

Entrapment of Permanent Pacemaker Ventricular Lead in a Loop Formed by a Temporary Pacemaker Electrode. How to Untie a Tight Knot?

Roman Nevzorov MD¹, Sergey Litvin MD², Michael Knizhnik MD², Boris Strasberg MD¹ and Moti Haim MD¹

¹Departments of Cardiology and ²Diagnostic Radiology, Rabin Medical Center (Beilinson Campus), Petah Tikva, associated with Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel

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An unusual obstacle happened during implantation of a ventricular permanent pacemaker (PPM) lead in a patient with a temporary pacemaker (TPM). The PPM electrode was entrapped in a knot created by a loop of the TPM electrode. The external part of the TPM electrode was cut and the lead with a knot was extracted by snaring it through the left femoral vein.

PATIENT DESCRIPTION

A 71 year old patient underwent transcatheter aortic valve implantation (TAVI)

and developed a complete atrioventricular block during the procedure. A temporary wire was introduced as back-up via the right subclavian vein and was left in place. During the following 24 hours the patient remained pacemaker-dependant and was transferred to the electrophysiology laboratory for permanent pacemaker implantation.

A dual chamber pacemaker was implanted through the left subclavian vein.

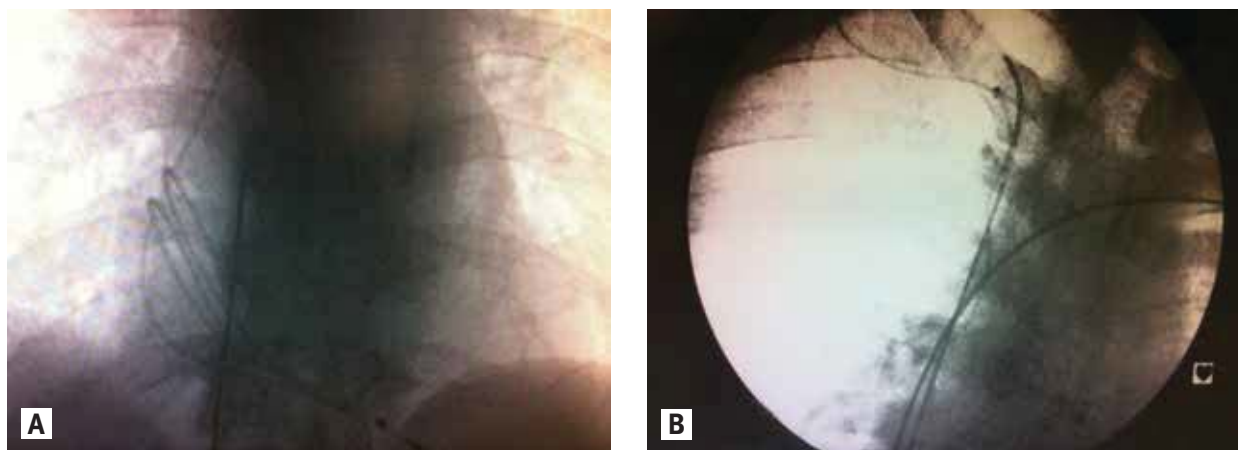
At the end stages of the procedure (when the electrodes were already positioned in the right ventricle apex and right atrial appendage) the temporary wire was carefully pulled out of the right ventricle (RV). However, a loop that was created while placing the temporary pacemaker caught the permanent ventricular lead that was inadvertently placed through the temporary wire loop [Figure 1A]. As we tried to pull out the temporary electrode, it formed a tight knot on the permanent ventricular lead, which

was partially extracted from the apex while pulling the temporary lead. We lost capture for a few seconds. The permanent lead was pushed forward and placed immediately back to the RV apex but then the previously open loop of the temporary wire created a knot that entrapped the permanent wire making it impossible to pull the temporary wire out without risking pulling the permanent lead as well [Figure 1B].

A second ventricular lead was placed in the RV apex via the left subclavian vein.

After multiple attempts, we succeeded in removing the trapped permanent ventricular lead from the loop of the temporal pacemaker electrode. We were left with the temporary wire through the right subclavian vein with a sizable knot making it hazardous to pull it through the vein, which could tear, making it difficult to control bleeding at the site. The external part of the temporary pacemaker electrode

Figure 1. [A] A loop that was created while placing the temporary pacemaker [B] A knot created by the previously open loop of the temporary wire



was cut and the lead with knot was successfully extracted by snaring through the left femoral vein approach. The patient did not develop a groin or pocket hematoma and was discharged the following day.

COMMENT

Atrioventricular block is a frequent complication of transcatheter aortic valve implantation [1]. Permanent pacemaker implantation after TAVI is safe and can be performed without an increased rate of complications [2]. We report the case of an unusual difficulty during the replacement of a temporary pacemaker with a per-

manent pacemaker. We have found only a few cases of intracardiac knotting of a transvenous pacemaker [3-5]. This complication should be anticipated and can be prevented by releasing the loop that was formed in the right atrium by the temporary wire before advancing the permanent electrode and also by making sure that the length of the electrode that is introduced into a cardiac chamber is not too long to prevent loop formation and potential knot formation [5].

Correspondence

Dr. R. Nevezorov

Dept. of Cardiology, Rabin Medical Center (Beilinson Campus), Petah Tikva 4941492, Israel

Phone: (972-3) 937-7100, **Fax:** (972-3) 921-3221
email: nevezorovroman1975@gmail.com

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Capsule

Physical activity to reduce fatigue in rheumatoid arthritis: a randomized controlled trial

Effective treatments for rheumatoid arthritis (RA) fatigue are limited. **Katz** and colleagues tested the effect of a pedometer-based intervention on increasing physical activity and decreasing fatigue among individuals with RA. Participants completed baseline questionnaires; engaged in 1 week of activity monitoring; were randomized to control (education [EDUC]), pedometer and step-monitoring diary (PED), or pedometer and diary plus step targets (PED+) groups, and were followed for 21 weeks. At week 10, questionnaires were administered by phone to all participants. During the final week, all participants again had 1 week of activity monitoring. A total of 96 individuals participated. Eight did

not complete the 21-week assessments. Both intervention groups significantly increased steps (+1441 [$P = 0.004$] for PED and +1656 [$P = 0.001$] for PED+), and the EDUC group decreased steps (-747 [$P = 0.14$]) (group-by-time interaction $P = 0.0025$). Between-group changes in fatigue were not significantly different (interaction $P = 0.21$). Mean changes in fatigue scores from baseline to week 21 were -1.6 (with-group $P = 0.26$), -3.2 ($P = 0.02$), and -4.8 ($P = 0.0002$) for EDUC, PED, and PED+ groups, respectively. Function and self-reported disease activity also improved in the PED and PED+ groups.

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Eitan Israeli

Capsule

CD22-targeted CAR T cells induce remission in B-ALL that is naive or resistant to CD19-targeted CAR immunotherapy

In chimeric antigen receptor (CAR) T cells targeting CD19 mediate potent effects in relapsed and/or refractory pre-B cell acute lymphoblastic leukemia (B-ALL), antigen loss is a frequent cause of resistance to CD19-targeted immunotherapy. CD22 is also expressed in most cases of B-ALL and is usually retained following CD19 loss. Fry and colleagues reported results from a phase 1 trial testing a new CD22-targeted CAR (CD22-CAR) in 21 children and adults, including 17 who were previously treated with CD19-directed immunotherapy. Dose-dependent antileukemic activity was observed, with complete remission obtained in 73% (11/15) of patients receiving $\geq 1 \times 10^6$ CD22-CAR T cells per kg body weight, including 5 of 5 patients with

CD19^{dim} or CD19⁻ B-ALL. Median remission duration was 6 months. Relapses were associated with diminished CD22 site density that likely permitted CD22⁺ cell escape from killing by CD22-CAR T cells. These results are the first to establish the clinical activity of a CD22-CAR in B-ALL, including leukemia resistant to anti-CD19 immunotherapy, demonstrating potency against B-ALL comparable to that of CD19-CAR at biologically active doses. These results also highlight the critical role played by antigen density in regulating CAR function.

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Eitan Israeli