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Case Report

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Sudden death of a female adolescent due to inferior vena cava thrombosis andpulmonary embolism

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ABSTRACT

Despite the fact that considerable advances were seen in diagnosis and treatment of pulmonary embolism since last decades, it is still a seriously mortal condition. Although pulmonary embolism is very rare in children and young people, it can be extremely deadly. Vena cava inferior thrombosis might give rise to pulmonary embolism. Association of vena cava inferior thrombosis and pulmonary embolism might be unthought of particularly in adolescents or young adults, and difficult to manage for the clinicians.

Keywords: Adolescent, pulmonary embolism, vena cava inferior thrombosis.

INTRODUCTION

Pulmonary embolism (PE) due to pulmonary bed obstruction may progress to severe acute right ventricular failure. Contrary to widespread belief, PE is not an uncommon condition. Hospital admissions due to PE was encountered as being 65 per 100.000, in USA, in 2012.[1] On the other hand, PE is reported to be diagnosed in two million cases globally with 3% mortality rate.[2] 5 to 10% of in-hospital deaths are attributed to PE.[3] Non-specific signs and symptoms of PE makes it difficult to diagnose and the clinicians also do not perform an extra effort not to overlook the PE. However, the diagnosis and emergent intervention of PE is of vital significance.

As it is well-known, PE mostly seen in the elderly population. The elderly over 70-year old are at risk of PE as high as 3-fold in comparison to those individuals between the ages of 45 to 69, and the individuals between the ages of 20 to 44 have a 3-fold lower risk of PE than those individuals who are between 45 and 69.[4] PE in adolescents is thought to be very rare, displaying an incidence as much incidence as 1/50 of the elderly population. As for the children, it is found to be between 8.6-57/100.000 in hospitalized children, and 0.14-0.9/100.000 in non-hospitalized children.[5,6]

We sometimes come across with the cases include both vena cava inferior (VCI) thrombosis and PE. Recently, a case is reported, which involves both the inferior vena cava thrombosis and PE simultaneously. However, that report associates of both clinical entities of VCI thrombosis and PE with adverse effects of SARS-CoV-2 vaccine. Accordingly, a 27-year-old young male is reported. He admits to the hospital with chief complaints of cough, hemoptysis and epigastric pain which are reported to begin 10 days later receiving the second dose of mRNA-1273 (Moderna, Cambridge).[7] It is reported that he has no history of trauma or surgical intervention. Also, he and his family are reported to have no history of venous thromboembolism and any coagulation disorder. In another study, a PE case due to VCI thrombosis in a 29-year-old male with chronic pancreatitis is reported.[8]

In this case report, a 17-year-old female adolescent with both PE and VCI thrombosis was presented.

CASE

A 17-year-old female adolescent admitted to the emergency department of Ersin Arslan Training and Research Hospital with acute abdominal pain complaint. Informed consent form was approved by the relatives of the patient. She reported that she had a widespread and vague abdominal pain with a feeling of shortness of breath, since last three to four hours. The patient was not obese and was of medium height. Her medical history revealed neither any surgical intervention nor pregnancy/miscarriage/abortion. The history of mRNA vaccination for SARS-COV-2 pandemic was questioned. The patient was understood to have a sum of four doses vaccination for SARS-CoV-2, firstly two doses of conventional type of vaccine and the two doses of mRNA type of vaccine. It

was revealed that about 10 months had passed since the last mRNA vaccination. According to the knowledge of her past medical history received from relatives, she had never smoked or used alcohol. They could not provide reliable information to exclude the existence of a kind of familial thrombophilia and could not precisely report whether there is venous thromboembolism case or not in their family. When the patient was asked, it was uncovered that she did not ever use oral contraceptives or any other regular medication. Vital signs were as follows: respiration was 18/min, heart rate was 98/min, body temperature was 38°C, blood pressure was 85/55 mmHg, and oxygen saturation was 95%. The hematological parameters were as follows: white blood cell count was 17.6×10^9/L (4-10×10^9/L), neutrophil count was 16.7×10^9/L (2-7×10^9/L), lymphocyte count was 0.5×10^9/L (0.8-4×10^9/L), monocyte count was 0.3×10^9/L (0.2-0.8×10^9/L), eosinophil count was 0.0×10^9/L (0.02-0.5×10^9/L), hemoglobin was 13.4 g/dL (11.5-14.7 g/dL), hematocrit was 39.5% (36.9-49.1%), red blood cell count was 4.35×10^{12}/L (4-6×10^{12}/L), thrombocyte count was 233×10^9/L (150-400×10^9/L), mean corpuscular hemoglobin was 30.8 pg (27-33 pg), mean corpuscular hemoglobin concentration was 33.9 g/dL (32-36 g/dL), red cell distribution width standard deviation was 40.7 fL (36-50 fL), and mean corpuscular volume was 90.9 fL (80-100 fL). According to the arterial blood gas parameters, pH was 7.246 (7.35-7.45), pO_2 was 20.5 mmHg (80-108 mmHg), and pCO_2 was 41.6 mmHg (32-48 mmHg). In biochemical profile, albumin was 43.3 g/L (35-52 g/L), urea was 18.8 mg/dL (17-43 mg/dL), alkaline phosphatase was 69 U/L (30-120 U/L), lipase was 15.8 U/L (0-67 U/L), indirect bilirubin was 0.88 mg/dL (0-1 mg/dL), direct bilirubin was 0.461 mg/dL (0-0.5 mg/dL), creatinine was 0.82 mg/dL (0.5-0.95 mg/dL), glucose was 142.9 mg/dL (70-100 mg/dL), Na+ was 141.2 mmol/L (135-145 mmol/L), K+ was 3.9 mmol/L (3.5-5 mmol/L), Cl- was 104.1 mmol/L (98-109 mmol/L), Ca^{++} was 2.28 mmol/L (2.17-2.51 mmol/L), and C-reactive protein was 6.48 mg/L (0-5 mg/L). D-dimer was 4.27 mg/L (0-0.55 mg/L). Prothrombin time was 14.5 s and active partial thromboplastin time was 36.5 s. Fibrinogen was 160 mg/dL (150-400 mg/dL).

While an abdominal ultrasonography screening was being planned to reveal the etiology of abdominal pain in the emergency department, an urgent computed tomography pulmoner angiogram (CTPA) screening was decided upon encountering a high D-dimer value. No finding was found in the lower extremity Doppler ultrasonography screening. According to the CTPA report, filling defects consistent with acute pulmonary thromboembolism were observed in the segmental and subsegmental branches of both main pulmonary arteries and thrombosis in inferior vena cava (**Figure 1, 2** and **3**). 6. The case was intervened with tissue plasminogen activator (100 mg/2 h).

After the patient was as diagnosed as PE, she was admitted to intensive care unit. The patient was also consulted the thoracic surgery department to get the patient considered for VCI filter insertion or aspiration thrombectomy. However, the blood pressure decreased to 75/50 mmHg, palpitations, and paradoxical pulse was observed. She passed away within ten minutes immediately after supportive care (hydration, oxygenation, ventilation etc.) started in intensive care unit.

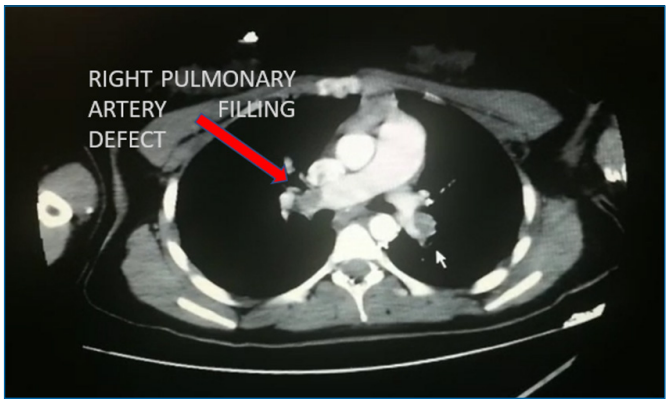

Figure 1: Right pulmonary artery filling defect in computed tomography pulmoner angiogram.

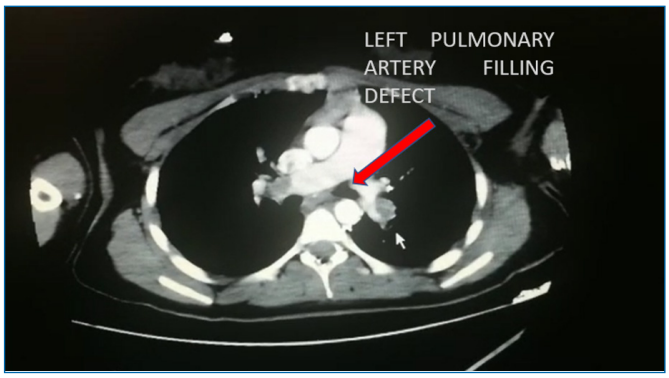

Figure 2: Left pulmonary artery filling defect in computed tomography pulmoner angiogram.

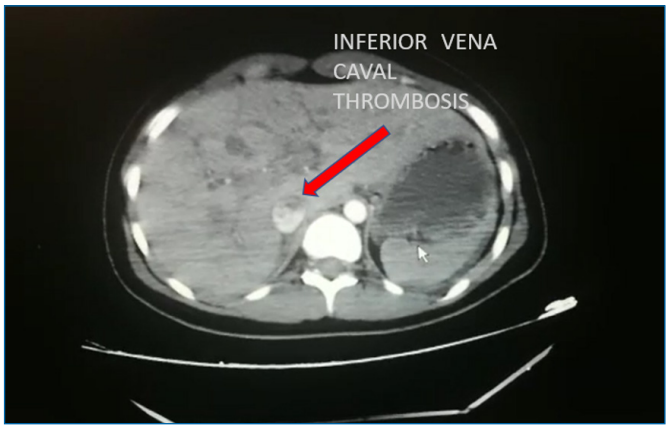

Figure 3: Observation of inferior vena caval thrombosis in computed tomography pulmoner angiogram.

DISCUSSION

According to a recent study, only 9 of 728 acute PE patients were in the adolescent age group, in a period of 16 years of follow-up. Despite the incidence rate of the PE is observed as nearly 1.5 times high in women as it is in men, there seems not to be a significant difference between genders, in the age range of 15 to 20. This is attributed to the less incidence of abortion and oral contraceptive usage, which are well-known contributors of PE, in that range of age.[6] In another study, in which the patients aged between 10 to 17 are 52.2%, most commonly detected risk factors of PE in children are revealed to be central venous catheter (39.2%), malignancy (34.8%), recent surgery (34.8%), infection (30.4%), drugs such as corticosteroids and asparaginase (21.7%), Nephrotic Syndrome (13.0%), congenital heart disease (8.7%), trauma (4.3%), polycythemia (4.3%), and splenectomy (4.3%). Unfortunately, the total number of the patients included in the study is 23, very low in order to be considered sufficient to

rely on the outcomes, like many other studies conducted with children or adolescents PE patients. 16 out of 23 patients are determined to have D-dimer levels above 0.5 mg/L.[9]

Although deep vein thrombosis (DVT) is the most significant one among the causes of PE, it should not be ignored that VCI thrombosis may also result in PE. Along with the certain recent case reports,[7,8] it is revealed that PE is one of leading outcomes among of acute or chronic complications of VCI thrombosis, with a ratio of 30%. It is also stated that the mortality of VCI is twice that of DVT.[10] Unfortunately, rapid loss of the patient in the case we presented did not allow us to carry out further investigations to elucidate the etiology.

CONCLUSION

Although Pulmonary Embolism is a disease that is known to be mostly specific to the elderly, it can cause dramatic results in adolescents as well. VCI thrombosis might simultaneously prompt to PE.

ETHICAL DECLARATIONS

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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