

# Implications of a Potentially Compressed Left-sided Inferior Vena Cava and Subsequent Systemic Venous Consequences

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**ABSTRACT**

**Background:** The inferior vena cava (IVC) is a major vein responsible for transporting deoxygenated blood to the heart from the lower body. It is typically located on the right side following development. However, variations may arise such as a double-sided IVC or a left-sided IVC. These anomalies, while perceived inconsequential, may have significant underlying contributions to clinical conditions.

**Results:** Examination of an 86-year-old female cadaver revealed an incomplete left-sided IVC (LIVC), characterized by its position on the left side of the

abdominal aorta before crossing over to the right side. The cadaver also exhibited hepatomegaly, potentially linked to this LIVC. The enlarged liver inferiorly displaced the left kidney, exacerbating angulation of the left renal vein. The presence of the LIVC may have clinical implications, such as an increased risk of deep vein thrombosis and impacts on adjacent organs.

**Conclusion:** This case illustrates the underlying role that variations in left-right developmental patterning have on susceptibility to diseases or disorders. Considerations of such variations are critical in clinical diagnoses, particularly in cases presenting symptoms with indirect causes. This study contributes to the growing knowledge on IVC anomalies and their broader implications in treating underlying and perceived harmless contributors.

**Keywords:** Hepatomegaly; Embryonic patterning; LIVC; Venous constriction

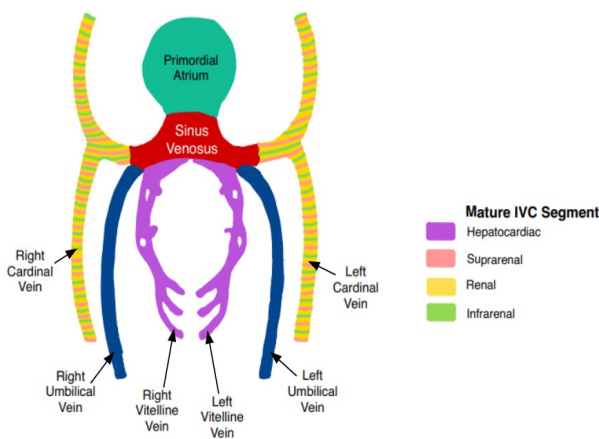
**INTRODUCTION**

This case report highlights the intersection of Left-Right (L-R) development patterning and anomalies through examination of an atypical left-sided inferior vena cava (LIVC). L-R patterning plays a crucial role in the organization of the human body during fetal development. Deviations in breaking L-R patterning symmetry may contribute to anatomical anomalies, potentially giving rise to disorders. For example, deviations from typical patterning may lead to significant anatomical vascular variations with underlying clinical implications.

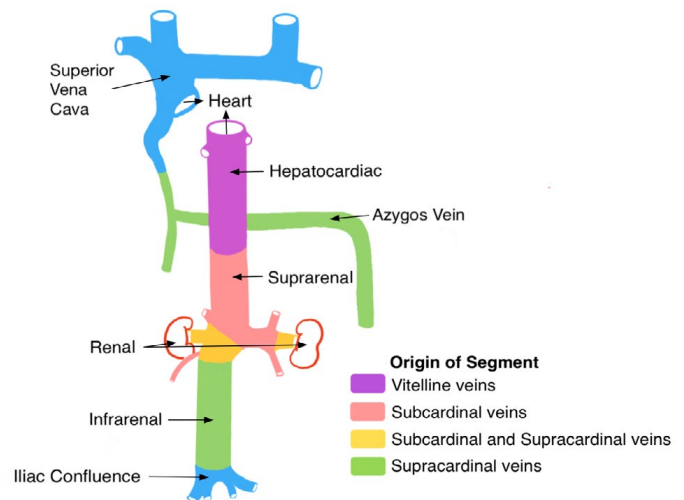
The IVC is a major vein, responsible for carrying deoxygenated blood from the abdomen and lower extremities to the right atrium of the heart. At approximately the L4/L5 vertebral level, the common iliac veins merge to form the IVC, which is located to the right of the abdominal aorta (AA). During gestational weeks 5 to 7, three venous systems are present: the vitelline, umbilical, and cardinal (Figure 1) [1]. All three systems present as

paired veins, converging towards the developing heart and modeling L-R symmetry in the early embryo [1]. As embryonic development progresses, there is a redirection of blood flow towards the right-sided veins as venous return enters the right atrium [1]. This left-to-right shunt of blood causes a breakdown of the embryonic venous symmetry through enlargement of the right-sided veins and regression of the left-sided veins. Finally, the IVC is formed from segmental contributions of these veins, including the hepatocardiac, suprarenal, renal, and infrarenal components (Figure 2) [2, 3].

The hepatocardiac segment is the most superior part of the IVC, formed by enlargement of the right vitelline and simultaneous disappearance of the left vitelline veins [1]. The suprarenal segment develops, through anastomosis of the sub cardinal veins. These sub cardinal veins form from the cardinal veins, which drain the embryonic kidney and anastomose with the supracardinal veins that drain the intercostal veins. The renal segment develops from the anastomosis of sub cardinal and supracardinal veins. Finally, the infrarenal



**Figure 1** Embryonic venous system at the gestational weeks 5-7, showing the left and right paired veins that contribute to IVC development. The cardinal veins give rise to the suprarenal (pink), renal (yellow), and infrarenal (green) segments later in development (modified from Figure 1 in Yagel, et al (2010)).



**Figure 2** Development of the mature IVC with each segment labeled (modified from Figure 2 in Li, et al. (2021)).

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Table 1) Major IVC anomalies and their deviations from typical development (synthesized from Sadler (2004, p. 266)[1], and Eldefrawy, et al., (2011, pp.4-8) [7].

Venous System Defects	Embryonic Morphology
Double Inferior Vena Cava (DIVC)	Left sacrocardinal veins remain connected with the left sub cardinal vein
Absence of the Inferior Vena Cava (AIVC)	Right sub cardinal vein fails to connect with the hepatocardiac segment
Left-sided Inferior Vena Cava (LIVC)	Regression of the right supracardinal vein and persistence of the left supracardinal vein

segment forms through an anastomosis of the supracardinal veins. The IVC forms from connection of the hepatocardiac segment and the right sub cardinal vein. The left-sided veins regress and disappear as the IVC continues to mature [1].

Variations in IVC development may arise as a result of improper breaking of embryonic venous symmetry. These anomalies of the IVC are generally rare [4]. Such variations are often considered to be incidental findings during diagnostic imaging [5]. Major variations include a double IVC (DIVC), a left-sided IVC (LIVC), and an absence of the IVC (AIVC) (Table 1).

DIVC is the most common variation (0.2-3%)[6]. and LIVC is the second most common with an incidence rate of 0.2-0.5% [5, 6]. In development of the DIVC, the left sacrocardinal veins remain connected to the left sub cardinal vein [1]. However, the LIVC develops through an atypical regression of the right supracardinal and persistence of the left supracardinal veins, primarily affecting the infrarenal segment of the IVC [7]. Two sub-variations of LIVC may arise: an incomplete and a complete LIVC. In an incomplete LIVC, the left common iliac vein ascends as a tributary to a retained LIVC, and crosses the AA before joining the proximal component of the LIVC on the right side [8]. In a complete LIVC, the LIVC drains the left renal vein and ascends as a preaortic trunk, emptying into the superior segment of the IVC [8].

**Procedures**

The cadaver donor was an 86-year-old female, who passed away from cerebrovascular disease. The cadaver donor was stored and maintained in the medical school’s dissection room, ensuring optimal care, respect, and dignity. During routine abdominal dissection, several anatomical variations were noted including: a LIVC, hepatomegaly, and a displaced left kidney. No visible surgical interventions were observed. With permission, this study was undertaken, images and measurements of these abnormalities were recorded, and safety protocols and ethical guidelines were adhered to. The study was conducted with a deep appreciation of the valuable contribution of the cadaver donor, acknowledging the significance of body donation to medical science and education.

There were specific limitations that are important to acknowledge here. Firstly, due to the privacy policy of the donor program, medical records were not made available. This lack of medical background information limited the ability to correlate observed anatomical features with any pre-existing health conditions or diseases. Secondly, we did not dissect the lower limbs to investigate the presence of deep vein thrombosis. Given that post-mortem blood clotting had already occurred, ante mortem thromboses would be indistinguishable from post-mortem thromboses. This limitation prevented us from comprehensively assessing the vascular health of the donor and potentially relevant pathologies.

**CASE REPORT**

This cadaver donor exhibits an incomplete LIVC, which originates from the convergence of the common iliac veins and courses along the left side of the AA. At the first lumbar vertebral level, the LIVC courses anteriorly over the AA to the right side (Figure 3). During its course anterior to the AA, the LIVC passes inferior to the superior mesenteric artery (SMA). This atypical course of the IVC is in contrast to the typical placement of the left renal vein between the SMA and AA.

The cadaver donor is visibly large and displaces the left kidney. For the cadaver’s sex and height range (152.4-160.0 cm), a typical liver measures 6.00-6.75 cm at the midclavicular line and 4.00-4.50 cm at the midsternal line [9]. This cadaver’s liver measured 15.00 cm at the midclavicular line and 12.00 cm at the midsternal line, consistent with hepatomegaly (Figure 4). The enlarged liver displaced the left kidney inferiorly. In contrast to the typical anatomical arrangement where the right kidney is positioned slightly inferiorly compared to the left, the enlarged liver and inferiorly-displaced left kidney resulted in an inferiorly-directed and angulated left renal vein (Figure 5).

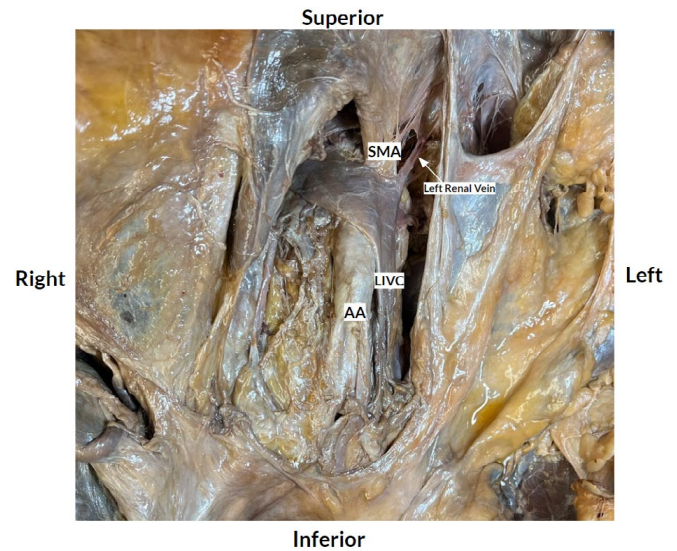


Figure 3) Anterior view of the posterior abdominal wall and the LIVC. (The parietal peritoneum has been removed).

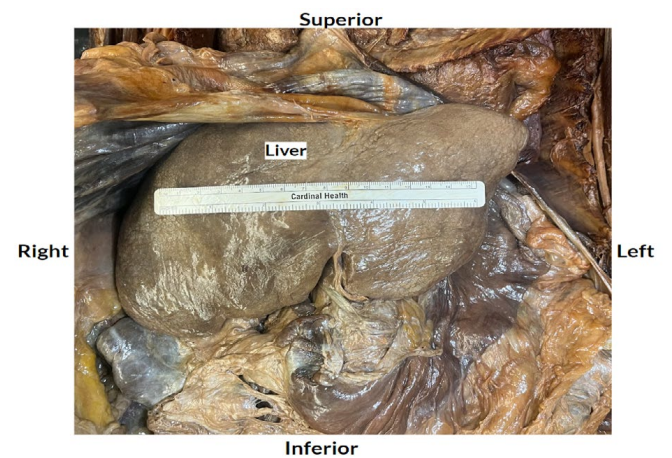


Figure 4) Anterior view of the enlarged liver in situ (ruler for scale).

**RESULTS AND DISCUSSION**

The presence of a rare LIVC in conjunction with hepatomegaly and kidney displacement raises questions regarding the underlying role of variations in clinical diseases and disorders. In this case, the LIVC passes between the AA and SMA, a typical site of anatomical compression of the left renal vein [10]. Particularly in males, left renal vein compression restricts blood flow from the left kidney and left gonadal vein, leading to Nutcracker Syndrome [10].

Although the LIVC courses anterior to the AA in this female cadaver, its compression was not apparent during dissection. However, the presence of hepatomegaly and subsequent displacement of the left kidney may suggest compression of the LIVC did occur. Other possible clinical implications associated with LIVC include a weaker right atrium myocardium, hypotension, and a greater risk of deep vein thrombosis (DVT) due to venous blood pooling [5]. Inhibited venous flow through a compressed IVC increases the risk of coagulation upstream of the venous system and in the deep veins of the lower limbs. The LIVC potentially puts younger patients at an increased risk for DVTs. In younger patients with tibiofemoral DVT, venous stasis was likely secondary to the IVC anomalies that were present



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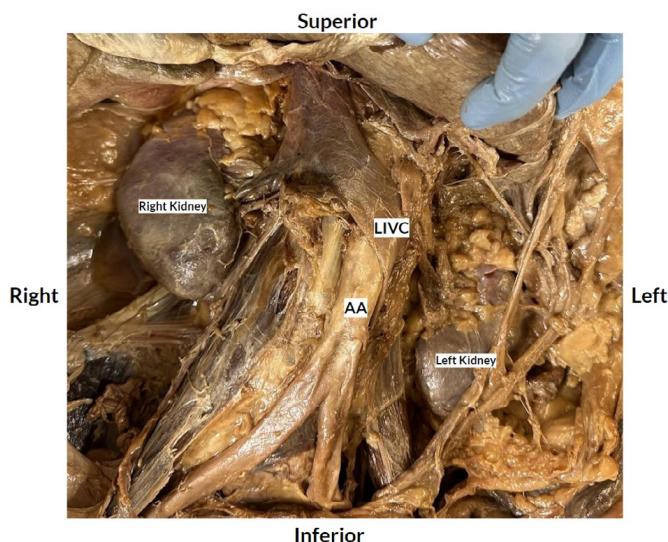


Figure 5) Anterior view of the IVC and other retroperitoneal structures.

in 5-16% of these patients [6]. Thus, prophylactic antithrombotic measures should be considered in these patients to lower the risk for venous stasis [2].

The height and liver measurements of the cadaver suggests the presence of hepatomegaly [9]. Hepatomegaly can present secondary to IVC compression or obstruction due to increased blood pooling in the venous system and end organs or from a thrombosis such as a DVT [11, 12]. The hepatomegaly displaced the left kidney inferiorly in the cadaver, relative to the right kidney, which is usually displaced more inferiorly. The inferiorly-displaced left kidney resulted in an inferiorly-directed and angulated left renal vein, which may affect venous drainage of the kidney. This left kidney may be considered to be an ectopic kidney due to the significance of the inferior displacement. These findings associated with the LIVC, highlight the importance of considering the role of anatomical variations as underlying causes of clinical disease and disorders [13].

### CONCLUSION

This example of a LIVC with potential complications highlights the importance of recognizing the underlying role of developmental abnormalities in diseases and disorders and their management. LIVC formation occurs through alterations in L-R developmental patterning, producing venous variations with an array of potential implications that may be overlooked. For example, symptom production and alleviation are frequently the focus of clinical treatment, while visually innocent variations are overlooked in their potential causal role.

Awareness of anatomical variations like a LIVC is also important beyond diagnostic processes and management for routine surgical interventions and procedures. For example, in contrast to an IVC filter insertion in a patient with typical IVC anatomy, a modified procedure should be considered for a patient with a LIVC because of the potential for injury from a misguided catheter. This case underscores the need for healthcare providers to integrate knowledge of anatomical variations along with considering preoperative imaging and planning in their clinical decision-making. Identifying anomalies during the diagnostic process, prior to procedures and throughout interventional treatments could improve patient care and may prevent serious complications during medical procedures. Finally, investigating the role of developmental abnormalities as a potential causal factor in diseases and disorders is highly relevant.

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### CONFLICT OF INTEREST

None.

### ETHICS APPROVAL STATEMENT

Permission granted by the Rutgers University Anatomical Association.

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