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Migration of a Pacemaker Lead to an Unusual Site

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INTRODUCTION

Various well-described complications such as lead dislodgement and migration can be observed although the most effective clinical treatment of cardiac conduction disturbance is permanent pacemaker implantation. In the current case report, we described the cause and management of a dislodged and migrated pacemaker lead to the hepatic vasculature.

CASE REPORT

An 80-year-old female patient with mechanical mitral valve prosthesis, complete heart block and atrial fibrillation was referred to our arrhythmia center for implantation of a pacemaker. Through the left pectoral region and axillary vein, a single chamber pacemaker with an active-fixation lead could eventually be implanted at the right ventricular base. Appropriate apical and septal regions could not be reached due to severe pulmonary hypertension, tricuspid valve regurgitation and dilated right heart chambers although various stylets and delivery catheters were used. Due to these reasons, the procedure and fluoroscopy times were much higher compared to a standard anatomy. Only 1 week after implantation, she was admitted to our emergency department with syncope. The electrocardiography showed complete heart block and baseline atrial fibrillation as in the previous diagnosis mentioned. Device interrogation demonstrated no sensing and capture. Chest X-ray showed the dislodged lead into the abdominal area. We performed a selective hepatic venography via the femoral vein and inferior vena cava and realized that the lead was in the hepatic vein and the distal tip embedded into the hepatic tissue (Figure 1). The patient has been treated with warfarin anticoagulation due to both mechanical heart valve and atrial fibrillation. Before the procedure the INR level was 2.1. No reversal of anticoagulation was considered. With the back-up of a general surgeon and reversal agents including intravenous vitamin K and prothrombin complex concentrate, we gently performed a simple traction to remove the lead. No important damage to the liver and bleeding occurred. During the same procedure, we tried to implant another lead into the right ventricle; however, all attempts failed. Therefore, using a coronary sinus implantation system a coronary sinus lead was implanted into the posterolateral branch of the coronary sinus with good sensing and pacing parameters without diaphragm stimulation (Figure 2). At the 1, 3, and 6 months follow-up, no change in sensing and pacing parameters were observed.

DISCUSSION

Dislodgement and migration of cardiac implantable electronic device leads are not uncommon. These conditions can be result from Twiddler's syndrome, Reel syndrome, Ratchet syndrome, severe atrioventricular valve regurgitation, inadequate fixation of leads and devices to the pocket, pocket hematoma causing enlarged pocket space, and pocket size/device mismatch.[1] Backward migration to the right atrium, superior vena cava, innominate vein, subclavian vein, and even the pocket can be generally seen although forward migration to more unusual locations including the inferior vena cava, pulmonary arteries, diaphragm, and the left ventricle (through a perforation) can occur. Various reasons of dislodgement in our case can be as follows; 1- Each right ventricular systole causes important backward blood flow from

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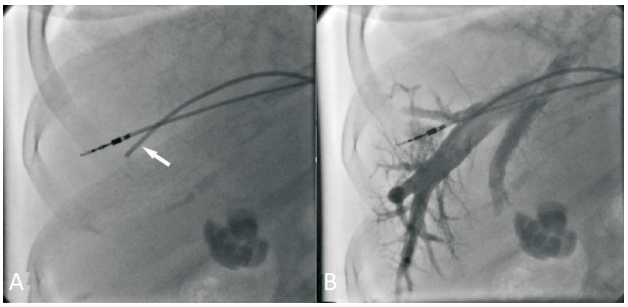

Figure 1. *Active-fixation pacemaker lead demonstrated in the liver before (A) and after contrast injection (B). The lead tip embedded in the hepatic tissue with the body located in the hepatic vein. White arrow, the multipurpose catheter.*

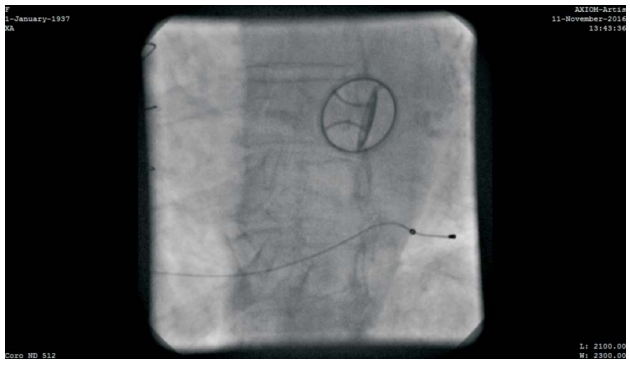

Figure 2. *Bipolar coronary sinus pacemaker lead implanted into the posterolateral branch.*

the tricuspid orifice. In addition, in severe cases with advanced right ventricular failure diastolic regurgitation can also be seen. With an incompetent valve and severe regurgitation, hemodynamic forces lead to backward pulling of the lead, 2- Active fixation leads with insufficient screwing into the myocardium can easily dislodge because they have no tines for stability, 3- Another reason for electrode dislodgement seems to be basal implantation of the electrode instead of apical or septal implantation. The majority of leads implanted < 1 year can be explanted with simple traction. However, after 1 year, especially > 5 years, lead extraction using locking stylets, telescopic sheaths, powered dilators, and different veins other than the implanted vein can be needed with on-site surgical back-up. Additional transcatheter tools including snares, steerable ablation and guiding catheters, pigtail catheter, and bioptomes are all important tools that should be available to retrieve the lead and its fractured components.[2,3] In some cases, implantation of the lead into the coronary sinus can be needed. Various conditions requiring implantation into the coronary sinus are as follows; 1- Anatomical barriers that preclude the passage through the valve such as atresia, stenosis and mechanical prosthesis,[4,5] 2- Failed implantation into the ventricle as in our case, 3- Presence of persistent left superior vena cava with absence of right-sided vein making the implantation near impossible, 4- Presence of abnormal ventricular substrate resulting in abnormal elevation of the capture threshold.

CONCLUSIONS

Dilated right ventricle and severe tricuspid regurgitation can make lead implantation impossible, therefore cardiac venous system becomes a good option.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this case report.

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