CASE REPORT **DOI: https://doi.org/10.18502/fem.v6i2.8722**

Double inferior vena cava with bilateral mild hydronephrosis: a rare case report

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Published online: 2021-11-05

Abstract: A 47-year-old female with a history of antiphospholipid syndrome and ischemic stroke was presented to the emergency department due to abdominal pain and bloody vomiting. Ultrasonography showed double inferior vena cava and bilateral mild hydronephrosis. Furthermore, the abdominal computed tomography (CT) scan did not show any evidence of urolithiasis. The ultrasound images of distinctive developmental variations of inferior vena cava and other veins are important to be known. Vascular anomalies, although rare, should be taken into account in the differential diagnosis of focal lesions within the abdominal cavity. Double IVC might have been the cause of hydronephrosis in our patient.

Keywords: Anatomical Variation; Emergency Department; Inferior Vena Cava; Ultrasound

Cite this article as: Balafar M, Parsian Z, Javad Rashid R, Elmdust N, Arjmandi H, Soleimanpour H. Double inferior vena cava with bilateral mild hydronephrosis: a rare case report. Front Emerg Med. 2022;6(2):e27.

1. Introduction

Inferior vena cava (IVC) abnormalities are typically diagnosed accidentally in asymptomatic patients. As these anomalies cause sluggish vascular flow and several collateral vessels, they are the potential cause of deep vein thrombosis. Veins with anomalous positions are also susceptible to become varicose because of compression (1). IVC is developed in the embryologic period between the 6th and 10th week of gestation, during which embryonic veins are developed, regressed, anastomosed, and replaced (2). One of the rare anomalies of IVC is duplicated IVC, which occurs in 1.5% (between 0.2 and 3 percent) of the normal population (3). According to one theory, IVC is formed from the regression of three paired embryonic veins that have numerous anastomoses. They are named the supracardinal, the subcardinal veins, and the posterior cardinal veins (4). Duplication of the inferior vena cave can be classified as incomplete or complete. Inadequate regression of supra cardinal vein causes incomplete duplication (5). A signed consent form was obtained from the patient in the study. This research is approved by Ethics Committee of Tabriz University of Medical Sciences, Tabriz, Iran. The approval ID of this case report is IR.TBZMED.REC.1400.525.

2. Case presentation

A 47-year-old female patient was presented to the emergency department of Imam Reza General hospital in Tabriz, Iran with the chief complaint of abdominal pain. The patient had mild intermittent abdominal pain for 2 days. Her pain was ambiguous in the lower part of the abdomen that was accompanied by vomiting the previous day, which was bloody the last time. She had a past medical history of ischemic stroke two months ago that was under treatment with warfarin and atorvastatin. She was diagnosed with antiphospholipid syndrome after her ischemic stroke. Her daughter and her cousin had coagulopathy disorder as well. The patient's primary laboratory findings in the emergency department were as follows: white blood cell count: 7500/μl, hemoglobin: 13.7g/dl, platelet: 312000/μl, blood sugar: 95mg/dl, blood urea nitrogen: 42mg/ml, serum creatinine: 3.7mg/ml, Na: 132mmol/L, K: 4.2mmol/L, amylase: 20 U/L, PT>6 seconds, PTT>120 seconds, INR>36 ISI.

In the bedside ultrasonography of the patient with General Electric device through convex 2-5 MHz probe, in B-mode, abnormal IVC was seen in the left side of aorta (Figure 1). The patient was sent for venous color Doppler ultrasonography, which showed duplicated IVC (Figure 2). The blood flow was normal in both right and left IVCs, and they both entered the right atrium. Additionally, mild bilateral hy-

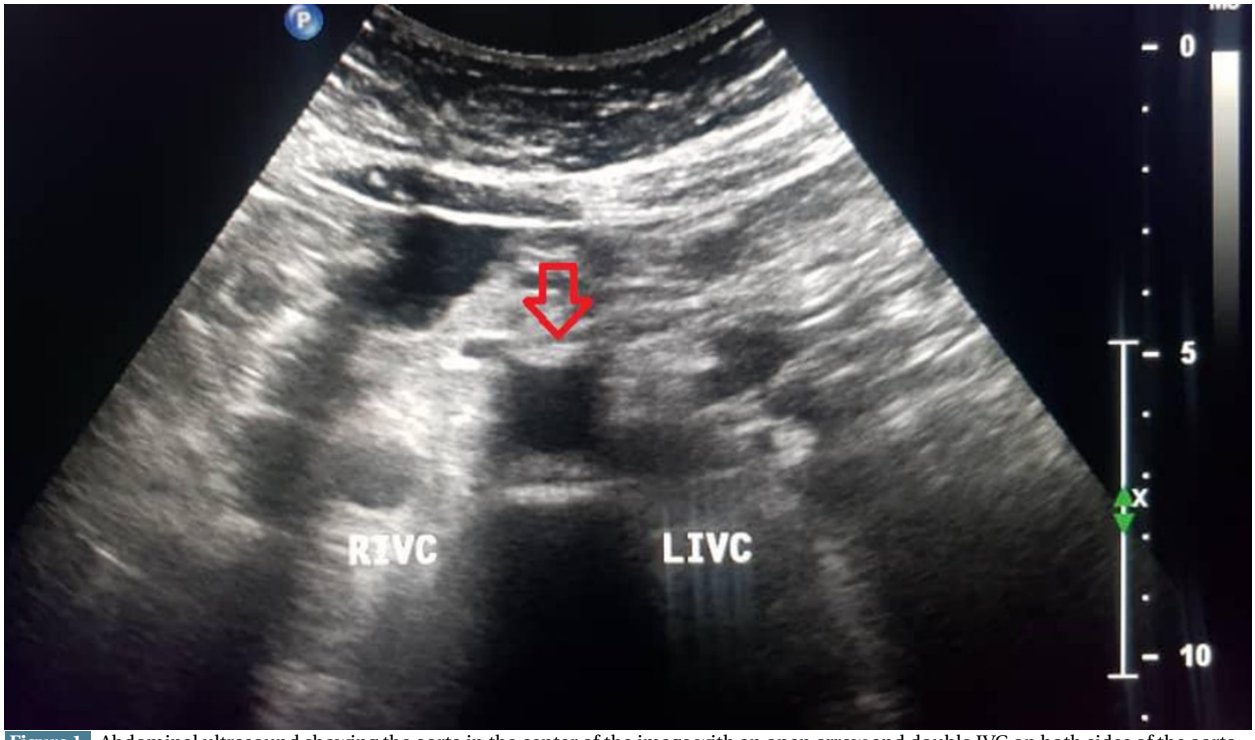

Figure 1 Abdominal ultrasound showing the aorta in the center of the image with an open arrow and double IVC on both sides of the aorta

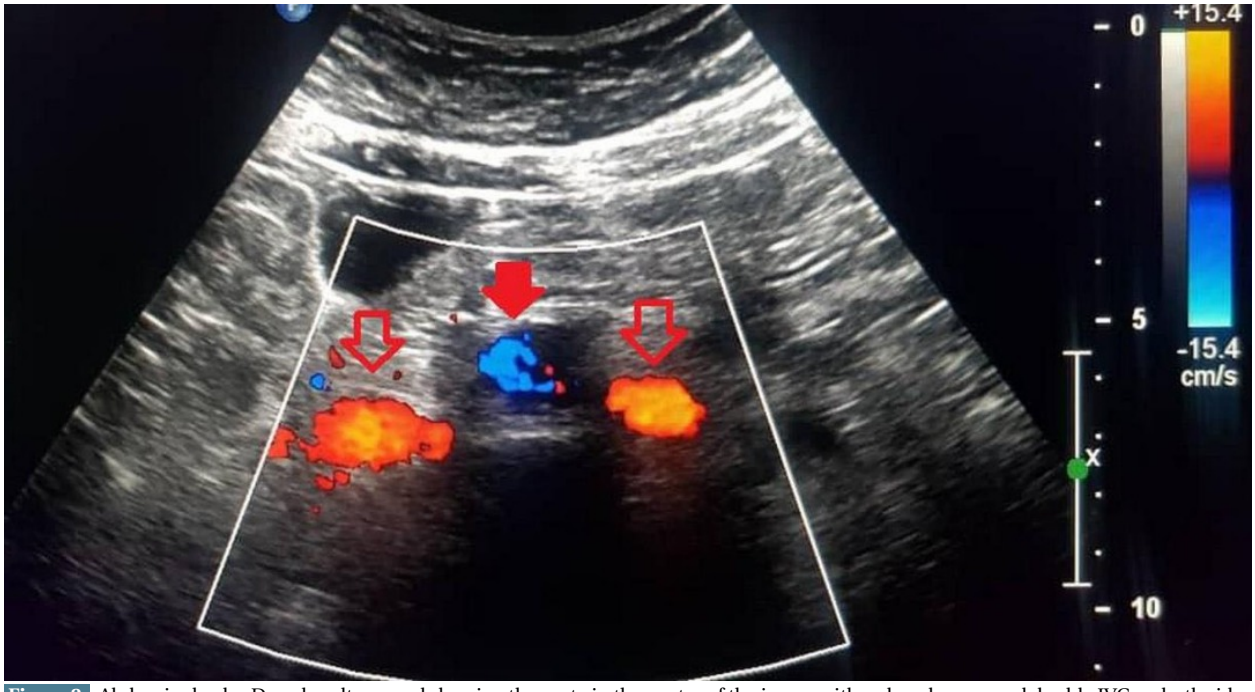

Figure 2 Abdominal color Doppler ultrasound showing the aorta in the center of the image with a closed arrow and double IVC on both sides of the aorta with two open arrows

droureteronephrosis was seen in the ultrasonography of the patient. For further evaluation of abdominal pain, the patient underwent an abdominal CT scan with only oral contrast, as the patient's creatinine level was high. Minimal free fluid was reported around both kidneys with insignificant pararenal fascia thickening, and duplication of IVC was reported. There was no evidence of urolithiasis in the abdominal CT scan (Figure 3).

The patient was admitted, warfarin was discontinued, and four units of fresh frozen plasma (FFP) were infused, and

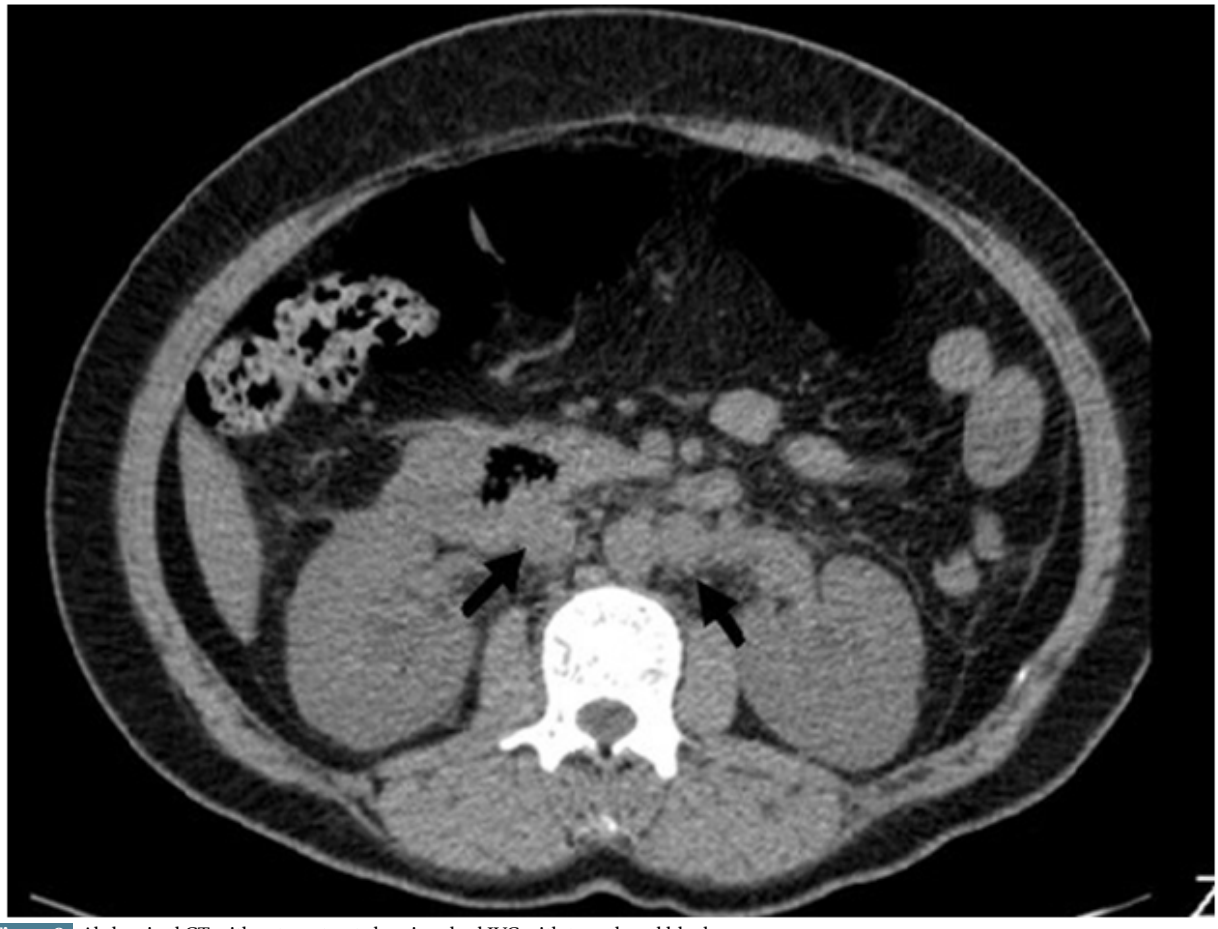

Figure 3 Abdominal CT without contrast showing dual IVC with two closed black arrows

the patient was hydrated with intravenous crystalloid. Upper endoscopy was performed, which was normal. Further evaluation was done, which showed elevated anti-cardiolipin immunoglobulin M (IgM) and immunoglobulin g (IgG). Her creatinine level decreased to 1.3 mg/ml over three days, and she was discharged by substituting warfarin with rivaroxaban.

3. Discussion

The reported case explains duplication of the inferior vena cava. The gold standard for diagnosing duplication of inferior vena cava is phlebography (6). However, as this is an invasive method, some authors have recommended that ultrasound with computed tomography (CT) scan is sufficient for making the diagnosis (7). In our case, the diagnosis was also made by these two modalities without phlebography. It is notable that misdiagnosis and needless intervention have been reported (8). Unfortunately, we could not precisely describe the anatomical variation of the veins. Many other case reports could not do this also as the most thorough descriptions are usually from studying anatomy by dissecting cadavers in medical schools (7). There is an argument that duplication of inferior vena cava may predispose patients to develop thrombosis (8). It seems that in the reported cases of deep vein thrombosis (DVT) with concomitant duplicated IVC, there has been a pre-existing disease such as cancer, history of abdominal surgery, lower limb fracture, using hormonal contraceptives, and a genetic tendency for thrombosis (7). Our patient did not have any predisposing factor for DVT and had not developed DVT in spite of having duplicated IVC similar to the cases without risk factors. Instead, there is a likelihood of connotation between DVT and other anomalies of the inferior vena cava (9). A case had been reported that duplication of IVC obstructed the ureter and caused hydronephrosis; in this case, hydronephrosis was moderate and unilateral (10). Our patient also had hydroureteronephrosis, but it was bilateral with mild severity. This is a rare complication of duplicated IVC, which was seen in our case as well. The absence of urolithiasis or any other pathology for ureteral obstruction in the abdominopelvic CT scan as the cause of hydronephrosis in our patient leads to the assumption that doubled IVC might be the cause of hydronephrosis in our patient.

4. Conclusion

Identifying duplication of IVC is important, as it can lead to confusion with aortic aneurysm, retroperitoneal cysts, and lymphadenopathy. According to what we have discussed, duplication of IVC should be considered as one of the differential diagnoses in patients with abdominal pain and as a rare cause of hydronephrosis in clinical practice. Double IVC might have a link with the patient's underlying antiphospholipid syndrome as well, but one case is not enough for such a conclusion.

5. Declarations

5.1. Acknowledgment

We would like to appreciate of the cooperation of Clinical Research Development Unit, Imam Reza General Hospital, Tabriz, Iran in conducting of this research.

5.2. Authors' contribution

HS, MB, RJR, ZP, NE and HE performed the clinical data collection, literature review, and drafting of the manuscript. All of the authors were involved in either managing the patients or writing the manuscript. All authors read and approved the final manuscript.

5.3. Conflict of interest

There is no conflict of interest.

5.4. Funding

None.

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