**Introduction**—Patients with MPO-AAV have an increased risk of deep vein thrombosis (DVT). However, the mechanism remains elusive. Recently, it has been reported that aberrant formation and disordered regulation of NETs could be implicated in the development of MPO-AAV. On the other hand, NETs interact with coagulant factors and can induce thrombosis. In this study, we carried out autopsy on an MPO-AAV patient complicated with DVT, and then the association of NETs and DVT was examined.

**Methods**—NETs in the glomeruli and thrombus were examined by immunofluorescent staining. To quantify the NETs volume in the thrombus, immunohistochemistry was performed using anti-citrullinated histone 3 antibody. The area of NETs was quantified by Image J software and then standardized by the numbers of neutrophils counted in the serial sections. The amount of NETs in the thrombus was compared to other thrombi derived from a patient who died of bacterial sepsis and from one who died of post-operative pulmonary embolism.

**Results**—NETs were detected in the glomerular crescents despite the absence of microbes in the MPO-AAV patient. Interestingly, abundant NETs were detected in the thrombus complicated with MPO-AAV, and the amount of NETs was significantly greater compared to other thrombi unrelated to MPO-AAV.

**Discussion**—It is reported that MPO-ANCA can induce NETs, and that NETs can induce thrombosis. This study suggests the possibility that the pathogenesis of thrombosis in MPO-AAV could be critically associated with the mechanism via NETs.

**Conclusion**—This study suggests the possibility that the pathogenesis of DVT in MPO-AAV could be critically associated with the mechanism via MPO-ANCA and NETs.

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**Abundant neutrophil extracellular traps in thrombus of patient with microscopic polyangiitis**

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**Introduction**—CD68+ B cells may be a novel immunological biomarker to follow induction of remission and impending flare in patients with ANCA vasculitis before and after treatment with rituximab.

**Reference**


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