Right ventricular pseudoaneurysm causing very late tamponade: The leftover of a previously perforated and replaced defibrillator lead

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Introduction
Lead perforations, as complication to device implantations, are encountered with a wide spectrum of clinical manifestations. A high degree of attention is required in detecting rare complications and preventing serious consequences in the clinical course. We present a very rare complication of a right ventricular pseudoaneurysm resulting from lead perforation, despite early intervention with lead removal and lead replacement after the initial incident.

Case report
A 43-year-old man with hypertrophic cardiomyopathy with moderate outflow tract obstruction and severe asymmetric left ventricular hypertrophy was implanted with a dual-chamber implantable cardioverter-defibrillator (ICD) (primary prophylactic indication). Postprocedure he developed chest pain. Echocardiography and fluoroscopy were unremarkable. Lead position was unchanged and lead parameters were very good. He was observed for another day. The symptoms resolved, and he was scheduled for close follow-up. Echocardiography was still without effusion. Three weeks after the implantation he reported to have muscular twitches in the intercostal muscles close to the apex of the heart. At evaluation in clinic, extracardiac stimulation was evident. The ICD lead was removed and a new ICD lead was implanted in a presumed septal position. A pericardial effusion was detected (Figure 1A), considered to be the result of leakage after removal of the perforated lead. The effusion was judged to be hemodynamically insignificant and not easily accessible for drainage percutaneously. The patient’s symptoms resolved after lead replacement. First and second lead position is shown in Figure 1B and 1C. The effusion was stable and after observation the patient was sent home for close echo follow-up. At the sequential follow-up, the effusion gradually diminished, and further follow-up was scheduled at a referring hospital. Even 4 months after lead replacement, despite continued regression, the effusion was not totally resorbed.

Six months after the removal of the perforated lead and replacement with a new lead, the patient collapsed at work and was urgently admitted in severe hemodynamic compromise with initial systolic blood pressure at 50 mm Hg, stabilized by inotropics to 110 mm Hg by the attending prehospital doctor, who by fast assessment ultrasound detected a significant pericardial effusion documenting cardiac tamponade. The patient died from the tuberculous effusion.

KEY TEACHING POINTS
- Right ventricular pseudoaneurysm is a very rare but potentially very serious complication to lead perforation, and can occur despite early intervention with lead replacement.
- A low threshold for using additional imaging modalities in lead perforations can potentially be advantageous.
- Pericardial effusions after lead perforations require close follow-up until resolution or consideration of early drainage.
- Device-related complications exhibit diverse presentations. A high level of suspicion in individual cases is needed for detecting rare complications.

KEYWORDS
Lead complications; CT imaging; Right ventricular pseudoaneurysm; Cardiac tamponade; Device implantation

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The patient was brought emergently to the catheterization lab for urgent pericardiocentesis; 500 mL of blood was drained immediately and a further 150 mL was produced in the drain during the next hours. Hemodynamic stabilization was obtained immediately after initial drainage. A computed tomography (CT) scan immediately after pericardiocentesis demonstrated a right ventricular pseudoaneurysm, measuring 1.5 cm in diameter with a fistulous connection to the right ventricular cavity close to a coronary artery branch (Figure 2). The preliminary description of the CT suspected a communication between a coronary artery branch and the pseudoaneurysm, but at a more thorough evaluation this was considered improbable. The pseudoaneurysm was located at the likely position of the lead tip of the previously extracted and perforated lead, and was not related to the existing lead. Surgical closure of the pseudoaneurysm was then performed. Intraoperatively the fistulous connection was verified with connection through the apical septum to the right ventricular cavity close to a left anterior descending branch, but without communication to the latter. The fistula was oversewn and closed. Stigmata of recent bleeding in the pseudoaneurysm was identified peroperatively. A postoperative CT scan showed persistent closure of the pseudoaneurysm and fistula. The ICD was tested and lead values were unremarkable postoperatively. The patient was then discharged without further complications. In retrospect he reported to have had reduced functional capacity ever since the ICD implantation, likely because the pericardial effusion had never been fully resolved.

Discussion
The case represents a very rare complication to ICD lead implantation. Penetrating cardiac injury can produce pseudoaneurysms in the affected cardiac chamber and has been reported in relation to myocardial biopsy,1,2 in relation to lead extraction of tightly adherent leads,3,4 and in the left ventricle after pericardiocentesis with accidental chamber puncture,5 as well as in relation to other traumatic penetrating lesions. It has, however, to our knowledge not been reported almost 6 months after removal of and replacement of the penetrating lead, with late rebleeding and cardiac tamponade.

High-volume centers regularly encounter lead perforations and have routines in handling these complications, even though hard evidence-based guidelines are difficult to establish for handling these events. Such complications are not very rare, but each case frequently contains different aspects, each in themselves rare. The best strategy for handling these cases must be differentiated and adapted to individual circumstantial factors, such as the location and consequence of the penetrating lesion per se.

Several issues are of interest in the present case. It is likely that the lack of complete resorption of the pericardial effusion during the months of follow-up could be attributed to minor rebleeding events. The patient did not receive any antiplatelet or anticoagulant drugs; only a beta blocker was taken. Leaving pericardial effusions that do not appear hemodynamically significant for spontaneous resorption is a frequent matter of discussion. It is speculative, even though plausible, that in the present case early drainage could have prevented the expansion of the aneurysm, by allowing agglutination of the pericardial parietal and visceral layers early after lead perforation. Furthermore, leaving blood in the pericardial space produces inflammatory reactions with potential risk for secondary inflammatory exudation, exacerbating the initial volume of effusion. Pericardial constriction is also possible later on. Weighing the risk of pericardial puncture in minor effusions against these considerations is important in handling such complications.
The role of imaging is central. Early cardiac CT could be considered. The pseudoaneurysm was not detected on the routine echo, which is usually focused on the pericardial effusion. Even in the preoperative transthoracic and transesophageal echo, it was difficult to fully visualize the pseudoaneurysm. It is most likely that complementary imaging modalities will contribute to the recognition of more details of mechanisms and collateral damages, influencing and individualizing the handling of the individual case. Whether early CT could have prevented the late complication by disclosing early signs of the pseudoaneurysm in the present case, allowing earlier intervention, is uncertain but not unlikely. In retrospect, the ICD lead should probably have been replaced at the time of initial complaint of chest pain the day after the first procedure, but was deferred because of symptom resolution.

Septal placement of pacing leads is a main priority for avoiding lead perforation, but fluoroscopic verification of this position is at best moderate. In the present case, the location of the fistula suggests that the initial position of the penetrating lead could have been at the apical septum. Lead migration through septal myocardium to the apical free wall and penetration in this location is a possible mechanism. Suggestively, fluoroscopy may be even more misleading for assessing lead tip position in hypertrophic cardiomyopathy patients when severe septal hypertrophy is present, distorting the anatomy, and in addition a comparatively small right ventricle is present, giving the impression of septal location when the true position is far more apical and even free-wall apical.

In summary, we present a case of a potentially fatal complication occurring at a time when the risk after lead perforation and lead replacement usually would be considered to be low. The case adds aspects to variation in presentations of lead complications. Possibilities for different follow-up and handling are discussed. The case illustrates the need for thorough assessment and a high degree of awareness for unusual presentations in evaluating potential lead complications. It is suggested that a low threshold for performing imaging with echocardiography and CT scan could be useful when symptoms occur post implantation, keeping attention to the diverse spectrum of complications after device implantation.

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Figure 2  Computed tomography (CT) images of the distal fistula containing contrast and communicating with the right ventricular pseudoaneurysm (yellow arrow in magnified segment, right). An overview is shown at left and the magnified view of the square section at right. The CT scan is acquired after pericardiocentesis with the pericardial drain in situ. Yellow triangle indicates a segment of the left anterior descending artery. The yellow star indicates the left internal mammary artery.