Acute inflammation 2nd degree burn case study biology essay

A 43 year old female presents with a burn on her left forearm. There is significant erythema that covers the entire proximal forearm in the shape of a circle. She complains of numbness and tingling down her pinky. There is only pain upon contact of the wound so she has to wear a short sleeve shirt. She burned herself while boiling hot water and spilling it on herself. The wound became red and swollen and gradually became worse over the course of two days. She has mild blistering and scalding across her forearm. She is determined to have a second degree burn because of the severity of the erythema and blistering, but the lack of charring and discoloration does not indicate a third degree burn. She presents with a classic case of a second degree burn, although many different variations have been documented. The first method of treatment is to clean and cool the wound. Once the burn has been cleaned and cooled, it can be managed by the use of antibiotics, analgesics, and sometimes local anasthetics. The prognosis for burns depends primarily on the age of the patient and the surface area of the wound. Also, since burn injuries often present as comorbid conditions, the presence of smoke inhalation injury, debris, or bone fractures will strongly influence the prognosis.

Skin burns can manifest in a variety of ways depending on the cause of the burn and also the severity of the burn. There are up to six degrees of burning, with each level of burn penetrating deeper into the skin layer. Burns can also be assessed in terms of total body surface area, which is the percentage affected by partial thickness or full thickness burns. Severe burns may require amputation, surgery, or skin grafting. When the first layer of skin (epidermis) is burned through and the second layer of skin (dermis) is

also burned, the injury is called a second-degree burn. Blisters develop and the skin takes on an intensely reddened, splotchy appearance. Underneath the epidermis is where the hair follicles, blood vessels, nerve endings and sweat glands reside. Underneath the dermis lie the muscles, nerves, larger blood vessels, and bones. Heart rate and peripheral vascular resistance increases following a major burn injury. This is due to the release of catecholamines from injured tissues, and the relative hypovolemia that occurs from fluid volume shifts. Initially, cardiac output decreases and approximately after 24 hours, cardiac output returns to normal and then increases to meet the hypermetabolic needs of the body.

Immediately following injury, vasodilation is the first step in acute inflammation. Arterioles become dilated and then new capillary beds are formed in the area. This causes heat and redness to form, which allows for increased blood flow. There is increased vascular permeability which causes exudate to flow out in the intravascular tissues. The increase of extravascular fluid leads to swelling, or edema. An important function of acute inflammation is to activate and send out leukocytes to the site of injury. In the lumen, there is margination, rolling, and adhesion to the endothelium so the leukocytes can bind to the lumen. The next step is called transmigration, which is the migration of leukocytes to the endothelium. Chemokines act on the adherent leukocytes to help the cells migrate through endothelial spaces toward the site of injury. This process occurs predominantly in the venules.

Next, leukocytes emigrate toward the injured area through a process called chemotaxis. All granulocytes, monocytes and lymphocytes respond to

chemotactic stimuli at their own unique rate. Phagocytosis involves three distinct but interrelated steps: recognition of the particle to be engulfed by the leukocyte, ingestion, and degradation of the ingested material. Binding of a particle to phagocytic receptors causes the process of active phagocytosis to occur. The particle to be engulfed is completely surrounded within a phagosome. The membrane of the vacuole combines with the limiting membrane of a lysosomal granule, which results in the release of the granule’s contents into the phagolysosome. Throughout the process, the neutrophils and monocytes become increasingly degranulated.

Microbial killing is largely carried out by oxygen-dependent mechanisms. Phagocytosis stimulates an increase in oxygen, increased glucose oxidation, and production of reactive oxygen intermediates. The regulators of inflammation have short half-lives and are manufactured in quick bursts, only for the amount of duration that the stimulus persists for. As inflammation progresses, the process also activates a variety of stop signals that actively terminate the reaction. These mechanisms cause a switch from the secretion of leukotrienes (pro-inflammatory) to lipoxins (anti-inflammatory).

References

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General Pathology

Chronic inflammation â€" Chronic Bronchitis

A 24 year old male presents with shortness of breath and an expectorating cough. He has been smoking hookah tobacco daily for the past six years. He has always had a weak immune system and has recently developed a constant cough within the last three months. A chest x-ray revealed hyperinflation of the lungs and auscultation of the lungs revealed prolonged expiration. The patient was determined to have chronic bronchitis due to the past history of chronic tobacco smoking and the consistent cough that has lasted for months. Common manifestations of chronic bronchitis include wheezing, productive cough, and occasionally chest pain, fever, and fatigue. There are a few different treatment protocols but the main one is smoking cessation and rest. In some cases, antibiotics or bronchodilators are prescribed. Early diagnosis of chronic bronchitis as well as smoking cessation, can drastically improve the probabilities of a good outcome.

Individuals with chronic bronchitis that quit smoking early on can greatly slow the progression of lung deterioration; otherwise they risk becoming permanently disabled at some point.

One major component of the mononuclear phagocyte system is macrophages, which consists of related cells such as tissue macrophages and blood monocytes. Monocytes migrate into many different tissues where they become macrophages. The lifespan of monocytes in the blood is about 24 hours, whereas tissue macrophages have a half-life of about several months to years. Early on during acute inflammation, monocytes emigrate into extravascular tissues, and within 48 hours they are established as the predominant cell type. The same factors that control neutrophil emigration, govern the extravasation of monocytes.

When the monocyte gets to the extravascular tissue, it is transformed into a much larger phagocytic cell, which increases its cell size, metabolism, and ability to properly ingest microbes. In acute inflammation, when the invading cell is destroyed, macrophages eventually undergo necrosis or migrate back into the lymphatic system. However, in chronic inflammation, macrophage accumulation continues, and is regulated by different mechanisms. This army of regulators makes macrophages important and also powerful mediators in the body’s defense system, but the same arsenal can also create detrimental tissue damage when macrophages are activated inappropriately. As a result, tissue necrosis is one of the main setbacks of chronic inflammation.

A wide range of substances in addition to the residual leftovers of macrophages can also contribute to tissue injury in chronic inflammation. Necrotic tissue can elevate the inflammatory response through the indirect activation of kinin, complement and fibrinolytic systems. Other cell types that contribute include mast cells, plasma cells, eosinophils, and lymphocytes. Mast cells are widely spread out in connective tissues and partake in both acute and chronic inflammatory conditions. Although neutrophils are mainly characteristic of short-term inflammation, many types of chronic inflammation continue to exhibit large numbers of neutrophils. Neutrophils are especially important in chronic lung damage induced by persistent tobacco smoking.

Granulomatous inflammation is a distinguished pattern of chronic inflammation most notably characterized by its focal accumulations of triggered macrophages, which often assume an epithelial-like appearance. It is only activated in a limited number of infectious and some noninfectious diseases. The lymphatic system along with the lymph nodes filters and mediates the extravascular fluids. Along with the phagocyte system, the lymphatic system represents a secondary or backup defense mechanism that is activated whenever a local inflammatory reaction fails to terminate or neutralize an invading cell.