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Perspective

Targeting NETs for Therapeutic Development in Acute Lung Injury (ALI)

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DESCRIPTION

There is mounting proof that platelets play a significant role in inflammatory processes by forming close relationships with innate immune cells. Here, we show that the most common cause of mortality following transfusion treatment, transfusion-related acute lung injury (TRALI), causes neutrophil extracellular traps (NETs) to develop as a result of activated platelets. The production of NETs involves the activation of neutrophils and release of their DNA, a process that may or may not end in neutrophil mortality. NETs are made of decondensed chromatin adorned with granular proteins that serve to capture extracellular pathogens. NETs developed in the lung microvasculature and NET component levels rose in the plasma in a neutrophil and platelet dependent animal model of TRALI. We found NETs in the lungs, plasma, and individuals with acute lung damage who had TRALI in humans.

There is growing proof that platelets play a significant role in acute inflammation and damage in diseases such rheumatoid arthritis, cerebral malaria, and ALI. Furthermore, we have demonstrated that platelets play a crucial role in the murine TRALI model. Neutrophil-dependent platelet sequestration in the pulmonary microcirculation prevents mice from suffering severe lung damage and passing away. Plasma thromboxane synthesis reduces when platelet activation is inhibited by aspirin, along with lung damage and death.

Blood samples from the Clinic TRALI case-control study taken before and after the event. Blood was also drawn from healthy individuals and people with acute cardiac problems who were hospitalised to the University of California, San Francisco (UCSF) emergency room. All blood samples were centrifuged at 800 g for 10 minutes and immediately frozen at -80°C after being cooled as soon as possible. A gamma counter was used to measure the radiation.

During a 20-minute incubation period at room temperature in the dark, absorbance at a wavelength of 405 nm was measured. Results for the production of soluble NETs are provided as an increase in absorbance over control stated as a percentage. Hoechst 33342 was used to stain the DNA, and Vectashield Mounting Media (Vector Laboratories) was used to mount the cells for imaging with a Nikon 6D fluorescent microscopes with National Inpatient Sample (NIS) Elements for image collection (Nikon Imaging Center).

There is a critical need for ALI therapeutics that work. We suggest that NETs be a fresh focus for drug development in ALI. One therapy method focuses on blocking the early production of NETs; we argue that antiplatelet drugs (aspirin, tirofiban) are effective approaches. Targeting specific NET components is an alternate strategy that may be more practically practical because NETs may emerge early in conditions like ALI and sepsis and are already causing damage at the time of initial clinical presentation. We disrupted the scaffold using DNase1 and used a blocking antibody against H2A and H4 histones to target specific components (Deoxyribonucleic I). Both methods had a positive impact in lowering lung injury and death. When administered 5 minutes after the start of TRALI, when NETs are already present and lung damage is taking shape, DNase1 was similarly effective. We suggest that NETs are a promising target for future preclinical and clinical testing since a better knowledge of pathophysiology will probably lead to therapeutic advancements in ALI.

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