

ICD Lead Migration: A Lesson to Learn

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Introduction

Implantable Cardioverter Defibrillator (ICD) was initially used for secondary prevention of sudden cardiac death due to VF/VT [1]. The first use of ICD to prevent sudden cardiac death was in 1980 [1]. Currently, ICD is indicated for secondary prevention of SCD due to sustained VT or VF (in whom there is no identifiable cause) and primary prevention of SCD in patients who are at risks of SCD due to VT/VF2. This includes patients with ischemic cardiomyopathy with EF \leq 30% or non-ischemic cardiomyopathy with NYHA class II/III and EF \leq 35%. ICD is not recommended if the patient survival is less than a year or if there are reversible causes [2]. More often, patients who are candidates for ICD are also candidate for Cardiac Resynchronization Therapy RCT (or biventricular pacing) if QRS duration \geq 120 milliseconds [3]. Compared to medical therapy, CRT improves survival according to CARE-HF trial [4]. Combination of both biventricular pacing and ICD is recommended to reduce mortality and morbidity in patients with heart failure and prolonged QRS complex [3]. According to COMPANION trial, the benefit of this combination is greatest in presence of LBBB and QRS \geq 150 milliseconds [5].

ICD insertion carries a risk of major complications like cardiac perforation, bleeding and infection. The overall rate of ICD lead insertion complication is around 3-6 percent [6,7]. One large cohort study has shown 5.4% incidence of major complications requiring reoperation or hospitalisation in the first 90 days after the procedure [8]. Cardiac perforation incidence is 0.14% and usually associated with significant mortality and morbidity [9].

In this case report, ICD lead migration with unusual presentation is discussed. The case was referred to our cardiothoracic unit at Blackpool Teaching Hospital for ICD lead extraction.

Case Report

A 53 years old non-smoking male, who works as decorator, presented with a history of infero-posterior Myocardial Infarction in 2012, which was managed by right coronary artery stenting. He also had an episode of Ventricular Tachycardia in 2013, whereby he had ICD inserted post incident. He has heart failure (Ejection Fraction 35%), hypertension and diverticulosis and was on medical therapy of Bisoprolol, Perindopril, Furosemide, Eplerenon and Ezetimibe.

Two weeks prior to his admission to our Cardiac centre in Blackpool, he was getting intermittent left sided chest pain especially when working with both arms over his head frequently. The initial most severe chest pain episode was sudden, sharp and scoring 8/10 in severity. Having visited his local Accident and Emergency, He received pain killers, had Chest X-Ray and blood tests which were considered normal. Later, he has been discharged once the pain had settled on the same day.

Two days later, whilst at home, he developed left sided facial herpes zoster and his chest pain is subsided. The following day, he developed another paroxysm of severe chest pain 10/10 in severity. After visiting Accident and Emergency unit for the second time in a week, his chest X-ray, EKG and cardiac enzymes were unremarkable. He had pain relief and discharged home the same day following pain control.

A day after his second admission, whilst sitting at home, he noticed a large bruise over the left side of his chest and abdomen (Figure 1). He then went back to the Accident and Emergency unit but this time there was no chest pain.

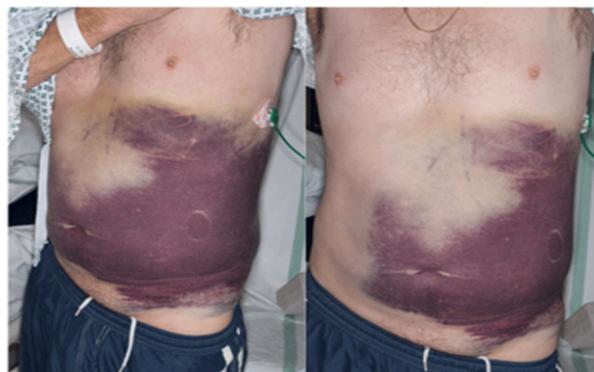


Figure 1: Massive Thoracoabdominal Haematoma due to ICD lead migration through the chest wall (The haematoma is extended downward possibly due to the gravity effect)

Investigations

Blood results: WBC $9.8 \times 10^9/L$, CRP 64 mg/L, Hb 110 g/L, Cr 56 $\mu\text{mol/L}$, Urea 6 mmol/L, AST 21 iu/L,

INR 1, Platelets $310 \times 10^9/L$, aPTT 31.7 Sec, Troponin I 4.3 g/L

EKG: Old LBBB, No significant new changes

ECHO: EF 35%, No Pericardial effusion

Imaging: Chest X-Ray followed by chest and abdominal CT scan

Differential Diagnosis

Acute Coronary Syndrome (chest pain), Herpes Zoster (the patient already have facial Herpes at the time of haematoma development), Trauma, Haemorrhagic pancreatitis (also causes abdominal wall haematoma), Retroperitoneal Haematoma.

Diagnosis

The Patient was diagnosed with ICD lead migration confirmed by CT scan (Figure 2). This caused a chest wall haematoma (Figure 1). He then referred to our cardiac centre in Blackpool

in normal range. His ICD check in the Cardiac Intensive Therapy Unit (CITU) revealed no ventricular capture with maximum output. We also noticed chest wall twitching during ventricular pacing. Referring back to his last ICD check reports, we noticed the loss of the ventricular intrinsic amplitude as well as gradual decrease in the ventricular pace impedance days prior to symptoms developments (Figure 3)

Management

Both surgical and cardiology teams agreed to remove the migrating ICD lead transvenously in the hybrid cardiac surgical theatre and in the presence of surgical team to accommodate for any possible complications like bleeding or tamponade.

The team has successfully extracted the lead transvenously (Figure 4) and replaced it with a new ICD lead (Figure 5) without untoward events. The patient then spent 24 hours in the CITU and further 24 hours in the ward prior to discharge.

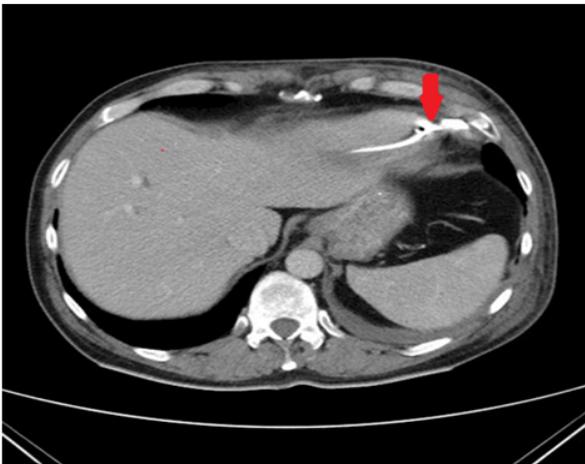


Figure 2: ICD lead (arrow) migration through the apex of right ventricle into the chest wall

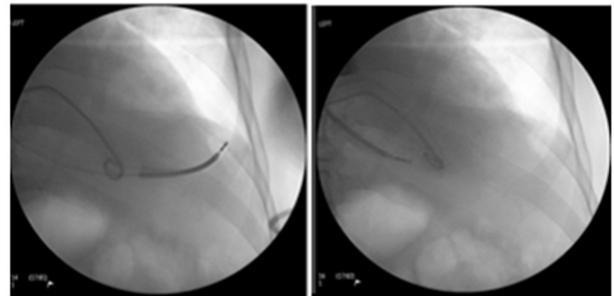


Figure 4: Transvenous lead extraction

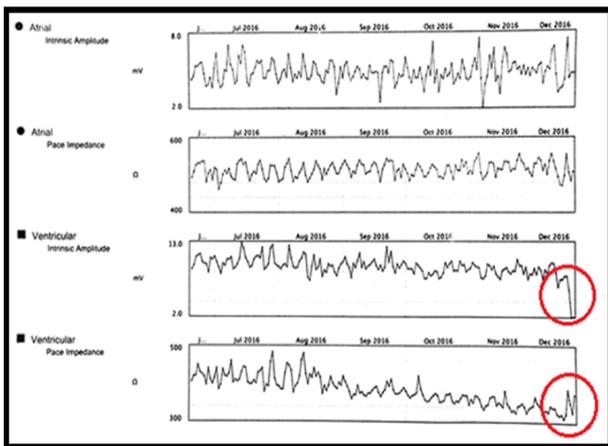


Figure 3: Loss of Ventricular Intrinsic Amplitude and Ventricular Pace Impedance days before development of chest pain and haematoma (red circles)

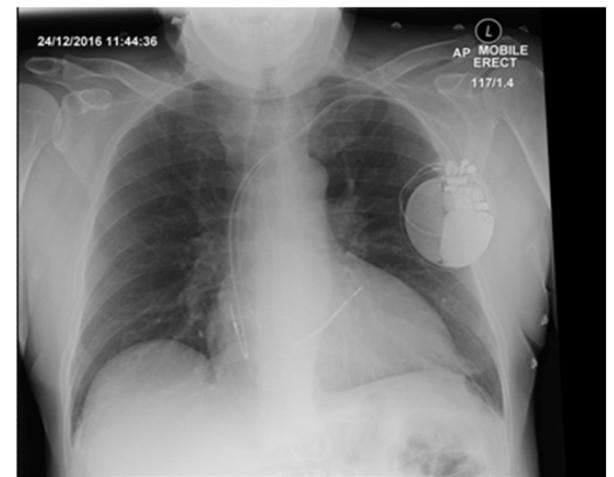


Figure 5: New ICD implantation

Discussion

The incidence of ICD lead perforation is ranging from 0.34% to 5.2% according to one study [10]. Literature has shown variety of ICD lead migration presentations ranging from no symptoms to fatal tamponade [10&11]. All cases in one study share a common feature which is altered lead parameters prior to

Victoria Hospital (BVH) for ICD lead removal. On admission to BVH, he was asymptomatic and stable with his blood results been

development of symptoms [12]. This confirms the importance of home monitoring to predict and prevent fatal complications [13].

The right atrium and right ventricular apex are the most frequent sites of perforation [14]. Most likely, the lead movement with systole is the culprit. This would render the thin and stiff leads to perforate the myocardium especially if mounted with helical screw. Perforation through the right ventricular apex is frequently an asymptomatic event [14]. Another explanation of perforation mechanism may be related to vertical lead mobility during the patient arms movement in upward direction (Twiddlers Syndrome) as in our case, whereby, hand movement above the head level might have led to vertical lead displacement over the years.

What makes our case unusual is the fact that Perforation symptoms started three years post ICD implantation with the main clinical sign being a massive anterior thoraco-abdominal haematoma which led to the diagnosis. Furthermore, previous loss of lead parameters was preceding the symptoms development; hence, an emphasis should be made to raise the index of suspicion of ICD lead migration in such cases.

Conclusion

Unexplained chest pain in a patient with ICD device with loss of ventricular amplitude and pace impedance should highlight the possibility of lead migration. Even if asymptomatic, loss of ventricular impedance and amplitude should be thoroughly investigated to rule out lead migration. An emphasis should be made on proper positioning of the ICD leads at the time of insertion.

Those who are in occupations or habits which require them to frequently move their arms upward during painting or decoration for example may need to avoid that position or manoeuvre to prevent lead migration (Twiddler's Syndrome).

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