

## Complete atrioventricular block and syncope during acute pulmonary thromboembolism: a case report

Akut pulmoner tromboembolizm sırasında atriyoventriküler tam blok ve senkop gelişimi: Olgu sunumu

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The most common electrocardiographic (ECG) findings in acute pulmonary thromboembolism (PTE) are right bundle branch block, T wave, and ST-segment changes. Complete atrioventricular (AV) block has hitherto been reported in only one patient with PTE. A 63-year-old female patient presented with recurrent syncope and sudden-onset dyspnea. There was complete AV block in the admission ECG. She never had similar complaints before and an ECG taken three months before was completely normal. A temporary pacemaker was placed through the femoral vein. Physical and laboratory findings were suggestive of acute pulmonary embolism. Pulmonary artery angiography demonstrated nearly total occlusion of the proximal right pulmonary artery. Her coronary arteries were normal. Thrombolytic therapy with streptokinase infusion followed by standard heparin infusion resulted in clinical improvement and resolution of complete AV block. The patient was discharged on the 15th day with oral warfarin treatment.

**Key words:** Electrocardiography; heart block/complications; pulmonary embolism/diagnosis/complications; syncope/etiology.

Acute pulmonary thromboembolism (PTE) is a serious disease that may be accompanied by electrocardiographic (ECG) abnormalities.<sup>[1]</sup> Incomplete or complete right bundle branch block (RBBB), S1Q3T3 pattern, T wave inversion, and ST-segment depression in leads V1-4, aVF, and DIII are the most common ECG findings in PTE.<sup>[2]</sup> In addition, sinus bradycardia and atrioventricular (AV) block can be seen due to vasovagal reflex during PTE.<sup>[3,4]</sup> Although certain ECG findings may raise the suspicion of pulmonary embolism (PE), ventilation-perfusion scintigraphy, pulmonary arterial angiography, and necropsy results confirm the diagnosis.<sup>[5]</sup>

Akut pulmoner tromboembolizmde sağ dal bloku, T dalga ve ST-segment değişiklikleri en yaygın görülen elektrokardiyografi (EKG) bulgularıdır. Tam atriyoventriküler blok ise sadece bir olguda bildirilmiştir. Altmış üç yaşında kadın hasta tekrarlayan senkop atakları ve ani başlayan dispne ile başvurdu. Yatış EKG'sinde tam atriyoventriküler blok saptandı. Daha önce benzer semptomları olmayan hastanın üç ay önce çekilen EKG'si tamamen normal idi. Hastaya femoral venden geçici pacemaker takıldı. Fizik ve laboratuvar bulguları nedeniyle akut pulmoner emboliden şüphelenilen hastaya yapılan pulmoner arter anjiyografisinde sağ pulmoner arter proksimalinin neredeyse tamamen tıkalı olduğu görüldü. Koroner arterler ise normal bulundu. Streptokinaz infüzyonu ile trombolitik tedavi ve arkasından standart heparin infüzyonu ile klinik düzelme sağlandı ve atriyoventriküler blokun çözüldüğü görüldü. Hasta 15 gün sonra, oral warfarin tedavisi verilerek taburcu edildi.

**Anahtar sözcükler:** Elektrokardiyografi; kalp bloku/komplikasyon; pulmoner embolizm/tanı/komplikasyon; senkop/etioloji.

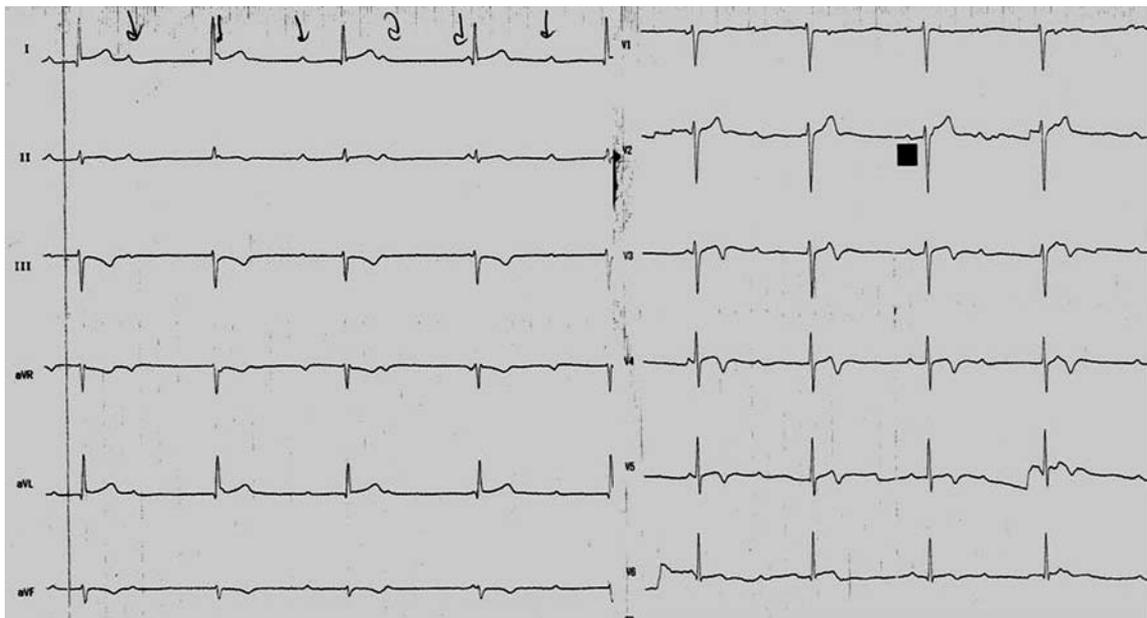
In this paper, we report on a patient who developed complete AV block and syncope during acute massive PE.

### CASE REPORT

A 63-year-old female patient presented with recurrent syncope and sudden-onset dyspnea lasting for three days. There was complete AV block in the admission

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**Figure 1.** Complete atrioventricular block observed in the admission electrocardiography.

ECG (Fig. 1). The patient was hospitalized and a temporary intravenous pacemaker was placed through the femoral vein. She was not taking any negative chronotropic drugs and no electrolyte imbalance was detected. On admission, physical examination revealed a systolic murmur of grade 2 at the lower left sternal border, and wide splitting of the second heart sound with an accentuated P<sub>2</sub> component. She had mild unconsciousness, distress, hypotension (systolic/diastolic blood pressure 70/50 mmHg), bradycardia, and tachypnea (respiration rate 42/min). She was taking lisinopril 10 mg/day for hypertension of 5-year history, had a history of smoking (12 pack-years) for 35 years, and underwent a previous operation for inguinal herniation. She never had similar complaints before and an ECG taken three months before was completely normal.

Laboratory tests showed normal troponin T and myocardial fraction of creatine kinase, but a fairly increased D-dimer level (2,300 mg/dl; normal 65-250 mg/dl). Hemoglobin was 10.6 g/dl (normal 12.0-15.0 g/dl) and white blood cell count was 11,000 mm<sup>3</sup>. Arterial blood gas analysis on room air yielded the following: pH 7.56, PCO<sub>2</sub> 23 mmHg, and PO<sub>2</sub> 45 mmHg. The telecardiogram revealed an increased cardiothoracic ratio and a remarkable pulmonary conus. Transthoracic echocardiography showed normal left ventricular systolic functions, mild tricuspid regurgitation, dilated right cardiac chambers, paradox movement of the intra-ventricular septum, and systolic pulmonary arterial pressure of 35 mmHg. Physical and laboratory findings and the history of the patient were suggestive of

acute PE. Pulmonary artery angiography demonstrated nearly total occlusion of the proximal right pulmonary artery (Fig. 2). Coronary angiography showed normal coronary arteries. After the diagnosis was made and at the second hour of admission to the coronary care unit, 48-hour streptokinase infusion was initiated with a rate of 100,000 U/hr following a bolus injection of 250,000 U in 30 min. At the sixth hour of streptokinase infusion, complete AV block resolved and the ECG showed incomplete RBBB, and inverted T waves in leads V1-4. During the first 24 hours, intermittent short AV block episodes were seen. Standard heparin infusion was started for seven days after the completion of streptokinase therapy. Clinical and laboratory findings improved during therapy. On the fourth day,



**Figure 2.** Pulmonary artery angiography showing near occlusion of the proximal right pulmonary artery.

lower extremity venous Doppler examination revealed a thrombus extending from the common femoral to the popliteal veins. The patient was discharged on the 15th day with oral warfarin treatment.

## DISCUSSION

Diagnosis of PE depends primarily on clinical suspicion. The ECG abnormalities are quite various in acute PTE and have a relatively minor importance at diagnosis. Combination of clinical and ECG findings increases the diagnostic sensitivity from 30% to 80% in patients at high risk after an operation.<sup>[6]</sup> Incomplete or complete RBBB and right axis deviation are associated with the extent of embolic obstruction. The most probable explanation for the appearance of RBBB in acute massive PTE is the inhibition of blood supply to the subendocardial blood vessels due to acute ventricular dilatation. Right bundle branch block is a characteristic ECG finding and shows involvement of the main pulmonary arteries.<sup>[2]</sup>

Bradycardia and AV block which are quite rare in PTE are responsible for the development of syncope. Syncope is seen in approximately 10% of patients with PTE and explained by three mechanisms. First, massive PTE can cause hypotension due to acute right ventricular failure and impaired left ventricular filling. Second, PTE can cause complete AV block in the setting of pre-existing left bundle branch block (LBBB). Wilner et al.<sup>[7]</sup> reported syncope as a consequence of paroxysmal AV block in two PTE patients with pre-existing LBBB. Marti et al.<sup>[8]</sup> reported complete AV block secondary to PTE in a patient who had LBBB on admission. Third, PTE can trigger vasovagal reflex syncope. Heart rate and myocardial contractility are increased due to increased sympathetic discharge from stimulation of the right atrial stretch receptors, decreased left ventricular filling, and hypoxia. In the presence of decreased left ventricular filling, this positive inotropic effect may result in stimulation of the left ventricular mechanoreceptors (vagal afferent C-fibers).<sup>[9]</sup> These fibers are located at the base of both ventricles and stimulated by passive distention of the right ventricle by a large embolus. Subsequently, increased parasympathetic discharge due to stimulation of vagal afferent C-fibers leads to bradycardia, AV block, and hypotension.

The occurrence of complete AV block and syncope in our patient without pre-existing ECG abnormalities suggest a vasovagal reflex as the most likely cause. Simpson et al.<sup>[10]</sup> reported two patients with transient sinus bradycardia and AV block causing syncope. Akinboboye et al.<sup>[11]</sup> reported a case with second-degree AV block and syncope during recurrent PE.

In conclusion, in the setting of acute PTE, common ECG findings such as RBBB, ST-segment changes, and T wave inversions may be accompanied by an increase in vagal tone, which in turn may result in sinus bradycardia and AV block leading to syncope by affecting the cardiac intrinsic conduction system at the level of sinoatrial and atrioventricular nodes. Therefore, acute massive PTE should be considered in the differential diagnosis in patients presenting with syncope or near-syncope. As in our case, a high index of suspicion, early diagnosis, and early treatment may prevent significant morbidity or mortality.

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