

# Duplicate Inferior Vena Cava Description and its Clinical Relevance: a Case Report

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## ABSTRACT

**Introduction:** anomalies of the inferior vena cava (IVC) are rare, with duplication being one of the most frequent variations, occurring in approximately 0.2% to 3% of the general population. The IVC develops through a complex embryological process involving the regression and persistence of supracardinal, subcardinal, and posterior cardinal veins. Duplication results from the persistence of bilateral venous structures and is typically asymptomatic, often identified incidentally during imaging exams. However, its recognition is crucial for surgical planning and preventing complications during invasive procedures.

**Case Report:** a 45-year-old male underwent magnetic resonance imaging (MRI) of the abdomen and pelvis to evaluate renal abnormalities detected on ultrasonography. MRI revealed the absence of common iliac veins, with the internal and external iliac veins ascending bilaterally to form two separate IVCs. The right and left veins followed a parallel course anterior to the psoas major muscle, anastomosing at the level of the intervertebral symphysis between T XII and L I, where they merged with the renal veins to form a single IVC. No additional anatomical abnormalities were observed.

**Conclusion:** IVC duplication is a rare anatomical variant with significant clinical implications. Although usually asymptomatic, it may complicate retroperitoneal and pelvic procedures, increasing the risk of iatrogenic injuries and deep vein thrombosis due to altered hemodynamics. Recognizing this variation through detailed imaging assessments is essential for precise diagnosis, surgical planning, and effective clinical management, minimizing potential complications and optimizing patient outcomes.

**Keywords:** Inferior vena cava duplication; Anatomical variation; Congenital vascular anomalies.

## Introduction

Anomalies and anatomical variations of the inferior vena cava (IVC) are rare, with IVC duplication being one of the most frequent alterations<sup>1,2</sup>. There is considerable controversy regarding who first described this condition. However, one of the earliest reports was published in 1881 in the Saint Bartholomew's Hospital Reports by Church and Langton<sup>3</sup>. Currently, the estimated prevalence of IVC duplication ranges from 0.2% to 3% in the general population<sup>4,5</sup>, with a global average of 0.7%<sup>6</sup>.

The development of the IVC is a complex process, involving the formation and coordinated regression of the supracardinal, subcardinal, and posterior cardinal veins<sup>7,8</sup>. Duplication is characterized by the presence of two inferior vena cavae, resulting from the persistence of segments of the cardinal veins during embryonic development<sup>9,10</sup>.

In most cases, IVC duplication is an asymptomatic condition, often identified incidentally during imaging exams performed for other reasons, such as abdominal pain or hepatic alterations<sup>10-12</sup>. Although it typically

does not cause direct symptoms, IVC duplication may interfere with clinical management and has been associated with other congenital anomalies, such as renal agenesis and variations in venous drainage patterns<sup>8,13</sup>.

The recognition of IVC duplication can be challenging due to the absence of specific clinical signs. Imaging techniques, such as computed tomography (CT) and magnetic resonance imaging (MRI), are the primary tools for its identification, allowing for a detailed analysis of anatomical variations<sup>4,5,14</sup>. Early diagnosis plays a critical role in surgical planning and the prevention of complications, particularly in cases of thrombosis or during invasive procedures<sup>15,16</sup>. Understanding this anatomical variation is essential for tailoring therapeutic strategies and avoiding misdiagnoses or complications related to altered vascular anatomy.

This study aimed to provide an anatomical description of a case report based on a magnetic resonance imaging examination of a patient, emphasizing the importance of the accurate diagnosis of this anomaly and its clinical implications.

### Case Report

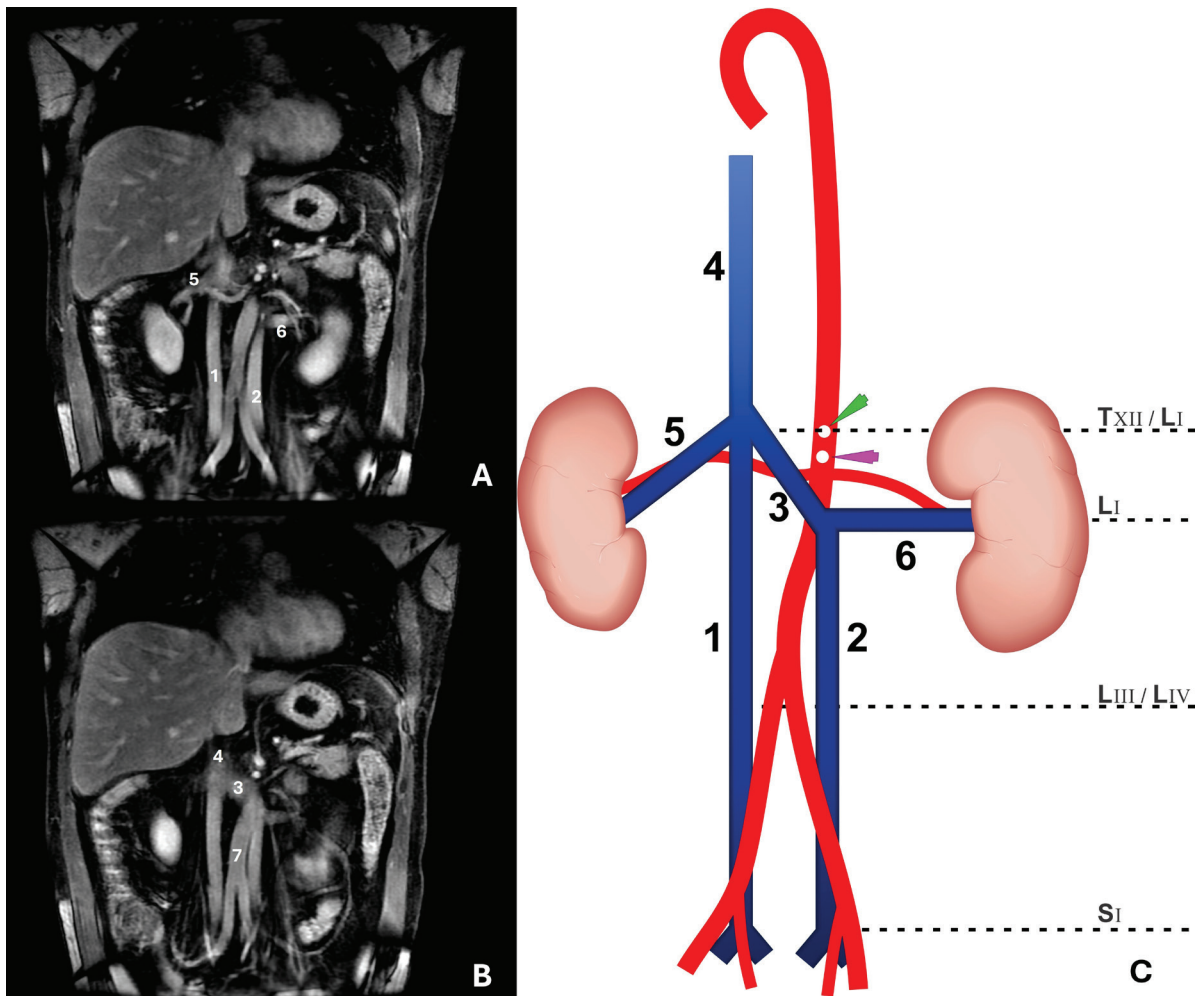
A 45-year-old man was referred for a magnetic resonance imaging examination of the abdomen and pelvis to evaluate renal abnormalities previously identified during an ultrasonography investigation. The MRI revealed that the internal and external iliac veins were located at the level of S I bilaterally, with the absence of the common iliac veins. Both veins followed a course anterior to the psoas major muscle, one on each side of the aorta, separated by 2.38 cm. The right vein measured 21.08 cm in length and 1.11 cm in diameter, while the left vein measured 21.57 cm in length and 1 cm in diameter.

On the left side, the vein anastomosed with the left renal vein (5.5 cm) at the level of L I, forming a right angle. Approximately 28 mm above this point, it anastomosed with the right side at an angle of 37.5°, at the level of the intervertebral symphysis between T XII and L I. Simultaneously, the right renal vein (5.43 cm) joined them at an angle of 55°, forming a single inferior vena cava. This single IVC continued to the right atrium of the heart (11.6 cm) without further peculiarities or variations (Figures 1 and 2).

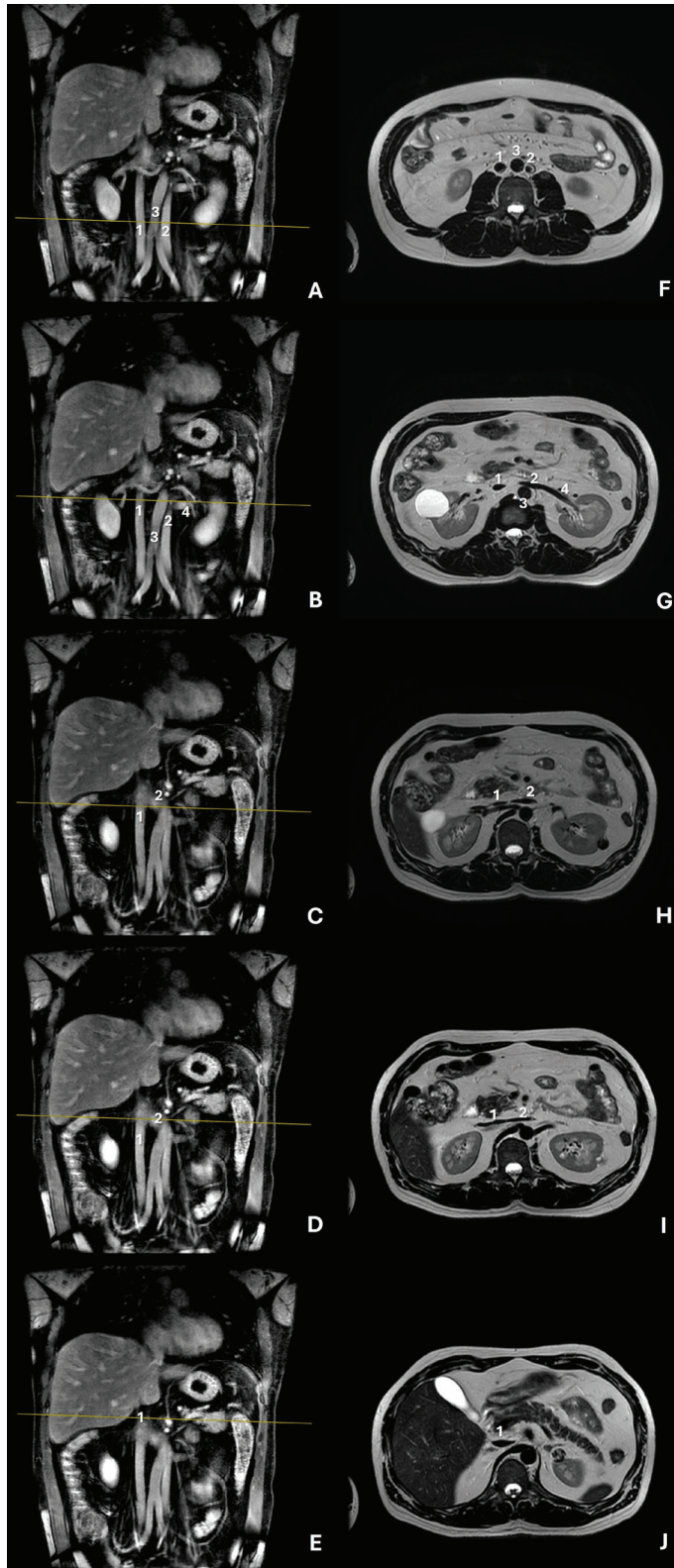
### Discussion

The inferior vena cava is an abdominal, retroperitoneal vessel that drains blood from the abdomen, pelvis, and lower limbs. It is formed by the union of the right and left common iliac veins<sup>17-19</sup>. This union typically occurs at the level of the fourth or fifth lumbar vertebra, and the IVC ascends superiorly, terminating at the floor of the right atrium after passing through the diaphragm via the aortic hiatus<sup>19-21</sup>. The normal vascular development of the IVC during embryogenesis involves four segments derived from different embryonic structures that anastomose between the fourth and eighth weeks of intrauterine life<sup>1,22,23</sup>. The three pairs of veins responsible for the formation of the IVC include the posterior cardinal, subcardinal, and supracardinal veins<sup>10,24-26</sup> (Figure 3).

The IVC develops through a series of sequential stages, beginning with the formation of primitive embryonic venous systems. Embryonic development is complex, and the formation of anastomoses among the three pairs of embryonic veins can result in congenital anomalies<sup>25,27</sup>. These anomalies may involve the bilateral persistence of subcardinal or supracardinal

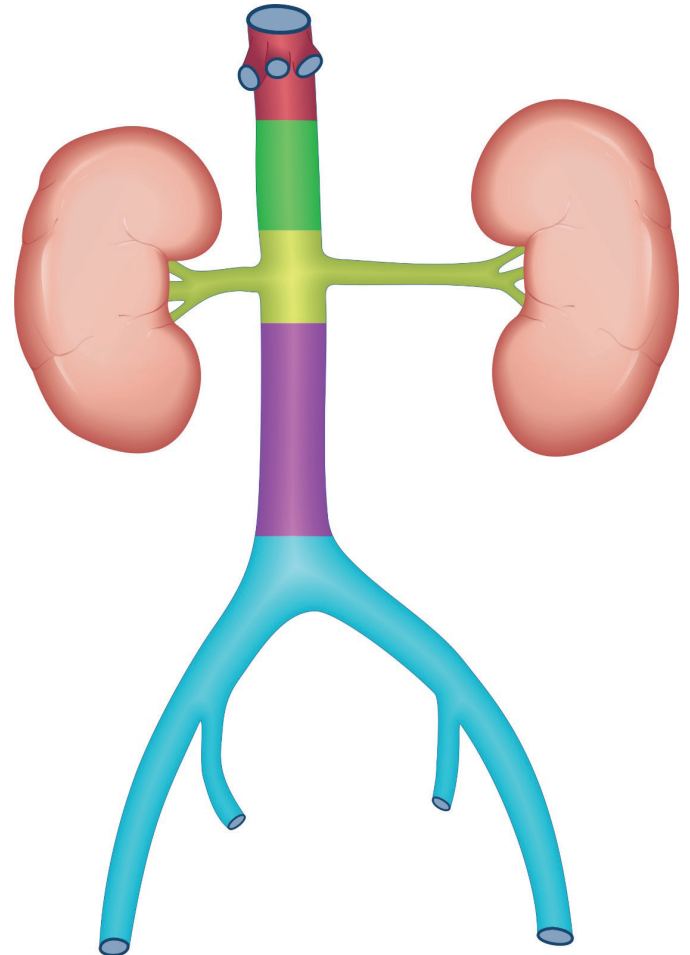


**Figure 1.** Magnetic Resonance Imaging (MRI) in the coronal plane, T1-weighted with fat saturation and after gadolinium-based contrast injection (A, B). Schematic drawing of the duplicated inferior vena cava (C). 1 - Right inferior vena cava (21.08 cm), 2 - Left inferior vena cava (17.42 cm), 3 - Course after anastomosis between the left inferior vena cava and the left renal vein (4.62 cm), 4 - Inferior vena cava (11.6 cm), 5 - Right renal vein (5.43 cm), 6 - Left renal vein (5.5 cm). Green arrowhead - Origin of the celiac trunk. Purple arrowhead - Origin of the superior mesenteric artery.



**Figure 2.** Magnetic Resonance Imaging (MRI) in the coronal plane, T1-weighted with fat saturation and after gadolinium-based contrast injection (A-E). Axial MRI images at the level of L III (F), at the intervertebral symphysis of L I/L II (G), at the level of L I (H, I), and at T XII (J), T2-weighted. 1 - Right inferior vena cava, 2 - Left inferior vena cava, 3 - Abdominal aorta, 4 - Left renal vein.

veins, interrupted development of the right subcardinal vein, or the persistence of a left supracardinal vein, leading to the formation of a duplicated IVC<sup>28-30</sup>. Other notable variations include azygos continuation of the



**Figure 3.** Schematic drawing showing the segments of the inferior vena cava and their embryological origins. Intrahepatic segment (red), origin: right vitelline vein; Suprarenal segment (green), origin: right subcardinal vein; Renal segment (yellow), origin: right subcardinal vein; Infra-renal segment (purple), origin: right supracardinal vein and anastomoses; Iliac veins (blue), origin: posterior cardinal veins.

IVC, circumaortic left renal vein, IVC agenesis, and situs inversus<sup>1,31,32</sup>. These anatomical variations may be associated with various congenital and pathological conditions<sup>23,33</sup>. Thus, variations in IVC development can have clinical implications, influencing both diagnosis and the management of medical conditions.

Although the exact causes of this condition are not fully understood, factors such as genetic predisposition, molecular signaling, and interactions with adjacent tissues appear to play a significant role in this process<sup>34</sup>. Altered anatomy can compromise blood flow and therapeutic approaches, making early diagnosis essential, particularly in patients with significant vascular comorbidities.

The findings in this case report reveal a rare and relevant configuration of the IVC, consistent with descriptions in the literature regarding congenital variations of this anatomical structure. The patient presented with a duplicated IVC, where the internal and external iliac veins on each side united to form the IVC at the level of S I, rather than at the typical level of L V, where the common iliac veins usually join to form the IVC<sup>18,22</sup>. The two IVC followed separate trajectories

until anastomosing with the renal veins, forming a single IVC. This pattern aligns with subtypes described by Morita *et al.*<sup>35</sup> and Shin *et al.*<sup>36</sup>, resembling types 2a and 6a, characterized by the absence of communication between the right and left common iliac veins. Studies by Vignesh *et al.*<sup>13</sup> and Sorin Hostiu *et al.*<sup>37</sup> indicate a prevalence of 0.2% to 3% for this condition. Additionally, Klinkhachorn *et al.*<sup>38</sup> highlighted the importance of the conformation of duplicated vessels, classifying this pattern as asymmetric duplication, where the right IVC has a larger diameter than the left.

These classifications aid in the precise description of anatomy, contributing to accurate diagnostic interpretation and appropriate clinical management. Furthermore, this type of variation is particularly relevant in retroperitoneal procedures and IVC filter placement, as altered anatomy may increase the risk of deep vein thrombosis (DVT), present in approximately 5% of cases related to IVC duplication, or create challenges during invasive procedures such as catheterization. These findings underscore the importance of detailed anatomical knowledge for diagnosis and surgical planning, reducing risks in patients with vascular variations<sup>15</sup>.

The etiology of DVT in these cases is linked to biophysical principles: IVC duplication can cause alterations in hemodynamic patterns, including turbulence and redistribution of blood flow, resulting in reduced velocities, particularly in areas of venous stasis. This slowing increases the likelihood of thrombus formation, following Virchow's triad, which associates abnormal blood flow, endothelial injury, and hypercoagulability with thrombogenesis<sup>39</sup>. For patients requiring IVC filters, identifying a duplicated vein is essential for proper placement, ensuring efficacy against embolic events. Moreover, knowledge of this variation is crucial in retroperitoneal and pelvic surgeries due to the risk of severe iatrogenic injuries, diagnostic confusion with retroperitoneal neoplasms or lymphadenopathy, and potentially fatal hemorrhages<sup>15,16,40,41</sup>.

Early detection of IVC duplication can be challenging, particularly due to the variability of its anatomical presentation and the possibility of

its existence without evident clinical signs. Given that IVC variations, including duplication, are often diagnosed incidentally through imaging studies such as computed tomography or magnetic resonance imaging<sup>15,40</sup>, surgeons and radiologists must remain vigilant in interpreting imaging findings, considering that vascular variations are possible and may be associated with abnormalities in other pelvic veins<sup>40</sup>.

In the study by Morita *et al.*<sup>35</sup>, approximately 82% of the individuals analyzed had a duplicated IVC (28 of 34), with the non-communicating pattern being the most prevalent (39.3%). In the study by Shin *et al.*<sup>36</sup>, among 2,488 patients, 40 cases (1.61%) of IVC variations were identified, of which 23 (0.9%) exhibited duplication. For Ito *et al.*<sup>42</sup>, the incidence of IVC variations was 20.9% (521 in 2,488), with 0.92% (23 individuals) presenting a duplicated IVC, with type C described by Morita *et al.*<sup>35</sup> being the most common. These data highlight the considerable prevalence of this condition in the population, reinforcing the need for detailed attention from healthcare professionals in managing and diagnosing patients with such anatomical variations.

## Conclusion

Duplication of the inferior vena cava is a rare anatomical condition that is generally asymptomatic but of significant clinical relevance. Its complex embryonic development and variations increase the risk of complications during retroperitoneal and pelvic procedures, where there is a higher likelihood of iatrogenic injuries, such as hemorrhages. Additionally, this condition is associated with a higher incidence of deep vein thrombosis due to altered venous flow, underscoring the importance of careful and detailed imaging evaluation. Knowledge of IVC duplication enables safer interventions, preventing misdiagnoses and severe complications, particularly in cases requiring IVC filters for protection against embolisms, for example. Thus, awareness of this variation among healthcare professionals is essential for precise surgical planning and the implementation of effective and safe interventions, ensuring better patient outcomes.

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## Mini Curriculum and Author's Contribution

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