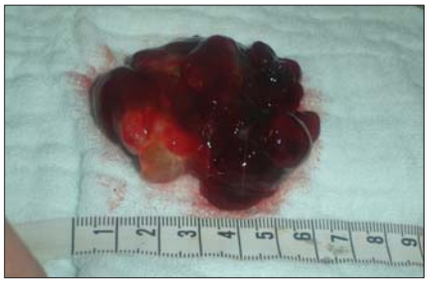

**Figure 4. Excised myxoma**

marginal branch of circumflex artery) were performed. Tricuspid valve was examined with saline test and severe insufficiency was seen, therefore, De-Vega annuloplasty was performed. The operation was completed without any complications and the patient was discharged on the 5th postoperative day. Pathological diagnosis was myxoma.

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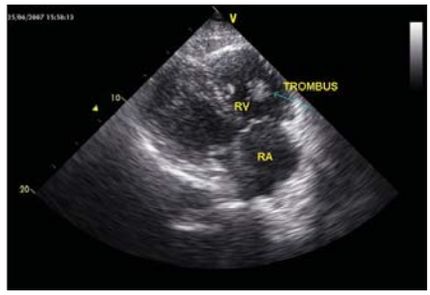
# Ekokardiyografi ile sağ ventrikülde mobil trombüs tespit edilen akut pulmoner tromboemboli olgusu

## *A case of acute pulmonary thromboembolism with a mobile thrombus in right ventricle detected with echocardiography*

Klinik olarak pulmoner tromboemboliden (PTE) şüphelenilen olguların tümünde akciğer perfüzyon sintigrafisi ve/veya pulmoner anjiografinin zamanında uygulanması mümkün olamamaktadır. Transtorasik ekokardiyografi (TTE), kalp boşluklarında trombüs tanısında oldukça önemli bir tanı yöntemidir. Acil serviste yatak başında uygulanabilen TTE yöntemi, PTE'li hastalarda erken tanı olanağı sağlayabilmektedir. Bu raporda, akut PTE kliniği gelişen bir olguda TTE ile sağ ventrikül trombüsünün gösterilmesi ve PTE tanısının doğrulanması anlatılmaktadır.

Asit etyolojisi araştırılmak üzere yatırılan morbid obez ve immobil olan 75 yaşında bayan hastada yatışının 3. gününde ani nefes darlığı, sırt ağrısı ve senkop gelişti. Hastanın genel durumu kötü, bilinç bulanık, dispneik ve takipneik idi. Kan basıncı 140/100 mmHg, nabız 100/dakika ritmik, vücut ısısı 37,4 ºC, arter kan gazı hipoksik ($PO_2$: 56,6), hipokapneik ($PCO_2$: 33) olup D-dimer 3414 mg/L olarak saptandı. Akut PTE ön tanısıyla yapılan TTE'de sağ ventrikül içinde lobüle mobil trombüs ve orta derecede pulmoner hipertansiyon (60 mmHg) tespit edildi. (Resim 1, Video 1. Video/hareketli görüntüler www.anakarder.com'da izlenebilir). Hastaya trombolitik ve antikoagülan tedavi başlandı. Genel durum bozukluğu nedeniyle trombektomi için ameliyata alınamadı. Takibinde solunum sıkıntısı artan hasta entübe edilerek mekanik ventilatöre bağlandı. Ancak akut olayın gelişimini izleyen 24 saat içinde exitus oldu.

Sağ kalp kökenli trombüslerin görülme olasılığı sol kalbe göre çok daha nadirdir ve sıklıkla mikroemboli veya masif PTE'ye yol açabildiklerinden kötü prognozludur. Bu olgularda erken tanı ve acil tedavi çok önemlidir. Bu nedenle, akut PTE şüphesi olan hastalarda, erken dönemde ekokardiyografi yapılarak PTE tanısının doğrulanması, akciğer perfüzyon sintigrafisi ve/veya pulmoner anjiyografi yapılmadan fibrinolitik tedaviye başlanması için zaman kazandırabilir.




**Resim 1. Ekokardiyografide sağ ventrikül kavitesi içinde trombüsün görünümü**

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# Interventricular septal perforation as a rare complication of temporary transvenous pacemaker

## *Geçici transvenöz pacemaker'ın nadir bir komplikasyonu: İnterventriküler septal perforasyon*

Interventricular septum perforation is a rare complication of pacemaker implantation, but it may cause death and may be misdiagnosed.

A 70-year-old woman was admitted to the emergency room of another hospital with complete atrioventricular (AV) block with a ventricular rate of 30 beats/min on electrocardiography (ECG). Ventricular tachycardia occurred during temporary pacemaker implantation via right subclavian vein. The patient referred to our hospital after D/C cardioversion. The ECG on admission showed right bundle branch block (RBBB) pacemaker rhythm with a rate of 68 beats/min. The pacemaker lead was not in normal position on chest X-ray.

The RBBB pattern is a useful marker of the left ventricular stimulation. The left ventricular stimulation after temporary or permanent pacemaker implantation is associated with connections formed by the coronary sinus and its branches, intracardiac defects (sinus venosus type defect, patent foramen ovale, atrial septal defect), perforation of interventricular septum and malposition due to inadvertent subclavian artery puncture. The lead position was assessed with transthoracic echocardiography.

The transthoracic echocardiography showed left ventricular hypertrophy (interventricular septum thickness of 16 mm), relaxation disturbance, pacemaker lead passing from the right atrium to the right ventricle and to the left ventricle at the interventricular plane (Fig. 1, 2). The tip of the lead was detected in the left ventricle and this was confirmed with transesophageal echocardiography (Fig. 3).

Echocardiography is very useful in the assessment of the lead location, diagnosis of the pacemaker complications and follow-up. Interventricular septum perforation should be suspected in the presence of RBBB on ECG after implantation of a temporary or permanent pacemaker.

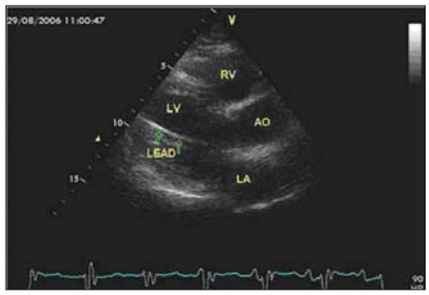


**Figure 1. The parasternal long-axis view of transthoracic echocardiography showing the lead of the temporary pacemaker**
Ao - aorta, LA - left atrium, LV - left ventricle, RV - right ventricle

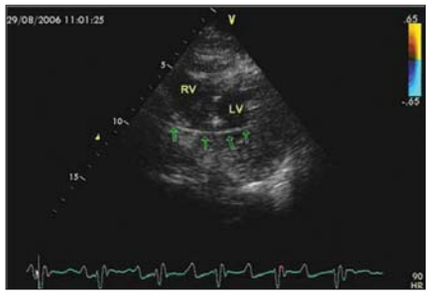


**Figure 2. Parasternal short-axis view of transthoracic echocardiography showing the lead in the left ventricle**
LV - left ventricle, RV - right ventricle

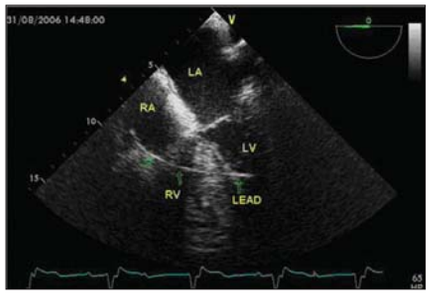


**Figure 3. Transesophageal echocardiography at midesophageal level showing the lead passing through interventricular septum to the left ventricle**
LA - left atrium, LV - left ventricle, RV - right ventricle

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# Catecholaminergic polymorphic ventricular tachycardia caused by a novel mutation in the cardiac ryanodine receptor

## *Kardiyak ryanodin reseptör genindeki yeni bir mutasyon sonucu oluşan katekolaminerjik polimorfik ventriküler taşikardi*

A 21-year-old male presented with a 9-year history of recurrent, exercise-related syncope. His past medical history was remarkable for cerebral palsy due to birth hypoxia. Twelve-lead electrocardiogram (QTc range 395-405 ms) showed frequent premature ventricular contractions with left bundle branch block morphology and inferior axis (Fig. 1). Within 24 hours of admission, the patient developed polymorphic ventricular tachycardia associated with loss of consciousness requiring 4 direct-current cardioversions (Fig. 2). Holter monitoring showed frequent narrow and wide QRS complex tachycardias and possible bidirectional ventricular tachycardia (Fig. 3). Transthoracic echocardiography was completely normal. There was no family history of syncope or sudden death. Genetic analysis demonstrated the presence of a novel, causative, de novo missense mutation in the cardiac ryanodine receptor gene (RyR2: p.L4105F) that was not present in either the parents of the patient or in 100 healthy control individuals; therefore, he was a sporadic carrier. Ventricular arrhythmias were controlled with the combination of beta-blocker (metoprolol 200 mgr/day) and calcium-channel blocker (verapamil 120 mgr/day) therapy, and the patient underwent successful placement of a dual-chamber implantable cardioverter defibrillator.

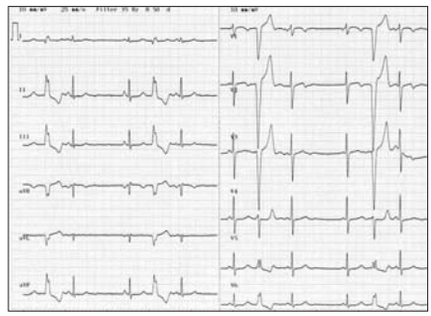

**Figure 1. Baseline 12-lead electrocardiogram**

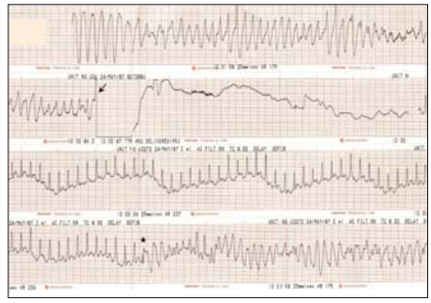

**Figure 2 . Termination of polymorphic ventricular tachycardia with direct current cardioversion (arrow) and degeneration of narrow complex tachycardia to polymorphic ventricular tachycardia (asterisk)**