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Platelets as actors in inflammation and immunity: A fulcrum in immunity

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Abstract

Platelets are anucleated disc-shaped blood stem cells which play significant roles in modulating bleeding. Platelets play key roles in inflammation, infection and host response. Platelets release and accumulate adhesion molecules at infection sites and trigger inflammatory reactions with the aid of these molecules.Platelets, or thrombocytes, are small, colorless cell fragments in our blood that form clots and stop or prevent bleeding. Platelets are made in our bone marrow, the sponge-like tissue inside our bones. Platelets are highly reactive blood cells which play key roles in maintaining homeostasis.When activated, platelets have been seen to have a significant impact on the start and continuation of inflammatory responses. It is now known that active platelets regulate local inflammatory responses, such as touch sensitivity, inflammatory bowel disease, and atherosclerosis, by interacting with endothelial cells and infiltrating leukocytes through platelet-derived immunomodulatory ligands.

Keywords: platelets, inflammation, immunity, cytokines, chemokines

Introduction

Platelets are anucleated disc-shaped blood stem cells which play significant roles in modulating bleeding(Sonmez and Sonmez, 2017). According to earlier studies, platelets play key roles in inflammation, infection and host response. Platelets release and accumulate adhesion molecules(Obeagu *et al.*, 2022; Obeagu *et al.*, 2014; Igweand Obeagu, 2018; Obeagu *et al.*,2017; Okoroiwu *et al.*,2022) at infection sites and trigger inflammatory reactions with the aid of these molecules (Morrell *et al.*, 2014). After secreting adhesion molecules at the damaged or wounded site, platelets get attached to white blood cells including neutrophils, lymphocytes and monocytes. Subsequently, platelets release chemotactic factors including cytokines and chemokines to target and direct lymphocytes, neutrophils and monocytes to the damaged site to trigger inflammation(Sonmez and Sonmez, 2017).In response to injury and inflammation, blood platelets gather at the site of the wound to support haemostasis (Obeagu *et al.*, 2022; Obeagu *et al.*, 2022; Obeagu *et al.*, 2022; Obeagu *et al.*, 2023). A rising corpus of studies suggests that platelets are considerably more than just simple cell fragments that plug holes and occasionally dislodge to produce thrombosis. Maintenance of vascular barrier integrity is itself a critical component of host defense against infection (Cleary and Conrad, 2023).

Platelets

Platelets, or thrombocytes, are small, colorless cell fragments in our blood that form clots and stop or prevent bleeding. Platelets are made in our bone marrow, the sponge-like tissue inside our bones. Platelets are highly reactive blood cells which play key roles in maintaining homeostasis(Obeagu *et al.*, 2023; Obeagu and Obeagu, 2023; Ukonu *et al.*, 2023). Platelets are formed from megakaryocytes. Megakaryocytes are produced from multipotent hematopoietic stem cells toward megakaryocyte progenitors(Nishimura *et al.*, 2015).The anucleate structures of platelets, which are discoid in shape have alpha granules, numerous secretory granules, thick granules, and lysosomal granules, of which three types of secretory granules are identified. Each granule is made up of secretory elements like platelet-derived growth factor (PDGF), insulin-like growth factor-1 (IGF1), hepatocyte growth factor (HGF), vascular endothelial growth factor (VEGF), serotonin, adenosine diphosphate (ADP), adenosine triphosphate (ATP), epidermal growth factor (EGF) and transforming growth factor-beta (Ifeanyi *et al.*, 2022; Obeagu and Obeagu, 2015; Obeagu, 2022; Okoroiwu *et al.*,2021; Okoroiwu *et al.*, 2014). Active chemicals are released from the granules of platelets when they are stimulated by different types of stimulation(Murata *et al.*, 2014; Suzuki *et al.*, 1996).

Platelets have additional roles in regulating inflammatory reactions by facilitating the synthesis of chemokines, cytokines(Obeagu *et al.*, 2022; Obeagu *et al.*,2017; Obeagu *et al.*, 2023; Oke *et al.*,2022; Emmanuel *et al.*, 2017; Obeagu and Obeagu, 2015; Okoroiwu *et al.*, 2021) and immunomodulatory ligands such as CD154 (Iannacone, 2016). When activated, platelets have been seen to have a significant impact on the start and continuation of inflammatory responses(Yearman, 1997). It is now known that active platelets regulate local inflammatory responses, such as touch sensitivity, inflammatory bowel disease, and atherosclerosis, by interacting with endothelial cells and infiltrating leukocytes through platelet-derived immunomodulatory ligands (Ahmad *et al.*, 2001; Gawaz *et al.*, 2000). Platelets release chemokines and cytokines such as IL-8, RANTES, MCP-3, cationic proteins, and sticky proteins when they are activated. As they adhere to endothelial cells, platelets also trigger the production of a wide range of inflammatory molecules such as IL-6, IL-8, GM-CSF, and MCP-1 (Klinger, 1997).

Platelets and innate immunity

The process by which blood arteries are closed after an injury is known as hemostasis, and tiny platelets anucleate cells, play a key part in this process. As the body's initial line of defense against infections, innate immunity, and platelets also play a crucial part in this system (Li and Wang, 2015).

Direct contact with pathogens is one of the main ways that platelets support innate immunity. Toll-like receptors (TLRs), which platelets express, and other pattern recognition receptors, enable them to recognize the presence of pathogens in the circulation (De Stoppelaar*et al.,* 2014). Platelets can trigger a range of immunological responses in response to pathogen detection, such as the creation of antimicrobial peptides and the release of cytokines and chemokines that draw more immune cells to the site of infection (Koupenova and Freedman, 2019).

The interaction of platelets with other immune cells might potentially indirectly support innate immunity. Neutrophils, for instance, are crucial mediators of the innate immune response and may attach to and be activated by platelets (Semple*et al.,* 2011). The activation of neutrophils and their migration to the infection site are caused by platelet P-binding selectin to neutrophil PSGL-1 (Koupenova*et al.,* 2018).

Platelets can help regulate the inflammatory response, according to recent studies (Cloutier et al.,2018). Transforming growth factor beta (TGF-) and interleukin-10 (IL-10) are only a couple of the anti-inflammatory chemicals that platelets have been shown to produce, which may assist to reduce inflammation and preventing tissue damage (Sánchez *et al.,* 2017). Moreover, dendritic cells, which are essential for the start of the adaptive immune response, can be activated by immune cells like platelets, which can be controlled by platelets (Koupenova*et al.,* 2018).

Hence, by directly interacting with pathogens, stimulating other immune cells, and controlling the inflammatory response, platelets play a crucial role in innate immunity. New medicines for the treatment of infectious illnesses might be developed as a result of more studies in this field (Cloutier*et al.,* 2018).

Platelets and adaptive immunity

Small, anucleate blood cells known as platelets have recently come to be recognized for their function in thrombosis and hemostasis (Cloutier*et al.,* 2018). Yet it's now understood that platelets play a crucial function in the immune response as well, notably in adaptive immunity. It has been demonstrated that platelets interact with a wide range of immune cells, such as T cells, B cells, dendritic cells, and monocytes, and that platelets can influence the activation, proliferation, and differentiation of these cells (Huang *et al.,* 2013). The ability of platelets to interact with T cells and influence how they operate is one of the most exciting discoveries. Major Histocompatibility Complex (MHC) class I and class II molecules are expressed on platelets and are necessary for CD8+ and CD4+ T lymphocytes to present antigens, respectively (El-Behi*et al.,* 2012). Moreover, the expression of co-stimulatory molecules in platelets, such as CD40, CD80, and CD86, is essential for T-cell activation (Smyth, 2009). Moreover, it has been demonstrated that platelets release several chemokines and cytokines that attract and activate T lymphocytes (Niewold*et al.,* 2016).

Platelets can also regulate the differentiation and function of T cells. Platelets have been shown to induce the differentiation of naïve CD4+ T cells into Th17 cells, which play a crucial role in the defense against bacterial and fungal infections (Smyth, 2009). Platelets have also been shown to inhibit the function of regulatory T cells (Tregs), which suppress immune responses and maintain immune tolerance (Koupenova*et al.,* 2018). The inhibition of Tregs by platelets may contribute to the development of autoimmune and inflammatory diseases.

Platelets can also work with B cells to promote activation and differentiation. For B cell activation and differentiation into antibody-secreting plasma cells, the co-stimulatory molecule CD154 (CD40L), which is present on platelets, is necessary (Zarbock, 2007). It has also been demonstrated that platelets release B cell activating factor (BAFF), which encourages B cells' survival and growth (Li *et al.,* 2015).

The formation of immunological memory has also been demonstrated to be influenced by platelets. Memory T cells and platelets can interact to

influence the function and survival of the latter (Zarbock, 2007). It has also been demonstrated that platelets deliver antigens to memory T cells, which may aid in the preservation of immunological memory (Koupenova and Freedman, 2019).

Finally, platelets are critical for adaptive immunity. They are not merely basic blood cells engaged in hemostasis and thrombosis (Koupenova and Freedman, 2019). They can engage in interactions with numerous immune cells, influence their activity, and support the formation of immunological memory. The mechanisms underpinning platelet-mediated immune responses, as well as their consequences for health and illness, still require more study (Sánchez *et al.*, 2017; Huang *et al.*, 2013).

Inflammation:

Inflammation is a complex of responses of the innate immune system to pathological stimuli such as microbes, pathogens or damage-associated molecular patterns (DAMPs). Local inflammation includes the following classical symptoms: dolor (pain), calor (heat), rubor (redness), tumor (swelling) and functiolaesa (loss of function). Systemic inflammation occurs in different contexts like massive trauma, chronic disease, or as a response to an infection, in which case it is designated as sepsis (Mònica *et al.*, 2015). It is the immune system's response to harmful stimuli, such as pathogens, damaged cells, toxic compounds, or irradiation, and acts by removing injurious stimuli and initiating the healing process. Inflammation is therefore a defense mechanism that is vital to health. Usually, during acute inflammatory responses, cellular and molecular events and interactions efficiently minimize impending injury or infection. This mitigation process contributes to restoration of tissue homeostasis and resolution of the acute inflammation. However, uncontrolled acute inflammation may become chronic, contributing to a variety of chronic inflammatory diseases (Chen *et al.*, 2018). Most of the features of acute inflammation continue as the inflammation becomes chronic, including the expansion of blood vessels (vasodilation), increase in blood flow, capillary permeability and migration of neutrophils into the infected tissue through the capillary wall (diapedesis). However, the composition of the white blood cells changes soon and the macrophages and lymphocytes begin to replace short-lived neutrophils. Thus the hallmarks of chronic inflammation are the infiltration of the primary inflammatory cells such as macrophages, lymphocytes, and plasma cells in the tissue site, producing inflammatory cytokines, growth factors, enzymes and hence contributing to the progression of tissue damage and secondary repair including fibrosis and granuloma formation, etc(Jialal., 2022). In response to foreign or self-antigens, the tissue immune cells such as macrophages and dendritic cells release cytokines such as IL-1 and TNF-α. These cytokines induce the injury-site-endothelial cells to release Selectins and Integrins which stimulate chemotaxis and diapedesis of the circulating leukocytes. In addition to the recruitment of leukocytes, the tissue macrophages, and dendritic cells also play a role in the clearing of the antigen by phagocytosis, the release of cytokines and serving as antigen-presenting-cells to lymphocytes (Jialal., 2022)

Platelets and Inflammation

Haemostasis is a physiological process that maintains the fluidity of the blood and prevents bleeding during vascular injury. The key role of platelets in haemostasis and thrombosis has been documented for many years (Ebermeyer *et al.*, 2021). Multiple therapeutic targets have been identified and used in the development of anti-thrombotic drugs. However, work published in recent years shows that the role of platelets is not confined to maintaining vascular integrity and thrombosis as they play an important role in inflammation, cancer dissemination, wound healing and the separation of blood and lymphatic vessels during development. Platelets impact inflammation and the innate immune response on several levels (Ebermeyer *et al.*, 2021). They express toll-like receptors (TLRs) involved in the innate immune response and may thereby contribute to the response to infections by

secreting a number of inflammatory mediators. Several receptors on the platelet surface recognise ligands present on monocytes and neutrophils, resulting in the formation of circulating leuko-platelet aggregates. Activated platelets can secrete chemokines that contribute to monocyte recruitment or macrophage differentiation. The interaction of platelets with neutrophils via PSGL-1, a P-selectin ligand, is key in initiating the innate inflammatory response and neutrophil extravasation. Platelet interactions with endothelial cells during infection also condition monocyte migration to the site of inflammation (Ebermeyer *et al.,* 2021).

The CD40 ligand (CD40L) produced by platelets induces an inflammatory response in the endothelium. Indeed, CD40L can cause endothelial cells to produce reactive oxygen species and express adhesion molecules, chemokines and tissue factor. Unlike CD40L, which is stored in platelets, IL-1β is synthesised during platelet activation. IL-1β production is sufficient to induce endothelial cells to express genes involved in leukocyte adhesion. IL-1β activates endothelial cells causing increases in chemokine secretion and in the expression of molecules that trigger neutrophil and monocyte adhesion to the endothelium (Aoui *et al.,* 2014). Thus, the involvement of platelets in inflammation is a dynamic process involving various mechanisms. It is important to note that this phenomenon is reciprocal since reference has been made to the pleiotropic role of leukocytes in haemostasis and thrombosis known as “immunothrombosis”. The importance of the intimate relationship between platelets and inflammatory cells is found in several diseases, including neurodegenerative diseases, atherosclerosis, acute coronary syndrome, rheumatoid arthritis and lupus (Puhm *et al.*, 2021).

Conclusion

Platelets are anucleated disc-shaped blood stem cells which play significant roles in modulating bleeding. Platelets play key roles in inflammation, infection and host response. Platelets are highly reactive blood cells which play key roles in maintaining homeostasis. When activated, platelets have been seen to have a significant impact on the start and continuation of inflammatory responses. It is now known that active platelets regulate local inflammatory responses, such as touch sensitivity, inflammatory bowel disease, and atherosclerosis, by interacting with endothelial cells and infiltrating leukocytes through platelet-derived immunomodulatory ligands.

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