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Recommended Citation
Leonhard, Emily, "Gout as a Significant Risk Factor for Cardiovascular Disease: A Case Study" (2015). Nursing Student Class Projects (Formerly MSN). 84.
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Gout as a Significant Risk Factor for Cardiovascular Disease: A Case Study

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Introduction

Gout, an inflammatory arthritis caused by elevated serum uric acid levels, is emerging as a significant risk factor for cardiovascular disease (CVD). Recently, a nurse practitioner (NP) was caring for a patient who was suffering from a particularly severe gout attack while being treated in the ICU for new onset Atrial Fibrillation. The patient reported that he had never had an attack this severe before. He asked the NP if his history of gout could be related to his recent heart problems. The nurse practitioner, in an attempt to answer the patient’s question, spent time researching the disease process of gout, as well as the possibility of association between gout and cardiovascular disease. She learned that current research provides clear evidence that gout is a risk factor for CVD. In the course of her research, she advanced her knowledge of the pathophysiology of gout and the significance of gout’s risk for CVD. Finally, utilizing this knowledge will help her to manage the care of patients with gout and cardiovascular disease.

Presentation of Case Study

L. B. is a 64 year-old male hospitalized in the Intensive Care Unit (ICU) for new onset Atrial Fibrillation. On his second day in the hospital, L. B. underwent a stress test, which he ultimately failed. Eight hours after the stress test, L. B. developed severe pain, swelling, and anemia in both hands, elbows, wrists, feet, and ankles. The patient was given Fisonsamide 20mg IV, which did not alleviate the swelling and pain. Finally, it was identified that he was experiencing an episode of acute inflammation of the left lower extremity (club foot). The hallmark symptom of gout was present.

Phase 1: Acute gout - episodes of acute inflammation

- sudden onset of symptoms
- pain - most severe in the first 12 - 24 hours
- erythema (localized redness)
- inflammation of soft tissue arthritis and cartilaginous joint structures
- commonly affected joint is the first metatarsophalangeal (MTP) joint of the lower limbs; classic podagra (the hallmark symptom of gout)
- most commonly affected upper joint limb in the olecranon bursa. Hands can also be affected.
- monosodium urate deposition (single structure involvement) is the most common.
- nocturnal onset most common

Figure 1. (A) Rapid development of extended intracapsular tophi in the fingertips in a patient with serum urate level >32 mg/dL caused by chronic kidney disease and chronic heart failure on high-dose diuretics Beites, A. M. (2014, p. 197).

Phase 2: Chronic gout – persistent or mononucleo

- palpable tophi (macroscopic aggregate of monosodium urate crystals (MUCs))
- gouty arthritis – persistent joint limitations
- chronic gouty arthritis – persistent joint swelling
- joint deformity
- oligoarticular or polyarticular distribution may occur if gout is severe, persistent and untreated for an extended length of time
- increased risk for cardiovascular disease

Underlying Pathophysiology

Pathophysiology of gout

High levels of serum uric acid, hyperuricemia, is the main cause of gout. Hyperuricemia is when serum uric acid levels are greater than 7.0 mg/dL. In treated serum uric acid levels can occur from two different causes: overproduction of uric acid urate through purine synthesis de novo and salvage pathways or renal under secretion of uric acid.

Additionally, there is a hereditary component to the ability of certain populations in renal uric acid handling of acid and hyperuricemia (87% for familial fractional excretion of uric acid, 60% for serum urate) (Merriam, Choi, & Dayer, 2014, p. 194).

As the level of uric acid in the blood increases, it precipitates and forms monosodium urate (MSU) crystals. MSU crystals, once deposited in the tissues and synovial space of the joint, initiate an acute inflammatory response. The inflammatory response attracts leukocytes to the synovial space and phagocytizes the MSU crystals. This release destructive enzymes that cause more inflammation and tissue damage.

Signs and Symptoms

Figure 2. 60 mg, Colchicine 0.6mg, Methelprednisone 60mg IVP. This relieved the swelling and pain. Finally, it was identified that he was experiencing an episode of acute inflammation of the left lower extremity (club foot). The hallmark symptom of gout was present. This is a picture of L.B.'s right foot and ankle shortly after discharge from the ICU. This picture was provided by L. B. and permission for use was obtained.

Phase 3: Established gout

- widespread tophi formation
- gouty arthritis
- chronic gouty arthritis
- joint pain
- joint deformity
- arthritis
- reduced mobility
- hyperuricemia
- gouty tophi
- urate monosodium
- urate crystals

Figure 3. Implications for Nursing Care

A thorough knowledge of the phases of the disease is required to effectively manage gout (Hardy, 2011, p. 197). Not only must the NP efficiently and effectively treat the Ego’s of the patient, but also manage the chronic inflammation as well. Furthermore, because of the well documented association between gout and cardiovascular disease, the nurse practitioner must evaluate a patient with the diagnosis of gout for the increased risk of CVD. For example, if a patient presents with acute symptoms of gout, there should be an automatic assessment for CVD. The assessment for cardiovascular risk factors should lead to patient education about the increased future risk for cardiovascular disease (Blakes & Krishnan, 2014, p. 141).

Significance of Pathophysiology

A direct and indirect association between the pathophysiology of gout and the risk for developing CVD has been well established by past and current research. According to Hardy, 2011, p. 197, “gout is a chronic disease. It is imperative that practitioners understand the pathophysiology of gout and the heart.” The direct pathway starts with hyperuricemia to deposition of MSU crystals (Bhole & Krishnan, 2014, p. 126). There are several mechanisms that explain the association between gout and CVD. MSU, monosodium urate (Bhole & Krishnan, 2014, p. 140).

Over time, this chronic form of inflammation [gout] can perceptually increase the risk of CVD (Blakes & Krishnan, 2014, p. 126). There are several mechanisms that explain the association between gout and CVD.

The direct pathway starts with hyperuricemia to deposition of MSU crystals into the synovial spaces of joints. This results in chronic low-grade inflammation which promotes atherogenesis and thrombogenesis. Additionally, there are reports that link hyperuricemia with greater coronary artery complications (Blakes & Krishnan, 2014, p. 126). Elevated uric acid levels in the blood increases the patient’s risk for CVD.

The indirect pathway proves that there are shared risk factors for gout and CVD. Research has shown that patients with gout also have the same risk factors for CVD. These include: male gender, age, diabetes, hypertension, obesity, alcohol consumption, metabolic syndrome, and hyperuricemia (Blakes & Krishnan, 2014, p. 179).

References Cited


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