Case Report

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Inferior Vena Cava Stenosis Following Orthotopic Liver Transplantation: Differentiating Points from False Positives

뇌사자 간이식 후 발생한 하대정맥 협착: 위양성과의 감별점

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Orthotopic liver transplantation has become the treatment of choice for patients with end-stage liver disease. Various early or delayed vascular complications, including arterial pseudoaneurysm, thrombosis, or stenosis, and venous stenosis or occlusion, may lead to graft failure. Early detection and prompt management of such complications are essential to achieve successful transplantation and prevent the need for retransplantation. This report presents differentiating points, using computed tomography and digital subtraction angiography findings and measurement of pressure gradient across the stenotic lesion, that require immediate intervention in patients with inferior vena cava stenosis after orthotopic liver transplantation.

Index terms Angiography, Digital Subtraction; Endovascular Procedures; Inferior Vena Cava; Liver Transplantation; Postoperative Complications

INTRODUCTION

Liver transplantation (LT) is the treatment of choice for patients with acute liver failure or

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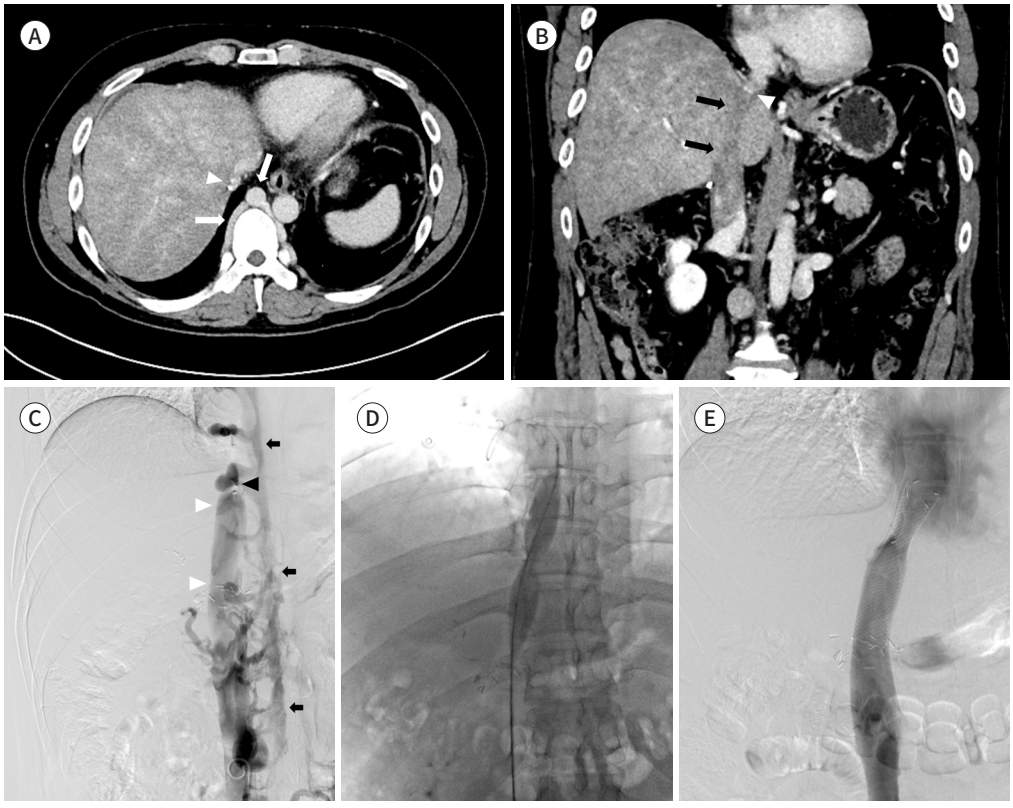
chronic liver disease. Several early or delayed postoperative complications have been reported, and early detection and management of such complications have increased graft survival rates (1-3). Vascular complications after LT are estimated to occur in 9% of patients. They include pseudoaneurysm of the hepatic artery and stenosis/thrombosis of the hepatic artery, portal vein, hepatic veins, and inferior vena cava (IVC) (3, 4). We report two cases of IVC stenosis following orthotopic LT detected on follow-up CT.

CASE REPORT

CASE 1

A 33-year-old male was referred for interventional radiology with suprahepatic IVC stenosis detected on follow-up CT. He underwent deceased donor LT 47 months prior due to hepatitis B virus infection and liver cirrhosis (LC). His laboratory findings were as follows: aspar-

Fig. 1. A 33-year-old male with suprahepatic IVC stenosis after orthotopic liver transplantation.
CT (A, B) and DSA (C-E) images were taken 3 years and 11 months after transplanation.
A. Severe stenosis of the IVC below the surgical clips (arrowhead), mosaic enhancement of the liver parenchyma, and engorgement of the paravertebral plexus and azygos vein (arrows) are seen.
B. On a coronal image, non-opacification contrast media in the IVC (arrows) below the clips (arrowhead) is observed.
C. IVC flow (white arrowheads) is obstructed below the clips (black arrowhead), and multifocal collateral flows drain into the RA (arrows).
D, E. After balloon angioplasty, a bare-metal stent (20 mm/80 mm) is inserted, following which the pressure gradient between the RA and IVC is 4 mm Hg, and collateral veins disappear.
DSA = digital subtraction angiography, IVC = inferior vena cava, RA = right atrium



tate transaminase (AST) and alanine aminotransferase (ALT) levels were not increased (29 IU/L and 17 IU/L, respectively), but the total bilirubin (TB) level was elevated (2.5 mg/dL). Contrast-enhanced CT revealed reticular enhancement of the liver parenchyma, suggesting hepatic congestion and non-opacification of the hepatic veins and intrahepatic IVC with bilateral lumbar vein engorgement (Fig. 1A, B). Stenosis of the anastomotic site between the suprahepatic and intrahepatic IVC was suspected.

Inferior vena cavography was performed using a 5-Fr pigtail catheter (Performa angiographic catheter; Merit Medical, South Jordan, UT, USA) via the right common femoral vein approach. A total of 20 mL of the contrast medium (Pamiray 300; Dongkuk Pharm., Seoul, Korea) was injected at a rate of 10 mL per second. The suprahepatic IVC near the surgical clips was stenosed, and reflux to the dilated right hepatic vein was observed. Multiple collateral veins, including the lumbar veins, drained into the right heart via the azygos vein (Fig. 1C). The pressure gradient between the right atrium (RA) and infrahepatic IVC was 17 mm Hg, which decreased to 12 mm Hg after angioplasty with an 18-mm/60-mm balloon (XXL balloon dilatation catheter, Boston Scientific, Natick, MA, USA) (Fig. 1D). A bare-metal stent (20 mm/80 mm; Niti-S, Taewoong Medical, Gimpo, Korea) was deployed across the stenotic lesion. The pressure gradient decreased to 4 mm Hg, and the collateral veins were not visible after stent placement (Fig. 1E). Six days after the procedure, the liver function test results were within the normal range (AST, 34 IU/L; ALT, 20 IU/L; and TB, 1.2 mg/dL). However, restenosis of the anastomosis site was detected on a 7-month follow-up CT, and another stent (20 mm/60 mm; Niti-S, Taewoong Medical) was placed.

CASE 2

A 42-year-old female was transferred for the evaluation of IVC stenosis. She underwent orthotopic LT for alcoholic LC 22 months prior. Sixteen months after transplantation, ascites occurred, and the patient was diagnosed with LC again following liver biopsy. CT demonstrated severe stenosis below the anastomosis of the infrahepatic IVC and deviation to the right side as the volume of the right hepatic lobe decreased (Fig. 2A-C). The patient's AST level was slightly elevated (51 IU/L), and the albumin level was low (2.8 g/dL).

An inferior vena cavography was performed using a 5-Fr pigtail catheter (Performa Angiographic Catheter; Merit Medical). In contrast to the CT findings, IVC stenosis was not observed, and venous flow in the IVC was well preserved (Fig. 2D). The pressure gradient between the RA and infrahepatic IVC was 2 mm Hg. IVC stenting was not performed, and a drainage catheter was inserted for massive ascites. On an 8-month follow-up CT, ascites was disappeared, and the IVC lumen was well maintained.

The Institutional Review Board at our institution approved this study (IRB No. 05-2021-061), and the requirement for informed consent was waived.

DISCUSSION

IVC stenosis or thrombosis occurs in less than 1% of transplantations (2, 4). It usually occurs at the site of surgical anastomosis. Acute IVC stenosis is caused by technical factors during surgery, such as a discrepancy in the size between the donor and recipient IVCs, or organ rota-

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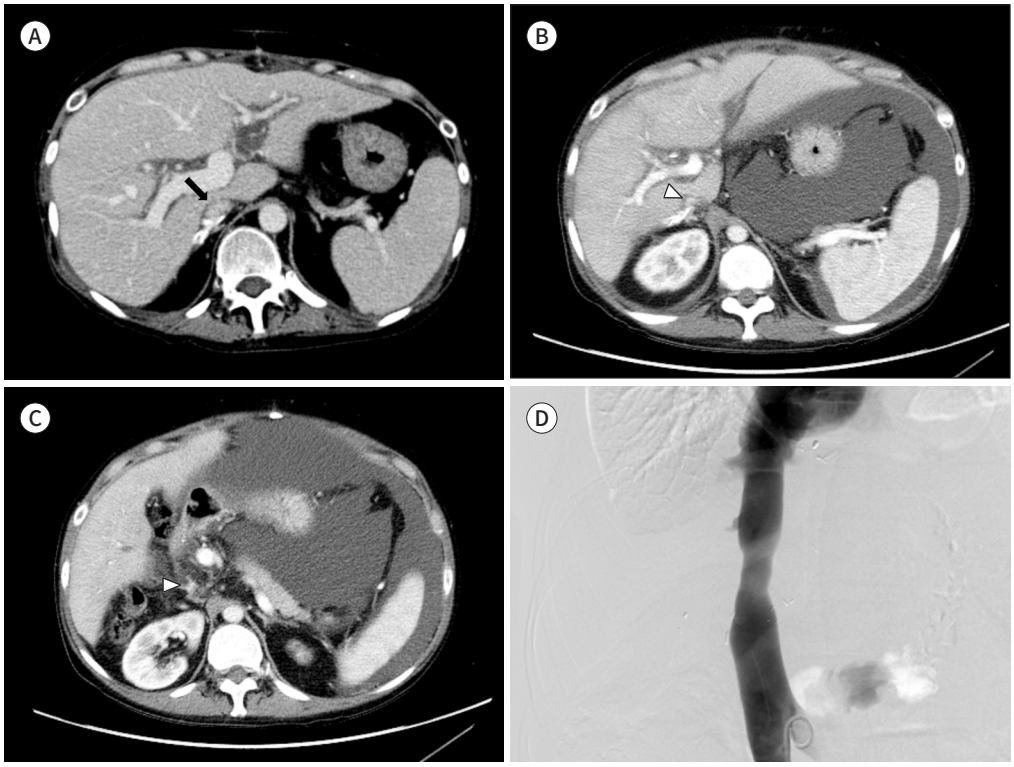
Fig. 2. A 42-year-old female with IVC pseudostenosis after orthotopic liver transplantation.
A. No abnormality is seen on axial CT 3 months after transplantation, and the IVC (arrow) is located in front of the vertebral body.
B, C. Relatively homogeneous enhancement of the liver parenchyma, absence of collateral flow, recurrent liver cirrhosis with massive ascites, and severe stenosis of the IVC below the anastomotic site of the infrahepatic IVC (arrowheads) are detected on axial CT 22 months after transplantation. The collapsed IVC appears deviated to the right relative to a CT image obtained at the 3-month follow-up (A).
D. The IVC lumen is well maintained without stenosis on conventional venography of DSA.
DSA = digital subtraction angiography, IVC = inferior vena cava



tion, which results in kinking of the supra- or infrahepatic IVC. Chronic thrombus, fibrosis around the anastomosis, neointimal hyperplasia, or extrinsic compression of the anastomotic site from a hypertrophied graft are thought to be causes of chronic IVC obstruction (2, 4-6). The clinical signs and symptoms include abdominal pain, pleural effusions, hepatic dysfunction, ascites, and lower extremity edema (2, 7).

Contrast-enhanced CT imaging is commonly used to confirm IVC stenosis. Stricture or thrombosis in the IVC, a mosaic pattern of hepatic enhancement, engorged pericaval and periaortic collateral formation including the thoracolumbar plexus and azygos system can be detected in patients with IVC stenosis (2), which coincides well with the CT findings in Case 1.

At Doppler US (DUS), reduction in the diameter of the IVC and prestenotic dilatation of the hepatic veins with impaired vascular flow are indirect findings of IVC stenosis. A sustained monophasic waveform is also a sensitive finding (6). However, depending on the location of the stenosis (infrahepatic, suprahepatic, or intrahepatic), DUS findings of the hepatic veins might be normal or abnormal. In addition, it is difficult to evaluate the IVC with DUS when the patient has a large volumes of ascites like case 2.

IVC pseudostenosis detected on CT images, which may worsen or disappear depending on

the posture of the patient, is caused by growth or twisting of the graft and compression by ascites (8). In our second case, even though severe stenosis of the infrahepatic IVC was found on CT, there was no evidence of flow obstruction in the IVC on inferior vena cavography. The collapsed lumen of the IVC was recovered by a large volume of contrast (10 mL/s, total 20 mL) injected into a pigtail catheter. Furthermore, another finding suggesting IVC stenosis, such as reticular hepatic enhancement or collateral formation, was not observed on CT. Compression by massive ascites and lateral deviation of the anastomotic site due to volume loss of the right hepatic lobe led to pseudostenosis of the IVC. Pressure gradient measurements following conventional venography can be helpful in distinguishing pseudostenosis from pathologically significant lesions. A pressure gradient > 5 mm Hg across the stenosis is considered significant (2).

Endovascular approaches are preferred for the treatment of IVC stenosis after LT. However, the role of balloon angioplasty alone is limited because it is ineffective in cases of stenosis caused by graft kinking. The possibility of anastomotic rupture and restenosis due to elastic recoil of the fibrotic tissue in chronic stenosis are also problems (9, 10). Several studies have indicated that stent placement for treating IVC stenosis is effective and has long-term patency. Donaldson et al. (9) reported that 96% of IVC stents have long-term patency, and symptoms were relieved in 85% of patients. In a study by Lee et al. (10), stent patency was maintained for > 5 years after IVC stenting, and clinical success was achieved in 86% of patients.

In conclusion, we experienced two cases of IVC stenosis suspected on follow-up CT after orthotopic LT. Collateral vascular structures in the paravertebral venous plexus or reticulated mosaic pattern of liver parenchyma on CT, and IVC venography with measurement of the pressure gradient across the stenotic lesion can help distinguish IVC stenosis from pseudostenosis.

Author Contributions

Conceptualization, all authors; data curation, K.J.H., K.T.U., R.H.; investigation, C.Y., J.J.Y.; project administration, C.Y., K.J.H., J.U.B.; resources: K.J.H.; supervision, K.J.H.; visualization, C.Y., K.J.H.; writing—original draft, C.Y., K.J.H.; and writing—review & editing, C.Y., K.J.H., J.U.B.

Conflicts of Interest

Ung Bae Jeon has been a Section Editor of the Journal of the Korean Society of Radiology since 2021; however, he was not involved in the peer reviewer selection, evaluation, or decision process of this article. Otherwise, no other potential conflicts of interest relevant to this article were reported.

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뇌사자 간이식 후 발생한 하대정맥 협착: 위양성과의 감별점

최연수[1] · 김진혁[1*] · 전웅배[1,2] · 장주연[1] · 김태언[1,2] · 류화성[1]

뇌사자 간이식은 말기 간질환 환자에서 최우선치료로 시행되고 있다. 간이식 후 가성동맥류 형성, 동맥 혈전증 및 협착, 정맥의 협착 및 폐색 등 다양한 초기 또는 후기 혈관 합병증이 발생할 수 있으며, 이는 이식 실패를 야기할 수 있다. 이러한 합병증을 빨리 발견하고 적절한 치료를 하는 것이 이식 성공을 달성하고 재이식을 방지하기 위해 중요하다. 이 증례 보고에서는 컴퓨터단층촬영 및 디지털감산 혈관조영술 영상 소견과 협착 부위 전후의 압력차 측정을 통해 뇌사자 간이식 후 발생한 하대정맥 협착 환자에서 즉각적인 인터벤션이 필요한 감별점을 제시하고자 한다.

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