

Clots without a cause: a case of large asymptomatic pacemaker lead-associated thrombi

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Pacemaker lead-associated thrombi are well-recognised phenomena that often remain clinically silent¹⁻⁴ but may present as heart failure or pulmonary embolism (PE).⁵ The aetiology is often unknown and management poorly defined. We report the case of a 61-year-old male with two large, incidentally detected right atrial thrombi attached to the pacemaker leads without clear predisposing factors, ultimately requiring surgical removal. This case highlights the challenges of managing asymptomatic lead-associated thrombi, particularly when very large.

Case

A 61-year-old gentleman underwent a routine annual follow-up transthoracic echocardiogram (TTE), which demonstrated two large right atrial masses associated with his pacemaker leads. There were no significant valvular abnormalities, and the left and right ventricular function were normal. His background includes a long-standing left bundle branch block and severe non-ischaemic cardiomyopathy, which had significantly improved following medical therapy and cardiac resynchronisation therapy with defibrillator (CRT-D) implantation 5 years ago. He is also in long-term remission from follicular lymphoma following chemotherapy and stem cell transplant in 2010. Transoesophageal echocardiography (TOE) 1 week later confirmed a 2.6×2.3cm mass encasing the leads and prolapsing through the tricuspid valve (Figure 1) with mild tricuspid regurgitation and no tricuspid stenosis, a smaller mass extending into the inferior vena cava and a patent foramen ovale (PFO) (Figure 2). Dabigatran was initiated.

Large thrombi related to the pacemaker leads, albeit atypical, were considered the most likely diagnosis. The differential diagnoses included vegetations, right atrial myxoma, metastatic malignancy, and lymphoma. Systemic wellness, a C-reactive protein (CRP) of 1mg/L (N<5) and negative

blood cultures made infective endocarditis extremely unlikely. Adherence of the masses to the leads rather than the interatrial septum argued against myxoma. A negative comprehensive procoagulant screen excluded antiphospholipid syndrome. Elevated lactate dehydrogenase (325U/L, N=120–150), low haptoglobin (<0.20g/L, N=0.30–2.00) and high reticulocytes (125x10⁹/L, N=10–100) raised concern for haemolysis as a prothrombotic factor; however, in absence of clinical features, this was attributed to secondary haemolysis from the thrombus itself. Positron emission tomography-computed tomography (PET-CT) demonstrated no increased fluorodeoxyglucose uptake to suggest malignancy. Thrombus was therefore considered most likely; however, repeat TOE after 3 weeks of dabigatran demonstrated no definite interval change in the size of the masses.

Cardiosurgical discussion unanimously favoured open-heart surgery over prolonged anticoagulation (including the option of switching dabigatran to subcutaneous enoxaparin or warfarin) or percutaneous aspiration, given the size of the masses and risk of complications—particularly the risk of paradoxical embolism across the PFO. The patient was bridged with intravenous heparin and underwent surgical removal of the two large masses (Figure 3), PFO closure, pacemaker lead extraction and implantation of a new epicardial CRT pacemaker to prevent future lead-associated complications. Histology confirmed simple thrombus without evidence of myxoma or malignancy, and there was no evidence of any structural abnormality of the pacemaker leads. The patient recovered well and was continued on lifelong dabigatran given the potential for a procoagulant state.

Discussion

The reported incidence of pacemaker lead-associated thrombus varies widely between 1.3 and 23%.^{6,7} Presentations range from incidental findings—as in this case—to obstruction, embolic

Figure 1: Mid-oesophageal four-chamber transoesophageal echocardiography (TOE), demonstrating a) the size of the larger right atrial thrombus.

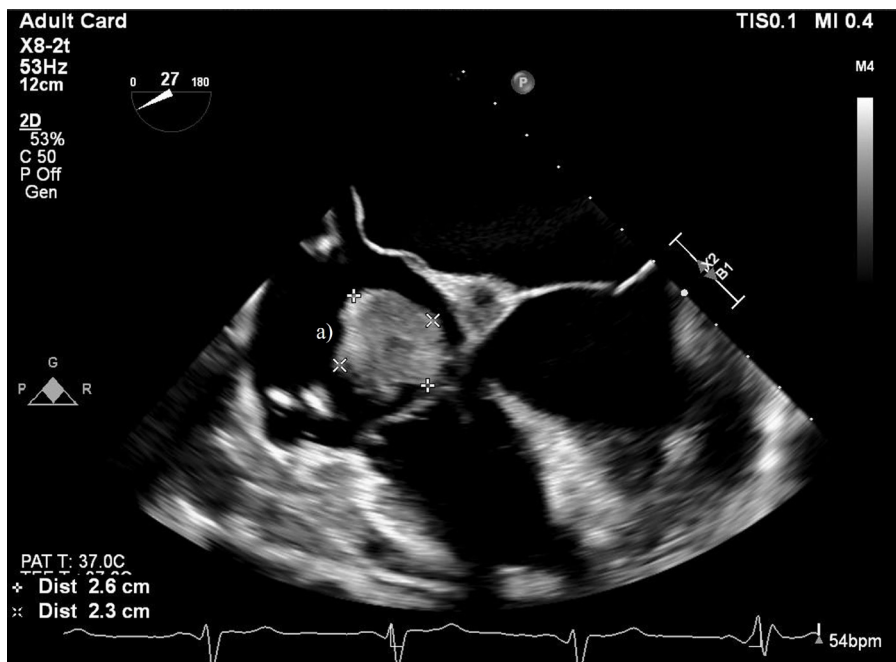


Figure 2: Mid-oesophageal right ventricular inflow-outflow tract transoesophageal echocardiography (TOE), highlighting a) the larger right atrial thrombus, b) the smaller thrombus extending into the inferior vena cava, and c) the patent foramen ovale.

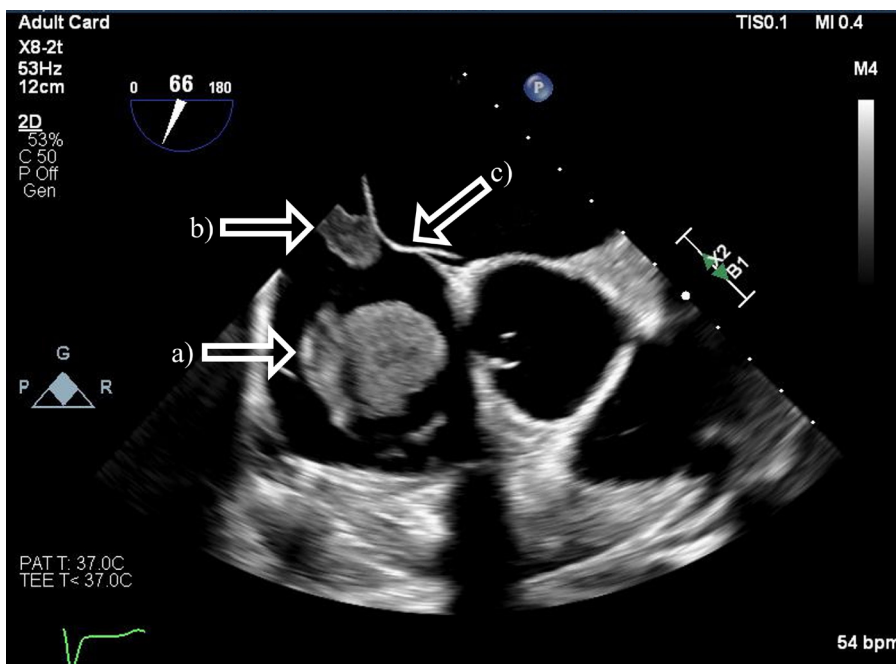
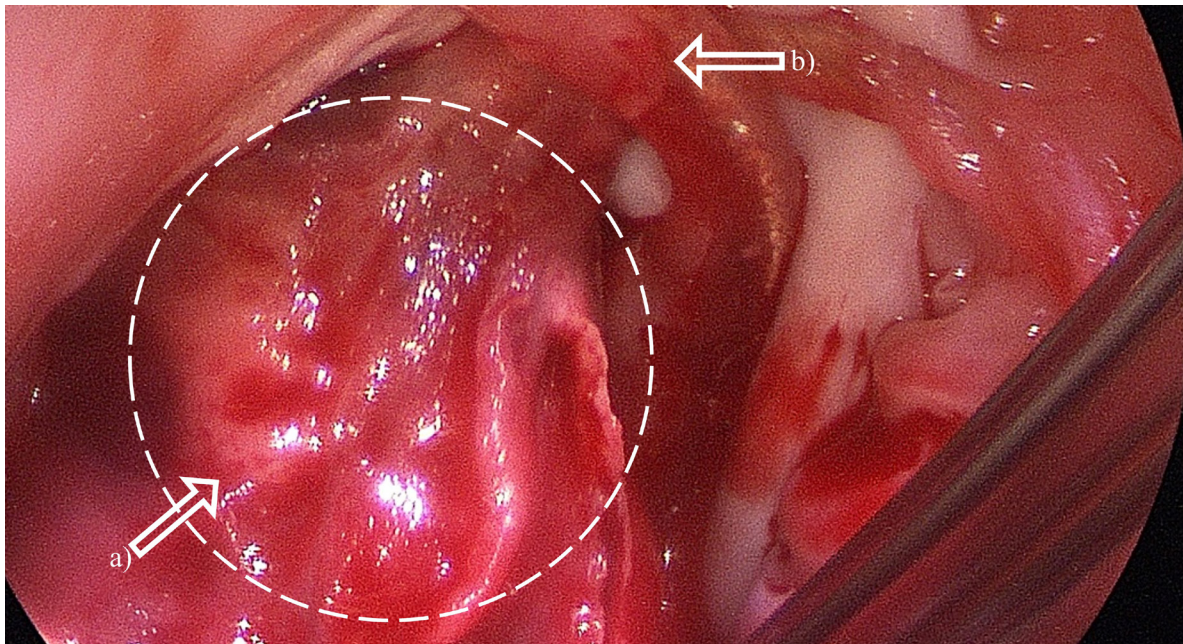


Figure 3: Intraoperative thoracoscopic view into the right atrium, highlighting a) the larger thrombus, and b) a pacemaker lead encased in thrombus.



events or right heart failure.¹⁻⁵

Thrombus formation is likely multifactorial, consistent with Virchow's triad.⁷ However, this patient had no active thrombotic disorder, malignancy or infection. The pacemaker leads were intact with no exposed prothrombotic surface. His preserved cardiac function made blood stasis less likely.

No consensus guidelines exist for the management of lead-associated thrombus. Thus, current practice is informed by case reports and expert opinion, rather than randomised data.⁸ Anticoagulation with warfarin or a direct oral anticoagulant is typically first-line therapy. Clinical outcomes vary; some thrombi resolve within weeks, while others persist chronically. Escalation of treatment to thrombolysis, percutaneous aspiration or surgical removal may be required.¹⁻⁴ The Heart Rhythm Society recommends lead extraction for clinically significant thromboembolism or venous obstruction,⁵ but the evidence is low-quality and provides no guidance for asymptomatic cases such as this.

Only four other case reports of large asymptomatic intracardiac thrombi have been published.¹⁻⁴ Three had identifiable risk factors; a 35-year-old female with chronic severe left ventricular dysfunction, and two males aged 71 and 62 years with non-anticoagulated permanent atrial fibrillation.

The fourth case involved an 83-year-old female with anticoagulated paroxysmal atrial fibrillation and no other apparent risk factors. The first two patients underwent surgical removal, the third patient was managed with prolonged anticoagulation after declining surgery, and the fourth patient underwent percutaneous thrombus aspiration.

Our patient's absence of identifiable risk factors and the persistence of his large thrombi despite anticoagulation for 3 weeks raised concerns for potential thrombus progression and related complications, including right heart failure from tricuspid valve obstruction or regurgitation, stroke from paradoxical embolism through the PFO,⁵ or PE.⁹ Given these risks, surgery was justified, although not without the inherent risks of embolism, bypass-related complications, and infection.

Conclusion

This case report of large, yet asymptomatic, pacemaker-related thrombi with no clear predisposing factors highlights the uncertainty of management in absence of established guidelines. Management remains individualised, requires shared decision-making and balances risks of complications with those of intervention. Surgical removal may be justified for large masses of

uncertain aetiology, even if asymptomatic, due to the potential of thrombus related complications of PE, right heart failure secondary to tricuspid valve regurgitation or stenosis, pacemaker lead

malfunction or stroke from paradoxical embolism across a PFO. Unusual masses should always prompt exclusion of malignancy.

COMPETING INTERESTS

Nil.

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