Thrombotic cardiac tamponade after transseptal puncture

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Introduction

Hemopericardium and subsequent tamponade is an insidious complication of cather ablation procedures for atrial fibrillation (AF).

Case report

An 84-year-old woman was admitted for catheter ablation of long-standing, permanent AF. Her previous medical history consisted of long-standing arterial hypertension and persistent AF diagnosed 3 years ago. She had undergone circumferential pulmonary vein (PV) isolation (ie, linear ablation around the PV antra with subsequent verification of PV isolation) and repeat ablation of a macroreentrant atrial tachycardia around the left superior PV. She had remained in sinus rhythm for 2 months postablation, and paroxysmal AF was detected after cessation of amiodarone therapy. On admission, clinical examination was unremarkable. She was in sinus rhythm, her blood pressure was 130/90 mm Hg, and echocardiography revealed normal left ventricular ejection fraction (65%), left atrial diameter of 32 mm, and grade I mitral and tricuspid regurgitation. She had no signs of myocardial hypertrophy or pericardial thickening. Her medications consisted of irbesartan/hydrochlorothiazide, bisoprolol, and low-dose aspirin. Her warfarin had been replaced with subcutaneous enoxaparin 3 days ago, and her international normalized ratio was 1.1.

She was taken to the electrophysiology laboratory for circumferential PV isolation of all PVs and ganglionated plexi ablation, according to our standard protocol.[1] Transseptal puncture was performed at a high atrial septum site, and difficulty was encountered in crossing the septum, which had become fibrosed after the previous 2 procedures. Immediately afterward and before heparin was given, the patient felt unwell. She became relatively hypotensive (80/50 mm Hg), and clinical signs of tamponade were obvious. Echocardiography demonstrated an echolucent space compressing the right atrium, considered to be a moderate pericardial effusion, with impaired right ventricular filling and mitral flow velocity paradoxus. Pericardiocentesis was attempted immediately under fluoroscopic and echocardiographic guidance, without success. Although the needle was clearly within the pericardial space, no blood was drawn. An emergency thoracotomy was performed, which revealed a large clot within the pericardium adjacent to the lateral wall of the left atrium and left ventricle, without ongoing bleeding. The patient experienced immediate relief and recovered uneventfully. Subsequent thrombophilia testing, including factor V Leiden and prothrombin mutations, and antiphospholipid antibodies revealed a borderline lupus anticoagulant, without anticardiolipin and anti–β_2-glycoprotein-1 antibodies (Table 1).[2,3] The patient refused further ablation and was placed on oral anticoagulation.

Discussion

Even though ablation procedures have evolved to offer increased success rate and reduced complications, hemorrhagic events remain an insidious complication. Among the complications, the development of hemopericardium and subsequent tamponade has been documented in approximately 1.2% of patients.[4–6] In most cases, pericardiocentesis and percutaneous drainage can provide effective treatment; however, surgical intervention is sometimes needed, mainly in the setting of uncontrolled pericardial bleeding.

Two underrecognized clinical entities that can lead to tamponade and can be difficult to identify with transthoracic echocardiography as well as to treat with percutaneous drainage are loculated hemopericardium (eg, in patients with prior cardiac surgery and preexisting intrapericardial adhesions) and coexisting huge pleural effusion.[7,8] Immediate pericardial thrombus is an extremely rare possibility because small amounts of blood in the pericardial space do not result in immediate clot formation.[8] In fact, intracavitary blood has long been known to be incoagulable,[9] and even though evidence for pericardial effusions does not exist, extrapolation of hemothorax characteristics suggests that hemopericardium is defibrinated and thrombocytopenic, and contains elevated levels of D-dimers.[10] Accordingly, early development of pericardial thrombus is an unlikely finding.

KEYWORDS Atrial fibrillation; Ablation; Transseptal puncture; Tamponade
ABBREVIATIONS **AF** = atrial fibrillation; **PV** = pulmonary vein
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KEY TEACHING POINTS

- Pericardial thrombus is an extremely rare possibility, but it may occur and, if accompanied by tamponade physiology, would require surgical evacuation.
- Female gender and older age confer increased complication risk.
- Repeat transseptal punctures are associated with a higher risk for cardiac tamponade because of scarring of the previously perforated septum.

Nevertheless, it may occur and, if accompanied by tamponade physiology, would require surgical evacuation.[8]

Our case also emphasizes the need for thorough preprocedural assessment of patient characteristics predisposing to complications and appropriate adaptations of treatment strategy. Several periprocedural complications have been associated with female gender, older age, and procedural complexity,[11] and previous ablation procedures and repeat transseptal punctures carry a 3-fold higher risk for cardiac tamponade because of scarring of the previously perforated septum.[5] When a difficult transseptal puncture is anticipated, intracardiac echocardiography has been shown to reduce the rate of hemopericardium and tamponade to 0.25%.[11]

Table 1 Thrombophilia testing

1. Blood cell count with peripheral smear, hepatic and renal function, and serum protein electrophoresis.
2. Genetic testing for *factor V Leiden* and *prothrombin* G20210A mutations, especially if an inherited thrombophilia is suspected.
3. *Antiphospholipid antibodies* (eg, lupus anticoagulant, anticardiolipin antibodies, anti–β2-glycoprotein-1 antibodies). Testing for antiphospholipid antibodies requires confirmation 12 weeks after an initial positive result.
4. *Antithrombin, protein C,* and *protein S,* at least 6 weeks after the event.

Note: Direct leukocyte genomic DNA testing for factor V Leiden and prothrombin G20210A mutations is unaffected by anticoagulation therapy. Heparin therapy can lower antithrombin activity and antigen levels and can impair interpretation of clot-based assays for a lupus anticoagulant. A delay of at least 5 days after heparin is stopped before testing usually is feasible. Warfarin therapy reduces the activity and antigen levels of vitamin K–dependent factors, including proteins C and S (up to 6 weeks). Non–vitamin-K-dependent oral anticoagulants (NOACs) may cause false-positive lupus anticoagulant (dilute Russell viper venom time) test results and falsely low antithrombin activity. Testing should be delayed until the effects of warfarin or NOACs therapy have resolved.

Furthermore, on occasion a higher puncture position or a larger-curve Brockenbrough needle may be needed.[12] In the presence of a dilated left atrium, a technique used in mitral valvuloplasty may be of help.[13] The issue of uninterrupted anticoagulation with either a vitamin K antagonist or Xa inhibitor,[14,15] which appears to be safer than bridging to heparin, or no anticoagulation 2–3 days before ablation also may be raised. However, the limited experience of just 1 case does not allow any definitive conclusions in this respect.

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