PaceMaker lead and atrial thrombosis, a rare event but of high clinical importance

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SOUHRN

Kontext: Vznik trombózy po zavedení transvenózní elektrody představuje podceňovaný problém. Přes její vysokou incidenci zůstává většina případů nediagnostikovaných, protože je klinicky němá. Byla popsána řada rizikových faktorů predisponujících k tvorbě sraženin na elektrodách přístroje. Protože nejsou k dispozici žádné doporučené postupy (guidelines) ani důkazy ohledně optimální léčby, záleží způsob odstraňování endokavitárních sraženin z elektrod kardiostimulátoru na individuální volbě mezi chirurgickou extrakcí katétrem, trombolýzou nebo antikoagulační léčbou. U některých pacientů může léčbu dále komplikovat křehká rovnováha mezi krvácivými a trombotickými příhodami.
Popis případu: Popisujeme případ 86letého muže se srdečním selháním a silně sníženou ejekční frakcí v důsledku chronické ischemické kardiomyopatie. Bylo u něj zjištěno několik komorbidit a v anamnéze měl hlubokou žilní trombózu i závažné krvácení. U pacienta došlo k rozvoji plicní embolie: v místě vzniku tromboembolie visela na elektrodě kardiostimulátoru sraženina.
Závěr: Antikoagulace s antagonisty vitaminu K umožnila poměrně bezpečné řešení problému s trombózou (nebo tromboembolií) a krvácením.

Keywords:
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Pace maker
Pulmonary embolism
Transesophageal echocardiography
Transthoracic echocardiography
Vitamin K antagonists

ABSTRACT

Background: Trans-venous lead-related thrombosis is an undervalued issue. Despite its large incidence, the majority of cases remain undiagnosed because clinically silent. Many risk factors are described as predisposing to clot formation on device leads. Since the lack of guidelines or evidences regarding the best therapeutic option, treatment of endocavitary clots on pacemaker wire is left to individual decision between surgical catheter extraction, thrombolysis or anticoagulation therapy. In some patients, the labile equilibrium between hemorrhagic and thrombotic events may further complicate management.
Case presentation: We report the case of a 86-y-o man with HF severely reduced EF due to chronic ischemic cardiomyopathy. He had several co-morbidities and both history of DVT and major bleeding. He developed PE: a clot hanging the PM wire was identified as the site of origin of thrombo-embolism.
Conclusion: Anticoagulation with VKA allowed a relatively safe control of either thrombotic (and thromboembolic) and bleeding issue.

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Introduction

Trans-venous lead-related thrombosis is undervalued. It is estimated to occur up to 45% among patients underwent device implant. Cumulative incidence of wire-related thrombosis was estimated around 23% at 1 year from implant with the majority of events occurred within the first three months after procedure without clinical sequelae.[1–8] Mostly events remain undiagnosed because clinically silent. Symptoms, which are generally related to deep vein thrombosis (DVT) in the electro-catheter (E-C) insertion site, occurs in only 1–3% of cases and sometimes appear lately, revealing a severe condition with congestive heart failure (CHF), shock and finally death.[2,9–13] Female sex, age >71 y-o, major surgery/immobilization, active cancer, high Body Mass Index (BMI), personal history of thrombosis, coagulation disorders and the absence of anticoagulant therapy are well known risk factors for DVT and, so long, for device lead-related thrombosis. According to Ata Soleimani Rahbar et al., patients with atrial fibrillation (AF) have 8 times higher (OR 8) risk to develop thrombus on endocavitary leads.[14–17] CHF and either pacemaker leads number and material have been described as important predisposing factors in clot formation on device leads.[3,14,18,19] Diagnosis is echocardiographic: classical Echo-Color-Doppler (ECD) may be sufficient, but in case of endocavitary thrombus trans-esophageal echocardiography (TEE) is required.[20,21] Sometimes differential diagnosis between thrombus and vegetation can be challenging. Positron emission tomography (PET) with fluorodeoxyglucose (FDG) or single photon emission, computed tomography (SPECT) can be helpful in making the correct diagnosis in these situations.[18,22–24] Since the lack of evidence about the better choice in terms of mortality, treatment of endocavitary clots on pacemaker wire is left on individual decision. Clot size and site and symptoms gravity are the main determinants of therapeutic approach. Surgical extraction is a suitable option for "fit" patients with big thrombus. Other possibilities are percutaneous intervention or medical therapy. Either anticoagulation or thrombolysis are reported in literature as effective in dissolving endocavitary clots.[2,9,14,25,26] Aim of this case report is to share our experience of pace maker (PM) wire-related thrombosis in order to add one more step, albeit very little, in the clinical management of similar situations.

Case report

A 86-year-old smoker, chronic kidney disease (stadium IIIa), BMI 28 and from ten years a history of psoriasis.[27-31] A 86-year-old patient was referred to our emergency department for atypical chest pain and dyspnoea. He had history of CHF with reduced ejection fraction (EF) (25%, New York Heart Assocation [NYHA] class II) secondary to chronic coronary artery disease (age-indeterminate silent inferior acute myocardial infarction, stenting of anterior descendent in 2007 and critical occlusion of obtuse marginal branch and first diagonal branch with no indication to treat).[5,6] In October 2019 he had a re-acutization of heart failure (HF) requiring hospitalization due to new-onset atrial flutter. Sinus rhythm was restored with intravenous Amiodarone infusion and he was successively discharged without oral anticoagulation. In the light of severe bradycardia episodes recorded during telemetry monitoring, he underwent single-lead PM implantation before discharge. One month later, at ambulatory evaluation, PM interrogation showed permanent AF and rivaroxaban 110 mg BID was than prescribed.[17,32,33] Three days later he came back with edema and pain at superior left limb, which was also warm and blushed. Patient reported to have started anticoagulation therapy just the day before. ECD demonstrated DVT *in situ* of PM electro catheter insertion.

He was discharged with indication to continue rivaroxaban. At three days, the ECD evaluation reported an improvement, either clinical and echocardiographic, of DVT.

In March he was hospitalized again, this time because of severe anemization (hemoglobin 6.4 g/dl) requiring transfusion. EGDS was performed without significant findings. At colonoscopy hyper-plastic bleeding polyps with no surgical indication were found. Due to his high bleeding risk profile, he was discharged with indication to

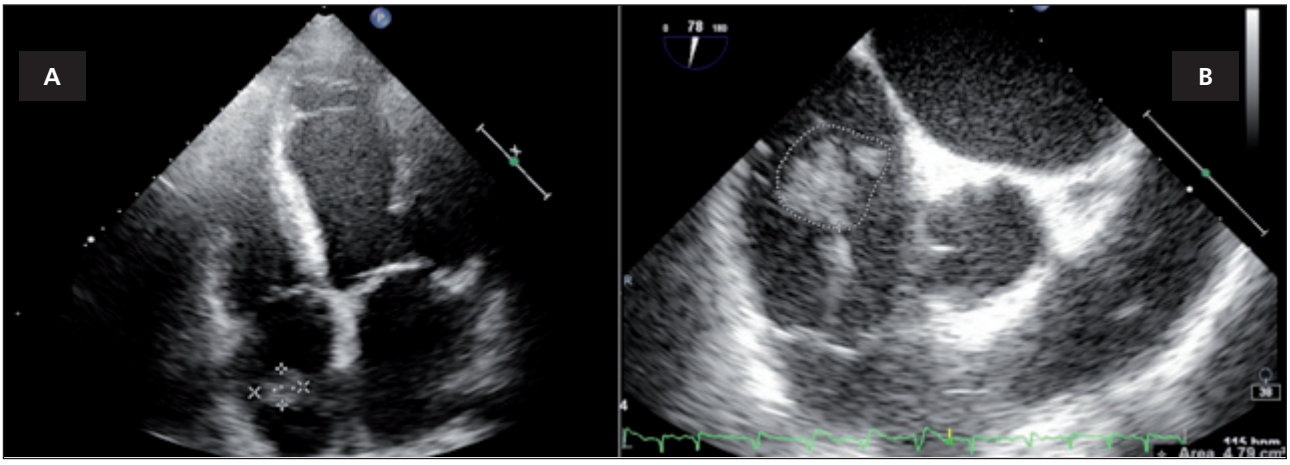

Fig. 1 – Right atrial thrombus seen in apical 4 chambers at transthoracic echocardiography (A) and at transesophageal echocardiography (B) at first presentation.

dismiss Rivaroxaban. 20 days later, dyspnoea and atypical chest pain led him to hospital again. 12-lead ECG showed AF with PM-induced ventricular rhythm. Hemoglobin was 12.6, C- reactive protein 8.1, NT-pro brain natriuretic peptide 30.000, glomerular filtration rate 46, troponin I was negative and thyroid function normal. Pulmonary angio computed tomography (CT) was performed with diagnosis of massive pulmonary embolism (PE). Trans thoracic echocardiography (TTE) enlightened a severe reduction in right ventricle function (tricuspidal anular plane systolic excurtion [TAPSE][11]) and a mass in right atrium hanging the wire. TEE was then performed showing hyper-reflective material with numerous "arborizations" (maximum cross-sectional size: 3 cm; area 5 cm^2). The mass had high excursion and motility. He never had fever at home (neither he had during hospitalization). No endocarditis-suggestive signs were enlightened at physical examination and PM pouch was clean, showing no signs of infection. Intravenous heparin (with activated partial thromboplastin time monitoring) was started as pulmonary embolism therapy.[34–36] In order to rule out endocarditis, PET with FDG and blood cultures were both performed and found negative. Infection was definitively excluded. Suspecting a paraneoplastic origin of the thrombus, patient underwent total body computer tomography, which showed meningioma and a primitive formation with evolutive nature in the right kidney (diameter: 14 mm).

Collegial discussion with cardiac surgeons excluded catheter extraction due to patient's multiple co-morbidities which made him too frail to underwent cardiac surgery. Thrombolysis was also excluded because of the intracranial neoplasm and the recent anemization. Oral anticoagulant therapy with vitamin K antagonists (VKA) was then started (international normalized ratio [INR] target 2.5–3.5).

Pre-demission echocardiography reported an improvement in right ventricle longitudinal function (TAPSE 16) in comparison to admission. At 1-month follow-up no further embolic neither hemorrhagic events were reported. Blood chemistry showed stable hemoglobin (12 g/dl) and INR was 2.9. Echocardiography showed a partially reduction in mass dimension (cross-sectional size 2.5 cm, area 4 cm^2).

Discussion

In clinical practice borders between thrombotic and bleeding risk is often blurred, making the decision challenging despite scores and guidelines. Particularly in elderly people with numerous co-morbidities, risk factors for thrombotic and hemorrhagic events are usually co-existing. This makes the choice to whether or not prescribe anticoagulant extremely difficult since it means substantially which event to risk: hemorrhagic or thrombotic. Sometimes there is no right or wrong answer, but consequences can still be severe. The case reported showed both sides of the coin: at first, therapy with direct oral anticoagulants (DOAC) led to a severe anemization requiring transfusion; then, interruption of anticoagulation therapy led to clot formation on PM wire, reflecting the labile balance between these two entities.

In this context, the evidence of a clot hanging the PM lead can be even more complex to manage. Despite the lack of guidelines, in clinical practice symptoms and thrombus dimensions are common criteria in choosing or not E-C surgical extraction. However, co-morbidities and advanced age made our patient not suitable for surgery.

Thrombolysis could be a valid option, but age and recent major bleeding (occurred less than four weeks before) and intracranial neoplasm (meningioma) were respectively relatives and absolute contraindications to the procedure.[37] VKA were the only option available. In fact, despite their effectiveness in DVT and PE is well established, DOAC have demonstrated less efficacy in preventing embolization from endocavitary thrombi compared to VKA.[38–40] Moreover, if, in one hand, DOAC have been shown to be burdened with a lower bleeding risk than VKA, on the other hand the presence of an effective, cheap and simply available antidote to VKA allows an easier management of future possible bleeding episodes. Even more, as weekly INR monitoring is required during coumadin treatment, a closer follow up is possible with VKA and this aspect can earn much more relevance in those patients who are neglected and more prone to not assume therapy correctly.

Conclusion

According to our experience, and in accordance to what is reported in some clinical cases, VKA can be an effective option a in case of lead-related thrombosis, especially when surgery is not suitable due to patient performance status or thrombus characteristics and criteria for thrombolysis are not met or the bleeding risk is assessed as too high. Further studies are needed to establish the real effectiveness of VKA in this setting and to draw up the most correct guidelines for management of E-C-related endocavitary thrombi.

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