diffuse ST depression in all other leads (Fig. 1). Troponin I was top normal. Chest X-ray findings were unremarkable. As a result, therapy for acute coronary syndrome was initiated and arrangements were made for emergency cardiac catheterization for suspected acute coronary occlusion. Results from an angiogram, however, failed to reveal coronary artery disease and was normal. After cardiac catheterization, transthoracic echocardiography revealed moderate right ventricular enlargement with mildly reduced function and displacement of the interventricular septum into the left ventricle; however, left ventricular systolic function was normal. In addition, pulmonary artery pressure was elevated moderately but the patient did not demonstrate "McConnell's sign". CT angiography revealed massive bilateral pulmonary embolism.

As stated previously, ST-segment elevation associated with PE is rare, and the direct relationship remains unclear. Anteroseptal or anterior ST-segment elevation was noted in few cases (1-3). Recently it is noted that the presence of STE in lead aVR in patients with APE is associated with poor prognosis. The presence of STE in lead aVR could be an easily obtainable and noninvasive ECG parameter, helpful in risk stratification of patients with APE (3, 4). Most ECG abnormalities associated with PE are thought to be a consequence of a sudden pressure overload on a non-compensatory right ventricle. This additional strain may induce global or focal myocardial ischemia. Therefore, another potential theory suggests that ST elevation in PE results from epicardial or micro-vascular coronary vasospasm induced by such strain. A third theory suggests that severe hypoxemia induces a catecholamine surge, which increases myocardial workload and results in ischemia (1, 3-5). Also the serum potassium level in our patient at the time of the ECG was 5.5 mEq/L, lower than that seen in some cases of ST-segment elevation (1, 5). In addition the severity of metabolic acidosis in our patient (bicarbonate, 20.3 mEq/L) is less than that seen in cases associated with ST elevation attributed to metabolic abnormalities. Indeed the most interesting finding in our case is ST elevation only in aVR lead that may be seen in few cases of acute coronary syndrome.

In conclusion; specific abnormal findings on ECGs may provide clues to the diagnosis and according to previous studies (3, 4) for risk stratification of PE in patients presenting with chest pain, dyspnea, or both; however, the present case illustrates the rare and interesting association of PE with ST-segment elevation in the aVR lead. Therefore, we recommend that physicians consider the presence of PE in patients with chest pain or dyspnea, even when ST-segment elevation is present in aVR lead.

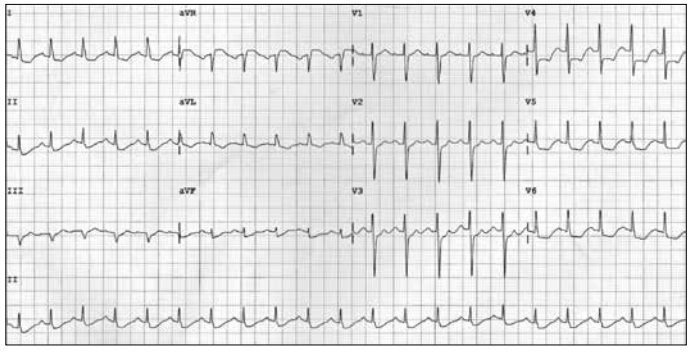

Figure 1. A 12-lead electrocardiogram in patient with acute pulmonary thromboembolism, note the ST segment elevation in aVR lead

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A deadly chain of events in a case; Deep venous thrombosis, pulmoner embolism, patent foramen ovale and cerebral embolism

Bir vakada ölümcül olaylar zinciri; derin ven trombüsü, pulmoner emboli, patent foromen ovale ve serebral emboli

A 67-year-old overweight female with history of hypertension was admitted to our emergency room due to mental confusion, weakness of the right side of the body and shortness of breath starting 12 hours ago. She had stable hemodynamic status with a blood pressure of 140/70 mmHg and heart rate of 106 bpm. Her physical examination was unremarkable with sinus rhythm.

She was diagnosed acute stroke and underwent a cranial magnetic resonance imaging (MRI). Her cranial MRI showed a large infarct in the left middle cerebral artery region (Fig. 1). For the determination of the source of embolus, her echocardiographic examination demonstrated moderate pulmonary hypertension of 65 mmHg accompanying mild right ventricle dilatation with spared apical motion (McConnell sign). The cardiac chambers including main pulmonary artery were clear, and ejection fraction was normal (65%). Transesophageal echocardiography was performed for further evaluation of the interatrial septum. The contrast study demonstrated a large amount of bubble passing through a tunnel like patent foramen ovale (PFO) (<10 mm) (Fig. 2-4. Video 1-3. See corresponding video movie images at www.anakarder.com). All tests were normal except for a slight elevation of D-dimer (500 mg/L). With suspected pulmonary embolism (PE), we performed a tomographic pulmonary angiography, and it revealed a large thrombus partially obstructing the proximal parts of

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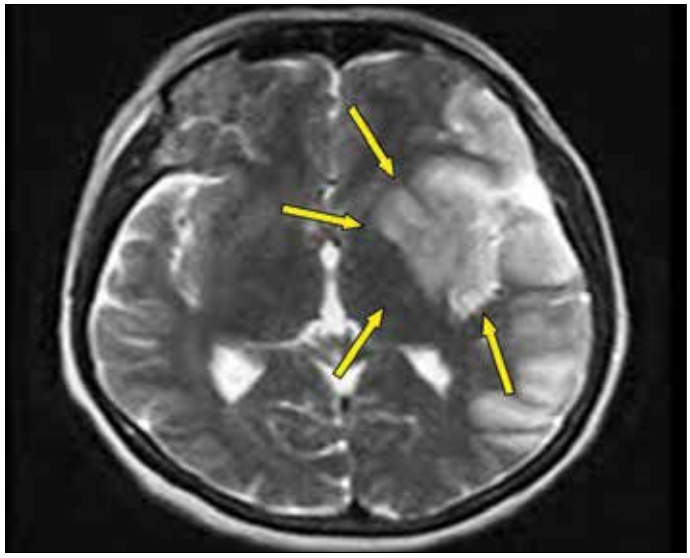

Figure 1. Brain MRI images of acute cerebral infarctions in the left middle cerebral artery territory (arrows)

MRI - magnetic resonance imaging, MCA - middle cerebral artery

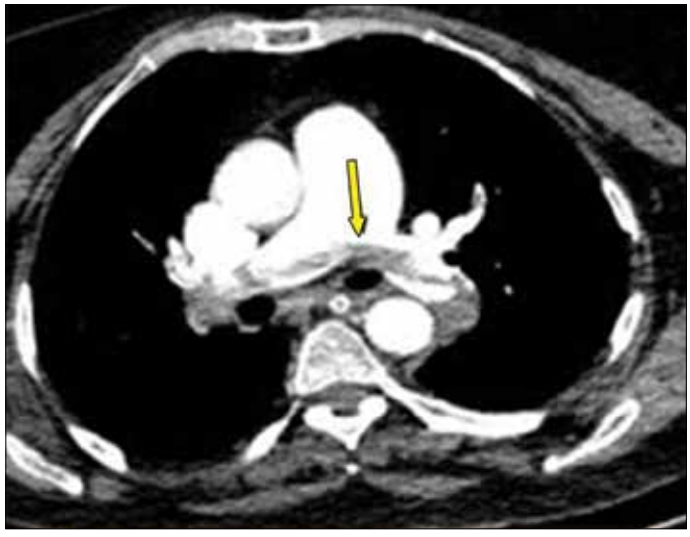

Figure 2. TEE image from 120 degree midesophageal level shows a PFO. (Arrow) After administration of agitated saline both spontaneously and Valsalva maneuver, plenty of bubble passed through right to left atrium (Arrow)

PFO - patent foramen ovale, TEE - transesophageal echocardiography

both pulmonary arteries (Fig. 5). Low-molecular-weight heparin with antiplatelet therapy was started. An extensive thrombus was also detected in the right femoral vein using lower extremity veins with duplex ultrasound. As a result, paradoxical cerebral embolism as well as PE caused by deep venous thrombosis (DVT) and its association with PFO was diagnosed evidently. During her follow-up period, her hemodynamic condition remained stable, and all imaging tests were repeated after 8 days. Her echocardiographic examination showed a decrease in the pulmonary artery pressure to 40 mmHg without any remarkable changes of the right heart size. In addition, a slight reduction of thrombus in the pulmonary arteries was detected by tomographic pulmonary angiography. After a short period of time, her clinical condition deteriorated and on her cranial tomography, severe hemorrhage was observed in previous cerebral infarct region. Unfortunately, the patient was eventually died.

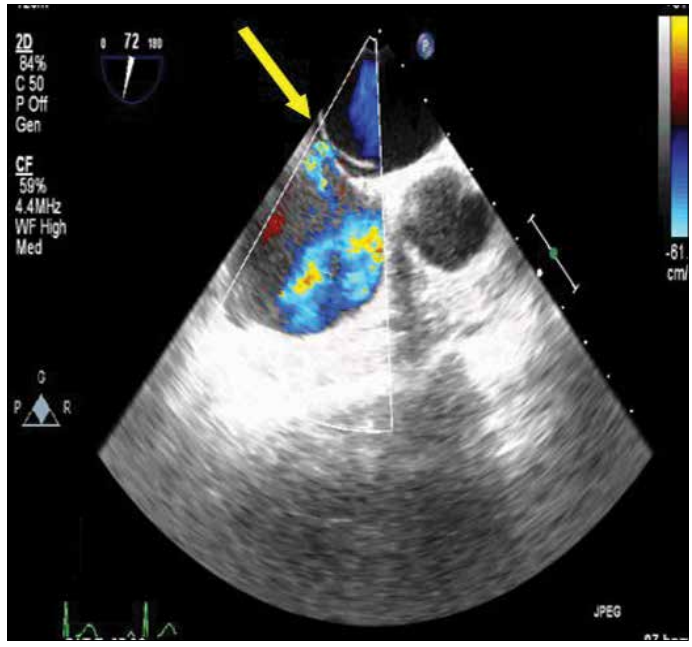

Figure 3. TEE image from 120 degree midesophageal level shows a PFO. (Arrow) After administration of agitated saline both spontaneously and Valsalva maneuver, plenty of bubble passed through right to left atrium (Arrow)

PFO - patent foramen ovale, TEE - transesophageal echocardiography

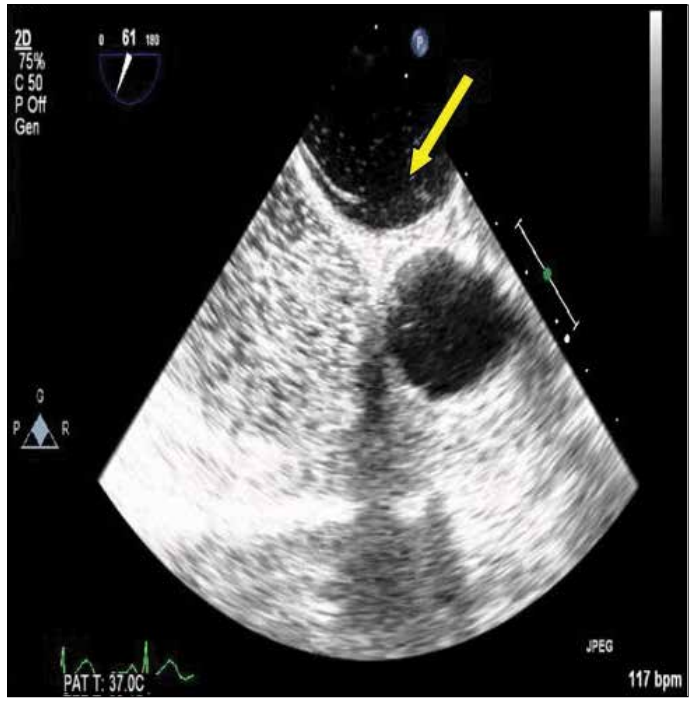

Figure 4. 3D Real time TEE image form 120 degree midesophageal level shows an PFO (Arrow)

PFO - patent foramen ovale, TEE - transesophageal echocardiography

PE is an important clinical entity characterized by still a high mortality rate if not diagnosed early. The most common cause of PE is DVT. The risk level of all patient with PE should be determined to plan an appropriate therapy on the basis of three crucial factors; hemodynamic status, right ventricular dilatation and myocardial injury (1). In case of PE, the right heart pressures will increase, and in the presence of PFO, the passage

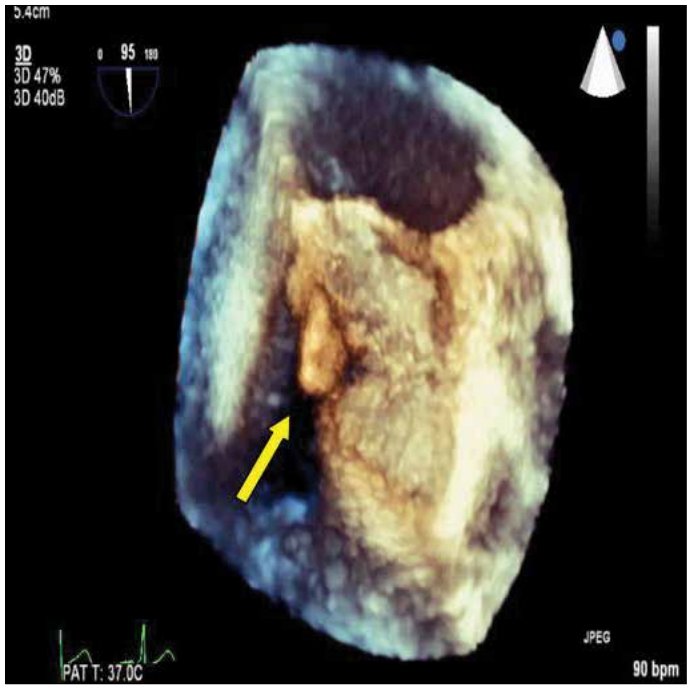

Figure 5. Computed tomographic scan of the chest with contrast agent. (A) Bilateral pulmonary embolism. Arrow point to the pulmonary emboli (Arrow)

become much easier (2, 3). It is always possible that DVT may lead to systemic embolism through the PFO, called as paradoxical embolism (4). If a cerebral embolism exists, the clinical condition may be predominantly neurologic, and PE may be overlooked. In the event of cryptogenic stroke, the existence of PFO and DVT is generally searched, respectively. In these patients, PE should be kept in mind and investigated. The presence of DVT, PE, PFO and systemic embolism in a case together seems to be the most frightening and deadly association, as seen in our case.

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Video 1. TEE color image from 120 degree midesophageal level shows an PFO

PFO - patent foramen ovale, TEE - transesophageal echocardiography

Video 2. TEE color image from 120 degree midesophageal level shows after administration of agitated saline both spontaneously and Valsalva maneuver, plenty of bubble passed through right to left atrium

PFO - patent foramen ovale, TEE - transesophageal echocardiography

Video 3. 3D Real time TEE 120 degree midesophageal level shows an PFO

PFO - patent foramen ovale, TEE - transesophageal echocardiography

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Komplike akut Tip B disseksiyonla başvuran Marfan sendromlu hastada başarılı endovasküler greft-stent tedavisi

Successful endovascular stent-graft treatment in a patient with Marfan syndrome presenting with complicated acute Type B dissection

Yirmi bir yaşında erkek hasta, acil servise ani başlayan sırt ağrısı, her iki bacakta uyuşma ve karın ağrısı yakınması ile başvurdu. Hastanın özgeçmişinde, iki yıl önce Ghent kriterlerine göre Marfan sendromu tanısı alıp, mitral valve prolapsusuna bağlı ileri derecede mitral yetersizlik, asendan aort anevizması ve orta dereceli aort yetersizlik nedeniyle de Benthall ve mitral protez kapak operasyonu hikayesi bulunmaktaydı. Yırtılır tarzda sırt ağrısı olan hastanın her iki femoral nabızının zayıf olduğu saptandı. Bunun üzerine yapılan bilgisayarlı tomografide sol subklavyan arter distalinde desendan aortada intimal yırtık ve diseksiyon flebi görüldü (Şekil 1A, B). Hasta; medikal tedaviye dirençli şiddetli karın ve sırt ağrılarının devam etmesi ve hemodinamik stabilitenin bozulması üzerine endovasküler greft stent uygulaması için kateter laboratuvarına alındı. Sağ koldan yapılan aortografide diseksiyon flebinin (yalancı lümenin) her sistolde kanla dolarak abdominal aortada lümünal daralmaya yol açtığı görüldü (Video 1. Video/hareketli görüntüler www.anakarder.com' da izlenebilir). İntimal yırtık alanı aortografide belirlendikten sonra (Video 2. Video/hareketli görüntüler www.anakarder.com'da izlenebilir) sağ femoral arter yolundan intimal yırtığın proksimalindeki sağlam dokuyu da içine alacak şekilde greft stent implante edildi (Video 3. Video/hareketli görüntüler www.anakarder.com'da izlenebilir). İşlem sonrasında kontrol aortografi ile endoleak olmadığı saptanan hasta, işlemin üçüncü gününde olaysız taburcu edildi (Video 4. Video/hareketli görüntüler www.anakarder.com' da izlenebilir).

Stanford Tip B diseksiyon komplike değilse bugün için kabul gören tedavi seçeneği medikal tedavidir. Ancak, komplike akut tip B diseksiyon (KATBD) hayatı tehdit eden bir klinik tablodur ve acil girişimsel tedavi gerektirir. Bu amaçla, açık cerrahi onarım uzun yıllardır başarı ile uygulanan konvansiyonel metoddur. Son dekatda ise, endovasküler greft stent (EGS) uygulaması da pratikte kullanılmaya başlanmıştır. KATBD'de açık cerrahi ve EGS'nin karşılaştırıldığı bir çalışmada EGS'nin hastane içi erken dönem sonuçlarının daha iyi olduğu ortaya konulmuştur (1). EGS'nin orta dönem sonuçlarının açıklandığı başka bir çalışmada ise; 1. ve 5. yıl sağ kalım oranları sırasıyla %79, %61 saptanmıştır (2). Beş çalışmanın değerlendirmeye alındığı meta-analizde ise; Yazarlar