Clinical Case Reports and Reviews

Case Report

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Dual-chamber ICD-CRT implantation in a patient with persistent left superior vena cava: testing skills

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Case report

A persistent left-superior vena cava (PLSVC) is an uncommon identification with a form of 0.3% to 0.5% of persons in the common population [1-5]. Nevertheless, it is the most common thoracic venous anomaly [6-10]. Typically, the left superior vena cava fades post embryological developement. The identification can be missed by the manifestation of a standard right superior vena cava. This subject did not have an ordinary or rest of a right superior vena cava. Furthermost of the individuals do not present the symptoms, and the presence of the persistent left superior vena cava is by the way found during or after insertion of a central venous catheter (CVC) or pacemaker electrodes. The correct report of a PLSVC and lack of a right superior vena cava has significant clinical repercussions in definite circumstances, such as oncological therapy, totally implantable vessels catheters, hemodynamic checking in intenive care unit (ICU) or the correct location of pacemakers [1-10]. The further clinical relevance of the described anomaly could be due to common tachyarrhythmia and conduction disturbances [11-15]. The PLSVC usually descends vertically, anterior, and to the left of the aortic arch and main pulmonary artery. It runs adjacent to the left atrium (LA) before turning medially, piercing the pericardium to run in the posterior atrioventricular (AV) groove [16]. In about 90% of cases, it drains into the coronary sinus (CS); alternative sites include the inferior vena cava, hepatic vein, and LA. The entry into LA is invariably associated with an atrial septal defect ASD [17,18].

In this case, we describe a female patient, 63 years old, with hypertension and dilated cardiomyopathy, without coronary artery disease. She was recovered from sudden cardiac death, with previous events of syncope, dyspnea on habitual exertion, and pre-syncope that began 6 months ago. She also was in use of acetylsalicylic acid 100 daily, carvedilol 25 twice a day, digoxin 0.25 mg per day, furosemide 40 mg daily, atorvastatin 40 mg daily, spironolactone 50 mg daily. The basal eletrocardiogram (ECG) presented sinus rhythm and QRS complex duration 170 ms. The 24-hour Holter monitoring showed sinus rhythm, with minimum – average – maximum heart rate (HR) of 39, 56 and 93 bpm, respectively, as well as, 6737 polymorphic ventricular ectopic beats and 5 episodes of non-sustained ventricular tachycardya, being the highest composed 16 beats at 180 bpm. The transthoracic echocardiogram showed: LA 4,3 cm, LVED 6,3 cm, LVES 5,8 cm, LVEF 17,2%, left ventricular mass index 139,3 g/m^2, andd diffuse hypokinesia of the left ventricle. The coronary angiography did not present any new obstruction.

The patient was submitted to general anesthesia by an anesthesiologist, and 2 g of cefazolin was administered intravenously. During the surery, a persistent left superior vena cava (PLSVC) was perceived. The left venography revealed a lack of contrast filling in an innominate vein (IV) and a quadripolar diagnostic catheter of electrophysiology within the CS introduced into the right femoral vein, as shown in Figure 1A and B, respectively. Post several efforts we succeeded in placing the "double-coil" shock lead into the right cephalic vein by dissection, then through the IV and right superior vena cava into the apex of the right ventricle (Figure 1C). After a double puncture of the right axillar vein, by one of them, a long sheath was positioned faced to the CS ostium, and the contrast was injected non-selectively filling this structure, showing how big was the CS in this case (Figure 1D). Subsequently, the angiography of coronary arteries was performed, the quadripolar diagnostic catheter was moved to the right outflow tract, and the long sheath was fully inserted into the CS (Figure 1E), then it was pulled back and selective injection of contrast was done with the aid of a Swan-Ganz catheter (Figure 1F). The guide wire was positioned inside the lower-posterior vein, and the quadripolar diagnostic catheter was pulled back to be removed (Figure 1G). A bipolar CS lead was placed into the vein selected, the "double-coil" shock lead was maintained in the same position, the atrial lead was inserted via the other right axillar puncture being actively fixed in the upper right atrium wall, the leads were fixed in the right pectoral muscle and connected to an implantable cardioverter defibrillator with cardiac resynchronization therapy (ICD-CRT), as shown in Figure 1H.

The following devices parameters were measured at the end of the procedure:

Leads	Sense (mV)	Impedance (Ω)	Threshold (V) @ 0.5ms
Atrial	P = 2.8	502	0.50
Right ventricular	Right R = 16.2	624	0.50
Left ventricular	Left R = 19.0	711	0.75

The biventricular pace measured was 129 ms. After 48 hours the patient was discharged, using the same medications; no AF episodes were recorded by the ICD+CRT, and this one presented normal parameters. Until the present time of follow up (1 month), the patient presented improvement of the symptoms, without arrhythmic events, and keeping the same parameters of the moment of the implant

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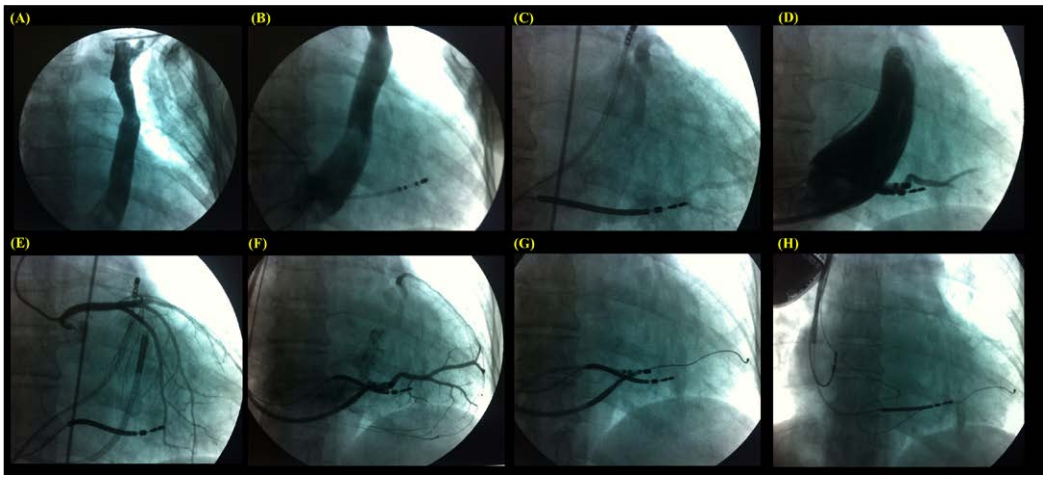

Figure 1. The left venography revealed a lack of contrast filling in an innominate vein (IV) and a quadripolar diagnostic catheter of electrophysiology within the coronary sinus (CS), A and B respectively. The "double-coil" shock lead was positioned in the apex of the right ventricle (C). Long sheath positioned faced to the CS ostium, and the contrast was injected non-selectively filling this structure (D). Subsequently, the angiography of coronary arteries was performed (E), and selective injection of contrast was done with the aid of a Swan-Ganz catheter (F). The guide wire was positioned inside the lower-posterior vein (G). A bipolar CS lead was placed into the vessel selected, the "double-coil" shock lead was maintained in the same position, the atrial lead was actively fixed in the upper right atrium wall, and connected to the implantable cardioverter defibrillator with cardiac resynchronization therapy (ICD-CRT) device (H).

References

1. Noheria A, Deshmukh A, Asirvatham SJ (2015) Ablating Premature Ventricular Complexes: Justification, Techniques, and Outcomes. *Methodist Debakey Cardiovasc J* 11: 109-120. [Crossref]
2. Kennedy HL, Whitlock JA, Sprague MK, Kennedy LJ, Buckingham TA, et al. (1985) Long-term follow-up of asymptomatic healthy subjects with frequent and complex ventricular ectopy. *N Engl J Med* 312: 193-197. [Crossref]
3. Agarwal SK, Heiss G, Rautaharju PM, Shahar E, Massing MW, et al. (2010) Premature ventricular complexes and the risk of incident stroke: the Atherosclerosis Risk In Communities (ARIC) Study. *Stroke* 41: 588-593. [Crossref]
4. Ataklte F, Erqou S, Laukkanen J, Kaptoge S (2013) Meta-analysis of ventricular premature complexes and their relation to cardiac mortality in general populations. *Am J Cardiol* 112: 1263-1270. [Crossref]
5. Nogami A (2011) Purkinje-related arrhythmias part I: monomorphic ventricular tachycardias. *Pacing Clin Electrophysiol* 34: 624-650. [CrossRef]
6. Nogami A, Naito S, Tada H, et al. Demonstration of diastolic and presystolic Purkinje potentials as critical potentials in a macroreentry circuit of verapamil-sensitive idiopathic left ventricular tachycardia. *J Am Coll Cardiol* 36: 811-823. [Crossref]
7. Scheinman MM (2009) Role of the His-Purkinje system in the genesis of cardiac arrhythmia. *Heart Rhythm* 6: 1050-1058. [CrossRef]
8. Adams JC, Srivathsan K, Shen WK (2012) Advances in management of premature ventricular contractions. *J Interv Card Electrophysiol* 35: 137-149. [CrossRef]
9. Lee GK, Klarich KW, Grogan M, Cha YM (2012) Premature ventricular contraction-induced cardiomyopathy: a treatable condition. *Circ Arrhythm Electrophysiol* 5: 229-236. [Crossref]
10. Kim RJ, Iwai S, Markowitz SM, Shah BK, Stein KM, et al. (2007) Clinical and electrophysiological spectrum of idiopathic ventricular outflow tract arrhythmias. *J Am Coll Cardiol* 49: 2035-2043. [Crossref]
11. Noda T, Shimizu W, Taguchi A, Aiba T, Satomi K, et al. (2005) Malignant entity of idiopathic ventricular fibrillation and polymorphic ventricular tachycardia initiated by premature extrasystoles originating from the right ventricular outflow tract. *J Am Coll Cardiol* 46: 1288-1294. [Crossref]
12. Santoro F, Biase LD, Hranitzky P, et al. Ventricular fibrillation triggered by PVCs from papillary muscles: clinical features and ablation. *J Cardiovasc Electrophysiol* 25: 1158-1164. [Crossref]
13. McLure HA, Rubin AP (2005) Review of local anaesthetic agents. *Minerva Anestesiol* 71: 59-74. [CrossRef]
14. Kiuchi MG, Zapata-Sudo G, Trachez MM, Ririe D, Sudo RT (2011) The influence of age on bupivacaine cardiotoxicity. *Anesth Analg* 112: 574-580. [CrossRef]
15. Becker DE, Reed KL (2012) Local Anesthetics: Review of Pharmacological Considerations. *Anesth Prog* 59: 90-102. [Crossref]