***Case Report* / Olgu Sunumu**

Reverse McConnell Sign in a Patient with Acute Pulmonary Embolism

Akut Pulmoner Embolili Hastada Ters McConnell Bulgusu

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There are various echocardiographic parameters for diagnosis of pulmonary embolism. Among these paramaters, "McConnell sign" is defined as right ventricular free wall hypokinesis in the presence of normal right ventricular apical contractility. We presented a patient with acute pulmonary embolism which caused reversible akinesia of the apex and right ventricular midfree wall, a finding we would like to term "reverse McConnell sign."

Key words: Acute pulmonary embolism; reverse McConnell Sign; cardiac arrest.

Pulmoner embolinin tanısı için birçok ekokardiyografik parametre bulunmaktadır. Bu parametreler arasında, sağ ventrikül apikal kasılması normalken sağ ventrikül serbest duvar hareketinde hipokinezi olması "McConnell bulgusu" olarak tanımlanmaktadır. Biz sağ ventrikül apeksi ve serbest duvarının reversibl akinezisine yol açan akut pulmoner embolili bir olgu sunduk ve bu bulguyu "ters McConnell işareti" olarak yorumladık.

Anahtar sözcükler: Akut pulmoner emboli; ters McConnell işareti; kardiyak arrest.

There are various echocardiographic parameters for diagnosis of pulmonary embolism. Among these paramaters, "McConnell sign" is defined as right ventricular free wall hypokinesis in the presence of normal right ventricular apical contractility. We presented a patient with acute pulmonary embolism which caused reversible akinesia of the apex and right ventricular midfree wall, a finding we would like to term "reverse McConnell sign."

CASE REPORT

A 42-year-old man presented with sudden cardiac arrest. Upon arrival in emergency room, the initial physical examination revealed hypoxemia (pulse oximetry was in low-mid 80's) and arterial blood gas showed pH 7.44, $PaCO_2$ 33 mmHg, PaO_2 42 mmHg on room air. He was intubated, resuscitated with normal saline and intravenous adrenaline and was transferred to the intensive care unit. Resuscitation continued in intensive care unit. Shortly afterwards, his blood pressure was 80/60 mm Hg, his heart rate was 115/min, and his respiratory rate was 22/min. Electrocardiography showed deep S waves in lead II, 1 mm of ST elevation in lead III and 2 mm of ST depression in aVL. An emergent bedside echocardiogram showed right ventricle (RV) dilatation, mild tricuspid regurgitation and a peak tricuspid regurgitation pressure gradient of 32 mm Hg, paradoxical motion of the interventricular septum, vigorous contractions of the basal segment of the RV free wall, and akinesia of the mid-RV free wall and RV apex (Fig. 1). D-dimer level from a blood sample taken during the arrest was grossly elevated at 1048 μg/L (Normal < 200 μg/L). Thorax computerized tomography or pulmonary angiography for the diagnosis of pulmonary embolism was not done due to hemodynamic instability of the patient. At this point pulmonary embolism was suspected and emer-

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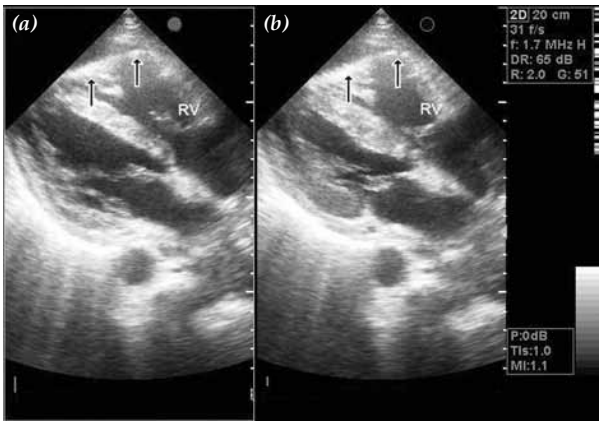

Fig. 1. *Apical 4-chamber view of right ventricle in diastole* ***(a)*** *and systole* ***(b)*** *showing akinesia of RV apex and midfree wall.*

gently 20 mg boluses of recombinant tissue type plasminogen activator were administered via femoral vein, followed by an infusion of 90 mg over one hour. Serial troponin I measurements were within normal range. A repeated echocardiogram two days later showed mild hypokinesia of the mid-RV free wall and RV apex, and normal RV wall motion at the base (Fig. 2). However, the patient died at the third day of hospitalization.

DISCUSSION

Pulmonary embolism is a possible noncardiac cause of cardiac arrest and has an extremely unfavorable prognosis.[1] The mechanism of cardiac arrest caused by pulmonary embolism is based on pulmonary mainstream obstruction and liberation of vasoconstrictive mediators from the thrombi, leading to increased right ventricular afterload. As the right ventricle fails, right atrial pressure rises and cardiogenic shock occurs. Overload of the right ventricle results in a leftward shift of the ventricular septum, leading to decreased left ventricular diastolic filling and diastolic volume. This situation causes circulatory failure.[2] Transthoracic echocardiography (TTE) is a noninvasive, painless and cost-effective examination, very useful in diagnostics of acute pulmonary embolism.[2]

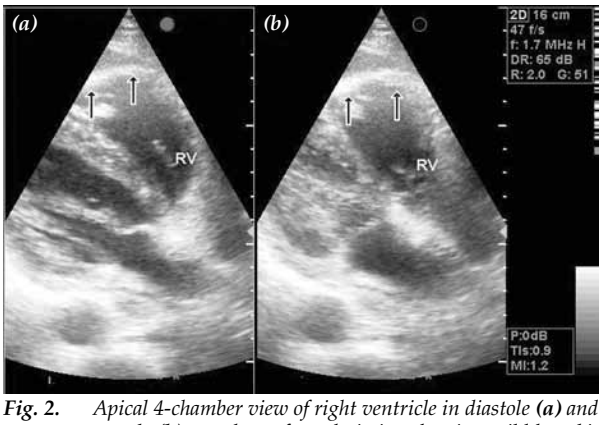

Fig. 2. *Apical 4-chamber view of right ventricle in diastole* ***(a)*** *and systole* ***(b)*** *two days after admission showing mild hypokinesia of RV apex and midfree wall .*

In hemodynamically unstable patients, TTE can be performed rapidly and may reveal evidence of RV failure suggestive of pulmonary embolism, as well as other conditions including myocardial infarction, aortic dissection, and pericardial tamponade. Echocardiographic findings among patients with pulmonary embolism include RV dilatation and hypokinesis, paradoxical interventricular septal motion toward the LV, tricuspid regurgitation, and pulmonary hypertension.[2,3] RV hypokinesis is an independent risk predictor for early death in patients with pulmonary embolism.[4] The finding of regional RV dysfunction with severe free wall akinesia or hypokinesia (McConnell sign) is specific for pulmonary embolism.[5] McConnell sign consists of normokinesia of the apical RV free wall segment and akinesia of the mid-RV free wall. We found hyperkinesia of the basal RV free wall, while the mid-RV free wall and RV apex were showing akinesia.[5] To the best of our knowledge, these findings observed in our patient has been reported in only one case by Haghi et al.[6] so far.

Haghi et al.[6] had defined these findings as a "reverse McConnell sign". We also defined the findings in our patient as a reverse McConnell sign being inspired by Haghi et al. McConnell sign was originally suggested as a useful criterion for the diagnosis of acute pulmonary embolism mostly because it was observed in the setting of acute but not chronic pulmonary hypertension.[6] Casazza et al.[7] reassessed the McConnell sign in a different clinical setting. They compared two-dimensional echo recordings of patients with acute pulmonary embolism and acute RV infarction. The data showed that the previously described abnormal pattern of regional RV contractility did not allow to differentiate between those two conditions.[7] In fact RV wall motion abnormalities in acute pulmonary embolism were virtually identical to those observed in acute RV infarction. As in the original report on McConnell's sign, Casazza et al.[7] were also unable to give a clear explanation of the cause of preserved RV apical contractility in acute pulmonary embolism and RV infarction.

In conclusion, presence of RV wall motion abnormalities can be more a rationalistic approach rather than localization of RV wall motion abnormality for suspicion of acute pulmonary embolism. However, large studies are necessary in order to practice this approach.

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