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Emily Leonhard
Otterbein University, emily.leonhard@otterbein.edu

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Gout as a Significant Risk Factor for Cardiovascular Disease: A Case Study

Emily Leonhard, R.N., B.S.N
Otterbein University, Westerville, Ohio

Introduction

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

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

- sudden onset of symptoms
- pain - most severe in the 1st 24 - 48 hours
- erythema (localised redness)
- inflammation of soft tissue of articular and periarticular joint structures

- Most commonly affected joint is the first metatarsophalangeal (MTP) joint of the lower limb – classic podagra (the hallmark symptom of gout)
- most commonly affected upper limb joint is the olecranon bursa.
- Hands can also be affected.
- monochromatous distribution (same structure involvement) is the most common.
- nocturnal onset most common

Phase 2: Chronic gout – persistent or nonacute

- palpable tophi (macrophage aggregate of monosodium urate crystals (MSUcs))
- gout arthritis – persistent joint irritation
- severe gouty arthritis – persistent joint swelling
- joint deformity
- oligoarticular to polyarticular distribution may occur if gout is severe, persistent and untreated for an extended length of time

- increased risk for cardiovascular disease

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. Additional, there are reports that link hyperuricemia with greater coronary artery calcifications (Boles & Krishnan, 2014, p. 145).
- The inflammatory response attracts leukocytes into the synovial and the tissues and synovial space of the joint, initiates an acute inflammatory response. The inflammatory response also leads to the neovascularization of the synovial tissue and phagocytes MSU crystals. This release destructive enzymes that cause more inflammation and tissue damage.

Additionally, there is a hereditary component to the ability of certain populations to renal acid handling of acid and hyperuricemia (87% for fractional excretion of uric acid, 60% for serum urate) (Merriam, Choi, & Dalbeth, 2013, p. 3).

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

Presentation of Case Study

L. B. is a 64 year old male hospitalized in the Intensive Care Unit (ICU) for non acute 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 Fisonsaline 20mg IV which did not alleviate the swelling and pain. Finally, he was identified that he was experiencing an episode of acute inflammation (EAI) of gout and he was given Melphesinone 60mg IV. This relieved the swelling and pain enough that he could be discharged. Ultimately, L. B. underwent cardiac catheterization where he received a stent for an 80% episode of acute inflammation (EAI) of gout and he was given Melphesinone 60mg IV. This relieved the swelling and pain enough that he could be discharged. Ultimately, L. B. underwent cardiac catheterization where he received a stent for an 80%

Figure 3. (A) Rapid development of extended intradermal tophi in the fingertips in a patient with serum urate level >12 mg/dL caused by chronic kidney disease and chronic heart failure on high dose diuretics (Boles & Krishnan, 2014, p. 197).

Signs and Symptoms

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 non acute Atrial Fibrillation. The patient had already had 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 cardiac catheterization where he underwent a stress test, which he ultimately failed. Ultimately, L. B. underwent cardiac catheterization where he received a stent for an 80%

Phase 2: Chronic gout

Conclusions

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 uric acid urine through purine synthesis de novo and salvaging pathways or renal excretion under excess of uric acid.

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. Additional, there are reports that link hyperuricemia with greater coronary artery calcifications (Boles & Krishnan, 2014, p. 145). Elevated uric acid levels have been associated with increased risk for CVD.

The inflammatory 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 age, diabetes, hypertension, obesity, alcohol consumption, metabolic syndrome, and monosopn (Boles & 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. Gout may be considered, in a common condition. In fact, gout is one of the most common conditions seen in 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 that they can prepare 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 EAs 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. This assessment should be performed to assess these risk factors to either prevent the patient from developing CVD or to early detect the disease. If the disease is detected in an early stage, the patient can be referred to a CVD specialist doctor. This early detection and assessment can lead to an early treatment of CVD.

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 EAs and for CVD. The patient was educated on how he can modify his diet, exercise, medications, etc. Utilizing this knowledge, his significance and practice implications, the nurse is better prepared to care for future patients suffering from gout and CVD.

References Cited


Additional Sources


Cognates

Figure 2. (B) Rapid development of extended intradermal tophi in the fingertips in a patient with serum urate level >12 mg/dL caused by chronic kidney disease and chronic heart failure on high dose diuretics (Boles & Krishnan, 2014, p. 197).

Figure 7. Direct and indirect causal pathways linking hyperuricemia, gout, and CVD. MSU: monosodium urate (Boles & Krishnan, 2014, p. 146).

Pathophysiology of gout as a risk factor for cardiovascular disease

Over time, this chronic form of inflammation (gout) can perceptively increase the risk of CVD. (Boles & 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. Additional, there are reports that link hyperuricemia with greater coronary artery calcifications (Boles & Krishnan, 2014, p. 126). Elevated uric acid levels have been associated with increased 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 age, diabetes, hypertension, obesity, alcohol consumption, metabolic syndrome, and monosopn (Boles & Krishnan, 2014, p. 139).