Right atrial thrombus secondary to pacemaker wires.

Alexander C Feuchter

Kenneth D. Katz MD
Lehigh Valley Health Network, kenneth_d.katz@lvhn.org

Published In/Presented At

Feuchter AC, Katz KD. Right atrial thrombus secondary to pacemaker wires. J Emerg Med. 2012 Sep;43(3):e185-7. doi: 10.1016/j.jemermed.2010.04.047. Epub 2010 Jul 24.

The Journal of Emergency Medicine, Vol. 43, No. 3, pp. e185–e187, 2012

0736-4679/$ - see front matter

ELSEVIER

doi:10.1016/j.jemermed.2010.04.047

Clinical Communications: Adults

RIGHT ATRIAL THROMBUS SECONDARY TO PACEMAKER WIRES

Alexander C. Feuchter, MD,* and Kenneth D. Katz, MD†

*University of Pittsburgh Affiliated Residency in Emergency Medicine, and †Department of Emergency Medicine, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania

Reprint Address: Alexander C. Feuchter, MD, Affiliated Residency in Emergency Medicine, University of Pittsburgh Medical Center, 230 McKee Place, Suite 500, Pittsburgh, PA 15213

☐ **Abstract—Background: Pacemaker-induced right atrial thrombus is a rare condition that has not been described in the Emergency Medicine literature. This is a potentially fatal condition that is diagnosed with an echocardiogram and treated with surgical removal, thrombolytics, or long-term anticoagulation. Objectives: This case report is designed to increase awareness among emergency physicians of this potentially fatal condition. Case Report: We describe the case of a patient with a massive right atrial thrombus secondary to pacemaker wire who presented to the Emergency Department with syncope, bradycardia, and rapid hemodynamic deterioration. Conclusion: Emergency physicians should be aware of this life-threatening entity. Emergency bedside cardiac ultrasound or echocardiogram may be of value in its early identification.** © 2012 Elsevier Inc.

☐ **Keywords—pacemaker; atrial thrombus; syncope; echocardiography; AICD**

INTRODUCTION

Implanted pacemakers are a common and safe treatment for bradydysrhythmias. However, serious thrombotic and embolic complications of implanted cardiac pacing occur in 0.6–3.5% of cases (1). Clinical presentations include: asymptomatic findings on routine echocardiogram, dyspnea, embolic stroke, and unstable cardiac dysrhythmias (2–5). Because this clinical entity has not been described in the emergency medicine literature, this case report serves to make emergency physicians aware of the potentially life-threatening complications.

CASE REPORT

A 66-year-old man with hemodialysis-dependent end-stage renal disease, diabetes mellitus, severe ischemic heart disease with dual chambered pacemaker, and automated implantable cardioverter-defibrillator (AICD) was transported to the Emergency Department (ED) by private vehicle after a syncopal episode earlier that evening. He reported walking to the kitchen when he suddenly lost consciousness. He was found within seconds by his wife, regained consciousness immediately, and was transported to the ED. The patient stated that he had been feeling generally unwell for the past 3 days without other accompanying complaints except for intermittent shortness of breath. The AICD had not fired, and he had been dialyzed on schedule 2 days before presentation. His medications included: digoxin, calcium acetate, gabapentin, aspirin, carvedilol, Humulin R (Eli Lilly and Company, Indianapolis, IN), allopurinol, colchicine, and NEPHRO-CAPS (Fleming Pharmaceuticals, Fenton, MO).

Initial vital signs included: paced heart rate of 44 beats/min, blood pressure of 102/53 mm Hg, room air pulse oximetry 99%, temperature 36.8 °C, and respiratory rate 16 breaths/min. In general, he was alert and oriented and in no acute distress, with a Glasgow Coma Scale score of 15. Physical examination revealed only

RECEIVED: 10 October 2009; FINAL SUBMISSION RECEIVED: 19 January 2010;
ACCEPTED: 11 April 2010

bradycardia and chronic bilateral pitting edema in the lower extremities. The patient was placed on a cardiac monitor, nasal cannula oxygen was administered, pacing pads were placed, and intravenous access was obtained. At that time, no additional intervention was performed due to the presence of stable bradycardia. An electrocardiogram (ECG) revealed a paced rhythm at 50 beats/min with unchanged ventricular morphology, compared with an ECG obtained 2 months prior. The QRS duration was 192 ms, and the QTc was 481 ms. Laboratory diagnostics were obtained and sent for analysis. However, before obtaining the results, the patient's clinical condition deteriorated rapidly within 30 min, with decreased responsiveness, severe dyspnea, and progressive bradycardia that deteriorated rapidly to asystole. Despite immediate endotracheal intubation, external pacing, Advanced Cardiac Life Support protocol implementation, and treatment of presumed hyperkalemia (avoiding calcium because serum digoxin level was unavailable at the time), the patient expired after a brief return of spontaneous circulation.

Laboratory studies, which were received after the time of arrest, included: sodium 129 mEq/L (136−146), grossly hemolyzed potassium 14 mEq/L (3.5−5) (repeat, also hemolyzed, 8.9 mEq/L), chloride 94 mEq/L (95−110), bicarbonate 17 mEq/L (21−31), blood urea nitrogen 77 mg/dL (5−20), creatinine 10.4 mg/dL (0.5−1.4), glucose 81 mg/dL (70−99), and troponin I 0.24 ng/mL (< 0.1). Complete blood count was normal, and serum digoxin level was undetectable. A chest radiograph done just before deterioration was unchanged from his previous study obtained approximately 2 months earlier that demonstrated cardiac enlargement, evidence of sternotomy and coronary artery bypass surgery, and a left subclavian dual-chamber pacing device.

Upon family request, an autopsy was performed, and a massive right atrial thrombus was found filling the entire right atrium, measuring 10.3 cm × 8 cm × 3 cm (Figure 1). Additionally, this thrombus extended proximally into the superior vena cava and distally into the right ventricle. Pacemaker lead wires were discovered and found to have contributed to thrombus formation. Prior right ventricular infarction was also found, as well as an old inferior wall infarction. The cause of death was determined to be the atrial thrombus secondary to pacemaker wire with ensuing obstructive sequelae.

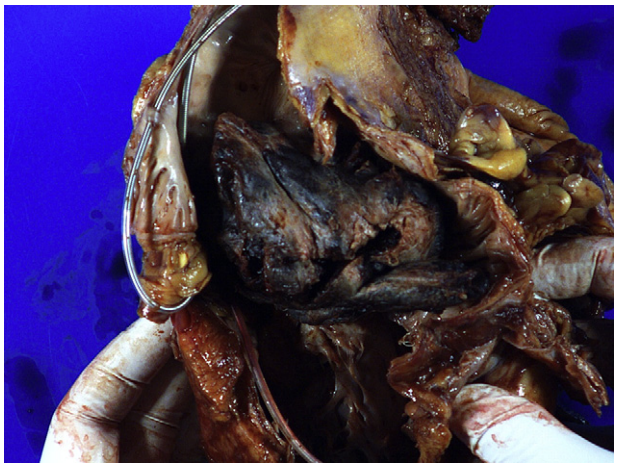

Figure 1. Postmortem photograph showing the large right atrial thrombus and pacer wires.

DISCUSSION

Clinically silent pacemaker lead thrombosis occurs in as many as 35−45% of all implants; however, serious complications from this are much rarer (1). Presenting complaints and clinical presentations vary and include: chest pain, cyanosis, edema, pyrexia, malaise, congestive heart failure, shortness of breath, and frank shock (5). Diagnosis is generally made via echocardiogram, which can demonstrate right atrial enlargement and hyperechoic masses in the right atrium (6). Thrombi are often seen with transthoracic echocardiography (echo), but transesophageal echo has greater sensitivity (3). Bedside transthoracic echo is probably the most appropriate initial diagnostic test, due to its easy availability and lack of dependence on patient stability. Cardiac computed tomography scan and cardiac magnetic resonance imaging have not been validated yet for this purpose (7). Treatment ranges from anticoagulation or thrombolysis to surgical removal. In the majority of cases, surgical removal has been the recommended and successful treatment, but anticoagulation, thrombolysis, and percutaneous extraction have been used with some success (7,8). Due to the rarity of this condition, treatment has not been extensively studied. Management decisions must be made on a case-by-case basis, but if a thrombus is detected, immediate cardiothoracic surgery and cardiology consultation is warranted. Anticoagulation is more likely to be considered in the stable patient. Factors predisposing individuals to development of pacemaker lead thrombosis include congestive heart failure and hypercoagulable states (2). Diagnosis is rarely made in the ED, because it is such a rare condition with a nonspecific clinical presentation.

Due to this patient's hemodynamic compromise and size of the thrombus, surgical removal probably would have been the only treatment. However, the broad differential diagnosis engendering the clinical presentation (that included hyperkalemia, acute coronary syndrome, digoxin toxicity, pericardial effusion, pneumonia), coupled with the patient's rapid decompensation, would have made surgical thrombectomy extremely difficult to perform in a way that would affect the patient's outcome.

CONCLUSION

Atrial thrombus is an important diagnosis to consider in the setting of non-specific cardiopulmonary complaints or unexplained hemodynamic compromise in a patient with an implanted pacemaker/AICD. Rapid bedside ultrasound or early cardiology consultation and bedside echocardiogram may be warranted in similar patients, especially if initial and typical diagnostics are unrevealing.

REFERENCES

1. Barakat K, Robinson N, Spurrell R. Transvenous pacing lead-induced thrombosis: a series of cases with a review of the literature. Cardiology 2000;93:142−8.
2. Schifter D, Kozer L, Saul B, et al. An unusual case of multiple right atrial thrombi in a patient with a dual-chambered pacemaker. Angiology 1999;50:855−8.
3. Tugcu A, Ozlem Y, Tayyareci Y, et al. Right atrial pacemaker lead thrombosis causing tricuspid inflow obstruction. Pacing Clin Electrophysiol 2009;32:262−4.
4. Ikonomidis I, Papadopoulos C, Flevari P, et al. Left atrial thrombus after biventricular pacemaker implantation. Eur J Echocardiogr 2009;10:582−4.
5. Coleman D, DeBarr D, Morales D, et al. Pacemaker lead thrombosis treated with atrial thrombectomy and biventricular pacemaker and defibrillator insertion. Ann Thorac Surg 2004;78:e83−4.
6. Parravicini R, Cocconcelli F, Barchetti M, et al. Massive thrombosis on right atrial pacemaker electrode. Tex Heart Inst J 1996;23:185.
7. Carda R, Almería C, Lennie V, et al. What to do with an atrial thrombus? Eur J Echocardiogr 2008;1:204−5.
8. Fabijanić D, Giunio L, Vujicić I, et al. Right atrial pacemaker lead thrombosis incidentally detected by transesophageal echocardiography. Coll Antropol 2005;29:159−61.