

A CASE OF DOUBLE INFERIOR VENA CAVA

ABSTRACT

Duplications of the inferior vena cava (IVC) are seen with an incidence of 0.2% to 3.0% in the general population. Embryogenesis of the IVC is a complex process involving the intricate formation and regression of numerous anastomoses, potentially leading to various anomalies. These anomalies are usually of no clinical significance however may predispose to thromboembolism, presumably by favoring venous stasis, in addition, aberrant vessels may be mistaken for lymphadenopathy and may be biopsied which can cause significant hemorrhage. In this report, we present a 14 years old boy, who was incidentally diagnosed to have double IVC from computed tomography (CT) done for other reasons.

INTRODUCTION

Abnormalities of the inferior vena cava (IVC) are extremely rare. With the increasing use of CT scans, these anomalies are more frequently diagnosed. The majority of venous anomalies are asymptomatic and they include left-sided IVC, duplicated IVC, and absent IVC. The embryological development of the IVC is complex and involves the development and regression of three sets of paired veins.

The first case of inferior vena cava duplication (IVCD) was described in 1916 in a male subject dissected during an autopsy by Lucas in London [1]. As reported in the literature, the incidence of IVCD is 1.5% (range 0.2%–3%), with intraoperative findings between 0.2% and 0.6% [2–4]. In this report, we discuss the embryology of the double IVC.

CASE REPORT

A 14 years old boy with no significant past medical history had a road traffic accident. Upon arrival to the casualty department, he had a full Glasgow coma scale, however, his vital signs showed episodes of hypotensive with tachycardia. Generally, he looks pink and he had tenderness over the abdomen. Focus assessment sonography for trauma (FAST) showed positive intrabdominal free fluid indicating intraabdominal injury. Computed tomography of the abdomen revealed that he sustained AAST classification grade 1 splenic injury, however, another pathology was incidentally noticed in the CT scan in which he has double inferior vena cava.

The CT showed double IVC below the renal veins. Both IVC were formed from the respective common iliac veins and ran upwards bilaterally to the abdominal aorta as far as the renal veins as shown in figure 1. The left IVC terminated on the left renal vein as shown in figure 2. The left renal

vein crossed posterior to the aorta to join the right IVC. The right IVC, once receiving the left and right renal veins, ran upwards as a single vein continuing superiorly as the azygos vein within the retro-rural space. The azygos vein drained into the superior vena cava in the right paratracheal space. The hepatic veins drained into a stump of the hepatic segment of the IVC, which opened cranially into the right atrium and caudally drained into the right renal vein, at the confluence with the right IVC.

Otherwise, he is asymptomatic for the double IVC and the splenic injury was successfully treated conservatively.



Figure 1: Double IVC ran upwards bilaterally to the abdominal aorta



Figure 2: The left IVC terminated into the left renal vein

DISCUSSION

The normal IVC is composed of four segments: hepatic, suprarenal, renal, and infrarenal. It derives from a complex embryogenic process beginning at the sixth week of gestation and involving three pairs of primitive veins (posterior cardinal, subcardinal, and supra cardinal veins) that appear and regress anastomosing in the final IVC. In particular, the postcardinal veins appear and remain in the pelvis as the common iliac veins, the right supracardinal vein persists to form the infrarenal IVC, and the right subcardinal vein persists to develop into the suprarenal segment by the formation of the subcardinal-hepatic anastomosis while the left subcardinal vein and the left supracardinal vein regress completely [2, 4, 6]. The renal segment develops from the anastomosis between the

subcardinal and supracardinal veins while the hepatic segment derives from the right vitelline vein [6, 7].

An alteration of one step of this process determines at least 14 different anatomic anomalies of the IVC and many classification systems have been proposed to group these variants.

As reported by Bass, major anomalies are double IVC (with a prevalence of 0.2–3%), left IVC (0.2–0.5%), retro aortic left renal vein (2.1%), circumaortic left renal vein (8.7%), and absence of the hepatic segment of the IVC with azygos continuation of the IVC (0.6% of cases).

The duplication of IVC results from the persistence of the right and the left supracardinal veins [5,6]. The persistence of both the right and left supracardinal veins leads to the development of a double IVC up to the level of the left renal vein. The duplicated left IVC joins the left renal vein, which in turn runs to the right to drain into the right IVC. The condition is asymptomatic and usually diagnosed incidentally following abdominal imaging.

Double IVC can be misdiagnosed as lymphadenopathy, especially in patients being evaluated for renal surgery or neoplasm. Patients with double IVC are at risk of vascular injury during retroperitoneal procedures therefore identification before surgery is essential to avoid such complications, highlighting the need for coronal reconstructions [7]. Contrast-enhanced CT or magnetic resonance imaging (MRI) is required to identify vascular structures.

Some reports suggest that patients with double IVC have a higher risk of developing thromboembolic events [8,9]. Abnormal veins can be dilated and tortuous; therefore, blood flow can be altered. This increases the chance of thromboembolic events in these anomalous veins. Recurrence of emboli from deep vein thromboses of the lower extremities in the presence of an IVC filter may suggest the presence of IVC duplication.

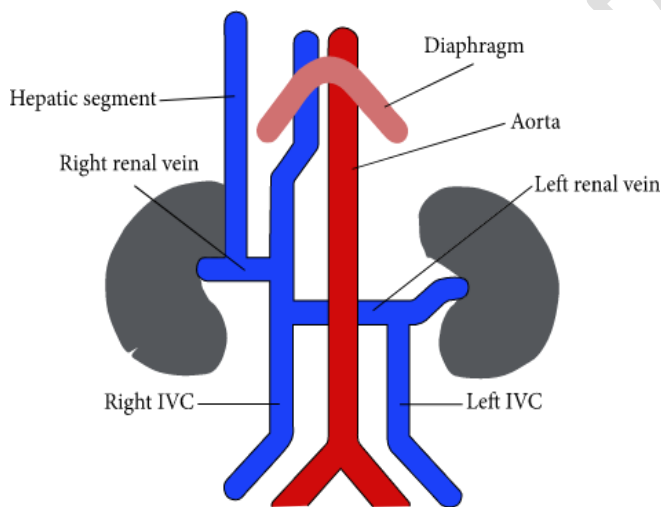


Figure 3: Schematic figure illustrating double IVC with a retro aortic left renal vein, azygos continuation

CONCLUSION

Anomalies of the IVC are rare anatomical variants that result from abnormal development/regression of fetal venous circulation. The majority of patients are asymptomatic and these variants are detected incidentally on CT/MRI. Awareness of the different anomalies of the IVC is necessary for radiologists to prevent misinterpretation of aberrant vessels such as paravertebral lymph node enlargement and mediastinal masses. The Multidetector CT technique is the preferred method for imaging the congenital vascular anomalies of IVC since it is less costly, less invasive than conventional

angiography, fast, easily applicable, and reliable in terms of identification of thoracoabdominal vascular structures.

Their presence may have significant implications for surgical procedures, as careful preoperative planning is required. This is particularly the case for procedures such as laparoscopic radical nephrectomy or laparoscopic donor nephrectomy.

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