Web Case Study: Acute Gouty Arthritis

Roshini Mathew, RN, BSN

Wright State University-Miami Valley College of Nursing and Health

NUR 7103

February 22, 2013
Web Case Study: Acute Gouty Arthritis

History and Physical

Source of Information

Information was obtained from the patient, who is alert and oriented and is a reliable source for providing data. He is accompanied by his wife and gives the nurse practitioner (NP) permission for the wife’s presence in the examination room. The patient is dual-lingual in English and Spanish, but the primary language is English.

Chief Compliant

“Throbbing/sharp pain and swelling in right big toe.”

History of Present Illness

This is a 49-year old Caucasian male presenting to the office with pain in the right great toe that suddenly started yesterday morning around 11:00 AM while he was getting ready for work. He reports it is a continuous pain at a pain level of six out of ten, but started at four out of ten on a pain scale of zero to ten. The pain is more severe at night, reaching to a pain level of ten out of ten, and is described by the patient as “like my toe is on fire!” Usually, the patient sleeps for six to seven hours at night but has slept for only three hours last night due to severe pain. He is unable to tolerate covering his right foot with bed sheets, wearing his regular dress shoes, and standing for more than five minutes. Due to the pain, he has not been to work since yesterday. He has been applying ice, elevating the right leg, and taking Tylenol 650 mg oral every four to six hours with minimal to no pain relief. The patient denies injury, falls, or any other trauma to the right foot. No other pain, redness, or swelling is reported. He reports no new changes in his care plan except for a new medication, hydrochlorothiazide, added by his cardiologist three weeks ago to better manage his blood pressure. He denies any other recent illnesses.
WEB CASE STUDY GOUT

Medications

Metoprolol 50 mg PO BID, hydrochlorothiazide 12.5 mg PO daily, omeprazole 20 mg PO daily, aspirin 81 mg PO daily, acetaminophen 650 mg PO every four to six hours PRN for pain. Denies using alternative or complementary therapies.

Medical History

Childhood illnesses. Chickenpox at age of nine; fractured left ankle playing tennis at the age of 14.


Surgeries/Procedures. Tonsillectomy and adenoidectomy at age of 14; back surgery for sciatica pain relief in 2010.

Allergies. Latex and peanuts. No known drug allergies.

Immunizations. Up-to-date. Last tetanus-diphtheria booster received 2011. Flu vaccine received in December 2012.

Personal/Social History

The patient has been married for 23 years and has two children: one 13-year old daughter and one 16-year old son. He has been a full-time accountant for Liberty Mutual firm for the past ten years and shares no concern of financial hardship or emotional and work-related stress. He describes his relationship with his wife as “pleasant and happy,” but shares concern about taking care of behavioral issues with their teenage son which is causing a lot of stress in the family. He admits to drinking alcohol more than usual with his friends at a local bar. For the past ten years, he has been drinking one to two 12-ounce cans of beer every other day. Currently, he is drinking
WEB CASE STUDY GOUT

two to three beer cans every day for the past four months. The patient denies using illicit drugs or smoking.

The patient reports that he is usually independent at home, but since the onset of the right foot pain, his wife is assisting with activities of daily living. They live in a two-floor home and the patient reports not being able to walk up/down the stairs because of the severity of pain. He is currently not driving and relies on his wife for transportation. With full-time jobs, children’s extracurricular activities, and church events, the family has a hectic lifestyle and usually eats fast food or in a restaurant five days of the week. Some of the favorite food choices of the patient include steak and lobster. His work mainly consists of sitting. Occasionally, he will exercise (i.e. walk on the treadmill) once a week on Saturdays when he is off from work. In his leisure time, he enjoys solving Sudoku puzzles and reading history books.

Family History

The patient’s mother passed away at the age of 70 from a massive stroke. She had a history of diabetes, hypertension, and osteoarthritis. The father is 77-years old and lives in a nursing home. Father’s medical history consists of hypertension, hyperlipidemia, chronic kidney disease, and macular degeneration. The patient has one sister, 44-years old, who was recently diagnosed with hypertension. Per patient, the causes of death of the patient’s grandparents are unknown.

Review of Symptoms

General. The patient believes he is generally in good health despite having chronic high blood pressure. He reports having fatigue due to not sleeping since yesterday. Denies fevers, chills, loss of appetite, unidentified weight loss or weight gain. Confirms taking medications as prescribed and is independent with medication management.
WEB CASE STUDY GOUT

**Neurological.** Denies numbness, tingling, hallucinations, forgetfulness, alternation in mentation, diminished concentration, dizziness, tremors, or falls. States he is limping on his right foot due to pain, but usually has a steady gait.

**HEENT.** Head: Denies any trauma to the head and face including eyes, ears, nose, or mouth. Reports having headaches at times when he is stressed, but Tylenol is effective for pain relief. Eyes/Ears: Denies visual disturbance, diminished hearing, itching, discharge, pain, redness, edema, or ringing in the ears. Nose: Denies nasal congestion, drainage, sinus pain, bloody nose, or dryness. Throat: Denies sore throat, throat tightness, throat congestion, difficulty swallowing or foul breath/taste. States visits the dentist for biannual teeth cleaning and exam; last visit was three months ago.

**Neck.** No stiffness, pain, or enlarged lymph nodes.

**Respiratory.** No coughing, shortness of breath, pain when breathing, or chest discomfort.

**Cardiovascular.** Denies chest pain, sweating, extra heart beats, palpitations, dizziness, or swelling in arms or legs.

**Gastrointestinal.** States has good appetite, adequate food and water intake, and usual flatulence. No nausea, heartburn, hiccoughs, diarrhea, constipation, abdominal tenderness, or rectal bleeding. Usual bowel pattern consists of daily evacuation of moderate soft, formed brown stool.

**Genitourinary.** Denies urinary urgency, frequency, hesitancy, or burning. No bladder fullness or spasms. Does not have stress/night incontinence or flank pain. No history of urinary tract infections. Urine appearance is clear, without odor, and straw colored. Prostate exam that was completed last month was normal.
WEB CASE STUDY GOUT

**Genitalia.** Uncircumcised male. Denies penile or scrotal lesions, lumps, swelling, or drainage. Sexually active with his wife. States performs self-testicular exams occasionally.

**Musculoskeletal.** Reports right great toe joint tenderness, stiffness, redness, and swelling which started suddenly yesterday morning. Current pain rating is six out of ten. Denies history of falls, fractures, trauma, or any other muscle or joint pain. He usually ambulates and transfers independently and safely without support or use of equipment, but has been using a cane for support since yesterday when the right great toe pain began.

**Integumentary.** Reports redness, swelling, and warmth on right great toe. Denies itching, burning, bruising, rash, open sores, lesions, blistering, or thinning of skin.

**Psychosocial.** Denies depression, alteration in emotional stability, suicidal ideations, impulsive behavior, social withdrawal, and angry outbursts. States usually does not have anxiety, but since yesterday has been anxious due to the right great toe pain. Also states he has been stressed from the unpleasant behavioral actions of his teenage son.

**Hematologic.** No bleeding issues, anemia, or weakness stated.

**Endocrine.** No evidence of altered mentation, weakness, diaphoresis, irritability, weight loss, weight gain, thinning of hair, or increased thirst.

**Physical Examination**

**General.** The patient is appropriate, pleasant, makes eye contact, and is well-groomed. Patient is sitting on the examination table, grimacing, and guarding the right foot, and is anxious. Pain level is six out of ten on zero to ten scale. Height is five feet six inches, weight is 178 pounds, and calculated Body Mass Index (BMI) is 28.7 kg/m2 classifying him as overweight.
WEB CASE STUDY GOUT

**Vital signs.** Blood pressure is 148/88 mmHg, left arm sitting; apical pulse is 76 beats per minute and regular without ectopic beats; respiratory rate is 16 breaths per minute with normal depth and effort; temperature is 99.1°F oral; oxygen level of 99% on room air.

**Neurological.** Patient is alert and oriented to place, person, time/date and able to follow commands appropriately. Cranial nerves II – XII intact. Face is symmetrical and speech is clear and concise, without aphasia or dysarthria. Bilateral pupils are 3mm, normal, reactive to light, corneal reflex present, central and peripheral vision intact. Cough and gag strong and intact. Gross and fine sensory and motor strength intact in bilateral upper extremities and left lower extremity. Weak fine motor strength and altered gait noted in right lower extremity to pain. Deep tendon reflexes intact. No numbness and tingling reported. Short and long term memory intact.

**HEENT.** Head is normocephalic, no trauma, lesions, or tenderness on palpation. Moderate amount of thick gray/brown hair is evenly distributed. Frontal and maxillary sinuses non-tender to palpation. Smooth movement of temporomandibular joint. No periorbital edema, clear conjunctiva and sclera. Pupillary light reflex and extraocular muscles intact. Ears in normal position without external tenderness. Tympanic membrane is gray, no cerumen impaction. No hearing loss, positive Rinne test. Nose slightly bulbous without polyps or masses. Nares patent without inflammation or discharge. Buccal mucosa and lips pink and moist. Uvula and tongue midline, tonsils absent, all teeth intact with slight yellow staining. Scar noted above left eyebrow which the patient acquired as a child from falling off a swing.

**Neck.** No pain, pulsations, distended veins, or enlarged glands. Lymph nodes non-tender. Trachea midline, swallowing intact. Bilateral +2 carotid pulses, no bruits auscultated.

**Chest.** No gynecomastia, masses, or lesions. Non-tender on palpation.
**Respiratory.** Anteroposterior-to-transverse diameter of 1:2, symmetrical chest expansion, tactile fremitus present bilaterally. Normal, effortless, regular, even breathing. Resonant lung fields. Clear lungs upon auscultation without adventitious breath sounds.

**Cardiovascular.** Bilateral +2 radial, brachial, posterior tibial, and dorsalis pedis pulses with brisk capillary refill (less than three seconds). Apical pulse regular, S1 and S2 present, S3 and S4 absent. No murmurs, clicks, snaps, or rubs. No edema in extremities except right great toe.

**Abdomen.** Umbilicus midline and inverted. No protrusion, bulging, pulsation, tenderness, rigidity, fluid wave or discoloration. Abdomen round, obese. Normal bowel sounds auscultated in all four quadrants without bruits. Negative for tenderness at the costovertebral angle. Hepatomegaly and splenomegaly not present.

**Musculoskeletal.** Erythema noted at the base of the right great toe in the metatarsophalangeal joint with non-pitting edema, warm and tender to touch. Skin is intact without drainage. Limited flexion, extension, plantar flexion, and dorsiflexion of right foot and toes due to pain, 4/5 strength. Full range of motion and 5/5 strength in bilateral upper extremities, left lower extremity, hip, and spine. Equal arm and leg lengths. No erythema, crepitation, or edema noted in any other joint. No kyphosis or scoliosis.

**Integumentary.** Skin smooth, warm to touch, dry, and intact without lesions, ulcers, overgrowths, or ecchymosis. Overall, red-pink skin tone, no cyanosis or pigmentation noted. Nail beds pink, no clubbing.

**Laboratory Findings**

Table 1. Comprehensive Metabolic Panel (CMP) with Creatinine Clearance, Magnesium, and Phosphorus
### Table 1: Laboratory Results

<table>
<thead>
<tr>
<th>Lab</th>
<th>Results</th>
<th>Normal Values</th>
<th>Lab</th>
<th>Results</th>
<th>Normal values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>139 mEq/L</td>
<td>135-148 mEq/L</td>
<td>Globulin</td>
<td>2.0 g/dL</td>
<td>1.9-3.6 g/dL</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.0 mEq/L</td>
<td>3.4-5.3 mEq/L</td>
<td>A/G Ratio</td>
<td>1.7</td>
<td>0.8-2.6</td>
</tr>
<tr>
<td>Chloride</td>
<td>101 mEq/L</td>
<td>96-110 mEq/L</td>
<td>Total Bilirubin</td>
<td>1.4 mg/dL</td>
<td>0.2-1.9 mg/dL</td>
</tr>
<tr>
<td>Carbon Dioxide</td>
<td>26 mEq/L</td>
<td>23-29 mEq/L</td>
<td>AST (SGOT)</td>
<td>16 U/L</td>
<td>10-34 U/L</td>
</tr>
<tr>
<td>Glucose</td>
<td>98 mEq/L</td>
<td>70-100 mg/dL</td>
<td>ALT (SGPT)</td>
<td>18 U/L</td>
<td>8-38 U/L</td>
</tr>
<tr>
<td>BUN</td>
<td>14 mg/dL</td>
<td>7-20 mg/dL</td>
<td>Alkaline Phosphatase</td>
<td>85 U/L</td>
<td>23-144 U/L</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.9 mg/dL</td>
<td>0.8-1.4 mg/dL</td>
<td>Anion Gap</td>
<td>11</td>
<td>10-20</td>
</tr>
<tr>
<td>BUN/Creat Ratio</td>
<td>10:1</td>
<td>10:1-20:1</td>
<td>Magnesium</td>
<td>1.9 mEq/L</td>
<td>1.6-2.4 mEq/L</td>
</tr>
<tr>
<td>Calcium</td>
<td>8.9 mg/dL</td>
<td>8.5-10.5 mg/dL</td>
<td>Phosphorus</td>
<td>3.5 mEq/L</td>
<td>2.5-5.2 mEq/L</td>
</tr>
<tr>
<td>Total Protein</td>
<td>8.0 g/dL</td>
<td>6.0-8.3 g/dL</td>
<td>Creatinine Clearance</td>
<td>104.5 mL/min</td>
<td>97-137 mL/min (male)</td>
</tr>
<tr>
<td>Albumin</td>
<td>4.9 g/dL</td>
<td>3.5-5.2 g/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(Normal values from Pagana & Pagana, 2010)

### Table 2: Complete Blood Count (CBC) with Differential

<table>
<thead>
<tr>
<th>Lab</th>
<th>Results</th>
<th>Normal Values</th>
<th>Lab</th>
<th>Results</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>12.3 k/mm3</td>
<td>4.5-10.5 k/mm3</td>
<td>Band Neutrophils</td>
<td>2.3%</td>
<td>2-5%</td>
</tr>
<tr>
<td>RBC</td>
<td>5.8 m/mm3</td>
<td>4.7-6.1 m/mm3 (male)</td>
<td>Lymphocytes</td>
<td>15.2%</td>
<td>14.0-51.0%</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>15.9 g/dL</td>
<td>14.0-17.5 g/dL (male)</td>
<td>Monocytes</td>
<td>2.5%</td>
<td>2-8%</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>46.3 %</td>
<td>40.7-50.3% (male)</td>
<td>Eosinophils</td>
<td>1.2%</td>
<td>1-3%</td>
</tr>
<tr>
<td>MCV</td>
<td>88.4 fL</td>
<td>80.0-100.0 fL</td>
<td>Basophils</td>
<td>0.1%</td>
<td>0.0-1%</td>
</tr>
<tr>
<td>MCH</td>
<td>28.6 pG</td>
<td>27.0-31.0 pG</td>
<td>Absolute Segmented Neutrophil</td>
<td>10 k/mm3</td>
<td>1.5-8.0 k/mm3</td>
</tr>
<tr>
<td>MCHC</td>
<td>33.7 g/dL</td>
<td>32.0-36.0 g/dL</td>
<td>Absolute Lymphocyte</td>
<td>1.2 k/mm3</td>
<td>0.9-4.1 k/mm3</td>
</tr>
<tr>
<td>RDW</td>
<td>13.5%</td>
<td>9.0-15.0%</td>
<td>Absolute Monocyte</td>
<td>0.6 k/mm3</td>
<td>0.2-1.1 k/mm3</td>
</tr>
<tr>
<td>Platelets</td>
<td>388,000 k/mm3</td>
<td>150,000-400,000 k/mm3</td>
<td>Absolute Eosinophil</td>
<td>0.2 k/mm3</td>
<td>0.0-0.6 k/mm3</td>
</tr>
<tr>
<td>Segmented Neutrophils</td>
<td>78.7%</td>
<td>40.0-76.0%</td>
<td>Absolute Basophil</td>
<td>0.1 k/mm3</td>
<td>0.0-0.3 k/mm3</td>
</tr>
</tbody>
</table>
WEB CASE STUDY GOUT

(Normal values from Pagana & Pagana, 2010)

Table 3. Additional Laboratory Findings: Erythrocyte Sedimentation Rate (ESR), Rheumatoid Factor (RF), Antibodies to Citrulline-containing peptides (anti-CPP), Serum Uric Acid (sUA), 24-hour Urine Uric Acid

<table>
<thead>
<tr>
<th>Lab</th>
<th>Results</th>
<th>Normal Values</th>
<th>Lab</th>
<th>Results</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>ESR</td>
<td>8 mm/hr</td>
<td>&lt;15 mm/hr (males under 50 yrs)</td>
<td>sUA</td>
<td>7.2 mg/dL</td>
<td>3.4-6.8 mg/dL</td>
</tr>
<tr>
<td>RF</td>
<td>17 U/L</td>
<td>&lt;60 U/L</td>
<td>24-hr urine uric acid</td>
<td>0.5 mmol</td>
<td>1.5-4.4 mmol</td>
</tr>
<tr>
<td>anti-CPP</td>
<td>6 U/L</td>
<td>&lt;20 U/L</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(Normal values from Pagana & Pagana, 2010)

Table 4. Radiographic Study

| X-ray of Bilateral Feet | Results: Asymmetrical and articular edema of the soft tissues surrounding the base of the right great toe in the first metatarsophalangeal joint. No evidence of fracture in either foot. No bone erosion noted in either foot. No edema or other abnormalities noted in left foot. |

Table 5. Synovial Fluid Analysis and Polarized Light Microscopy from Arthrocentesis of Right Foot Metatarsophalangeal Joint

<table>
<thead>
<tr>
<th>Results</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clarity</td>
<td>Translucent</td>
</tr>
<tr>
<td>Color</td>
<td>Yellow</td>
</tr>
<tr>
<td>Viscosity</td>
<td>Low</td>
</tr>
<tr>
<td>WBC</td>
<td>3500 mm(^3)</td>
</tr>
<tr>
<td>PMNs</td>
<td>58%</td>
</tr>
<tr>
<td>Gram Stain Culture</td>
<td>Negative</td>
</tr>
<tr>
<td>Total Protein</td>
<td>4 g/dL</td>
</tr>
<tr>
<td>Glucose</td>
<td>29 mg/dL</td>
</tr>
<tr>
<td>Presence of Crystals</td>
<td>Monosodium urate crystals</td>
</tr>
</tbody>
</table>
WEB CASE STUDY GOUT

(Normal Values from Mundt & Shanahan, 2011)

**Diagnosis**

After consideration of findings from detailed history and physical, laboratory, and radiography results, the patient is diagnosed with acute gouty arthritis. The American College of Rheumatology (ACR) (ACR, 2012) identifies the definitive diagnosis of gout as the presence of tissue deposition of monosodium urate crystals in extracellular fluids of the joint, as noted in the synovial fluid. Other diagnostic criteria of gout include sudden onset of monoarticular arthritis, progression of maximal inflammation within one day, and unilateral attack, pain, redness, and swelling involving the first metatarsophalangeal joint (ACR, 2012; Peláez-Ballestas et al., 2010; Withers, George, & Keenan, 2012).

In addition, 90-100% of gout cases can be attributed to hyperuricemia (sUA > 6.8 mg/dL), which is acquired by the patient from eating a high-purine diet and taking low-dose salicylate (aspirin 81 mg) and thiazide diuretic (hydrochlorothiazide) medications (Schlesinger, 2005). Diuretic-induced hyperuricemia is caused by the undersecretion and promotion of urate reabsorption by the proximal tubules of the kidneys (El-Sheikh, van den Heuvel, Koenderink, & Russel, 2008). The laboratory finding of the 24-hour urine uric acid suggests that decreased levels of uric acid is excreted by the kidneys, thus increasing sUA levels to 7.2 mg/dL and promoting uric acid precipitation. Four clinical stages of gout exist: asymptomatic hyperuricemia, acute gouty arthritis, intercritical gout (frequency between gout attacks), and chronic tophaceous gouty arthropathy. The patient was likely maintained in an asymptomatic clinical stage until recently when the cardiologist added the medication hydrochlorothiazide.

**Differential Diagnosis**
Differential diagnoses are important to consider in evaluating and treating the disease. Some of the other possible diagnoses for this patient include rheumatoid arthritis, septic arthritis, osteoarthritis, reactive arthritis, trauma/soft tissue injury, viral infection, cellulitis, and calcium pyrophosphate dehydrate deposition disease (CPPD) (Sundy, 2012).

Rheumatoid arthritis and reactive arthritis are usually clinically diagnosed when other causes of diseases are eliminated. The lab findings indicate insignificant values of both RF and anti-CPP, strongly suggesting against a diagnosis of rheumatoid arthritis. In addition, a symmetrical pattern of inflammation that is exacerbated after a long period of rest (e.g. on awakening in the morning) is indicative of rheumatoid arthritis (National Institute of Arthritis and Musculoskeletal and Skin Diseases [NIAMS], 2013). A lack of preceding infection caused by Chlamydia trachomatis, Yersinia, Escherichia coli, or Clostridium difficile pathogens can rule out the diagnosis of reactive arthritis (Hannu, 2011; Townes, 2010).

The synovial fluid analysis can assist in eliminating other differential diagnosis. Polarized light microscopy detected the presence of monosodium urate crystals and not calcium pyrophosphates dehydrate precipitation, thus eradicating the diagnosis of CPPD (Malik, Schumacher, Dinnella, & Clayburne, 2009). Non-inflammatory articular conditions, such as osteoarthritis, trauma, and viral infection, can be ruled out because the WBC count is <2000 mm3 and PMNs are <75% in the synovial fluid. Furthermore, no evidence of trauma was noted on the x-ray of the right foot. Although the disease process of septic arthritis and cellulitis are inflammatory, similar to gouty arthritis, a negative fluid culture leads to the elimination of any infectious causes of the clinical manifestations (ACR, 2012; Mundt & Shanahan, 2011). Unlike gouty arthritis, the erythema and edema associated with joint cellulitis enlarges beyond the joint area and is polyarticular in nature (Simosen et al., 2006).
Plan

According to the ACR recommendations, the core therapeutic measure in gout is educating the patient on diet and lifestyle modification and eliminating prescription medications that induce hyperuricemia. Urate-lowering therapy (ULT) is initiated if a patient meets one of the recommended criteria which includes presence of tophi noted in the physical examination or an imaging study, two or more gout attacks a year, stage II or worse chronic renal disease, and history of urolithiasis (ACR, 2012). The criterion for increased sUA is not specified in the guidelines because during an acute gout attack, sUA is usually increased and is not a reliable indicator. Furthermore, ULT is not recommended during an acute phase of a gout attack because gout attacks are exacerbated during the initial phases of the treatment (Curiel & Guzman, 2012). The patient does not have a history of renal disease or urolithiasis and tophi are not visible in any joint including the right great toe. The patient is also not a candidate for ULT as this is the first presenting incidence of gouty arthritis. Appropriate treatment according to the guidelines at this time consists of an anti-inflammatory medication regime for this patient (ACR, 2012).

Non-Pharmacological Therapy

The most effective long-term management is healthy lifestyle adaptation (ACR, 2012). A diet high in red meats, seafood, and alcohol contain purines which metabolizes into an end product of uric acid (Schlesinger, 2005). The patient will be instructed to avoid his favorite foods of lobster and steak and to consume a low-purine diet (four ounces of meat daily), especially during an acute attack. Other foods to limit are organ meats, herring, anchovies, mackerel, and other items high in saturated fats which decrease the body’s capacity to excrete uric acid. Plant-based protein such as nuts, beans, and legumes and low-fat dairy products are low in saturated fats and are encouraged (ACR, 2012). Educating the patient to abstain from alcohol is also
WEB CASE STUDY GOUT

critical for averting future gout attacks. The patient will be offered a family counselor to assist with the current stress the patient is experiencing. Encouraging patient to stay well hydrated by drinking eight to ten, eight-ounce glasses a day can assist with uric acid elimination (ACR, 2012; Schlesinger, 2012).

Other non-pharmacologic therapies focus on resting the foot for one to two days and continuing to apply ice packs for comfort. Two risk factors of gout that the patient can self-manage are losing weight to achieve a normal BMI of less than 25 kg/m2 and controlling blood pressure by restricting salt intake and exercising (ACR, 2012; Schlesinger, 2012). Soriano, Rothenbacher, Choi, and Rodriguez (2011) suggested 62% of overweight individuals and 18% of hypertensive individuals are more likely to develop gout than individuals who do not have these diagnoses.

The ACR (2012) recommendations also state to carefully consider eliminating potential urate-elevating medications, such as thiazide diuretics, if disease processes can be managed by another drug. For this patient, the drug hydrochlorothiazide will be discontinued and the patient will be instructed to follow-up with the cardiologist for a more appropriate plan to control the high blood pressure. Losartan, an angiotensin II receptor blocker, is suggested as an alternative treatment to thiazide diuretics in hypertensive gout patients (Gibson, 2013; Ruilope, 2012). The current recommendations state low-dose salicylates (aspirin <325 mg/daily) do not effect plasma urate significantly and can be continued as cardiovascular disease prophylaxis in gout patients (ACR, 2012; Greener, 2011).

**Pharmacological Treatment**

For mild or moderate gout attacks (pain level that is less than or equal to six) and those involving one or a few small joints, the first-line recommendation is to initiate monotherapy with
WEB CASE STUDY GOUT

anti-inflammatory drugs such as oral non-steroidal anti-inflammatory drugs (NSAIDs), systemic corticosteroids, or oral colchicine (ACR, 2012). The treatment chosen should consider patient’s preferences and associated comorbidities. To optimize care, therapy should be started within 24 hours of acute gout attack onset. If patients do not adequately respond to initial therapy, a second agent is an appropriate option (ACR, 2012).

**Drug of Choice.** After careful consideration from data gathered from history and physical, the diagnosis, insurance coverage, and current guidelines for the diagnosis, the only approved Food and Drug Administration (FDA) single agent colchicine (Colcrys™) in the class of anti-gout will be the drug of choice for this patient.

**Administration.** The medication can be taken without regards to meals, but with adequate water. The ACR (2012) recommends to treat acute gout attacks with a loading dose of 1.2 mg of colchicine (Colcrys™), followed by 0.6 mg one hour later for a total of 1.8 mg maximum recommended dosage in one hour period (FDA, 2009; Lexi-Comp, 2013). The dose cannot be repeated for at least three days (Yang, 2010).

**Rationale.** Due to the history of PUD, the patient is not a candidate for treatment with NSAIDs. Corticosteroids were not chosen as first-line treatment for this patient after considering past medical history of hypertension. Some of the side effects of oral corticosteroids include fluid retention, increased blood pressure, and weight gain (Sarnes et al., 2011).

This drug was also chosen because symptom relief usually occurs within 18-24 hours, unlike NSAIDs and corticosteroids (Lexi-Comp, 2013; Yang, 2010). In randomized control trials (RCTs) for drug efficacy in acute gout treatment, the use of NSAIDs (etoricoxib, indomethacin, and naproxen) or prednisone resulted in 50% pain reduction in 48-90 hours (Janssens et al.,
Pharmacodynamics. Colchicine reduces leukocyte chemotaxis and phagocytosis and inhibits the construction and release of glycoprotein, a protein generated from phagocytosis of urate crystals. The medication also decreases pH in the tissues by inhibiting oxidation of glucose and lactic acid from leukocytes, thus resulting in inhibition of urate crystal accumulation (Lexi-Comp, 2013; Micromedex, 2013).

Pharmacokinetics. Onset of action of colchicine is 18-24 hours, and time to peak is half an hour to three hours (Lexi-Comp, 2013). Terminal half-life of 1.8 mg of colchicine is 23.6 hours (Yang, 2010). The medication is distributed in leukocytes, kidney, spleen, and liver (volume of distribution is 5-8 L/kg). Thirty-nine percent of the drug is bound to protein. Metabolism of the drug occurs via hepatic system, in CYP3A4, and 40-65% of the unabsorbed, unchanged drug is excreted via urine (Lexi-Comp, 2013).

Dosage Forms and Strengths. Colchicine (Colcrys™) is available in 0.6 mg tablets (FDA, 2009).

Uses. Colchicine is approved by the FDA for the indication of prophylaxis and treatment of gout and Familial Mediterranean Fever (FMF) in adults. The medication is a non-analgesic and should not be treated for other pain symptoms (FDA, 2009).

Contraindications. Concomitant use of colchicine with p-glycoprotein (e.g. cyclosporine) or strong CYP3A4 inhibitors (e.g. clarithromycin and erythromycin) in patients with renal or hepatic impairment can result in colchicine toxicity by tripling colchicine blood levels, leading to fatality (FDA, 2009; Lexi-Comp, 2013; Terkeltaub et al., 2010).
WEB CASE STUDY GOUT

**Storage and Stability.** The medication should be stored in a tight container and protected from light at room temperature between 68-77° Fahrenheit (Lexi-Comp, 2013).

**Cost.** Colchicine (ColcrySTM) oral 60 tablets of 0.6mg costs $349.20 (Lexi-Comp, 2013).

**Adverse Effects.** The most common adverse reactions for treatment of gout attacks are related to gastrointestinal disorders such as diarrhea (23%) and abdominal cramping or vomiting (26%) (Terkeltaub et al., 2010). Another common side effect is pharyngolaryngeal pain. Less common side effects include alopecia, aplastic anemia, pancytopenia, and muscle weakness (FDA, 2009; Lexi-Comp, 2013). Colchicine toxicity is possible and symptoms include severe diarrhea or vomiting, muscle weakness, numbness/tingling in toes or fingers, increased infections, unusual bleeding/bruising, and paleness or gray color of tongue, lips, or hands (FDA, 2009).

**Drug-Drug Interactions.** Dose adjustment is required for HMG-CoA reductase inhibitors because concomitant administration with colchicine may increase the levels of these medications. In contrast, colchicine decreases the effects of aripiprazole, cyanocobalamin, axitinib, and saxagliptin (Lexi-Comp, 2013). Levels of colchicine may be increased by the following: CYP3A4 moderate inhibitors, CYP3A4 strong inhibitors, digoxin, fibric acid derivatives, P-glycoprotein/ABCB1 inhibitors, telaprevir, and fosamprenavir. However, P-glycoprotein/ABCB1 inducers decrease the effect of colchicine (Lexi-Comp, 2013).

**Nutrition/Herbal Interactions.** Grapefruit juice should not be taken with colchicine to avoid increased serum concentrations of colchicine. Macrocytic anemia or neurologic changes may occur due to the effect of colchicine on cyanocobalamin absorption (Lexi-Comp, 2013).

**Monitoring Parameters for Colchicine.** Dose adjustment is not required for the treatment of gout attacks in mild to moderate renal or hepatic dysfunction, but colchicine
treatment course should not be administered for more than once every two weeks. The patient will be monitored for fatal colchicine toxicity resulting from severe renal (creatinine clearance <30 mL/min) and hepatic impairment. Depending on the frequency of course treatments, routine renal and hepatic function tests will be performed every three to six months. A routine CBC with differential is also required for monitoring possible blood dyscrasias, such as myelosuppression, pancytopenia, and aplastic anemia, which can be caused by colchicine (FDA, 2009).

Follow-Up/Patient Education. The patient will be educated on the correct administration of the treatment course, possible side effects to report, and drug-drug/drug-nutritional interactions. The patient will be advised that gout is an ongoing disease and that medication to treat acute or chronic gout will have to be taken infinitely despite improvement of symptoms (ACR, 2012; Schumacher et al., 2009). The patient will be informed to contact the prescriber in two days if the signs and symptoms of acute gout do not resolve for reassessment of the treatment plan. To ensure the patient took the medication correctly, the patient will be re-educated on the treatment course and possible drug-drug and drug-nutritional interactions. If indicated, the therapy will be repeated in three days after evaluating hepatic and renal function, and CBC for blood dyscrasias (Yang, 2010). The ACR (2012) also suggested to consider either adding or supplementing the initial treatment with a second recommended agent if the initial therapy was inadequate (defined by the ACR as <20% improvement in pain score within 24 hours or <50% improvement in pain score >24 hours after initiating therapy).

APN Authority to Prescribe. An Advanced Practice Nurse (APN) with a current CTP/CTP-E is able to prescribe anti-gout agents, including colchicine, within APN scope of practice (Ohio Board of Nursing [OBN], 2013).
Clinical Study

Despite the extensive use of colchicine, the evidence basis for the drug’s efficacy remains limited. Only two randomized, placebo-controlled trial studies evaluating oral colchicine therapy for acute gout have been conducted. The earlier study conducted in 1987 used a different colchicine dosing regimen than the current FDA approved dose, therefore the study’s results are outdated. The objective of the 2010 AGREE study conducted by Terkeltaub et al. (2010) was to compare low-dose colchicine and high-dose colchicine with placebo in acute gout flares. The multicenter (incorporated 54 centers in the United States), randomized, double-blind, placebo-controlled, parallel-group, dose-comparison study was performed between April 2007-October 2008.

A total of 575 patients were randomized into the trial, but only 185 of those patients had an eligible gout attack and took the study medication. The participants considered in the study were male and postmenopausal female patients ≥ 18 years of age with an ACR (2012) confirmed diagnosis of acute gout, and history of two or more gout flares within the previous 12 months. Demographic (age, sex, race) and other baseline characteristics (concomitant use of ULT, alcohol use, number of gout attacks) were comparable. However, the majority of the participants were ~50 year old white men with a mean elevated sUA of 8.5 mg/dL. An eligible gout attack included flare onset and study medication administration within 12 hours, presence of inflammation, and a pain rating of four or more on a zero to ten pain scale. Randomization occurred in three treatment groups: 1) low-dose colchicine, 1.2 mg followed by 0.6 mg in one hour, followed by placebo doses every hour for five hours 2) high-dose colchicine, 1.2 mg followed by 0.6 mg every hour for six hours or 3) placebo, two placebo capsules initially, followed by one placebo capsule every hour for six hours (Terkeltaub et al., 2010).
WEB CASE STUDY GOUT

**AGREE Efficacy Findings**

The AGREE study suggested both treatment group regimens were statistically significant than the control group. The high-dose colchicine group and the low-dose colchicine group had 32.7% (odds ratio [OR] 2.64 [95% CI 0.22-0.92], \( P=0.034 \)) and 37.8% (OR 3.3 [95% CI 1.41-7.77], \( P=0.005 \)), respectively, of participants that responded to ≥ 50% reduction of pain at 24-hour after the administration of the first dose versus the placebo group (Terkeltaub et al., 2010).

**AGREE Safety Findings**

The most common adverse events (AEs) noted in the study were diarrhea, nausea, and vomiting. However, all AEs in the low-dose and placebo groups were reported as mild to moderate in intensity while the high-dose group had severe cases of the AEs. Diarrhea occurred in 76.9% (OR 21.3 [95% CI 7.9-56.9]) of participants in the high-dose group, 23% (OR 1.9 [95% CI 0.8-4.8]) in the low-dose group, and 13.6% in the placebo group. Nausea presented in 17.3% (OR 3.9 [95% CI 1.0-15.3]), 4.1% (OR 0.8 [95% CI 0.2-4.1]), and 5.1% of the high-dose, low-dose, and placebo groups, respectively. Participants in high-dose group (17.3%) also reported vomiting. Vomiting did not occur in the low-dose and placebo groups (Terkeltaub et al., 2010).

In conclusion, low-dose colchicine treatment efficacy and safety is as effective as high-dose colchicine treatment, and both regimens were suggested as more effective for treatment of acute gout than placebo. Another benefit of using low-dose colchicine regimen is the possible reduction of strong drug-drug interactions that can cause fatality from colchicine toxicity. The low-dose treatment course exposes patients to two thirds less colchicine than a higher-dose exposure (Terkeltaub et al., 2010).

**AGREE Ethics**
WEB CASE STUDY GOUT

All participants were given written informed consent and the study was approved by the Sterling Institutional Review Board. The study upheld FDA regulations and International Conference on Harmonization guidelines. The AGREE study design recognized the ethical issues of not adequately treating the placebo group. Therefore, the study limited the primary end point to 24 hours that a patient would be left untreated (Terkeltaub et al., 2010). In addition, the researchers allowed the administration of other rescue drugs (e.g. NSAIDs) if the pain was intolerable after taking at least the first dose of the study drug. The patients who took a rescue drug were excluded from the study data. A significantly increased number of patients receiving placebo treatment took at least one rescue drug when compared to the high-dose (OR 0.53 [95% CI 0.25-1.14], P=0.103) and low-dose group (OR 0.45 [95% CI 0.22-0.92], P=0.027), but difference between the placebo and high-dose group was insignificant (P=0.103) (Terkeltaub et al., 2010).

Issues

In selecting the drug of choice, the practitioner must consider the correct diagnosis, allergies, previous effective treatment (if any), and existing comorbidities. Other factors such as patient’s willingness and ability to adhere with treatment regimen, level of literacy, ability to afford treatment costs, and extent of family support (e.g. for transportation to appointments/pharmacy) is also critical for assessment. The patient is well-educated, denies financial stress, receives sufficient assistance from his wife, and is conscious of his medical care as observed by the practitioner during the history and physical exam. Due to the patient’s history of hypertension and PUD, other first-line treatment agents of corticosteroids and NSAIDs were eliminated. The two-dose administration of colchicine is convenient for the patient who states he
WEB CASE STUDY GOUT

has a hectic lifestyle. Greater adherence is associated with the ease of medication administration, and can lead to better treatment outcomes (Schumacher et al., 2009).

A medical practitioner should routinely be updated on current guidelines of treatment and practice by accessing RCTs and evidenced-based practice research studies. Although colchicine is an approved drug of choice as a first-line treatment for gout and has been used since antiquity, FDA has recognized only two RCTs (first study conducted in 1987; the second study in 2010 as described above) have been completed to evaluate the efficacy of the drug in the treatment of acute gout. In the United States, approximately eight million people are diagnosed as having gout. Gout is increasing in prevalence due to associated comorbidities, such as hypertension, obesity, and chronic kidney disease (ACR, 2012). Further studies are warranted to validate and compare the efficacy of colchicine with other ACR-approved first-line medications for acute gout. Additional studies are also necessary to determine if concomitant ULT, sites of arthritis, or other doses affect the efficacy and safety of the drug.

According to the Arthritis Foundation (2013), the fundamental goals of acute gout treatment include providing rapid relief of pain and inflammation, averting future attacks, and preventing the development of tophi and comorbidities (i.e. kidney stones, renal disease). Symptoms of acute gout are debilitating, involve extreme pain and can take up to one week to resolve and are associated with decreased quality of life (ACR, 2012). As observed in this case study, acute gout can result in loss of mobility, interrupted sleep, emotional stress, and work and social limitations (Schumacher et al., 2009). If patient is unable to work due to pain, financial strains can also evolve. Therefore, the ACR (2012) recommends initiation of treatment that can alleviate disease symptoms rapidly, within 24 hours, to improve quality of life. Colchicine was
WEB CASE STUDY GOUT

an appropriate choice for the treatment of this patient since the ACR recommends initiation of the drug within 36 hours of symptom onset (ACR, 2012).

The ACR (2012) reports a significant shortfall in gout patient education. Patient education is of critical importance in treatment adherence. A combination of verbal, written, and teach-back methods should be utilized for optimum learning (Mayoux-Benhamou et al., 2008). Medical jargon should be avoided and disease process and treatment should be explained in simple, preferably sixth to eighth grade language (Castro, Wilson, Wang, & Schillinger, 2007). During the office visit, the patient will be given pamphlets and educated on the triggers of acute gout attacks and the importance of maintaining healthy weight with diet and lifestyle modifications. Avoiding alcohol and fast-food and following a low-purine diet will be reiterated. Further education will be provided on some commonly prescribed drugs (e.g. clarithromycin and erythromycin) that interact with colchicine and can result in fatality. A referral to a family counselor will be offered to assist with alleviating patient’s stress level. The patient will also be instructed to initiate colchicine treatment regimen upon first clinical signs and symptoms of an acute gout attack (within 24 hours) without needing to consult the practitioner first (but should call if the current signs and symptoms do not resolve in two days) (ACR, 2012). However, the practitioner should be immediately notified of the acute gout occurrence in order to schedule a follow-up appointment to assess the need for ULT. The patient will be notified that ULT is a possible option if the current treatment plan is not effective for gout management and/or if the patient has two or more gout attacks in a year (ACR, 2012).

A potential professional issue can arise between practitioners since the medication hydrochlorothiazide was discontinued by a practitioner who did not initially prescribe the medication. A courtesy follow-up call to the cardiologist is optional to provide an update on
WEB CASE STUDY GOUT

patient condition and the reason for the discontinuation of the medication. If appropriate and if preferred by the patient, an appointment with the cardiologist can be scheduled for the patient to ensure proper follow-up of blood pressure management.

**Legal and Ethical Considerations**

An APN must consider legal and ethical implications when prescribing treatment. A valid, current certificate to prescribe (CTP) is required and all care provided should be within the APN’s standards, scope, and practices (Kleinpell, Hudspeth, Scordo, & Magdic, 2012; OBN, 2013). The APN is solely responsible for understanding the current treatment and formulary guidelines, notifying patients of medication recalls, and adjusting the treatment plan with alternate therapy if required. In diagnosing and treating this patient, OBN (2013) medication formulary and the ACR (2012) guidelines for gout classification and management were utilized.

Ethical principles should be upheld to prevent legal allegations and to respect all persons. The philosophy of the *Belmont Report* was followed during the APN-patient interaction. The value respect for all persons was incorporated by treating the patient as an autonomous agent, empowering him to make own choices concerning his health (Polit & Beck, 2012). An informed consent was also signed by the patient before the start of the office visit, promoting voluntariness of the decision to receive care. Information, such as patient education, was provided to the patient to facilitate understanding of the disease process to assist in positive decision making. Education included, but was not limited to, the disease process, personal risk factors, and treatment plan (i.e. goals of treatment, colchicine administration, common side effects to report, precautions, when to follow-up, etc.). Providing adequate education results in greater adherence to the treatment plan, and improves patient outcomes.
WEB CASE STUDY GOUT

An APN is obligated to provide treatment that aligns with the standards of beneficence and non-maleficence, doing no harm while maximizing benefits and decreasing associated risks (Polit & Beck, 2012). Confidentiality and privacy of the patient was maintained to reduce harm caused by the unintentional disclosure of private information. Before prescribing the medication colchicine, a detailed history and physical examination including pertinent laboratory findings were obtained to assist in diagnosis and to evaluate the general health of the patient. The administration of FDA-approved colchicine (Colcrys™) was an appropriate choice after considering normal hepatic and renal function tests, and existing comorbidities of hypertension and PUD. Furthermore, in hope of preventing future gout attacks, the potential harmful medication hydrochlorothiazide was discontinued and the patient was recommended to follow-up with the cardiologist to assess for alternate drug therapy for blood pressure management. An update via phone was provided to the cardiologist to ensure continuity of care and accurate data keeping. Finally, the patient was treated fairly and justly without regards to sex, age, race, or other characteristics such as personal lifestyle choices. Valid, truthful information on the prognosis of the disease was discussed with the patient and the possibility of altering the medication regime to include ULT if current treatment is unsuccessful.

An APN must consider multiple factors in order to select the most appropriate treatment regime that will result in optimum patient outcomes. The core of the nursing culture is to advocate for patients and to constantly seek opportunities to improve patient-delivered care. A knowledgeable practitioner, with expertise and evidenced-based practice, can provide high-quality, competent patient care which integrates aspects of psycho-emotional, biophysical, socio-environmental, ethical, and legal factors.
WEB CASE STUDY GOUT

Discussion Questions

The patient returns to the clinic six months later within the year with another acute gout attack. The serum uric acid (sUA) level is now 8.6 mg/dL and tophi are not detected on exam or x-ray. After assessment of the patient, it is established that an urate-lowering therapy (ULT) is warranted.

1) According to the ACR (2012) guidelines, what is the appropriate first-line ULT pharmacological treatment for this patient? Include the preferred target sUA level for treatment, drug’s efficacy, initial drug dose, titration parameters, follow-up monitoring/precautions, and mechanism of action.

2) Often with ULT, concomitant use of colchicine is prescribed for prophylaxis of attacks of acute gout. Will the patient be able to continue treatment with colchicine for acute gout attacks if prophylaxis colchicine therapy is initiated? Explain your answer. Also, explain why the prophylaxis treatment is indicated and what is the recommended dosage and duration of the prophylaxis therapy according to the ACR (2012) guidelines.
WEB CASE STUDY GOUT

References


WEB CASE STUDY GOUT


WEB CASE STUDY GOUT


*Rheumatoid arthritis*. Retrieved from
http://www.niams.nih.gov/Health_Info/Rheumatic_Disease/default.asp

Ohio Board of Nursing (OBN). (2013). *The formulary developed by the Committee on Prescriptive Governance*. Retrieved from


Ruilope, L. M. (2012). Antihypertensives in people with gout or asymptomatic hyperuricemia: Losartan and calcium channel blockers are most effective owing to their uricosuric properties. *BMJ: British Medical Journal*, 344 (7843), 9-10.
WEB CASE STUDY GOUT


WEB CASE STUDY GOUT
