"Mechanisms and clinical significance of neutrophil extracellular traps (NETs) in thrombosis"

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Neutrophil extracellular traps (NETs) are supramolecular chromatin-based assemblies released by activated neutrophils and composed of decondensed DNA decorated with histones, neutrophil elastase, myeloperoxidase, and other granular proteins. Although initially identified as an antimicrobial defense mechanism, NETs are now recognized as key mediators linking inflammation and thrombosis. From a structural perspective, the DNA backbone of NETs acts as an extracellular polyanionic scaffold that supports multivalent protein binding, electrostatic interactions, and the retention of coagulation components, thereby promoting platelet adhesion, fibrin deposition, and thrombus stabilization.

Recent evidence indicates that specific DNA regions within NETs may directly interact with thrombin, while histones and neutrophil-derived enzymes amplify endothelial injury, platelet activation, and inflammatory signaling. These molecular events shift the hemostatic balance toward a prothrombotic state and contribute to thrombus persistence, resistance to fibrinolysis, and the progression of both venous and arterial thrombosis.

Clinically, NETs have been detected in thrombus specimens and are increasingly viewed as potential biomarkers and therapeutic targets in thrombotic disease. A deeper understanding of NET biology may therefore improve risk stratification and support the development of novel strategies for the diagnosis and treatment of thrombosis.