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CORRESPONDENCE

Clues to the diagnosis of inferior vena cava agenesis in patients with deep venous thrombosis

Claves para el diagnóstico de la agenesia de la vena cava inferior en pacientes con trombosis venosa profunda

To the Editor,

The exact incidence of congenital anomalies of the inferior vena cava (IVC) in the general population has not been established, but is estimated to be close to 0.2–1%.[1] However, in a study involving 749 cadaver dissections *Grigorescu et al*. reported a 14.7% overall rate of IVC anomalies.[2] Such great disparity raises the question of whether these vascular anomalies may be underdiagnosed. IVC agenesis (IVCA) is a rare congenital abnormality in which venous return from the lower limbs is achieved through collateral veins. Most IVCA patients are asymptomatic, but they occasionally present with venous thromboembolic complications. In fact, the incidence of IVCA in persons younger than 30 years with lower limb deep venous thrombosis (DVT) is around 6% and congenital IVC malformations have been reported in 16.2% of young patients with iliac vein thrombosis.[1–4] Nonetheless, the information on this condition is scant, mainly consisting of case reports, also in the few spanish studies.[5,6] Identification of IVCA is important to avoid misdiagnoses, and is essential in preoperative planning of surgical and interventional radiology procedures. We analyzed the characteristics of a series of IVCA patients with acute symptomatic venous thromboembolism (VTE) attended in a university hospital, with the aim of determining clinical clues to establish the diagnosis.

Patients with acute symptomatic VTE and an imaging diagnosis of IVCA were identified in a retrospective review of the medical records of patients with VTE between 1980 and 2010. Six cases were retrieved; four were males (66.6%) and mean age at the diagnosis was 23.5 (18–30) years. All patients presented isolated DVT and no symptoms of pulmonary embolism. In all cases, iliac veins were affected, and five out the six (83.3%) patients had bilateral DVT. VTE was considered idiopathic in all but one patient, who was taking oral contraceptives. On physical examination, superficial collateral veins of the abdominal wall were detected in five (83.3%) cases. Four (66.6%) patients showed azygos vein dilation on chest radiography (Fig. 1). The diagnosis of IVCA was confirmed by computed tomography (CT), which disclosed involvement of the suprarenal IVC in all cases. No other vascular malformations were detected in any patient.

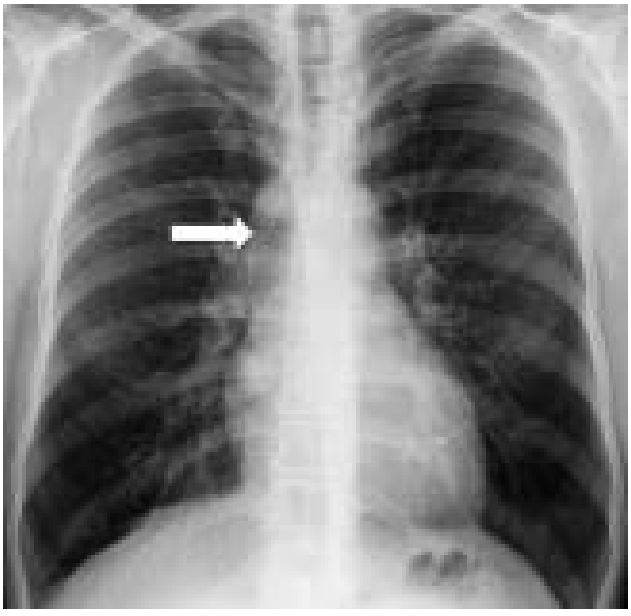

Figure 1 Chest radiography showing azygos vein dilation (white arrow).

Our findings are in accordance with previous studies in which most IVCA patients with DVT were males with bilateral DVT, and essentially, iliac veins were affected.[4,7] *Lambert et al*. described ten IVCA patients with DVT and carried out a literature review that included 62 additional patients, reported up to 2010. Most were isolated cases. DVT was bilateral in six out of ten patients reported by Lambert and in 35.4% of reviewed cases. Furthermore, in eight of Lambert's cases and 20.6% of cases from the literature, the patients had undertaken major physical exertion before the onset of DVT.[7] Unfortunately, data about this circumstance were not available in our patients.

In addition, we have identified other clues obtained from the clinical and basic imaging data to suspect the diagnosis of IVCA in patients with DVT. As occurred in our patients, IVCA usually affects the renal and post-renal segments, and induces the development of collateral circulation with dilated vessels.[7] There are various collateral pathways of

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venous return in IVCA, the main one being thorough the azygos and hemiazygos veins.[6] This pathway is particularly common when the suprarenal segment of the IVC is affected,[7,8] in accordance with the findings in our series. The related azygos vein dilation can usually be seen on chest radiography, as occurred in four out of six patients. In addition, a part of the collateral circulation may be detected on physical examination as superficial collateral veins of the abdominal wall, present in most of our patients. Despite presenting proximal DVT that is often bilateral, the venous return through a collateral network may make it difficult for clots to reach the pulmonary circulation. Hence, in patients with IVCA,symptoms of pulmonary embolism are uncommon.[7] When IVCA is suspected, CT is diagnostic of IVCA without the need for other examinations, and magnetic resonance imaging is also useful.[3,9,10]

In conclusion, IVCA should be suspected in young patients, particularly males, with unprovoked, usually bilateral DVT affecting the iliac veins and no evidence of pulmonary embolism. Superficial collateral abdominal veins on physical examination and azygos vein dilation on chest radiography may be other clues to the diagnosis of IVCA. CT examination in these patients will confirm the suspected diagnosis.

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Hematoma esofágico intramural y antiagregación plaquetaria

Intramural esophageal hematoma and platelet anti-aggregation

Sr. Director:

El hematoma esofágico es una enfermedad esofágica poco frecuente[1]. Entre sus factores predisponentes figuran la hipertensión portal, los procedimientos de escleroterapia esofágica, los tratamientos antiagregantes o anticoagulantes, los vómitos repetidos o la ingestión de cuerpos extraños[2]. Clínicamente suele presentarse como una tríada consistente en dolor torácico, odinofagia/disfagia y hematemesis, siendo poco habitual la aparición de la tríada completa[3]. En el diagnóstico diferencial se considerará el síndrome coronario agudo, la enfermedad aórtica, el tromboembolismo pulmonar, la úlcera péptica perforada y la pancreatitis. Las pruebas diagnósticas indicadas son la endoscopia digestiva oral y la tomografía axial computarizada (TAC) de tórax[3]. El tratamiento recomendado es conservador con dieta absoluta y medidas de soporte sintomático, presentando evolución favorable en la mayoría de los casos[4]. La cirugía estaría indicada ante sangrados masivos o perforación esofágica. El hematoma esofágico como efecto adverso al tratamiento antiagregante es sumamente infrecuente y hay pocos casos clínicos publicados[5].

Varón de 86 años con antecedentes de hipertensión arterial, diabetes mellitus tipo 2, dislipemia, hiperplasia benigna de próstata y temblor esencial, que mantenía una buena situación basal con independencia total para las actividades básicas de la vida diaria. Consultó por presentar dolor precordial opresivo, sin irradiación ni cortejo vegetativo de 48 h de evolución, de intensidad creciente y sin otra clínica asociada. Mantenía buen estado hemodinámico con aceptable estado general, bien hidratado y perfundido, con presión arterial (PA) 146/74 mmHg, FC 71 latidos por minuto (lpm). La auscultación cardiopulmonar mostraba tonos rítmicos a 70 lpm, sin soplos, y un murmullo vesicular normal con algunos crepitantes inspiratorios en