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Gouty Arthritis

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Introduction

Gouty arthritis is a relatively common diagnosis within the United States. It is estimated that 9.2 million Americans suffer from gout (Golenbiewski & Keenan, 2019). Gouty arthritis is also believed to be on the rise (Golenbiewski & Keenan, 2019). This may correlate with the aging baby boomer generation since age is a risk factor. The rise of conditions that increase the risk for gouty arthritis is another correlation (Harding, 2016). These conditions include hypertension (HTN), type 2 diabetes mellitus, and chronic kidney disease (Harding, 2016). It is very probable that gouty arthritis will be encountered within the primary care setting. Gouty arthritis is a condition that may be treated and managed in primary care by a family nurse practitioner (FNP). FNPs should be knowledgeable of when to refer or consult with rheumatology specialists.

Case Study

The following is a fictitious case study that represents a scenario that could be encountered in primary practice:

- 48-year-old male presents with severe pain ("toe is on fire" description) in his right big toe, which is accompanied by inflammation and erythema
- Denies any fever, chills, or trauma
- Current prescription regimen: simvastatin 40 mg daily for hyperlipidemia for 6 years and hydrochlorothiazide 25 mg daily for HTN for 5 years
- Other factors include consistent weight gain for the past 5 years; he is about 50 pounds overweight
- Social history includes drinking a six-pack of beer daily

The remainder of this presentation will provide the FNP with the knowledge and tools on how to address and treat such a case.

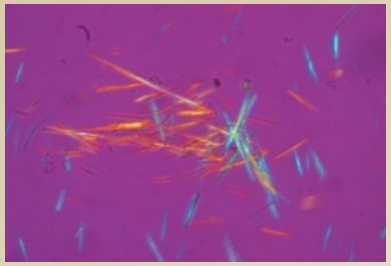

Figure 1: MSU crystals
Credit: By Bobjgalindo - Own work, CC BY-SA 4.0, https://commons.wikimedia.org/w/index.php?curid=5653456

Signs & Symptoms

- Common initial presentation is redness, swelling, and pain of the big toe (Golenbiewski & Keenan, 2019)
- Feet, ankle, hands, wrists, elbows may also be affected (Golenbiewski & Keenan, 2019)
- Associated symptoms: shiny skin, mild fever or chills, loss of appetite (Golenbiewski & Keenan, 2019)

Underlying Pathophysiology

Gout is a metabolic disorder that results in painful, sometimes debilitating, arthritis (Harding, 2016). The cause of this condition is a buildup of uric acid in the blood and synovial fluid (McCance & Huether, 2018). It is associated with purine metabolism and kidney function. Purines and pyrimidines in the body become converted to uric acid (McCance & Huether, 2018). One enzyme that plays a role in the breakdown of uric acid is xandine oxidase (Golenbiewski & Keenan, 2019). Uric acid is then filtered in the blood and excreted in the urine. Uric acid has a limited solubility and the threshold of uric acid is about 6.8 mg/dL (Ragab, Elshahly, & Barin, 2017). Uric acid loses a proton at a pH of about 7.4 and then becomes a urate ion (Ragab et al., 2017). The urate ion binds sodium and forms monosodium urate crystals (MSU) (McCance & Huether, 2018). MSU crystals are then deposited around the joints (McCance & Huether, 2018). MSU crystals that are in the joint cavity initiate the inflammatory process(Golenbiewski & Keenan, 2019). Leukocytes will migrate to the affected site to release inflammatory chemicals such as cytokines (Golenbiewski & Keenan, 2019). Acute attacks are generally self-limited as mast cells help end the inflammatory period by engulfing debris and crystals (Raga et al. 2017).

Hyperuricemia can occur if there is decreased or overproduction of uric acid. Renal failure, HTN, and diuretics can lead to decreased excretion of uric acid (Berkowitz, 2007). Causes of overproduction of uric acid include an overconsumption of purines in meats and alcohol, increased cellular turnover (hemolytic anemias or malignancies), and hypoxanthine guanine phosphoribosyltransferase (HGPRT) deficiency (Lesch-Nyhan syndrome) which is an inherited X-linked disorder (Berkowitz, 2007). 90% of cases are caused by renal underexcretion (Ragab, et al, 2017).

Significance of Underlying Pathophysiology

There are four stages to gouty arthritis: asymptomatic tissue deposition, acute flares, inter-critical segments (continued crystal deposition), and chronic gout (American College of Rheumatology [ACR], 2019). Key points to these phases include:

- Asymptomatic hyperuricemia may be an incidental finding and does not necessarily warrant treatment for gout (Golenbiewski & Keenan, 2019)
- SUA levels may be normal during an acute attack and require obtaining a baseline after the attack resolves (Harding, 2016)
- In the inter-critical segment, crystals remain and may require ongoing treatment for hyperuricemia (Ragab et al., 2017).
- Repeated acute flare ups lead to chronic gouty arthritis. This leads to tissue destruction and deformity caused by tophi which permanent deposits of urate crystals along the joints (Ragab et al., 2017).
- Chronic gout leads to an increased risk for kidney stones composed of urate and urate nephropathy (Harding, 2016). Urate nephropathy is a buildup of crystals in the kidney interstitium and tubules leading to renal failure.

Tophus of the knee

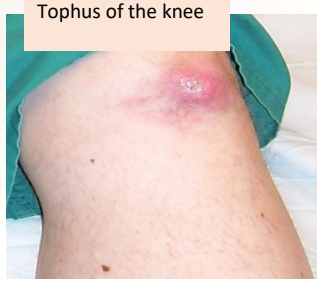

Figure 2: By NickGorton - NickGorton, CC BY 2.5, https://commons.wikimedia.org/w/index.php?curid=1324804

Implications for Nursing Care

- Diagnosis can only be confirmed by identifying MSU crystals within synovial fluid (Ragab et al., 2017)
- **Normal SUA levels are 3.5-7.2 mg/dL (ACR, 2019)**
- **Target SUA is <6 mg/dL (ACR, 2019)**
- Treatment is aimed at lowering urate levels and making dietary changes. Management of acute flares are aimed at managing inflammation and restoring function (Golenbiewski & Keenan, 2019).
- Not all respond to ULT and have refractory gout

- **Medications for Acute Gouty Arthritis (Hanier, Matheson, & Wilkes, 2014)**
  - NSAIDs (first line of therapy)
  - Colchicine (no analgesic properties; should not be administered after 36 hours of onset)
  - Corticosteroids (preferred for NSAIDs and colchicine contraindication)
  - Do not initiate urate lowering therapy (ULT) during an acute attack

- **Medications for Prevention of Chronic Gouty Arthritis (Hanier et al., 2014)**
  - Colchicine
  - Pegloticase
  - Xanthine oxidase inhibitors: Allopurinol, febuxostat

- **Dietary recommendations**: reduction or avoidance of foods that can increase SUA (Golenbiewski & Keenan, 2019):
  - Alcohol
  - Meats (organ, red and/or processed)
  - High fructose corn syrup

- **Risk factors (ACR, 2019):**
  - **Lifestyle and other factors:**
    - Diet
    - Alcohol consumption
    - Male
    - Family history
  - **Health conditions:**
    - HTN
    - Obesity
    - Hyperthyroidism
    - Renal insufficiency
    - Hemolytic anemia
    - Kelley-Seegmiller syndrome
    - Lesch-Nyhan syndrome
  - **Medications:**
    - Diuretics
    - Salicylate containing drugs
    - Niacin
    - Cyclosporine
    - Levodopa

Hyperuricemia Management

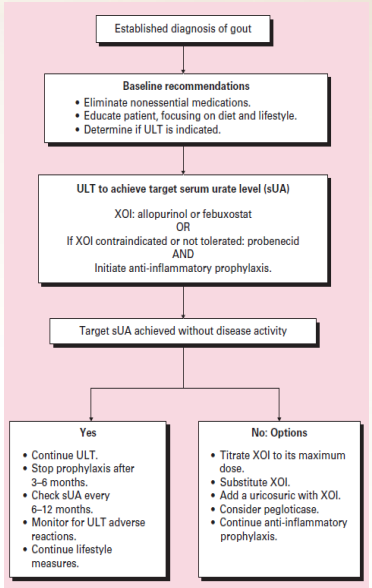

Figure 3: Credit: Harding, 2016

Revisit the Case Study

Within the case study and the information provided, it would be reasonable for the FNP to suspect an acute gout attack. His age, sex, location/description of pain, inflammation of the big toe, history of HTN, prescription of the diuretic HCTZ, increased weight, and alcohol use are all factors that influence the development of gout. The acute attack should be managed with appropriate pharmacologic therapy. Changing the HCTZ to another option to manage HTN should be considered. A referral to a rheumatologist is appropriate to identify MSU crystals within the joint and to determine the long-term plan. Patient education regarding diet, weight, and alcohol use will be vital to help prevent future attacks and joint damage.

Conclusion

Though gout is well understood, it generally remains poorly managed. The keys reasons for this are poor diet, widespread use of diuretics, comorbidities such obesity and HTN (Mead, Arabindoo, & Smith, 2014). Studies show that less than half of patients on urate lowering therapy are compliant with medication (Mead et al., 2014). To help combat this issue, providers can increase follow-up appointments (Mead et al., 2014). Acute flares are aimed at managing inflammation and restoring function (Golenbiewski & Keenan, 2019). Once the acute attack subsides, treatment is aimed at lowering urate levels and making dietary changes. If non-compliance is not thought to be an issue and the patient is not responding to treatment, refractory gout may need to be considered and treated accordingly. Finally, it is important for the FNP to note that guidelines for treatment may change in the future. Current ACR guidelines were published in 2012. These guidelines are currently under review and updated recommendations can be expected in 2020 (ACR, 2019).

References

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