AUTOMATIC INTERNAL CARDIAC DEFIBRILLATOR LEAD IN THE LEFT VENTRICLE

SVETLANA BARBARASH, MD
ANN TONG, MD

(from the Department of Cardiology, Southwest Medical Associates, Las Vegas, Nevada, USA and the Cardiac &Vascular Institute, Gainesville, Florida, USA)

Introduction: Inadvertent insertion of a pacemaker/defibrillator lead into the left ventricle is an uncommon event, and its actual incidence is probably unknown. It may be underestimated and underreported because of a possible asymptomatic course.

Case presentation: We report a case of a 63-year-old Caucasian man with a malpositioned ICD lead into the left ventricle via a subclavian artery that was not suspected during implantation and went undiagnosed for 3 months without complications. The patient remained asymptomatic. He underwent an uncomplicated removal of the device and subsequent placement of a new ICD in the right ventricle.

Conclusions: Inadvertent insertion of a pacemaker/defibrillator lead into the left ventricle is a potentially dangerous complication that may occur under fluoroscopic guidance and may be overlooked by routine device interrogation. Careful attention to lead position on CXR posteroanterior and lateral views as well as 12-lead ECG in ventricular paced (magnet mode) after device implantation is recommended.

Abbreviations and acronyms:

ICD = internal cardiac defibrillator
CXR = chest x-ray
ECG = electrocardiogram
LV = left ventricle
RBBB = right bundle branch block
RV = right ventricle
LIMA = left internal mammary artery
LAD = left anterior descending artery
SVG = saphenous vein graft
PDA = posterior descending artery
PLV = posterior left ventricular branch
AV = atrio-ventricular
BNP = brain natriuretic peptide
PA = posterior-anterior

Introduction

Implantation of transvenous pacing and implantable cardioverter-defibrillator leads is one of the most common procedures involving the heart.[1] It is estimated that more than 100,000 implantable cardioverter-defibrillator and more than 200,000 permanent cardiac pacemaker implantations are performed in the USA annually.[2] This procedure is performed by cardiologists, cardiothoracic surgeons, intensivists and general surgeons worldwide. Malposition of a ventricular lead into the LV is an uncommon event, and its actual incidence is probably unknown. It may be underestimated because of underreporting. Inadvertent LV pacing can result from unintentional placement of the ventricular lead into the LV through a patent foramen ovale or atrial septal defects, or after perforating the interatrial septum, especially at the fossa ovalis.[3] This may occur more frequently in patients with dilated hearts, which may make fluoroscopic examination difficult and misleading. In these conditions, the lead passes through the atrial septum to the left atrium, then to the LV through the mitral valve. LV pacing after permanent transvenous pacemaker implantation has also been reported after ventricular septum or RV free wall perforation by the lead with subsequent LV pacing.[4,5] Moreover, unintentional placement of the ventricular lead into the distal coronary sinus or other cardiac veins has also been reported and may present with an ECG pattern of RBBB in paced mode.[6] Misplacement of the lead via the subclavian artery through the aortic valve into the LV may also result in LV pacing and a subsequent RBBB pattern shown on an ECG in paced mode.[7]

Case presentation: SH is a 63-year-old gentleman with a history of 6-vessel coronary artery bypass graft surgery in August 2014.

He had LIMA to first diagonal and sequentially to LAD, SVG to first obtuse marginal and second obtuse marginal, SVG to PDA and PLV in sequential fashion. Ejection fraction was 20% before and 25% after bypass.

He had a single lead ICD placed in September 2015 when he was admitted in the hospital for congestive heart failure exacerbation. His echocardiogram at that time showed ejection fraction of 20-25% with mild pulmonary hypertension.

The patient presented for his first visit in the Heart Failure Clinic on December 28, 2015. At that time he was complaining of leg edema, increase in abdominal girth despite being compliant with medications and low-sodium diet. He could not walk 1 mile in 20 minutes, and denied chest pain.

His ECG (figure 1) showed sinus rhythm with first-degree AV block, left atrial enlargement, right axis deviation, 94 bpm.

Admission laboratory tests showed BNP of 2416 ng/L, hemoglobin A1c 7.3, normal complete blood count, creatinine 1.04 mg/dl, potassium 5.6 mmol/L, sodium 138 mmol/L.

Past Medical History: Hyperlipidemia, hypertension, and diabetes for 20 years.

Medications: Spironolactone 25 mg once a day, lisinopril 2.5 mg once a day, furosemide 40 mg once a day, atorvastatin 20 mg once a day, aspirin 81 once a day, metformin 1000 mg twice a day, carvedilol 6.25 twice a day,

Social history: Denies smoking, alcohol or drugs use. He is a retired construction worker.

Family history and review of system: Noncontributory

Physical examination: elderly gentleman in no acute distress, well nourished and normally developed. Blood pressure 90/64, heart rate 56 bpm, respiration 16/m, oxygen saturation 97%, height 5 feet 7 inches, weight 184 pounds. No scleral icterus or corneal arcus. He had elevation of jugular venous pressure. Carotid upstrokes were brisk bilaterally without bruits. Lungs were clear to auscultation with symmetrical chest excursion. Cardiac exam revealed non-displaced point of maximal cardiac impulse. Rhythm was regular. First and second heart sounds normal, no murmurs, rubs or gallops. ICD device in left pectoral region – incision is well healed, no erythema or drainage. Abdominal exam revealed hepatomegaly and possible ascites. Extremities: 1+ pedal edema, both pedal pulses were normal. Cold extremities.

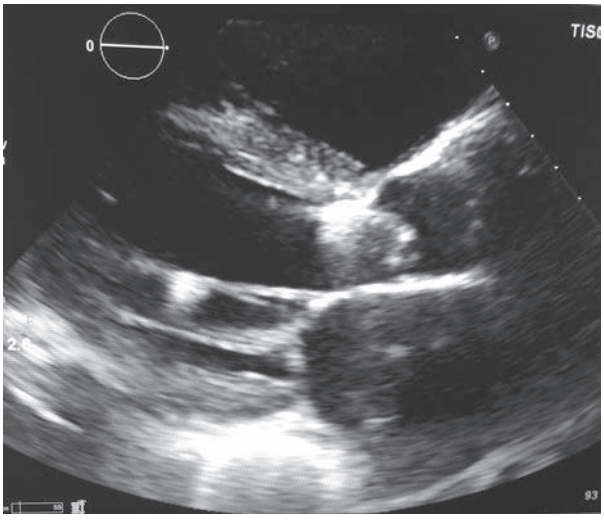
Patient was felt to be hypervolemic. He was given educational materials about low-sodium diet. His furosemide was changed to torsemide 40 mg daily, and repeated blood work and echocardiogram were scheduled.

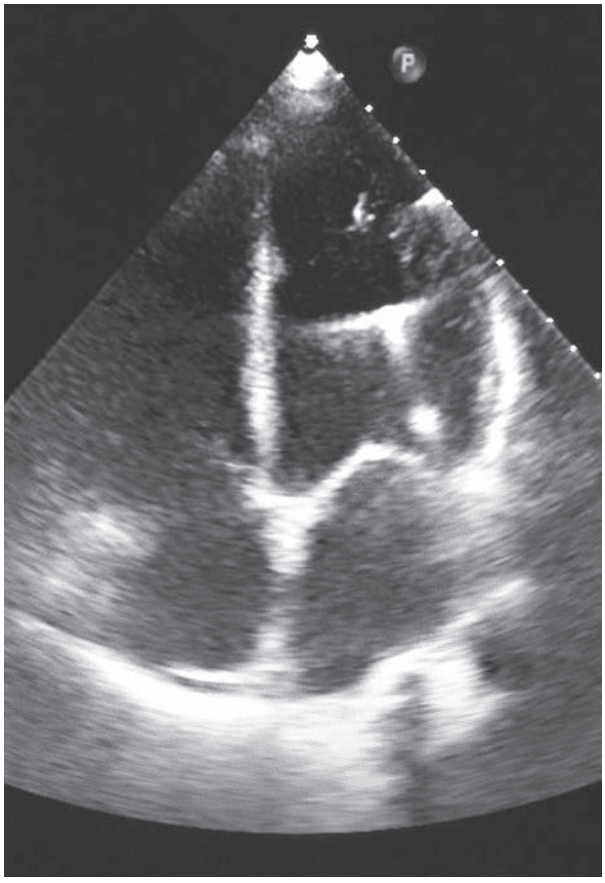
Echocardiogram on 1/11/2016 showed the following: Left ventricle: ICD lead appeared in LV cavity, unclear pathway to determine lead origin (figure 2,3). There was no ICD wire visualized in right-sided cardiac structures. The cavity size was at the upper limits of normal. Systolic function was severely reduced. The estimated ejection fraction was 15-20%. Hypokinesis of the anteroseptum. Akinesis of the inferoseptal , apical , inferior and basal-mid anterolateral myocardium. Anterior wall was not well visualized. Diastolic function could not be evaluated. Right ventricle: moderately dilated, severely hypokinetic. Left atrium: moderately dilated. Pulmonary pressure: systolic pressure was mildly increased.

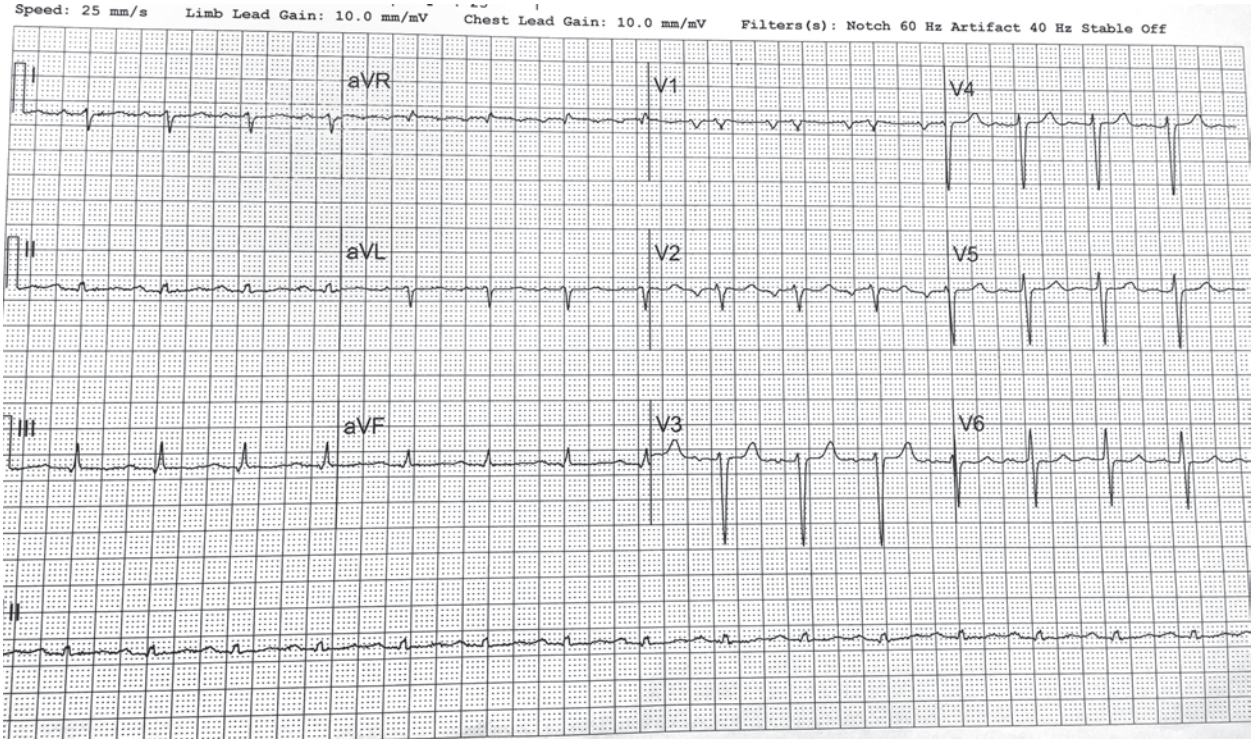
The patient was admitted to the hospital on 1/13/2016.

Transesophageal echocardiogram showed device lead in the ascending aorta through the aortic valve into the left ventricle. The findings were subsequently confirmed by computer tomography of the chest.

On 1/19/2016 the patient underwent surgical lead extraction. Intraoperative findings revealed that the ICD lead was inserted into the left subclavian artery. The lead was retracted under fluoroscopy. The tip was identified and the entire lead was removed from the







left ventricle and then removed from the artery. The patient tolerated the procedure well without complications.

The patient was discharged home on 1/21/2016 with an external defibrillator, "Life Vest".

New single chamber ICD was placed on 2/26/ 2016 via the right subclavian vein without complications.

He continued to be followed in the Heart Failure Clinic for optimization of medical therapy.

Discussion

The diagnosis of an inadvertently misplaced lead in the LV is simple but requires a high index of suspicion. Chest radiographs with posteroanterior and lateral projections should help differentiate RV from LV lead position. In our case, we could not locate the report of the initial chest X-ray done outside of our institution.

Also, the diagnosis of malpositioned device leads could easily be missed during electrocardiography and routine device interrogation because the patient was not pacemaker-dependent. Lead position was discovered by routine echocardiography.

Although unusual, serious complications may develop secondary to lead misplacement into the LV. These complications include systemic thromboembolism, perforation of the mitral valve leaflets, mitral insufficiency, aortic valve endocarditis, diaphragmatic pacing and loss of capture.[3,8] The exact risk of thromboembolism secondary to the presence of a pacing lead in the LV is unknown, but the incidence may reach up to 37% as suggested by previous reports.[9] On the other hand, there have been several reports in the literature about inadvertently placed pacemakers and ICD leads in the LV that were accidentally discovered after up to 17 years without systemic thromboembolic events in the absence of anticoagulation therapy.[10]

The therapeutic options for a misplaced lead in the LV are limited. If misplacement is diagnosed early after implantation, lead removal or adjustment is usually feasible. Adequate lifelong anticoagulation with warfarin is the therapeutic option of choice if the lead has been in place chronically. Lead extraction should be reserved for failure of anticoagulation or during other concomitant cardiac surgery.[10] In our patient, it was decided to extract the lead.

Conclusion

Inadvertent insertion of pacing and internal cardioverter defibrillator wires into the left ventricle is a potentially dangerous complication that may happen even in the most experienced hands. Fluoroscopy during implantation could be difficult and misleading in localizing the site of the ventricular leads. Device interrogation after implantation does not help differentiating between RV and LV pacing. Pacing thresholds are usually normal at the time of implantation and behave normally at follow-up. It is advisable that every patient receive a 12-lead ECG in ventricular paced

(magnet mode) during or immediately after implantation. A CXR PA and lateral should be performed and examined to determine the course of the pacing wire. Alternatively, limited echocardiography may be considered to confirm lead position.

REFERENCES

1. Bauersfeld UK, Thakur RK, Ghani M, Yee R, Klein GJ: Malposition of transvenous pacing lead in the left ventricle: radiographic findings. *AJR Am J Roentgenol* 1994, 162:290–292.

2. Bollinger BC, Heidenreich J: From cardiac arrhythmias. In *Current Diagnosis & Treatment Emergency*. 6 edition. Edited by: Stone CK, Humphries RL. New York: Lange Medical Books/McGraw-Hill; 2008:578–607.

3. Schiavone WA, Castle LW, Salcedo E, Graor R: Amaurosis fugax in a patient with a left ventricular endocardial pacemaker. *Pacing Clin Electrophysiol* 1984, 7:288–292.

4. Villanueva FS, Heinsimer JA, Burkman MH, Fananapazir L, Halvorsen RA Jr, Chen JT: Echocardiographic detection of perforation of the cardiac ventricular septum by a permanent pacemaker lead. *Am J Cardiol* 1987, 59:370–371.

5. Ormond RS, Rubenfire M, Anbe DT, Drake EH: Radiographic demonstration of myocardial perforation by permanent endocardial pacemakers. *Radiology* 1971, 98:35–37.

6. Meyer JA, Millar K: Malplacement of pacemaker catheters in the coronary sinus: recognition and clinical significance. *J Thorac Cardiovasc Surg* 1969, 57:511–518.

7. Mazzetti H, Dussaut A, Tentori C, Dussaut E, Lazzari JO: Transarterial permanent pacing of the left ventricle. *Pacing Clin Electrophysiol* 1990, 13:588–592.

8. Konings TC, Koolbergen DR, Bouma BJ, Groenink M, Mulder BJ: Iatrogenic Perforation of the posterior mitral valve leaflet: a rare complication pacemaker lead placement. *J Am Soc Echocardiogr* 2008, 21:512.e5–512.e7.

9. Van Erckelens F, Sigmund M, Lambertz H, Kreis A, Reupcke C, Hanrath P: Asymptomatic left ventricular malposition of a transvenous pacemaker lead through a sinus venosus defect: follow-up over 17 years. *Pacing Clin Electrophysiol* 1991, 14:989–993.

10. Van Gelder BM, Bracke FA, Oto A, Yildirir A, Haas PC, Seger JJ, Stainback RF, Botman KJ, Meijer A: Diagnosis and management of inadvertently placed pacing and ICD leads in the left ventricle: a multicenter experience and review of the literature. *Pacing Clin Electrophysiol* 2000, 23:877–883.

Статья поступила 26.04.2016

For correspondence:

Barbarash Svetlana
Address: 888 S Rancho Dr. Las Vegas, NV 89106
Tel. (702) 877-8600
E-mail: sbarbarash@yahoo.com