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Role of Neutrophil Extracellular Traps in Animal Models of Thrombosis

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Introduction

Hemostasis is a physiological host defense mechanism that prevents injury-related bleeding by keeping blood flow contained and under pressure. The cell and atomic peculiarity related with hemostasis is coagulation. The pathological process that arises from hemostasis is thrombosis. It is linked to numerous diseases, including cancer-related thrombosis, myocardial infarction, stroke, pulmonary and cerebral embolism, and phlebitis of the lower limbs. As a result, it is one of the leading causes of death worldwide. Different mechanisms, primarily those involving the subendothelium, endothelium, platelets, neutrophils, and, more recently, neutrophil extracellular traps (NETs), can initiate thrombus formation [1].

Description

A subpopulation of leukocytes known to play a significant role in initial immunity is neutrophils. In point of fact, neutrophils are the first cells to migrate to the site of infection or, at the very least, of the organism's recognized danger. There are numerous defence mechanisms in neutrophils. The most popular instrument, which is imparted to monocytes, is phagocytosis. Phagocytosis is a biological process in which a neutrophil engulfs, digests, and kills a bacterium after it has been identified; Its granules contain bactericidal proteins that drive these actions. Degranulation, also known as the release of proteins, is the straightforward second mechanism. The neutrophil is able to degranulate, or release its surface molecular components that were initially contained in its granules, once it is activated by the recognition of a threat. Myeloperoxidase, neutrophil elastase and cathepsin G are examples of some of these molecular components that are toxic and bactericidal. The generation of NETs is the final and most recently described mechanism. When neutrophils are subjected to excessive stimulation, significant intracellular modifications occur. They expel this ensemble into the extracellular environment by combining their granular content with their deoxyribonucleic acid (DNA).The purpose of this chromatin that has been adorned with neutrophilic serine proteases is to bind bacteria and prevent their spread throughout the body. Prior to platelets, neutrophils were the first cells present at the site of thrombosis following laser beam injury. Tissue factor (TF) is expressed on the surface of activated neutrophils, which also produce thrombin. The activation of platelets by thrombin results in the creation of platelet thrombi and fibrin. The presence of TF on the neutrophil surface appears to be the source of the procoagulant activity in this model. Neutrophils are involved in the formation of thrombosis and in the activation of the coagulation cascade in other models of thrombosis, such as the deep vein thrombosis (DVT) model. Finally, serine proteases found in neutrophils are thought to be able to hydrolyze the TFPI (tissue factor pathway inhibitor molecule) and, as a result, prevent the coagulation cascade's extrinsic activation pathway from occurring. The component proposed by the creators is that: (1) TFPI that is circulating and/or released by cell actors like platelets and the endothelium is recruited to the site of the injury, where it is then (2) incorporated into the thrombus that is forming, where it can be broken down by serine proteases from the neutrophils that are present.

The white lineage of blood cells makes up neutrophils. They are leukocytes that look like granulocytes and play a big role in immunity. They are the most common type of immune cells in human blood. The first cells or effectors recruited to the site of inflammation are neutrophils, which serve as the host first line of defense. In the beginning, neutrophils are formed in the bone marrow, where they remain for five to seven days. Neutrophils are immune cells that migrate to the infected target organ during an infection. Neutrophils, on the other hand, migrate to the liver and spleen for elimination when there is no infection and are released from the bone marrow into the general circulation of the blood. There, they patrol for six to nine hours [2].

Neutrophils are cells with a polylobed core fragmented into two to five curves with thick chromatin. There are a lot of granules in the clear cytoplasm. They are cells with a size of seven microns in mice and approximately fifteen microns in humans. The mechanisms of thrombosis are studied through the use of animal models. Depending on the species and the vascular area being targeted, numerous models have been developed. Mice, rats, rabbits, hamsters, guinea pigs, pigs, dogs and baboons are the primary species utilized for thrombosis research. For a number of reasons, the mouse is still the standard animal model for the study of cardiovascular disease: 1) It is a mammalian species that shares many physiological characteristics with humans; 2) It is cost-effective to house; 3) It is simple to manipulate; and 4) Its genome is simple to change, which makes it possible to create a large number of transgenic lines. Understanding the cellular and molecular mechanisms underlying the pathogenesis of thrombus formation makes use of animal thrombosis models. As a result, a variety of mouse preclinical models with various mechanisms exist: There are two vascular domains in which some induce denudation and others induce activation of the endothelium: veins and arteries [3-5].

Conclusion

This study reveals that, in addition to their immune activity, neutrophils have been shown to play a significant role in thrombosis, resulting in the phenomenon known as immuno thrombosis. Finally, neutrophils express the PDI protein on their surface, triggering the activation of neutrophil surface tissue factor and granting neutrophils triple coagulant activity. Neutrophils and NETs are strongly involved in thrombosis. The study of the composition of thrombi allows the design of new targeted and innovative therapeutic strategies to reduce mortality due to these thrombotic events.

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Conflict of Interest

The authors declare that there is no conflict of interest associated with this manuscript.

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