

during the intervention. The ACT should be between 300 ms and 350 ms. Inferior vena cava filters are usually endothelialized after 3 weeks of device deployment. Crossing IVC filters after three or more weeks is thus relatively safe if a careful technique is utilized (9). In our case report, the filter had been placed 8 years prior to the EP study. We felt that the filter was in a stable position, and potential risk for migration or dislodgment was low. Contrast injection before crossing the filter will ensure there is no significant thrombus that might be dislodged (10). Each time the catheters are passed through the filter strict fluoroscopic monitoring must be performed. The balloon of the EnSite catheter should be fully deflated. To prevent against guidewire entrapment, the EnSite catheter and guidewire should be removed together. Straight guide wires or guide wires with soft tips should be chosen (4).

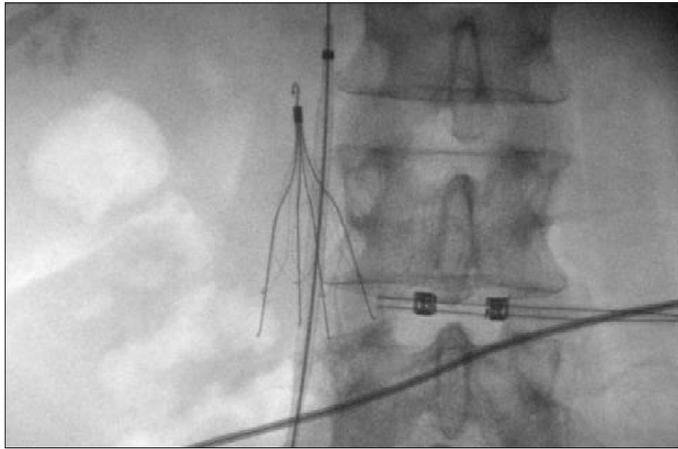


Figure 1. EnSite catheter with deflated balloon is being negotiated across the inferior vena cava filter

An EP study in a patient with an IVC filter system can be safely carried out from the femoral vein, even several times, without any complications if appropriate precautions are taken.

Acknowledgements

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References

1. Morady F. Radio-frequency ablation as treatment for cardiac arrhythmias. *N Engl J Med* 1999; 340: 534-44.
2. Dixit S, Callans DJ. Role of contact and non-contact mapping in the curative ablation of tachyarrhythmias. *Curr Opin Cardiol* 2002; 17: 65-72.
3. Sing RF, Adrales G, Baek S, Kelley MJ. Guide wire incidents with inferior vena cava filters. *J Am Osteopath Assoc* 2001; 101: 231-3.
4. Kusssmaul WG 3rd, Secaira R, McCormick DJ, Cohen M. Right heart catheterization in the presence of an inferior vena cava filter. *Catheter Cardiovasc Interv* 2001; 52: 479-80.
5. Recto MR, SobczykWL. A novel technique to prevent displacement of inferior vena cava filter during cardiac catheterization with subsequent transcatheter closure of a patent foramen ovale in a patient with cryptogenic shock. *J Invasive Cardiol* 2002; 14: 471-3.
6. Singer I. Catheterisation and electrogram recordings. In: Singer I, editor. *Interventional Electrophysiology*. Baltimore, Maryland, USA: William & Wilkins; 1997. p. 35-8.
7. Erenberg FG, Shim D, Beekman RH 3rd. Intraoperative hemorrhage associated with transhepatic cardiac catheterization: a report of two cases. *Catheter Cardiovasc Diagn* 1998; 43: 177-8.
8. Sinha SK, Harnick D, Gomes JA, Mehta D. Electrophysiologic interventions in patients with inferior vena cava filters: Safety and efficacy of the transfemoral approach. *Heart Rhythm* 2005; 2: 15-8.
9. Rhodes JF, Miller SG, Lane GK, Latson LA. Transcatheter interventions across an inferior vena cava filter. *Catheter Cardiovasc Interv* 2003; 59: 333-7.
10. Schoeffler M, Ringewald J, Schechter E. Transfemoral venous access through inferior vena cava filters for interventions requiring large sheaths. *Catheter Cardiovasc Interv* 2007; 69: 47-51.

Combined heterozygote factor V Leiden mutation and anticardiolipin antibody positivity in a young patient with spontaneous deep vein thrombosis

Kombine heterozigot faktör V Leiden mutasyonu ve antikardiyolipin antikor pozitifliği olan genç bir hastada derin ven trombozu

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Introduction

In this report, we present an interesting case of a combined heterozygote factor V Leiden mutation and anticardiolipin antibody positivity in a young patient with spontaneous deep vein thrombosis, which emphasizes importance of thrombophilia.

Case Report

A 21-year-old male was referred to our hospital because of an acute onset pain on his lower extremities. He had no previous medical history and no precipitating condition such as trauma, surgical procedure or immobility. Also he had no history of alcohol or cigarette consumption.

His father had a coronary artery disease and his mother had a history of two spontaneous abortions.

On physical examination, he had painful thighs and positive Homan's signs on both lower extremities. There was no other abnormal sign. On venous Doppler ultrasound, deep venous thromboses were established, extending from the calf veins through the popliteal, superficial, and common femoral veins and into external iliac veins (Fig.1, 2). After taking venous blood samples, low molecular weight heparin was implemented to the patient.

Abnormal laboratory tests were as follows: a polymerase chain reaction revealed heterozygosity for factor V Leiden. Among other tests, lupus anticoagulant test was negative. The patient had normal antithrombin III, protein S and C levels as well. Anticardiolipin antibody (aCL) immunoglobulin M (IgM): 46.3 MPL units (normal < 9 MPL units) and aCL immunoglobulin G (IgG) 23 MPL units (normal < 9 MPL units).

His symptoms subsided in few days after the anticoagulant treatment. He was then switched to warfarin treatment and discharged on warfarin to be taken indefinitely. In the follow up period his symptoms completely resolved and he had not any further thromboembolic event during one year of follow-up.

Discussion

Thrombophilia is an either inherited or acquired tendency for venous thromboembolism. An early age (<45 years) thrombosis without any

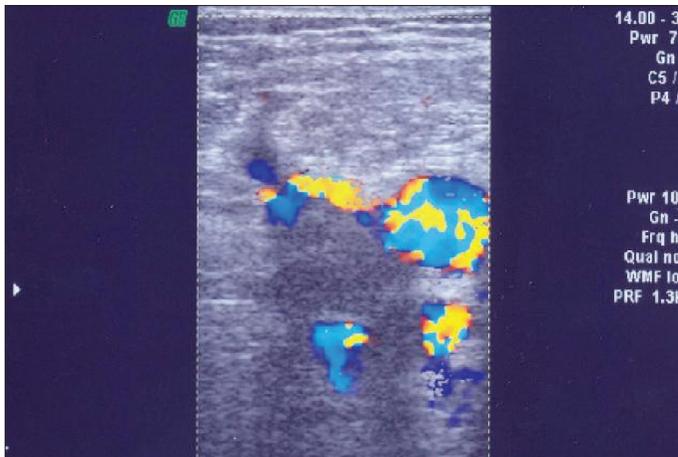


Figure 1. Color venous Doppler ultrasound of patient with thrombus

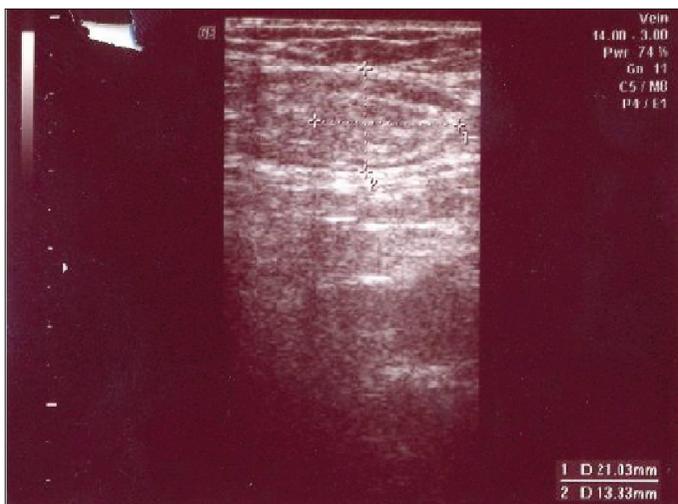


Figure 2. Venous Doppler ultrasound of patient with thrombus

precipitating condition, especially at unusual sites are the clinical features suggesting thrombophilia.

Factor V Leiden mutation is present in the 4-6% of the general population and is the most common inherited cause of the syndrome accounting for the 40-50% of cases (1). The heterozygosity of factor V Leiden is associated with three to seven fold increased risk of venous thromboembolism (2). Other less common causes of inherited thrombophilia are the antithrombin III, protein C and S deficiencies, and rare conditions such as plasminogen and heparin cofactor-II deficiencies and dysfibrinogenemia. However, the lifetime probability of developing thrombosis and the severity of the thromboses seem to be considerably less in heterozygotes with the factor V Leiden mutation than in patients with the less common inherited thrombophilias (3). Moreover, there is an increased incidence of factor V Leiden among thrombotic patients with other thrombophilic defects, as compared to the general population. The presence of additional inherited defects increases the thrombotic risk comparing to single factor V Leiden mutation (4, 5). The acquired risk factors such as oral contraceptives, hormone replacement therapy and pregnancy also increase the risk of thrombosis when they are together with factor V Leiden mutation (6).

Although there are numerous reports regarding both inherited and acquired thrombotic risk factors combined with Factor V Leiden mutation, we have not noticed any data about the association of ACA and Factor V Leiden mutation. Our search for the Medline revealed a case report about an association of Factor V Leiden mutation and lupus anticoagulant in a young man who developed lower extremity deep vein thrombosis (7). The present report about the association of high ACA and Factor V Leiden mutation is therefore puts forward an uncommon association in the risk of thrombophilia. Yet, there is some conflicting data about the role of ACA or lupus anticoagulants on the risk of thrombophilia. Antiphospholipid antibodies can be detected in approximately 5 to 21 percent of all patients with venous thrombosis, although this does not necessarily point out a casual relationship. There are however, reports regarding the increased risk of deep venous thrombosis due to the presence of lupus anticoagulant or ACA, and a recent meta-analysis showed that the risk for antiphospholipid associated thrombosis demonstrated a higher risk in patients with the lupus anticoagulant and aCL than in other patients (8).

Because of the above data, we considered that the cause of the massive deep vein thrombosis of our case was the combination of Factor V Leiden mutation and high aCL IgG and IgM levels. As the patient had two permanent risk factors of venous thrombosis we decided to continue the anticoagulation indefinitely.

Bili et al. coworkers (9) represented the largest study to date evaluating the association between aCL and a β 2GPI antibodies and recurrent cardiac events in patients after myocardial infarction. Factor V Leiden is a risk factor for MI in young people, because of its high prevalence compared with other genetic mutations relevant to thrombosis, the effect of factor V Leiden in populations of other age and sex, in association with other risk factors (10). These data also revealed an inverse association of IgM aCL antibody levels with recurrent cardiac events (9). Thus, patients with elevated IgG aCL and low IgM aCL antibodies had the highest risk compared with all other aCL antibody groups.

Conclusion

We conclude that young patients with spontaneous venous thrombosis should be meticulously investigated for the presence of both hereditary and acquired thrombotic risk factors. This situation suggested these patients should be evaluated by physicians for cardiovascular events.

References

1. Nicolaes GA, Dahlback B. Activated protein C resistance (Factor V Leiden) and thrombosis: factor V mutations causing hypercoagulable states. *Hematol Oncol Clin North Am* 2003;17: 37-61.
2. Price DT, Ridker PM. Factor V Leiden mutation and the risk for thromboembolic disease: a clinical perspective. *Ann Intern Med* 1997; 127: 895-903.
3. Lensen RP, Rosendaal FR, Koster T, Allaart CF, de Ronde H, Bertina RM. Apparent different thrombotic tendency in patients with factor V Leiden and protein C deficiency due to selection of patients. *Blood* 1996; 88: 4205-8.
4. Koeleman BPC, Reitsma PH, Allaart CF, Bertina RM. Activated protein C resistance as an additional risk factor for thrombosis in protein C-deficient families. *Blood* 1994; 84: 1031-5.
5. Koeleman BPC, van Rumpft D, Hamulyak K, Reitsma PH, Bertina RM. Factor V Leiden: an additional risk factor for thrombosis in protein S deficient families? *Thromb Haemost* 1995; 74: 580-3.
6. Lensen RPM, Bertina RM, de Ronde H, Vandenbroucke JP, Rosendaal FR. Venous thrombotic risk in family members of unselected individuals with factor V Leiden. *Thromb Haemost* 2000; 83: 817-21.
7. Lopez FF, Sweeney JD, Blair AJ, Sikov WM. Spontaneous venous thrombosis in a young patient with combined factor V Leiden and lupus anticoagulant. *Am J Hematol* 1999; 62: 58-60.
8. Wahl DG, Guillemin F, de Maistre E, Perret-Guillaume C, Lecompte T, Thibaut G. Meta-analysis of the risk of venous thrombosis in individuals with antiphospholipid antibodies without underlying autoimmune disease or previous thrombosis. *Lupus* 1998; 7: 15-22.
9. Bili A, Moss AJ, Francis CW, Zareba W, Watelet LF, Sanz I. Anticardiolipin antibodies and recurrent coronary events: a prospective study of 1150 patients. *Thrombotic Factors, and Recurrent Coronary Events Investigators. Circulation* 2000; 102: 1258-63.
10. Rosendaal FR, Siscovick DS, Schwartz SM, Beverly RK, Psaty BM. Factor V Leiden (resistance to activated protein C) increases the risk of myocardial infarction in young women. *Blood* 1997; 89: 2817-21.

Early postoperative left atrial thrombosis in a biatrial orthotopic heart transplant recipient successfully treated by intravenous heparin

Biatrilyal ortotopik kalp nakil alıcısında intravenöz heparin ile başarılı tedavi edilmiş erken postoperatif sol atriyal trombus

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Introduction

Thrombus formation and spontaneous echocontrast (SEC) within the left atrium (LA) are harmful intermediate to long term complications of the standard orthotopic heart transplantation (OHT) (1). We present an OHT recipient with extremely mobile multiple LA thrombi diagnosed early after the operation by transthoracic echocardiography (TTE) and treated successfully without thrombectomy.

Case report

A 24-year-old man, with a history of idiopathic dilated cardiomyopathy, underwent OHT by the biatrial anastomosis approach. During the operation, the patient was given 800 mg protamine and 3 units of whole blood. After that, he had no hemorrhagic complication and no need for any other coagulation factor. A large pericardial effusion developed immediately after the operation for which close clinical and TTE follow-up on a daily basis was undertaken while we avoided anticoagulant and antiplatelet agents. On the 10th postoperative day a mobile mass within the LA was noted on TTE. Transesophageal echocardiography (TEE) confirmed the presence of multiple, homogeneous, dense, extremely mobile masses suggestive of thrombi along the LA suture line together with SEC (Fig. 1). Left atrial size was 4.0x7.0 cm. Left ventricular (LV) ejection fraction was 42% and mitral valve was normal. These LA masses were not apparent on the TTE examination the day be-

fore. On the same day, the patient suffered from severe abdominal pain that subsided quickly after vomiting. On physical examination his blood pressure was 140/90mmHg, pulse rate was 88bpm and regular, he had no fever. Abdominal examination was unremarkable. The 12-lead electrocardiogram showed normal sinus rhythm. Abdominal X-ray and computerized tomography revealed no pathology. No clinical evidence of peripheral embolization or neurological deficit was detected. Due to the unstable immediate postoperative course and relatively low LV ejection fraction, we were reluctant for surgical removal of thrombi and decided to put the patient on systemic anticoagulation with intravenous heparin upon detection of LA thrombi. A bolus heparin dose of 5000U followed by 1000U/hour was started as intravenous infusion. Subsequent heparin dose was adjusted by activated clotting time with a target of 200-250ms. LA masses dramatically reduced in size as depicted on serial echocardiographic studies. Control TEE on the 5th day of active heparinization showed a completely clear LA (Fig. 2) without any increase in the pericardial effusion and the patient did not suffer any embolic complication. Meanwhile repeat biopsies did reveal no rejection.

Discussion

Stasis within the atria due to enlarged cavities and prominent sutures, electrical discordance (2), atrial arrhythmias, LV dysfunction, increased platelet aggregation (3), acute rejection (4) and the surgical technique itself (1) are considered as predisposing factors for thrombus formation. The