

Heart perforation in patients with permanent cardiac pacing – pilot personal observations

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Submitted: 15 November 2010

Accepted: 24 February 2011

Arch Med Sci 2012; 8, 1: 70-74

DOI: 10.5114/aoms.2012.27284

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Abstract

Introduction: Heart perforation is a rare complication of pacemaker (PM)/implantable cardioverter-defibrillator (ICD) implantation.

Material and methods: In our clinic in 2005-2010, 6 patients with heart perforation were hospitalized (3 women, 3 men), mean age 58.6 ±20.8 years (17 to 73 years). The indication to PM/ICD implantation was tachy-brady syndrome in 3 cases, second-degree atrioventricular block, advanced with losses of consciousness, vaso-vagal syndrome type II B with asystole lasting 12 s and recurrent non-sustained ventricular tachycardia in 1 patient. We analyzed patient's medical records, X-rays, echocardiography, computed tomography (CT) and procedure protocols.

Results: The incidence of heart perforation was 0.09%. Symptoms developed 4 to 990 days (mean 186.3 ±394.3) after PM/ICD implantation. The perforation site was found in the right atrial wall in 1 case and the right ventricular wall in 6 cases. The TTE revealed an accumulation of fluid in the pericardium over 10 mm behind the posterior wall of the left ventricle in all patients. The CT scan confirmed perforation of the heart chambers (atrium and in 6 cases ventricle). In 5 cases the whole device was removed by direct traction or percutaneous lead extraction with pericardiocentesis when necessary (pericardium drainage in 3 cases) while in 1 case cardiac surgery was needed.

Conclusions: The perforating lead may be removed by direct traction in the operating room with cardiocirculatory, anesthesiological and echocardiographical backup. In case of the lead perforation outside the pericardial sac or its atypical location, cardiac surgery is a safer method. The most important diagnostic method remains computed tomography.

Key words: heart perforation, pacemaker/cardioverter-defibrillator implantation.

Introduction

Implantation of a pacemaker (PM)/cardioverter-defibrillator (ICD), as well as reimplantation and repair operations, is associated with a low risk of complications, which usually occur early after the procedure. Possible complications include the following: PM pocket haematoma, stimulation of the chest wall muscle, diaphragm, phrenic nerves, lead dislocations within the heart, infections (bacterial endocarditis of the tricuspid valve, of the lead tip area, pacemaker pocket), subclavian vein thrombosis and/or its supplies, and in rare cases right ventricle or right atrium perforation with possible tamponade symptoms. The most common late complication is lead fracture, which requires its repair or removal. Another possible com-

plication is PM dislocation under the skin (which may cause decubitus or even skin necrosis) and skin puncture by a lead or PM [1].

Heart perforation in patients after PM implantation is a rare but potentially fatal complication. It may involve large veins, atrial or ventricular walls or the coronary sinus. Perforations may be acute (up to 24 h after implantation, which may lead to tamponade or death), subacute, which occur up to 1 month after implantation, or chronic, which appear at a later time. Another classification distinguishes between early (symptoms occur up to 1 month after implantation) and delayed perforations. The estimated rate of this complication is 1% and depends mainly on the surgeon's experience [2-13]. Main risk factors are listed in Table I [1, 4, 5].

The paper presents cases of life-threatening subacute heart tamponade and delayed perforations in patients after PM/ICD and atrial/ventricular lead implantation.

Material and methods

In our clinic in 2005-2010, 6 patients with heart perforation were hospitalized (3 women, 3 men), mean age 58.6 ± 20.8 years (17 to 73 years). In 2 of the patients, implantation was performed outside our clinic. During this period 3600 PM and 1007 ICD implantations were performed. Characteristics of the patient population are presented in Table II.

We analysed patients' medical records, physical examination, X-rays, echocardiography, computed tomography (CT) and protocols of the implantation procedures.

Table II. Characteristics of the patient population

| Parameter | Patient 1 | Patient 2 | Patient 3 | Patient 4 | Patient 5 | Patient 6 |
|---|-----------|-----------|-----------|-----------|-----------|-----------|
| Age [years] | 70 | 73 | 67 | 60 | 65 | 17 |
| Sex | Male | Male | Female | Female | Male | Female |
| Tachy-brady syndrome | + | + | + | | | |
| Second-degree atrioventricular block, advanced with losses of consciousness | | | | + | | |
| Vaso-vagal syndrome type IIB with asystole lasting 12 s | | | | | | + |
| Recurrent non-sustained ventricular tachycardia | | | | | + | |
| Dual chamber pacemaker | + | + | + | + | | + |
| Dual chamber ICD | | | | | + | |
| Coronary heart disease | + | + | + | + | + | |
| Arterial hypertension | + | + | + | + | + | |
| Dyslipidaemia | + | + | + | | | |
| Hyperthyreosis | | | + | | + | |
| Diabetes mellitus | | + | | | + | |
| Obesity | | + | | | | |

Results

In the present study, the incidence of heart perforation was 0.09%. Symptoms developed 4 to 990 days (mean 186.3 ± 394.3) after device implantation. There was no case of acute heart tamponade/perforation up to 24 h after implantation. The perforation site was found in the right atrial wall in one case and the right ventricular wall in 6 cases (Figures 1-2). Ventricular leads were placed in the right ventricular outflow tract and in 1 case in the apex. Atrial leads were placed in the right auricle. Heart wall damage was caused by pacing

Table I. Risk factors of lead perforation

| |
|---|
| I. Type of lead, mode of stimulation, lead tip location |
| Temporary stimulation |
| Atrial stimulation |
| Active fixation |
| Defibrillator leads: <ul style="list-style-type: none"> • Double spirals • Number of shocks delivered • Excessive length of the lead • Small diameter of the lead • High resistance (small tip surface) • Apical position |
| II. Propriety of the heart muscle |
| Congestive cardiomyopathy |
| III. The elderly, women, patients with low body mass (< 20 kg/m²) |
| IV. Anticoagulation therapy and steroid use within 7 days of implantation |
| V. Chest trauma, especially soon after implantation |

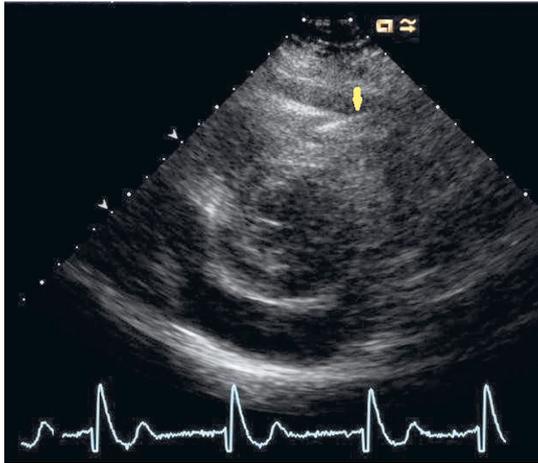


Figure 1. Transthoracic echocardiography view. Ventricular lead penetrating the wall of the right ventricle

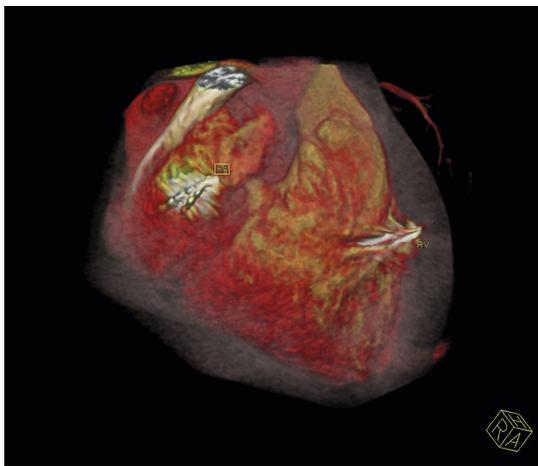


Figure 2. Computed tomography scan. Tips of both atrial and ventricular leads outside the heart

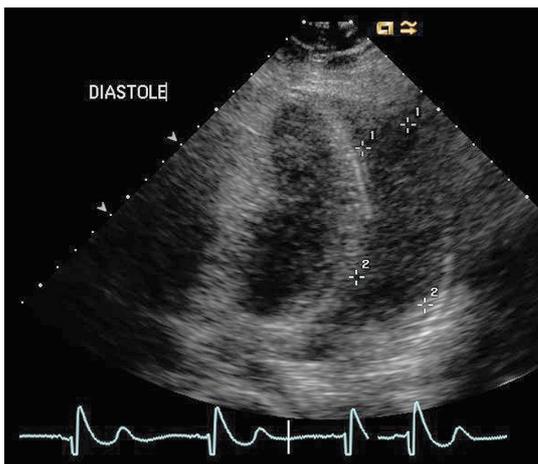


Figure 3. Transthoracic echocardiography view. Pericardial effusion up to 29 mm behind the left ventricular lateral wall

ventricular and atrial leads (Biotronik Setrox S60, S53, Vitatron Crystalline, Medtronic CapSure-FixNovus) in 5 cases and by a 1-coil defibrillator lead (Medtronic Sprint Quattro) in one case. All

leads were active fixation. Patients complained of stabbing pain in the left side of the chest, increased by movement (in 5 cases), dyspnoea (1 case), diaphragm stimulation (1 case), or remained asymptomatic (1 case). Standard follow-up of the electrical parameters showed pacing failure (in 4 patients), and sensing failure (in 6 cases). Transthoracic echocardiography showed pericardial effusion over 10 mm behind the posterior wall of the left ventricle in the parasternal long-axis view in all patients (Figure 3). Computed tomography scanning confirmed heart perforation (of the atrium and in 6 cases of the ventricle). In the operating room, with full cardiac surgery and anaesthesiology backup, the whole device was removed by direct traction, with pericardiocentesis when necessary (pericardium drainage in 3 cases) in 4 patients, and in one case (Figure 4) cardiac surgical extraction was used (in this patient, the lead penetrated the pericardial sac, injuring the parietal pleura and ended in the intercostal muscles, but without pneumothorax); 1 patient with delayed perforation underwent device removal by percutaneous lead extraction outside our department in a centre specializing in percutaneous lead extractions. Five patients had a new PM/ICD implanted, with lead tip location different from the previous one. One patient (patient no. 6) did not agree to have a new PM implanted.

Discussion

Heart perforation is a rare, but potentially fatal complication of permanent cardiac pacing systems. Subacute and chronic perforation may be asymptomatic, or it may cause dangerous symptoms resulting in death [2, 3, 9, 11, 13]. The common symptoms are chest pain, chest muscle or diaphragm contraction, hiccough, abdominal pain, dyspnoea, faintness or losses of consciousness (because of pacing failure), inadequate shocks of the ICD, symptoms of heart failure or tamponade [2, 3, 9, 12, 13]. Heart tamponade is a life-threatening complication, caused by rapid blood accumulation in the pericardial sac. It leads to pressure growth in the pericardium, which in turn impairs blood return to the atria and ventricles, lowering ejection volume and causing haemodynamic collapse with symptoms of cardiogenic shock, arrhythmias or death [10]. In our population, there was no case of an acute heart tamponade.

The most common perforation symptoms described in the literature are improper electrophysiological parameters; however, there have been cases with normal electrical function [1]. Failure of the lead to pace (increased pacing threshold) or sense (decreased R wave amplitude) or decreased lead impedance may suggest perforation and require further diagnosis [5, 7]. In our material, in

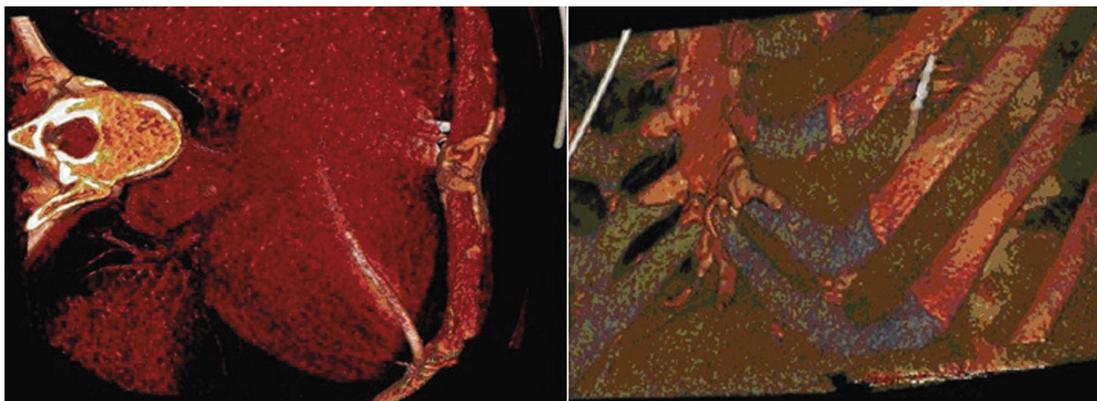


Figure 4. Computed tomography scan. Ventricular lead perforating pericardial sac, parietal pleura and ending in intercostal muscles

each case we observed a high pacing or sensing threshold.

Diagnostic methods in heart perforation involve chest X-ray and fluoroscopy (to show lead dislocation outside the border of the heart), transthoracic and transoesophageal echocardiography (TTE and TEE) (presence of the lead tip in the pericardium or pericardial effusion). The most important diagnostic method remains CT, which is the gold standard. It reveals both lead dislocation and even small pericardial effusion [4, 9, 12, 13]. Each of our patients had pericardial effusion.

In case of symptomatic heart perforation, the best method of treatment is extraction of the whole device. Various techniques of lead removal are used depending on the lead construction and the time since its implantation [5]. Active fixation leads of homogeneous diameter up to 2 years after implantation (6 months in the case of defibrillator leads) may be removed by direct traction, as in 4 of our patients. In case of failure, Byrd dilators are used. Active fixation leads of uneven diameter and/or implanted over 2 years before or other lead types (passive fixation) and implanted over 1 year before (6 months in the case of defibrillator leads) should be removed by a classical set of Byrd dilators [1, 5, 14], as occurred in our 1 patient with delayed perforation.

Most authors believe that patients with heart perforation should be transported to a centre specializing in percutaneous lead extraction and the best treatment is direct traction or percutaneous lead extraction in the operating room, with monitoring of basic life functions. Cardiosurgical, anaesthesiological and echocardiographic teams ready at hand are always necessary [1, 5]. It is also advised to have TEE monitoring during and after the operation. Percutaneous extraction of the perforating lead, not requiring general anaesthesia, has been performed in cases of a very high operation risk.

American guidelines recommend surgical removal of the perforating leads [6, 8]. The report-

ed cases of surgical lead removal were digestive tract perforation, massive heart tamponade, and atypical location of the perforating lead in the intercostal muscles [9, 12], as in our patient who required cardiac surgical operation.

In most of these cases, a new pacemaker was implanted, placing the lead tip in a different location than before. In the case of open surgery, epicardial leads have been used [4].

Diagnosis should be based on the patient's medical records, telemetric control of electrical parameters and visual examinations. Computed tomography remains the gold standard and it confirmed perforation in all our patients.

The main limitation of this study is the small group: only 6 patients in 5 years. None of the above-mentioned predictors appeared in our patients with a significant frequency.

In conclusion, in case of symptomatic heart perforation, the best method of treatment is extraction of the whole device. The perforating lead may be removed by direct traction in the operating room with cardiosurgical, anaesthesiological and echocardiographic backup. In case of lead perforation outside the pericardial sac or its atypical location, cardiac surgery is a safer method. The most important diagnostic method remains CT.

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