4. Ruiz-Hernández PM, Wangüemert-Pérez F. Sensibilidad y valor predictivo negativo de la ergometría para el diagnóstico de la taquicardia ventricular polimórfica catecolaminérgica. Rev Esp Cardiol. 2015;68:545–6.
5. D'Argenio V, Frisso G, Precone V, Boccia A, Fienga A, Pacileo G, et al. DNA sequence capture and next-generation sequencing for the molecular diagnosis of genetic cardiomyopathies. J Mol Diagn. 2014;16:32–44.
6. Jiménez-Jáimez J, Álvarez M, Algarra M, Macías-Ruíz R, Peñas R, Valverde F, et al. Baja penetrancia clínica en sujetos portadores de mutación patogénica para las canalopatías cardiacas. Rev Esp Cardiol. 2013;66:275–81.

http://dx.doi.org/10.1016/j.rec.2016.04.007

Unexpected Interrupted Inferior Vena Cava Diagnosed During Failed Transcatheter Left Atrial Appendage Closure

Diagnóstico inesperado de una interrupción de la vena cava inferior durante el fallo de un cierre percutáneo de la orejuela auricular izquierda

To the Editor,

An 88-year-old man with a history of biological aortic valve replacement, pacemaker implantation and chronic atrial fibrillation with thromboembolic protection (dabigatran 110 mg x 2) was admitted for serious intestinal bleeding. Bowel angiodysplasia was diagnosed and the anticoagulation therapy was discontinued. The patient was scheduled to undergo endovascular left atrial appendage (LAA) closure with the Amplatzer Amulet device (St Jude Medical)[1] given that the patient had a score of 4 on both the HAS-BLED and CHA_2DS_2-VASc scales. After sheath insertion in the right femoral vein, a 0.035″ conventional J-tip wire repeatedly crossed to the left side of the spine. Contrast media injection confirmed the absence of a right inferior vena cava (IVC) and showed a left IVC combined with a hemiazygos continuation (Figure 1), confirmed by computed tomography angiography (Figure 2). Consequently, the procedure was aborted and finally cancelled. Of note, the transgastric IVC long-axis view by transesophageal echocardiography showed the hepatic veins and strongly suggested an IVC interruption (Figure 1).

All isolated congenital variations of the IVC are a consequence of abnormal embryologic development, affecting approximately

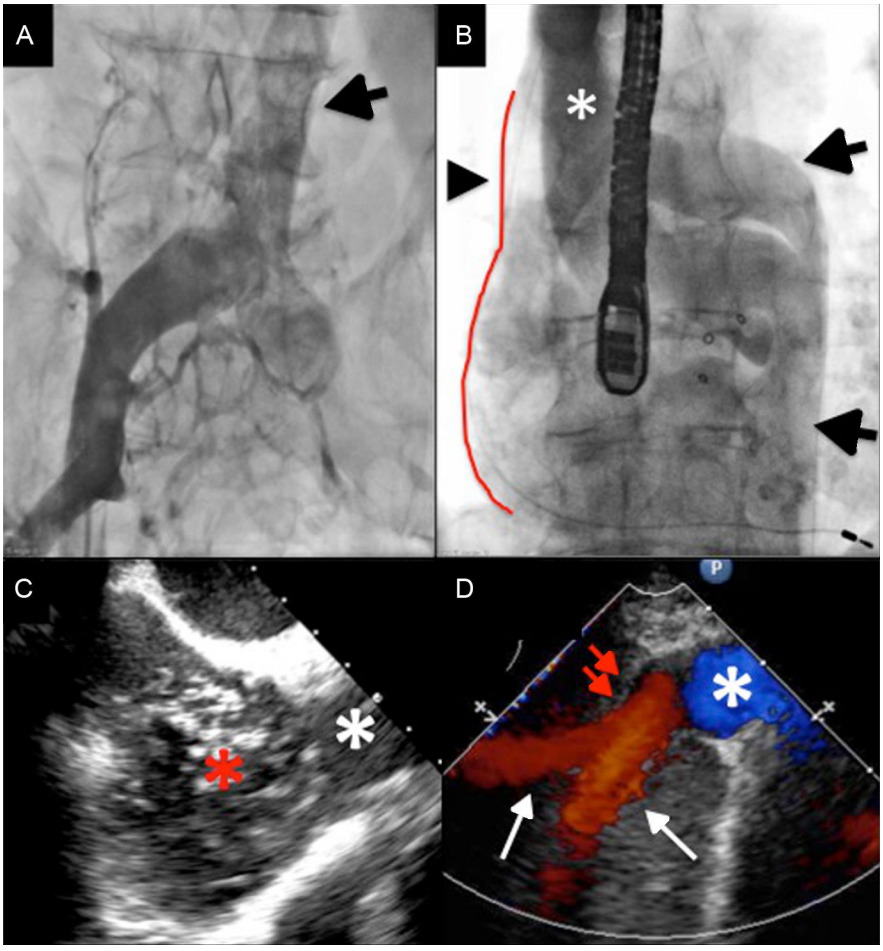

Figure 1. A: Venography showing a left IVC (arrow) and the absence of the right IVC. B: Hemiazygos continuation (2 arrows) and, due to the azygos cross (asterisk), the inferior systemic venous return enters the superior vena cava (arrow head and limited by the red line). C: Transgastric IVC long-axis view by transesophageal echocardiography. Superior vena cava (white star); microbubbles in the left atrium coming from the superior vena cava after injection in the left IVC (red star). D: IVC (white star), hepatic veins (white arrows); IVC interruption (red arrows). IVC, inferior vena cava.

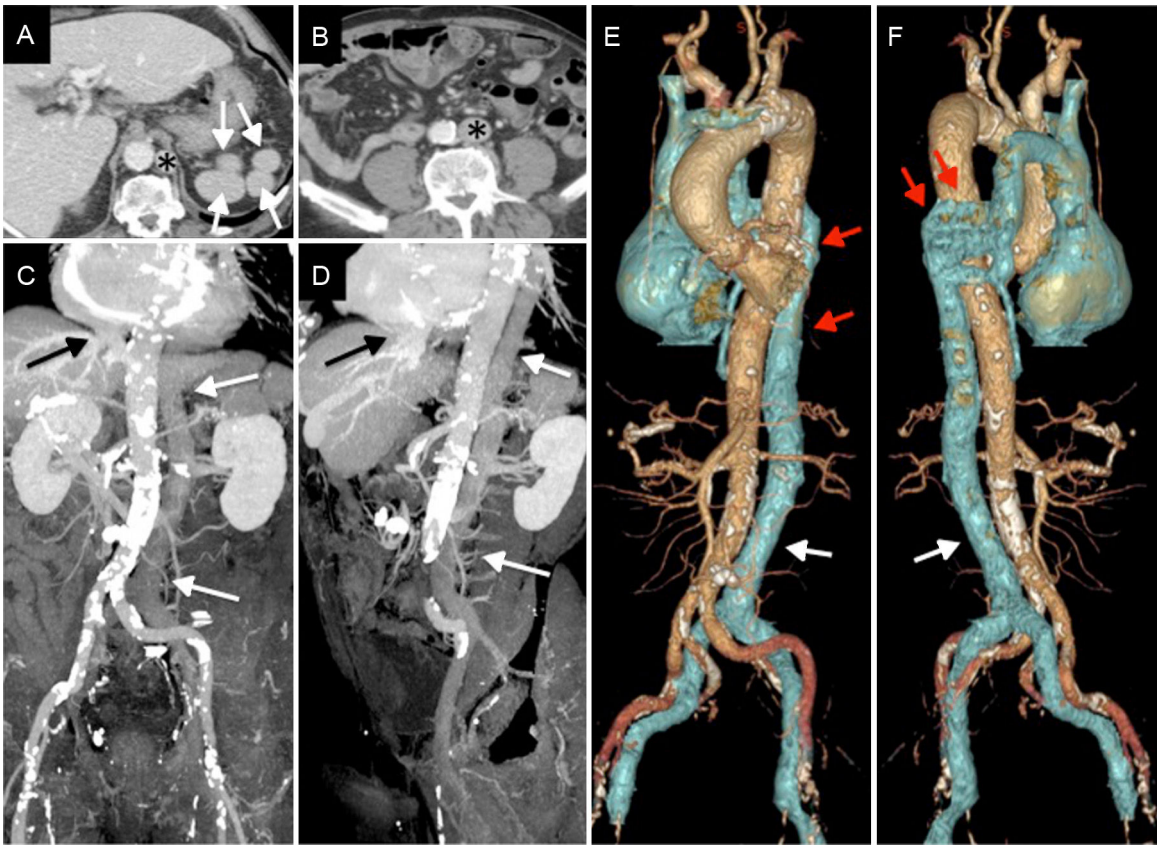

Figure 2. Computed tomography angiography. A: Polysplenia (arrows), hypertrophy of the hemiazygos vein (asterisk). B: Absence of IVC on the right and left position of the IVC (asterisk). Coronal (C) and sagittal reconstructions (D) showing the interruption of IVC (black arrow) with hemiazygos continuation and left-sided IVC (white arrows). E and F: Volumetric rendering images showing the abnormal systemic venous return from the lower body with a left-sided IVC (white arrow) and a hemiazygos continuation (red arrow). IVC, inferior vena cava.

4% of the population.[2] Because of the wide variety of anomalous persistence, regression, or anastomoses between vitelline, posterior cardinal, subcardinal, and supracardinal veins, several congenital abnormalities may exist alone or in combination.[2] The most frequent are double IVC (0.2%-3%), azygos and hemiazygos continuation of the IVC (0.6%), and left-sided IVC joining the left renal vein (0.2%-0.5%).[2,3] Indeed, more than 1 anomaly can coexist, such as in our patient, in whom the left-sided IVC was combined with an interrupted right IVC and hemiazygos continuation. These 2 rare combined abnormalities resulted in an abnormal lower systemic venous return via the azygos venous system to the superior vena cava. In addition to the abnormal venous return, polysplenia (Figure 2) was diagnosed and enabled the complex diagnosis of left isomerism syndrome. Indeed, azygos and hemiazygos continuation of the IVC has been associated with situs anomalies, asplenia or polysplenia, and congenial heart malformations.[3,4]

The consequence of our patient's venous anomalies was a circuitous and abnormally long trajectory to the heart from the femoral approach, and thus the failure of endovascular LAA closure. In such anatomies or in the absence of patent IVC, transjugular or subclavian approaches may be challenging alternatives considering the transseptal puncture and LAA cannulation. Other approaches such as thoracoscopic appendage exclusion may also be considered, but our 88-year-old patient refused any further procedures.[5] Of note, in a similar anatomy, only 1 successful right-sided catheter ablation has been described in a child using the transfemoral approach.[6]

Although often asymptomatic, IVC congenital variations are an important condition to recognize before or during any endovascular procedure such as structural interventions (eg, MItraClip, LAA closure), electrophysiological interventions or IVC filter insertion.

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REFERENCES

1. Cruz-González I, Arzamendi D, Rama-Merchan JC, Piña-González P, Sánchez PL, Serra A. Left Atrial Appendage Occlusion With the New Amulet™ Device: Feasibility, Safety and Short-term Efficacy. Rev Esp Cardiol. 2015;68:724–6.
2. Smillie RP, Shetty M, Boyer AC, Madrazo B, Jafri SZ. Imaging Evaluation of the Inferior Vena Cava. Radiographics. 2015;35:578–92.
3. Malaki M, Willis AP, Jones RG. Congenital anomalies of the inferior vena cava. Clin Radiol. 2012;67:165–71.
4. Rameshbabu CS, Gupta KK, Qasim M, Gupta OP. Heterotaxy Polysplenia Syndrome In An Adult With Unique Vascular Anomalies: Case Report With Review Of Literature. J Radiol Case Rep. 2015;9:22–37.

5. Benussi S, Mazzone P, Maccabelli G, Vergara P, Grimaldi A, Pozzoli A, et al. Thoracoscopic appendage exclusion with an atriclip device as a solo treatment for focal atrial tachycardia. Circulation. 2011;123:1575–8.
6. Kilic A, Amasyali B, Kose S, Aytemir K, Kursaklioglu H, Lenk MK. Successful Catheter Ablation of a Right-Sided Accessory Pathway in a Child With Interruption of the Inferior Vena Cava and Azygos Continuation. Int Heart J. 2005;46:537–41.

http://dx.doi.org/10.1016/j.rec.2016.04.008

Usefulness of Reciprocal Changes in the Diagnosis of Myocardial Infarction With Minimal ST-segment Elevation

Utilidad de las alteraciones especulares en el diagnóstico del infarto con elevación mínima del segmento ST

To the Editor,

Minimal ST-elevation often leads to inappropriate diagnoses and delayed interventions. Recent studies have shown that between 11% and 23% of infarctions do not reach the cutoff for accepted electrocardiographic criteria for infarction, and the absence of such criteria is not associated with a more favorable prognosis.[1,2] The main objective of this study was to analyze the prevalence of reciprocal changes (RC) as a diagnostic tool in myocardial infarction with minimal ST-elevation.

The study was based on a prospective registry of 480 consecutive patients with a definitive diagnosis of infarction who underwent emergency coronary angiography as part of a systematic primary angioplasty program between 2009 and 2011. The present study comprised 75 patients with a maximum ST-elevation of 0.01 to 0.1 mV and who could be assessed for RC. The indication for catheterization was based on persistent symptoms of ischemia. No patient received thrombolysis during the study period.

Reciprocal changes were defined as J point depression $\geq$ 0.05 mV in the TP segment, in at least 1 lead other than the aVR lead. All patients provided informed consent prior to participation.

Variables were compared using the chi-square test, the Fisher exact test, and the Mann-Whitney *U* test. The variables associated with RC with $P < .1$ (age, site, multivessel disease) were included in the multivariate logistic regression model to assess whether they were independently associated.

In total, 51 patients had RC (prevalence 68%, 95% confidence interval [95%CI], 57%-79%), which was attributed to an ischemic cause in all of them. In 27 patients, a depression $\geq$ 0.1 mV was observed and, in all patients except 1, the slope was horizontal or decreasing.

Among the 24 remaining patients, 17 showed an ST-depression of 0.01-0.04 mV (n = 15) or negative or symmetric T waves (n = 10). Overall, 68 patients had some sort of RC that supported diagnosis (91%; 95%CI, 84%-97%) (Figure).

The Table shows the characteristics of the groups according to the presence of RC. In all patients, initial thrombolysis in myocardial infarction (TIMI) flow was 0/1 or creatinine kinase was elevated to more than 3 times the upper limit of normal. Although the enzyme concentrations were similar, patients with RC showed a tendency toward a higher incidence of heart failure during hospitalization (31% vs 12%; P = .080).

In the analysis by infarction site, the prevalence of RC was 38% (95%CI, 8%-69%) in anterior acute myocardial infarction, 70% (95%CI 35%-100%) in lateral or inferobasal infarction, and 75% (95%CI, 63%-87%) in inferior infarction (P = .041). In the multivariate analysis, nonanterior site was independently associated with the presence of RC (odds ratio = 4.6; 95%CI, 1.3-16.1; P = .017).

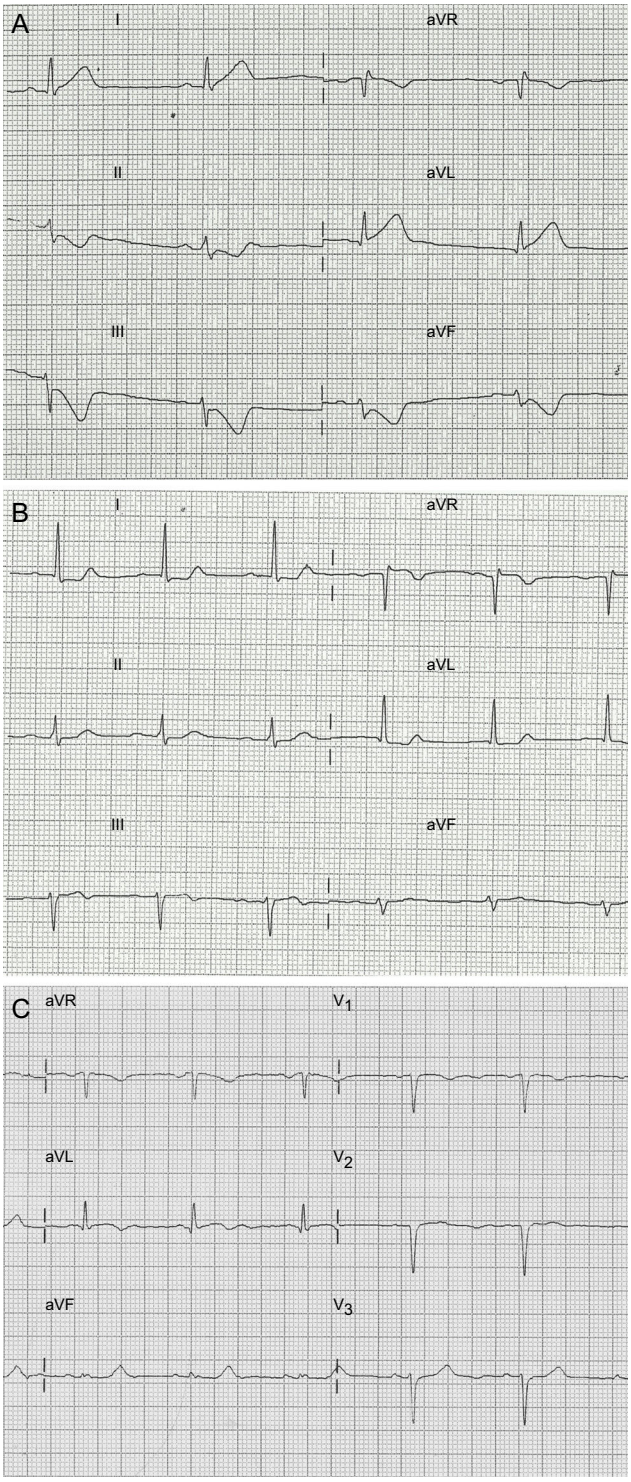

Figure. Electrocardiographic patterns of patients included in the study. A: Reciprocal ST-segment depression > 0.1 mV. B: Reciprocal depression of 0.05 mV. C: Reciprocal depression < 0.05 mV and symmetric T wave change.