# CASE REPORT

# Unusual case of colonized pacemaker lead presenting with endocarditis, hemoptysis and tricuspid valve stenosis

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J-F Morin, R Sheppard, P Chamoun. Unusual case of colonized pacemaker lead presenting with endocarditis, hemoptysis and tricuspid valve stenosis. Can Respir J 2011;18(3):e48-e49.

The present report is the first to describe a case of hemoptysis caused by an endocardial pacemaker lead. In addition, the patient presented with endocarditis and tricuspid valve stenosis. Aggressive treatment consisted of surgical extraction of two pacemaker leads and one pacemaker battery, replacement of the tricuspid valve and implantation of a DDD-R epicardial pacemaker.

Key Words: Endocarditis; Hemoptysis; Infection; Pacemaker

The present case illustrates the potential complications associated with endovascular pacemaker leads. Clinical signs may be subtle; therefore, the threshold to investigate with echocardiography and treat aggressively should be low.

#### CASE PRESENTATION

A 60-year-old man was diagnosed with Wegner's granulomatosis in 2001. At that time, he underwent insertion of a left pectoral VVI pacemaker for sick sinus syndrome. Several months later, the pacemaker battery became colonized. The battery was removed and a new right pectoral VVI pacemaker was implanted.

In early November 2004, he was admitted to hospital for fever and hemoptysis. Chest radiography (Figure 1) revealed a free pacemaker lead floating into the right ventricular outflow tract and directed toward a consolidated right upper lobe.

Two blood cultures were positive for coagulase-negative staphylococcus. An echocardiogram revealed a left ventricular ejection fraction of 60%. There was moderate tricuspid stenosis, with a peak and mean gradient of 13 mmHg and 4 mmHg, respectively. There was moderate to severe tricuspid regurgitation, with a pulmonary artery systolic pressure of 46 mmHg. There was also moderate right atrial enlargement and mild to moderate pulmonary valve regurgitation. A pacemaker wire was seen in the right ventricular outflow tract. A computed tomography scan of the thorax (Figure 2) confirmed the pacemaker lead wedging into a segmental pulmonary artery branch with parenchymal consolidation distally. A coronary angiogram did not demonstrate significant coronary artery stenosis.

The patient was started on cefazolin 1 g intravenously every 8 h. Extraction of the floating pacemaker lead using the Cook-Byrd technique was unsuccessful. On November 12, 2004, the patient underwent open heart surgery with extraction of the two pacemaker leads and one pacemaker battery, in addition to tricuspid valve replacement with a 27 mm St Jude metallic prosthesis (St Jude Medical, USA) and insertion of a DDD-R epicardial pacemaker. There was massive fibrosis of the tricuspid valve leaflets and subvalvular apparatus, with severe tricuspid valve stenosis. Histology showed fibrous tissue with chronic inflammation, local acute inflammation and granulation tissue formation.

His postoperative course was uncomplicated. A repeat echocardiogram five days postoperatively revealed a well-seated tricuspid Un cas inhabituel de dérivation de stimulateur colonisée, entraînant une endocardite, une hémoptyse et une sténose de la valvule tricuspide

Le présent rapport est le premier à décrire un cas d'hémoptyse causé par la dérivation d'un stimulateur endocardique. De plus, le patient présentait une endocardite et une sténose de la valvule tricuspide. Le patient a subi un traitement énergique, composé de l'extraction chirurgicale de deux dérivations et d'une pile du stimulateur, du remplacement de la valvule tricuspide et de l'implantation d'un stimulateur épicardique DDD-R.

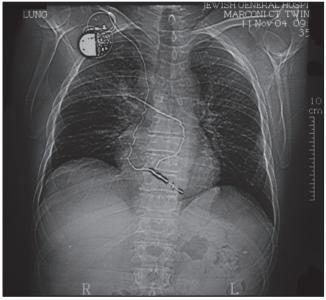


Figure 1) Chest radiograph showing the free pacemaker lead floating into the right ventricular outflow tract

prosthesis with moderate ventricular hypokinesis and a left ventricular ejection fraction of 55%. One week after surgery, he was discharged home on acetylsalicylic acid, the same preoperative dose of azathioprine and warfarin, and vancomycin 500 mg intravenously every 12 h for an extra five weeks. At six weeks follow-up, he was asymptomatic, with satisfactory pacemaker and prosthetic valve function. His chest radiography was normal.

## **DISCUSSION**

Tricuspid valve stenosis related to endocardial pacemaker lead migration is uncommon (1). No cases of hemoptysis caused by endocardial leads have been reported. In the present case, hemoptysis and positive blood cultures triggered further investigation of the patient. The

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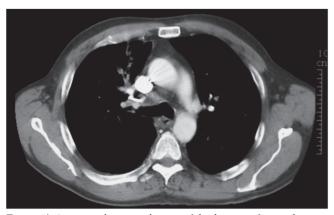


Figure 2) A computed tomography scan of the thorax confirming the pacemaker lead wedging into a segmental pulmonary artery branch with parenchymal consolidation distally

hemoptysis was related to parenchymal lung inflammation or ischemia (occlusion or embolic) due to the tip of the endocardial lead migrating into a branch of the right pulmonary artery (Figure 2). The combination of endocarditis, free-floating leads in the right ventricular outflow tract and hemoptysis mandated the extraction of the pacemaker lead.

With the Cook-Byrd technique (2) being unsuccessful, surgical extraction of the pacemaker leads was the only remaining option.

Moderate tricuspid valve stenosis and severe tricuspid regurgitation complicated the management of this patient. Despite the absence of symptoms or signs of tricuspid valve dysfunction, the valve had to be replaced at the time of the lead extraction. Among patients who have undergone pacemaker or defibrillator implantation, very few developed tricuspid valve fibrosis. The predisposing factor may have been perforation of a tricuspid valve leaflet at the time of the implantation.

Endocarditis caused by infection of pacemaker leads necessitates extraction of all leads. An echocardiogram cannot accurately determine which lead is colonized. In addition, the implantation of a prosthetic valve in the tricuspid position precludes the implantation of an endovascular lead. The present case illustrates the potential complications associated with endovascular pacemaker leads. Clinical signs may be subtle; therefore, the threshold to investigate with echocardiography and treat aggressively should be low.

### REFERENCES

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