Cardiac perforation due to delayed migration of a chronic dialysis catheter: a case report

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Background
Tunneled haemodialysis catheters are commonly used to perform haemodialysis. Rare complications of these catheters include perforations of major blood vessels or the heart. Albeit rare, these complications can lead to significant morbidity and mortality.

Case summary
We present a case of late migration of a tunneled haemodialysis catheter causing a right atrial perforation with subsequent pericardial tamponade, haemodynamic shock, and cardiac arrest. A 51-year-old female patient with end-stage renal disease presented with hypotension and lactate acidosis, indicating circulatory shock, during ambulatory intermittent haemodialysis. Dialysis was performed through a tunneled haemodialysis catheter that had been implanted more than 1 year ago. Upon admission to the hospital, initial diagnostics, including transthoracic echocardiography and computed tomography scan, showed a circumferential pericardial effusion which was not haemodynamically significant and no other pathological findings. After being transferred to the intensive care unit, the patient again showed signs of haemodynamic shock at the start of another dialysis session which deteriorated to cardiac arrest. Ultimately, using multi-modality imaging, migration of the catheter tip through the right atrial wall into the pericardial space was diagnosed. Emergency sternotomy and surgical extraction of the tunneled haemodialysis catheter were performed and the patient recovered completely.

Discussion
Migration and perforation of a tunneled haemodialysis catheter can occur late after implantation and lead to circulatory shock, thus requiring immediate diagnostic workup and surgical therapy. Routine diagnostic procedures may be insufficient for making a correct diagnosis. More specific approaches, such as multi-modality imaging including contrast echocardiography, should be implemented upon clinical suspicion.

Keywords
Tunneled haemodialysis catheter • Cardiac perforation • Pericardial tamponade • Cardiopulmonary resuscitation • Case report
Introduction

The prevalence of end-stage renal disease has increased over the past decades. In 70–90% of these cases, intermittent haemodialysis (IHD) is used for renal replacement therapy, requiring a safe vascular access. For patients where a creation of an arteriovenous fistula is unfavourable, a long-term tunnelled cuffed haemodialysis catheter (THC) is an alternative. Tunnelled cuffed haemodialysis catheter complications (e.g. catheter-associated infection, pneumothorax, vein stenosis, haematoma, thrombosis, or vascular injury) are associated with increased morbidity and mortality. Cardiac perforations remain a rare and potentially life-threatening complication in adults and usually occur in the early course. This case report demonstrates a delayed migration of a THC with subsequent atrial perforation and cardiac arrest.

Timeline

Admission to the emergency department in beginning circulatory shock upon start of a hemodialysis session.

TTE demonstrating a pericardial effusion without signs of hemodynamic compromise. CT scan is without major additional findings.

Contrast echocardiography by injecting fluid through the catheter which becomes immediately visible in the pericardial space and leads again to the development of a pericardial effusion.

Thoracotomy and recovery of the hemodialysis catheter, reconstruction of the right atrium and implantation of a pacemaker.

Start of another hemodialysis session. Sudden event of shock resulting in asystole and cardiopulmonary resuscitation.

TTE showing a pericardial tamponade. Urgent pericardiocentesis is performed. CT scan is suspicious of a catheter migration.

Discharge home in full recovery.

Learning Points

- Migration of tunnelled haemodialysis catheters may occur a long time after implantation, leading to potentially life-threatening complications, and should be taken into consideration in patients with unexplained pericardial effusions or haemodynamic compromise during or after haemodialysis.
- Detection of migrated tunnelled haemodialysis catheters may be challenging, emphasizing the value of multi-modality imaging, including contrast echocardiography.
- Considering a myocardial perforation by a migrated catheter, withdrawal of the catheter in an uncontrolled environment may have fatal complications. Thus, urgent removal should be performed by open thoracotomy.
Case presentation

We present the case of a 51-year-old woman suffering from end-stage renal disease after kidney transplantation, which had become necessary because of systemic lupus erythematosus. Her immunosuppressive therapy consisted of 1 mg of tacrolimus twice daily. Moreover, her drug history included amlodipine, bisoprolol, pravastatin, candesartan, levothyroxine, cholecalciferol, and calcitriol but no anti-coagulation. Due to a history of multiple arteriovenous fistula placements, the patient had been implanted into the right internal jugular vein more than 1 year ago. Lately, high-dose urokinase was used to lock the catheter’s proximal lumen post-dialysis because of catheter malfunction and suspicion of catheter thrombosis. Moreover, the inflow and outflow lumina were switched so that the proximal cannula was now used for blood outflow in order to perform IHD in ambulatory care (Figure 1). Nevertheless, high flow rates could not be achieved by these measures. Ambulatory IHD could only be performed without fluid removal and at a very low blood flow rate.

The patient was admitted to the emergency department because of discomfort and stabbings chest pain at the start of the regular ambulatory IHD treatment. Upon admission to the hospital, she was hypertensive (90/60 mmHg), in sinus tachycardia (110 b.p.m.) and presented with cool extremities, suggestive for circulatory shock. There were no appreciable murmurs. Arterial blood gas analysis, obtained in the emergency department, revealed a concomitant lactate acidosis and hyperkalaemia (serum lactate: 5.5 mmol/L, normal: 0.5–2.2 mmol/L, serum potassium: 8 mmol/L, normal: 3.5–4.6 mmol/L). All other laboratory values, including markers of myocardial ischaemia, were low, respectively within normal range (Troponin I 13 pg/mL, normal < 3 pg/mL; CK 52 U/L, normal <145 U/L).

The patient was transferred to the intensive care unit (ICU) for further diagnostic evaluation and treatment. Transthoracic echocardiography (TTE) showed a moderate circular pericardial effusion without echocardiographic signs of tamponade or haemodynamic significance. Left and right ventricular functions were normal. A chest radiograph revealed no clear pathological findings (Supplementary material online). A computed tomography (CT) scan ruled out pulmonary embolism and confirmed the circular pericardial effusion, measuring up to 1.2 cm (Figure 2). Beyond that, the scan was considered unremarkable. The THC tip could not be located due to contrast agent overlay.

Due to severe hyperkalaemia, a short continuous veno-venous haemodialysis (CVVHD) with a low blood flow rate (100 mL/min) without fluid removal was performed after admission to the ICU until normalization of potassium values. During the treatment, the patient complained of chest pain.

The following day sustained low efficiency dialysis (SLED; Genius™ system, Fresenius) with a higher blood flow rate of 300 mL/min was performed using the THC. Immediately at the start, the patient showed severe hypotension, tachycardia, dyspnoea, and chest pain. This acute situation progressed within minutes to asystole. Cardiopulmonary resuscitation was started, an endotracheal intubation was performed without complications and a return of spontaneous circulation was observed after three minutes. An electrocardiogram showed no signs of electrical alternans. Initial atrial fibrillation with a rapid ventricular response quickly converted into normal sinus rhythm. A blood analysis revealed an elevated serum lactate of 7.1 mmol/L and a mild hypercapnia (pCO2 of 54.7 mmHg, normal: 34.0–45.0 mmHg, other values: pO2 164 mmHg, normal: 75.0–100.0 mmHg; pH 7.35, normal: 7.36–7.44; HCO3− 27.6 mmol/L, normal: 20.0–26.5 mmol/L; BE 4.3 mmol/L, normal: -2.0 to 3.0. Na 146 mmol/L, normal: 135–145 mmol/L; Hb 9.9 g/dL, normal: 12.3–15.3 g/dL; K 5.1 mmol/L). Mild therapeutic hypothermia was induced to minimize the risk of secondary brain injury. The patient remained stable with low doses of catecholamines.

Urgent bedside TTE demonstrated an enlargement of the circular pericardial effusion up to 2 cm with the diastolic collapse of the right atrium and the free right ventricular wall, indicating cardiac compression and tamponade.

Immediate pericardiocentesis drained 350 mL of sanguineous fluid (Hb 7.2 g/dL). This led to a rapid clinical improvement in the patient’s condition, but the source of bleeding remained unclear. Significant coronary artery disease was ruled out by invasive coronary angiography. Due to primary asystole, a temporary pacemaker wire was inserted through the femoral vein.

The patient remained stable without recurrence of pericardial effusion. A CT was repeated in order to further evaluate the origin of the intra-pericardial blood. Based on the sequence of events, repeated imaging now raised the first suspicion for catheter migration through the right atrial wall into the pericardium (Figure 3, see also Supplementary material online). As the exact location could not be determined by CT, contrast echocardiography was performed, injecting contrast agent through the proximal lumen of the THC which became immediately visible in the pericardial space and led again to the development of a circular pericardial effusion (Figure 4). Hence, the diagnosis of a THC migration was made, the injected fluid was aspirated immediately and an open thoracotomy was performed urgently.

Surgical exploration confirmed an atrial perforation of the proximal tip of the catheter (Figure 5). The THC was removed (chest radiograph after thoracotomy, Supplementary material online) and the atrial laceration was closed with a bovine pericardial patch. Due to intermittent sick sinus syndrome, a permanent epicardial two-chamber pacemaker was implanted during the same procedure.

Further recovery was uneventful with extubation on the first post-operative day. The patient was transferred to the nephrological ward in stable condition and was discharged home on post-operative day 13. At follow-up, 9-month later, the patient is doing well without any neurological or cardiovascular problems. An upper arm A–V fistula is used for haemodialysis.

Discussion

Cardiac perforations associated with a THC remain a very rare (incidence from 0.0001% to 1.4%) but life-threatening complication with reported mortality ranging from 65% to 100%, mostly occurring during or early after catheter implantation. Risk factors for perforation include trauma during insertion and contact-induced or chemical erosion leading to injury of the myocardial wall over time. Furthermore, catheter type, line tip position and angle, anatomical variations, and prolonged use may play a role. Overall, 80% of all perforations are located in the right atrium and right ventricle, followed by the superior vena cava.
As, in this case report, signs and symptoms may be unspecific and occur with sudden onset, making quick identification and diagnosis challenging. A strong clinical suspicion and the use of multimodality imaging facilitated the correct diagnosis in this case. Repeated CT raised suspicion for atrial perforation of the THC, but only contrast echocardiography reliably determined the exact location of the catheter tip and identified the perforation site in the right atrium. We assume that the malfunctioning proximal tip had already been transiently located in the myocardium upon admission to the hospital but had not yet migrated permanently.
This would explain the feasibility of a short CVVHD session with a low blood flow rate. We assess that during the second dialysis session on ICU, performed with a higher blood flow rate, complete migration and permanent myocardial perforation took place. A reason for this may be the higher blood flow in the cannula, resulting in cardiac tamponade and immediate haemodynamic collapse. Previous fibrinolytic treatment with urokinase may have aggravated myocardial erosion and catheter migration.

**Figure 3** Repeated computed tomography of the thorax raising suspicion for a migration of the tunnelled cuffed haemodialysis catheter through the right atrial wall (indicated by red arrows).

**Figure 4** Transthoracic echocardiography in subcostal view obtained before (A) and shortly after (B) application of fluid through the proximal lumen of the tunnelled cuffed haemodialysis catheter, leading to rapid development of a circular pericardial effusion (marked with red arrow).
On the assumption of a catheter migration, the main objective in the management is to secure its safe and controlled retrieve. Leaving the catheter in place could lead to septicaemia, thromboembolism, and further dislocation. However, utmost caution is required when removing the catheter. Considering a perforation of the myocardium, uncontrolled withdrawal may have fatal consequences. Thus, removal should be performed directly by open thoracotomy or at least with on-site cardiac surgery backup.

In the presented case, early diagnosis of the underlying problem and urgent operative treatment yielded a positive outcome for the patient. Due to the extreme rarity of this complication, routine imaging for asymptomatic patients with a THC to evaluate for catheter migration may not be useful. However, a high alertness for potentially life-threatening THC complications is warranted in patients with recent catheter dysfunction, acute symptoms in temporal relationship with IHD and an unexplained pericardial effusion. In these cases, a multi-modality imaging approach, including CT and contrast echocardiography, is required, even if THC implantation was performed long time ago.

**Lead author biography**

Charlotte Jahnke graduated in Medicine at the Medical University of Münster (Germany) in 2014 and is currently in her specialist training for cardiology and internal medicine at the University Heart and Vascular Center Hamburg (Germany). Her main scientific focus is on cardiovascular imaging.

**Supplementary material**

Supplementary material is available at European Heart Journal—Case Reports online.

**Slide sets:** A fully edited slide set detailing these cases and suitable for local presentation is available online as Supplementary data.

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**Consent**

The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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**Data availability**

The data underlying this article are available in the article and in its online supplementary material.

**References**

1. Philipponnet C, Aniort J, Pereira B, Azarnouch K, Hadj-Abdelkader M, Chabrot P et al. Systematic review of atrial vascular access for dialysis catheter. Kidney Int Rep 2020;5:1000–1006.
2. Santoro D, Benedetto F, Mandello F, Pipito N, Barillà D, Spinelli F et al. Vascular access for hemodialysis: current perspectives. Int J Nephrol Renovasc Dis 2014;7:281–294.
3. Vascular Access 2006 Work Group. Clinical practice guidelines for vascular access. Am J Kidney Dis 2006;48 Suppl 1:S176–S247.
4. dos Santos Modelli ME, Cavalcanti FB. Fatal cardiac tamponade associated with central venous catheter: a report of 2 cases diagnosed in autopsy. Am J Forensic Med Pathol 2014;35:26–28.
5. Wong K, Marks BA, Qureshi A, Stemm J. Migration of a central venous catheter in a hemodialysis patient resulted in left atrial perforation and thrombus formation requiring open heart surgery. A A Case Rep 2016;7:21–23.
6. Orme RM, McSwiney MM, Chamberlain-Webber RF. Fatal cardiac tamponade as a result of a peripherally inserted central venous catheter: a case report and review of the literature. Br J Anaesth 2007;99:384–388.

7. Booth SA, Norton B, Mulvey DA. Central venous catheterization and fatal cardiac tamponade. Br J Anaesth 2001;87:298–302.

8. Domino KB, Bowdle TA, Posner KL, Spittellie PH, Lee LA, Cheney FW. Injuries and liability related to central vascular catheters: a closed claims analysis. Anesthesiology 2004;100:1411–1418.

9. Tayal VS, Kline JA. Emergency echocardiography to detect pericardial effusion in patients in PEA and near-PEA states. Resuscitation 2003;59:315–318.