eISSN 1308-4038

International Journal of Anatomical Variations (2014) 7: 77–79

Case Report

Report of two cases of double inferior vena cava with an emphasis on embryogenesis and clinical relevance

Published online June 14th, 2014 © http://www.ijav.org

Marjeta TANKA ✦

MEDICARE Imaging Diagnostic Center, Tirana, ALBANIA.

✦ Marjeta Tanka, MD
MEDICARE Imaging Diagnostic Center
Rruga e Barrikadave Nr 69
Tirana, ALBANIA.
☎ +355 672066592
✉ marjeta.tanka@yahoo.com

Received June 11th, 2013; accepted February 3rd, 2014

Abstract

Double inferior vena cava is a congenital variation caused by an unusual embryological development of the inferior vena cava. Most malformations of inferior vena cava are accidental findings in abdomen imaging. We report two cases with double inferior vena cava diagnosed during routine computed tomography in two patients referred to our clinic for abdominal pain. In both cases the double inferior vena cava was considered to be a persistence of both the right and left supracardinal veins. Although this variation is not common it becomes significant when planning surgical and radiological vascular procedures.

Key words [double inferior vena cava] [inferior vena cava] [venous variation]

Introduction

Double inferior vena cava (IVC) is a congenital variation caused by an unusual embryological development of the inferior vena cava [1, 2] with a prevalence of 0.2-3% [3]. Knowledge of the embryogenesis of the IVC is helpful for understanding its variation. The inferior vena cava originates between the 6th and 8th weeks from a dynamic process of development, regression, anastomosis, and replacement of the vitelline vein, subcardinal and supracardinal veins. When the left supracardinal vein fails to regress, it results in a double IVC. Although this variation is not common, it has clinical importance. We report two cases of double IVC with review of the embryogenesis of the IVC and the clinical relevance of this anatomical variation.

Case Report

The first case was a 22-year-old woman who referred to emergency clinic with recurrent lower abdominal pain. Physical examination and laboratory tests provided no significant findings. Abdominal contrast enhanced computed tomography depicted double IVC (Figure 1). In addition to the usual right-sided IVC, another IVC was seen on the left side of the abdominal aorta with smaller size compared the right IVC (Figure 1). The left IVC draining the left renal vein, crossed the aorta anteriorly, and joined the right inferior vena cava (Figure 2). Both IVC were formed from the respective common iliac veins. No other vascular variations were noted.

The second case was a 59-year-old man with acute abdominal pain. We proceeded with CT imaging of the abdomen which revealed portal vein and superior mesenteric vein thrombosis associated with ischemic change in the ileocecal region. We incidentally observed a double IVC (Figure 3), on both sides of the aorta. As in the first case, he had a right-sided IVC and a vascular structure on the left side of the aorta draining the left renal vein which resulted to be the duplicated IVC (Figure 4).

Discussion

Different variations of the IVC have been described [3]. Study of embryology of inferior vena cava allows for explaining its multiple anatomical variants .The inferior vena cava originates between the 6th and 8th weeks from a dynamic process of development, regression, anastomosis, and replacement of the vitelline vein, subcardinal and supracardinal veins. The posterior cardinal vein appears first and disappears except for the distal segment that becomes the iliac bifurcation.

During the fifth week the subcardinal veins, which drain the mesonephrons, are formed. The anastomosis between the subcardinal veins forms the left renal vein. The left subcardinal vein disappears and the right subcardinal vein becomes the renal segment of the IVC. One week later, during the sixth week the supracardinal veins are formed. While the left supracardinal vein regress, the right supracardinal vein becomes the infrarenal portion of inferior vena cava. The right vitelline vein forms the hepatic segment of the IVC. It connects to the renal segment and the infrarenal segment and the IVC is complete. When the left supracardinal vein fails to regress, a duplicate IVC results. Radiologically the characteristic CT appearance of double IVC is a normal inferior vena cava along the right side of the spine and a left-sided IVC ascending to the level of the renal veins to join the right-sided IVC through a vascular structure that may pass either anteriorly or posteriorly to the aorta at the level of the renal veins. In our two cases the IVCs were situated bilaterally to abdominal aorta. The left IVC ascended to the level of the renal veins, crossed the aorta anteriorly, joined the right IVC and then ran upwards as a single vein in its normal anatomical position. In both cases the double IVC is considered to be a persistence

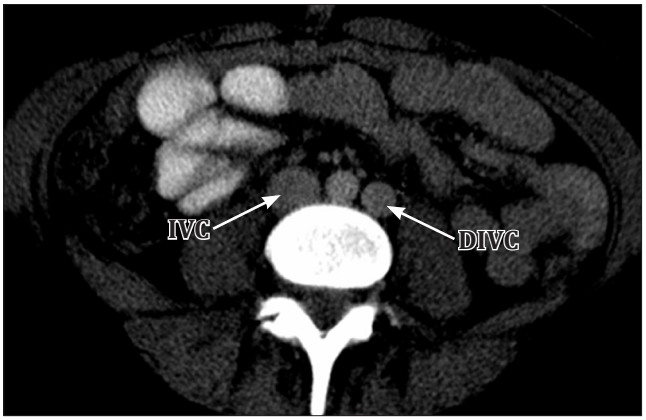

Figure 1. Case 1. Contrast-enhanced axial CT shows double inferior vena cava, *the first one at a normal anatomic location (**IVC**)* and *the second one (**DIVC**)* on the left side of the abdominal aorta with smaller size compared the right inferior vena cava.

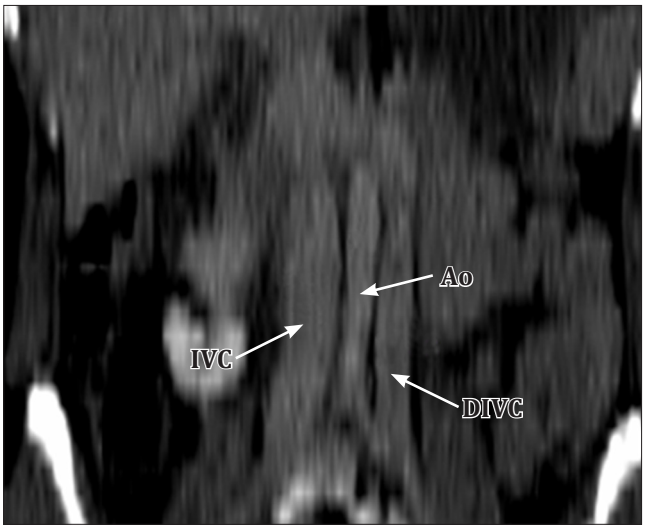

Figure 2. Case 1. Coronally reformatted CT image shows double inferior vena cava, bilateral to the aorta. *The left IVC (**DIVC**)* draining the left renal vein, crossed the *aorta (**Ao**)* anteriorly, and joined *the right inferior vena cava (**IVC**).*

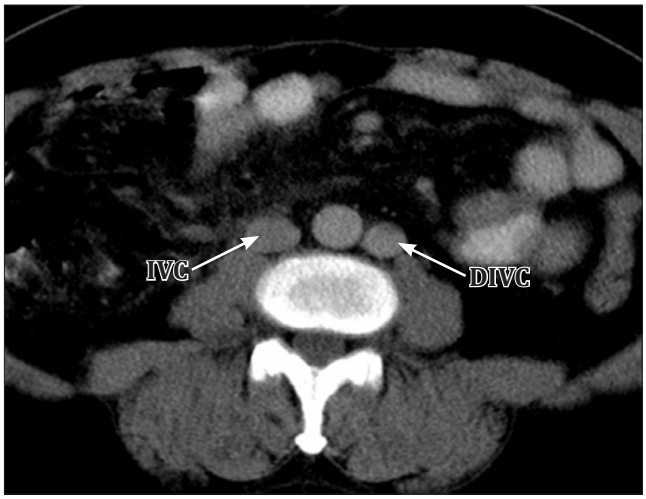

Figure 3. Case 2. Contrast-enhanced axial CT in the second patient shows a vascular structure on the left side of the aorta draining to the left renal vein which resulted to be the duplicated IVC. *(**IVC**: inferior vena cava; **DIVC**: double inferior vena cava)*

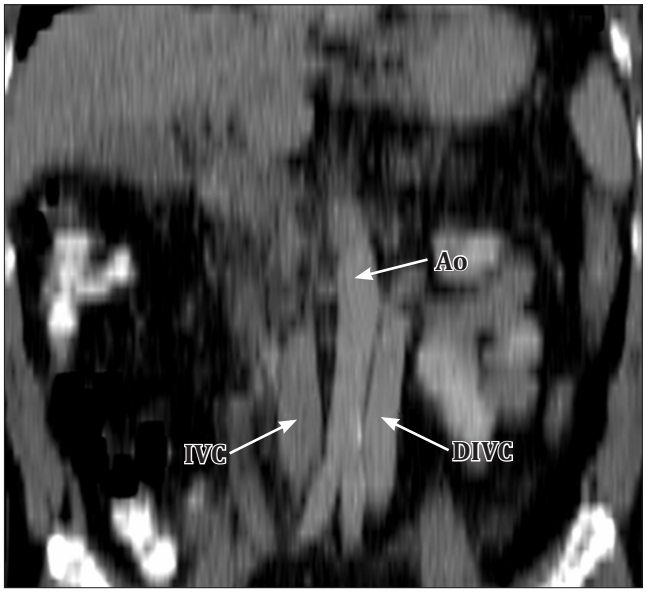

Figure 4. Case 2. Coronally reformatted CT image shows double inferior vena cava seen running on both sides of *aorta (**Ao**). (**IVC**: inferior vena cava; **DIVC**: double inferior vena cava)*

of both the right and left supracardinal veins. Although, double IVC variation is not common, it has clinical importance consequence in certain setting. Radiologically double IVC can be mistaken with a retroperitoneal mass or paravertebral lymph-node enlargement [4, 5] and lack of knowledge of these variations might result in a misinterpretation of the radiologic images of double IVC, leading to surgical errors such as bleeding during retroperitoneal lymph node dissections. These diagnostic pitfalls can be avoided by performing helical CT with IV contrast administration. Also patients with IVC variations have high risk of developing deep vein thrombosis through an inadequate blood return which increase the blood pressure in vein with venous stasis and consequent venous thrombosis of the lower extremities [6, 7] Treatment of patients with peripheral thromboembolic diseases include double IVC observation, placing filter in either system or coil-embolization of the duplicated segment plus placing a filter in the right IVC. Failure to diagnose the double IVC may lead to recurrent embolism in these patients [8, 9]. Recognition of a double IVC is also important during whole organ transplantation or radical nephrectomy. Imaging examination, CT or MRI plays an important role in the diagnosis of different variations of inferior vena cava.

Conclusion

Double IVC is an uncommon congenital variation. Detailed knowledge of this variation becomes significant to differentiate it from other pathologic conditions and is of extreme importance for surgeons to avoid complications during retroperitoneal surgery or interventional procedures [10].

Acknowledgements

The author would like to express her special thanks to her Mentor and teacher, Prof. Dr. Sofika Qamirani, to his colleague Nikollaq Leka, MD, for his great effort in preparing this manuscript; and to her colleague Eriona Abazaj, MD for assistance in the preparation of the figures.

References

[1] Sadler TW. Langman's Medical Embryology. 9th Ed., Philadelphia, Lippincott Williams &Wilkins. 2004; 261–266.

[2] Moore KL, Persaud TV. The Developing Human. Clinically Oriented Embryology. 8th Ed., London, Saunders Elsevier. 2008; 285–336.

[3] Bass JE, Redwine MD, Kramer LA, Huynh PT, Harris JH. Spectrum of congenital anomalies of the inferior vena cava: cross-sectional imaging findings. Radiographics. 2000; 20: 639–652.

[4] Kleberg I, Wajsman Z. Duplicated inferior vena cava simulating retroperitoneal lymphadenopathy in a patient with embryonal cell carcinoma of the testicle. J Urol. 1986; 136: 678–679.

[5] Ng WT, Ng SS. Double inferior vena cava: a report of three cases. Singapore Med J. 2009; 50: 211–213.

[6] Reinus WR, Gutierrez FR. Duplication of the inferior vena cava in thromboembolic disease. Chest.1986; 90: 916–918.

[7] Kondo Y, Koizumi J, Nishibe M, Muto A, Dardik A, Nishibe T. Deep venous thrombosis caused by congenital absence of the inferior vena cava: report of a case. Surg Today. 2009; 39: 231–234.

[8] Ruggeri M, Tosetto A, Castaman G, Rodeghiero F. Congenital absence of the inferior vena cava: a rare risk factor for idiopathic deep-vein thrombosis. Lancet. 2001; 357: 441.

[9] Gayer G, Luboshitz J, Hertz M, Zissin R, Thaler M, Lubetsky A, Bass A, Korat A, Apter S. Congenital anomalies of the inferior vena cava revealed on CT in patients with deep vein thrombosis. AJR Am J Roentgenol. 2003; 180: 729–732.

[10] Babaian RJ, Johnson DE. Major venous anomalies complicating retroperitoneal surgery. South Med J. 1979; 72: 1254–1258.