“You Did This To Me”: a Case of Acute Gouty Attack During Acute Decompensated Heart Failure Treatment

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Learning Objectives

1) Recognize that aside from direct diuretics effect, clinical factors such as hypovolemia and acute kidney injury, can also lead to hyperuricemia.

2) Recognize that gout flare does not necessitate discontinuation of loop diuretics. Clinicians need to be aware the various considerations in selecting agents for treating gout flare, in patients with acute decompensated heart failure.

Case Presentation

A Caucasian female in her 60s with a past medical history of paroxysmal atrial fibrillation, coronary artery disease and obesity, initially presented to the emergency department with palpitation, found to be in atrial fibrillation with rapid ventricular response, acute decompensated heart failure and acute kidney injury. She received amiodarone loading and intravenous furosemide. On hospital day five, she was referred to the home hospital (HaH) program. On admission, the patient was found to have pain, erythema and swelling involving the right MTP joint. Pain progressively worsened over the last 24 hours. Physical examination and review of system revealed no tophi nor history of urolithiasis.

“I was told this is gout. You (clinicians) did this to me because you picked Lasix”, said the patient. Serum uric acid was 11.9 mg/dL (ref range 2.3- 6.6 mg/dL) on HaH admission and 12.6 mg/dL on repeat. HaH team continued intravenous furosemide and started the patient on renally dosed colchicine. Pain subsequently improved and finally subsided. On discharge, the patient was scheduled to follow up with PCP to consider urate-lowering therapy.

Hyperuricemia can result both from direct diuretics effect as well as other clinical processes such as volume depletion and acute renal injury

Clinicians also should astutely recognize gout flare and be thoughtful on treatment strategy given the patient’s comorbidities, therapy side effects and interactions

Team-based non-pharmacological interventions including patient dietary education play important roles in both acute decompensated heart failure and gout management

Table: Non-pharmacological management with focus on dietary education

	Gout	Heart Failure
Goals	Prevention of flare and disease progression •Avoid flare triggers •Manage comorbid conditions •Urate lowering (ACR 2020 recommends serum urate concentration less than 6 mg/dL and less than 5 mg/dL for + tophi; while ACP 2017 deemed insufficient evidence for such targets)	Reduce symptoms Prevent disease progression Avoid complications
Risk Reduction	Dietary changes Weight reduction Alcohol intake reduction Medication substitution (such as thiazide diuretics)	Smoking cessation Alcohol intake reduction Sodium intake reduction Avoid illicit drug (such as cocaine) Weight reduction Activity & Exercise Cardiac Rehabilitation
Comorbid Management	Hypertension Obesity Diabetes Hyperlipidemia Atherosclerosis Metabolic syndrome Chronic kidney disease	Ischemic heart disease Valvular disease Cardiomyopathy Hypertension Diabetes Obesity Arrhythmias Others: Anemia & Iron deficiency
Dietary Education	Weight loss reduced calories and weight loss can lower the amount of gout attacks and reduce uric acid levels, lessen stress on the joints Dietary composition •Generalized dietary purine restriction is neither practical nor necessary •Adequate protein intake, esp. that from low-fat dairy +/- plant sources •DASH diet •Fruits, vegetables and whole grains Some studies have showed these may be beneficial •Coffee; in moderation especially regular caffeinated •Vitamin C (500 mg daily) •Cherries •Limited consumption of natural sweet fruit juice •Moderate consumption of wine Certain foods to use in moderation or to avoid •Organ and glandular meats •Some seafood like anchovies, shellfish, sardines and tuna •Alcohol, especially beer and distilled liquors •Foods and beverages with high fructose corn syrup	Limited sodium - 2 g/day during inpatient - AHA/ACC 2022: < 2.3 g/day - European Society of Cardiology: avoid > 6 g/day Restrict fluid intake in refractory HF or symptomatic hyponatremia to 1.5-2L/day Weight loss Dietary composition •Limit the saturated fats, avoid the trans fats •DASH diet Omega-3 polyunsaturated fatty acid (PUFA) supplementation

Other reference: UpToDate, Mayo Clinic patient education

Discussion

Physiology of Urate Excretion & Hyperuricemia

- Physiologically, 75% of urate is excreted by the kidney while the rest by the GI tract. With renal excretion specifically, urate reabsorption and secretion take place at proximal tubules.
- Hyperuricemia can result from both under-excretion and overproduction of urate. In heart failure, under-excretion is mainly associated with impaired renal function. Alcohol consumption and obesity are associated with not only increased urate production but also reduced renal excretion.
- Interestingly, salt restriction, both short- and long-term, has also been related to hyperuricemia. Tissue hypoxia, common in heart failure, is a stimulus for urate production.

Diuretics-induced hyperuricemia mechanism

- Two mechanisms proposed: indirect and direct.
- Indirect effect: secondary to volume depletion by diuretics. Volume depletion results in an appropriate increase in proximal tubule urate reabsorption.
- Direct effect: a few mechanisms have been proposed. 1) Diuretics and urate compete for proximal tubule secretion 2) Diuretics inhibit secretion of urate at a luminal transporter protein 3) Thiazide selectively enhances urate reabsorption.

Decision on Diuretics

- Most clinicians do not discontinue diuretics because of gout flares during an ADHF hospitalization
- Clinically gout usually develops after 20-30 years of sustained hyperuricemia

Choice of therapy for acute gout flare

- Oral NSAID is almost always prohibited given its volume retention and renal injury effects, as well as its association with poor heart failure hospitalization outcomes. In this particular patient, NSAID use is also prohibited by concurrent DOAC medication.
- Colchicine can effectively reduce inflammation and pain. Oral formulation is associated with a high risk of diarrhea. Intravenous formulation has a high risk of side effect including bowel marrow suppression and CNS effects. The dose needs to be renally and hepatically adjusted.
- Intra-articular injection of glucocorticoids, if available, can be effective.
- Oral glucocorticoid can be used with caution, considering its heart failure-specific effects including fluid retention and pro-arrhythmia, as well as other side effects
- Controversies exist on the indications, timing, and whether to treat to target for Allopurinol, the urate-lowering therapy (ULT). If to initiate, allopurinol can be started during a gout flare. The practice of delaying the ULT start until after flare resolution is based on observations and common practice, but lacks concrete evidence. Allopurinol should be renally dosed. Flare prophylaxis, with the above-discussed agents, can be considered as co-therapy.

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