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Case report

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Late atrial perforation from tunnelled haemodialysis catheter

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Introduction

Central venous cannulation is a commonly performed procedure in HD patients. One complication associated with such a procedure includes cardiac tamponade, thought to arise from the guidewire, dilator or venous cannula piercing the myocardial wall. This particular risk may be minimized by the use of soft j-tipped wires, ensuring cannulae are not advanced too far or against resistance and the use of post-procedure chest radiographs to confirm correct catheter positioning. Most such complications, however, occur at the time of insertion. We report here a rare case of a soft silicone HD catheter migration 2 years following insertion.

Case report

A 48-year-old Afro-Caribbean lady, on long-term HD, presented in accident and emergency with retrosternal chest pain and shortness of breath of 12 h duration. There was no previous history of angina, ischemic heart disease, trauma or thromboembolic disease. She suffered from established renal failure from hypertensive nephrosclerosis and had been treated by regular HD for 5 years, initially through a left brachial cephalic fistula. This had failed 2 years previously after which a central venous catheter (Permcath) had been inserted in the ® internal jugular, under fluoroscopy guidance. She took Doxazosin, Nifedipine and Metoprolol for her long-standing hypertension.

On examination, her blood pressure was 210/110 mmHg with normal pulse and respiratory rate. She was apyrexial and had normal oxygen saturation on room air. There were minimal basal crepitations bilaterally and normal heart sounds. There was slight pedal oedema. Her ECG showed left ventricular hypertrophy with ST elevation in V_2, V_3 and T wave inversion in lateral leads all of which had been noted on previous recordings. Her haemoglobin on admission was 8.4 g/dl (normal 12–14 g/dl), serum creatinine 1000 μmol/l (normal) and albumin 26 g/l. Blood gas examination was normal. Her D-dimer was 703 ng/ml and Troponin-I was borderline at 0.055 ng/ml. She was immediately treated according to acute coronary syndrome protocol that included the administration of subcutaneous Clexane. Her chest pain subsided with sublingual GTN spray. The same day she had her routine HD session that was uneventful. Some hours later she experienced a further episode of retrosternal chest pain that subsided with sublingual GTN spray. An ECG was unchanged. She underwent two further uneventful HD sessions during the following 72 h. During the latter session, 2.7 l of fluid were removed, and her post-dialysis blood pressure was 160/97 mmHg.

A few hours following this session, she suddenly became agitated and confused and her Glasgow Coma Scale deteriorated. She developed dysarthria, aphasia, left-sided weakness and bilateral plantar extensors. Her anticoagulant treatment was stopped. An urgent CT head scan was unremarkable. At this stage she had a markedly elevated leucocyte count 23.2×10^9/l, and a rapidly progressive lactic acidosis with pH 7.03, bicarbonate 8 mmol/l and lactate 28 mmol/l and base excess of −38 mmol/l. She required intubation and ventilation for maintenance of her airway. Her liver function tests revealed deranged parameters with ALT of 6858 IU/l. There was severe hypoglycaemia and grossly deranged coagulation. She required high doses of inotropic support to maintain her blood pressure. Her condition continued to deteriorate over the next few hours, despite full support and she died from progressive multiple organ failure.

Post-mortem examination revealed erosion of the right atrial wall by one arm of the HD catheter that had caused contusion of the overlying pericardium. There was a bloody pericardial effusion of ~100 ml. Atrial muscle necrosis was evident relating to the area of damage by the catheter. There was marked left ventricular hypertrophy but no evidence of recent or previous myocardial infarction. The coronary arteries revealed mild atherosclerosis. The cardiac valves were within normal limits. There was a type 2 dissection of the ascending aorta 7 cm in length, with no evidence of re-entry or adventitial rupture. Histology showed features of myxoid changes in the arterial wall. The lungs showed pulmonary oedema. The small bowel showed haemorrhagic infarction. The main arterial branches showed atherosclerosis but were patent. There was no evidence of venous thrombosis.

Discussion

Central venous catheter migration may be primary or secondary.[1] Primary migration occurs during insertion and the incidence depends on the approach of insertion ranging from 30 to 5% in the external jugular and the subclavian, respectively. Secondary migration occurring weeks to months after insertion is widely variable too and is reported in 5–55% of cases. In such migrations, the right atrium is eroded into in ~9% cases. Secondary migration in subclavian vein catheters are reported less often although catheter fracture and embolization of the distal fragment are well-known complications of long-term subclavian vein cannulation.[2]

Secondary migration of subclavian central vein catheters with atrial perforation is unusual.[3–6] We have found no reported cases of this occurring as late as 2 years after insertion as occurred in our case. Factors associated with cardiac perforation have been summarized[5,7,8] and include catheter construction, catheter-induced endocardial injury leading to thrombus formation and adherence of the catheter to the heart wall leading to wall erosion accentuated by cardiac contractions, positioning of the tip of the catheter post-insertion, length of the superior vena cava and poor fixation of catheter to the skin.

Significant cephalad migration of HD catheters may occur even induced by such changes as movement of the patient from supine to upright position[9,10] especially in obese patients and women with pendulous breasts.[10,11] In our case though there was distal migration after 2 years. Atrial placement may minimize catheter migration associated with changes in posture. In our patient, atrial perforation occurred despite the use of the softer silicone material.[4,12] In our case, atrial perforation was not associated with prior evidence of catheter malfunction, and perforation was a subacute or chronic event judged by the findings on post-mortem. The degree of pericardial fluid accumulation was not sufficient to cause tamponade but may, in addition to her dissecting aneurysm, have been partly responsible for her chest pain.

To minimize such problems, it has been suggested that catheter placement takes place by the right internal jugular route, when this is possible, with the catheter tip being placed at the junction of the superior vena cava and the right atrium or in the proximal right atrium.[13] The tip of soft silicone-tunnelled catheters should be placed within the atrium that should span the atrio-caval junction in an inspiratory film, further confirmed at fluoroscopy,[14,15] which also minimizes recirculation.[4] Clearly, AV fistulas are the preferred form of venous access and their use represents the optimal means of avoiding all catheter-related problems.

This patient on regular HD presented with chest pain probably relating to a dissecting aneurysm of the ascending aorta that was unrecognized pre-mortem. Subsequently she deteriorated suddenly and died from multiple organ failure probably related to small bowel infarction, which in the absence of occlusive disease of the superior mesenteric vessels, was most likely from thrombotic microangiopathy or disseminated intravascular coagulation. The post-mortem findings of a dissecting aneurysm of the ascending aorta and atrial perforation with associated bloody pericardial effusion could both have been triggers for this thrombotic state. Although our explanation of events leading to the patient's death is speculative in nature, the aspect of the case we wish to focus on is the late migration of the tunnelled central venous HD catheter causing atrial perforation, which is extremely rare.

In conclusion, we present a rare case of soft silicone HD Permcath migration and atrial perforation occurring 2 years after insertion. When HD patients using these devices as dialysis access present with chest pain, such complications should be considered as part of the differential diagnosis, even when considerable time has elapsed after catheter placement.

Conflict of interest: None declared.

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