An 84-year-old woman was admitted for catheter ablation of atrial fibrillation (AF). She had become symptomatic, and differential pulmonary vein isolation was performed at a high atrial septum site, but no blood was drawn. Echocardiography demonstrated an echolucent space within the pericardial space, which was confirmed under fluoroscopic and echocardiographic guidance, without success. Although the needle was inserted into the pericardium, 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-β2-glycoprotein-I antibodies (Table 1). 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. 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. Immediate pericardial thrombus is an extremely rare possibility because small amounts of blood in the pericardial space do not result in immediate clot formation. In fact, intracavitary blood has long been known to be incoagulable, 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. Accordingly, early development of pericardial thrombus is an unlikely finding.
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

Furthermore, on occasion a higher puncture position or a larger-curve Brockenbrough needle may be needed.12 In the presence of a diluted 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.

References

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.