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Perspective

Innate Immune Mechanisms of Tissue Injury

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DESCRIPTION

Inflammation is a process by which body's white blood cells and the things they make protect from the infection from outside invaders, such as bacteria and viruses. But in some diseases, like arthritis, body's defense system triggers inflammation when there are no invaders to fight off. In these autoimmune diseases, immune system acts as if regular tissues are infected or somehow unusual, causing damage. Inflammation is a reaction to infections and tissue damage. Inflammation was previously defined by the clinical manifestations, later by the presence of leukocytes, and now by the expression of "inflammatory" cytokines and chemokine. However, white blood cells and cytokines often have more anti-inflammatory, regeneration-promoting, and homeostatic effects. The capabilities of the "inflammatory" innate immune system mediator/regulator, which determines the tissue environment to meet tissue needs while re-establishing post-injury homeostasis. Inflammation is a reaction to infections and tissue damage. Inflammation is one of the most important controlled programs of histopathology conserved in the process of evolution so far, and its main purpose is to remove infection, repair tissue damage, and maintain tissue homeostasis. This is a very complex but very well coordinated process, classically caused by infections and tissue damage. Historically, "inflammation" was originally defined as an early component of the healing process after tissue injury by Hippocrates. This definition was challenged by the discovery of the microscope in the 19th century, and the microscopic presence of leukocytes at the site of infection or injury has since been called "inflammation". However, this simple definition of "inflammation" no longer applies in the 21st century, largely due to advances in immunology and leukocyte biology over the last decade. Leukocytes have been shown to exhibit a number of immunomodulatory phenotypes, including M2 macrophages, regulatory T and B-cells, and fibrous cells with anti-inflammatory function. This means that the presence of leukocytes observed by a pathologist at the site of infection or injury does not necessarily indicate "inflammation", at least without further characterizing their functional phenotype. Therefore "inflammation" based on the expression of inflammatory cytokines and the presence of the phenotype of inflammatory leukocytes, it is associated with high recurrence rates, including chronic myelogenous leukaemia in adults. Donor lymphocytes are important in preventing tumor recurrence, but they also cause graft-versus-host disease, a major complication. Clinical manifestations of acute graft-versus-host disease include rash, diarrhoea, gastrointestinal bleeding, and jaundice due to immune-mediated damage to the epithelial cells of the skin, gastrointestinal tract, and liver. In severe cases, graft-versus-host disease can be fatal and is the leading cause of death for allogeneic hematopoietic stem cell transplantation recipients. Trauma is the leading cause of death for young people in industrialized countries. Recent clinical and experimental studies have increased evidence that activation of the innate immune system contributes to the etiology of traumatic and adverse consequences. As a "front line of defense," the complement system represents a powerful effector arm of innate immunity and is involved in mediating the early post-traumatic inflammatory response. Despite commonly useful functions such as pathogen removal and immediate response to danger signals, complement activation has post-traumatic effects. Post-traumatic ischemia/reperfusion injury represents the classic entity of complement-mediated tissue damage, exacerbating local and systemic inflammation and contributing to "antigen loading" by releasing toxic mediators. These pathophysiological results have been shown to persists systemic inflammatory response syndrome after major trauma and ultimately contribute to distant organ damage and death. Numerous experimental models have been developed in recent years with the aim of mimicking the post-traumatic inflammatory response and enabling testing of new pharmacological approaches that include new concepts of site-specific complement inhibition. Treatment for inflammatory diseases may include medications, rest, exercise, and surgery to correct joint damage. Treatment plan will depend on several things, including type of disease, age. Treatment for inflammatory diseases may include medications, rest, exercise, and surgery to correct joint damage. To heal the cut, body sends inflammatory cells to the injury. These cells start the healing process. Body continues sending inflammatory cells even when there is no danger from the outside. For example, in

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rheumatoid arthritis inflammatory cells and substances attack joint tissues leading to an inflammation of the joints and also cause severe damage to joints with pain and deformities.