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Case Report

Anomalous development of the inferior vena cava: Case reports of agenesis and hypoplasia

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ABSTRACT

We reported the cases of two adult male patients who were admitted to our emergency room with abdominal pain and dyspnea caused by gallstones and pulmonary embolism respectively. During the radiological investigations, as collateral findings, we found two anomalous development of the inferior vena cava. These conditions affect about 4% of population and, although asymptomatic or mildly symptomatic, are associated with thrombotic manifestations as deep vein thrombosis and pulmonary embolism. The prompt recognition of these anomalies is necessary in order to prevent the complications associated with these conditions and to set the best therapy for patients.

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1. Introduction

Anomalous embryologic development of the inferior vena cava (IVC) is a rare congenital abnormality that affects approximately 4% [1] of the population [2]. IVC agenesis is one of the less prevalent variants, with an approximate incidence from 0.005% to 1% [3].

These malformations can be unique or can be associated with other abnormalities such as situs inversus, dextrocardia, or polysplenia and asplenia [4].

Thrombotic manifestations such as deep vein thrombosis or pulmonary embolism (PE) are the most common clinical oc-

currence in these patients but usually this condition is completely asymptomatic.

We report a series of two cases of anomalous development of IVC in a patient with PE and in an asymptomatic patient.

2. Cases presentation

2.1. Patient #1

An abdominal magnetic resonance imaging (MRI) examination was performed to assess the diameter of the primary

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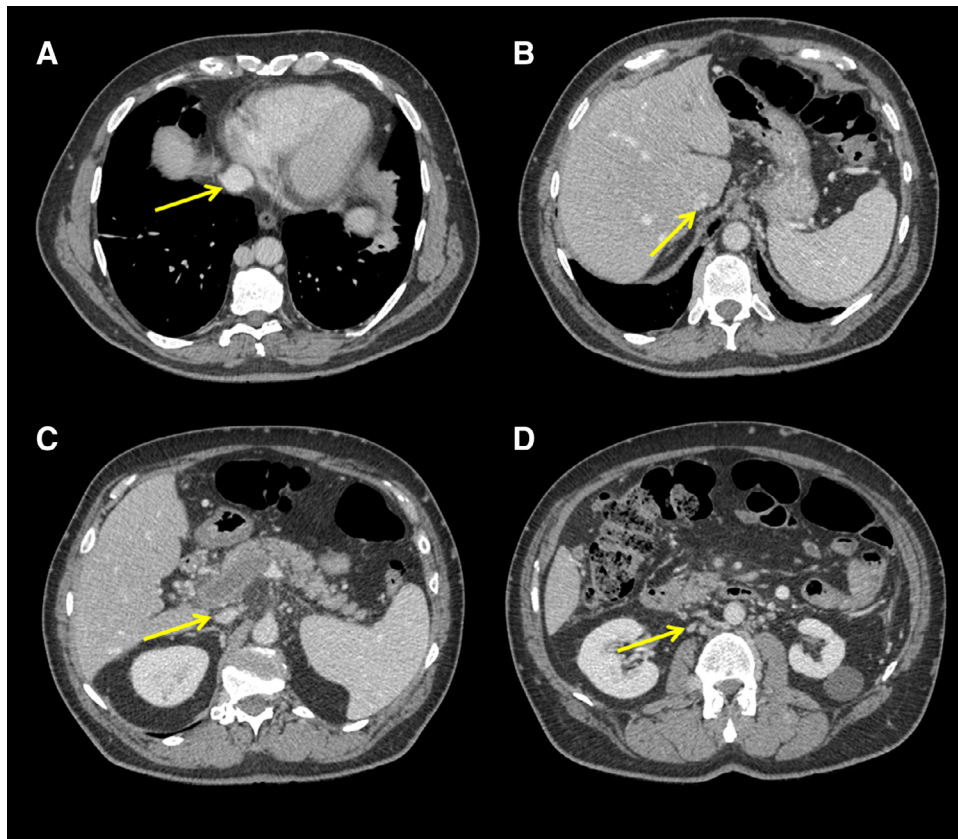


Fig. 1 – Computed tomography, portal phase, axial sections; (A) regular aspect of suprahepatic inferior vena cava (IVC) (yellow arrow); (B) intrahepatic IVC (yellow arrow); (C) reduction caliber of infrahepatic IVC (yellow arrow); (D) hypoplastic aspect of infrarenal IVC (yellow arrow). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

biliary duct. MRI was performed by an MR scan operating at 1.5 T (Intera 1.5 T; Philips Healthcare, Best, The Netherlands) with four channels phased array coil. The imaging protocol included a T2-weighted axial (with breath-hold, repetition time [TR] shortest, echo time [TE] 80 ms, slice thickness 7 mm, slice gap 1 mm), T2-weighted axial spectral presaturation with inversion recovery (TR shortest, TE 80 ms, slice thickness 7 mm, slice gap 1 mm), T1-weighted axial (TR shortest, TE 4.6 ms, slice thickness 8 mm, slice gap 1 mm), DUAL (spoiled gradient echo, within and out-phase echoes time; out of phase: TR shortest, TE 2.3 ms, slice thickness 5 mm, slice gap 1 mm; in-phase: TR shortest, TE 4.6 ms, slice thickness 5 mm, slice gap 1 mm), T2-balance on coronal plane (TR shortest, TE shortest, slice thickness 7 mm, slice gap 1 mm), T1-high resolution isotropic volume excitation (TR shortest, TE shortest, slice thickness 2 mm at 0 in, 30 in, 90 in, and 150 in after administration of contrast medium intravenous (Gadobenato dimeglumina 0.5 M; volume: 15 mL; flow rate: 2.5 mL/s). The examination was completed by a T1 post Gadolinium Contrast (Gd) (TR shortest, TE 4.6 ms, slice thickness 8 mm).

The examination confirmed the gallstone disease, showing a normal diameter of the primary biliary duct and, additional finding, IVC agenesis with dilated paravertebral vein system (Fig. 1).

In order to assess the structure and the diameter of the abdominal venous vessels a contrast-enhanced CT examination was performed by a 64-slice CT scanner (Lightspeed; General Electric Healthcare, Waukesha, WI). The protocol included a noncontrast CT scan, with 5 mm of thickness, and dynamic acquisition at 35 and 90 seconds after administration of iodine contrast medium (Iobitridol; volume: 120 mL; flow rate: 2.5 mL/s), slice thickness: 2.5 mm.

In addition to IVC agenesis and dilation of paravertebral vein system, CT showed deep venous ectasia and specifically of azygos and hemiazygos veins (Fig. 2), renal veins (Fig. 3), external iliac veins, pudendal veins, obturator veins, testicular veins, gluteal veins, and lumbar ascending veins (Fig. 4), caused by blood flow redistribution (Fig. 5).

2.2. Patient #2

A 65-year-old male was admitted to the Emergency Department for upper quadrants abdominal pain and dyspnea. Pulmonary embolism was suspected and a contrast-enhanced chest CT was performed.

At the moment of the CT examination blood laboratory tests showed: Creatinine: 0.9 mg/dL (normal value (nv): 0.7–1.3 mg/mL); Fibrinogen: 632.00 mg/dL (nv: 200–400 mg/dL), D-

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