CASE REPORT

# Pacemaker lead thrombosis – a rare cause of breathlessness

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**How to cite this article:** Kelly MA, Davidson A, Griffiths K, Pessotto R, Leslie SJ. Pacemaker lead thrombosis – a rare cause of breathlessness. Arch Clin Cases. 2024;11(2):51-55. doi: 10.22551/2024.43.1102.10288

**ABSTRACT**

As the number of pacemaker insertions increases to manage numerous cardiac arrhythmias, the number of complications is also increasing as a result. More common complications such as infection and lead displacement are routinely discussed with patients before they undergo the procedure. However rare complications such as superior vena cava syndrome are not discussed during the consenting period. But they do occur, as seen in this case of a 69-year-old male. This fit and active man had a right-sided dual-chamber pacemaker inserted due to sinus node disease and presented 5 years later with symptoms suggestive of superior vena cava obstruction (SVCO). Despite anticoagulation and before surgical intervention could be performed, the patient developed a right-sided chylothorax which was drained. An autologous pericardial patch repair of the SVC and a thrombectomy of SVC clots was subsequently performed. This was only partially successful and the SVCO recurred. A low fatty chain diet was initiated to manage the chylothorax, which remains stable. This rare complication has left the patient with a small pleural effusion and chronic pleural thickening. They can still exercise with mild breathlessness. The management of such a complication, which requires the input of many specialists, is challenging and often does not completely resolve all symptoms. For this reason, superior vena cava obstruction should be considered as a risk during the consenting procedure for a pacemaker insertion.

**KEYWORDS:** chylothorax; pacemaker; SVCO; pleural effusion; complication

## INTRODUCTION

Superior vena cava obstruction (SVCO), also known as superior vena cava (SVC) syndrome, is a condition that occurs when there is obstruction of blood flow through the superior vena cava [1]. The diagnosis is made clinically with imaging being used as confirmation. The development of a collateral circulation means patients can compensate for a long time or can be entirely asymptomatic [2]. However, if symptoms are present, they often include shortness of breath and oedema of the head, neck, and arms. Patients also report flushing and headaches when bending forward [2,3].

External pressure, in the form of a mass in the mediastinum, is the most common cause of SVC obstruction. This mass can be malignant or non-malignant [1]. There is however an increasing number of cases caused by thrombosis of the SVC due to the insertion of pacemakers, implantable defibrillators, and central venous catheters [3]. Device-related complications are now responsible for 20-40% of cases [4]. As the pacemaker leads are advanced through the venous system, vessel wall inflammation and thrombus formation can occur which can result in obstruction [2].

**Received:** December 2023; **Accepted after review:** May 2024; **Published:** June 2024.

Treatment depends on the cause of the obstruction. One paper reports that balloon angioplasty and stenting can safely be used to treat pacemaker-induced SVCO, without disrupting the pacemaker wires – giving examples of three separate cases where this intervention was successful [5]. Surgery is currently used if endovascular interventions are not successful [2].

## CASE PRESENTATION

A fit and active 69-year-old man had a right-sided dual-chamber pacemaker implanted to treat sinus node disease causing symptomatic bradycardia. Venous access was obtained via the right subclavian vein as the patient was left-handed. A post-pacemaker chest x-ray showed adequate positioning of the pacemaker and no symptoms were noted.

Five years later, the patient presented with breathlessness and features suggestive of a superior vena cava obstruction (SVCO). These were exacerbated by exercise and lifting his arms above his head. Symptoms included edematous upper limbs, distended jugular veins and facial plethora.

An initial contrast CT of the neck and chest failed to identify an occlusion, in part due to artefact from pacing wires. High-dose direct oral anticoagulation (Rivaroxaban 15mg bd) was started, but despite this, symptoms worsened

and an invasive venogram with a 4 French sheath and pigtail catheter at the confluence of the brachiocephalic veins confirmed the presence of a tight web-like stenosis at the azygous vein level (Figure 1).

After a multidisciplinary team discussion with the pacing team, interventional radiologist and cardiac surgeons, open surgical intervention was thought to be a preferable approach to balloon angioplasty due to the long length of occlusion (6cm) and presence of clot.

Prior to surgery the patient re-presented with worsening breathlessness and was found to have a large right-sided pleural effusion (Figure 2). A Seldinger drain was inserted, which removed a total of 6.5 liters of lipid-rich fluid (Figure 3). The fluid was milky, pink, and grossly lipemic, with a triglyceride content of 24.6 mmol/l, which confirmed the diagnosis of a chylothorax (triglyceride concentration of $>$ 6.1 mmol/l) [6].

Further investigations were performed to rule out malignancy. No malignant cells could be identified under microscopy. A CT of the neck, thorax, abdomen, and pelvis showed no adenopathy or splenomegaly to suggest haematological malignancy (excluding lymphoma) and confirmed the chylothorax was caused directly by a superior vena cava (SVC) thrombosis. This was thought to be caused by pressure on the venous system, preventing chylous fluid from draining into the right subclavian vein above.

The patient underwent autologous pericardial patch repair of SVC and thrombectomy of SVC clots. However, this was only partially successful and ultimately the SCVO recurred.

To manage the chylothorax, the patient commenced a low-fat diet. Over the next 18 months, the small pleural effusion remained static, and a normal diet was slowly reintroduced with no worsening of the pleural effusion, presumably due to the development of collaterals.

At follow-up, the patient remains well, and despite right sided costophrenic angle blunting on imaging, likely due to small effusions/chronic pleural thickening (Figure 4), can exercise, albeit with ongoing breathlessness.

## DISCUSSION

Common complications of permanent pacemakers such as the formation of a pocket hematoma, lead dislodgement or infection, are discussed regularly with patients and can occur in up to 6% of all pacemaker insertions [7]. However, patients are not routinely informed of less common complications such as pacemaker-lead thrombosis and SCVO [8]. SVCO occurs in $<$0.1% of patients [9].

This patient initially attended with a suspected SVCO and imaging showed greatly reduced flow through the SVC, the patient was therefore anticoagulated. There were several therapeutic challenges in this case which warrant further discussion.

Initially, it was hoped that thrombus might be causing a significant proportion of the SVCO and that anticoagulation might result in clinical benefit. However, despite anticoagulation, symptoms continued and indeed worsened, an invasive venogram confirmed ongoing SVCO.

The choice of anticoagulant was also debated. The patient had been started on a direct oral anticoagulant (DOAC) Rivaroxaban at standard dosing. When symptoms persisted, a switch to warfarin was considered; although DOACs are licenced for the treatment of deep vein thrombosis (DVT) of the lower limbs and in pulmonary thromboembolic disease, there is no strong evidence to support their use in SVCO secondary to pacemaker lead placement [10]. With advice being general, advising the use of anticoagulation whilst targeting the underlying cause of SVCO, without specifying which medication to use [11].

Nevertheless, there are several advantages to DOAC use such as having "fixed dosing… rapid onset" and requiring "no monitoring" [12]. For this reason, the anticoagulant was

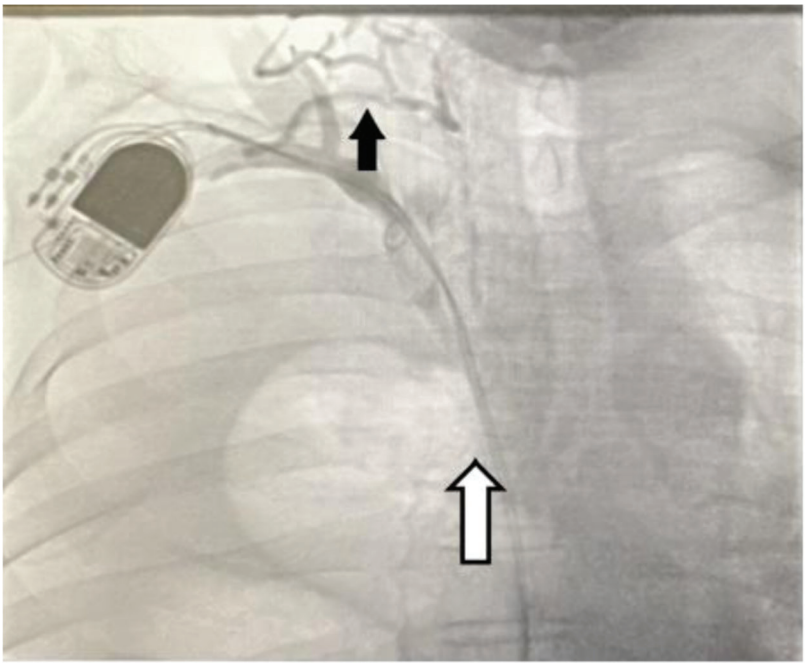

**Fig. 1.** Venogram: Upper arrow (black) shows collateral vein formation. The lower arrow (white) shows the normal anatomical position of SVC as it enters the right atrium.

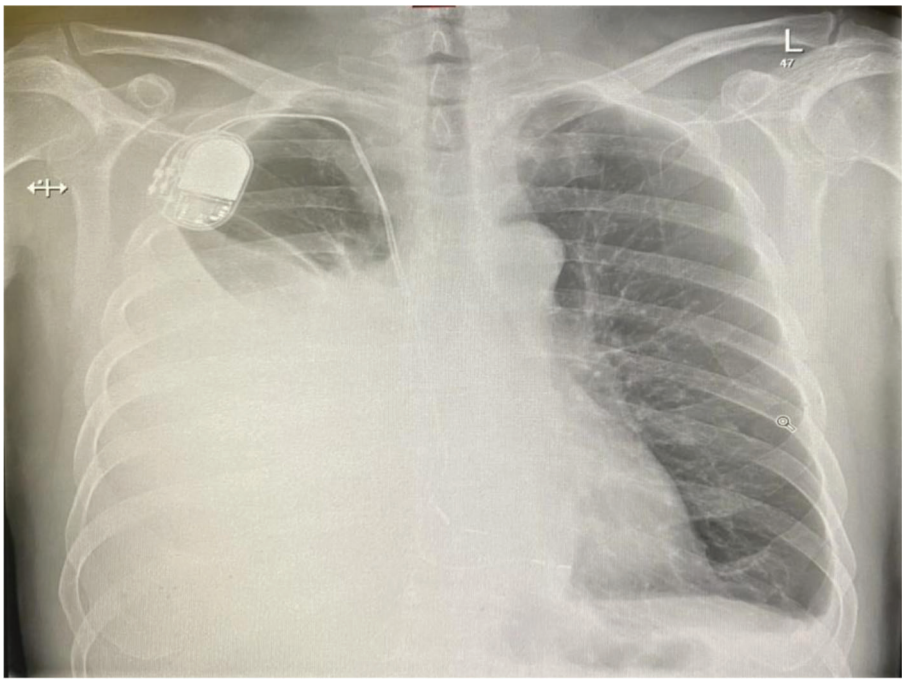


**Fig. 2.** Chest X-ray demonstrating a large right-sided pleural effusion requiring Seldinger drain insertion.

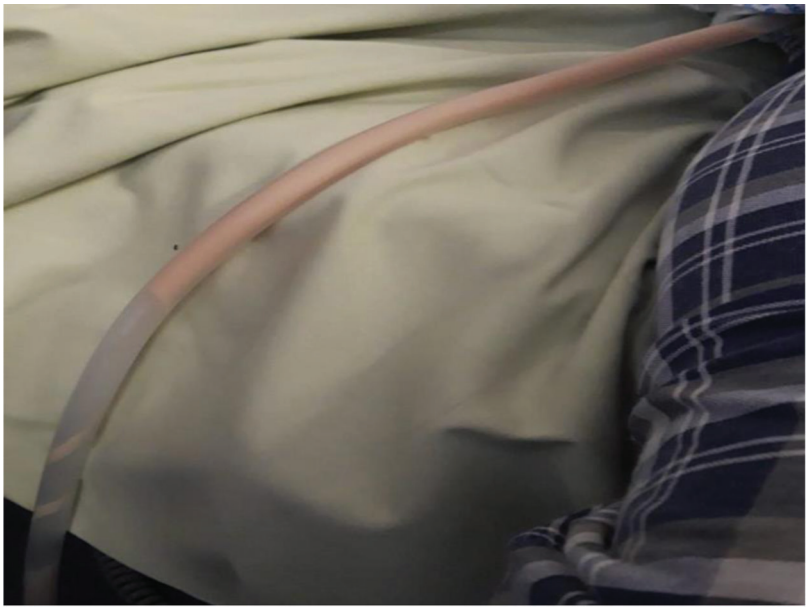

**Fig. 3.** Chest drain demonstrating pink milky fluid.

not changed but the dose was increased at the suggestion of the cardiothoracic team when the SVCO was shown to persist. This was a pragmatic, but non-evidence-based, decision.

The presence of ongoing SVCO obstruction raised further therapeutic discussions and three approaches were initially considered, namely conservative, percutaneous, and surgical.

Given that the patient was being treated at a remote non-cardiac surgery centre with no prior experience of this condition, an electronic multidisciplinary team discussion was started between a cardiologist, interventional radiologist, and cardiac surgeon.

A conservative approach was initially considered; there was already evidence of collateral formation in the upper limbs both clinically and radiologically and over time the expectation might have been that these would continue to develop. However, this would be a slow process and complete resolution of symptoms was not certain.

A percutaneous approach was also considered as initially there appeared to be a non-total obstruction associated with a focal stenosis. A percutaneous approach would be less invasive than a surgical approach but would only likely be successful if a balloon venoplasty was followed by stenting

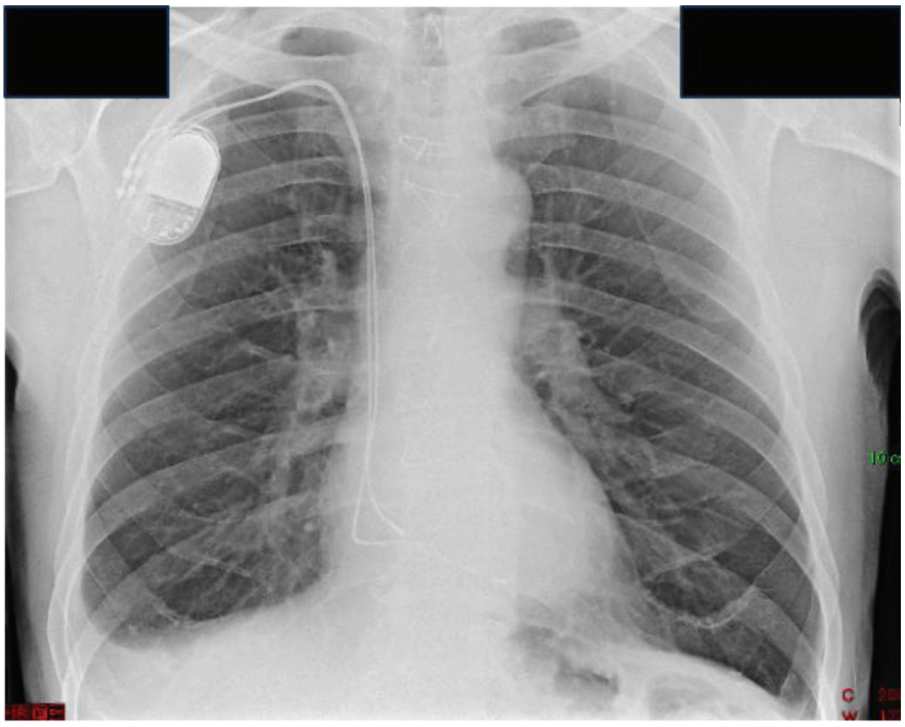

**Fig. 4.** Convalescent chest X-ray demonstrating right-sided blunting at the costophrenic angle.

to prevent the tissue from recoiling, as witnessed in another case of SVCO [5]. This being the case, for a stent to be deployed the existing transvenous pacing lead would need to be extracted [13].

One known complication of transvenous lead removal is tears to the SVC which can result in haemothorax or a pericardial effusion, with complete vascular lacerations often resulting in the need for urgent surgical intervention whilst carrying a large mortality risk of 50% [13]. This case was thought to carry a high risk of SVC compromise given that the leads were likely already adherent to the SVC as the site of obstruction. Indeed, this had been the experience in a recent case where the pacemaker leads could not be safely removed and a percutaneous approach to a SVCO was abandoned in favour of surgery. The fact that the SVCO appeared on repeat imaging to be totally occluded and the likely presence of clot also added concern that a percutaneous approach might afford less control of embolism of thrombotic material which would have the risk of pulmonary embolism (PE).

The insertion of a leadless pacemaker, first created to avoid the known complications of the transvenous pacing leads, was considered in this patient [14]. However, the patient had a dual chamber pacemaker inserted for sinus node disease and was physically active. Therefore, a leadless right ventricular pacemaker was thought a suboptimal pacing choice as it provides only right ventricular pacing and would result in the loss of future atrioventricular synchrony.

Surgery was considered as an option to try and spare the pacemaker, offering a more durable outcome for the patient. During these discussions, there was increasing concern about the risk of thrombus and uncontrolled embolism, and despite consideration of the post-operative complications of a thoracotomy (which carries a 3-4% mortality risk), it was thought that the patient would benefit from surgery [15].

The patient was then offered an operation with the intention that it would be undertaken as an elective case, but the patient was admitted with breathlessness after developing a large chylothorax and surgery as an inpatient was expedited.

During the immediate post-operative recovery period and after, the patient had ongoing dyspnoea. A CT showed recurrent occlusion of the SVC (August 2021) and an ongoing right-sided chylothorax.

Advice was sought from the respiratory team regarding the ongoing chylothorax and the dieticians concerning a low-fat diet. At this point, various options were again considered. If symptoms were intolerable then repeat surgery or a percutaneous option could have been considered but would have likely required removal of the pacemaker.

Fortunately, the patient's symptoms were tolerable, and follow-up has continued since with 6 monthly chest x-rays for 2 years. After 2 years, the low-fat diet was relaxed because the patient was finding this very restrictive. Chest x-rays were again repeated, both 6 and 12 months after relaxing the low-fat diet which showed no increase in the right effusion. This suggests that a more relaxed diet is being tolerated, possibly due to the formation of collaterals.

## CONCLUSION

This case highlights a rare complication which can accompany the insertion of a pacemaker – SVCO. It highlights the difficulties faced in investigating and treating such a complication which involves repeated patient exposure to tests requiring radiating and contrast. The benefits and drawbacks of medical, percutaneous, and surgical management have all been explored with the individual patient at the centre of each decision. Despite undergoing difficult corrective surgery, this patient has still

been left with long-term symptoms which will require further management.

The impact of such a procedure, its complications and future management should be considered before all pacemaker device insertions and for this reason, we suggest that although SVCO is a rare complication, the potential consequences for the patient are significant. Implanters should consider discussing rarer complications during the informed consent procedure before the patient decides to agree to pacemaker implantation.

## Declarations of interest

None.

## Informed consent

This case report has been seen and approved by the patient involved in the case who has provided written approval for it to be submitted for publication.

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