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Massive pulmonary embolism with extreme hypoxia and hypocarbia caused by knee bandage in young male

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Summary

Pulmonary embolism (PE) caused by obstruction of the pulmonary arterial bed is an acute, life threatening, cardiovascular emergent situation. It is a reversible cause of right ventricular failure. The initial diagnosis majority may be missed due to symptoms and signs are nonspecific. Because of late detection of diagnosis is fatal, PE should be thought when acute dyspnea occurs. PE and deep vein thrombosis (DVT) are clinical situations related to venous thromboembolism (VTE). PE accompanies 50% to DVT.[(1)] The real incidence of DVT and PE is not known due to nonspecific clinical situation. Pulmonary embolism occurs in 0.4% of hospitalized patients.[(2)]

Keywords: Pulmonary embolism, deep vein thrombosis, ventricular failure.

Case

A 34 years old professional basketball player was admitted to our emergency service with ambulance service due to acute dyspnea. The dyspnea had started suddenly after basketball training and the general condition of the patient has rapidly deteriorated. The patient had an injury of left knee anterior cruciate ligament two month ago. A knee bandage had been advised by an orthopedist. Except this injury there was no special characteristic on his background. On physical examination, he was unconscious and extremely cyanotic. He had severe dyspnea and tachypnea. First and second heart sound were normal, but there was right ventricular third heart sound (S3) and jugular venous distention. The blood pressure was 80/50 mmHg. The ECG

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showed S1Q3T3 pattern. There was a bandage on his left knee. Also on the left thigh there was swelling and stiffness. The artery blood gas showed extreme hypoxia, hypocarbia and deep acidosis (**Figure 1**).

On laboratory examination D-dimer was extremely high. Transthoracic echocardiography (TTE) revealed right ventricular dilatation (RV diameter:3.47 cm), systolic dysfunction (TAPSE:0.92 cm and tricuspid valve lateral annulus systolic velocity:0.7 cm/sec) , and high estimated systolic pulmonary artery pressure (sPAP:55 mmHg) (**Figure 2 and 3**). Finally we suspected strongly acute pulmonary emboli. The patient was considered at high risk of PE due to hemodynamic instability. So we decided urgently to start intravenous thrombolytic therapy. He was intubated because of poor blood gasses results. 100 mg t-PA was given in two hours. Unfractionated heparin (UFH) was started with t-PA and continued after thrombolytic therapy. After thrombolytic therapy the hemodynamic situation and artery blood sample were improved (**Figure 4**). The patient was extubated.

When he was stable, pulmonary CT angiography was performed. Huge thrombus bulks were observed in both the pulmonary artery and distal branches. Warfarin was started to patient. Bilateral lower extremities venous Doppler and coagulation parameters (Factor Leiden V, protein C and S etc.) were normal. So we thought that the PE was secondary to venous blood restriction by knee bandage and decided to give warfarin therapy for six months. The patient was discharged without complaint to be followed up as outpatients.

Figure 1. *Initial blood gases results.*

Discussion

Pulmonary embolism (PE) caused by obstruction of the pulmonary arterial bed is an acute, life threatening,

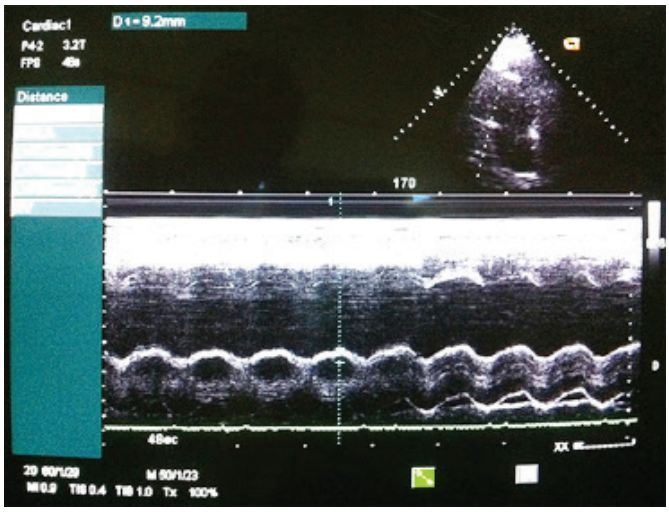

Figure 2. *Right ventricular enlargement in transthoracic echocardiography parasternal long axis view.*

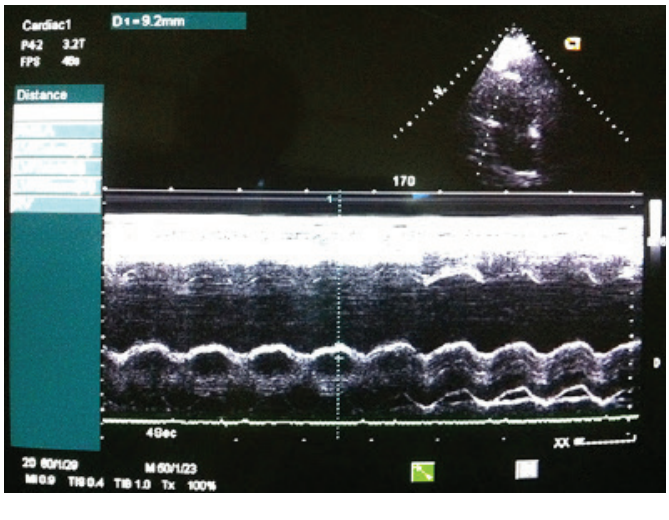

Figure 3. *Decreased TAPSE in transthoracic echocar diography.*

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cardiovascular emergent situation. PE and deep vein thrombosis (DVT) are clinical situations related to venous thromboembolism (VTE). On patients who admitted to the clinic with PE, DVT is the underlying cause of %70 as a result of further investigations.[3]

Figure 4. *After thrombolytic therapy and extubation blood gases results.*

In the presence of a number of predisposing factors that facilitate the exit is called secondary PE. If there is any underlying cause is called idiopathic PE. Compose 20% of all cases were idiopathic PE according to ICOPER.[4] Facilitating factors that are associated with the patient or conditions include certain; advanced age, prior VTE, active malignancy, neurological disease with extremity paralysis such as heart disease and respiratory failure situations that require a long time immobilization, congenital or acquired thrombophilia, hormone replacement therapy (HRT) or combined oral contraceptive treatment, hip or leg fracture, hip or knee joint replacement, major surgery and trauma.[5]

Conclusion

In our patient there was no any immobilization history. But he had history of knee injury. Although it did not cause immobilization there was a bandage on his knee which restricting venous blood flow. So we keep in mind that deep vein thrombosis and PE may be occurred without immobilization.

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Conflicts of interest were not reported.

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