A case report of a late left atrial appendage perforation 4 months after occluder implant: reason for or caused by a resuscitation?

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Background

Atrial fibrillation (AF) is a common disease and can lead to cardioembolic stroke. Stroke prevention according to the CHA2DS2-VASc score is achieved via oral anticoagulation. In recent years, interventional occlusion of the left atrial appendage (LAA) has become a common alternative. Besides showing non-inferiority in large trials compared with warfarin, interventional LAA occlusion can lead to serious adverse events with most of them occurring peri-interventionally.

Case summary

A 75-year-old man with AF and recurrent gastrointestinal bleedings was referred for an interventional closure of the LAA. The intervention was successful with an ABBOTT V R Amulet device. Four months later, the patient had to be resuscitated. Return of spontaneous circulation occurred after 10 min. On hospital arrival, echocardiography revealed a pericardial tamponade and 2 L of blood were drained. A coronary angiogram revealed a lesion with active leakage of contrast agent in the proximal circumflex artery. The patient was transferred to the cardiac surgery department immediately. Intra-operatively a perforation of the tissue at the basis of the LAA close to the left main coronary artery was discovered. The occluder was excised and the LAA was closed by endocardial sutures.

Discussion

In this report, we review the literature concerning interventional LAA occlusion and the reported cases of LAA perforation. Retrospectively, it remains unclear whether the perforation caused the resuscitation or was induced by it. To our knowledge, this is the first reported case of a laceration of a coronary artery by an occlusion device.

Keywords

Case report • Atrial fibrillation • Stroke prevention • Cardiac tamponade • LAA occluder device • Resuscitation

Introduction

Atrial fibrillation (AF) is a common disease and can lead to cardioembolic stroke.1 In patients with known AF and an elevated CHA2DS2-VASc score stroke prevention is advised using an oral anticoagulant (OAC).2 Owing to the observation that the majority of
cardiogenic thrombi originate in the left atrial appendage (LAA), percutaneous interventional occlusion of the LAA has been introduced as an alternative for patients that have a high bleeding risk or are intolerant to OAC. For such patients, the current European Society of Cardiology (ESC) guidelines recommend the interventional LAA closure (LAAC) with a class of recommendation of IIb and a level of evidence B. Several studies demonstrated non-inferiority compared with warfarin. However, interventional LAAC can lead to serious adverse events most commonly occurring peri-interventionally. In a European registry, a procedure-related complication rate of 4% at 30 days has been reported. In this registry of 1021 patients undergoing LAAC, there were seven cases of pericardial effusion with three of them requiring pericardiocentesis within 30 days of the procedure.

### Timeline

| Date       | Event                                                                 |
|------------|----------------------------------------------------------------------|
| November 2016 | Successful percutaneous interventional closure of the left atrial appendage (LAA) with an ABBOTT Amulet device (18 mm) |
| April 2017  | Patient complained of acute progressive dyspnoea and called the emergency medical system (EMS) |
| 8:30 a.m.   | On EMS arrival, the patient is conscious, vital signs show: blood pressure of 80/50 mmHg, heart rate of 90/min, oxygen saturation of 85% |
| 9 a.m.      | Cardiac compressions and application of catecholamines led to a return of spontaneous circulation |
| 10 a.m.     | In the ED Ultrasound revealed a cardiac tamponade, a drainage tube was installed and the patient transferred to the intensive care unit |
| 3 p.m.      | More than 2 L of blood have been drained |
| 6 p.m.      | Intra-operatively a perforation of the tissue at the basis of the LAA close to the left main coronary artery was discovered |
| June 2017   | The occluder was excised and the LAA was closed by endocardial sutures, two pledget sutures were used to seal the epicardial lesion at the LAA basis which stopped the bleeding |

### Case presentation

A 75-year-old man with persistent AF, a CHA2DS2-VASc score of 4 and a HAS-BLED score of 4 was referred to a tertiary clinic for a LAAC. His medical history consisted of a chronic obstructive pulmonary disease (COPD), coronary artery disease, and a two-thirds gastric resection performed in the 1980s. The intervention was successfully performed with an ABBOTT Amulet 18 mm device being implanted (Figure 1A). After the implantation, he received acetysalicylic acid (ASA) and Clopidogrel for 6 months. Approximately 5 months post-intervention, the patient expressed acute dyspnoea and called the emergency medical services (EMS).

On arrival of the EMS, the patient was conscious, severely distressed and hypotensive with a blood pressure of 80/50 mmHg and a heart rate of 90/min. Peripheral oxygen saturation of 85% measured percutaneously was noted. After the administration of bronchodilator medication for a presumed COPD exacerbation, the patient’s clinical condition deteriorated with a resultant cardiac arrest with pulseless electrical activity. The patient received cardiopulmonary resuscitation including chest compressions, endotracheal intubation, and the administration of 3 mg Adrenaline. Return of spontaneous circulation occurred approximately 10 min later at which point he was transferred to a local hospital. During the transport, the cardiorespiratory parameters were relatively stable with a systolic pressure around 100 mmHg. The capnometry after return of spontaneous circulation measured a PCO2 of 60 mmHg. Upon arrival at the hospital, the physical examination showed jugular vein distension but no peripheral oedema.

Echocardiography performed in the emergency department revealed a pericardial tamponade requiring pericardiocentesis via the subxiphoidal route, 800 mL of blood were initially evacuated. The differential diagnosis included primary respiratory failure or a sudden cardiac death with secondary pericardial effusion induced by cardiac compressions under double antiplatelet therapy, a ruptured aortic aneurysm, a myocardial rupture, or a primary laceration of a cardiac structure by the LAA occluder. A ruptured aneurysm of the ascending aorta was excluded by ultrasound. Electrocardiogram examination did not demonstrate any ST-segment elevation or low voltage. Blood gas analysis documented a stable respiratory situation with a PaO2 of 230 mmHg and a PaCO2 of 55 mmHg, the pH was 7.3 and Lactate elevated with 10 mmol/L (<2 mmol/L). The haemoglobin was measured at 9 g/dL (12–16 g/dL). The chest X-ray demonstrated the proper placement of the endotracheal and pericardial tube and ruled out a pneumothorax or pulmonary oedema.

In the hours following pericardiocentesis, approximately 2 L of blood were evacuated. Conventional methods of coagulopathy management including application of tranexamic acid, fresh frozen plasma, and fibrinogen did not stop the bleeding. To stabilize the patient a total of eight packs of red blood cells were transfused.

In preparation of cardiac surgery, a coronary angiogram was performed (Figure 1B). It revealed active leakage of contrast agent in the proximal circumflex artery and the patient was transferred to the cardiac surgery department immediately. Intra-operatively a perforation of the tissue at the basis of the LAA close to the left main coronary artery was discovered. Under protection of cold blood cardioplegia on cardiopulmonary bypass the occluder was excised through the opened left atrium. The LAA was closed by endocardial sutures and
two pledget sutures were used to seal the epicardial lesion at the LAA basis which stopped the bleeding. Fortunately, after prolonged treatment in the intensive care unit and weaning ward the patient was discharged and alive 4 months after the event without neurological deficit and on ASA monotherapy. Transthoracic echocardiogram in the weaning ward ruled out a persistent pericardial effusion.

Discussion

In any resuscitation point of care ultrasound is needed to exclude treatable courses of cardiac arrest. In this case, it revealed a pericardial tamponade and after insertion of a drainage tube the patient stabilized. As for the differential diagnosis, several reasons for a pericardial effusion need to be considered. Probably, the most common cause is an iatrogenic rupture of cardiac structures which is a possible complication of most cardiac interventions, i.e. coronary stenting, pacemaker implantation, or electrophysiology study. Ruptured aortic aneurysm can be diagnosed via ultrasound or computed tomography scan. A rare complication of a myocardial infarction is a rupture of a left ventricular aneurysm and is often fatal.

In the literature, there are reported cases of an acute perforation of the LAA during occluder implantation leading to cardiac tamponade. Sarcon et al. report a case of an unintended sheath motion during release of the device leading to a LAA perforation. Another case report of an implantation of an Amulet device describes a perforation of the pulmonary artery by the hooks of the device leading to a cardiac arrest 8 h after the implantation.

Cases of delayed pericardial perforations have been reported following interventional septal occlusion. One case report describes a delayed pericardial tamponade by an atrial septal defect (ASD) occluder 79 days after implantation leading to cardiac arrest. Taggart et al. report a case of a pericardial perforation occurring 6 years after interventional patent foramen ovale (PFO) closure. In the setting of interventional septal occlusion, it has been postulated that these late pericardial perforations have been induced by the rigid material of the occluder that can potentially erode the left atrial roof or the aortic root. This complication is very rare but life-threatening after interventional placement of PFO and ASD closure devices, the incidence ranging from 0.1% to 0.3%. Patients that received an oversized septal occluder or have a short anterosuperior rim are at higher risk of device induced erosion.

In review of the clinical information in the reported case, it remains unclear whether a primary perforation of the LAA led to the ambulatory cardiac arrest. Since there were more than one laceration of the LAA, it seems also possible that the thoracic compressions led to the perforation and consecutive pericardial tamponade while the initial collapse was caused by an exacerbated COPD.

To our knowledge, this is the first reported case of a potential late laceration of the circumflex artery by an LAA occluder. This possible complication has to be kept in mind when treating a patient with cardiac arrest and a history of LAA occluder placement.

Lead author biography

Dr Karsten Schenke was born in 1976 in Hannover. He is an Interventional Cardiologist working in Hamburg at academic hospital AK Barmbek. His fields of interests are radial access for coronary interventions, implantation of PFO/LAA occluder and intensive care.
Supplementary material

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

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

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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