Anomalies of the inferior vena cava in patients with deep venous thrombosis. Pictorial essay.

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Abstract

The incidence of agenesis of inferior vena cava in the general population was estimated at 0.6 - 0.8%. In patients with deep vein thrombosis the frequency of agenesis was estimated between 5 and 6.7% in young people. In this pictorial essay, we present the ultrasound findings obtained in 8 patients with high deep venous thrombosis and inferior vena cava anomaly (absence of a segment), compared with images obtained by angio-computed tomography or magnetic resonance imaging. At the end we discuss the place of abdominal ultrasound in the evaluation of these patients.

Keywords: inferior vena cava, agenesis, deep venous thrombosis

Introduction

The annual incidence of deep vein thrombosis (DVT) associated or not with pulmonary thromboembolism (PTE), was estimated in the U.S. between 70 and 113 cases/100,000 inhabitants/year [1-6]. In Europe the incidence of DVT in general population was estimated at 160 cases /100,000 inhabitants /year in Malmo, Sweden [7], 182 cases /100,000 inhabitants /year in Göteborg, Sweden [8], 124 cases /100,000 inhabitants /year in Bretagne France [9]. The incidence of DVT and PTE in U.S. was higher in Caucasians and Afro-American subjects compared with Hispanic and Asian subjects [5].

The incidence of DVT and PTE increases with age [5,9]. Pathogenetic mechanisms involved in DVT are basically contained in the Virchow triad: venous endothelial damage, venous stasis, and hypercoagulable states (congenital or acquired). In a retrospective study on 1,230 patients with DVT or PTE, at least one risk factor

Received 29.12.2011 Accepted 20.01.2012 Med Ultrason 2012, Vol. 14, No 1, 53-59 Corresponding author: Sorin Pop 46F/8 Mircea Eliade str, 400364 Cluj Napoca, Romania Email: popsorin98@yahoo.com for thrombosis was identified in 96% patients [9].

Abnormalities of inferior vena cava (IVC) in the general population were described in a proportion of 0.2 -1% [11]. In patients under 30 years old presenting with DVT the incidence of IVC anomalies reach a rate of up to 5% [12,13]. In most of the cases the positive diagnosis was established by angio-computer tomography (angio-CT) [14, 5]. Ultrasound diagnosis is less used for detecting IVC anomalies and, according to some authors, usefulness in the diagnosis of ultrasound IVC anomalies is limited [16,17].

In this pictorial essay we aim to present some imagistic aspects, especially ultrasonographic aspects, of abnormalities of the IVC found in 8 patients with DVT.

Normal aspect of IVC

IVC arises from the reunion of the common iliac veins, lies on the right part of the spine, and has an upward trajectory parallel to the abdominal aorta. The main tributary veins are renal and the three hepatic veins. By ultrasound IVC is examined in transverse and longitudinal scans using gray scale and color Doppler imaging (fig 1). Color Doppler examination in the longitudinal plane provides the best information on IVC continuity



Fig 1. a) Normal IVC, longitudinal scan for identification of the venous segments: 1 – liver segment, 2 – prerenal segment, 3 – renal segment (compared with right renal artery), 4 – part of the infrarenal segment (caudal IVC); b) color Doppler examination, longitudinal scan of the postrenal (caudal, infrarenal) segment of IVC; c) underrib recurrent scan of the hepatic segment of IVC: L-left hepatic vein, M-medium hepatic vein, RA-right atrium.

[18]. The average diameter of IVC is 17.5 mm and, under normal conditions, does not exceed 25 mm [18].

From cranial-caudal direction IVC is embryologic formed from the liver segment (formed from the right viteline vein), prerenal segment (consisting of the union of the right subcardinale vein with the hepatic vein), renal segment (formed by supracardinale - right subcardinal veins anastomosis), postrenal/infrarenal segment (consisting of supracardinal vein), and iliac veins confluensa segment (formed by posterior cardinal veins) [14,19-21].

The right supracardinal vein forms the azygos vena, and the left supracardinale vena forms the hemiazygos vena [19]. Common iliac veins are embryologic formed from the caudal end of posterior cardinal vein [21].

Diagnosis of anomalies

The classification of IVC anomalies is based on the modifications of the final vein segment [21]. From the anomalies described by Mathews et al [21] we found atresia or interruption of the IVC in 8 patients with high DVT. In these cases, development of retroperitoneal collateral circulation (lumbar veins, azygos and hemiazygos vein) or abdominal wall collateral circulation (cavocaval collateral circulation) was associated

Ultrasound diagnosis of atresia or interruption of the IVC is based on two aspects:

 lack of continuity of the IVC segment (direct visualization of interruption);

- the presence of collateral venous circulation in the abdominal wall or retroperitoneal location (fig 2).

Venous collateral circulation develops for replacing the atresic or absent venous segment and can be easily demonstrated by color Doppler ultrasound (fig 3). Sometimes, in cases with IVC interruption or complex anomaly, venous collateral circulation can be found in relation to the visceral surface of the liver and upper pole of right kidney (fig 3-5). The venous collateral circulation can be achieved through several pathways, most commonly through retroperitoneal lumbar longitudinal veins continued cranial with azygos and hemiazygos veins (fig 6), veins that are finally ending in superior cava vein.

Collateral circulation is identified by ultrasonography based on the particular aspects of dilated, irregular venous trajectory with an unusual location. In figures 7 and 8 are depicted the color Doppler ultrasound findings compared with ango-CT or angio-MRI imagines. Note that both imaging methods, correctly identified the aberrant vein pathway, such as the abnormal continuation of right common iliac vein presented in fig 8.

In patients with interruption of the IVC, collateral venous circulation can be also realized through the abdominal wall veins (fig 9).

The collateral venous dilatation may have, in cases of complex anomaly of the ICV, large dimensions. For example the giant collateral venous dilatation with subhepatic location showed in figure 10 was initially interpreted as a possible arterial aneurysm.

One of our patient with absence of the postrenal IVC segment have, apart for sequelae of DVT, left renal vein partial thrombosis (fig 11).

The exact frequency of the IVC abnormalities in the general population has not been established. Grigorescu et al in 749 cadaver dissections establish an overall rate of 14.68% of IVC anomalies [22]. In 6 cases (0.8%) an abnormal IVC trunk was found, with interruption or absence of a segment of the IVC.

The absence of an IVC segment creates venous stasis in the lower limbs, venous drainage being difficult due to



Fig 2. Color Doppler ultrasound and angio-CT identification of the absent IVC segments: a) color Doppler ultrasound, longitudinal scan: patient with right deep vein thrombosis and absence of the postrenal segment of IVC (segment marked with arrows); the cranial segment of IVC is refill through a posterior collateral vein (asterisk); posterior to IVC the presence of collateral veins are noted; b) color Doppler ultrasound, longitudinal scan in a 12 years-old girl with bilateral lower limb deep vein thrombosis- absence of the IVC postrenal segment (arrows); c) color Doppler ultrasound, longitudinal scan: patient with bilateral deep vein thrombosis, absence of the IVC postrenal segment (between calipers), and venous collateral circulation (arrow); d) color Doppler examination, transverse scan of the distal abdominal aorta (Ao): patient with right leg deep vein thrombosis and absence of the IVC (normally situated in the right side of aorta -arrow). Asterisks - collateral venous circulation on the left side of the aorta; e). angio-CT examination, late venous time, three-dimensional reconstruction: patient with bilateral DVT sequelae and partial thrombosis of left renal vein: the complete absence of the IVC, postrenal segment, caudal to the right renal vein.



Fig 3. Color Doppler examination of the retroperitoneal collateral venous circulation, in the absence of a segment of the IVC: a) longitudinal left paraaortic scan: venous dilatation with irregular pathways, more likely to be lumbar longitudinal veins (the patient shown in fig 2c); b) slightly oblique scan in the left lower abdomen showing the venous collateral circulation in left paraaortic space - dilated veins with irregular pathway (patient from figure 2a).



Fig 4. Absence of the postrenal IVC with particular location of the collateral venous circulation, longitudinal scan of the right lobe of the liver (patient with prior bilateral deep vein thrombosis): a) gray scale ultrasound, dilated veins on the visceral liver surface (arrows); b) color Doppler ultrasound, emphasize the venous collateral circulation.



Fig 5. IVC interruption with collateral venous circulation, particular location: a) gray scale transverse scan in the right upper quadrant – "venous lakes" between the right lobe of the liver and upper pole of right kidney (patient shown in fig 2c); b) color Doppler ultrasound, transverse scan, right upper quadrant: venous collateral circulation in relation with right renal vein and liver; c) and d) patient with complex anomalies of IVC with drainage into the superior vena cava, azygos and hemizygos veins: venous collateral circulation localized under the liver.



Fig 6. Interruption of the IVC, renal and postrenal segments, with collateral circulation development through the azygos and hemiazygos veins, angio-CT examination, late venous time sections: a) transverse section, 1-normal hepatic segment of the IVC, 2-dilated azygos vein, 3-dilated hemiazygos vein; note the absence of IVC caudal to the hepatic segment, Ao- aorta; b) reconstruction in the coronal plane: 1-dilatated azygos and 2-hemiazygos veins.



Fig 7. Correlation between color Doppler ultrasound and angio-MRI examination in a patient with high right leg DVT, anomaly of the IVC formation, and absence of the postrenal IVC segment: a) and b) transvers scan in the distal portion of the abdominal aorta, highlights the absence of IVC and collateral venous circulation on both sides of the abdominal aorta (arrows); c) angio-MRI – IVC absent, paravertebral (asterixis) and paraaortic (arrow) collateral circulation.



Fig 8. Correlation between color Doppler ultrasound and angio-MRI, patient with high right leg DVT, anomaly of the IVC formation, and absence of the postrenal IVC segment: a) color Doppler oblique scan in right iliac fossa- common iliac vein (CIV) is interrupt and continue posteriorly with an aberrant venous paths (arrow); b) angio-MRI section of the aberrant venous path of the common iliac vein.

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Fig 9. Angio-MRI examination – collateral venous circulation in the abdominal wall in the patient showed in fig 7 and fig 8.



Fig 10. Left renal vein with partial thrombosis (arrow)-the same patient as in fig 2d.



Fig 11. Giant venous dilatation with subhepatic localization (the same patient as in fig 5c,d and fig 6).

the blood deviation through collateral venous pathways. The association between the absence of a segment of the IVC and DVT is described by several authors in a small number of cases [11-13,16,17,19,23-27]. Lamber et al reported 10 cases of DVT associated with IVC agenesis and identified 62 published cases until 2010 [16].

The reported cases had in common the higher location of thrombosis, younger age of onset (mostly under 40 years), and a male predominance (81.9%). Also, the authors found more frequently bilateral DVT and absence of pulmonary embolism (the thrombus could be ensnared into the collateral venous network) [16].

IVC agenesis incidence in young people (under 30 years old) with lower limb DVT was estimated to be between 5 and 6.7% [11,13].

In seven years, between 2004 and 2011, we have diagnosed 8 patients with high DVT associated with IVC agenesis, cases presented in this pictorial essay. Due to the lack of epidemiological data, we can not determine which is incidence of IVC anomalies in the patients with diagnosed DVT.

Angio-CT and angio-MRI are the imagistic methods that can provide the diagnosis [13,23- 27]. In all cited studies the authors consider that abdominal ultrasound have a minor contribution for IVC abnormalities assessment as comparison with angio-CT or angio-MRI [11-13,16,17,19,23-27]. Lambert et al states that ultrasound could not establish the diagnosis of agenesis of the IVC in any of the 10 patients studied [16].

In our cases the initial diagnosis of IVC anomaly was established by abdominal ultrasound. Complementary imaging confirmed the diagnosis, and could determine the exact location of collateral circulation. Ultrasound diagnostic criteria for IVC agenesis were those above mentioned. It should be emphasized that ultrasound can identify the collateral venous circulation, but can not specify exactly which veins are involved.

In conclusion, a segment of IVC agenesis is rare for patients with DVT. In young male people with DVT, IVC agenesis should be taking into account. In our opinion, the diagnosis of IVC anomaly can be suggest by ultrasound as the initial evaluation, but this diagnosis must be confirmed by angio-CT or angio-MRI.

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