

CASE REPORT OF PACEMAKER IMPLANTATION IN A PATIENT WITH ANOMALOUS DRAINAGE OF THE SUPERIOR VENA CAVA

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ABSTRACT

Background and objective: Persistent left superior vena cava (PLSVC) is a rare embryologic remnant anomaly. Mostly both superior vena cava coexist, however if there is Agenesis of the Right Superior Vena Cava (ARSVC) the venous drainage to the heart will be made to the right atrium, through the coronary sinus. Usually asymptomatic, this malformation can be detected when patients undergo procedures involving the superior vena cava. This is a case report of a patient with PLSVC and ARSVC who underwent pacemaker implantation with greater technical difficulties due to lack of preparation for the anatomical anomaly, as well as the difficulty in handling sedoanalgesia. **Case report:** Male patient, 65 years old, with Coronary Disease, 5 years before undergoing myocardial revascularization, with 1st degree atrioventricular block (AVB). Admitted for permanent cardiac pacemaker implantation. He was admitted to the Surgical Center and received monitoring, venipuncture with a 20 G catheter, and sedoanalgesia with fentanyl and propofol was initiated. Venous puncture was performed in the right subclavian vein, but when checking the position of the guide wire, an anomalous ventricular pathway was visualized. After multiple repositioning attempts, the anomalous intraventricular pathway persisted. During manipulation, the patient presented supraventricular extrasystoles and increased demand for sedatives. When revisiting preoperative exams and found Angiotomography of the Coronary Arteries with a report of "probable PLSVC and ARSVC with anomalous drainage into the coronary venous sinus".

KEYWORDS: PERSISTENT LEFT SUPERIOR VENA CAVA; ARTIFICIAL PACEMAKER; CONSCIOUS SEDATION

INTRODUCTION

The persistence of the left superior vena cava (PLSVC) is a rare and sparsely described embryological remnant anomaly in the literature. Its incidence varies from 0.1 to 0.3%¹ in healthy adults to 4.3% in patients with congenital heart diseases². Mostly, both superior vena cavae coexist, but if there is embryological regression and degeneration of the right anterior cardinal vein, it will result in the absence of the right superior vena cava, and venous drainage to the heart may be carried out by the left superior vena cava into the right atrium through the coronary sinus². Due to the frequency of asymptomatic carriers, this malformation is often incidentally detected when patients undergo central venous catheter placement, pacemaker implantation, or open-heart surgery³.

The embryological venous system has, as its essential structures, the cardinal veins. The upper and lower cardinal veins combine to form the duct of Cuvier, which drains into the bicarotid venous sinus and develops into the right atrium. The duct of Cuvier, along with the caudal part of the right superior cardinal vein, will form the right superior vena cava (RSVC), while the left common cardinal

vein and the caudal part of the left superior cardinal vein will regress³. If this regression does not occur, it results in PLSVC³. One hypothesis to justify this event involves various embryological conditions leading to a reduction in the size of the left atrium. Consequently, it will not have the necessary dimensions to compress the coronary sinus and the left cardinal vein, culminating in PLSVC³.

This is the report of a case of a patient with PLSVC undergoing pacemaker implantation due to atrioventricular block (AVB), with the discovery of agenesis of the right superior vena cava (ARSVC), resulting in greater technical and analgesic management challenges.

CASE REPORT

Male patient, 65 years old, with a history of coronary artery disease, underwent myocardial revascularization 5 years ago, and currently has first-degree atrioventricular block (AVB) while on aspirin 100 mg/day and enalapril 20 mg/day. The patient was admitted for the implantation of a permanent cardiac pacemaker. In the pre-anesthetic evaluation, a complete blood count, renal function tests, 12-lead electrocardiogram showing first-degree AVB, and

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transthoracic echocardiography revealing concentric ventricular hypertrophy with preserved ventricular function were assessed. The chosen anesthetic technique was light sedation and local anesthesia. Upon entering the operating room, the patient was monitored with pulse oximetry, non-invasive blood pressure, and cardiac monitoring. Cardiac monitoring showed the presence of first-degree AVB with a heart rate of 42 beats per minute, maintaining adequate capillary filling time and blood pressure. A 20 G catheter was inserted for venous access, and sedation was initiated with a bolus of 50 mcg fentanyl and continuous infusion of Propofol using the Schnider model, with a target concentration of 1.0 microgram/ml. After sedation, local anesthesia with 100 milligrams of lidocaine was administered, followed by a right infraclavicular incision and venous puncture under direct visualization of the right subclavian vein. During the advancement of the guide wire, there was no resistance. However, when checking the position of the guide wire through radiographic images, an anomalous ventricular trajectory was visualized (Figure 01).

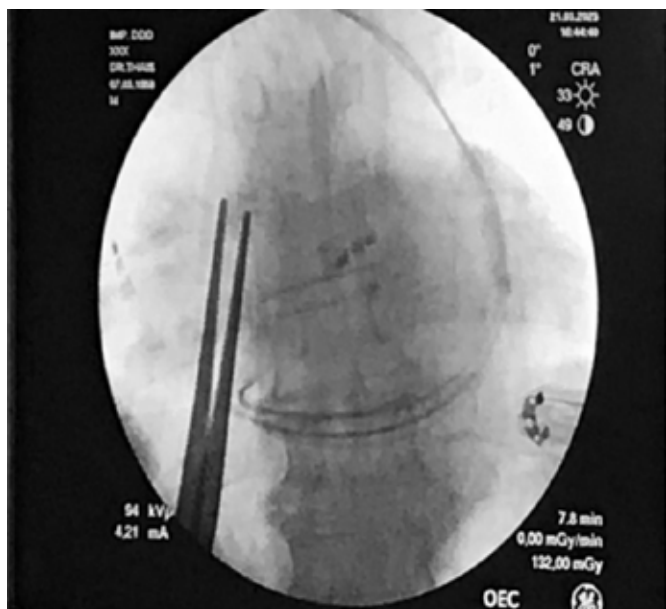


Figure 01: radiological image taken using a surgical arch showing the positioning of the electrodes.

After multiple attempts to reintroduce it into the proper position, the decision was made to change the puncture site. Ultrasound-guided venous puncture was performed in the left subclavian vein. During the introduction of the guide wire, resistance was encountered. Upon checking the X-ray, it was observed that even with the change in the puncture site, the anomalous intraventricular trajectory persisted. During the frequent manipulations, the patient experienced multiple episodes of supraventricular extrasystoles due to the introduction and reintroduction of in-

tracavitary wires, in addition to an increased demand for sedatives to maintain adequate sedoanalgesia, considering the prolonged painful stimulus and time.

The responsible cardiologist decided to revisit preoperative exams and found coronary artery angiogram (figure 02) with a report of "probable ARSVC agenesis and PLSVC with anomalous drainage into the coronary sinus." Keeping in mind the diagnosis of PLSVC, they resumed the procedure at the initial site and repositioned the electrodes, requiring multiple stimulus tests to find the optimal positioning for both electrodes and, consequently, for the pacemaker.



Figure 02: Three-dimensional representation from coronary angiogram of the patient's heart in a right posterolateral view shows the enlarged coronary sinus (blue arrow) above the right coronary artery (red arrow) and right atrium (white arrow), with the right superior vena cava not visualized in this view.

DISCUSSION

PLSVC can be relatively common, as it is the most frequent among thoracic venous anomalies. While PLSVC is mostly isolated, its coexistence with ARSVC constitutes a rare congenital venous malformation⁵. In a normal adult, about one-third of venous return is through the superior vena cava, and in cases of obstruction, venous content will flow through collateral circulation to the lower part of the body, reaching the atrium through the inferior vena cava. This usually takes several weeks for collateral vessels to dilate sufficiently to accommodate the flow from the superior vena cava, leading to an increase in cervical venous pressure by 20 to 40 mmHg⁶. Difficulty in draining the superior vena cava can result in edema in the upper part of the body, potentially causing cerebral edema and airway edema⁶. AR-

SVC is closely related to PLSVC, as diverting drainage to the left allows venous return with less resistance and, therefore, with fewer or even no clinical repercussions. However, generally asymptomatic ARSVC with PLSVC can be a component of more complex cardiac pathologies or, in stressful situations, may lead to significant issues such as various arrhythmias³.

PLSVC, in addition to the vascular changes already described, is often associated with other congenital heart lesions, especially when it drains into the left atrium⁴. Approximately 92% of PLSVC drain into the right atrium through the coronary sinus; however, the remaining 8% drain into the left atrium independently of the coronary sinus, creating a left-to-right atrial shunt⁷. Atrial septal communication has been the most common cardiac anomaly associated with the left superior vena cava⁴. Other cardiac lesions accompanying this anomalous vessel include single atrium, ventricular septal defect, Eisenmenger complex, Tetralogy of Fallot, truncus arteriosus, pulmonary stenosis, tricuspid atresia, aortic coarctation, anomalous pulmonary venous return, and absent right superior vena cava⁴. In cases diagnosed with Tetralogy of Fallot and Eisenmenger syndrome, there is a 20% and 8% probability of PLSVC, respectively⁸. There is also a frequent association with situs inversus or partial transposition of the viscera and/or levocardia⁴. A high incidence of left superior vena cava has been reported in asplenia⁴.

In cases where right drainage is predominant, the coronary sinus typically shows an expansion that can lead to the compression of the atrioventricular node and its surroundings. Other possible complications resulting from the volumetric increase of the coronary sinus include compression of the left atrium with reduced cardiac output and complications associated with mitral valve surgery due to anatomical proximity³.

In patients with PLSVC, complications related to central venous puncture, regardless of the technique used, result from the tortuous path crossing structures under mechanical stress and therefore include angina, hypotension, and perforation, which can progress to severe arrhythmias, cardiogenic shock, and cardiac tamponade. Another relevant risk described in the literature is stenosis of the coronary sinus, which can lead to the failure of coronary drainage with consequences for the arterial flow of the right and left coronary arteries³. In the specific case of pacemaker implantation, there are difficulties in fixing and positioning intracavitary electrodes.

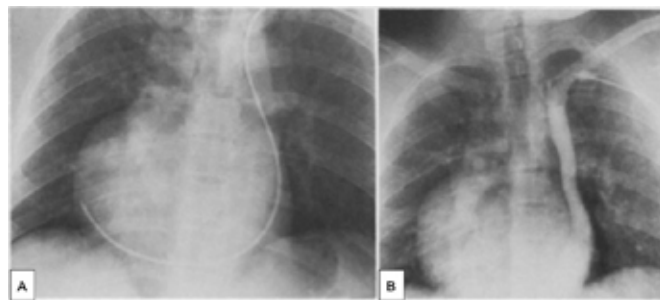


Figure 03: A: Shows that the catheter travels through the left paramediastinum, the left cardiac border in the left superior vena cava, and in the coronary sinus. B. Contrast infusion highlights the left superior vena cava and the coronary sinus .

CONCLUSION

PLSVC is an infrequent anatomical condition, so routine screening in preoperative evaluation is not necessary. Due to its predominantly asymptomatic nature, PLSVC may not be diagnosed until invasive procedures involving the vena cava occur. Although routine screening is not recommended, it is prudent to carefully observe exams that may show and facilitate early diagnosis, such as X-rays, computed tomography, echocardiography, and others. Thus, incidental findings, when diagnosed, should be communicated to the entire team, allowing for the proper prevention of associated complications. The anesthetist, as a participant in patient care, should be able to recognize and assist in preparing the team and managing the patient.

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