Rheumatoid arthritis (ra) and guillain-barre syndrome (gbs)

Inflammation is a protective reaction associated with vascular tissues in response to different stimuli such as irritants and pathogens. In addition, other causes of inflammation may include physical injuries and immune reactions on body cells and tissues. Therefore, inflammatory reactions serve to eliminate the stimuli and start the process of healing on damaged cells, tissues, and organs (Ferrero-Miliani et al., 2007, p. 227). Conversely, these inflammatory reactions can be chronic or acute. This essay presents the etiology, pathogenesis, diagnosis, clinical manifestations, prognosis, and the treatment of Rheumatoid Arthritis (RA) and Guillain-Barre Syndrome (GBS).

Rheumatoid Arthritis (RA)

Scenario:

45 years old woman started with severe pain in her hands and feet. She noticed that she could not shake her wrists whilst she was doing PE with her students in the gym. About 2 month later, when she was working in her yard, the pain became even more pronounced. She was digging the yard for make a path way. The garden working was difficult to do because every day her feet hurt and the strength in her hands was so reduced that even lifting a large boiling pot in the staff kitchen was difficult. At night times she put her hands under her pillow to try to reduce the pain so that she could sleep.

Chronic immune inflammatory reactions can occur on synovial tissues in response to the synovitis, synovial cells, and the accumulated synovial fluid in the joints. This type of autoimmunity causes Rheumatoid arthritis (Majithia & Geraci, 2007). The symptoms of RA are not only limited to the articular regions but they can also spread to other parts of the body. Therefore, RA affects the joints, skin, lungs, kidneys, blood vessels, heart, and other

systemic tissues. In addition, the disorder leads to destruction of the ankylosis and cartilage lining the joints. It also causes nodular lesions on the skin and diverse inflammatory reactions on different systemic tissues (Majithia & Geraci, 2007, p. 937). The clinical diagnosis of RA involves physical examination of symptoms, blood tests, x-ray radiographic imaging, and other differential diagnoses, which are aimed at distinguishing the symptoms of RA from other disorders.

Moreover, the pathogenesis of RA entails proliferation and fibrosis of cells; the destruction of cartilage and bones; and pannus formation. These changes are caused by the activities of proteolytic enzymes, cytokines, and prostanoids in the synovial region (Majithia & Geraci, 2007, p. 937). Here, inflammation is mediated by Tumor Necrosis Factor-alpha and Interleukin-1 (IL-1), which are the most notable pro-inflammatory cytokines in the disease process of RA. The two cytokines enhance the production of other inflammatory elements such as nitric oxide (NO) and prostaglandin E2 (PGE2).

However, IL-1 has shown prominence in the pathogenesis of RA. Initial IL-1 release stimulates osteoblasts, synoviocytes, and chondrocytes. The cells take part in the inflammatory reactions, bone destruction, and pannus formation. Furthermore, the inflammatory reactions elevate the secretion of IL-1 relative to the progress of the disease. In addition, IL-1 stimulates the movement of neutrophils into the synovial region; the production and differentiation of lymphocytes; and finally the activation of macrophages. Additional IL-1 production leads to severe erosion of bone and cartilage, produces pain, and impairs tissue repair (Majithia & Geraci, 2007).

Lastly, Rheumatoid arthritis can be treated using medications such as analgesics, steroids, and disease-modifying antirheumatic drugs (DMARDs). In addition, non-pharmacological therapies such as physical therapy and nutritional therapy can halt the development of the disease. Conversely, the prognosis of RA shows varied symptoms in different patients such as disabilities, poor prognostic factors, and sometimes death (Majithia & Geraci, 2007, p. 939).

Guillain-Barre Syndrome (GBS)

Scenario:

A 33 years old man have a burning, sensitive, irritated sensation under his skin that spread throughout his arms and upper body over few months in the beginning of 2010. He noticed his sense of balance was lost. Then over a several weeks more symptoms presented themselves. His hands began to shake and tremor, his ears began buzzing, tickly in his left foot and the muscle spasms appeared, and muscle strength getting weak and pain grew in his thighs. His speech became jumbled and his left pupil dilated.

Acute infections of the peripheral nervous system can cause an autoimmune reaction in response to the pathogens and the host tissues. These immune responses are targeted at pathogens such as bacteria and the influenza virus but instead they attack the gangliosides of the nerve tissues (Hughes et al., 1999). This is the basis of GBS, which leads to inflammatory demyelination of the nerves and multiple neuropathies. Consequently, GBS is characterized by impaired sense of position, paralysis, absence of fever, areflexia, and symmetrical weaknesses that begin with the legs and spread to the upper limbs and finally to the face. Conversely, analyses of the cerebrospinal fluid

and electrodiagnostics provide important insights into the diagnosis of GBS. In addition, observable paralysis and areflexia can be used as the immediate indicators of GBS. However, additional differential diagnoses are important to distinguish the symptoms of GBS with other disorders such as the Motor Neuron Disease (Hughes et al., 1999, p. 74).

The pathogenesis of GBS is associated with immune responses targeted at an acute infection. However, the pathogens involved in the infection contain epitopes resembling some components of the peripheral nervous system. Therefore, the immune reaction attacks the nerve components causing acute inflammation on the myelin sheath or the axon (Hughes et al., 1999). Furthermore, the inflammatory reactions cause severe demyelination in the nodes of Ranvier and nerve roots. These inflammatory reactions are mediated by both the cellular and humoral immune components such as activated T-lymphocytes, which invade the demyelinated regions and attract macrophages that destroy the nerve membranes. Additional demyelination is thus, mediated by the macrophages and components of the complement system.

Lastly, the treatment of GBS entails providing supportive care for patients with paralyzed diaphragms and intravenous injections of immunoglobulin for stable patients. In addition, administration of plasmapheresis is recommended. Conversely, except for isolated cases of persistent areflexia, the prognosis of GBS shows that most patients begin recovering at the forth week after the onset and they can be completely healed after a few months or one year.

Conclusion

The essay presents a detailed discussion on two inflammatory conditions, which are caused by immune responses that target cells, tissues, and organs in the body. Therefore, the essay examines the etiology, clinical manifestations, diagnosis, pathogenesis, treatment, and the prognosis of Rheumatoid arthritis and Guillain-Barre Syndrome (GBS). From the discussions above, it can be deduced that inflammation is a serious complication, which occurs in the whole body or within a specific tissue and causes acute or chronic symptoms. However, most inflammatory conditions are treatable and preventable.