

A Case of Pulmonary Sarcoidosis Misdiagnosed as a Miliary Tuberculosis

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Sarcoidosis is a multisystemic granulomatous disorder of unknown etiology that affects individuals worldwide and is characterized pathologically by the presence of noncaseating granulomas in involved organ. Sarcoidosis most frequently involves the lung, the common presenting symptoms include cough, dyspnea, and chest pain. The typical radiologic findings are bilateral hilar lymphadenopathies and reticular opacities. We herein report a case with pulmonary sarcoidosis that misdiagnosed as a miliary tuberculosis. A 29 year old male presented with cough and dyspnea. Because the chest radiograph showed randomly distributed miliary nodules in both lungs and his sputum TB-PCR was positive once, we diagnosed as military tuberculosis initially. Despite of anti-tuberculous medication, his respiratory symptoms and radiologic findings were not improved. Finally by the open lung biopsy, we could diagnose the pulmonary sarcoidosis. After taking corticosteroid, the patient was improved gradually. Key Word: Pulmonary sarcoidosis, miliary tuberculosis

폐혈색전증을 동반한 Factor VII 유전자의 프로모터 -401G/A 다형성 1례

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This is the case report of factor VII gene-401 G/A polymorphism in the patient having pulmonary thromboembolism. The patient was 71-year-old woman who visited hospital with acute onset dyspnea. She had CT scan, which showed pulmonary thromboembolism. Even though she was treated by low-dose warfarin, as anticoagulation therapy, there was excessive prolongation of prothrombin time(PT). We examined if there was any deficiency of coagulating factor and found decreased activity of Factor VII. We analyzed the DNA base sequencing to determine the cause of decreased activity of Factor VII, found that there was a substitution from G to A on promotor -401. Furthermore, after doing RT-PCR of DNA of Factor VII gene in the patient and the control, we compared them using gel-electrophoresis. It seemed that the low transcription level made the activity of Factor VII decreased. The transcription level in the patient was lower because the genetic bands of the patient were thinner than those of the control. Comparing the results with those of Human Factor VII gene full sequencing, there was no difference between them except the substitution of G to A on promotor -401, assuming that the transcription abnormalities by promotor polymorphism is the cause. The follow-up CT scan of the patient shows none of the findings of pulmonary thromboembolism anymore after anticoagulation treatment. And the patient is followed-up in an outpatient clinic. We were examining the underlying disease in the patient with pulmonary thromboembolism, found that there was decreased activity of Factor VII. We experienced the case of Factor VII DNA polymorphism with excessively decreased activity of Factor VII, accompanying PT prolongation after low-dose warfarin therapy. We are reporting this case with references.