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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 order 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, redness, and swelling in bilateral hands, wrists, feet, and ankles. The patient was given Furosemide 20mg IVP, which did not alleviate the swelling and pain. Finally, it was identified that he was experiencing an episode of acute inflammation (EAI) of gout and he was given Methelprednisone 60mg IVP. This relieved the swelling and pain enough that he could be discharged. Ultimately, L. B. underwent cardiac catheritization where he received a stent for an 80% occlusion to his circumflex artery.

L. B. has a medical history of atrial fibrillation, gouty arthritis, hyperlipidemia, and GERD. Surgical history reveals left knee replacement (2015), left rotator cuff repair (2008), right rotator cuff repair (2004), and right knee replacement (1997). Current medications are Plavix 75mg, Sotalol 80 mg, Colchicine 0.6mg, Allopurinol 300mg, and Coumadin 2mg.

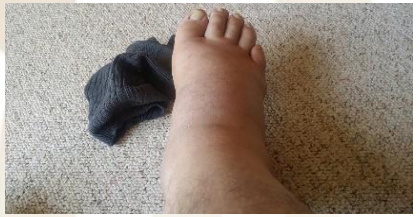

Figure 1.
This is a picture of L.B.'s right foot and ankle shortly after discharge from the ICU. This picture was provided by L. B. and permission for the picture to be printed was obtained from L. B.

Signs and Symptoms

There are two phases of gout: acute and chronic. Each phase has distinct signs and symptoms (Perez-Ruiz, Castillo, Chinchilla, & Herrero-Beites, 2014, p. 194).

Phase 1: Acute gout- episodes of acute inflammation (EAI)

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

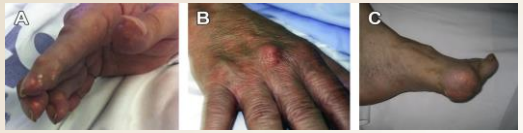

Fig. 3. (A) Rapid development of extended intradermal tophi in the fingerpads in a patient with serum urate level >12 mg/dL caused by chronic kidney disease and chronic heart failure on high-dose diuretics (Perez-Ruiz et al., 2014, p. 197).

Phase 2: Chronic gout – persistent or nonacute

- palpable tophi (macroscopic aggregate of monosodium urate crystals [MSUCs])
- gouty arthropathy – persistent joint limitations
- chronic gouty arthritis – persistent joint swelling
- joint deformity
- oligoarticular and 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 urate, hyperuricemia, is the main cause of gout. Hyperuricemia is when serum uric acid levels are greater than 7.0 mg/dL. Elevated serum uric acid levels can occur from two different causes: overproduction of hepatic urate through purine synthesis de novo and salvage pathways or renal under excretion of uric acid.

Additionally, there is a hereditary component to the ability of certain populations in renal uric handling of acid and hyperuricemia (87% for fractional excretion of uric acid, 60% for serum urate) (Merriman, Choi, & Dalbeth, 2014, p. 280).

As the level of urate 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, initiates an acute inflammatory response. The inflammatory response attracts leukocytes to hte synovial space and phagocytize the MSU crystals. This releases destructive enzymes that cause more inflammation and tissue damage.

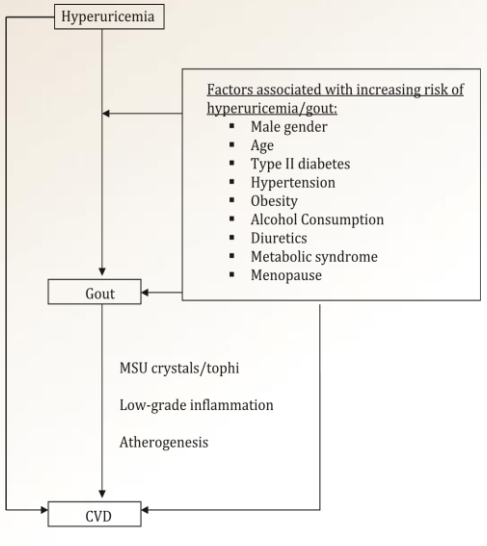

Fig. 7. Direct and indirect causal pathways linking hyperuricemia, gout, and CVD. MSU, monosodium urate. (Bhole & Krishnan, 2014, p. 140).

Pathophysiology of gout as a risk factor for cardiovascular disease

Over time, this chronic form of inflammation [gout] can perceivably increase the risk of CVD (Bhole & 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 calcifications (Bhole & Krishnan, 2014, p. 126). Ultimately, untreated high levels of uric acid in the blood will increase 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 menopause (Bhole & Krishnan, 2014, p. 139).

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. Unfortunately, gout is a common condition. In fact, gout is one of the most common conditions seen by practitioners. Its prevalence is higher than rheumatoid arthritis and in some studies it's equal to fibromyalgia and more prevalent than kidney disease, liver disease and prostate cancer in annual primary care visits in the United States (Vannucchi, 2012, p. 192). Therefore, as the incidence of gout increases in the population, so is the risk for CVD increasing. It is imperative that practitioners recognize this vital association so they can be prepared to treat a patient's gout and assess for CVD risk.

Implications for Nursing Care

A thorough knowledge of the phases of the disease is required to effectively manage gout (Hardy, 2011, p. 19). Not only must the NP efficiently and effectively treat the EAIs of gout, 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 then lead to patient education about the increased future risk for cardiovascular disease (Bhole & Krishnan, 2014, p. 141).

Conclusion

Thorough research has provided knowledge about gout and its association with increased risk of cardiovascular disease for both the nurse and the patient. After sharing what she learned, the nurse and the patient discussed how to decrease his risk for future EAIs and for CVD. She provided education on how he can modify his diet, exercise, medications, etc. Utilizing this knowledge, its significance and practice implications, the nurse is better prepared to care for future patients suffering from gout and CVD.

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