Intra-atrial endothelial lesion resulting from transseptal puncture for catheter ablation of atrial fibrillation

Conrad Genz, Hans D. Esperer, Alexander Schmeisser, Ruediger C. Braun-Dullaeus, Samir M. Said

Otto-von-Guericke University, University Hospital Magdeburg, Division of Cardiology, Section of Electrophysiology, Magdeburg, Germany

Abstract

Thromboembolic events are known complications of left atrial ablation therapy. We describe a complication which may also lead to systemic thromboembolism that has not been reported so far: the formation of a moving structure attached to the fossa ovalis after an attempted transseptal puncture in a 66-year old patient with symptomatic paroxysmal atrial fibrillation.

Introduction

Atrial fibrillation (AF) is the most common permanent cardiac arrhythmia with an increased risk of stroke, congestive heart failure and overall mortality. Therefore, a curative approach is the primary therapeutic goal. Recent studies have shown the superiority of catheter ablation methods compared to antiarrhythmic drug therapy[1-3] with an even smaller rate of treatment-related adverse events with the invasive approach (4.9% *vs* 8.8%).[3] Anne *et al.* classified known complications of AF ablation therapy into four partly overlapping categories with operator experience and patient profile always acting as modulating factors.[4] One of these complications is systemic thromboembolism eventually resulting in apoplexia, acute myocardial infarction or peripheral vascular compromise.[5,6] Causes of thromboembolic events may be: i) activation of the extrinsic coagulation cascade due to endothelial damaged caused by ablation;[7] ii) thrombus formation on catheters and sheaths; iii) char formation during the ablation process; iv) detachment of a pre-formed thrombus in the left atrium.[5] In this paper, we describe a hitherto unreported type of adverse event after attempted transseptal puncture (TSP), namely the formation of a floating structure attached to the interatrial septum. This finding could be classified as a procedure-dependent or device-dependent complication with a potential risk of systemic thromboembolism.

Correspondence: Samir M. Said, Otto-von-Guericke University, University Hospital, Division of Cardiology, Section of Electrophysiology, Leipziger Str. 44, 39120 Magdeburg, Germany.
Tel. +49.391.67.15206 - Fax: +49.391.67.15211.
E-mail: samir.said@med.ovgu.de

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Case Report

An otherwise healthy 66-year old patient with a heavy burden of paroxysmal attacks of AF was admitted to our hospital for catheter ablation because prior antiarrhythmic drug therapy with multiple agents had failed. Routine blood tests did not show any abnormalities with all relevant parameters being in the normal range. Pre-procedural transesophageal echocardiography (TEE) revealed a structurally normal heart and the absence of thrombi in both atria as well as a persistent foramen ovale (Figure 1).

Catheter ablation

During the TSP, emission of radiopaque material into the pericardial cavity became evident (Figure 1) upon which the procedure was aborted immediately. Echocardiography showed that the attempted TSP had not resulted in greater damage and excluded pericardial effusion. The patient was discharged a day later and oral anticoagulation (OAC) therapy restarted seven days post discharge. He was scheduled for a second attempt at catheter ablation eight weeks later. At the time of readmission, low-molecular-weight heparin was administered subcutaneously at a therapeutic dose after OAC had been stopped five days before. This time, TSP was TEE-guided but not further pursued after TEE revealed the presence of a filiform floating structure 4 mm in length that was attached to the right atrial side of the fossa ovalis (Figure 2). The procedure was aborted and OAC was reinitiated with overlapping low-molecular-weight heparin in therapeutic dosage given the presumed thromboembolic genesis of the structure and the intention to attempt re-ablation again should the structure disappear. We referred the patient to our outpatients clinic for TEE reassessment after effective OAC for two months after which TEE again demonstrated the presence of the above described structure (Figure 2).

Discussion

There are several possible reasons for the formation of the intracardiac structure we observed. The failed attempt at TSP might very well have caused an endothelial alteration which resulted in thrombus formation. However, if the structure we saw was a thrombus, one would have expected it to disappear after two months of efficient oral anticoagulation. Instead, the structure was unchanged at the time of re-evaluation. Another possible reason is bacterial endocarditis. However, the patient had remained in a clinically healthy condition during the entire follow-up period and had never developed any clinical or laboratory signs of inflammation or infection. In addition, no valvular or endocardial abnormalities had been revealed by TEE that would have favored bacterial adherence. Moreover, TEE had not shown any evidence for the existence of the filiform structure prior to the attempted ablation.

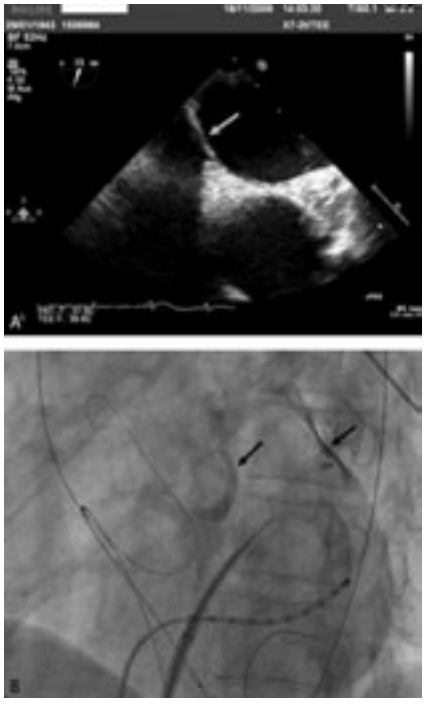

Figure 1. (A) Transesophageal echocardiogram before starting transseptal puncture showing intact intra-atrial septum (arrow); (B) emission of radiopaque material into the pericardial cavity (arrows) during the transseptal puncture.

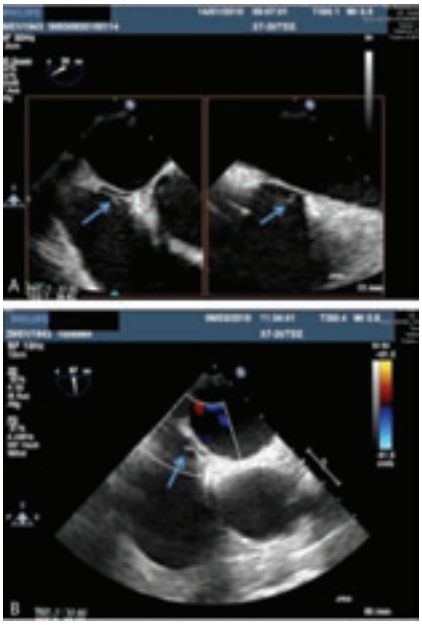

Figure 2. Transesophageal echocardiogram eight (A) and 16 (B) weeks after the attempted transseptal puncture. Note the filiform structure (arrow) the tip of which is floating in the right atrium and whose base is attached to the fossa ovalis.

Therefore, it is very unlikely that bacterial endocarditis was involved in the genesis of this structure. We, therefore, believe that the most likely reason for the observed filiform structure was that a piece of endothelial tissue inadvertently peeled off during the futile TSP attempt. This complication of TSP has never previously been reported and, if it went unnoticed, it could result in thromboembolic events. Although minor tissue detachment in the right atrium does not normally pose a serious problem, a major complication may arise if, during a repeated TSP, a piece of the detached tissue is displaced into the left atrium across the canal of the sheat.

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