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Do neutrophil extracellular traps contribute to the heightened risk of thrombosis in inflammatory diseases?

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Abstract

Thrombotic events, both arterial and venous, are a major health concern worldwide. Further, autoimmune diseases, such as systemic lupus erythematosus, ANCA-associated vasculitis, and antiphospholipid syndrome, predispose to thrombosis, and thereby push the risk for these morbid events even higher. In recent years, neutrophils have been identified as important players in both arterial and venous thrombosis. Specifically, chromatin-based structures called neutrophil extracellular traps (NETs) play a key role in activating the coagulation cascade, recruiting platelets, and serving as scaffolding upon which the thrombus can be assembled. At the same time, neutrophils and NETs are emerging as important mediators of pathogenic inflammation in the aforementioned autoimmune diseases. Here, we first review the general role of NETs in thrombosis. We then posit that exaggerated NET release contributes to the prothrombotic diatheses of systemic lupus erythematosus, ANCA-associated vasculitis, and antiphospholipid syndrome

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