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DUPLICATED INFERIOR VENA CAVA WITH INTERRUPTION AND AZYGOS CONTINUATION: A CASE REPORT AND LITERATURE REVIEW

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ABSTRACT

Background: *Congenital anomalies of the inferior vena cava (IVC) are rare vascular variants that can have significant clinical implications, especially during surgical or interventional procedures.* ***Case presentation:*** *We present a rare case of a 39-year-old female who was found incidentally to have a duplicated IVC associated with interruption of the retrohepatic segment and azygos continuation. Multidetector computed tomography (MDCT) with contrast revealed bilateral IVCs running alongside the abdominal aorta, connected by a transverse venous bridge below the aortic bifurcation.* ***Conclusion:*** *This anomaly results from atypical persistence and regression of embryonic venous precursors, including the subcardinal and supracardinal veins. Awareness of such variants is crucial, as they may complicate retroperitoneal surgery, cardiopulmonary bypass, or IVC filter placement and can predispose to venous stasis and thrombosis. MDCT with multiplanar reconstruction remains the modality of choice for accurate diagnosis and anatomical delineation.*

Keywords: *CT, duplicated IVC, azygos continuation, congenital.*

I. INTRODUCTION

The inferior vena cava (IVC) is the principal venous conduit for blood return from the subdiaphragmatic region, draining the abdomen, pelvis, perineum, and both lower extremities. It is formed by the confluence of the two common iliac veins at the anterolateral aspect of the fifth lumbar vertebra, ascends along the spine to the right of the aorta, courses posterior to the liver, and traverses the central tendon of the diaphragm at approximately the level of the eighth thoracic vertebra [1].

Congenital anomalies of the IVC are uncommon, with reported prevalence ranging from 0.07% to 8.7%, no specific age peak, ethnicity or gender relation is noted [2]. Some variants are clinically relevant as potential underlying causes of deep venous thrombosis (DVT), particularly in younger patients [3]. Moreover, awareness of these developmental variants of the IVC is essential to minimize complications during retroperitoneal surgery and endovascular interventions involving the IVC.

Here, we report a case with a rare and complex IVC anomaly characterized by suprarenal IVC interruption with azygos continuation associated with an unusual infrarenal bifurcation and retroaortic venous branch. According to literature, there have been few cases of IVC interruption with azygos continuation reported. Among them, Khadija et al. described duplicated IVC with azygos continuation and symmetrical bilateral venous ascent, and Géraud et al. reported a left-sided IVC variant terminating via retroaortic renal

venous crossover into a dominant right IVC [4], [5]. Our case uniquely combines features of both suprarenal interruption and infrarenal branching with inter-venous communication. This distinctive configuration expands the spectrum of reported IVC developmental anomalies and underscores the importance of detailed pre-procedural venous mapping to avoid potential diagnostic and interventional pitfalls.

II. CASE PRESENTATION

A 39-year-old female patient presented with chest pain, dyspnea and was admitted to the hospital for evaluation at the Cardiovascular Center. She had Graves' disease; her family history showed no relevant abnormality. Her electrocardiogram and echocardiogram showed normal findings. Therefore, a non-contrast chest computed tomography (CT) scan was performed, which demonstrated dilatation of the azygos vein, measuring up to 21 mm in diameter. Superiorly, the dilated azygos vein drains into the superior vena cava (SVC), while inferiorly it is continuous with the IVC. The hepatic veins converge at their confluence and drain directly into the right atrium. Based on these findings, the radiologist suspected an IVC anomaly and recommended contrast-enhanced thoracoabdominal CT for further assessment.

On contrast-enhanced CT, the suprarenal segment of the IVC was found to be continuous with the azygos vein, without direct drainage into the right atrium. Below the level of the L1 vertebral body, the IVC bifurcates into two branches: the left branch courses posterior to the abdominal aorta, while both branches descend along either side of the aorta. Each branch receives drainage from the ipsilateral renal vein as well as normal inflow from the common iliac veins bilaterally. A venous bridging connection between the two branches is identified just beneath the bifurcation of the abdominal aorta. The hepatic veins drain directly into the right atrium via a separate confluence, without communication with the IVC.

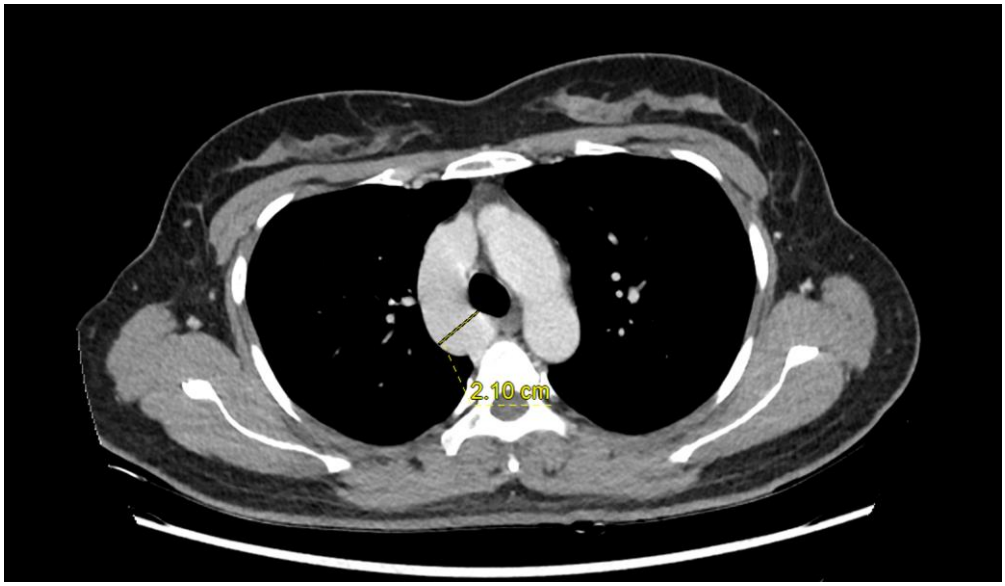

Figure 1. Dilated azygos vein

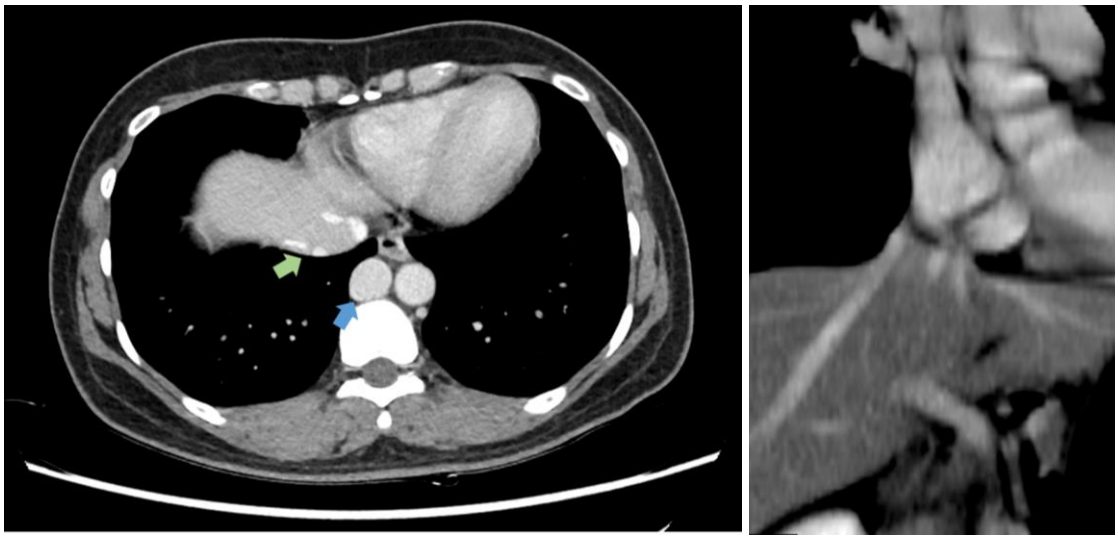

Figure 2. The hepatic veins (green arrow) draining into the right atrium via a separate confluence, without communication with the IVC (blue arrow)

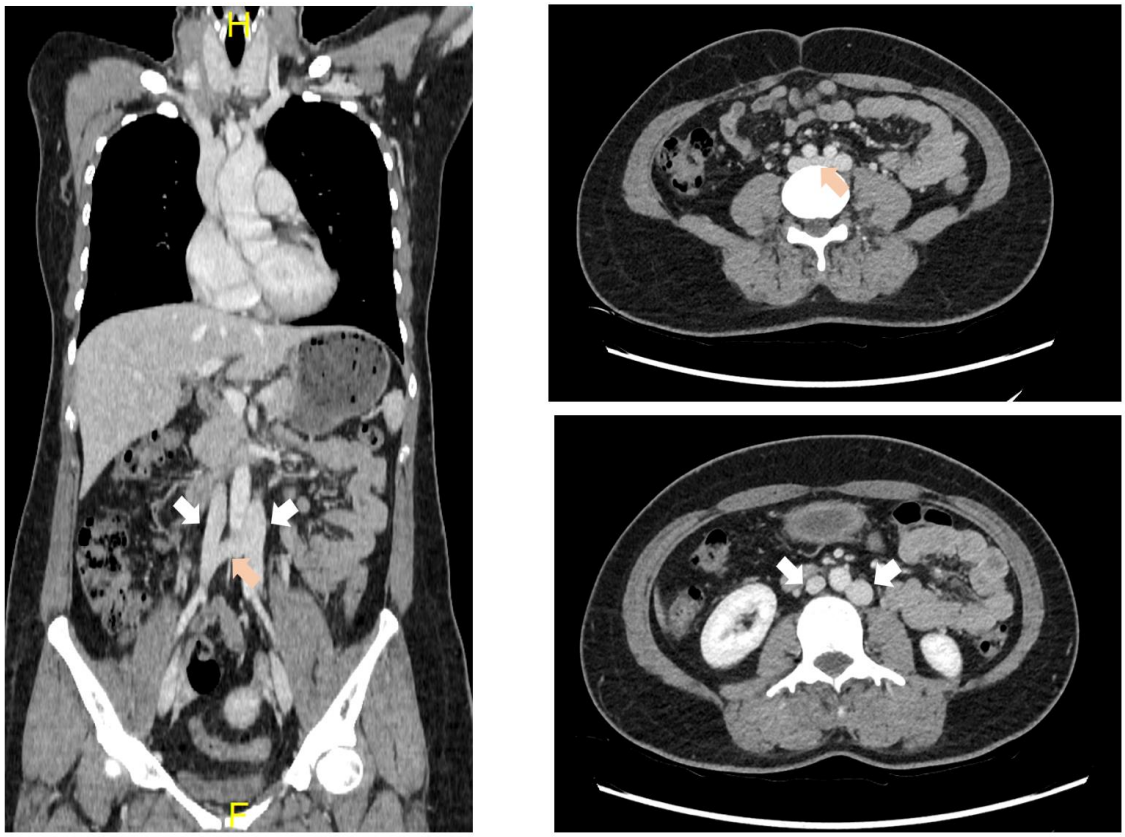

Figure 3. Bilateral IVCs (white arrow) course along the abdominal aorta with a communicating vein (orange arrow) beneath the bifurcation of the abdominal aorta

III. DISCUSSION

Knowledge of IVC embryology is crucial for understanding these congenital anomalies. The development of the IVC is a complex process that originates from three

paired embryonic venous systems: the posterior cardinal, subcardinal, and supracardinal veins. These channels appear sequentially during early embryogenesis, and the definitive right-sided IVC emerges as a composite vessel through selective persistence, anastomosis, and regression of these precursors. Specifically, the suprarenal segment derives from the right subcardinal vein, the infrarenal segment from the right supracardinal vein, and the renal segment from the anastomosis between the right and left subcardinal veins. Variations in this process, due to atypical persistence or regression, underlie the spectrum of congenital IVC anomalies. This developmental process occurs between the sixth and eighth weeks of embryonic life and determines the final anatomy of the IVC [6].

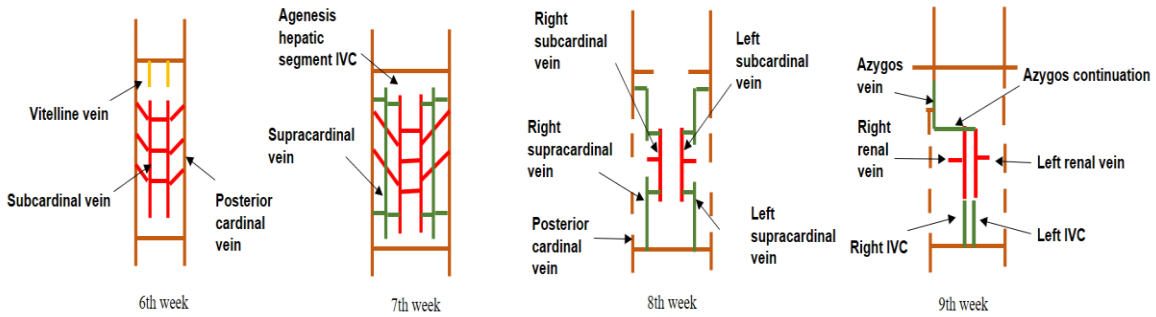

Figure 4. Diagrams illustrating the embryologic development of IVC duplication with azygos continuation at 6th week, 7th week, 8th week and 9th week of fetal development, adapted from Khadija ElAitari et al. [4]

By the sixth week of development, venous return from the caudal embryo is carried by the paired posterior cardinal veins, which later form the common iliac veins. The vitelline and umbilical systems contribute to the hepatic plexus, with the right vitelline channel persisting as the hepatic segment of the IVC. In the following weeks, the subcardinal and supracardinal veins arise and form key anastomoses that generate the renal and suprarenal segments of the IVC, as well as the gonadal and suprarenal veins. Regression of the left-sided channels establishes right-sided dominance, with the right supracardinal vein forming the infrarenal IVC and the cranial supracardinal system developing into the azygos–hemiazygos network.

Duplication of the IVC results from persistence of the connection between the left sacrocardinal and left subcardinal veins, with or without formation of the left common iliac vein, while the left gonadal vein develops normally. This variant has a prevalence of 0.2–3% [2]. A bridging vein may connect the common iliac veins at the inferior origin of the duplicated IVC. Rarely, the duplicated segment can continue as the hemiazygos vein and drain directly into the superior vena cava, as demonstrated in our case.

In some case reports, unrecognized duplication of the IVC has been associated with recurrent pulmonary embolism despite routine infrarenal IVC filter placement [7]. This can be explained by the fact that placing a filter in the right IVC does not protect against emboli originating from the left side. Therefore, alternative strategies have been proposed, including bilateral infrarenal IVC filter placement, suprarenal IVC filter placement, or steel coil embolization.

Table 1. Comparison between cases of interrupted IVC with azygos continuation [4], [5]

Feature	Our case	Khadija ElAitari *et al.* (2022)	Géraud Léra Akpo *et al.* (2021)
Venous drainage	Bilateral IVC branches drain into azygos vein → SVC. Hepatic veins drain directly to RA.	Two IVCs drain into azygos vein → SVC. Hepatic veins drain directly to RA.	Left venous system drains into left renal vein → crosses behind aorta → right IVC → RA.
IVC branch origin	Two IVCs with one originating from right CIV and one from left CIV	Two IVCs with the right one originating from right EIV and the left one from right IIV + left CIV	Two IVCs with the right one originating from right CIV + left IIV and the left one from left EIV.
IVC course	Two branches ascend on both sides of aorta. Left branch passes behind aorta. Branches connected by a venous bridge.	Two parallel IVCs run on both sides of aorta. No connecting branch.	Left venous trunk ends at renal vein; right IVC remains main channel. No connecting branch.
Relation to aorta	Two veins run on each side aorta with one connecting branch behind aorta beneath bifurcation of aorta level.	Two veins run on each side of aorta.	One left vein crosses behind aorta at renal level.

IVC, inferior vena cava; SVC, superior vena cava; RA, right atrium; AV, azygos vein; IBV, inferior vena cava (dominant right-sided venous trunk); EIV, external iliac vein; IIV, internal iliac vein; CIV, common iliac vein; LRV, left renal vein; RRV, right renal vein;

Interrupted IVC with azygos continuation occurs when the right subcardinal vein fails to establish continuity with the liver, diverting venous return from the lower body through the azygos system to the superior vena cava. Meanwhile, the hepatic veins drain directly into the right atrium at the expected IVC junction. This congenital anomaly occurs in approximately 0.6% of the population, usually is diagnosed incidentally in adolescents or adults [2]. It is not associated with a specific genetic mutation. However, it can accompany with polysplenia, cardiovascular malformations, and situs anomalies, which involve genetic factors [8].

DVT can be associated with this congenital IVC anomaly. Interrupted IVC without adequate collateral pathways may similarly result in vascular complications such as deep venous thrombosis and venous insufficiency. In a registry of more than 50,000 DVT patients, IVC agenesis was identified in approximately 0.06% [3]. Young age, unprovoked DVT, and proximal or bilateral DVT were found to be associated with this condition. The underlying mechanism is largely attributed to altered venous hemodynamics. In the absence or interruption of the IVC, venous return from the lower extremities is rerouted through collateral pathways, including the lumbar, azygos–hemiazygos, and gonadal venous systems. These channels are physiologically smaller in caliber and not designed to accommodate the entire lower body venous outflow, resulting in increased venous pressure and reduced flow velocity. The combination of venous hypertension and stasis creates a prothrombotic environment that predisposes affected patients to DVT.

If unrecognized, this developmental abnormality can lead to adverse outcomes in patients undergoing thoracic or cardiopulmonary bypass surgery. A fatal case of hypovolemic shock following ligation and partial excision of an anomalous vessel has been reported, although this occurred prior to the widespread availability of multidetector computed tomography (MDCT).

Duplication of the IVC with interruption is an extremely rare congenital IVC variation. Only a few cases of this variant have been documented in the literature. Each case illustrates an unique anomaly of the IVCs. In our case, the bilateral internal and external iliac veins drained into their respective common iliac veins, which continued as bilateral IVCs. A communicating vein between the two iliac veins was identified just beneath the bifurcation of the abdominal aorta. Khadija ElAitari et al. described an unusual case in which the right internal iliac vein drained into the left common iliac vein without connection between the right and left iliac veins [4]. In contrast, Géraud Léra Akpo et al. reported a case of IVC duplication with interruption in which the left internal iliac vein drained into the right IVC [5]. Furthermore, differences in IVC calibers have been noted in Géraud's case while in our case and in Khadija's report, the calibers of both IVCs were similar.

MDCT provides the most valuable information for detecting IVC anomalies and allows multiplanar reconstructions that further characterize the abnormality. According to the literature, additional modalities such as conventional venography, Doppler ultrasound, and magnetic resonance imaging can also be used. Conventional venography is traditionally regarded as the gold standard for detecting IVC anomalies, however, it is invasive. Doppler ultrasound is often suboptimal in obese patients. MRI has limitations due to higher cost, motion artifacts, limited availability, and contraindications in patients with implanted metallic devices or claustrophobia.

IV. CONCLUSION

Duplicated IVC associated with interruption of the retrohepatic segment and azygos continuation is a highly rare IVC anomaly. Recognition of such anomalies is critical, as they may predispose patients to deep venous thrombosis and complicate surgical or interventional procedures. Multidetector computed tomography with multiplanar reconstruction remains the most effective modality for diagnosis and anatomical characterization. Awareness of these variants among clinicians and radiologists is essential to prevent misdiagnosis and guide appropriate management strategies.

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