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Pulmonary embolism in young adult associated with Protein S Deficiency: A Case Report

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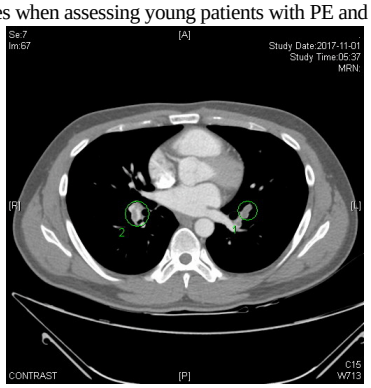
Abstract Acute pulmonary embolism(PE) is a component of venous thromboembolism(VTE), which proven fatal if not suspected and treated. The common risk factors for PE and VTE include prolonged immobility, older age, inherited clotting factors, and malignancy. Young man who are not have medical history and active are considered to be a lower risk group. We present an rare case of thromboembolism in young male associated with protein S deficiency. Case Report A 25-year-old male presented to the out patient department with an episode of hemoptysis 2 weeks ago. He had fever, chill, sputum. The patient was agency manager. His past medical history were unremarkable. His vital signs were normal. He had left calf painful swelling. Laboratory tests revealed leukocytosis(11300/uL), elevated D-dimer(3.73ug/mL), C-reactive protein(3.29ug/dL) levels and normal aPTT(36.2sec), and PT(12.7sec). Chest X-ray revealed opacity of right lower lung field. An ekg showed a regular rhythm. He was treated with cephaloporin and macrolide. The chest CT showed nodular consolidation on both lower lobe and the multiple PE in both lobar and segmental pulmonary artery. On lower extremity venography, multifocal VTE of left leg was seen. On family history, his brother had history of treatment with upper extremity VTE. Considering the patient's young age and family history, physician evaluated other risk factors for VTE including genetic causes. Protein S activity and protein S free level was decreased to 17% and 21.7%. The patient received anticoagulation treatment with unfractionated heparin and an oral anticoagulant. Life-long anticoagulation therapy continued and the patient was followed up for 10 months without any clinical events. Conclusion We reported a uncommon case of thromboembolism associated with protein S deficiency in young adult. Identifying predisposing factors for PE is important in deciding treatment duration and in preventive strategies for recurrence. It may be reasonable to check genetic causes when assessing young patients with PE and VTE.



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Rapid progression of silicosis combined with mixed dust pneumoconiosis

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Background: Radiologic progression of silicosis, a type of pneumoconiosis, takes about one decade. Rapidly progressive pneumoconiosis (RPP) frequently occurs in coal workers, particularly those with high exposure to silica. However, RPP has not been reported in other situations. Goal: To investigate the case of a patient with RPP that developed 25 years after he left his position in a stone processing factory. **Case Report:** A 64-year-old male miller had a persistent cough producing sputum for one month. He was clinically diagnosed with pulmonary tuberculosis in 2011. He worked in a stone processing factory from ages 20 through 37 and has owned his own mill for the past 25 years. His chest radiograph showed significant increases in the size and number of lung nodules since his last follow-up in 2013. Pulmonary function test showed moderate pulmonary insufficiency of the obstructive type with bronchodilator response. Percutaneous needle lung biopsy was performed for histologic diagnosis, and the nodular lesion showed diffuse infiltration of phagocytic macrophages and birefringent particles with polarizing microscopy. The patient was finally diagnosed with RPP of mixed dust pneumoconiosis combined with silicosis and chronic obstructive pulmonary disease. **Conclusion:** In this case, mixed dust pneumoconiosis with silicosis progressed rapidly even after the patient ended stone processing work. Pneumoconiosis might be accelerated by persistent exposure to grain dusts from working in a mill environment.