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REVIEW

Injury to the coronary arteries and related structures by implantation of cardiac implantable electronic devices

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Damage to the coronary arteries and related structures from pacemaker and implantable cardioverter-defibrillator lead implantation is a rarely reported complication that can lead to myocardial infarction and pericardial tamponade that may occur acutely or even years later. We summarize the reported cases of injury to coronary arteries and related structures and review the causes of troponin elevation in the setting of cardiac implantable electronic device implantation.

Keywords Pacemaker • ICD • Coronary artery • Perforation • Troponin

Introduction

Major complications from implantation of permanent pacemakers and implantable cardioverter-defibrillators (ICDs) [cardiac implantable electronic devices (CIED)] such as pneumothorax, cardiac perforation, and infection can occur in 4–7% of patients.[1–3] While cardiac troponin is important in the diagnosis of myocardial infarction, in the setting of recent device implantation, raised levels may also indicate direct trauma to coronary arteries, inadvertent placement of leads into the arterial system, pulmonary embolism, and takotsubo cardiomyopathy.[4] The objectives of this manuscript are to: (i) characterize the presentation and treatment of lead injury to coronary arteries and related structures, and (ii) review the causes of troponin elevation in the setting of recent device implantation.

Methods

A systematic search of the SCOPUS, MEDLINE, and Pubmed databases up to October 2014 was conducted for case reports of damage to coronary and bypass graft arteries as well as troponin elevations following insertion of a CIED. The MeSH search string was (artery [All Fields] and troponin [All Fields] and pacing [All Fields] and ICD [All Fields] and takotsubo cardiomyopathy [All Fields] and ('humans' [MeSH Terms] and English [Lang]). The literature search was limited to studies in humans and articles published in peer-reviewed journals in English. Reference lists from reviews and articles were also hand searched for relevant publications.

Damage to coronary arteries and related structures

Acute perforation of the right atrium (RA) or right ventricle (RV) by pacemaker or ICD leads occurs in 0.5–2% of patients.[5–7] It can result in chest pain, pericardial tamponade, and potentially death. Delayed perforation months to years post-implantation may lead to pneumothorax, pericardial tamponade, and migration of leads to the lung or chest wall.[7–12] Damage to coronary arteries is a less well recognized sequelae of lead perforation. Cardiac implantable electronic devices leads are deployed using fluoroscopy, a two-dimensional imaging modality, where the proximity of lead tips to coronary arteries is not well appreciated unless simultaneous contrast angiography is performed.

We recently characterized the position and proximity of CIED lead tips to coronary arteries using multiplanar cardiac computed tomography (CT)[13] and described a schema for defining lead tip position in the RV.[14] In this schema, the long axis of the RV is divided into thirds (RV outflow tract, middle RV, and RV apex), and the short-axis walls of the RV are defined as per *Figure 1*. In our analysis, a majority of RV 'septal' lead tips were not implanted on the 'septum', but instead on the RV antero-septal junction (*Figure 1*).[14] The left anterior descending (LAD) coronary artery runs within the interventricular groove superficial to the RV antero-septal junction. In our series, leads in the RV antero-septal junction were only a few millimetres (median 4.7 mm) away from the LAD (*Figure 2*).[13]

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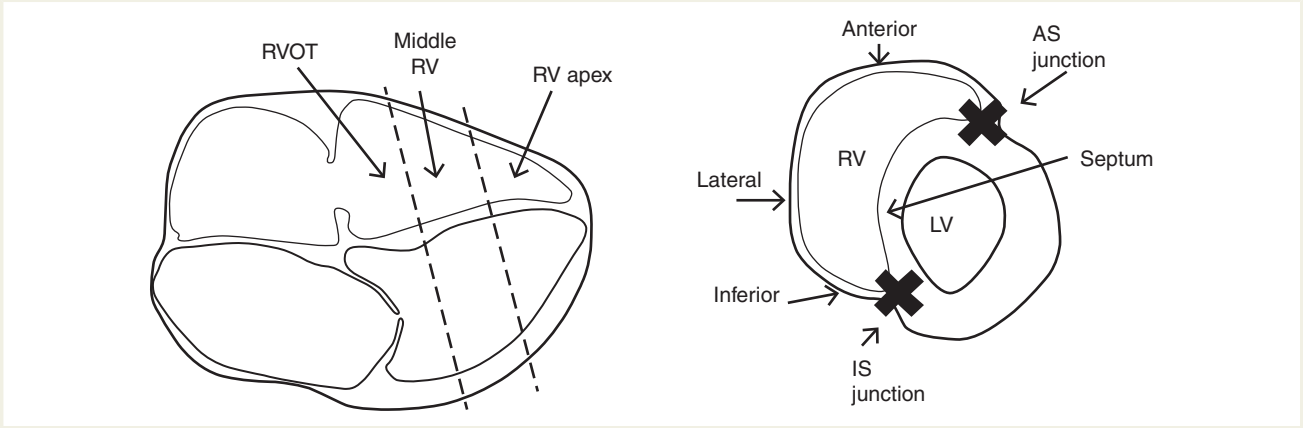

Figure 1 Schema for characterizing pacemaker lead location in the cardiac CT long and short axis. (Left) Computed tomography four-chamber long-axis view, classifying lead position into the RV outflow tract (RVOT), middle RV or RV apex. (Right) Computed tomography short-axis view RV pacing lead position: septum, anterior RV wall, antero-septal (AS) junction, lateral wall, inferior wall, and infero-septal (IS) junction.[11]

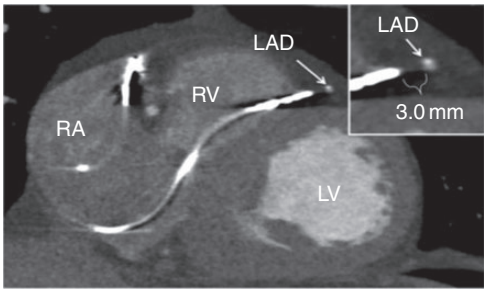

Figure 2 Parasternal short-axis CT view. The RV lead is in the antero-septal junction in close proximity to the LAD coronary artery. (Inset) The measured distance between the RV lead tip and LAD coronary artery is 3.0 mm.[13]

There have been two reports of injury to the LAD and two cases of damage to the right coronary artery (*Table 1*)[15–18] from lead perforation. The three perforated active-fixation leads presented within 36 h of implantation,[17,19,20] and a passive-fixation lead was incidentally discovered 4 years later.[17]

Damage to the LAD resulted in acute ST elevation myocardial infarction. In both cases, fluoroscopic images showed lead placement in the RV antero-septal junction short-axis position and middle RV long-axis position. In the case by Parwani *et al.*,[16] the helix of the pacing lead was deployed into the LAD coronary artery. It was treated by simultaneous removal of the lead and insertion of a covered stent. Nishiyama *et al.*[15] described a case where an ICD lead caused myocardial infarction secondary to coronary artery spasm distal to the lead. Spasm resolved when the lead was removed percutaneously.

The two cases of lead injury to the right coronary artery required surgical repair. In the case by Nagakawa[18] implantation of an active-fixation RA lead was complicated 3 h post-procedure by pericardial tamponade and haemodynamic collapse. At surgery, only the helix was seen protruding out of the RA wall and there was active bleeding from a lacerated right coronary artery. Pericardium was used to repair the coronary artery and to cover the helix protruding from the RA. Khoueiry *et al.*[17] described the incidental finding on chest X-ray of a RA mass 4 years post-pacemaker implantation. Cardiac CT showed a perforated passive-fixation lead that had resulted in a RA pseudoaneurysm with an arteriovenous fistula connected to the right coronary artery. Treatment consisted of surgical ligation of the artery, closure of the RA pseudoaneurysm, repair of the RA perforation, and replacement of the atrial lead.

In the previous cases, despite penetrating into coronary arteries, pacing characteristics were within the normal range and only one case was associated with a pericardial effusion.[18]

Damage to the aorta

Using cardiac CT, we found RA pacing lead tips on the anterior RA medial wall in close proximity to the aorta (*Figure 3*).[13] Aortic root puncture and perforation are known rare complications of RA trans-septal catheterization for left atrial access. A review of 5520 patients undergoing trans-septal catheterization reported five cases (0.09%) of aortic puncture and of these, three had aortic perforation.[21] There have been four reports of pericardial tamponade occurring secondary to active fixation pacing leads perforating through the RA wall and lacerating the adjacent aorta.[22–25] Clinical presentation ranged from hours[25] to 2 weeks[22] post-implantation. The aortic root[25] and the ascending aorta[22–24] were lacerated. All required surgical correction of the aorta. In one case the RA pacing lead was repositioned[23] and in the other three cases, the lead was left and the atrium surrounding the pacing lead was plicated.[22–25]

Inadvertent implantation of leads into coronary arteries

Mistaken introduction of leads into the arterial rather than venous system is a rarely reported complication. There are reports [19,20,26,27] of implantation of leads into the lumen or adjacent to the ostium of coronary arteries: two into the right coronary artery, one into the

Table 1 Case reports of inadvertent injury to coronary arteries

Study	Country	Age and sex	Lead fixation	Artery	Damage	Complication	Treatment
Parwani *et al.*[16]	Germany	49 M	V Active	LAD	100% occlusion	Ant STEMI	Covered stent
Nishiyama *et al.*[15]	Japan	68 M	V Active	LAD	75% occlusion	Coronary spasm and Ant STEMI	Percutaneous reposition
Nakagawa[18]	Japan	72 F	A Active	RCA	Laceration	Pericardial tamponade	Surgery
Khoueiry *et al.*[17]	USA	67 M	A Passive	RCA	AV fistula	RA pseudoaneurysm and AV fistula	Surgery

F, female; M, male; A, atrial lead; V, ventricular lead; STEMI, ST elevation myocardial infarction; LAD, left anterior descending coronary artery; RCA, right coronary artery; AV, arteriovenous.

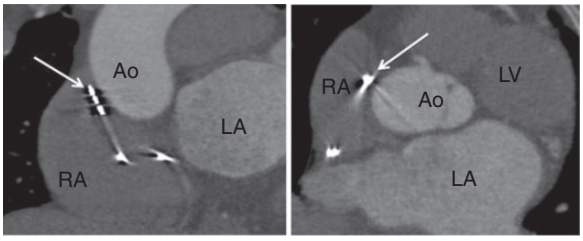

Figure 3 (Left) CT coronal view. (Right) Computed tomography axial view. The RA pacing lead tip is on the superior part of the medial wall of the RA appendage in close proximity to the aorta (Ao). LA, left atrium.[13]

circumflex artery, and two attached to the wall of the aorta. These were accompanied by leads positioned across the aortic valve onto the endocardium of the left ventricle (LV).

Meier[28] and Heinroth *et al.*[29] have demonstrated that it is possible to acutely pace the myocardium from leads placed into coronary arteries with low thresholds. In the above cases, pacing from the circumflex artery,[27] right coronary artery,[19,20] and even in the aorta near the ostium of the right coronary artery[20] was associated with satisfactory RA pacing capture and sensing thresholds.

Two cases were complicated by ST elevation myocardial infarction: one at the time of implantation into the right coronary,[27] and the other 6 months post-implantation into the circumflex artery.[20] The delayed presentation likely represents gradual migration of the lead distally into the vessel. Both cases were treated with percutaneous lead extraction. In another case 4 weeks post-pacemaker lead implantation into the right coronary artery, the patient was treated with lead removal and bypass grafting of the residual occluded right coronary artery.[19] The authors did not explain why surgery was chosen over percutaneous coronary intervention.

There are two reported cases[20,26] of intended RA active-fixation leads being implanted into the endocardium of the aorta near the ostium of coronary arteries. In the case reported by Issa *et al.*,[26] 5-year post-device implantation, an intended RA lead was found perforated through the wall of the aorta just below the ostium of the left main coronary artery and attached to the epicardium of the left atrium. The patient was treated conservatively due to the patient's poor clinical status and advanced age. Kosmidou *et al.*[20] presented a case where 12-month post-device implantation, the intended RA lead was found attached to the wall of the aorta, just below the ostium of the right coronary artery. The lead was removed percutaneously with no adverse clinical events. Despite leads being implanted into the arterial system in the above cases, there were no reported thromboembolic complications.

Inadvertent injury to the internal mammary artery

The right and left internal mammary arteries (LIMA) arise from the medial end of the subclavian artery, traverse behind the subclavian vein, and then run along the sides of the sternum.[30] The proximal internal mammary artery is vulnerable to inadvertent damage during attempted subclavian venous puncture as it passes behind the subclavian vein in the space between the clavicle and first rib (*Figure 4*).

In patients without bypass grafts, damage to the internal mammary arteries secondary to pacemaker implantation can result in adverse events. Case reports have detailed pseudoaneurysms that either: (i) bled acutely with haemothorax and haemomediastinum,[31] (ii) caused compression of the adjacent phrenic nerve (*Figure 4*) with diaphragmatic paralysis,[32,33] or (iii) heart failure due to the development of an arteriovenous fistula between the LIMA and the innominate vein.[34] In one case a lead perforated the RV and lacerated the overlying LIMA, resulting a left haemothorax and hypotension.[35] A thoracotomy was performed with surgical ligation of the LIMA and extraction of the lead.

There are two reports of damage to LIMA bypass grafts during pacemaker implantation leading to acute myocardial infarction.[36,37] In one case,[36] injury to the LIMA was treated by coronary angioplasty and stenting. In the other,[37] the resultant large anterior myocardial infarct was complicated by a large ischaemic ventricular septal defect, cardiogenic shock, and eventually death of the patient. These complications could be avoided by cephalic or axillary venous access or attempting subclavian venous access on the contralateral side that has not been used for grafting.

Left internal mammary artery injury secondary to laser sheath-assisted pacemaker lead extraction has been complicated by pseudoaneurysms and arteriovenous fistulas.[38–41] Chronic leads in contact with endothelium and endomyocardium provoke localized inflammatory changes that evolve to fibrous tissue that can extend and adhere to nearby vessels.[42] During the extraction of these

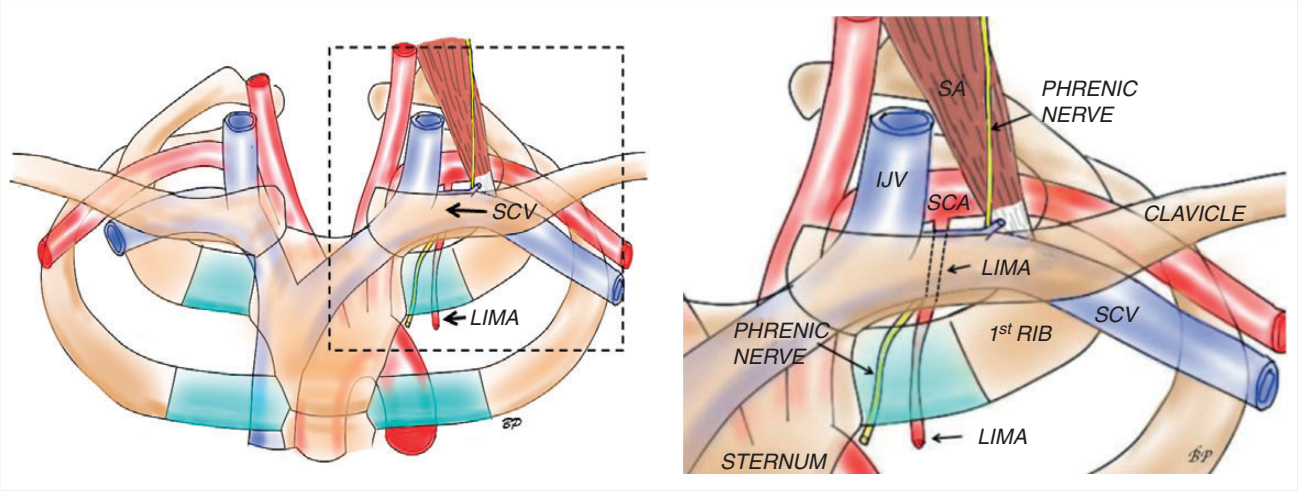

Figure 4 (Left) Diagram showing the relationship of the LIMA to the subclavian vein (SCV). (Right—the insert in Left is expanded) The LIMA arises near the medial end of the subclavian artery (SCA). It passes posterior (dashed line) to the SCV and first rib. It then runs inferiorly (close to the phrenic nerve) lateral to the sternal border. The scalenus anterior (SA) muscle and phrenic nerve lie between the SCA and SCV.

leads, the cutting energy of laser sheaths increases the risk of pseudoaneurysm and arteriovenous fistula formation.[38,43] Arteriovenous fistulas are usually recognized at the time of procedure by chest or back pain and bright red blood flowing around the sheath,[43] but can also present late with symptoms of congestive heart failure.[44] In both implantation- and extraction-associated cases, pseudoaneurysms and arteriovenous fistulas were managed with percutaneous coil embolization[31–34] or covered stents.[39,41]

Takotsubo cardiomyopathy

Takotsubo cardiomyopathy is a reversible cardiomyopathy with ECG changes, modest troponin elevation, and clinical symptoms that commonly occur following a physical or psychological stressor.[45,46] There have been 15 case reports of takotsubo cardiomyopathy following pacemaker implantation.[47–58] Similar to other published case series of takotsubo cardiomyopathy, the majority were women (79%), and their age ranged between 54 and 89 years old. Patients presented with chest pain and dyspnea and were in acute pulmonary oedema. There was either a modest or no troponin elevation and minor ST elevation in the precordial ECG leads. The time of presentation varied from a few minutes to 3 days post-device implantation.[47] Golzio *et al.*[59] reported a case where takotsubo cardiomyopathy occurred as a result of pacemaker syndrome secondary to a malfunctioning atrial lead. The majority (10 of 13) of patients had complete return to normal LV systolic function by a few days[55] to 2 months later.[50] Two patients did not recover normal systolic function,[47] and one patient died from intractable ventricular tachycardia and cardiogenic shock.[57] In all cases, coronary artery disease or damage to coronary arteries was excluded by coronary angiography.

Cardiac troponin elevation

Cardiac troponin T and I are specific and sensitive biomarkers of cardiac necrosis that are important in the diagnosis of myocardial infarction.[60] However, in the setting of recent CIED implantation, raised troponin levels can be secondary to myocardial trauma and inflammation from lead attachment to myocardium.[61] Other causes include: tachyarrhythmias, acute decompensated and chronic heart failure, sepsis, pulmonary embolism, and takotsubo cardiomyopathy.[4]

The majority of troponin is bound to actin and myosin and smaller amounts lie free in the cytoplasm. Myocardial damage causes an initial small short-lived peak followed by a slowly rising larger sustained elevation. The first peak is thought to represent troponin release from the cytosolic pool of troponin and the later larger peak is thought to be due to necrosis of myocardial tissue.[62]

Nikolaou *et al.*[63] studied the patterns of troponin release following CIED lead implantation. They demonstrated an early small short-lived peak of troponin I release[63] that contrasted with the larger sustained elevations seen with myocardial infarction.[64] In a subsequent larger trial,[65] 59% of 283 patients had elevated troponin levels (>0.1 ng/mL) at 6 h post-pacemaker implantation. Of these, five had 'markedly elevated' levels (>1.5 ng/mL). These five patients did not display ischaemic ECG changes and had normal coronary angiograms. The natural history of such patients is unknown.

Dworschak *et al.*[66] studied troponin T release in the setting of ICD lead implantation and defibrillation threshold testing. The level of troponin T correlated with the duration of the procedure, but had no relationship with the number of applied shocks or cumulative applied defibrillation energy from ICD threshold testing.

Non-coronary causes of elevated troponin levels are markers of poor prognosis. In pulmonary embolism, elevated levels correlated with clinical severity and 30-day mortality.[67] While symptomatic pulmonary embolism following device implantation is an uncommon complication (estimated to occur in 0–5% of patients[68]), the incidence of asymptomatic pulmonary embolism is thought to be much higher. Ventilation perfusion lung scans performed prior to and 14 days post-pacemaker implantation have found a 15% incidence of asymptomatic pulmonary embolism.[69]

Conclusion

In line with the increasing number of CIED implantations worldwide, there have been sporadic reports of inadvertent damage to coronary arteries and related structures. Damage to coronary arteries can have a varied clinical presentation: chest pain, respiratory distress, symptoms of heart failure, and cardiogenic shock. Clinical presentation can occur at the time of implantation, or even years later. While raised troponin levels may indicate damage to coronary arteries, it may also represent alternative diagnoses such as takotsubo cardiomyopathy, pulmonary embolism, or simply direct mechanical trauma from lead implantation.

Conflict of interest: none declared.

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