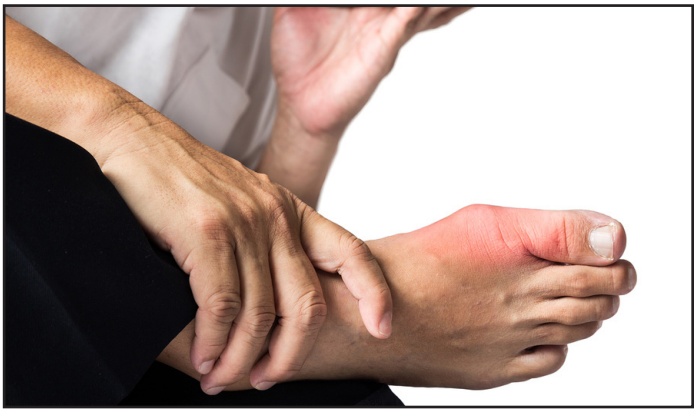
CE PRN

PHARMACY CONTINUING EDUCATION FROM WF PROFESSIONAL ASSOCIATES

"Update: Gout"

June 2016



We periodically review & update consideration of gout for two reasons: First: it is the most common inflammatory joint disease; 2nd—because the number of prescriptions for treating gout are on the increase. Our goals are to revisit this important topic & review treatment options. This lesson provides 1.25 hours (0.125 CEUs) of credit, and is intended for pharmacists & technicians in all practice settings. **The program ID # for this lesson is 707-000-16-006-H01-P for pharmacists & 707-000-16-006-H01-T for technicians.**

Participants completing this lesson by May 31, 2019 may receive full credit. Release date June 1, 2016.

To obtain continuing education credit for this lesson, you must answer the questions on the quiz (70% correct required), and return the quiz. Should you score less than 70%, you will be asked to repeat the quiz. Computerized records are maintained for each participant.

If you have any comments, suggestions or questions, contact us at the above address, or call 1-847-945-8050. **Please write your name, NABP eProfile (CPE Monitor®) ID Number & birthdate (MM/DD) in the indicated space on the quiz page.**

The objectives of this lesson are such that upon completion participants will be able to:

Pharmacists:

1. Recognize the symptoms of gout.
2. Explain how certain foods contribute to gout.
3. List the steps that lead to the metabolism of purine & formation of uric acid.
4. List the risk factors that contribute to gout.
5. Discuss medications used in treating gout.

Technicians:

1. Describe symptoms associated with gout.
2. Explain how certain foods contribute to gout.
3. Discuss medications used in treating gout.

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NOTE: ONE OF OUR FUTURE TOPICS THIS YEAR WILL BE ON THE OPIOID CRISIS OF ABUSE.

OVERVIEW

Gout is a complex metabolic type of inflammatory arthritis that can develop in some individuals with uric acid blood concentration of 6.8 mg/dL and over. However, in many cases elevated uric acid in the blood is asymptomatic and may never lead to gout. The disease is characterized by episodic attacks of sudden, acute, excruciating, burning pain, redness, warmth and tenderness in a joint, often at the base of the big toe. It can affect the insteps, knees, ankles, heels, wrists and elbows. Gout is the most painful type of arthritis and greatly impacts the quality of a patient's life. The word "gout" was derived from the Latin word "gutta," meaning a drop. In the middle ages it was stipulated that the disease occurs as a result of diffusion of a toxic substance from the blood into a joint. The inflammatory reaction in a joint is triggered by needle-like crystals of **monosodium urate (MSU)** that are deposited in and around a joint, bone, bursa, ligament or tendon. The MSU crystals may be deposited in other body tissue such as the kidneys which may result in the formation of kidney stones (calculi). The initial inflammation usually begins uniarticularly (single bone) affecting the 1st metatarsophlangeal joint located between the metatarsal bones of the foot and proximal (nearest) phalanges (bones that make up the toes).

PATHOPHYSIOLOGY

Gout is associated with build-up of uric acid in the blood (hyperuricemia). The formation of uric acid occurs as a result of breakdown of purines, some of which are produced by the body, and the remainder is generated from foods. MSU concentration in the body is always near its limit of solubility and is in a fluctuating balance between its production in the body and

its elimination by the kidneys. Deposition of MSU in a joint occurs when urate concentration exceeds its solubility in the interstitial fluid and plasma. Under normal conditions uric acid in the blood is excreted through the kidneys into the urine.

Gout is exclusively present in humans and some primates due to the absence of uricase, an enzyme that breaks down uric acid and converts it to allantoin, a very soluble product that is eliminated in the urine. When the concentration of uric acid in the blood exceeds 6.8 mg/dL, precipitation of SMU may take place in the joints of certain individuals to form gouty arthritis. Over time, the MSU crystals in the joints may aggregate to form microscopic masses termed tophi. Gradually these microscopic tophi increase in size and can be seen as nodules or lumps under the skin of a joint. The crystals or tophi elicit, amplify and intensify an inflammatory process that occurs as a result of stimulation of the synthesis and release of inflammatory mediators that lead to characteristic gouty arthritis. Cytokines, chemokines and protease are associated with crystals or tophi—induced inflammation that may become chronic leading to synovitis (inflammation of the synovial membrane of the joint), bone and cartilage damage. Following their deposition, the MSU crystals may remain dormant in the tissue for a long time before unpredictably provoking an intense and abrupt episode of acute, painful arthritic gout. A flare-up manifests itself by characteristic sharp pain, swelling, redness, and tenderness in the joint area.

Uric acid is the normal end product of purine metabolism. Purines (adenosine and guanosine) are naturally occurring chemicals found in practically all body cells and in foods from plants and animals. They are an essential part of the chemical structure of genes (DNA, RNA) in humans, animals and plants. Purine-rich foods include organ meats such as liver and kidney, sardines, anchovies, wine, beer, peas, pork, red meat, cauliflower, asparagus and sweetbreads. Uric acid is useful as it acts as an antioxidant to protect damage to blood vessels. Faulty metabolism of purine nucleosides (adenosine, and guanosine) can lead to overproduction of uric acid. Purine metabolism occurs as a result of several steps. Adenosine is converted to inosine through the action of the enzyme, adenosine deaminase. Inosine, in turn, is converted to hypoxanthine by the enzyme, purine nucleoside phosphorylase. The enzyme, xanthine oxidase, converts hypoxanthine to xanthine which converts to uric acid. The pathway for the metabolism of guanosine to uric acid is somewhat similar to that of adenosine. It begins with the conversion of guanosine to guanine by the enzyme, purine nucleoside phosphorylase. Guanine is then converted to xanthine by the enzyme, guanine deaminase. Ultimately, xanthine is converted to uric acid through the enzyme, xanthine oxidase. The uric acid produced as a result of the pathway of adenosine and guanosine remains in the blood due to the absence of the enzyme, uricase, whose function is to breakdown uric acid. About half of the purine found in the body is destroyed and replaced each day endogenously and exogenously via the intake of foods. Strict avoidance of a purine-rich diet may reduce serum uric acid by at least 1 mg/dL.

RISK FACTORS

1. **Hyperuricemia is a major risk factor for causing gout.** That being stated, there are persons whose uric acid blood level is more than 6.8 mg/dL, yet they do not develop gout. It is estimated that 1 in 5 individuals with hyperuricemia will not encounter gouty arthritis. Patients with uric acid blood levels of 9 mg/dL have a 50% probability of experiencing gout. The chance to develop gout for a person with uric acid blood level concentration of 12 mg/dL is 75%.

2. **Family history of gout.**
3. **Gender and age:** Even though gout may occur at any age, it mostly develops in men over 45 years of age and in women after menopause. The incidence is higher in men. However, after menopause the incidence among men and women becomes almost equal.
4. **Obesity and excess body fat** especially in persons with body mass index (BMI) of 30 and over.
5. **Excessive consumption of alcohol.**
6. **Significant ingestion of foods rich in purine.** It has been reported that such foods are responsible for 12% of gout cases.
7. **Comorbidity with metabolic syndrome** (hypertension, hyperlipidemia and diabetes).
8. **Hereditary factors.**
9. **Decreased urinary excretion of uric acid.** The majority of gout patients experience a deficit in urinary elimination of uric acid. This may result from decreased glomerular filtration, decreased tubular excretion, or enhanced tubular reabsorption.
10. **Medications such as diuretics, cyclosporine and niacin** have been associated with triggering acute gouty attacks.

EPIDEMIOLOGY

Incidence of gout among American adults in 2007-2008 was 3.9% (8.3 million), (6.1 million males and 2.2 million females). The number has increased by 1.2% over the last 20 years. The incidence increases with age. About 12% of men 70-79 years of age have gout compared to about 3% of men younger than 50. The incidences in men is 9%, and 6% in women over age 80. Presence of metabolic syndrome increases the occurrence of gout. About 58% of patients with gout have hypertension, 45% have lipid disorders, 33% have both hypertension and lipid disorders, and 20% have diabetes. The incidence among African Americans is twice that among whites. The fatality rate from gout as the direct cause of death is nil. About 2.3 million patients made ambulatory care visits to hospitals every year from 2001-2005. Between 2005 and 2011 the medical expenditure, including ambulatory care and medications attributed to gout, was $1.7 billion.

CLINICAL PRESENTATIONS

One of the most prominent clinical signs of gout is hyperuricemia. However, that does not indicate that gout is inevitable. This sign may be asymptomatic and could remain silent for years without triggering a gouty attack. When, and if, symptoms flare up, they begin acutely and abruptly, and often follow a trigger such as the intake of diuretics, excessive amounts of alcoholic or foods rich in purine. Some mild pain in the joint may precede the attack, which is characterized by excruciating, throbbing pain usually at the base of the big toe, but it can occur in other lower body joints. The skin of the affected joint appears red to purplish in color, swollen, tender and warm with a sensation that the toe is on fire. Stiffness in the affected joint may occur.

The first attack usually strikes without warning and often occurs at night. The pain may last from 7 to 10 days, after which it ceases or becomes mild but constant. Subsequent attacks may occur intermittently. However, the patient may not experience additional episodes for periods ranging from months to years. Some patients may experience only one attack during their life time, while others may have repeated attacks. If left untreated, gout attacks may become chronic and more frequent and intense, resulting in damage to the bones, deformity of the joint and morbidity. The deformity becomes more severe as tophi grow in size. Systemic symptoms such as mild fever, headache, weight loss and bradycardia may occur. Once the gouty attack subsides, pruritus as well as peeling of the skin covering the joint may follow.

DIAGNOSIS

To differentiate gout from other types of arthritis, one or more of the following tests may be performed.

1. Joint fluid test: MSU crystals may be revealed by drawing synovial fluid from the affected joint and microscopically testing for the presence of urate crystals.
2. Blood test to determine the level of uric acid. However, as indicated earlier, certain individuals may have high uric acid blood level but never experience gout.
3. X-ray imaging is beneficial in diagnosing chronic gout.
4. Dual energy CT scan can be helpful in detecting the presence of urate crystals. This test is considered more expensive than the other three.

TREATMENT

Gout is treatable by a combination of pharmacological and non-pharmacological measures. Pharmacological measures consist of employing medications that:

1. Control gouty attacks
2. Block uric acid production in the body
3. Lower serum uric acid
4. Enhance excretion of uric acid by the kidneys

The non-pharmacological measures involve lifestyle changes.

PHARMACOLOGICAL MEASURES

Nonsteroidal anti-inflammatory drugs (NSAIDs)

These medications include ibuprofen, naproxen and indomethacin. These medications act as analgesics, antipyretics and anti-inflammatories. Ibuprofen and naproxen are more commonly used than indomethacin due to the adverse effects of the latter.

Ibuprofen exerts its therapeutic effects by inhibiting cyclooxygenase (COX), an enzyme officially known as prostaglandin-endoperoxide synthase. It plays a role in the formation of prostaglandins and thromboxane A2 and B2, two enzymes that play a role in clot formation by facilitating platelet aggregation. Prostaglandins are lipids found in most body tissues and organs. They control functions such as inflammation, blood flow and formation of blood clots at the site of injury to blood vessels. In case of infection or damage to body tissue,

prostaglandins are synthesized at the site where they cause pain, inflammation, redness, and swelling. Inhibition of the COX enzymes by NSAIDs results in blocking the formation of prostaglandins, resulting in reduction of pain, redness, swelling and inflammation. Adverse effects of ibuprofen include an increase in the risk of fatal heart attacks or strokes especially when used in large doses and over a prolonged period of time, stomach or intestinal bleeding, nausea, drowsiness and dizziness. The usual dose of ibuprofen for gouty flare ups is 400-800 mg three to four times daily until the attack has subsided. At doses of 1200 mg daily, ibuprofen has low incidence of GI disturbances.

Naproxen is a NSAID whose mechanism of action and uses are similar to those of ibuprofen. However, ibuprofen has a faster onset than naproxen but a shorter duration of action. Naproxen provides better relief for strained muscles, sprain and arthritis. Furthermore, naproxen has less harmful effects on the heart than ibuprofen. People with a history of heart disease or heart attack are recommended to take naproxen instead of ibuprofen as it has less risk of causing cardiac complications. Plasma half-life of ibuprofen ranges from 2-4 hours, whereas that of naproxen is from 10-20 hours. The usual dose of naproxen for treating gout is 750 mg one time, followed by 250 mg 2-3 times daily until gouty flare-up ceases.

Indomethacin has a similar mechanism of action as other NSAIDs, but it is more potent. It is used mostly for managing moderate to severe attacks of gouty arthritis. Its limitations relate to adverse effects. It should not be used for minor aches and pains or fever. It causes gastric and CNS adverse effects in 30 to 60% of patients. GI problems include gastric ulceration and bleeding, nausea, vomiting, flatulence, epigastric pain, gingival ulcers and diarrhea. CNS adverse effects include headache which may be accompanied by ataxia, tremor, dizziness and insomnia. Reduction of the dose may diminish the intensity of the side effects, but it makes the drug less effective. To reduce GI side effects, the drug should be taken after meals or with antacids. Like ibuprofen and naproxen, it increases the risk of cardiac complications.

Colchicine

Colchicine is a botanical alkaloid obtained from dried corns and seeds of *Colchicine autumnale*, and is used primarily after an attack begins. It is occasionally used in very small doses between attacks to prevent recurrence of flare-ups, but is not very effective for prevention. If taken when the first sign of an attack appears, colchicine is capable of aborting the progression of the attack. The precise mechanism of action of colchicine is not fully understood. It has been speculated that its action is due to reduction of lactic acid production by white blood cells which leads to diminished deposition of uric acid in the joints. Furthermore, it acts as an anti-inflammatory agent by reducing phagocytosis. Colchicine has no analgesic activity per se. Thus it is not used to treat pain that is not caused by gout. Health care practitioners have been prescribing single ingredient colchicine (monotherapy) for many years, but this practice had not been approved by the FDA, as such, until 2009. The use of colchicine in combination with an ingredient that increased excretion of uric acid in the urine was approved by the FDA in 1939. The usual dose for reduction of pain and prevention of acute attacks is 2 tablets each containing 0.6 mg of colchicine. This dose is followed by one tablet every hour until pain is relieved or diarrhea takes place. After the initial dose, 0.6 mg tablets are taken every 2-3 hours. The total amount of colchicine needed to manage pain usually ranges from 4-8 mgs. The intake of the drug must be stopped in case of GI pain and diarrhea. Side effects include bone marrow depression, aplastic anemia, thrombocytopenia and agranulocytosis following long-term use, vomiting, diarrhea, headache, dizziness, and anorexia. It is contraindicated in renal

failure. Patients should limit alcohol consumption since alcohol tends to reduce effectiveness.

Medications that block uric acid production in the body

Allopurinol is a xanthine oxidase inhibitor (XOI), an enzyme that leads to the production of uric acid in the body. Blocking this enzyme will result in reducing the level of uric acid in the blood. Thus, the drug is used in the prevention of attacks rather than to treat them when they occur. Allopurinol is also used for treating kidney stones. It is recommended that the drug be taken with a full glass of water. Patients are instructed to also drink plenty of water during the day to minimize the risk of kidney stone formation. FDA pregnancy category is C. Full benefit of allopurinol may take a few months. Side effects include upset stomach, diarrhea, hypersensitivity, drowsiness, diarrhea, painful urination and loss of appetite. The usual dose is typically a starting amount of 200 mg once daily, and then a maintenance dose of 200 to 300 mg daily for mild gout or 400 to 600 mg for moderate to severe tophaceous gout in divided doses.

Febuxostat reduces uric acid production in the blood by inhibiting the enzyme xanthine oxidase. It was approved by the FDA in 2009 for treatment of hyperuricemia and chronic gout. It is more effective than allopurinol in regular doses, but not more efficacious in higher doses. It is ineffective in stopping flare-ups already in progress. It is recommended for patients who cannot tolerate allopurinol. Adverse effects include nausea, diarrhea, headache, increased hepatic serum enzyme levels, skin rash and shortness of breath. Febuxostat is taken orally in a daily adult dose of 40 or 80 mg with or without food. It should be used with caution in patients younger than 18 years of age.

Drugs that lower serum uric acid

Pegloticase, which was approved by the FDA in 2010, is a recombinant uricase, a uric acid specific enzyme that catalyzes oxidation and conversion of uric acid to water soluble allantoin, thereby reducing serum uric acid level. Allantoin is readily eliminated in the urine. It can be used in treating tophi. The drug is administered by IV infusion under the supervision of a health care provider. It is used mostly for treating moderate to severe chronic gout. The usual adult dose is 8 mg every 2 weeks. The drug is not recommended for use in asymptomatic hyperuricemia. Adverse effects include anaphylaxis, nausea, vomiting, constipation, stuffy nose and easy bruising.

Drugs that enhance excretion of uric acid by the kidneys

Probenecid is a uricosuric agent that increases the excretion of uric acid in the urine, thereby reducing uric acid level in blood plasma. This action occurs due to inhibition of tubular reabsorption by probenecid. By reducing uric acid blood concentration, the drug is capable of preventing gouty attacks from occurring, but not to treat an attack once it occurs. The initial adult dose is 250 mg twice daily for a week, and then 500 mg twice daily. Dosage between attacks should be adjusted by reducing the intake of the medication by 500 mg every 6 months until uric acid blood level has normalized. Probenecid intake should not be initiated if an attack is in progress. The drug may cause upset stomach. It is recommended that probenecid be taken with antacid or food. Adverse effects include headache, upset stomach, vomiting, dizziness, skin rash, anorexia, renal colic and increased urinary frequency.

Sulfinpyrazone is a uricosuric agent that lowers uric acid concentration in the blood by competitively preventing reabsorption of uric acid in the proximal tubule of the kidneys. Like other uricosuric agents, it prevents gouty attacks, but does not abort attacks when they are in progress. To reduce upset stomach, which it may cause, it should be taken with food or an antacid along with drinking plenty of water every day. Water or other liquids help prevent formation of kidney stones. Side effects of sulfinpyrazone include allergic reactions, nausea, vomiting, seizures, stomach pain, lower back and/or side pain and painful urination. The starting adult dose is 100 to 200 mg daily. The dose is increased by 100 or 200 mg every few days, up to 800 mg daily. After a period of time, and depending on the amount of blood uric acid, the dose may be adjusted.

Lesinurad was approved by the FDA in December 2015 for use together with XOIs such as allopurinol and febuxostat to treat hyperuricemia. Not every patient with hyperuricemia develops gout, but if uric acid crystals (MSU) are formed in the body, the risk of gout development increases. The mechanism of action of lesinurad is achieved by assisting the kidneys to excrete uric acid by inhibiting the formation of transporter protein involved in uric acid reabsorption in the kidneys. It is targeted for gout patients who failed to achieve target uric acid blood level with XOI alone. Lesinurad is not indicated for treating asymptomatic hyperuricemia and should not be used alone as monotherapy. The recommended dose is 200 mg once daily, which is the maximum dose, along with a XOI. Renal adverse effects may occur if lesinurad is taken without a XOI. The drug should be taken in the morning with food and plenty of water. Adverse reactions include headache, gastroesophageal reflux, renal adverse effects such as renal failure, increased blood creatinine and kidney stress.

NON-PHARMACOLOGICAL MEASURES EMPLOYED IN THE TREATMENT OF GOUT CONSIST OF GOUT FRIENDLY DIET AND HEALTHY LIFESTYLE CHANGE.

Gout-friendly diet

Diet has been linked to gout for centuries. It was stipulated that overindulgence with meat, seafood and wine can contribute to gout development. Physicians realized the benefits to be gained from a restricted diet to control gout even before understanding the cause of the disease. Gout-friendly diet in principle is similar to healthy diet i.e. moderate consumption of vegetables, fruits, whole grain, low-fat dairy products and lean white meat. Limiting and, if possible avoiding consumption of the following foods is recommended: liver, kidney, sweetbreads, lamb, pork, game meat, beef in large quantities, anchovies, sardines, herring, scallops, oysters and oatmeal. Healthy foods include green vegetables, tomatoes, fruits, especially cherries, breads, non-whole grain cereal, chocolate, nuts and peanut butter, low-fat milk and yogurt. Foods that contain small amounts of purine can be eaten in moderation and include salmon, bacon, turkey and trout. Regardless, animal protein consumption should be limited to 4-6 ounces daily.

Water consumption

Increasing daily water intake can enhance flushing of uric acid and in reducing the risk of kidney stones. It is recommended that a patient should drink about 16 fluid ounces of fluid a day.

Alcoholic beverages

Alcohol can interfere with excretion of uric acid. Additionally, alcoholic beverages contain high levels of purine which eventually is converted to uric acid. As a result, avoidance of

alcohol, if possible, should be attempted, especially during an attack.

Weight

Obesity or being overweight increases the risk of development of gout. Restricting caloric intake and losing weight can reduce uric acid level as purine intake becomes less. Losing weight also reduces stress on joints.

SUMMARY

Gout is a common metabolic type of inflammatory arthritis that may develop in some individuals with elevated uric acid blood level. The disease may be asymptomatic, but usually is characterized by excruciating pain in a joint, particularly that of the big toe. The inflammation is triggered by the presence of needle-like crystals of MSU in the joint. The disease affects all ages, but is mostly encountered in persons 50 years of age and older. It is more common among males than females. In 2007-2008, the incidence of gout in the U.S. was 8.3 million. The disease is treatable by a combination of medications and lifestyle changes. Medications used include those that control gouty attacks, block uric acid production, lower serum uric acid, or enhance excretion of uric acid by the kidneys.

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LESSON EVALUATION

Please fill out this section as a means of evaluating this lesson. The information will aid us in improving future efforts. Either circle the appropriate evaluation answer, or rate the item from 1 to 7 (1 is the lowest rating; 7 is the highest).

1. Does the program meet the learning objectives?
   - Recognize the symptoms of gout YES NO
   - Explain how certain foods contribute to gout YES NO
   - List steps that lead to metabolism of purine & formation of uric acid YES NO
   - List risk factors that contribute to gout YES NO
   - Discuss medications used in treating gout YES NO
2. Was the program independent & non-commercial? YES NO

	Low Relevance						Very Relevant
3. Relevance of topic	1	2	3	4	5	6	7

4. What did you like most about this lesson?_____
5. What did you like least about this lesson?_____

Please Mark the Correct Answer(s)

1. **Which statement is true?**
   A. Gout only effects the big toes
   B. Gout is characterized by presence of MSU crystals in a joint
   C. Uric acid in the blood occurs as a result of breakdown of protein
   D. Uric acid is eliminated by the liver
2. **Which statement is false?**
   A. Foods are the only source of purines in the blood
   B. Lumps of MSU crystals are called "tophi"
   C. Cytokines are responsible for initiation of synovitis
   D. Xanthine oxidase converts hypoxanthine to xanthine, which converts to uric acid
3. **Which of these in not a risk factor for development of gout?**
   A. Family history B. Intake of fat rich food
   C. Age D. Diuretics
4. **Which is true about naproxen?**
   A. More harmful effects than ibuprofen
   B. Longer effect than ibuprofen
   C. Half-life of 2 – 4 hours
   D. Usual dose for gout is 100 mg tid
5. **The mechanism of action of ibuprofen is:**
   A. Constriction of blood vessels
   B. Stimulation of epinephrine
   C. Suppression of chemical mediators released from mast cells
   D. Inhibition of cyclooxygenase
6. **Which statement is true?**
   A. Diagnosis of gout is confirmed only via blood test
   B. Incidence of gout higher in women than men
   C. About 58% of gout patients are hypertensive
   D. Hyperuricemia is always accompanied by gouty attacks
7. **Which statement is false about colchicine?**
   A. Prevents acute gouty attacks
   B. Its MOA may be reduction of lactic acid production by white blood cells
   C. Used to treat pain not caused by gout
   D. Long term use may cause bone marrow depression
8. **Which drug is an XOI?**
   A. Pegloticase B. Probenecid
   C. Sulfinpyrazone D. Allopurinol
9. **Which statement is false about lesinurad?**
   A. FDA approved in 2015
   B. MOA is by assisting kidneys to excrete uric acid
   C. Indicated for treating asymptomatic gout
   D. Renal adverse effects may occur if used as monotherapy
10. **Which statement is false?**
    A. Uricase function is to breakdown tophi
    B. Uric acid in the blood is converted to allantoin
    C. Only humans & some primates develop gout
    D. Gout is treatable

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