature. Cureus. 2023;15(11):e48361. doi:10.7759/cureus.48361.
6. Jeong JH, Ojha U, Lee YM. Pathological angiogenesis and inflammation in tissues. Arch Pharm Res. 2021;44(1):1-15. doi:10.1007/s12272-020-01287-2.
7. Miura D, Shindo M, Fukuda Y. Hepatic falciform ligament appendagitis evaluated by ultrasound: A report of 2 cases. Radiol Case Rep. 2022;17(11):4425-30. doi:10.1016/j.radcr.2022.08.090.