**NURS 6501Week 8: Module 5 Assignment: Concepts of Neurological and Musculoskeletal Disorders**

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Due Date

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**An Overview of the Case Study**

A 58-year-old obese male reports to the emergency department with chief complaints of fever, chills, pain, and swelling in the right great toe. Further, he states that the symptoms were sudden, and he could not put any weight on his foot. A physical examination reveals exquisite pain when assessing the first right metatarsophalangeal (MTP) joint. Besides these clinical manifestations of a musculoskeletal disorder, the patient has a medical history of hypertension and type 2 diabetes mellitus (T2DM). Based on the reported chief complaint of fever, chills, pain, and swelling in the right great toe, it is valid to confirm that the patient is suffering from gout.

**Neurological and Musculoskeletal Pathophysiological Processes of Gout**

The pathophysiology of gout entails an inflammatory response to excessive quantities of uric acid (hyperuricemia) in the blood and other body fluids. According to McCance & Huether (2019), hyperuricemia in synovial fluid is essential in explaining gout's pathophysiology. For instance, elevated levels of uric acid in the blood contribute s to the formation of monosodium urate (MSU) crystals around joints. When explaining the pathophysiology of gout, it is essential to consider the neurological process of an inflammatory response. Ragab et al. (2017) contend that the deposition of MSU crystals around joints triggers the inflammation process through engulfment by synovial phagocytic cells, leading to the production of lysosomal enzymes and inflammatory chemokines. Equally, the overproduction of urate and the subsequent monosodium urate crystallization signal TLR2 and TLR4 receptors, leading to the chronic inflammatory response.

From a perspective of musculoskeletal pathophysiology of gout, excessive quantities of uric acid in the blood and other fluids facilitate the crystallization of monosodium urate. As a result, the deposition of MSU crystals in the joint cavities triggers reactions with neutrophils and monocytes. McCance & Huether (2019) contend that the reactions between urate crystals and neutrophils and monocytes lead to tissue damage, especially when neutrophils release phagolysosome content. Tissue damages manifest through bone erosion, swelling, Achilles tendonitis, and olecranon bursitis.

**Racial/Ethnic Variables that May Impact Psychological Functioning**

Excessive alcohol consumption, obesity, lead toxicity, age, certain medications like thiazides, and dietary intake of purine-rich foods, including organ meats, pork, and fish, are modifiable and non-modifiable factors for gout (McCance & Huether, 2019). These risk factors are disproportionate to populations grappling with other health issues, such as hypertension. According to Sun et al. (2018), African Americans are at-risk of incident gout due to their susceptibility to hypertension. Equally, African Americans are vulnerable to obesity, diabetes, and chronic kidney disease, increasing the risk of gout.

**Conclusion**

Gout is a musculoskeletal disorder that emanates from excessive quantities of uric acid (hyperuricemia) in the blood and other fluids, including the synovial fluid. Hyperuricemia leads to monosodium urate crystallization and MSU deposition around joints. Notably, gout's neurological and musculoskeletal pathophysiology encompasses the triggering of inflammatory response after the deposition of MSU crystals around joints. An inflammatory response in joint cavities leads to bone erosion, swelling, pain, Achilles tendonitis, and olecranon bursitis. Ethnicity, unhealthy diet, medication use, obesity, diabetes, and lead toxicity are modifiable and non-modifiable risk factors for gout.

**References**

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