

Pelvic lipomatosis associated with portal vein thrombosis and hydronephrosis: a case report

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Özkacmaz Sercan 

Abstract

Pelvic lipomatosis is an uncommon disease with no clear etiology and it occurs secondary to deposition of a large amount of fatty tissue in the pelvis. This deposition causes compression to the rectum, bladder, and venous structures. Because of this compression, various symptoms, such as recurrent urinary infections, dysuria, tenesmus, and constipation, have mostly been reported. However, iliac or superior vena cava thrombosis secondary to vascular compression of pelvic lipomatosis is rare. This report describes a case of pelvic lipomatosis, which was associated with right-sided mild hydronephrosis and portal vein thrombosis.

Keywords

Pelvic lipomatosis, computed tomography, portal vein thrombosis, hydronephrosis, bladder compression, fatty tissue, splenomegaly

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Introduction

Pelvic lipomatosis is a rare disease, which leads to various symptoms because of compression of pelvic structures by extensive deposition of mature tissue in the pelvis. Clinical manifestations of pelvic lipomatosis vary depending on the compressed structures of the urinary system, intestinal tract, and venous system.¹

Although effects of pelvic lipomatosis on urinary and systemic venous systems are well known, such possible results on the

liver–portal venous system and potential mechanisms have not been previously discussed. This report presents computed tomographic (CT) findings of a case of pelvic lipomatosis associated with portal

Kırşehir Ahi Evran University, Kırşehir, Turkey

Corresponding author:

Sercan Özkaçmaz, Department of Radiology, Kırşehir Ahi Evran University, Faculty Of Medicine, Bağbaşı Street, 40100, Kırşehir, Turkey.

Email: Sercanozkacmaz@hotmail.com



vein thrombosis and right-sided hydronephrosis due to compression.

Case report

A 73-year-old woman was admitted to the emergency department with the complaint of nausea and vomiting for 4 days. She also reported epigastric pain for several months. Her medical history was unremarkable, except for mild hypertension for 15 years. A physical examination showed an enlarged spleen that extended 4 cm below the costal margin, mild abdominal distension, and tenderness in the right upper quadrant. She was an obese patient with a body mass index of 32 kg/m^2 (160 cm in height and 82 kg in weight). Laboratory examinations showed a mild elevation in transaminase levels (aspartate aminotransferase level: 132 U/L; alanine aminotransferase level: 118 U/L). A transabdominal ultrasound examination showed free intraperitoneal fluid, splenomegaly (length: 162 mm), and a dilated portal vein (2 cm in diameter) and portal vein thrombosis. Contrast-enhanced CT scanning showed portal vein thrombosis (Figures 1–5), splenomegaly (Figure 4), ascites (Figures 1, 4), collaterals consistent with intraperitoneal varices (Figure 4), and right-sided mild hydronephrosis (Figure 5). Additionally, homogenous fatty tissue that surrounded the rectum, and compressed and pushed the bladder–uterus anteriorly was detected (Figures 2–4). All laboratory examinations for determination of the possible cause of portal vein thrombosis, including inherited thrombophilia, malignant conditions, viral hepatitis, pancreatitis, cholangitis, inflammatory bowel disease, and chronic liver disease, showed negative results. The patient denied a history of drugs. CT findings with areas of symmetric fat density in the pelvic region indicated the diagnosis of pelvic lipomatosis, and imaging findings indicated the diagnosis of portal vein

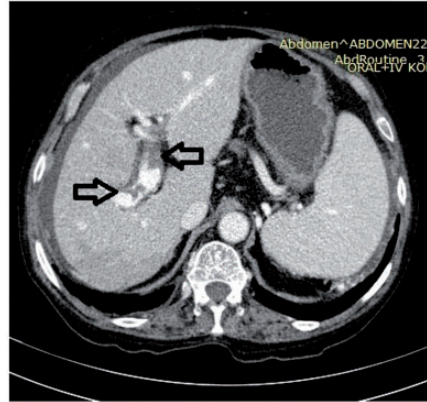


Figure 1. Axial contrast-enhanced computed tomographic image shows partial thrombus formation within the lumen of the right (mild) and left (marked) portal veins (arrows).

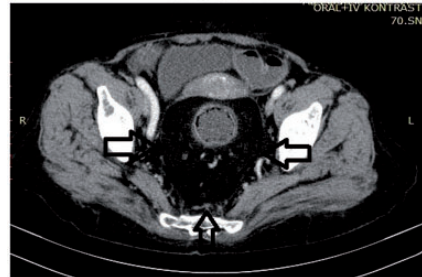


Figure 2. Axial contrast-enhanced computed tomographic image shows a homogenous fatty lesion that surrounds the rectum, and pushes the rectum, uterus, and bladder anteriorly.

thrombosis and pelvic lipomatosis. Therefore, a histopathological examination was not required. Two months after anticoagulant therapy was started, a follow-up ultrasound examination showed mild regression in thrombus size with a portal vein diameter of 18 mm. No marked change was noted for the size of the spleen (length: 164 mm). Six months after the initial diagnosis, the thrombus was completely resolved with a portal vein diameter of 16 mm by ultrasound. Additionally, a marked decrease in spleen size (size:141 mm) was observed.

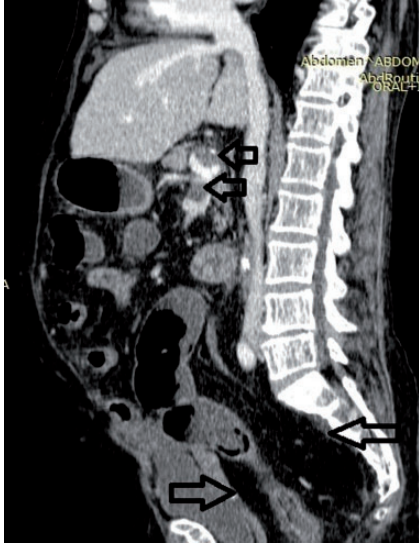


Figure 3. A sagittal reformatted contrast-enhanced computed tomographic image shows a pelvic fatty lesion (long arrows), which surrounds and narrows the rectosigmoid colon, and compresses the uterus and bladder. Additionally, thrombus formation within the lumen of the portal vein can be seen (short arrows).

However, right mild hydronephrosis persisted. During this last visit, the patient's aspartate aminotransferase and alanine aminotransferase levels were 97 U/L and 86 U/L, respectively.

Local Ethics Committee approval was not necessary because this was a case report and specific information about the patient was not included in the report. Written consent was obtained from the patient.

Discussion

Pelvic lipomatosis is a rare disease, which was first described in 1959 by Engles.² This disease occurs secondary to infiltration of a large amount of fatty tissue in the pelvis. The exact incidence of pelvic lipomatosis is unclear, but it is most prevalent in men and patients with a dark-skinned phenotype. Although pelvic lipomatosis is



Figure 4. Coronal reformatted contrast-enhanced computed tomographic image shows a fatty lesion, which surrounds the rectum, and compresses the uterus and bladder (arrows). Additionally, thrombus formation within the portal vein lumen can be seen (arrow head).



Figure 5. Sagittal reformatted contrast-enhanced computed tomography shows portal vein thrombosis (arrow) and right-sided mild hydronephrosis (arrow head).

found most commonly in the third or fourth decade of life, it is rarely reported in children.¹ Obesity, endocrine dysfunction, such as diabetes mellitus and Cushing's disease, lower abdominal or pelvic trauma, prior abdominal surgery, allergic conditions, and venous stasis, have been proposed for the cause of pelvic lipomatosis. However, the exact etiology of this disease remains unclear.³ The clinical presentations of pelvic lipomatosis vary depending on the compressed tissues. Common manifestations are dysuria, pollakiuria, nocturia, urgency if structures of the urinary system are compressed, constipation, tenesmus, and diarrhea if structures of the lower intestinal tract are compressed. Additionally, compression of the venous system leads to lower limb edema and thrombophlebitis.¹ Because areas of symmetric fat density in the pelvic region on CT scans are suggested as diagnostic, surgery or a histopathological examination is often not required. Making a specific diagnosis of pelvic lipomatosis without an invasive procedure is useful.^{4,5} For preventing pelvic lipomatosis, weight loss is suggested, but this is controversial.³

CT urography is also an important imaging modality for showing the typical presentation of a pear-shaped bladder. This feature is suggested to be important for indicating pelvic lipomatosis, as well as hydronephrosis secondary to vesicoureteral obstruction.⁶ In a study by Chen et al.,⁷ 20 of their 45 patients with pelvic lipomatosis had hydronephrosis.

Venous thrombosis and lymphatic obstruction associated with pelvic lipomatosis have previously been described. Van Heurn et al.⁸ reported a case of pelvic lipomatosis, which led to recurrent deep venous thrombosis and subsequent iliac venous obstruction. Scheshter⁹ reported a 36-year-old man with pelvic lipomatosis, which was associated with complete obstruction of the inferior vena cava and

a large thrombus that was completely occluded the left external iliac vein. Yamaguchi et al.¹⁰ described a 29-year-old man with pelvic lipomatosis associated with lower extremity edema secondary to compression to lymph vessels in the inguinal area. Locko and Interrante reported a 30-year-old man with pelvic lipomatosis, which caused vena caval obstruction.¹¹

In the current case, right hydronephrosis was detected, probably because of bladder compression. Thrombus formation was also observed in the lumen of a dilated portal vein, which led to splenomegaly and intra-peritoneal collaterals. This is the second report of pelvic lipomatosis associated with portal vein thrombosis. Similar to the current patient, Bhatia et al.¹² reported a case of portal vein thrombosis with portal hypertension. They also detected pelvic lipomatosis, which compressed the urinary bladder, rectum, and sigmoid colon, and confirmed portal vein thrombosis on CT imaging of the patient. El-Feky¹³ showed CT images of a 65-year-old cirrhotic male patient with pelvic lipomatosis in whom calcified plaques were present at portal and mesenteric veins walls. Because vascular compression is suggested as the cause of venous occlusions, which are observed in the patients with pelvic lipomatosis, portal vein thrombosis may occur secondary to compression and inflammation of distal veins, such as the superior rectal vein. The inferior mesenteric vein begins in the rectum as the superior rectal vein, and forms the portal vein with the superior mesenteric vein and splenic vein. Another hypothesis is that lipomatosis can extend to the retroperitoneum, causing portal vein thrombosis. Such possible associations with pelvic lipomatosis and portal vein thrombosis need to be further investigated. Additionally, effects of pelvic lipomatosis on liver parenchyma should be examined.

Conclusion

We suggest that hepatic parenchyma and the portal-mesenteric vein must be carefully examined in radiological examinations of patients with pelvic lipomatosis. Additionally, pelvic lipomatosis should be kept in mind in patients with portal vein thrombosis with an unknown etiology.

Declaration of conflicting interest

The author declare that there is no conflict of interest.

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ORCID iD

Özkacmaz Sercan  <http://orcid.org/0000-0002-9245-0206>

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