a muscular narrowing, leading to a tunnel shaped left ventricular outlet. The prevalence of discrete subvalvular membrane in adults has been reported as 6.5% by Oliver et al. (2). Ventricular septal defect (14.9%), aortic coarctation (12.7%) and bicuspid aortic valve (2.2%) may accompany this anomaly (2). A case of SAS associated with Gerbode type defect has also been reported (3). The clinical course of SAS is generally progressive. A CW Doppler derived peak instantaneous gradient of more than 50 mm Hg is considered severe and subsequent aortic valve damage and aortic regurgitation may develop in time (4). Suspicion of discrete subaortic stenosis arises either when the membrane is seen directly as an echodense structure, or when increased transaortic gradients are detected with morphologically normal aortic valves. However, in some patients only the associated heart diseases may be diagnosed and treated during childhood whereas SAS may remain undiagnosed till adulthood.

Valvar, supravalvar or subvalvar pathologies should be investigated in case of presence of high LVOT gradients. In differential diagnosis of subaortic obstruction, discrete subvalvar membranes, tunnel-like obstruction, hypertrophic cardiomyopathy, accessory mitral valve, anomalous chordal or papillary muscle insertion into the septum, accessory endocardial cushion tissue should be considered. If there are more than one possible obstructive lesions in the LVOT, as in our case, it may be impossible which one is the dominant lesion that causes the increased velocities. In this situation, calculation of the areas of the anatomical structures may be required which can be done with 3-D echocardiographic imaging (5, 6). In addition, investigation of the orientation of the accessory tissues and relation of these structures with surrounding cardiac structures is important in differential diagnosis of rare congenital abnormalities, such as in our case. To our opinion, the membrane defined in this report is unique because it is attached to the posterior aspect of the LVOT and also to the posterior LV wall. It does not extend anteriorly to the anterior mitral valve, thus does not cause a gradient on the LVOT. In addition, it has no anatomical relation with the mitral valve which rules out the diagnosis of accessory mitral valve.

Conclusion

In this report, we defined a unique type of subaortic membrane accompanied by BAV. Real time 3-D TEE was used to identify the anatomical properties and hemodynamic consequences of the membrane. The areas of the membrane, LVOT and aortic valve were successfully measured using 3-D images and the primary pathology was shown to be confined to the aortic valve which was replaced by a mechanical prosthetic valve.

Video 1. Two-dimensional TEE image in long-axis showing the membrane originating from the interventricular septum extending laterally

Video 2. Two-dimensional TEE image showing the membrane beneath the mitral valve as a thick chord extending laterally

Video 3. Video of the cropped full volume RT 3-D TEE dataset showing relation of the left atrium (LA), left ventricle (LV) and aorta (AO) to the membrane (Asterisk, tips of the yellow arrows point to septal and lateral sides of the membrane

References

1. Bhaya M, Mutluer FO, Mahan E, Mahan L, Hsiung MC, Yin WH, et al. Live/Real time three-dimensional transesophageal echocardiography in percutaneous closure of atrial septal defects. Echocardiography 2013; 30: 345-53. [CrossRef]
2. Oliver JM, Gonzalez A, Gallego P, Sanchez-Recalde A, Benito F, Mesa JM. Discrete subaortic stenosis in adults: increased prevalence and slow rate of progression of the obstruction and aortic regurgitation. J Am Coll Cardiol 2001; 38: 835-42. [CrossRef]
3. Akpınar I, Karabağ T, Sayın MR, Kalkan ME. An unusual defect and a rare combination: Gerbode defect and subaortic membrane. Anadolu Kardiyol Derg 2013; 13: E7-8.
4. Gersony WM. Natural history of discrete subvalvar aortic stenosis: management implications. J Am Coll Cardiol 2001; 8: 843-5. [CrossRef]
5. Marechaux S, Juthier F, Banfi C, Vincentelli A, Prat A, Ennezat PV. Illustration of the echocardiographic diagnosis of subaortic membrane stenosis in adults: surgical and live three-dimensional transoesophageal findings. Eur J Echocardiogr 2011; 12: E2. [CrossRef]
6. Bandarupalli N, Faulkner M, Nanda NC, Pothineni KR. Erroneous diagnosis of significant obstruction by Doppler in a patient with discrete subaortic membrane: correct diagnosis by 3D-transthoracic echocardiography. Echocardiography 2008; 25: 1004-6. [CrossRef]

Address for Correspondence: Dr. Barış Güngör,
Mehmet Akif Mah. Hicret Sok. Bulvar Sit.
A Blok D 3 Ümraniye, İstanbul-*Türkiye*
Phone: +90 216 545 86 95
Fax: +90 216 459 27 66
E-mail: drbarisgungor@gmail.com

Available Online Date: 06.05.2014

DOI:10.5152/akd.2014.5256

Successful treatment of a pulmonary embolism with low dose prolonged infusion of tissue typed plasminogen activator in a 37 year old female in early postoperative period

Ahmet Çağrı Aykan, Faruk Boyacı, Engin Hatem
Department of Cardiology, Ahi Evren Chest and Cardiovascular Surgery Education and Research Hospital; Trabzon-*Turkey*

Introduction

Deep vein thrombosis is life threatening disease which may cause pulmonary embolism (PE) (1). Thrombolytic therapy (TT) is indicated in patients with massive PE (1). Herein we reported a case of massive PE, associated with proximal deep vein thrombosis extending into the right atrium, in patient with recent major internal bleeding and hysterectomy operation that was successfully treated with low dose prolonged infusion of tissue type plasminogen activator.

Case Report

A 37-year-old woman was admitted to our clinic with dyspnea and chest pain. She had hysterectomy due to uterus rupture 6 days ago. On physical examination she was pale, afibrile and dyspnea. Her blood pressure was 85/45 mm Hg, heart rate was 112 beats/min and oxygen saturation was 86% at room air. She had mild respiratory difficulty. Her jugular veins were distended and lung fields were clear.

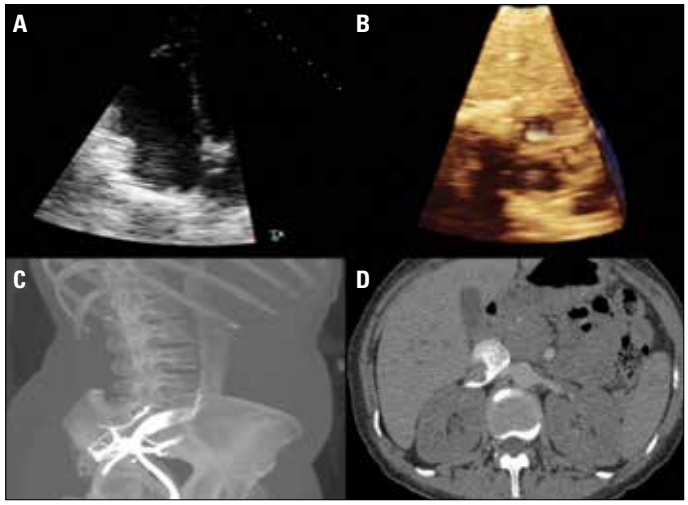

Figure 1. (A) The thrombus in the right atrium was evident in apical four chamber view. (B) The thrombus extending from inferior vena cava to right atrium was demonstrated in 3-dimensional transthoracic echocardiography. (C, D) A thrombus in the iliac veins extending to inferior vena cava was shown in computed tomography venography

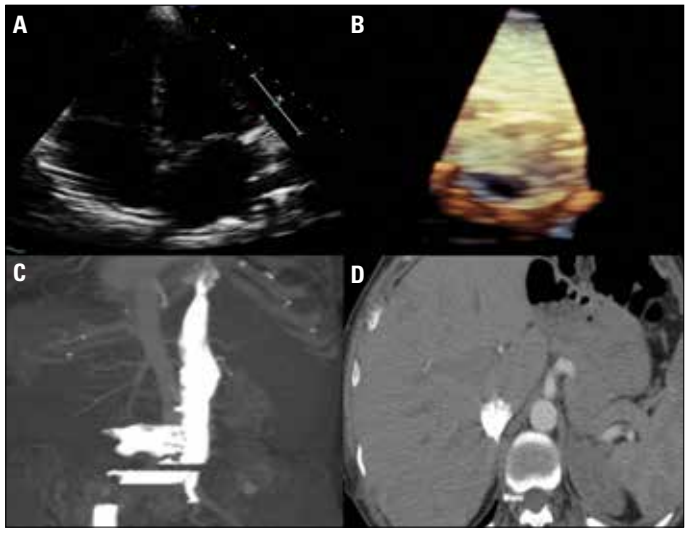

Figure 2. (A, B) Successful lyses of thrombus in right atrium was shown in transthoracic echocardiography. (C, D) Successful lyses of thrombus in inferior vena cava and iliac vein was demonstrated in computed tomography venography

Electrocardiography showed sinus tachycardia. Telecardiography was normal. Arterial blood gas analysis obtained at room air showed a pH of 7.42, partial pressure of carbon dioxide of 34 mm Hg, and a partial oxygen pressure of 80 mm Hg. Her plasma D-dimer level was 7000 ng/mL, Troponin-T was 0.20 mg/dL and NT pro-BNP was 650 mg/dL. A free floating thrombus in the inferior vena cava moving into right atrium was shown in transthoracic echocardiography examination (Fig. 1A, B). The right ventricle was enlarged. Doppler examination revealed moderate tricuspid regurgitation and pulmonary hypertension (pulmonary artery systolic pressure 40 mm Hg). Computed tomography demonstrated a huge thrombus in iliac veins extending to right atrium (Fig. 1C, D) and PE in lobar pulmonary arteries. Supplemental oxygen was started. Considering her medical conditions and for decreasing the possibility of hemorrhage we decided to administer prolonged infusion (in 6 hours) of low dose (25 mg) tissue type plasminogen activator without concomitant use of heparin. At the end of TT thrombus in the right heart was completely lyses; pulmonary artery systolic pressure was decreased to 20 mm Hg and nearly 90% of the thrombus in inferior vena cava and iliac veins lyses (Fig. 2A-D). Heparin bolus was administered and infusion was started after the end of TT. Patient was followed with heparin infusion till achieving effective anticoagulation with warfarin (a target INR level of 2.5). The patient was discharged uneventfully and well on consecutive follow up visits.

Discussion

TT decreases mortality and morbidity in patients with massive PE (1). The approved TT protocol is 100 mg t-PA given during a 2-hour infusion (1). TT is associated with significant complications including major bleeding and mobilization of intracardiac thrombotic mass, with resultant massive and potentially life-threatening events (1). Right heart thromboembolism is a rare but life treating condition but the optimal management of right heart thromboembolism is controversial (1). Mortality rates associated with no therapy, anticoagulation therapy, surgical embolectomy, and thrombolysis were 100.0%, 28.6%, 23.8%, and 11.3%, respectively in right heart thrombus (2).

TT achieved >50% clotlysis more often than heparin alone in proximal DVT patients with a trend toward reduced post thrombotic syndrome. However, major bleeding was increased significantly with use of systemic thrombolysis (1). The administration of standard regimen of 100 mg tissue type plasminogen activator in 2 hours is relatively contraindicated in patients with recent major surgery or major bleeding (1). But due to huge thrombus burden in iliofemoral vein and inferior vena cava the risk of recurrent PE was high in this patient, too. Moreover TT was indicated due to right heart thromboembolism and massive PE. There is no data in the literature regarding the safety of low dose prolonged infusion of tissue type plasminogen activator in patients with recent major bleeding and surgery. Recently Özkan et al. (3) showed that low dose prolonged infusion of tissue type plasminogen activator was effective and safe in the treatment of prosthetic valve thrombosis (4). Especially the low dose prolonged infusion of tissue type plasminogen activator significantly decreased the major complications including major bleeding and stroke compared to full dosed rapid protocols (3). Yıldız et al. (5) reported that low dose prolonged infusion of tissue type plasminogen activator may be an effective alternative in very elderly patients with PE. Considering her clinical situation and for decreasing complications such as bleeding, we decided to administer a prolonged infusion of low-dose tissue plasminogen activator. The thrombus in right heart chamber was resolved completely and >90% of thrombus was resolved in the inferior vena cava and iliofemoral veins.

Conclusion

In conclusion, low dose prolonged infusion of tissue type plasminogen activator may be safe and effective treatment alternative in patients with massive PE and DVT having high bleeding risk with conventional TT.

References

1. Jaff MR, McMurtry MS, Archer SL, Cushman M, Goldenberg N, Goldhaber SZ, et al. American Heart Association Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation; American Heart Association Council on Peripheral Vascular Disease; American Heart Association Council on

Arteriosclerosis, Thrombosis and Vascular Biology. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: a scientific statement from the American Heart Association. Circulation 2011; 123: 1788-830. [CrossRef]
2. Rose PS, Punjabi NM, Pearse DB. Treatment of right heart thromboemboli. Chest 2002; 121: 806-14. [CrossRef]
3. Özkan M, Gündüz S, Biteker M, Astarcıoğlu MA, Çevik C, Kaynak E, et al. Comparison of different TEE-guided thrombolytic regimens for prosthetic valve thrombosis: The TROIA Trial. JACC Cardiovasc Imaging 2013; 6: 206-16. [CrossRef]
4. Özkan M, Çakal B, Karakoyun S, Gürsoy OM, Çevik C, Kalçık M, et al. Thrombolytic therapy for the treatment of prosthetic heart valve thrombosis in pregnancy with low-dose, slow infusion of tissue-type plasminogen activator. Circulation 2013; 128: 532-40. [CrossRef]
5. Yıldız M, Karakoyun S, Acar RD, Özkan M. Effectiveness of low-dose prolonged infusion of tissue plasminogen activator in a nonagenarian patient with acute pulmonary embolism and main pulm onary artery thrombus. Blood Coagul Fibrinolysis 2013; 24: 95-6. [CrossRef]

Address for Correspondence: Dr. Ahmet Çağrı Aykan,
Ahi Evren Göğüs ve Kalp Damar Cerrahisi
Eğitim ve Araştırma Hastanesi,
Soğuksu Mah., Çamlık Cad.
61040 Trabzon-*Türkiye*
Phone: +90 505 868 94 61
Fax: +90 462 231 04 83
E-mail: ahmetaykan@yahoo.com
Available Online Date: 06.05.2014

DOI:10.5152/akd.2014.5273

Explantation of an atrial septal occluder device in a patient with nickel hypersensitivity

Emrah Uğuz, Kemal Erdoğan, Erol Şener
Clinic of Cardiovascular Surgery, Atatürk Training and Research Hospital; Ankara-*Turkey*

Introduction

Percutaneous closure of cardiac defects has become increasingly popular among cardiologists. Nitinol containing devices for transcatheter closure of atrial septal defects (ASD) have been used worldwide over the past decade (1, 2). These nitinol devices not only provided excellent results, but also made for safe and easy device implantation (1, 2). Meanwhile, there are certain contraindications and limitations to this relatively popular technique that should be acknowledged. Here, we report a case of nickel hypersensitivity after an ASD device closure requiring device explantation.

Case Report

A 26-year-old woman with a known history of percutaneous closure of ASD, presented with headache, shortness of breath and retrosternal pain and chest compression and her discomfort was exacerbating with inspiration. The secundum ASD had been closed using an Amplatzer Septal Occluder device (AGA Medical, Golden Valley, MN) in another institution a year ago. Within days after deployment, the patient developed symptoms. She also experienced episodes of shortness of breath and palpitations, usually lasting a few minutes. Symptoms progressed in severity and became constant after weeks. During her evaluation, no shunting was documented. She reported a severe metal allergy since childhood, to an extent that wearing any metal jewelry resulted in severe contact dermatitis. Reaction to the device was presumed to be the primary cause of her symptoms after an extensive work up. Since, patch testing is currently the gold standard for evaluating patients with nickel allergy, consultant Dermatology physician recommended to proceed with patch testing. Skin patch testing demonstrated hypersensitivity for nickel. Her symptoms continue to worsen and resulted in multiple hospital admissions. A course of prednisone and clopidogrel was attempted. Her symptoms persisted, requiring visits for control. In this follow up process, the patient was also evaluated at the institution where the device was implanted and they recommended surgical explanation of the device with the diagnosis of nickel hypersensitivity. She subsequently underwent uncomplicated device removal a year after her transcatheter ASD closure (Fig. 1-3) in our institution. Surgery was performed through a standard median sternotomy approach. After removal of the device, the defect in the atrial septum (2.5x2.0 cm) was closed with an autologous pericardial patch. We used polydioxanone sutures for sternal closure after the procedure in order to avoid steel wires. Postoperatively she experienced dramatic improvement of her symptoms. She remains symptom free now at 3 months after her operation.

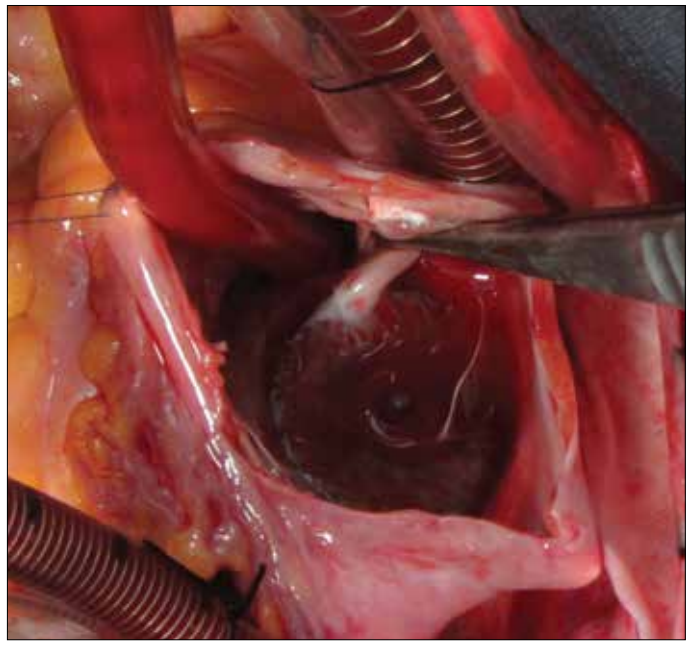

Figure 1. Amplatzer ASD occluder, right atrial surgical view

Discussion

The amplatzer ASD occluder device consists of nitinol which is a metallic alloy composed of 55% nickel and 45% titanium, giving it superior elasticity and shape memory (3). Since 8.6% of the population demonstrates skin sensitivity to nickel (4), the issue of biocompatibility of nitinol implants remains controversial. Patch testing is currently the gold standard for evaluating patients with nickel allergy (4).

Although device closure of an ASD has been reported to be safe, it has been associated with serious complications that required surgical