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Research Article

Inflammatory Response: An Update Overview of a Traverse between Health and Disease

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Abstract:

The inflammatory process is an important response to injury, infection, trauma, and many other insults. It is made up of a detailed cascade of both pro-inflammatory and anti-inflammatory mediators. The balance between these mediators determines a disease development. Further understanding of the endogenous mechanisms that control the inflammatory response is required to facilitate development of therapeutic options. In this review, we discuss the current knowledge of the mechanisms leading to development of acute and chronic inflammatory response as well as the factors that regulate this response.

Keywords: Inflammation, Mechanism, Acute, Chronic, Diseases

Introduction:

Inflammation was originally derived from the latin word 'inflammare' meaning burned. It is a protective response usually triggered by noxious stimuli which helps to eliminate pathogens, repair damaged tissue, and to restore homeostasis.1,2 Several factors can cause inflammatory process such as microbial, viral, or fungal infection, tissue damage, or autoimmune reactions.3 The inflammatory process are well-known for hundreds of years and are recognized clinically by five cardinal signs which includes pain (dolor), warmth or heat (calor or hyperthemia) redness (rubor), swelling of tissues (tumor), and loss of function laesa (laesa).4,5 These signs are as a result of immunological, biochemical and physiological changes following injury, trauma or foreign invasion, triggering the release of chemical mediators at the injured site, thus increasing blood flow, vascular permeability, and recruitment of leukocytes. Chemicals including histamine, bradykinin, and prostaglandins are release by the damaged cells. These chemicals cause blood vessels to leak fluid into the tissues, causing swelling, and this helps isolate the foreign substance from further contact with body tissues.

Types of Inflammation

Inflammation can be of two major types i.e. acute or chronic.6-9 Acute inflammation is an early but a short term response in the host body produced by cells of the innate immunity. It may occur in seconds or for some days or weeks. Acute inflammation starts after the release of a specific injury causing soluble mediators like cytokines, acute phase proteins, and chemokines which promote the migration of neutrophils and macrophages to the site of injury.10,11

If this does not resolve after six weeks, it is termed chronic form of inflammation. Chronic inflammation could cause harmful effects on host and is characterized by the invasion leukocytes (the primary inflammatory cells), secreting inflammatory cytokines, development factors and proteins.[12,17] Excessive or uncontrolled inflammation is devastating for normal homeostatic processes of the body. Most of the modern human diseases such as asthma, allergy, autoimmune diseases, hepatitis, coeliac disease, inflammatory bowel disease and glomerulonephritis are associated directly or indirectly to different inflammatory processes. [18-26]

Causes of Inflammation

The causes of inflammation are classified into two main groups: exogenous and endogenous factors. [19]

1. Exogenous Factors.

This could further be subdivide into two classes; microbial and non-microbial exogenous factors.

A. Microbial Factors.

There are two classes of microbial inducers of inflammation. The first class is pathogen-associated molecular patterns (PAMPs), which are carried by all microorganisms and are easily recognized as foreign by the host while the second class is virulence factors which are restricted to pathogenic microbes.[22]

B. Non-Microbial

Non-microbial causes of inflammation include allergens, toxic compounds, irritants, and foreign bodies that are too large to be digested or cause phagosomal damage in macrophages. Examples of foreign bodies include silica and asbestos, as well as signals released by damaged cells and tissues.[27]

2. Endogenous Factors.

These are substances released by dead, damaged, malfunctioned, or stressed tissues. They could be divided into two groups namely the infectious factors and the non-infectious factors.

A. Infectious factors:

This category includes substances released by bacteria, viruses, and other microorganisms.

B. Non-Infectious factors:

This involves physical injuries such as burns and exposure to substances like ionizing radiation, chemical compounds such as toxins, alcohol, and chemical irritants such as nickel and other trace elements.

Mechanism of Inflammation

Inflammation is a complex biochemical process involving secretion of chemical agents by immune cells like cytokines, chemokine, histamine, bradykinin, prostaglandins and reactive oxygen species which cause blood vessels to leak fluid into the tissues, resulting in swelling.[28-33] This helps isolate the foreign substance from further contact with body tissues. These substances are synthesized locally by cells at the site of inflammation or produced by the liver to circulate in the plasma as inactive precursors, most of which are activated by the contact with specific receptors at the site of inflammation.[34.35] While some mediators have direct enzymatic activity, others may cause oxidative damage to the foreign substance and surrounding tissues. Although inflammatory response depends on the exact nature of the initial stimulus and its location in the body, they all share a common mechanism which is summarized as follows: 1) cell surface pattern receptors recognize detrimental stimuli; 2) inflammatory pathways are activated; 3) inflammatory markers are released; and 4) inflammatory cells are recruited.[36,37]

Mechanism of Acute Inflammation

Many mediators play a pivotal role in initiating the cascade in the acute inflammatory process. The first group of mediators is the toll-like receptors (TLRs), which are membrane-spanning proteins found on the surfaces of the innate immune system cells like macrophages and dendritic cells.[38] They recognize the pathogen-associated molecular patterns (PAMPs) or danger-associated molecular patterns (DAMPS).The second group of mediators are arachidonic acid (AA) which act through the cyclooxygenase pathway.[39,40] Arachidonic acid is a phospholipid that constitutes the membrane of the body's cells. The third group is the Mast cells which are derived from precursors in the bone marrow and are widely distributed in the connective tissues. These cells become activated when there is tissue damage. The fourth group is the complements. Complements are a set of proteins that interact with one another creating a cascade that forms membrane attack complex which eliminates pathogens.[34] Complements can become activated through several pathways like the classical, alternative, or mannose-binding lectin pathways.[41]

Mechanism of Chronic Inflammation

In response to foreign or self-antigens, the tissue immune cells such as macrophages and dendritic cells release cytokines. These cytokines induce the injury-site-endothelial cells to release Selectins and Integrins which stimulate chemotaxis and diapedesis of the circulating leukocytes.[22] 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.10 Once the circulating leukocytes enter the local injury site, they are activated by various cytokines and chemokines secreted by the macrophages and dendritic cells. On activation, the leukocytes further release cytokines and mediators of inflammation.

Resolution of Inflammation

To prevent progression from acute inflammation to chronic inflammation, the inflammatory process must be suppressed to prevent tissue damage. Inflammation resolution is a well-managed process involving the spatially- and temporally-controlled production of mediators, during which chemokine gradients are diluted over time and presents a good therapeutic target for diseases.[42-45] During resolution, circulating white blood cells eventually no longer sense these gradients and are not recruited to sites of injury.[44] Dysregulation of this process can lead to uncontrolled chronic inflammation and development of diseases.

Conclusion

Inflammatory response is a common feature that explains the pathologies underlying the development of diseases. In this sense, understanding the inflammatory processes will allow for the development and production of improved targeted therapies.

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