Case Report

Persistent left superior vena cava in a 29-year-old lady with Ebstein's anomaly and complete heart block. A case report and literature review

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ABSTRACT

Introduction: Persistent Left Superior Vena Cava (PLSVC) is a rare congenital vascular anomaly that may occur alone or in combination with complex congenital heart anomalies and dangerous arrhythmias. We report the first case in the literature of combined PLSVC, Ebstein's Anomaly (Type A) and complete atrioventricular block in an adult female, being successfully managed with permanent pacemaker implantation in Sierra Leone.
Case summary: We present an interesting case of a 29-year-old female, referred to the cardiology clinic on account of breathlessness, dizziness, and recurrent syncope. Physical examination revealed a pulse rate of 39 bpm, jugular venous pulse with occasional cannon waves, and grade 3/6 pansystolic murmur in the tricuspid valve area. An electrocardiogram confirmed complete atrioventricular block with junctional escape rhythm, while Transthoracic Echocardiogram (TTE) confirmed Ebstein's Anomaly (Type A) and moderate tricuspid regurgitation. PLSVC was discovered as an incidental intraprocedural finding. Deploying a pacemaker lead through this venous anomaly from the left side was futile. Nevertheless, we used a right sided approach that resulted in a successful permanent pacemaker implantation with optimal and stable parameters.
Conclusion: This rare case report highlights the practical challenges often encountered in the practice of cardiology during pacemaker and other cardiac device implantation. Cardiologists and critical care physicians should be acquainted with the venous anomaly of PLSVC, its variants, and procedure-associated risks, for better clinical decision making.

1. Introduction

Persistent Left Superior Vena Cava (PLSVC) is a rare cardiac anomaly that is usually asymptomatic [1]. It is present in 0.3–0.5% of the general healthy population and up to 4.5% of individuals with congenital heart disease (Ebstein's anomaly, ventricular septal defect, atrial septal defect, coarctation of aorta, cor triatriatum, etc) [2,3].

PLSVC may co-exist with Ebstein's anomaly, a rare congenital malformation of the heart that accounts for <1% of all cases of congenital heart disease [4]. Ebstein's anomaly is characterized by apical displacement of the tricuspid valve, and atrialization of the right ventricle. Based on the variable degree of displacement of the anterior tricuspid valve leaflet and size of the right ventricle, Carpenter et al. classified Ebstein's anomaly into four subgroups (Type A to Type D) [5].

About 30–70% of individuals with Ebstein's anomaly may have Atrial Septal Defect, Patent Foramen Ovale, Tetralogy of Fallot or other congenital anomalies. In the absence of an accompanying congenital anomaly, most Ebstein's anomaly individuals may have normal life span that would require no treatment intervention [6].

Even though PLSVC is benign in most cases, its presence may have several clinical implications as it may pose challenges and complicate invasive procedures like pacemaker (PM)/Implantable Cardioverter Defibrillator (ICD) implantation, central venous access, and cardiothoracic surgery [7]. PLSVC may be associated with an abnormal electrophysiological function due to anatomical and architectural abnormalities of the heart. The clinical implication is either manifested as tachyarrhythmias (atrial flutter/fibrillation, Wolff-Parkinson-White syndrome, Supraventricular tachycardia) or bradyarrhythmia

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(atrioventricular conduction blocks) [8,9]. Since PLSVC may co-exist with other congenital heart diseases, an early diagnosis is important for patient's prognosis.

This case report highlights the practical challenges that are often encountered with fluoroscopic C-arm X-ray machine during permanent pacemaker implantation in a resource-limited country. Furthermore, we report a case of combined PLSVC and Ebstein's anomaly in a young lady presenting with complete heart block. This case report is the first to be described in the current literature of cardiovascular medicine. It is registered under Research Registry https://www.researchregistry. com/browse-the-registry#home/ with the unique identifying number researchregistry7940 and reported in line with the Surgical CAse REport (SCARE) 2020 Criteria [10].

2. Case report

A 29-year-old Sierra Leonean lady was referred by his primary physician to the Specialist Out-Patient Cardiology Clinic (Connaught Hospital) with two-month history of irregular heartbeats, breathing difficulty and easy fatiguability that progressively worsened over time. She reported a recent history of dizziness, with recurrent presyncope and syncopal attacks. She is gravida 3, para 3, with the last baby being one year old. Her primary physician managed her for a presumptive diagnosis of heart failure with Spironolactone, Furosemide, and carvedilol without an initial cardiologist evaluation. She is not diabetic or hypertensive. She is a petty trader who does not smoke cigarettes or drink alcohol.

On examination, her pulse rate was 39/min regular full volume and non-collapsing. All arterial pulses were present with no radio-radial or radio-femoral delay. Jugular Venous Pulse (JVP) revealed occasional cannon waves. Her blood pressure was 130/60 mmHg, while the apex beat was in the fifth intercostal space mid-clavicular line. On auscultation, first (S1) and second (S2) heart sounds were heard with variable intensity of S1. Grade 3/6 pansystolic murmur was heard in the tricuspid valve area. There was adequate air entry into both lung fields with vesicular breath sounds. The liver was enlarged and pulsatile.

A 12 lead ECG showed a ventricular rate of 36/min with atrioventricular dissociation. A diagnosis of complete heart block with junctional escape was confirmed [Fig. 1A]. Transthoracic Echocardiogram (TTE) reported normal left ventricular dimension with preserved systolic function (Ejection Fraction = 61%), right atrial enlargement with moderate tricuspid regurgitation and a displaced septal leaflet of the tricuspid valve towards the right ventricular apex, suggestive of Ebstein's anomaly (Type A) [Fig. 2B]. She was transferred to an affiliated hospital of the University of Sierra Leone Teaching Hospital Complex (Choithrams Memorial Hospital) for pacemaker implantation. While on admission, reversible causes for complete heart block were ruled out. Her thyroid function test was compatible with pre-clinical hypothyroidism. Thyroid Peroxidase Antibodies [0.8iu/ml (0.0–66.0iu/ml)] and thyroid gland scan were normal. An informed consent form was signed by the patient for permanent pacemaker (PPM) implantation. The procedure was performed with a cine fluoroscopic C-
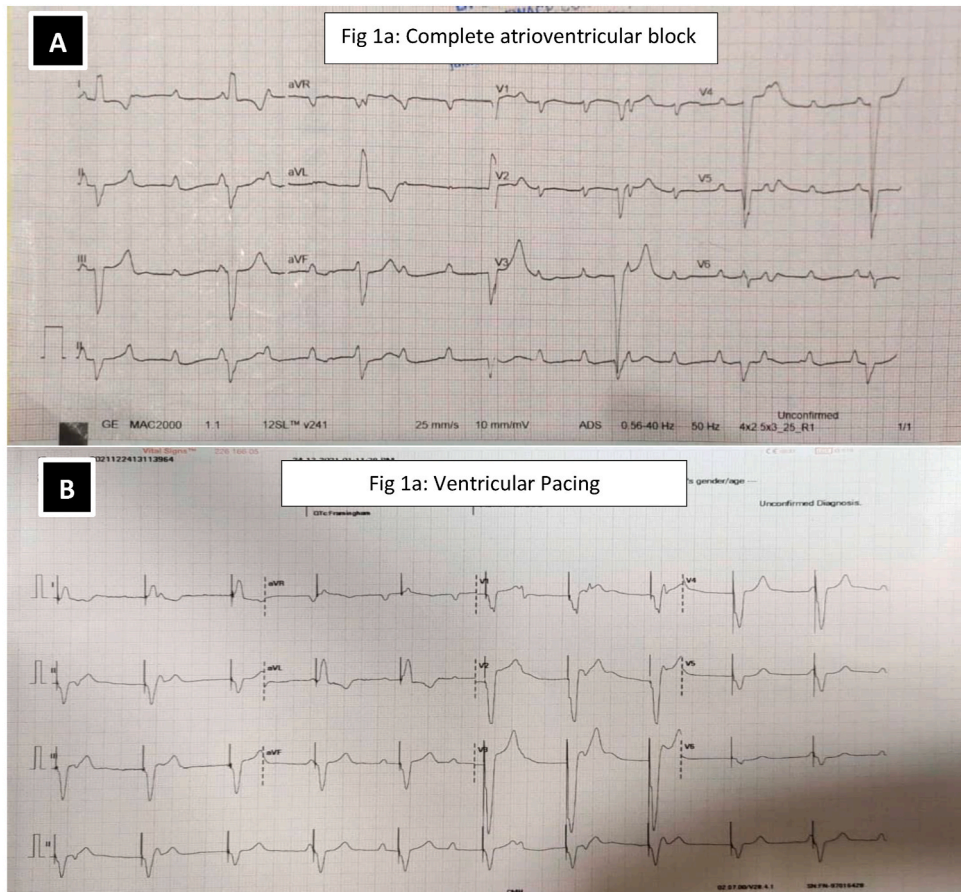
Figure 1: Electrocardiogram (ECG)
    A.   ECG shows complete atrioventricular block
    B.   ECG shows ventricular pacing

Fig. 1. Electrocardiogram (ECG)
A. ECG shows complete atrioventricular block
B. ECG shows ventricular pacing.
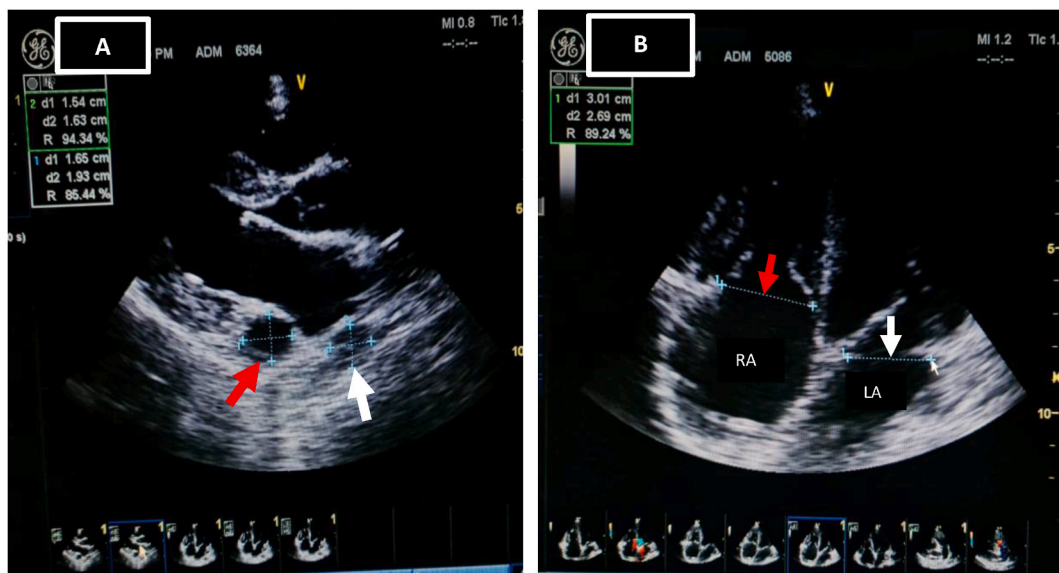
Fig. 2. Transthoracic Echocardiogram

A. Parasternal long axis view: red arrow is pointing to a dilated coronary sinus. White arrow is pointing to the Descending Aorta

B. Apical four chamber view illustrating a downward displacement of the tricuspid valve (red arrow) relative to the mitral valve (white arrow), indicative of Type A Ebstein Anomaly. Grossly dilated right atrium (RA). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

arm X-ray machine in an operating theatre, that serves as the only facility for cardiac device implantation in Sierra Leone.

A permanent pacemaker implantation was performed by a cardiologist with more than six years' experience and a team of doctors/ pacemaker technician. Venous access for PPM implantation was achieved through a left cephalic vein cut down, and this was followed by deploying a 7 French sheath. An abnormal trajectory of the guidewire along the left para-mediastinal region was observed on cine fluoroscopy, while introducing the guidewire into the left subclavian vein. [Fig. 3A, B, 3C]. After several attempts, it was rather impossible to deploy the guidewire across the vertebrae towards the right side. On two occasions the guidewire took an abnormal spontaneous track into the left internal jugular vein. In view of the prevailing scenario, a suspicion of PLSVC was raised and subsequently confirmed by injecting contrast into the left subclavian vein. The course of a PLSVC was clearly demonstrated as it opened into the right atrium via the coronary sinus [Fig. 3D]. Once the anatomy of PLSVC was established, technical difficulties and challenges were encountered on every attempt made to deploy the pacemaker lead through the PLSVC into the right atrium via the coronary sinus. To prevent intraprocedural complications related to the patient's complex venous anomaly, we concluded that the left-sided access was not feasible. Subsequently, we switched over to a right-sided approach as it was the best available option for pacemaker implantation. Venous access was achieved through the right subclavian vein. Once again, contrast was injected into the right subclavian vein to establish patency of the right superior vena cava, as agenesis of the RSVC may occur with PLSVC. An active bipolar lead was successfully implanted into the right ventricle.

Our initial plan was to implant a dual-chamber pacemaker. However, during the invasive procedure, it was rather tricky and challenging to implant an active fixation pacemaker lead into the right atrium, as it dislodged after every fixation attempt. The technical challenge encountered can be related to the abnormal atrialization of the right ventricle and tricuspid regurgitation in Ebstein's anomaly. We decided to implant a VVI pacemaker because the recommended time under fluoroscopy for dual-chamber pacemaker implantation has considerably exceeded. The implanted VVI pacemaker had a pacing threshold of 0.5V, stimulus duration of 0.4ms, an impedance of 760 Ω, and ventricular

sensing of 12.8mV.

Post-operative ECG was normal with ventricular pacing spikes [Fig. 1A] while post-implant chest x-ray confirmed correct placement of an active lead in the right ventricle. Meanwhile, the most important sign (dilated coronary sinus) of PLSVC on transthoracic echo was never documented in her echo report prior to pacemaker implantation. Subsequent review of her echo images post-implantation confirmed a dilated coronary sinus [Fig. 2A]. She was discharged the next day after an uneventful recovery. A follow-up angio-computed tomogram of the thoracic/neck vessels and review by an endocrinologist was recommended. Reconstructed CT images confirmed PLSVC and RSVC (Double Superior Vena Cavae), with a bridging brachiocephalic vessel and enlarged right atrium [Fig. 4A and B].

3. Literature review

PLSVC is an uncommon congenital anomaly in which the remnant vein of Marshall persists during the early weeks of embryonic development. Throughout normal fetal development, the embryonic venous system undergoes various developmental stages. At eight weeks of gestation, the internal jugular veins are formed from the cephalic portion of both superior cardinal veins which then anastomose to form the brachiocephalic vein [1]. The right-sided caudal portion of the cardinal veins will form the RSVC, while the left caudal portion will regress to form the ligament of Marshall but failing to do so, it will persist as the left superior vena cava that empties into the coronary sinus [1].

PLSVC may occur in isolation or in combination with other cardiac or vascular malformations and is frequently encountered in patients undergoing PPM or ICD implantation [11]. The high prevalence of PLSVC detection during implantation of PPM or ICD, is mainly due to the treatments of arrhythmias that are frequently associated with congenital venous anomalies [9]. Several proposed classifications of PLSVC have been reported in the literature, and these are established in relation to: (a) the anastomotic ramus between superior venae cavae and the azygos venous system by Uemura et al. [12], (b) presence or absence of the left brachiocephalic vein (LBCV) bridging both superior vena cavae (SVC) [13], and (c) the venous drainage pattern of the left superior vena cava
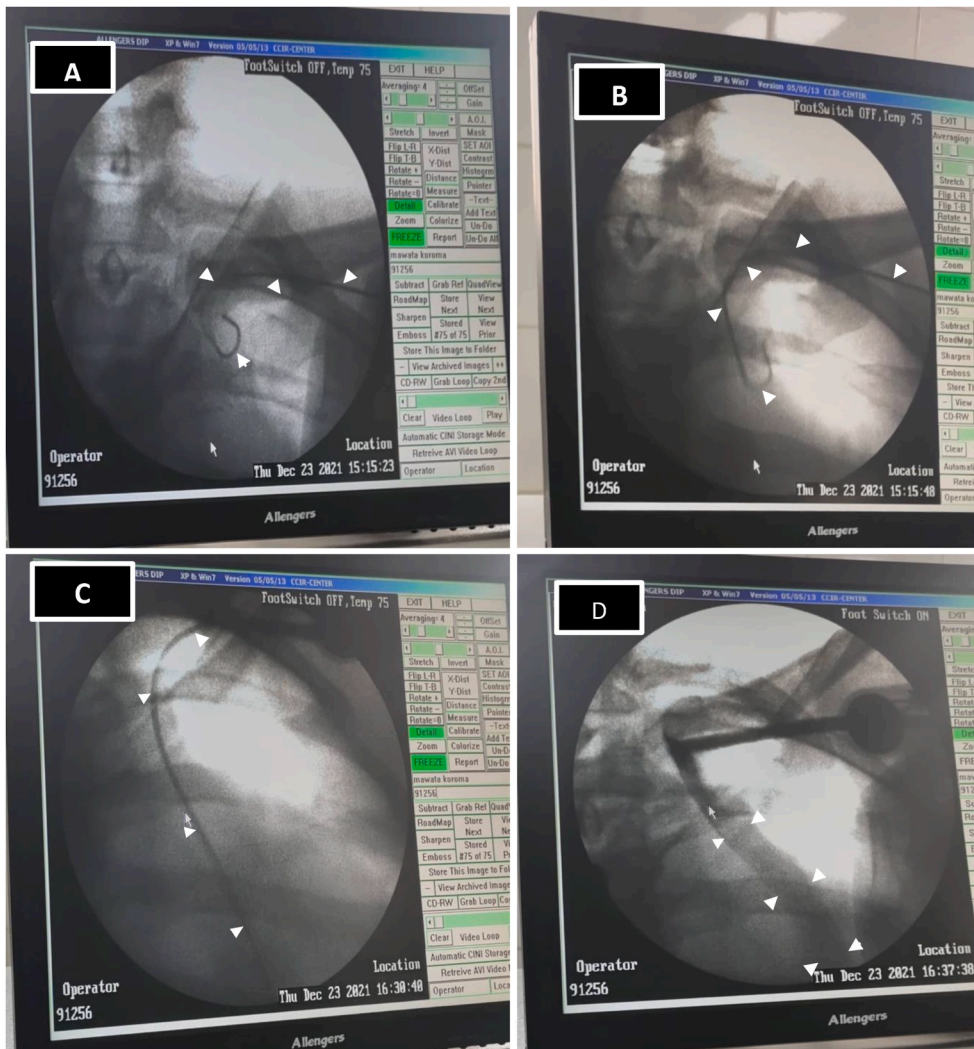
Fig. 3. Flouroscopic C-arm X-ray machine demonstrating the guidewire pathway and intravenous contrast

A. Failure of the guidewire to cross the midline with initial bending along the left paramediastinal region

B. Pathway of the guidewire along the left paramediastinal region

C. Full deployment of the guidewire along the left paramediastinal region

D. Domonstration

of Persistent Left Superior Vena Cava after injection of constrast.

(LSVC) by Zhang et al. [14].

According to Zhang et al. a rational and easy classification of PLSVC based on the venous drainage sites was sub-grouped into four types:

Type I – PLSVC drains into the right atrium through the coronary sinus.

Type II – PLSVC drains into the right atrium through the coronary sinus but has a connection with the left atrium, thus generating the right to left shunt.

Type III – PLSVC directly drains into the left atrium, resulting in a right to left shunt.

Type IV - PLSVC directly connected to the left pulmonary vein.

Ninety percent (90%) of all PLSVC classified as type I are also called Double SVC. Type 1 PLSVC may cause no hemodynamic consequences in the absence of dilated coronary sinus ostium [1]. A higher incidence of arrhythmias and conduction system abnormalities have been reported among patients with PLSVC. The postulated mechanism for this association is related to the stretching of atrioventricular nodal tissues by the dilated coronary sinus, which may also serve as a substrate for re-entry tachycardia [9]. Another mechanism for a sinus node dysfunction in PLSVC, may be due to the proximity of the cardinal venous tissue to the conduction tissues [8,9]. The patient described in this case report has type I PLSVC with conduction abnormality (complete heart block).

Goyal et al. reported that 65% of individuals with type 1 have no interconnecting vessel between RSVC and LSVC. The connection between the left and right superior vena cavae is called the brachiocephalic

vein or bridging vein [15]. Our case report highlighted this bridging vein [Fig. 4B]. In addition, PLSVC in association with other cardiac congenital abnormalities, is classified into several different groups: 1. Shunt Lesions 2. Conotruncal Malformation 3. Left-sided obstructive lesions. 4. Right-sided lesions 5. Ventricular anomalies, 6. Aortic arch anomalies, and 7. Heterotaxic forms [16–18]. Cardiac anomalies such as atrial septal defect, bicuspid aortic valve, coarctation of the aorta, coronary sinus ostial atresia, and cor triatriatum are reported in 40% of PLSVC patients, with most of these cardiac anomalies co-existing with right SVC agenesis [17]. Hence clinicians must search for associated congenital cardiac anomalies in patients with PLSVC. In our patient, an aortogram was performed to rule out coarctation of the aorta [Fig. 4D].

In recent publications, specific cardiac abnormalities such as Ebstein's anomaly have also been reported with PLSVC. Chahine et al. were the first group to publish the combination of PLSVC with Ebstein anomaly, and this was recently followed by another case report by Kwon et al. [19,20]. Nevertheless, none of these two cases described any associated conduction abnormality. Lam et al. published a case report of combined PLSVC and Ebstein's anomaly in a patient presenting with Wolff-Parkinson-White Syndrome [21].

A search in PubMed and Google Scholar databases using the keywords "persistent left superior vena cava" and "Ebstein's anomaly" generated only a few publications [19–22]. In contrast, another search in the same databases using the keywords "persistent left superior vena cava," "Ebstein's anomaly", and "Complete Heart block" produced zero
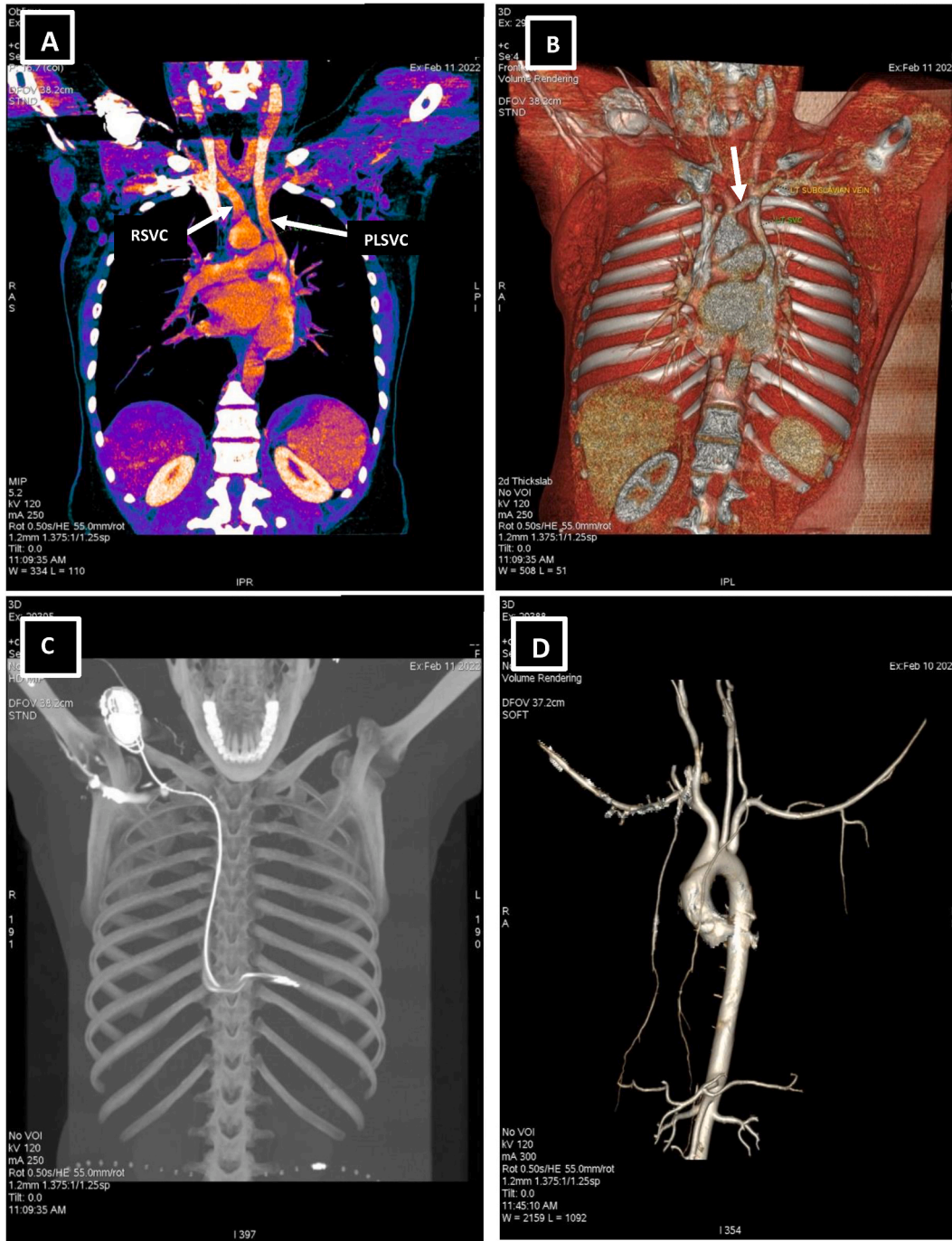
Fig. 4. Angio-Computed Tomography Scan
A. CT image showing Right Superior Vena Cava (RSVC) and Persistent Left Superior Vena Cava (PLSVC)
B. 3D reformatted CT Imaging showing Bridging Brachiocephalic Vessel (white arrow)
C. 3D reformatted CT imaging showing permanent pacemaker on the right side
D. 3D reformatted CT imaging showing a normal aortogram.

citations. To the best of our knowledge, we are the first authors to report in the literature; a case of combined PLSVC, Ebstein's anomaly and complete heart block in a patient in whom a VVI pacemaker was successfully implanted with the aid of a fluoroscopic C-arm X-ray machine.

The clinical implication of PLSVC is directly dependent on the drainage site and the accompanying cardiac anomalies. PLSVC is frequently asymptomatic in the absence of cardiac anomaly and can be detected as an incidental finding. In PLSVC, expansion of the coronary sinus may potentiate cardiac arrhythmias and may also complicate mitral valve surgery due to the close anatomic relationship of an existing

coronary sinus dilation [8,23–25]. Due to the tortuous course of PLSVC or atresia of the coronary sinus ostium, PM/ICD implantation may be complicated by fixation difficulties of the electrode. It may also result in serious complications like cardiogenic shock, heart perforation, cardiac tamponade, and lethal arrhythmia [1,11].

Drainage of PLSVC into the left atrium is crucial in the clinical presentation of these individuals. In cases where a right to left shunt is more pronounced, significant desaturation may occur. It will manifest as severe cyanosis, easy fatiguability, and reduced exercise intolerance. Paradoxical embolic events and even brain abscesses will occur in cases

where the PLSVC drains into the left atrium [1,14]. A large right to left shunt may require surgical correction.

Whenever PLSVC is inadvertently discovered during invasive procedures, the recommendation is to switch from left-sided access to the right subclavian vein for easy lead placement, especially in a setting, where deploying a lead through the PLSVC via the CS into the right atrium becomes challenging [3]. However, there are situations wherein a right-sided approach is not feasible, as in congenital absence of the right-sided SVC or previous traumatic injury to the right side [11]. In this clinical scenario, central venous access should be made from the femoral vein in patients with PLSVC.

In our patient, it was rather difficult to deploy the pacemaker lead through the PLSVC via the ostium of the CS into the right atrium after several attempts. The main limitations of this invasive procedure are related to the suboptimal radio-images of the fluoroscopic c-arm x-ray machine and the absence of specific instruments like steerable stylets and special sheaths. Nonetheless, there are several case reports of successful implantation of various cardiac devices (pacemakers/ICD/CRT-D) from the left subclavian approach in patients with PLSVC [26–28].

The ECG of PLSVC patients may show left axis deviation of the P wave, with a normal or shortened PR interval. The mechanism of the left axis deviation is related to the geometric changes of the LA [9]. The chest X-ray may show a crescent-shaped shadow of the PLSVC at the aortic knob or left upper mediastinum. The characteristic finding of PLSVC on 2-D Echo (parasternal long-axis view) is the identification of a large CS with a diameter > 1cm. In our case study, the patient's coronary sinus diameter was 1.54cm × 1.63cm (Fig. 2A), but not documented in the echo report prior to PPM implantation. The clinical implication is that a dilated coronary sinus on the echocardiography report should have alerted us (Clinicians) towards the possibility of PLSVC. A large CS is not pathognomonic for PLSVC as it can be seen with increased RA pressure, anomalous systemic or pulmonary venous system or fistulous connection with coronary arteries [24]. The diagnosis of PLSVC can be obtained on TTE by the injection of agitated saline. This procedure usually results in the opacification of the right atrium, but in the presence of PLSVC, the CS opacifies first before the RA. With recent advances in imaging, several techniques (venous angiography, CT, MRI) can now be used to directly visualize and confirm this venous anatomy [13,14]. TEE may also show a large CS, while a mid-oesophageal view on TEE will show the PLSVC near the LA appendage and left pulmonary vein.

4. Conclusion

We report the first case in the literature of combined PLSVC and Ebstein's anomaly (type A) in a young lady, who presented with a complete heart block that was successfully managed with the implantation of a VVI pacemaker by using a fluoroscopic C-arm Xray machine. Though PLSVC is usually asymptomatic, it may complicate cardiac device implantation even in an experienced hand. Additionally, PLSVC should be thought of every time the course of a guidewire or catheter inserted in the upper central venous system takes an unusual left-sided para-mediastinal downward path or if there is the presence of dilated coronary sinus on echocardiogram.

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Ethical Approval

Case reports are exempted from ethical approval in my institution Approval was obtained from the patient.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author's contributions

TRK, VYC, SC, KB and JC, OZM participated in the writing of the manuscript. JWBR reviewed all stages of the drafted manuscript for important intellectual content.

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Declaration of competing interest

Authors of this article have no conflict or competing interests. Final version of the manuscript was approved by the authors.

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