more decreased than men, requires higher levels of TG than men to stimulate production of CRP, TLR4 mediated, and that TG constitutes a clinical biomarker of impaired antiinflammatory or atheroprotective function in women, while in men it would be CRP, whose release would not depend on TG; in agreement with the findings of Onat et al. (1), about the usefulness of CRP and TG, among others, as clinical biomarkers of impaired or atheroprotective high-density lipoprotein antiinflammatory function for men and women, respectively.

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Author Reply

Sex divergence in rise of serum CRP to pro-inflammatory mediators
Proinflamatuvar mediyatörlerin serum CRP artışında cinsiyet farkı

Dear Author,

Regarding prediction of coronary heart disease, for which we tried to identify clinical biomarkers in the population at large in our recently published study (1), Lozano et al. (2) commented on the influence of gender on circulating C-reactive protein (CRP), representing the inflammatory response of the organism. Concurring with our conclusion that, added to fasting triglyceride concentrations, elevated CRP was proposed to mark high-density lipoprotein (HDL) dysfunction in cardiometabolic diseases among men but not in women (in whom elevated complement C3 was identified as independent marker), the author pointed out that, in the long-term treatment of schizophrenic patients with clozapine, a drug that consistently raises CRP levels, women respond with about half as great a rise in CRP as men.

We have reached the opinion since our study was concluded, that this sex difference on CRP is related to the association of circulating lipoprotein [Lp] (a) with triglycerides and the possible concomitant existence of autoimmune activation, a slow process which comprises aggregation between certain damaged proteins and protective serum proteins, and predominates in females (3). HDL dysfunction in males is usually, though not exclusively, due to simple pro-inflammatory state whereas it is associated in women commonly with inflammation-induced autoimmune process as well which often involves Lp(a) and apolipoprotein A-I. The immune complex formation is associated with complement C3 elevation (4) and with less independence from other clinical markers of systemic inflammation (including fasting triglycerides) and a lesser degree of rise in CRP levels. Hence, the divergence between sexes in rise of CRP levels to certain pro-inflammatory mediators. One may speculate whether clozapine, beyond stimulating CRP release, affects in women the induction of autoimmune processes.

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Can isolated ST elevation in aVR lead be a sign of acute pulmonary embolism?

aVR'de izole ST yükselmesi akut pulmoner emboli belirtisi olabilir mi?

Dear Editor,

Pulmonary embolism (PE) remains the major challenge of acute chest disease. The clinical and electrocardiographic manifestations may deviate to a diagnosis of acute coronary syndrome and even myocardial infarction. The current report documents the case of a 42-year-old woman who presented to the emergency department with chest pain and dyspnea. She had no known history of ischemic heart disease, cardiomyopathy, arrhythmia, or central nervous system disease. She had a 6-year history of minimal tobacco use. On examination, the patient's blood pressure was 100/60 mm Hg in the supine position; oxygen saturation, 96%; and heart rate, 123 beats per minute and regular. Her electrocardiogram showed interestingly isolated ST elevation in aVR lead and

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.

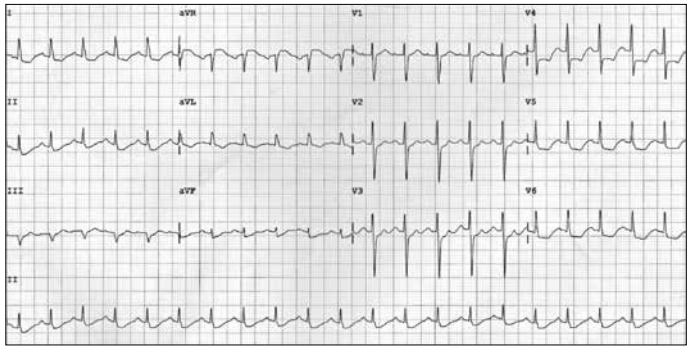

Figure 1. A 12-lead electrocardiogram in patient with acute pulmonary thromboembolism, note the ST segment elevation in aVR lead

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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