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Short Communication

Host-environment interface, host defense, and mast cell: autoimmunity, allergy, inflammation, and immune response

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Submitted: 30 April 2021

Accepted: 13 June 2021

Published: 16 June 2021

ISSN: 2379-9498

Abstract

Numerous factors such as environmental, physiological, and genetic factors have a deep association with mast cells. These cells not only govern the immune response but also enhanced the defense mechanism naturally and prepared a host defense system. These cells recognized pathogens via the involvement of adhesion and immune receptors. Mast cells can act as inflammatory cells and their role in allergic disease, autoimmunity, propagation of diseases, and exacerbation of various types of diseases explored. These cells promote the recruitment of neutrophils in the location of autoimmune destruction at the time of origin of diseases. Mast cells displayed a potential role in various biological routes and processes such as the secretion of cytokines, release enzymes, acquiring immunity against parasitic infections, in the process of fibrosis, allergy, inflammation, and the pathology of autoimmune diseases.

BACKGROUND

Dr. Paul Ehrlich was awarded the Nobel Prize in 1908 for the discovery of Mast cells. Earlier perception about the biological role of these cells was that mast cells only mediate allergy routes and participate in wound healing. Later on, varied scientific studies revealed that these cells also governed the phenomena of inflammation, control of infectious, and act as a key component in host defense mechanism.[1] These cells also deal with disease progression. Furthermore, mast cells are classified according to their features i.e. phenotypic characteristics and their anatomic locations. Reber et al. expressed the phenotypic characteristics of mast cells and subcategorized them into tryptase-positive, tryptase- and chymase-positive mast cells. This classification of mast cells was supported by their functioning styles too.[2] As tryptase- and chymase-positive mast cells displayed their affiliations with small intestinal submucosa and muscular is mucosa. Though, tryptase-positive mast cells have the potential to inhibit the mucosa of the stomach, colon, and small intestine. Few components of cellular systems and environment such as chemokines, nerve growth factor cytokines, and transforming growth factor-β (TGF-β) controlled mast cell physiology and survival. The process of activation of these cells depended on i.e. high-affinity IgE receptor and independent manner. The activation occurred via the routes initiates various specific signaling cascade mechanisms. These phenomena initiate more biochemical transformations such as intracellular calcium influx, and activation of certain transcription factors, including cytokine production, mast cell degranulation.[3] Apart from the activities induced by high-affinity IgE receptor, the β2-integrin, serotonin receptor, and intracellular adhesion molecule-1, the surface markers are other components of mast cells that support these cells for responding toward diverse stimuli. During the process of degranulation, mast cells unconfined lipid mediators and cytokines.[4] Simultaneously, these cells stored histamine, proteases, and heparin as well as promote neighboring tissues in storing the bioactive substances containing cytoplasmic lipid bodies and granules. Simultaneously, the process of release of these mediators is also dependent on several factors.

The anatomic residency and location of mast cells were fixed by specific cell subtypes and marked them to build a defense and enhance the immune system of the host [Fig.1]. At the time of an attack of microbial, while initiating the infection, mast cells immediately respond to it and act as innate immune cells. Therefore, mast cells deal with the different routes of allergic reactions and are also identified as the initiator of the immune response. Observed scientific shreds of evidence in the area of autoimmunity also underlined some demerits of these cells, and it was believed that sometimes, these cells can exacerbate disease while initiating the immune response for preventing infection.[5] The pathogenesis of multiple sclerosis, and rheumatoid arthritis illustrated here as an example. The role of mast cells and the route of their participation elaborated at the time of infection and discussed here. Similarly, the role of mast cells in infection is conferred, but it displayed different styles of functioning.

Herein, mast cells recruit neutrophils when any inflammation occurred. It produces a separate subset of immune responses. [6] The neutrophil response at the site of infection is defensive and protects the cellular compartments but during inflammation, therefore the role of mast cells varied every so often. It was observed that both types of blood components i.e. mast cells and neutrophils altogether enhanced tissue destruction at target sites by stimulating local vascular permeability. This phenomenon supported the inflammatory cells in the aforementioned activities. Besides it, there is little evidence available in the literature that can prove the participation of these cells in enhancing the disease. Herein, the role of mast cells in autoimmune disease is separately discussed. The route of mast cell actions and factors that modulate these cells functioning style in autoimmunity also elaborated in the next section.

OBSERVATIONS

Numerous genetic and environmental factors induce autoimmune diseases in the host because of the host-environment interface by evading host defense. The participation of mast cells elaborated here in the origin of an autoimmune disease, which can be defined as autoimmunity. These phenomena can also initiate allergy by enlarging the inflammation by suppressing its natural immune response [Fig. 2]. A literature survey confirmed that a definite route of etiology of these diseases was not yet discovered, but it was also predicted that mast cells had displayed some certain association of the pathways of the origin of the autoimmune disease with environmental, physiological, and genetic factors.[7] These observations will be helpful to clear the persistent confusion. The route and pathways of these diseases are interlinked with the pathology of mast cells. It was believed that mast cells could enhance autoimmune diseases via the secretion of the cytokine. As, mast cells have cytoplasmic granules and have the potential to release the enzyme, therefore, by releasing these types of enzymes these cells can easily propagate any autoimmune disease. Moreover, these cells can secrete proinflammatory mediators that also be harmful to healthy cells and can easily enhance the possibilities for further promotion of such activities of these mediators to stimulate the spreading of autoimmune diseases.[8] Therefore, it is an excellent area of research to be underlined for further investigation to explore the role of mast cells in the propagation of autoimmune diseases. It was also reported that these cells are also participating and influencing the hypersensitivity responses. Besides it, the role of mast cells in the origin of the route of allergic disease and their participation in inflammatory conditions is also illustrated.

The author tried his best by putting stress on exploring different aspects of mast cells and underlined a few of them and marked various other involvements of these cells in different routes such as their involvement in inhibiting bacterial infection, intensifying autoimmunity, their participation in atherosclerosis, and path of promoting cancer progression for further research. Besides all, mast cells inhibit the immune response via interleukin-10 production. The mast cells stimulate the migration of many cell types by producing inflammatory cytokines, for example, in the pathways of tumor necrosis factor. Mast cells are actively engaged with those cellular events, which transpired tissue damage during autoimmune disease. The immune system always governs cellular networks for regulating immune homeostasis properly in a normal environment. When the foreign antigen attacks, the immune responses counter it for restoring normal homeostasis to maintain normal physiological conditions. [9] In adverse conditions, proper functioning of the immune response lost its natural flow and, as a result, this phenomenon got overexcited or under reacted. In both conditions, the immune response-enhanced pathology followed infection. It was defined as an immune disorder that can initiate autoimmune disease (multiple sclerosis) and enhance viral infection [Fig. 3]. Dysregulation of immune response must be treated well by the immunotherapies for restoring immune homeostasis for proper functioning.[10] This interpretation encouraged researchers to determine the significant reason for the query, how mast cells participate in immune homeostasis. Moreover, a lot of proof is available that confirmed the role of mast cells in autoimmune disease. The propagation of these fatal events was reinforced by mast cells.[11] In many cellular events occurring at the time of origin of human autoimmune diseases (multiple sclerosis, and rheumatoid arthritis), and the same was observed. Mast cells also participated in the progression of these diseases.

Overall, antibodies, intrinsic danger signals, and pathogens are among those receptors that can be associated with mast cells and can propagate disease. Besides, mast cells initiate responses for dealing with infection settings by requiring neutrophils, the immune cells, at the location of the autoimmune destruction. Herein. It can be stated that the potent immune cells enhanced autoimmunity and by targeting them by any therapeutic, the propagation of the disease can be stopped.[12] Mast cells not only govern homeostasis via degranulation but also produced IgE-mediated allergic reactions, and perform as a bridge between innate and adaptive immune responses. Rheumatoid arthritis and multiple sclerosis are those diseases that are considered under the category of chronic inflammatory diseases [Fig. 4].[13] Recent advances identified mast cells to be targeted by remedies for treating these diseases. An advanced remedy, Tyrosine-kinase inhibitors, was recently prescribed for treating these diseases and are very effective too. Multiple sclerosis is defined as a neurological autoimmune disorder that persisted in the central nervous system. It was testified by the presence of inflammatory demyelination and subsequent axonal deterioration. The existence of aforesaid disease proved by testing of demyelinated plaque, a hypo-cellular area, which can easily be marked because of the formation of astrocytic scars that occurred within the region. The infiltration of mononuclear cells is the process that transpired at the time of disease propagation. It was also reported that the innate and adaptive immune cells also contribute to the progression of the pathogenesis of these diseases. The route of formation of neuroinflammation was initiated by mast cells by upholding demyelination via interactions.[14] These cells also promote neurons, and other immune cells to be part of these propagation routes. It was also evidenced that mast cells disrupt the blood-brain barrier, and permit the entry of inflammatory cells and mediators to nervous systems.

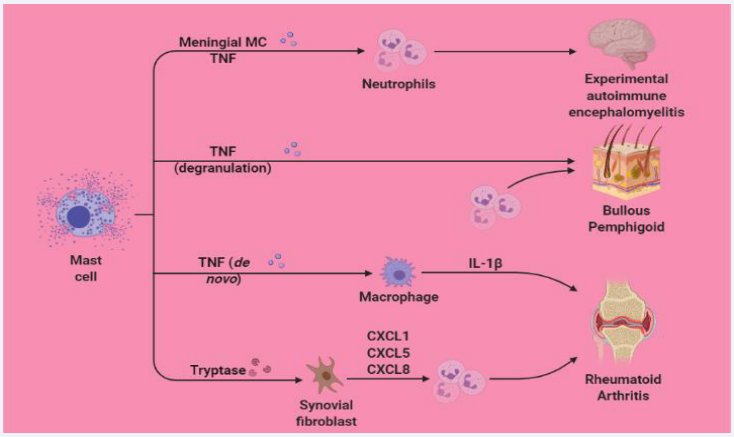

Figure 1 Illustrate the phenomenon and role ole of mast cells in autoimmune disease models. "Adapted and created with permission from [biorender.com] and acknowledged.

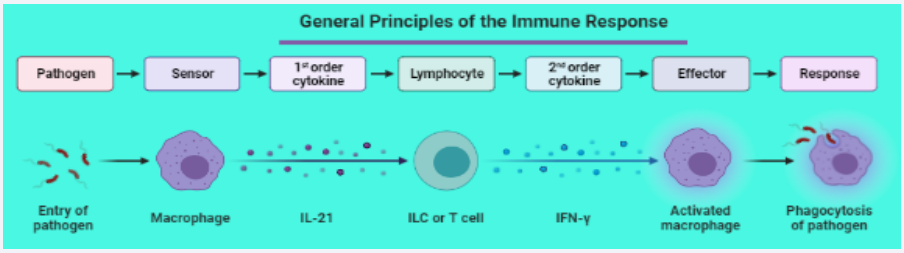

Figure 2 Illustrate the phenomenon of general principles of the immune response. "Adapted and created with permission from [biorender.com] and acknowledged.

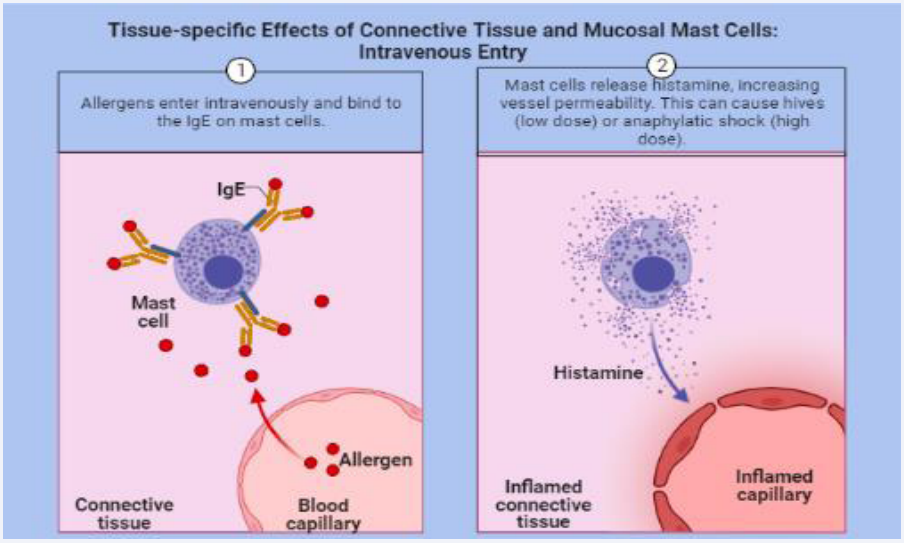

Figure 3 Illustrate the phenomenon of tissue-specific effects of connective tissue and mucosal mast cells: intravenous entry. "Adapted and created with permission from [biorender.com] and acknowledged.

Adopting the process of depletion or controlling the concentration of these cells can be an effective method to treat rheumatoid arthritis and multiple sclerosis.[15] The understanding of the molecular mechanism underlying can provide a route that can be followed at the time of designing the strategies that can be prescribed in treating autoimmune and inflammatory diseases.

CONCLUSION

Mast cells can also be defined as effector cells, which play a

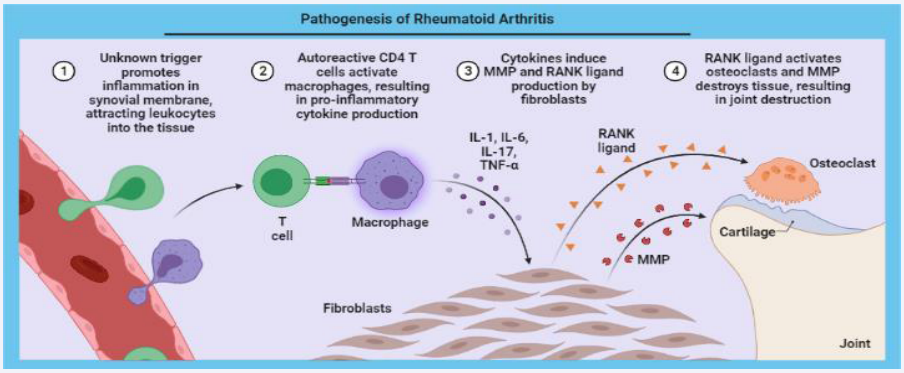

Figure 4 Illustrate the phenomenon of the pathogenesis of rheumatoid arthritis. "Adapted and created with permission from [biorender.com] and acknowledged.

key role in the recovery of tissue-injury, initiate via infectious agents, toxins, and metabolic states. These cells displayed their character as pro- to anti-inflammatory, in both the ways. According to the need of the physiological response, mast cells demonstrate their role, it could be in favor of inflammation for repair or destruction. Therefore, dysregulation of these cells leads to various pathology of the disease. Mast cells have pro-inflammatory mediators, and they can release it with the following degranulation. During atopic dermatitis, psoriasis, multiple sclerosis, and other inflammatory diseases, these cells participated in cross-talk with T cells. Any stress condition, these cells will further degrade the physiological conditions. It was also evidenced that mast cells also have immunomodulatory properties and can perform immunosuppressive actions. The mast cell inhibitors can be applied in the development of novel therapeutic applications. The hypersensitive immune responses initiate autoimmune and allergic diseases.

ACKNOWLEDGMENT

One author (Rajiv Kumar) gratefully acknowledges his younger brother Bitto. The author acknowledges bio render to illustrate the online diagram (**Figure 1, 2, and 3**). The authors acknowledge km. Babita Pundir, Host of FM Rainbow 107.1 MHz, Bombay, India for the kind support and encouragement.

REFERENCES

1. Rajiv K. Elucidation of the origin of autoimmune diseases via computational multiscale mechanobiology and extracellular matrix remodeling: theories and phenomenon of immunodominance. Curr Med Drug Res. 2021; 5.
2. Rajiv K. Cell Shrinkage, Cytoskeletal Pathologies, and Neurodegeneration: Myelin Sheath Formation and Remodeling. Arch Med Clin Res. 2021; 1: 1–5.
3. Kumar R, Chhikara BS, Gulia K, Chhillar M. Cleaning the molecular machinery of cellsviaproteostasis, proteolysis and endocytosis selectively, effectively, and precisely: intracellular self-defense and cellular perturbations. Molecular Omics. Royal Society of Chemistry; 2021. 11–28.
4. Taylor ML, Metcalfe DD. Mast cells in allergy and host defense. Allergy and asthma proceedings. . 2001; 22: 115–119.
5. M. F. Neuroimmunology and inflammation: Implications for therapy of allergic and autoimmune diseases. Ann Allergy Asthma Immunol. 2003; 90: 34–40.
6. Gri G, Frossi B, D'Inca F, Danelli L, Betto E, Mion F, et al. Mast cell: An emerging partner in immune interaction. Front Immunol. . 2012.
7. Lalani I, Bhol K, Ahmed AR. Interleukin-10: Biology, role in inflammation and autoimmunity. Ann Allergy Asthma Immunol. 1997; 79: 469–483.
8. Weller CL, Collington SJ, Williams T, Lamb JR. Mast cells in health and disease. Clinical Sci (Lond). 2011; 120: 473–484.
9. Morita H, Saito H, Matsumoto K, Nakae S. Regulatory roles of mast cells in immune responses. Semin Immunopathol. 2016: 38: 623–629.
10. Gilfillan AM, Beaven MA. Regulation of mast cell responses in health and disease. Crit Rev Immunol. 2011; 31: 475–529.
11. Rao KN, Brown MA. Mast cells: Multifaceted immune cells with diverse roles in health and disease. Ann N Y Acad Sci. 2008; 1143: 83–104.
12. Kumar R, Chhikara BS, Gulia K, Chhillar M. Review of nanotheranostics for molecular mechanisms underlying psychiatric disorders and commensurate nanotherapeutics for neuropsychiatry. Nanotheranostics. 2021; 5: 288–308.
13. Kumar R, Gulia K. The convergence of nanotechnology-stem cell, nanotopography-mechanobiology, and biotic-abiotic interfaces: Nanoscale tools for tackling the top killer, arteriosclerosis, strokes, and heart attacks. Nano Sel. 2021; 2: 655–687.
14. Dietrich N, Rohde M, Geffers R, Kröger A, Hauser H, Weiss S, et al. Mast cells elicit proinflammatory but not type I interferon responses upon activation of TLRs by bacteria. Proc Natl Acad Sci U S A. 2010; 107: 8748–8753.
15. Ohno T, Nakamura T, Nakae S, Morita H, Matsumoto K, Saito H, et al. TSLP is a negative regulator of RANKL-induced osteoclastogenesis. Biochem Biophys Res Commun. 2020; 530: 508–512.

Cite this article

Rajiv Kumar (2021) Host-environment interface, host defense, and mast cell: autoimmunity, allergy, inflammation, and immune response. J Clin Pharm 5(1): 1018.