

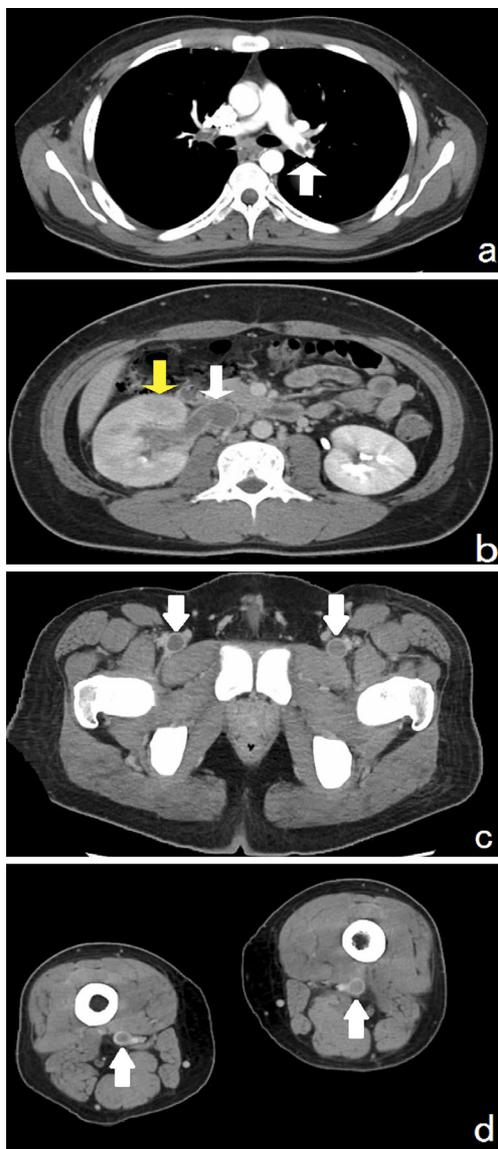
Thromboembolism Supervened on Eosinophilia Induced by Mycoplasma Pneumonia

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Key words: catastrophic thromboembolism, pulmonary embolism, enhanced computed tomography imaging, mycoplasma pneumonia, eosinophilia

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Picture.

An 18-year-old man, who was diagnosed with mycoplasma pneumonia 3 weeks previously, visited our hospital with sustained pain on the bilateral inferior limbs and back. Laboratory investigations revealed eosinophilia ($18 \times 10^2/\mu\text{L}$) and an elevated D-dimer level ($21 \mu\text{g}/\text{mL}$) in the plasma. A line of data excluded the possibility of drug allergies. In addition, there were no drug and other allergy related events. His eosinophil count in the peripheral blood had remained within the normal range. When the patient was diagnosed with mycoplasma pneumonia, the value was markedly elevated around $50 \times 10^2/\mu\text{L}$, which was strongly correlated with the disease activity during the course of treatment. Contrast-enhanced computed tomography demonstrated left pulmonary embolism (Picture a, white arrow) and right renal vein thrombosis with a swollen kidney (Picture b, yellow and white arrows). Bilateral femoral and popliteal veins thromboses were also detected (Picture c and d, white arrows). No supporting data were observed regarding abnormalities of coagulation or fibrinolysis, including the presence of pertinent antibodies. The extension of systemic thromboembolism was well correlated with the change in eosinophil count in the circulation, suggesting that the thromboembolism was caused by mycoplasma pneumonia. The patient completely recovered following anticoagulant therapy with normalization of eosinophilia and the D-dimer level. It has been reported that approximately 10% of patients with mycoplasma pneumonia are complicated with transient eosinophilia (1). Cytoplasmic granules of differentiated eosinophils are known to evoke hypercoagulability via the inhibition of thrombomodulin cofactors (2). To the best of our knowledge, this is the first demonstration of thromboembolism induced by transient eosinophilia in a case of mycoplasma pneumonia.

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The authors state that they have no Conflict of Interest (COI).

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References

1. Defilippi AC, Silvestri M, Giacchino R, Di Pietro P, Rossi GA.

Changes in blood eosinophil numbers during *Mycoplasma pneumoniae* infection in wheezing and non-wheezing, atopic and non-atopic children. *Pediatr Int* **50**: 718-721, 2008.

2. Mukai HY, Ninomiya H, Ohtani K, Nagasawa T, Abe T. Major basic protein binding to thrombomodulin potentially contributes to the thrombosis in patients with eosinophilia. *Br J Haematol* **90**: 892-899, 1995.

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