Successful treatment of a huge thrombus with thrombolytic therapy in a patient with normal left ventricle function and Takayasu arteritis

Takayasu arterit ve normal sol ventrikül fonksiyonu olan hastada dev trombüsün trombolitik ile başarılı şekilde tedavisi

Taner Şeker, M.D., Ahmet Oytun Baykan, M.D., Abdurrezzak Börekçi, M.D., Mustafa Gür, M.D., Murat Çaylı, M.D.

Department of Cardiology, Adana Numune Training and Research Hospital, Adana, Turkey

Department of Cardiology, Kafkas University Faculty of Medicine, Kars, Turkey

Summary-- We report a case of thrombus treated with thrombolytic therapy in a patient with normal cardiac functions and Takayasu arteritis. A 27-year-old man with a history of Takayasu arteritis was admitted to our out-patient clinic with a complaint of both right and left foot pain and weakness. In a Doppler ultrasound examination, a subtotal thrombotic occlusion was found in the bilateral popliteal arteries. A transthoracic echocardiography revealed a left ventricular apical thrombus, although both cardiac functions and heart dimensions were normal. Surgical excision was recommended to avoid further embolization, but this was refused by the patient. We decided to perform thrombolytic therapy. The thrombus in the left ventricle was fully resolved by the third day. Perfusion in both feet also returned to normal.

CASE REPORT

A 27-year-old male with a history of Takayasu arteritis was admitted with worsening foot pain of one hour duration. History showed once-daily use of Azathioprine 50 mg BID and prednisolone 4 mg. Initial blood pressure in right arm was 120/70 mmHg and 110/60 mmHg in left arm. Physical examination was normal except for bilateral lower extremity, which was pale and pulseless, and left upper extremity pulse was weak compared to the other. The ECG was normal. Lower extremity Doppler ultrasound (USG) examination revealed subtotal popliteal occlusion by thrombus and collaterals, as well as weak arterial flow in left and right foot. Transthoracic echocardiography revealed a mobile heterogeneous mass (38x16 mm) attached to the left ventricle apical wall with a short stalk (Figure 1). Examination of his subclavian MR angiography reported compatible with Takayasu arteritis (Figure 2a). The cerebral MR was normal. A complete blood
count, prothrombin time, activated partial thromboplastin time, liver enzymes, creatinine, c-reactive protein, ESR (erythrocyte sedimentation rate), Loups anticoagulant, and ANA (antinuclear antibodies) were unremarkable. Protein S activity 114% (normal 60%-130%) and protein C activity 96% (normal 70%-140%) and were within normal range. Also, fibrinogen and homocysteine levels were normal. Since, there was a simultaneous thrombi formation in both popliteal arteries, the mass in the apex of the left ventricle was accepted as thrombus. An urgent surgical thrombectomy for ventricle thrombus and popliteal thrombus was recommended in consideration of the known potential for coronary, cerebral, and other systemic embolism. However, the patient refused surgical treatment, and therefore thrombolytic therapy was considered, and a total dose of 1.5 millions unit streptokinase infusion was administered to the patient. 250.000 IU of streptokinase was applied as bolus infusion over 30 minutes, followed by a continuous intravenous infusion of streptokinase 100.000 IU/h over 12 hours. Minimal gum bleeding occurred during the infusion of streptokinase. Following the streptokinase infusion, we started subcutaneous low molecular weight heparin (Enoxaparin, 6000 IU, BID) with oral administration of warfarin, maintaining an international normalized ratio (INR) of 2.0-3.0. One day

![Figure 1. Initial 2-D transthoracic (A, B) and 3-D transthoracic (C) echocardiograms show pedunculated thrombi attached to LV apical wall. M-mode parasternal long- axis (D) shows normal LV function and dimensions. LV: Left ventricle.](image)

![Figure 2. Contrast-enhanced magnetic resonance angiography revealing the area of subclavian stenosis.](image)
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After the streptokinase infusion, thrombus size was significantly reduced (Figure 3). Also, feet perfusion and temperature became normal. On the third day, an echo exam showed that the thrombus was fully disappeared (Figure 4).

DISCUSSION

Left ventricular thrombus usually occurs in the presence of impaired LV function, such as in dilated cardiomyopathy, LV aneurysms or following a myocardial infarction.[2] Thrombi in the left ventricle must be treated to prevent life-threatening coronary, cerebral and systemic emboli. Although left ventricle thrombus occurs in the presence of impaired left ventricle function, there are some case reports of ventricular thrombus in patients with preserved ejection fraction.[3] Left ventricular thrombi can form in normal ventricles due to hyper-aggregable platelets, hypercoagulation, malignancy, idiopathic causes, pheochromocytoma or rheumatic diseases and can be detected by echocardiography.[4,5] It is well known that vasculitis is associated with hypercoagulability and thrombus formation.[6] In the present case report, vasculitis and endothelial dysfunction are supposed to have caused thrombosis, but there is no clear data on the role of hemostatic mechanisms in the development of thrombosis for our patient. Zöller B. et al showed disequilibrium between inducers and inhibitors of coagulation, and fibrinolysis pathways due to pro-inflammatory mediators have been suggested as contributors to hypercoagulability in these syndromes.[7] Arterial thrombus formation is common in patients with Takayasu arteritis and include ischemic stroke, transient ischemic attacks and acute coronary syndromes.[8,9] Although atherosclerosis and arterial anatomic abnormalities are the major determinants of increased risk of thrombotic events in Takayasu arteritis, a hypercoagulable state has been demonstrated, which is mainly attributed to enhanced platelet and coagulation activities, as well as complex interactions between inflammation and hemostasis involving pro-inflammatory cytokines, chemokines, adhesion molecules, tissue factor expression, platelet and endothelial activation, and micro particles.[10,11] Moreover, inflammation may lead to endothelial damage, causing the loss of intrinsic anticoagulant, antiag-
grettant and vasodilatory properties of endothelium. Thrombi in ventricle related to Takayasu arteritis are mostly accompanied by left ventricle systolic dysfunction. To the best of our knowledge, this is the first report about left ventricular thrombus in a patient with preserved ejection fraction due to Takayasu arteritis. In the literature, ventricular thrombi cases are usually treated with surgery or oral anticoagulation. Koç et al reported a case in which thrombus in the left ventricle due to peripartum cardiomyopathy was successfully treated with thrombolytic therapy. Thrombolytic treatment may be an option for some ventricular thrombus that causes systemic embolism.

**Conclusion**

We treated a rare case of left ventricular thrombus in a patient with a structurally and functionally normal heart, who presented with peripheral thromboembolism due to Takayasu arteritis. In the present report, our patient was admitted to hospital in one hour, presented with systemic embolization caused by intra-cardiac thrombus and successfully treated with thrombolytic therapy.

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**REFERENCES**


**Key words:** Takayasu arteritis/therapy; thrombosis; thrombolytic therapy; ventricle, left.

**Anahtar sözcükler:** Takayasu arterit/tedavi; trombüs; trombolitik tedavisi; ventrikül, sol.