Management of acute ischemic stroke occurred during thrombolytic treatment of a patient with prosthetic mitral valve thrombosis: Continuing thrombolysis on top of thrombolysis

Protez mitral kapak trombozu olan bir hastada trombolitik tedavisi sırasında gelişen iskemik inmeye yaklaşım: Trombolizisin üstüne devam edilen trombolizis

Introduction

Prosthetic heart valve thrombosis (PHVT) is one of the major causes of primary valve failure. Even with the use of warfarin, risk of thromboembolism is 1-2% per year, but the risk is considerably higher without or inadequate treatment with warfarin (1). Although surgery is the first-line treatment modality in symptomatic PHVT (2), thrombolytic therapy has recently evolved as an effective substitute to surgery (3-6). Cerebral thromboembolism associated with thrombolytic therapy of left-sided PHVT seems to be the main limitation. Acute ischemic stroke may be managed with thrombolysis in selected cases (7, 8).

Case Report

A 25-year-old woman with a history of mitral valve replacement 2 years ago was admitted to our hospital with severe dyspnea. Physical examination revealed blood pressure 80/50 mmHg, a regular apical pulse of 130 beats/min and suggested probable thrombosis of the valve prosthesis, with a muffled first prosthetic valve click. She was inadequately anticoagulated for the last 2 months. International Normalized Ratio was 1.4. Transthoracic echocardiogram (TTE) revealed high transmitral gradients (maximum: 25 mmHg, mean: 19 mmHg) with a valve area of 0.9 cm^2. Two dimensional (2D TEE) and real time 3 dimensional transesophageal (RT-3D TEE) echocardiographic examination revealed a large thrombus, 2 cm^2 in area, that impaired one of the occluder movements (Fig. 1A, 1B and Video 1, 2. See corresponding video/movie images at www.anakarder.com). Thrombolytic therapy-low-dose and prolonged infusion of tissue-type plasminogen activator (tPA) (25 mg in six hours)- was initiated as previously described (5). At the end of 2 hours of thrombolysis (8 mg), the patient experienced dysarthria with power being 2/5 in right sided limbs. Transthoracic echocardiography revealed significantly decreased transvalvular gradients (maximum: 9 mmHg, mean: 6 mmHg) with an increased valve area of 2.8 cm^2. 2D TEE and RT-3D TEE demonstrated normally functioning mitral valve, without evidence of thrombus (Fig. 1C and 1D). Urgent 64-slice cranial computerized tomography (CT) scan was definitely normal and excluded intracranial hemorrhage, and thrombolysis was continued with 12 mg tPA for 1 hour (totally 20 mg) for the management of stroke. Informed consent was obtained. Four hours after the stroke, the patient regained the ability to speak with power being 4/5 in right sided limbs. A week later, she had slight weakness of right sided limbs and was discharged with recommendation of physiotherapy

Discussion

PHVT is a life-threatening complication. Although surgery is recommended by the recent guidelines (2) for patients in New York Heart Association class III-IV unless surgery is high risk, it has high mortality up to 64% (9). Thrombolysis is an effective therapy for PHVT. There is no consensus concerning the therapeutic agents and dose of the thrombolysis. We hypothesized that successive, low dose (25 mg) and slow infusion (6-hours) of tPA would induce thrombolysis and limit the risk of hemorrhage and embolization. These strategy have provided safer thrombolysis in patients with PHVT as described previously (5). The most feared complication is the risk of cerebral embolism that can be up to 5-6% for left sided PHVT (6). Acute ischemic stroke may be managed with intravenous thrombolysis, combination of thrombolytics with other antiaggregants like glycoprotein IIb/IIa inhibitors, intraarterial thrombolysis and other catheter based approaches such as mechanical thrombectomy (7, 10). CT is mandatory to exclude intracranial hemorrhage for administration of the thrombolytic agents (7). In this case, the cerebral complication was a major one; the absence of the thrombus that was demonstrated previously on the mitral valve suggested it was highly likely of an ischemic origin. Regarding normal CT findings, consulting with a neurologist and radiologist, a decision was made in favor of carrying on IV thrombolysis which resulted in striking neurological improvement. The recommended tPA dose for acute ischemic stroke regarding to current guidelines is 0.9 mg/kg (maximum dose 90 mg) over 60 minutes with 10% of the dose given as a bolus over 1 minute (7) with the caveat that IV tPA has been associated with lower recanalization rates (10). Although the protocol we have chosen was lower than recommended doses, the success may be due to the early diagnosis and treatment of the thromboembolic complication that occurred during hospitalization. Besides this, the patient was young and this might have predisposed her to a good outcome.

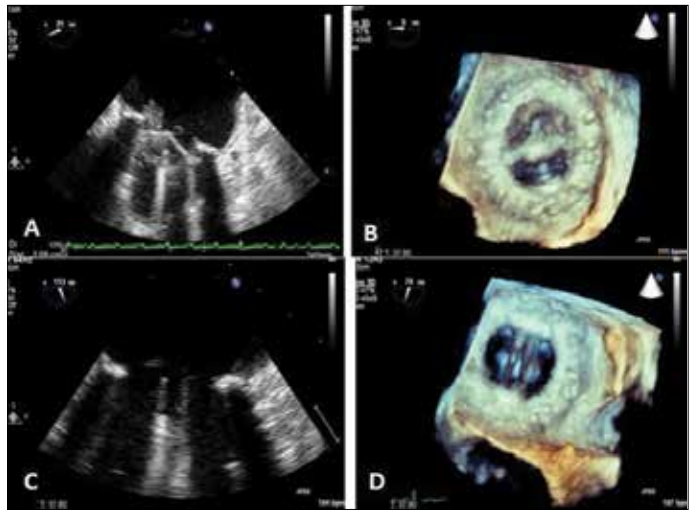

Figure 1. Obstructive thrombosis impairing occluder movement shown by two dimensional transesophageal echocardiography (A) and real time transesophageal echocardiography (B). After thrombolysis, 2D-TEE (C) and RT-3D TEE (D) demonstrated normally functioning mitral valve, without evidence of thrombus

RT - real time, TEE - transesophageal echocardiography

Conclusion

We present a unique case of mitral PHVT, performing a low dose and prolonged infusion of thrombolytic therapy, and continuing the therapy for the coincident acute cerebral ischemic complication, which was resolved with almost complete success. Thrombolysis in acute ischemic stroke is an effective therapy in selected cases.

Acknowledgments: We gratefully acknowledge the contributions of Mustafa Yıldız MD, PhD and Ahmet Ç. Aykan MD.

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Video 1, 2. Obstructive thrombosis impairing occluder movement shown by two dimensional transesophageal echocardiography (1) and real time transesophageal echocardiography (2)

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Available Online Date/Çevrimiçi Yayın Tarihi: 18.09.2012

doi:10.5152/akd.2012.222

Starr-Edwards caged ball valve pursuing to astonish us-38 years in mitral position

Starr-Edwards kafesli top kapak bizi şaşırtmaya devam ediyor-mitral pozisyonda 38 yıl

Introduction

Until invention of a caged ball valve prosthesis, rheumatic cardiac valve failure had been a deadly disease with death rate of 12/100.000 in 1958. The first time successful Starr-Edwards caged ball valve implantation performed in 25 August 1960 is accepted as a milestone for cardiac valve surgery (1). Several centers published their experiences with this valve in both aortic and mitral positions. Encountered complications with caged ball valve such as; systemic embolisation, ball variance, high pressure gradient, growth of pannus and chronic hemolysis; lead to new valve designs (2-5). Nowadays these investigations still goes on for perfect valve.

Despite its' old fashion design in some cases impressive durability of Starr-Edwards caged ball valves astonishes investigators and this case is one of them.

Case Report

A 58-year-old male patient with lower extremity edema and dyspnea during minimal exercise admitted to the hospital. 38 years ago the patient had mitral valve replacement with Starr-Edwards Caged Ball prosthesis for severe mitral valve regurgitation. According to New York Heart Association classification, the patient was in class 3. Echocardiography documented severe tricuspid valve regurgitation with right atrial (9.7 cm) and right ventricular (5.4 cm) enlargement. Pulmonary artery peak pressure was 40 mmHg. Left atrium diameter was 8 cm. Left ventricular diastolic diameter was 5.2 cm and systolic function was normal with a 60% ejection fraction rate. Peak and mean gradients were 13 mmHg, 7 mmHg respectively over the mitral valve caged ball prosthesis. Hemolysis or anemia were not observed in laboratory tests. Diuretic medications were prescribed and because of symptoms did not relieve drug doses were progressively increased. Despite intensive medical therapy, echocardiographic and clinical right cardiac symptoms did not improve patient underwent tricuspid ring annuloplasty (Carpentier Edwards) and mitral valve re-replacement (St. Jude mechanical valve) operation. Operation was achieved through median sternotomy with mild hypothermic cardiopulmonary bypass. Operative and postoperative courses were uneventful. Control echocardiography before discharge revealed normal functioning valves. Caged ball mitral valve prosthesis inspected as macroscopically at the end of the operation. Although gradients were reported in preoperative transthoracic echocardiography, there were neither growth of pannus and structural integrity loss nor lipid infiltration over the valve (Fig. 1).

Discussion

Caged ball valve design was inspired from a wine bottle stopper, which was invented in 1858. Harken-Sorroff, Starr-Edwards, Magovern-Cromie fabricated and implanted caged ball valves in 1960. Only Starr-Edwards valve was designed for mitral position and others were designed for aortic position. Until appearance of tilting disc valve,