Falciform Ligament Necrosis and Respiratory Distress due to Acute Pancreatitis

Asiye Perek[1], Nurdan Altan[2], Yağmur Özge Turaç Kösem[1], Onur Tutar[3], Vedat Durgun[1]

[1]Department of General Surgery, İstanbul University-Cerrahpaşa, Cerrahpaşa Faculty of Medicine, İstanbul, Turkey
[2]Department of General Surgery, Sarıkamış State Hospital, Kars, Turkey
[3]Department of Radiology, İstanbul University-Cerrahpaşa, Cerrahpaşa Faculty of Medicine, İstanbul, Turkey

Cite this article as: *Perek A, Altan N, Turaç Kösem YÖ, Tutar O, Durgun V. Falciform Ligament Necrosis and Respiratory Distress due to Acute Pancreatitis. Cerrahpaşa Med J 2021; 45(1): 53-56.*

Abstract

Ligamentum falciforme separates liver from the left lateral and medial segments along the umbilical fissure and suspends the liver on the anterior wall of the abdomen. The ligamentum teres hepatis is a remnant of the obliterated umbilical vein, which lies on the free edge of the falciform ligament. Moreover, it contains paraumbilical venules, muscle fibers, various fatty tissue amounts, and two mesothelial layers. Pathologies of the falciform ligament are considerably rare. The ligament is usually secondarily affected by surrounding inflammatory diseases. Because of its rarity, necrosis, or abscess of the falciform ligament is usually misdiagnosed as an abdominal wall abscess, and inappropriate treatment leads to the lesion's persistence and complications. Surgical resection is the therapy of choice. The main purpose is to remind that respiratory problems can be the primary complaint of the patient who applies to the emergency room with falciform necrosis secondary to acute pancreatitis.

Keywords: Acute pancreatitis, falciform ligament, falciform ligament necrosis, ligamentum falciforme, pleural effusion

Akut Pankreatite Bağlı Falsiform Ligament Nekrozu ve Solunum Sıkıntısı

Öz

Ligamentum falciforme, karaciğeri umblikal fissür boyunca sol lateral ve medial segmentlere ayırır ve karaciğeri karnın ön duvarında askıya alır. Ligamentum teres hepatis, falciform ligamentin serbest kenarında yer alan oblitere umbilikal ven kalıntısıdır. Ayrıca paraumbilikal venüller, kas lifleri, çeşitli miktarda yağlı dokuları ve iki yaprak mezotel tabakası içerir. Falciform ligamentin patolojileri oldukça nadirdir. Ligament genellikle çevredeki iltihaplı hastalıklara ikincil olarak etkilenir. Nadir görülmesi nedeniyle, falciform ligament apsesi ya da nekrozu genellikle yanlışlıkla karın duvarı apsesi olarak tanı alır ve uygun olmayan tedavi lezyonun kalıcı ve komplike hale gelmesine neden olur. Tedavide tercih edilen cerrahi rezeksiyondur. Bu olgu sunumunun ana amacı; akut pankreatite ikincil olarak falsiform ligament nekrozu ile acil servise başvuran hastanın başlıca şikayetinin solunum problemleri olabileceğini göstermektir.

Anahtar kelimeler: Akut pankreatit, falsiform ligament, falsiform ligament nekrozu, ligamentum falsiforme, plevral efüzyon

Ligamentum falciforme separates the liver from the left lateral and medial segments along the umbilical fissure and suspends the liver on the anterior wall of the abdomen. The ligamentum teres hepatis is a remnant of the obliterated left umbilical vein, which lies on the free edge of the falciform ligament. Additionally, it contains paraumbilical venules, muscle fibers, various fatty tissue amounts, and two mesothelial layers. Pathologies of the falciform ligament are quite rare. The ligament is generally secondarily affected by surrounding inflammatory diseases.

Because of its rarity, necrosis, or abscess of the falciform ligament is usually misdiagnosed as an abdominal wall abscess, and inappropriate treatment leads to persistence of the lesion and its complications. Furthermore, the frequency of accompanying respiratory complications is not acknowledged because they were not mentioned in other previous publications.

Surgical resection is the therapy of choice. The main purpose of this case is to emphasize that the respiratory problems can be the major complaint of the patient who applies to the emergency room with falciform necrosis secondary to acute pancreatitis.

Received/Geliş Tarihi: 08.07.2020 **Accepted/Kabul Tarihi:** 26.10.2020
Available Online Date/ Çevrimiçi Yayın Tarihi: 22.02.2021
Address for Correspondence/Yazışma Adresi: Asiye Perek, Department of General Surgery, İstanbul University-Cerrahpaşa, Cerrahpaşa Faculty of Medicine, İstanbul, Turkey
E-mail/E-posta: aperek@istanbul.edu.tr
DOI: 10.5152/cjm.2021.20024

Case Presentation

A 70-year-old woman referred to a peripheral hospital with nausea, vomiting, and abdominal pain. She had medications for hypertension, rheumatoid arthritis, and hypothyroidism. Her initial computed tomography (CT)

scan revealed choledocholithiasis, intra-abdominal and pelvic fluid, cholelithiasis, and intact pancreas. The amylase value was above 5,000 U/L. The patient had no identified lung disease or a history of smoking; she was transferred to our clinic because of the progressed respiratory problems. After informing the patient about possible operation varieties, follow-up in the intensive care unit and interventional procedures; written consent was obtained from the patient and patient's first degree relative.

On physical examination, abdominal tenderness and distension were present, bowel sounds were diminished, and respiratory rate was increased. The laboratory tests exhibited leukocytosis: 16.700, amylase: 1907 U/L, total bilirubin: 1.74 mL^{-1}, AST (aspartate aminotransferase): 548, ALT (alanine aminotransferase): 267, and CRP (C-reactive protein): 155. Arterial blood gas values were pH: 7.33, sO_2: 89.5%, pO_2: 54.3 mmHg, pCO_2: 30.4 mmHg, lactate: 0.9 mmol/L, and HCO_3: 15.6 mmol/L. On the chest X-ray, there was bilateral fluid collection; the plain abdominal radiography showed no abnormality. Medical therapy for pancreatitis administered. Because of opacity on chest X-ray, she had a thoracic CT scan. Bilateral pleural effusion was observed; there was no sign of pneumonia or pneumothorax.

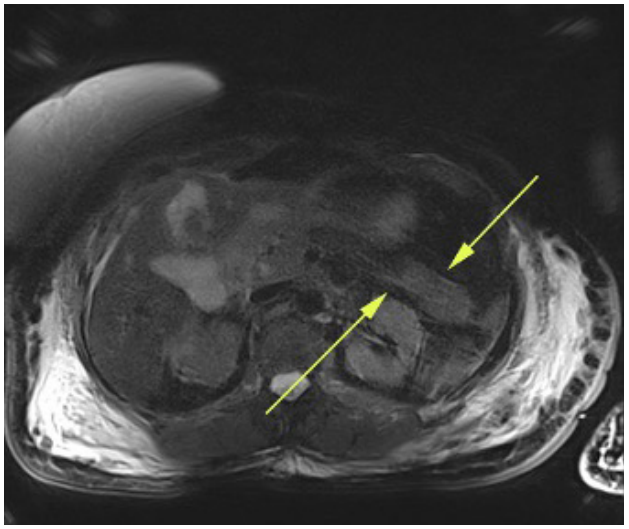

Figure 1. Pancreatic integrity was preserved, and thin linear T2 hyperintense effusions were noticed in the peripancreatic area (arrow)

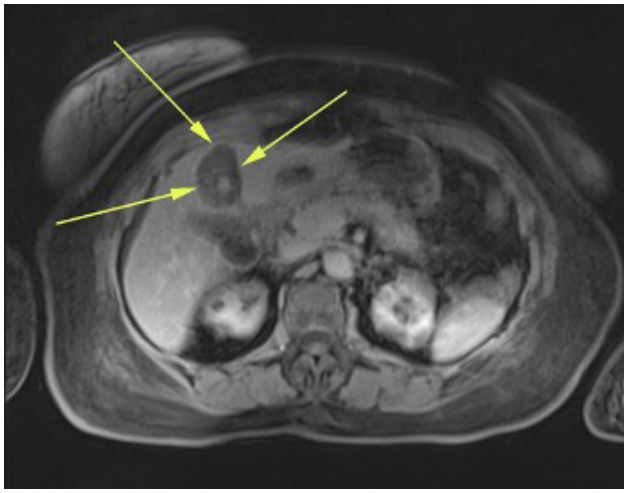

Figure 2. Falciform ligament was wider than normal and edematous (arrows)

The collections in both pleural cavities were drained by inserting a 10-FR catheter with ultrasound guidance; the fluid was transferred for microbiological and laboratory evaluation. The patient's total serum protein was 6 g/gL, and concurrently, the pleural fluid protein was 2 g/dL. Moreover, serum lactate dehydrogenase (LDH) level was 290 IU/L, and pleural fluid LDH level was 124 IU/L. According to Light's criteria, none of them have met the criteria; therefore, we inferred that it has been likely transudative effusion [1]. No pathogen was cultured, and the amylase level of the fluid was 41 U/L.

A magnetic resonance cholangiopancreatography (MRCP) was performed and failed to evaluate the common bile duct and extrahepatic bile ducts. Nonetheless, it confirmed that the pancreas assessment was normal, and fine linear T2 hyperintense effusions were remarked in the peripancreatic area (Figure 1). The falciform ligaments were recognized to be enlarged and edematous (Figure 2). Effusions were extending directly to the pelvis in both paracolic areas (Figure 3).

Endoscopic ultrasound was performed; bilateral pleural effusions were furthermore seen. The abdominal aorta and main vessels were within the normal range. The pancreatic parenchyma was slightly heterogeneous, and there was no dilatation in the duct of Wirsung; its diameter was 3.5 mm at the head of the pancreas. The diameter of the common bile duct (6.5

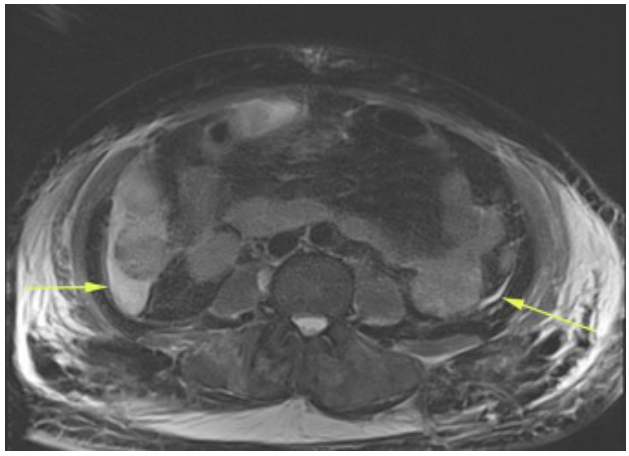

Figure 3. There are effusions in both paracolic areas extending toward the pelvis

mm) and intrahepatic bile duct were normal. The final diagnosis was acute pancreatitis and bilateral pleural effusions.

Her respiratory problems were exacerbated, and she was followed in the intensive care unit (ICU) for 6 days. In the ICU, the patient was given noninvasive ventilation support on account of respiratory problems, and approximately 1,500 cc of fluid was drained daily from the drainage catheters. Besides, with the infectious disease specialists' recommendation, antibiotherapy was applied as ceftriaxone 2 g/day and meropenem 3 g/day. The amylase level regressed to 66. After alleviating her condition, the operation was scheduled.

Surgical Procedure

The patient was prepared in a supine position under general anesthesia. Open laparotomy with right subcostal incision was performed. The falciform ligament was identified at the operation site, and it was 12´10´8 cm, edematous, with necrotic areas. There was no free fluid in the abdominal cavity. The falciform ligament was resected, and the cross sections exhibited abscess and necrosis in it. Although the gallbladder wall had normal looking without any sign of acute inflammation, cholecystectomy was completed in the same session. There was no stone in the common bile duct. The pancreas was intact to touch, and there was no necrosis on the hepatotactic and the gastrocolic ligaments and bursa omentalis. The abdomen was drained by a subhepatic drain.

Postoperatively, the patient transferred again to the ICU, and intravenous antibiotherapy was maintained, oral intake was begun, and the abdominal drain was removed on day 2 at the ICU. Remarkable improvement in respiratory conditions was perceived when the patient returned to the general surgery ward on day 5. The patient's catheter thoracotomies were removed on day 9, and she was discharged after removing sutures on day 11. The patient has been followed up in the outpatient department with an uneventful postdischarge period.

Histopathologies of the specimens were consistent with chronic cholecystitis and infarct, which holds all layers in the gallblader's focal area. Excision material, which was registered as falciform ligament, was explained such as enzymatic fat necrosis areas, fibrosis, necrosis, vascular proliferation, organization findings, and suppurative inflammation abscess formation of the xanthomatous reaction. Thus, the mechanism of abscess formation, in this case, remains unclear.

The review of the literature showed that all of the cases underwent surgical treatment. The patient did not develop any complaints during the 3-year follow-up.

Discussion

The falciform ligament is a remnant of the primitive ventral mesentery. It runs along the abdominal wall and extends to the anterior surface of the liver and the diaphragm. It attaches the liver to the diaphragm and to the abdominal wall. Its free edge has the round ligament of the liver, which is the remnant of the embryogenic left umbilical vein. The falciform ligament layers separate over the liver and form the anterior and the posterior layers of the coronary ligament. It includes some fatty tissue and represents a potential space.

The falciform ligament abscess, necrosis, or infections are very rare. The pathophysiology of infection is poorly understood, and primary infection is extremely rare.

The infection may be secondary to biliary tract infections and pancreatitis by bacterial contamination through the lymphatic and hematogenous pathways or aberrant biliary ducts [2].

In addition, septic sources in the liver, diaphragm, retroperitoneum, abdominal, and thoracic wall may reach the falciform ligament through hematogenous or lymphatic channels.

Its arterial supply originates from the right hepatic artery, and venous drainage goes to the paraumbilical vein and the portal system. The lymphatic drainage is to the retroperitoneum and precardiac, superior phrenic, periesophageal, and celiac nodes.

It most reasonably will be found as a coincidental finding in radiological tests done for patients with abdominal pain, biliary, or pancreatic disease symptoms. Pathologic lesions of the falciform ligament were first described in 1909 [2], and so far, 10 cases of falciform ligament abscess have been reported.

Falciform ligament necrosis may also develop because of the occlusion of the arterial supply of draining veins by instrumentation during laparoscopic surgery, trauma, or torsion of the preexisting cyst [3].

Falciform ligament necrosis following cholangitis from an obstructive ampullary carcinoma was reported [4].

The clinical picture usually points toward biliary pancreatic disease, and the falciform ligament lesion is a coincidental finding.

In ultrasonography, a hyperechoic, oval mass along the falciform ligament might be shown. Diagnosis is performed with CT or magnetic resonance screening. The round, cylindrical mass of the fatty density at the side of the falciform ligament can be shown. At the center of the mass, there is a well-defined hyperdense area [5, 6].

Associations and pathophysiological propositions for acute respiratory distress due to acute pancreatitis have been revealed in previous publications [7, 8]. Notwith-

standing, the connection between falciform necrosis and respiratory distress, particularly pleural effusion, which we significantly observed in this case, has never been addressed before.

Our patient's primary complaint was respiratory problems besides mild abdominal tenderness and distension. There was no sign or symptom of severe pancreatitis or biliary disease. The bilirubin level was normal, which excludes biliary obstruction, but the amylase level was very high. The MRCP did not help exclude biliary channel lithiasis. During surgery, no acute process was found in the pancreas and the gallbladder. An edematous, necrotic mass in place of the falciform ligament was found and resected. In our case, because in surgical exploration, the pancreas and the gallbladder appeared normal, the abscess formation mechanism was vague. Nevertheless, whatever is the mechanism, these patients must have surgical treatment because of necrotic tissue, and we suggest that it should be kept in mind that this rare falciform ligament necrosis condition may develop secondary to acute pancreatitis and can have a challenging respiratory complication.

Informed Consent: Written informed consent was obtained from patient and patient's first degree relative who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – A.P.; Design – A.P., N.A., V.D.; Supervision – A.P., V.D.; Resources – A.P., N.A., Y.Ö.T.K.; Materials – Y.Ö.T.K., O.T.; Data Collection and/or Processing – N.A., Y.Ö.T.K.; Analysis and/or Interpretation – A.P., N.A., Y.Ö.T.K., O.T., V.D.; Literature Search – A.P., N.A., Y.Ö.T.K.; Writing Manuscript – A.P., N.A., V.D.; Critical Review – V.D.; Other – O.T., V.D.

Conflict of Interest: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

Hasta Onamı: Yazılı hasta onamı bu çalışmaya katılan hastadan ve birinci derece yakınından alınmıştır.

Hakem Değerlendirmesi: Dış bağımsız.

Yazar Katkıları: Fikir – A.P.; Tasarım – A.P., N.A., V.D.; Denetleme – A.P., V.D.; Kaynaklar – A.P., N.A., Y.Ö.T.K.; Malzemeler – Y.Ö.T.K., O.T.; Veri Toplanması ve/veya İşlemesi – N.A., Y.Ö.T.K.; Analiz ve/veya Yorum – A.P., N.A., Y.Ö.T.K., O.T., V.D.; Literatür Taraması – A.P., N.A., Y.Ö.T.K.; Yazıyı Yazan – A.P., N.A., V.D.; Eleştirel İnceleme – V.D.; Diğer – O.T., V.D.

Çıkar Çatışması: Yazarlar çıkar çatışması bildirmemişlerdir.

Finansal Destek: Yazarlar bu çalışma için finansal destek almadıklarını beyan etmişlerdir.

References

1. Light RW. Pleural effusion. N Engl J Med 2002; 346: 1971-7. [CrossRef]
2. Henderson MS. III. Cyst of the Round Ligament of the Liver. Ann Surg 1909; 50: 550-1. [CrossRef]
3. Lloyd T. Primary torsion of the falciform ligament: computed tomography and ultrasound findings. Australas Radiol 2006; 50: 252-4. [CrossRef]
4. Warren LR, Chandrasegaram MD, Madigan DJ, Dolan PM, Neo EL, Worthley CS. Falciform ligament abscess from left sided portal pyaemia following malignant obstructive cholangitis, World J Surg Oncol 2012; 10: 278. [CrossRef]
5. Singh AK, Gervais DA, Hahn PF, Rhea J, Mueller PR. CT appearance of acute appendagitis. AJR Am J Roentgenol 2004; 183: 1303-7. [CrossRef]
6. Tsukuda K, Furutani S, Nakahara S, Tada A, Watanabe K, Takagi S, et al. Abscess formation of the round ligament of the liver: report of a case. Acta Med Okayama 2008; 62: 411-3
7. Browne GW, Pitchumoni CS. Pathophysiology of pulmonary complications of acute pancreatitis. World J Gastroenterol 2006; 12: 7087-96. [CrossRef]
8. Raghu MG, Wig JD, Kochhar R, Gupta D, Gupta R, Yadav TD, et al. Lung complications in acute pancreatitis. JOP 2007; 8: 177-85.