

Right-ventricular pacemaker lead induced severe tricuspid regurgitation with right heart failure immediately after pacemaker implantation: a case report

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Received Date : June 14, 2022
Accepted Date : July 16, 2022
Published Date : Aug 08, 2022
Archived : www.jcmimagescasereports.org
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Abstract

Background: We report a rare but severe pacemaker complication of a pacemaker lead induced severe tricuspid valve regurgitation. This led to acute right heart failure and decompensation within three days after pacemaker implantation.

Case presentation: An 80-year-old female patient presented with a pacemaker indication for sick sinus syndrome (SSS) documented with an implantable loop recorder (ILR). Preoperative transthoracic echocardiography (TTE) studies showed mild tricuspid regurgitation, moderate pulmonary hypertension with well-preserved right ventricular (RV) function. The patient received dual-chamber pacemaker implantation and ILR explantation. After the surgical procedure, the patient deteriorated day by day, both respiratory and hemodynamically. Interestingly, TTE showed evidence of new-onset high-grade tricuspid regurgitation, severe pulmonary hypertension, and right heart decompensation. After ruling out differential diagnoses (e.g., pulmonary embolism, ACS), it was decided to perform an AAI downgrade, after which the patient improved abruptly. An intraoperative TEE showed evidence of lead perforation or displacement of the tricuspid valve leaflets. In the further course, intermittent AV block III° appeared, whereupon the patient received a two-chamber-upgrade by left-ventricular (LV)-, instead of RV-lead implantation.

Conclusion: This case shows that the pre-operative selection of patients with an indication for LV- instead of RV-pacemaker lead implantation has a very high value. In most cases, a simple clarification by TTE provides the decisive indication for making the correct decision. The goal should finally be to use LV- instead of RV-lead implantation more generously when indicated, to avoid future short- and long-term complications.

Keywords: Pacemaker; tricuspid valve regurgitation; right heart failure; case report.

Background

The number of pacemaker implantations continues to increase, with a variety of techniques described and devices available. RV apex is often the site of choice for ventricular pacing [1]. Short- and long-term complications are not uncommon, and careful attention must be paid to avoid potential risks and to identify and treat all types of adverse events [2-6]. In particular, the use of an LV-lead instead of RV-lead in patients with appropriate risk factors has been studied and discussed for several years [2-3, 7]. Although the use of an LV-lead has shown a significantly better outcome, because beside of direct lead-induced trauma at tricuspid valve, the long-term RV pacing deleterious the LVEF (dyssynchrony) and heart fail-

ure [2, 8]. It seems to fail due to the surgical risk (e.g., the use of contrast media, long surgical time) and the performing centers, as LV-lead implantation does not yet seem to be part of the surgical standards in most centers. Therefore, the indication and implantation of an LV lead instead of an RV lead should be made more generous. Although this would mean referring some cases to pacemaker specialists who are performing LV-lead implantation, if the indication makes it justifiable (e.g., SSS, asymptomatic AV block).

This case reports a pacemaker-lead induced RV decompensation immediately after pacemaker implantation. Despite the already known evidence found in the literature that a specific choice of implantation method and device could have pre-

Citation: Thomas Aschacher, Daniela Geisler, Verena Lenz, Martin Grabenwöger. Right-ventricular pacemaker lead induced severe tricuspid regurgitation with right heart failure immediately after pacemaker implantation: a case report. *J Clin Med Img Case Rep.* 2022; 2(4): 1209.

vented the short-term poor outcome. Clear guidelines are lacking in these patients, which is especially true for patients with right heart failure (RHF), pulmonary hypertension (PH) and severe tricuspid valve regurgitation (TVR). Therefore, we report this case of an effective surgical approach in a patient with corresponding risk factors to reconsider and point out the importance of the corresponding guidelines.

Case Presentation

TAn 80-year-old female patient was admitted to the department of cardiac surgery for surgical implantation of a conventional dual-chamber pacemaker. She had a history of recurrent syncope and implantation of an ILR two years prior. The report of the ILR showed a high rate of symptomatic supra-ventricular tachyarrhythmia (SVTs) events (>30/month), who showed improvement on continuous beta blocker therapy. In the further follow-up, several sinus arrests (duration >3.8 sec) and bradycardia phases (> 10 sec with 30 beats/minute) were observed. b-blocker therapy is left in place for known tachy-brady-syndrome. Therefore, pacemaker implantation is indicated for recurrent SVTs, recurrent bradycardia up to 3.8 sec, and syncope in the history. Preoperatively, the patient was asymptomatic, dyspnea NYHA I-II, arterial hypertension, type II diabetes, chronic renal failure (GFR 38 mL/min/1.7m²; Creatinine: 1.3 mg/dL) and hyperparathyroidism. Preoperative transthoracic echocardiography (TTE) showed mild tricuspid regurgitation, moderate pulmonary hypertension (sPAP: 41.0 mmHg, PV vmx: 69.9 cm/sec), with well-preserved RV function. Global left ventricular function was normal, both atria were enlarged, and the mitral valve was minimally insufficient. The pulmonary valve is unremarkable. The inferior vena cava is borderline wide with diameter 22mm. The chest X-ray at admission revealed no signs of right heart decompensation, non-significant enlarged heart, or pleural effusions (**Figure 1A**).

Dual-chamber implantation was performed. The venous access on the left sided vena subclavia and vena cephalica was not accessible after multiple attempts. As next step, the left sided access was closed, and the ILR explanted. Because of the excessive time of surgical procedures, a sedoanalgesia and laryngeal mask intubation was performed. Followed by pacemaker lead implantation by preparation of the right Vena cephalica. The atrial lead was positioned into the right heart appendix, the RV-lead to the apex of the right ventricle. Measured values of the pacemaker-leads showed regular functions. The patient was hemodynamically and respiratory stable and extubated immediately.

Initially symptom-free, the condition worsened overnight. The patient showed increasing thoracic pain and cough irritation. She was cold sweaty, auscultatory wet rales, and sO₂ on room air was only 82%, which increased to 96% saturation on 4 liters of O₂. The patient remained hemodynamically stable. In X-ray, pneumothorax could be excluded (**Figure 1B**). The ECG was bland, cardiac enzymes and the rest of the laboratory were unremarkable except for a d-dimer of 2.35. The values of the pacemaker were stable and did not indicate a perforation. Antibiotics were started if silent aspiration was suspected.

Bedside TTE presented with a) left thoracic pleural effusion, and circumferential minimal pericardial effusion (without hemodynamically relevance), b) unremarkable left ventricular function (LVF) at normofrequency, minimal TVR, and severe pulmonary hypertension (sPAP: 59.0 mmHg; PV Vmx: 79.9 cm/s). Because of the sudden onset, and elevated d-dimer, pulmonary embolism was ruled out by computer tomography.

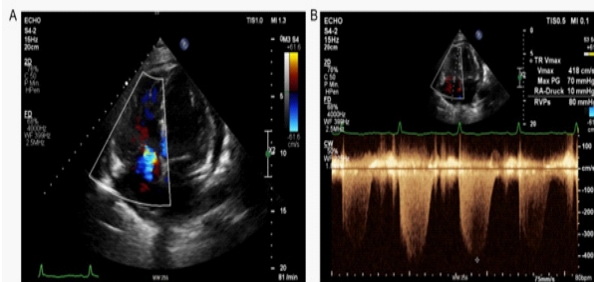


Figure 1: Preoperative vs. postoperative (48 hrs.) chest X-ray. Blue arrow: postoperatively (B) new described angular effusion left and retrocardiac consolidation left, with left accentuated mild to moderate signs of congestion, compared to prior surgery. Red arrow: RV-lead through the tricuspid valve.

From the third to fifth postoperative day, the patient developed fever, antibiotics were started, blood cultures were taken, which showed a negative result in the inpatient course. The laboratory showed increasing renal insufficiency, NT-proBNP and troponin-T increased, and C-reactive protein (CRP) and leukocytes showed continued increased values. Oxygen saturation remained limited with 84% O₂ on room air. Another TTE during the course presented the following finding: evidence of increasing right heart failure. The right ventricle appears enlarged from pre-examination, as well as an increase in TVR to now at least moderate, with two jets and concomitant severe pulmonary hypertension (sPAP: >80 mmHg) (**Figure 2**). The ECG showed a new onset atrial fibrillation not responding to amiodarone. There is also an increase in pericardial effusion, as well as conspicuous findings in the X-ray (**Figure 3**). Therefore, it is decided to revise the patient immediately. Re-measurement of the pacemaker data showed an atrial pacing rate of more than 78%. On the eighth day after initial surgery, the transient downgrading to AAIR was performed without complications by explanting the RV lead. A pericardial drainage via sub-xiphoidal access and a left thoracic drainage were performed. An intra-operative TEE showed an immediate improvement of TVR from mild to moderate insufficiency. Due to the increased inflammatory parameters, immediate re-implantation of a dual-chamber pacemaker using an LV-lead instead of an RV-lead was not considered.

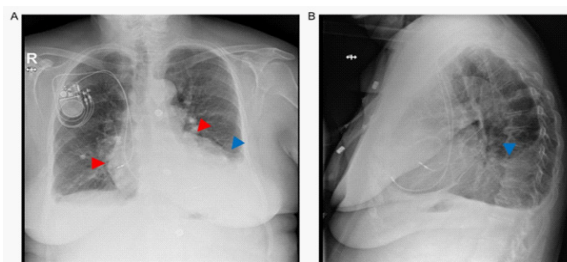


Figure 2: Postoperative transthoracic echocardiography (48 hrs. post-surgery). Left (A): A few days after pacemaker implantation, TEE showed a marked increase in tricuspid valve regurgitation from mild to moderate. Right (B): Measurements of TVR of TTE.

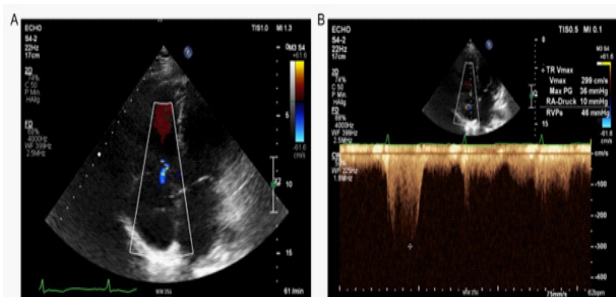


Figure 3: Preoperative chest X-ray prior to RV-lead revision. Blue arrow: Significant increase of retrocardiac consolidation left, with left accentuated moderate to severe signs of congestion. Red arrow: Signs of pericardial effusion with extension of the heart shade.

The postoperative course after the second surgical procedure was unremarkable. Symptoms such as dyspnea, massive leg edema and fatigue immediately changed to an upward trend. During the operation, the laboratory values as well the TTE were normalized, only the renal values stagnated at a value of 1.2 mg/dL creatinine, in the sense of a preoperatively known slight renal insufficiency. After removal of the drainage, there was no further accumulation of pericardial or pleural effusion in the further observation period of 7 days (**Figure 4**).

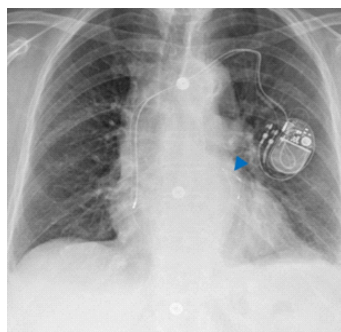


Figure 4: Transthoracic echocardiography (TTE) after pacemaker lead explantation. Left (A): Duplex TEE of tricuspid valve regurgitation showed significant reduction of TVR after RV-lead explantation. Right (B): Measurements of TVR of TTE.

The pacemaker implantation site was conspicuous for chronic *Staphylococcus epidermidis* infection with chronic pain within the next three month. Due to partially persistent symptomatic SSS, as well as new-onset AV-block II° type Wenckebach, the patient underwent further revision with complete system explantation, as well as contralateral implantation of a dual-chamber implantation with use of an LV-lead for ventricular pacing (Evity 8 DR, Fa. BIOTRONIK SE & Co. KG; Germany) (**Figure 5**). Further existing atrial fibrillation was not observed during follow-up.

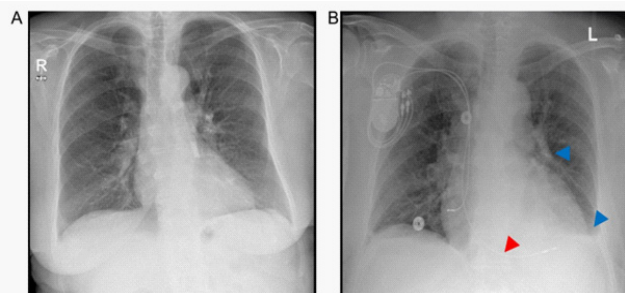


Figure 5: Postoperative chest X-ray two days after two-chamber pacemaker-upgrade with a LV-lead instead of a RV-lead. Blue arrow: Position of sinus coronarius-lead showed no need for left-ventricular position for adequate pacing with excellent pacemaker values.

Discussion and Conclusion

This particular case highlights the importance of RV- or LV(CS)-lead implantation in patients with corresponding risk factors. Of course, this postoperative course was not predictable or 100% avoidable with alternative choice of optional ventricular lead placement, but based on the prior literature, it was at least a case to reconsider the decision of proper ventricular lead placement or to address it interdisciplinarily. A meticulous review of the literature reveals several indications that, in appropriate cases, a well-considered decision based on current literature, at least in interdisciplinary terms, is necessary. Most previously, a variety of authors describe in selected case reports, as well as large-scale studies, that pacemaker leads positioned in the RV by passing the tricuspid valve, led to i) iatrogenic cause of RV dilatation and dysfunction [3,7,9] and ii), a higher incidence of lead-induced TVR or worsening of TR severity [2,4-6,9].

Even though the current guidelines have not yet addressed it. This may be due to the fact that the studies either have a very short follow-up (<2 years), nor that most of the studies fail to focus on the percentage of the stimulus rate. Thus, an 80% RV pacing is more susceptible to impending RV insufficiency because of abnormal physiology compared with only a 15% RV pacing. Therefore, we highlighted the importance of alternative ventricular lead placements including passing the coronary sinus instead of going through tricuspid valve in patients with corresponding risk factors to avoid a TVR and improve clinical long-term outcome. According to the literature, some cases of worsening of existing minimal to moderate TVR, pre-existing pulmonary hypertension and right heart dilatation, which ultimately leading to RHF, can be prevented.

In summary, this suggests that in patients at risk of worsening TVR, particularly those with preexisting TV disease, left ventricular pacing may be considered to reduce the likelihood of worsening TVR. According to the 'minimalist' recommendations of various cardiac societies, the use of an LV lead in patients with appropriate risks should be adopted in the appropriate guidelines. Just because LV leads are sometimes more difficult to place should not be a reason to accept a possibly worse clinical outcome.

Acknowledgements: Not applicable.

Funding: Not applicable.

Contributions: TA, DG, and VL collected patient data. TA and DG analyzed patient imaging. All authors were active in manuscript drafting. All authors read and approved the final manuscript.

Ethics declarations

Ethics approval and consent to participate: Informed consent was obtained from the patient for publication of anonymized case details.

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

Competing interests: The authors declare that they have no competing interests.

Data Availability Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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