

Venous system anomaly as a reason for misplacement of pacemaker lead (RCD code: IV-1C.1)

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Abstract

We present a case of a 45-year old patient with history of surgical ASD II closure and pacemaker implantation who was only recently diagnosed with lead misplacement due to anomalous venous return. JRC D 2016; 2 (6): 196–198

Key words: atrial septal defect, anomalous venous return, rare disease, tachy-brady

Background

Ostium secundum type atrial septal defect (ASD II) is a congenital heart disease fairly frequently found in adults whereas ASDs of ostium primum, sinus venosus or coronary sinus types are much

less common. ASD II may coexist with other congenital abnormalities involving anomalous pulmonary veins connection to right atrium (RA) in <10% of cases and, much less often, systemic veins drainage to left atrium (LA) [1]. We present a case of a patient with anomalous subclavian vein branch draining into LA.

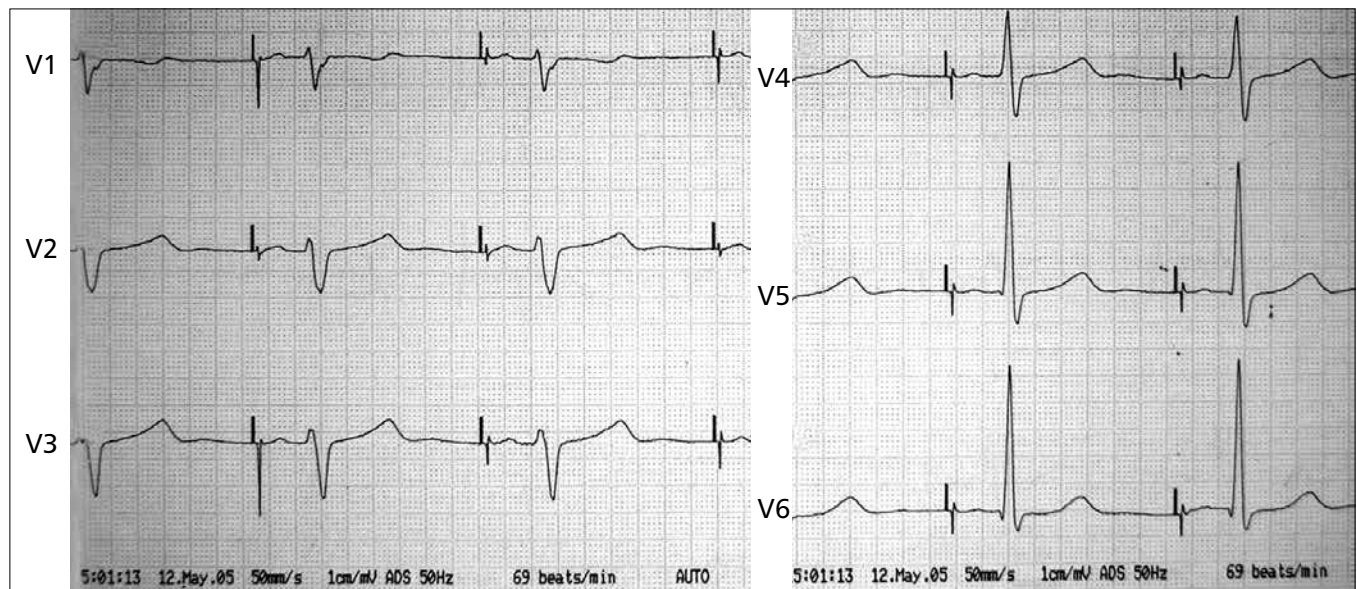


Figure 1. Electrocardiogram. Normal sensing and stimulation in AAI pacing mode is shown

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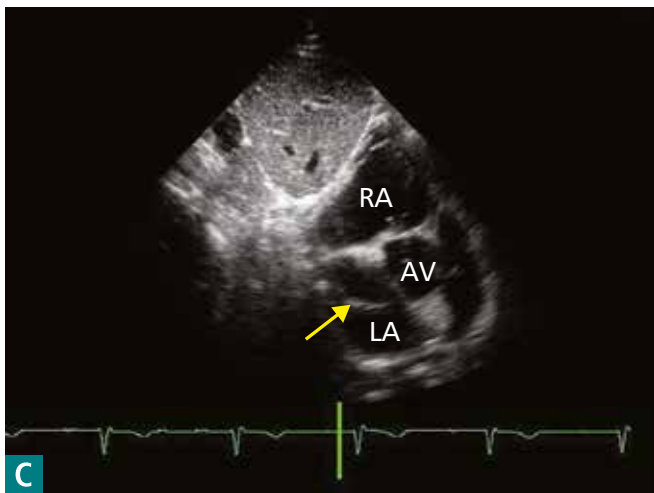
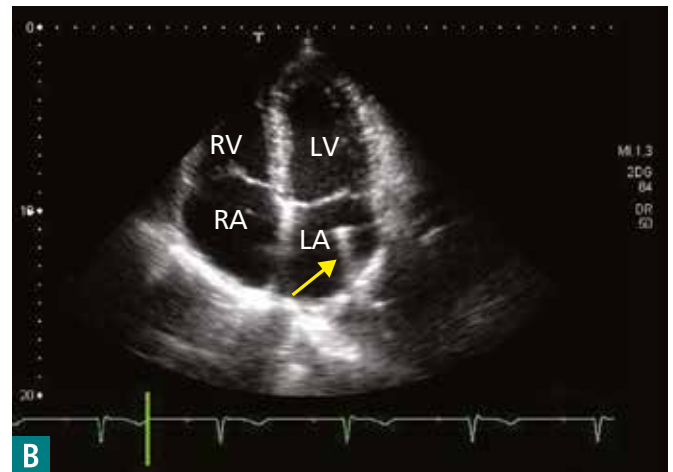
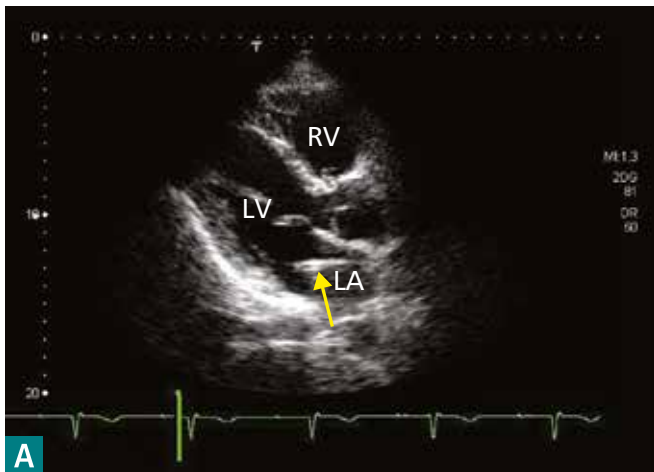


Figure 2. Transthoracic echocardiography. Pacemaker lead in the left atrium (arrow) is depicted. A. Parasternal long-axis view. B. Apical four chamber view. C. Subcostal short-axis view. RV – right ventricle, LV – left ventricle, RA- right atrium, LA – left atrium, AV - aortic valve

Case description

A 45-year old male, with a history of surgical ASD II closure (1996) and AAIR pacemaker (PM) implanted due to brady-tachy syndrome with Morgagni-Adams-Stokes (MAS) attacks (2002), was currently admitted to Department of Cardiology because of uncharacteristic chest pain of moderate intensity. In 2003 the patient was also hospitalized following similar in nature but more severe chest pain. Since myocardial necrosis markers had then been negative and no ischemia had been provoked during the exercise testing the coronary angiography (CA) was not performed. Now electrocardiogram (ECG) showed proper AAI stimulation with correct sensing (Figure 1). No significant arrhythmia was registered during 24-hour Holter ECG monitoring.

Transthoracic echocardiography (TTE) revealed presence of PM lead in left atrium (LA) (Figure 2). There were borderline right ventricle (RV) and RA dimensions of 28 mm and 19 cm², respectively. A moderate tricuspid regurgitation was also visible with calculated RV systolic pressure of 25 mmHg. No additional abnormalities were found and pulmonary to systemic flow ratio was normal.

Transesophageal echocardiography (TEE) documented course of PM lead within LA (Figure 3). No signs of ASD II recanalisation could be found. Sequential administration of 1st generation contrast

agent into right and then left antecubital vein resulted in opacification of right heart only.

CA did not demonstrate any stenotic lesions. Left ventriculography showed normal size and function of left ventricle (LV) and no mitral regurgitation. Oxymetry test was not suggestive of a shunt. PM lead course along an anomalous vessel connecting left subclavian vein and LA was visualized (Figure 4). Left-sided location of the lead could also be noticed on previously performed chest X-rays (Figure 5).

Taken all the above into account the patient was scheduled for conservative treatment including oral anticoagulation and discharged from hospital.

Discussion

The presented case is a rare example of coexistence of two congenital abnormalities– ASD II and anomalous systemic vein drainage.

Interestingly, despite previous cardiac surgery and PM implantation, the anomaly was only diagnosed after PM lead was accidentally noticed within LA during TTE performed due to uncharacteristic chest pain. Such finding could suggest recanalisation of ASD II with following dislocation of the lead. Multimodality imaging

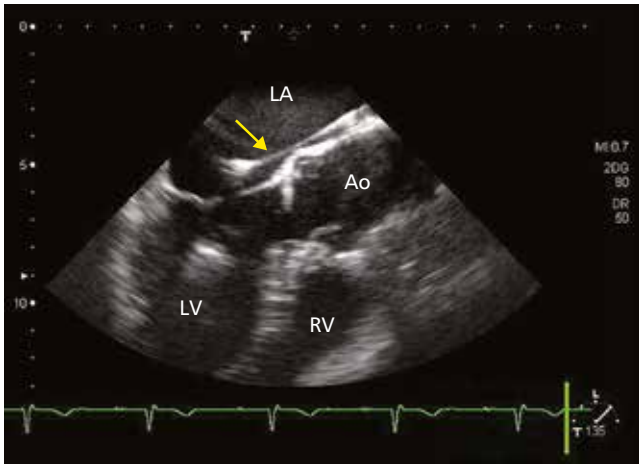


Figure 3. Transesophageal echocardiography. Pacemaker lead in the left atrium (arrow). RV – right ventricle, LV – left ventricle, LA – left atrium, Ao – aorta

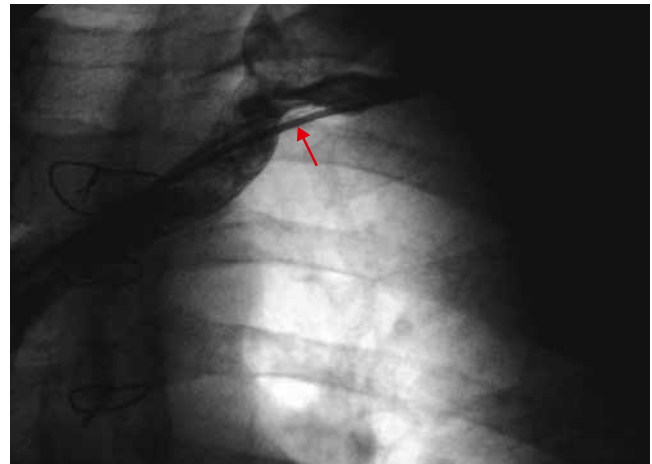


Figure 4. Venography. Contrast media was injected into the left basilic vein. Pacemaker lead reaches the left atrium via the left subclavian vein (arrow). A narrow occlusion containing the lead was not opacified – probably due to its occlusion

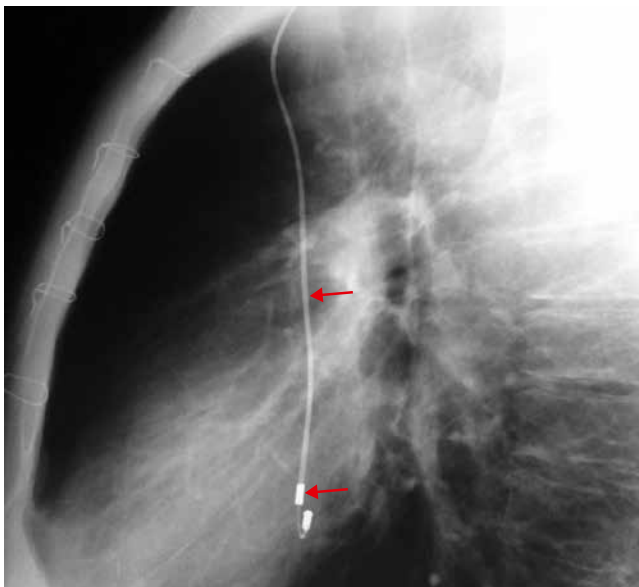


Figure 5. Chest X-ray; lateral view. Arrows indicate abnormal course of atrial pacemaker lead

(chest X-ray, TEE, angiography) enabled visualization of atypical lead course and its reason.

Venous anomaly was not revealed during diagnosing the ASD II before surgical correction. One might speculate that shunting volume was dependent on that additional connection and therefore fairly moderate. Tests performed after ASD II closure and PM implantation (TTE, TEE, oxymetry) did not show the shunt either. That is most probably due to the fact that the lead within the anomalous vessel caused its lumen to obliterate. Normal opacification of the RA only after contrast agent administration into both antecubital veins would seem consistent with such explanation.

Atypical chest pain reported by the patient and not verified during previous hospitalization was the reason for performing CA, which turned out to be normal.

Literature reports on erroneous PM lead implantation into the left heart describe mostly puncture of the left subclavian artery with lead entering LV or penetration of the interatrial or interventricular septum during lead anchoring [2, 3]. On the other hand, one should remember that in patients with biventricular or biatrial stimulating systems a lead positioned on the left side of the heart can be a normal finding (e.g. within coronary sinus in CRT patients).

Anomalous venous return may be present in patients with ASD II and should be considered during imaging testing and invasive treatment.

Our patient underwent surgical correction of ASD II before PM implantation, which might have changed anatomical conditions in the chest. That would explain the fact that the operator did not observe the improper course of the lead on fluoroscopy during the implantation, particularly as anchoring of the RA lead is known to be sometimes more challenging than the RV lead [2].

Since presence of a foreign body in the left heart increases the risk of embolism oral anticoagulation was started.

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