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Repeated fracture of pacemaker leads with migration into the pulmonary circulation and temporary pacemaker wire insertion via the azygous vein

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ABSTRACT
Repeated implantation of pacemaker in the same patient is a common occurrence because of the increased longevity of patients. However, repeated lead fracture in the same patient and migration of the pacemaker lead into the pulmonary circulation is rare. We describe a 56-year-old gentleman who had undergone pacemaker implantations thrice due to repeated lead fractures (thrice) and also had migration of the pacemaker lead into the pulmonary circulation. He also had an azygous vein which was noticed while placing the temporary pacemaker wire.

KEY WORDS: Pacemaker, lead fracture, migration

Pacemaker lead fracture is a known complication of permanent pacemaker implantation occurring in 4% of patients.[1] We describe a male patient who was referred to us for pacemaker implantation for the fourth time. He had experienced lead fracture thrice in the past [twice with endocardial leads and once with epicardial lead]. He also has migration of one of the endocardial leads into the pulmonary circulation and presence of an azygous vein was noticed during temporary pacemaker wire insertion which is rare.

Case Report
A 56-year-old gentleman came for checkup at our center with one-day history of recurrent syncope and giddiness. He was a farmer by occupation and his work involved vigorous manual labor. His heart rate was 36/ min and examination of the cardiovascular system was unremarkable. The electrocardiogram revealed complete heart block with ventricular rate of 36/ min and a non-functioning (battery- end of life) pacemaker implanted elsewhere.

He was a known case of complete heart block diagnosed in 1982 and had undergone a permanent pacemaker implantation in March 1982. A VVI pacemaker implantation [CPI Microlith Demand type] along with porous tined endocardial lead [CPI Unipolar, length 59 cm] was implanted via the subclavian vein in the right infraclavicular area. Subsequently, in 1989, the patient presented with continuous pectoral stimulation, however, details about the pacemaker parameters at that time were not available. The lead insulation was found to be broken at its attachment to the pulse generator. The patient underwent re-implantation of a new pacemaker [VVI, CPI Microlith Dx] along with a unipolar porous silicone tined lead [CPI tined lead] in the right infraclavicular region in October 1989 via the subclavian vein approach. At the time of the second implant, it seems that the lead end was clipped as per the image on the chest X-ray, however, it was not fixed to the subcutaneous tissue. The patient again followed up with similar complaints in October 1994. The pacemaker lead was found to have a complete fracture with failure to capture and sense, however, the pacemaker had not reached its ‘end of life’. The other ends of both the earlier leads were situated in the subclavian region, The same pacemaker was re-implanted in the epigastric region along with an epicardial lead. Though the previous pacemaker was explanted again, the leads were again left free without being fixed to the subcutaneous tissue as it was not possible to locate the cut end of the leads in the subcutaneous tissue. The patient was subsequently discharged after which he did not follow-up regularly for checkup and device interrogation.

This time, in November 2005, he followed up with us with recurrent syncope. On interrogation of the device, it was found to have reached its ‘end of life’. The chest radiogram showed
the pacemaker generator in the epigastric region; however, a lead fracture of the epicardial lead was noted. The pacemaker generator had reached its ‘end of life’. This was the third incident of lead fracture in the same patient though the last time the lead was epicardial. The X-ray chest also revealed the two endocardial leads, one of which was in its original position while the other had migrated into the pulmonary circulation. The tip of the intracardiac lead seemed fixed at the right ventricular site while the body of the lead had migrated into the main pulmonary artery forming a loop there and had subsequently migrated into the right pulmonary artery [Figures 1 and 2]. The computed tomography (CT) of the chest confirmed the position of the lead in the pulmonary artery [Figures 3 and 4].

The patient underwent a successful pacemaker implantation at our hospital. A VVI pacemaker was implanted [Medtronic Sigma] along with a tined steroid eluting lead [Capsure SP Novus] in the left infra-clavicular area via the subclavian vein approach. Incidentally, during the temporary pacemaker wire insertion, it was noted that the patient had an azygous vein draining into the superior vena cava [Figure 5]. The procedure was uneventful. No attempt was made to extract the earlier endocardial leads in view of the stable nature of the leads. The epicardial pacemaker was also not explanted as it was shown to have migrated deep into the abdominal wall and explanting it would involve a major surgery. The patient was discharged subsequently with an advice to regularly follow up and avoid heavy manual work.

This case demonstrates the repeated (thrice) lead fractures which occurred in the same patient though it was placed at different sites (endocardial and epicardial) and also the possibility of migration of the pacing leads into the pulmonary artery, especially when there are multiple abandoned pacemaker leads.
with epicardial lead systems. The etiology of epicardial lead fracture was most commonly seen in younger patients, rather than the cephalic vein. Calkins has demonstrated that entrapment by the subclavius muscle or the costo-clavicular ligament could cause repeated flexing of the leads during movement of the pectoral girdle. As most of the adults undergo infraclavicular endocardial pacemaker implantation, epicardial lead fracture is seen in the pediatric group in which the pacemakers are placed in the epigastric region. Cohen et al. and Epstein et al., studied the long-term performance of epicardial leads in the pediatric population and reported a lead fracture rate of 5.5% over five years and 3% over two years respectively. Fortescue et al., found that lead fracture was most commonly seen in younger patients, patients with structural congenital heart disease and those with epicardial lead systems. The etiology of epicardial lead fracture may be similar to the endocardial lead fracture. Soft tissue entrapment imposes a static load upon the leads and reported flexure at the point of entrapment may be responsible for the damage.

In a study by Antonelli, the subclavian route was associated with more failure rates than the cephalic route for polyurethane insulated leads. However, the route of insertion did not affect the failure rate for silicone leads. Pellethane 80A insulated leads are more prone to insulation failure than 55D and silicone rubber, especially when the subclavian route of entry is used rather than the cephalic vein. Calkins et al., found that there was no significant difference in complication rates and lead failure rates on follow-up when using cephalic cutdown and subclavian vein puncture approaches.

It’s necessary to diagnose problems like lead fracture while interrogating such patients. It’s imperative to test all parameters and also an X-ray chest to diagnose lead fractures. ‘End of Life’ can be diagnosed by the inability of the pacemaker to achieve its intrinsic native rate on magnet application.

Migration of the pacing leads is known in patients in whom the leads have been abandoned in the intra-cardiac cavity. Case reports of leads migrating out of the pulmonary arterial circulation causing pulmonary infarction and/or pulmonary abscess have been reported earlier. Migration of the pacemaker lead into the hepatic vein has also been seen. If the lead is free and mobile, it is usually quite possible to snare the lead and extract it with complete resolution of the problem. Suga et al., observed that patients with three or more abandoned pacemaker leads, four or more total leads and a younger age at initial pacemaker implantation had a higher incidence of pacemaker-related complications.

This case demonstrates the occurrence of repeated lead fracture in some patients and increased incidence of complications like lead migration in patients with multiple abandoned pacemaker leads.

Discussion

Re-implantation of pacemaker more than two or three times in a lifetime has become a common occurrence because of the increased longevity. The two issues of importance in this case are the occurrence of (a) repeated lead fracture and (b) migration of the lead into the pulmonary artery.

Lead fracture is one of the complications seen in up to 3.9% of cases of permanent pacemaker implantations with an average rate of 1.2% per patient year. Approximate 93% of all pacemaker lead fractures occur in the segment of the lead lateral to the venous entry and costo-clavicular compression has been implicated for the lead fracture. Anatomical studies demonstrated that entrapment by the subclavius muscle or the costo-clavicular ligament could cause repeated flexing of the leads during movement of the pectoral girdle. As most of the adults undergo infraclavicular endocardial pacemaker implantation, epicardial lead fracture is seen in the pediatric group in which the pacemakers are placed in the epigastric region. Cohen et al. and Epstein et al., studied the long-term performance of epicardial leads in the pediatric population and reported a lead fracture rate of 5.5% over five years and 3% over two years respectively. Fortescue et al., found that lead fracture was most commonly seen in younger patients, patients with structural congenital heart disease and those with epicardial lead systems. The etiology of epicardial lead fracture may be similar to the endocardial lead fracture. Soft tissue entrapment imposes a static load upon the leads and reported flexure at the point of entrapment may be responsible for the damage.

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References


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