

بِسْمِ اللّٰهِ الرَّحْمٰنِ الرَّحِیْمِ
(وَفَوْقَ كُلِّ ذِي عِلْمٍ عَلِيمٌ)



جراح

Pharmacology | FINAL 6

Drugs for Gout



Written by : DST

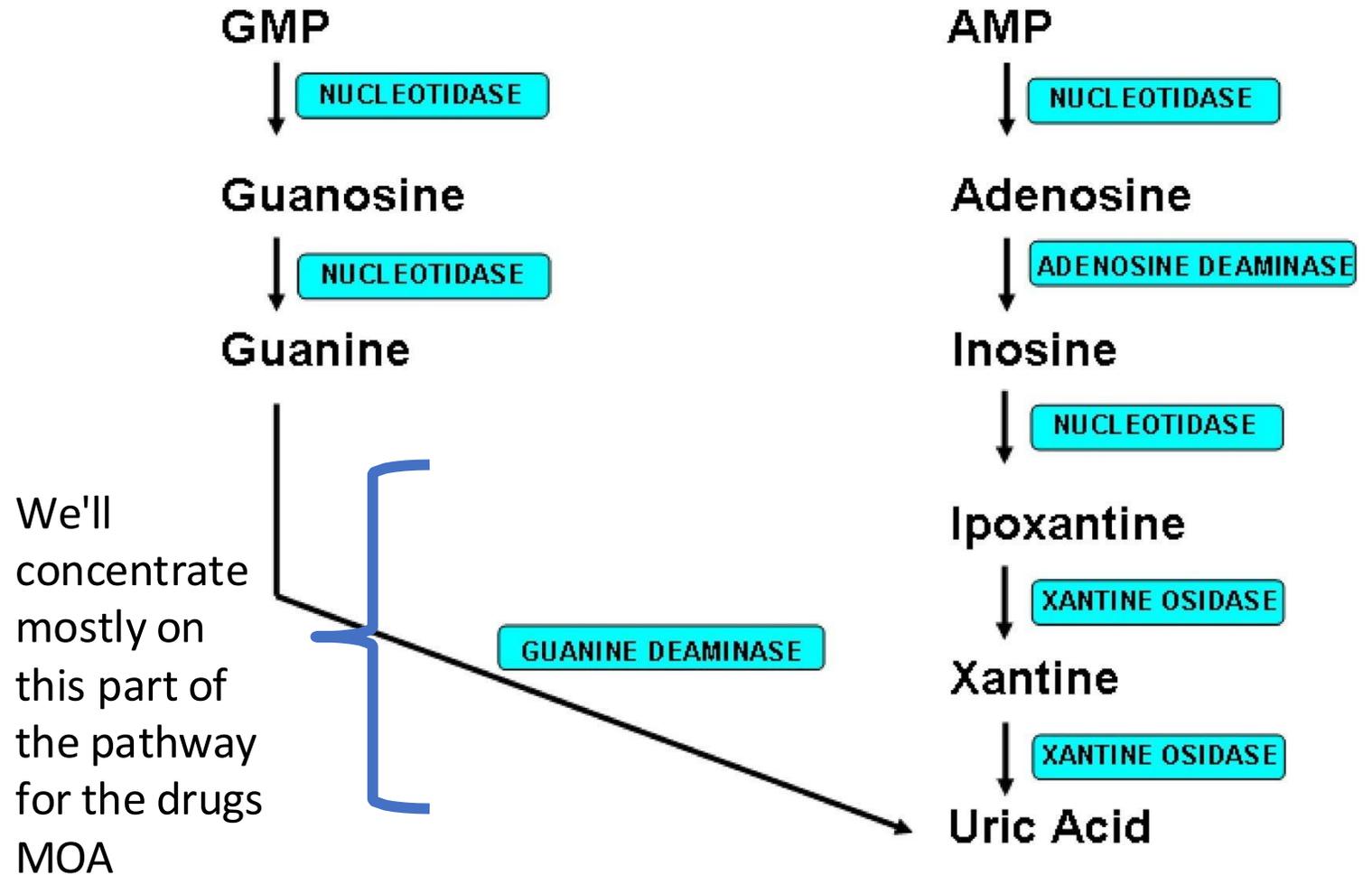
Reviewed by : Mousa Al-Neimat

Quiz on the previous lecture!



REMEMBER FROM GENERAL PHARMACOLOGY

- Get back and revise purine metabolism .
- You can watch the following video :
<https://youtu.be/iA0eROhF3pE?si=UnMDd5e68cX47VzP>



Drug Therapy of Gout

Drug therapy of gout

What Is Gout?

- Gout is a form of complex arthritis, characterized by **sudden, severe attacks of pain, swelling** in the joints, **redness**, and tenderness.
- It can affect **one or more joints**, with the **big toe** being **one of the most** commonly inflamed joints during a gout attack.
- Attacks can **occur suddenly**, often in the middle of the night when someone is sleeping, causing them to feel as if their **big toe is on fire**.
- The affected joint will become **hot, swollen, and painful**. These symptoms are typically **the result of an inflammatory reaction at that site**.

Drug therapy of gout

What Is Gout?

In Summary

Gout is an **inflammatory disease**. But what causes it? Gout can also be considered a **metabolic disorder**. It occurs due to inflammation, or what we refer to as arthritis, which is caused by the **deposition of monosodium urate crystals**. These crystals can deposit in the joints, as mentioned, but they **can also accumulate in other areas**, such as the **cartilage**. Additionally, uric acid deposits **can form in the kidneys**, leading to **renal calculi**. This can also result in **intestinal nephritis** (Although the doctor mentioned “intestinal nephritis” in the lecture, it is probable that “interstitial nephritis” was the intended term).

Gouty arthritis-characteristics

- sudden onset
- middle aged males
- severe pain
- distal joints
- Intense inflammation
- recurrent episodes
- influenced by diet
- bony erosions on Xray

As mentioned :

- gout can start in the middle of the night (**Suddenly**).
- It **usually affects males**, particularly **middle-aged men**.
- It is characterized by **severe pain** in the **distal joints**, such as the **toes**.
- The condition involves **intense inflammation**, and episodes can **recur at any time**.
- It is **influenced by diet** (since it is considered metabolic disease), and **changes in the bone can be identified through X-rays**.

Crystal-induced inflammation

hyperuricemia



crystal deposition



protein binding



receptor binding



inflammation



crystals engulfed



influx of PMN's



cytokine release

PMN is critical component of crystal-induced inflammation

Explanation of the previous slide Pt.1 :

Show does gout start?

what is the cascade of events that lead to this inflammation?

- As mentioned previously, we usually have **high levels of serum uric acid**, which we characterize as **hyperuricemia**.
- Uric acid is a **poorly soluble substance** and the major end product of purine metabolism.
- Many mammals possess an enzyme called **uricase**, which typically converts **uric acid into the more soluble substance allantoin**. **Unfortunately, humans do not have this enzyme.**
- Therefore, we must control the levels of **uric acid through its excretion by the kidneys**. In any situation where there is an **imbalance** between the uptake and elimination (or excretion) of uric acid, we may have **elevated levels** of uric acid in the serum, leading to **deposition** of the substance in joints, kidneys, intestines, and sometimes even in cartilage.

Explanation of the previous slide Pt.2

❑ **Show does gout start?**

what is the cascade of events that lead to this inflammation?

- These **urate crystals** will bind to certain proteins and receptors on the surface of the cells lining the joints, which are called **synoviocytes**.
- **Synoviocytes** will **engulf** these **urate crystals**, leading to the release of many **cytokines**, **prostaglandins**, **lysosomal enzymes**, and **interleukin-1**.
- This cytokine release causes the attraction of various cells, including **polymorphonuclear leukocytes**.
- These cells will **migrate into the joint space** and **amplify** the ongoing **inflammatory process**.
- **Polymorphonuclear leukocytes** are a critical component of crystal-induced inflammation.

Gouty arthritis-characteristics

- sudden onset One of the important characteristics of acute gout arthritis is that it can occur at any time.
 - middle aged males.
 - severe pain This pain is due to the release of cytokines, especially interleukin-1 and prostaglandins.
 - distal joints It typically occurs in the distal joints, although it can affect other areas as well.
 - intense inflammation
- recurrent episodes
 - influenced by diet
 - bony erosions on Xray
 - hyperuricemia

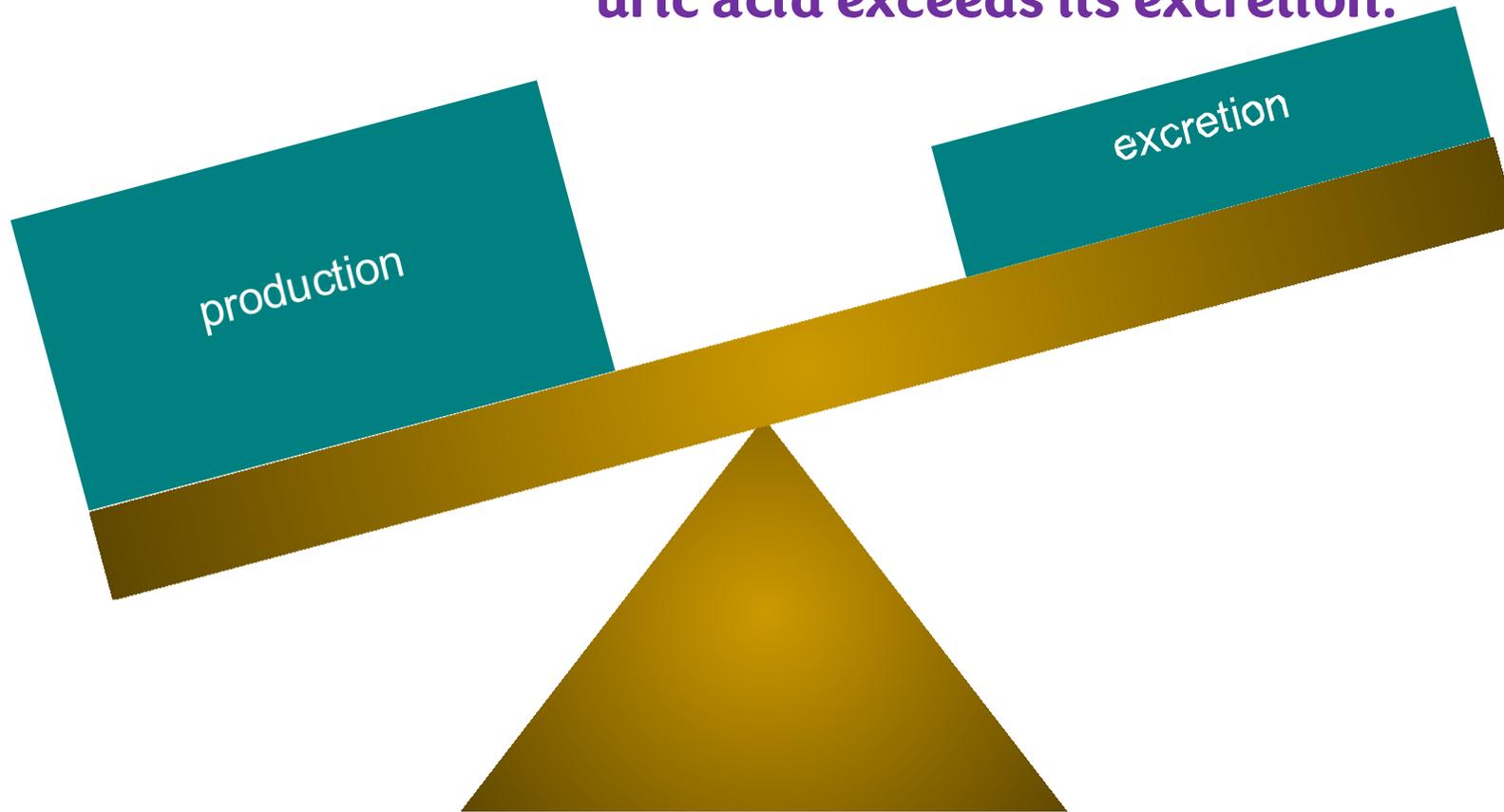
How can we diagnose acute gout arthritis? We can observe bony erosions on an X-ray, but it is also characterized by hyperuricemia.

The condition is associated with intense inflammation, which is further propagated by the presence or chemotaxis of various cells, mainly polymorphonuclear leukocytes. In the later stages of the attack, there is an increased number of mononuclear phagocytes or macrophages, which begin ingesting the urate crystals and releasing more inflammatory mediators.

Hyperuricemia

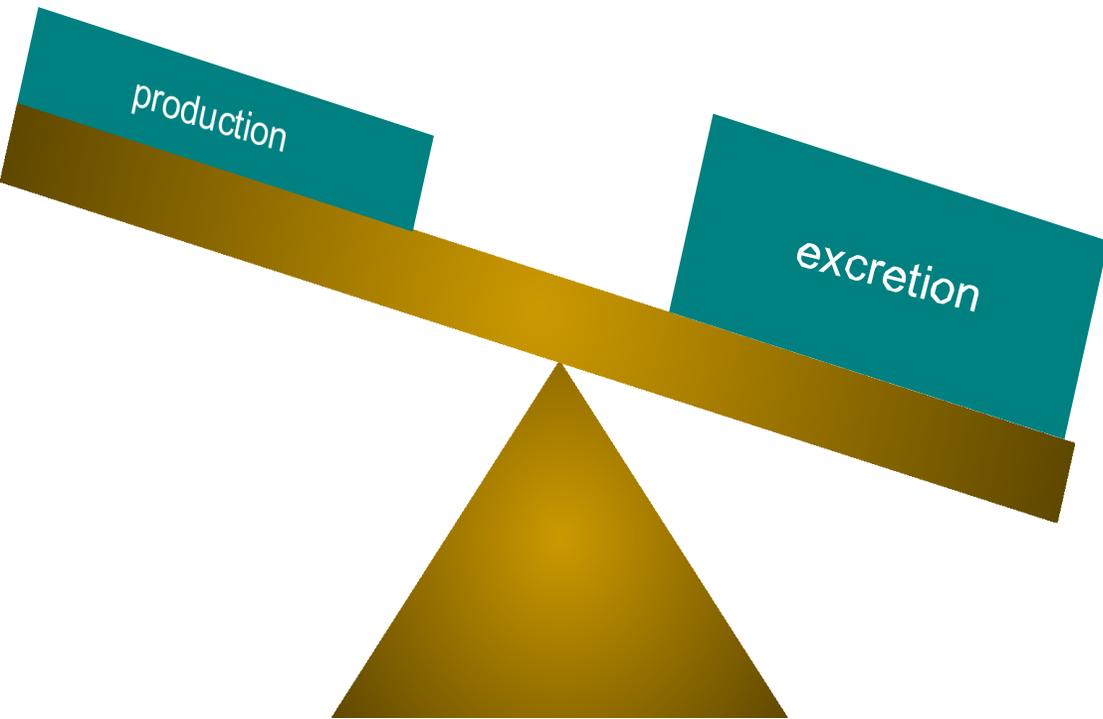
One important note is that it is **influenced by diet**, and this is very significant. How so?

There are two reasons why hyperuricemia occurs : The first is **when the production of uric acid exceeds its excretion.**



hyperuricemia results when **production exceeds excretion**

Hyperuricemia



The other reason is when the **excretion exceeds production**. This what the doctor said in the lecture but I think she means that hyperuricemia can also result when **the uric acid loss is impaired (abnormal excretion)**.

So, we usually have a balance. If we intake too many substances that are converted into uric acid in our body, what kind of substances are we talking about? Typically, red meats are digested in our body and produce purines. **These purines are then metabolized into uric acid.** Therefore, people with gout who **consume too much red meat or protein often end up with hyperuricemia.**

The other factor we're discussing here is when there's an **imbalance in the excretion process**. If the kidneys do not **excrete enough uric acid**, this leads to a **buildup of uric acid** in the bloodstream, again resulting in **hyperuricemia.**

net uric acid loss results when **excretion exceeds production**

Chronic tophaceous gout

gout doesn't only present as arthritis.

It can also present in other forms, such as **chronic tophaceous gout**. Tophaceous refers to **localized deposits of monosodium urate crystals**. Tophaceous gout is a **chronic form of gout** where we see **nodular masses, or nodes, of uric acid crystals deposited in various tissues and areas of the body**.

These tophi are present as **hard nodules, most commonly found around the fingers, as shown in this picture**.

This condition is not only a **painful inflammatory process** but also **disfiguring for the patient**. As we mentioned, tophi can be present in the **toes, fingers, and elbows**.

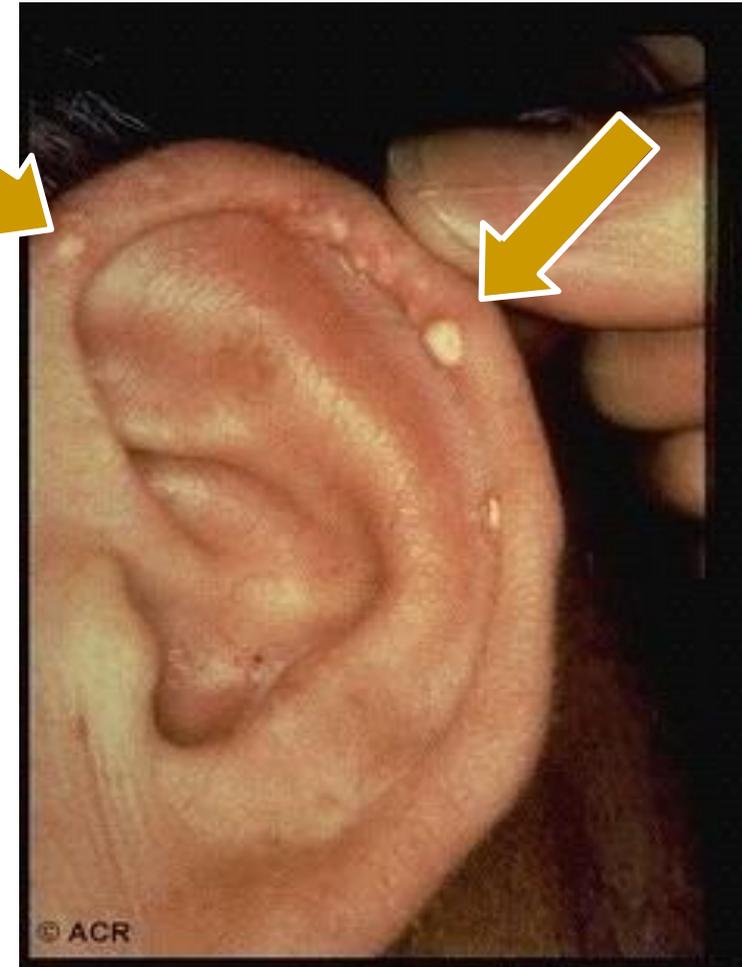


tophus = localized deposit of monosodium urate crystals

Gout - tophus

Here, we can see a classic location of the tophi on the helix of the ear.

classic location of tophi on **helix of ear.**



Gout - X-ray changes

DIP (*Distal interphalangeal joint*)
joint destruction
phalangeal bone cysts

Additionally, we talked about the changes that can be detected on an X-ray.

Notably, in the distal interphalangeal joint, we can observe joint destruction, and we can see the formation of **cysts** in the bone.



Gout - X-ray changes

Bony erosions

Here, we can see **bony erosions** in the joints.

What we have mentioned in this and the previous slides are some of the clinical manifestations of gout in the body. Let's not forget that it can also occur in other areas. As we mentioned, uric acid renal calculi can form, meaning stones in the kidneys.



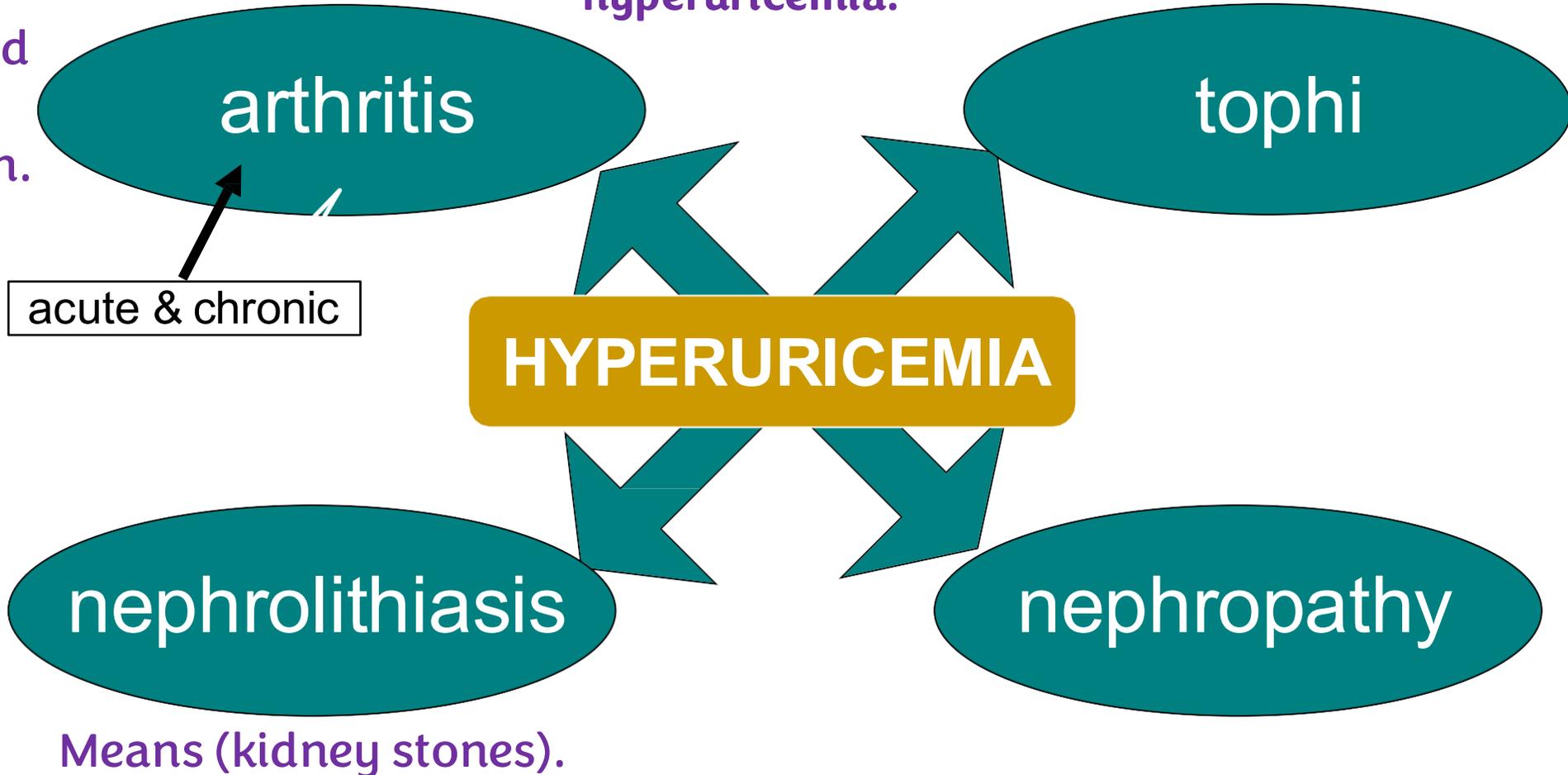
One important note to mention :

- ❖ clinical gouty episodes are usually associated with hyperuricemia.
- ❖ However, most individuals with hyperuricemia **may never develop clinical symptoms from urate crystal deposition.**
- ❖ Before starting chronic urate lowering therapy for gout patients where hyperuricemia is associated with gout and urate stone formation, **it is essential to distinguish** between individuals who have **asymptomatic hyperuricemia** and those who **experience gouty episodes due to it.**
- ❖ However, **Long-term drug treatment for asymptomatic hyperuricemia has not been proven to be effective**, meaning some individuals with high uric acid levels may go through life without **developing any adverse consequences.**

Gout - cardinal manifestations

All of these conditions are associated with elevated uric acid levels in the bloodstream, known as hyperuricemia.

Characterized by joint inflammation.



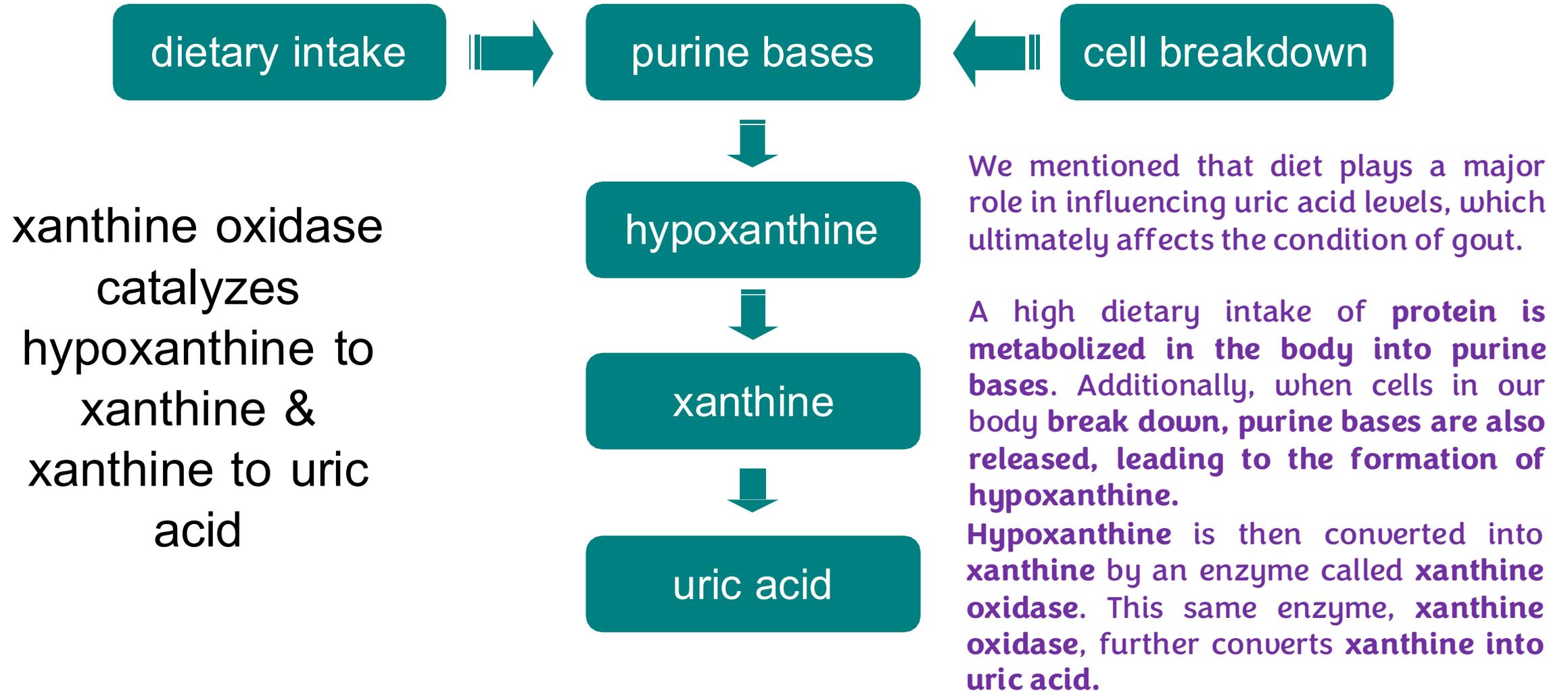
acute & chronic

Means (kidney stones).

Drug therapy of gout

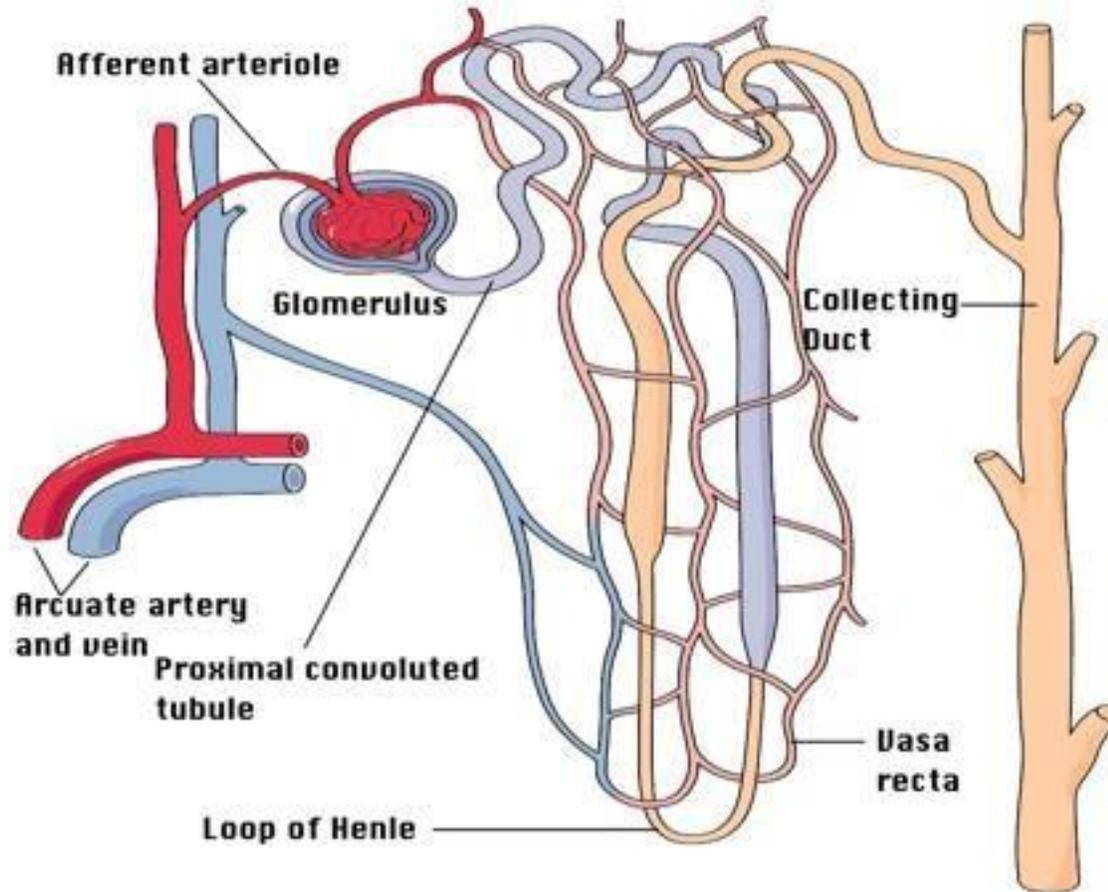
*The Role of Uric
Acid in Gout*

Uric acid metabolism



Renal handling of uric acid

- glomerular filtration ↓
- tubular reabsorption ↑
- tubular excretion ↓
- post-secretory reabsorption ↑
- net excretion



Explanation of the previous slide:

In the kidneys, how is uric acid handled?

- One key point to remember is that **the net effect of all the processes occurring in the renal glomeruli is the excretion of uric acid.**
 - 1) The process begins with **glomerular filtration**, which **decreases the concentration of uric acid in the plasma.**
 - 2) Later, in the proximal convoluted tubule, **uric acid undergoes reabsorption**, followed by a stage of **tubular excretion.**
 - 3) Finally, there is some **post-secretory reabsorption**. However, **the overall net effect remains the excretion of uric acid.**
- ✓ To regulate this process, we can use **certain pharmacological agents to inhibit tubular reabsorption or post-secretory reabsorption**, thereby **promoting uric acid excretion.**

Gout - problems

- **excessive** total body levels of uric acid.
- **deposition** of monosodium urate crystals **in joints & other tissues.**
- crystal-induced **inflammation.**

Treating acute gouty arthritis

- Colchicine
 - NSAID's
 - Steroids
 - rest, analgesia, ice, time
- Even though colchicine is **not the first-line drug therapy** for acute gouty arthritis, it was the primary treatment for many years.

Drugs used to treat gout

Acute Arthritis Drugs

colchicine

steroids

NSAID's

Urate Lowering Drugs

allopurinol

probenecid

febuxostat?

In addition to drug treatments for acute gouty arthritis, **bed rest, analgesics for pain relief, and time** play a crucial role in resolving the condition.

rest + analgesia + time

Drugs used to treat gout

NSAID's

- Indomethacin (Indocin) 25 to 50 mg four times daily.
- Naproxen (Naprosyn) 500 mg two times daily.
- Ibuprofen (Motrin) 800 mg four times daily.
- Sulindac (Clinoril) 200 mg two times daily.
- Ketoprofen (Orudis) 75 mg four times daily.

✓ All of them share the same MOA : **inhibiting urate crystal phagocytosis.**

Since most of the other NSAIDs we mentioned also have this MOA, they are used for gout treatment, except for one drug: aspirin. **Aspirin is not used in the treatment of acute gouty arthritis because it can cause renal retention of uric acid when used at low doses (less than 2.6 grams per day). On the other hand, at high doses, it is uricoseuric, but these are doses higher than 3.6 grams per day.**

✓ So, what do we usually use? **Indomethacin is commonly used in the initial treatment of gout as a replacement for colchicine.**

Drugs used to treat gout

NSAID's

- Indomethacin (Indocin) 25 to 50 mg four times daily.
- Naproxen (Naprosyn) 500 mg two times daily.
- Ibuprofen (Motrin) 800 mg four times daily.
- Sulindac (Clinoril) 200 mg two times daily.
- Ketoprofen (Orudis) 75 mg four times daily.

✓ How do we administer Indomethacin? We can use 25 to 50 milligrams, up to four times a day, usually for 5 to 7 days.

Colchicine - plant alkaloid

colchicum autumnale
(autumn crocus or
meadow saffron)

- Colchicine is a plant alkaloid that comes from a plant called *Colchicum autumnale*.
- This plant is also known as autumn crocus or meadow saffron.



Colchicine

- “only effective in gouty arthritis”.
- not an analgesic.
- does not affect renal excretion of uric acid.
- does not alter plasma solubility of uric acid.
- **neither raises nor lowers serum uric acid.**
- Opposite to NSAIDs, in addition to their **anti-inflammatory** effect, are also **painkillers**, meaning they have **analgesic** properties.

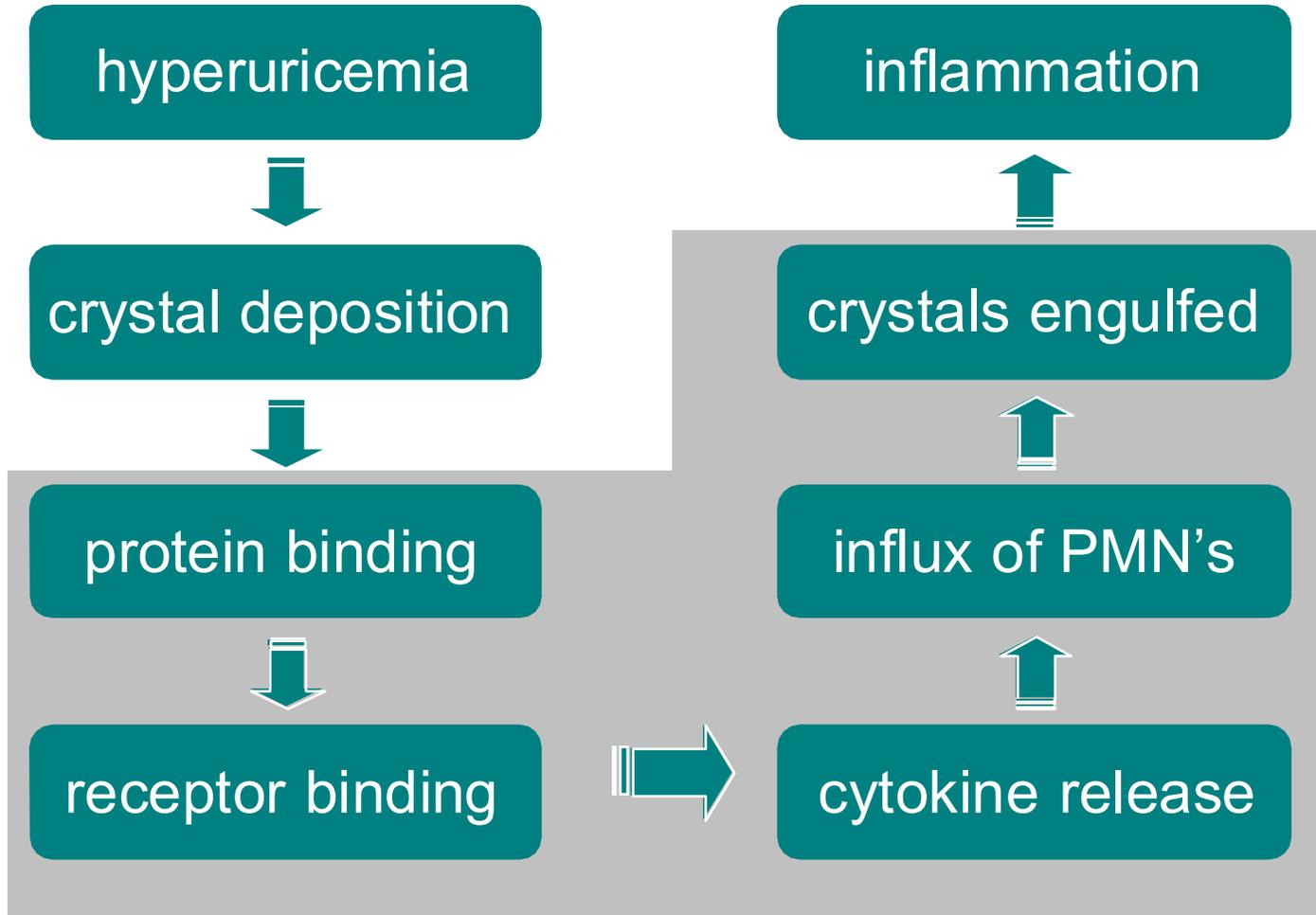
Colchicine

How Does Colchicine Help in Gouty Arthritis?

Colchicine works by preventing the polymerization of tubulin subunits, which is necessary for the formation of microtubule.

- Colchicine inhibits microtubule polymerization by binding to tubulin, one of the main constituents of microtubules.
- reduces inflammatory response to deposited crystals.
- diminishes PMN phagocytosis of crystals.
- blocks cellular response to deposited crystals.
- We previously mentioned that **phagocytosis** plays a major role in the inflammatory process associated with gouty arthritis. **For cells to engulf urate crystals, they need continuous microtubule formation and polymerization to move and perform phagocytosis.**
- By **inhibiting** microtubule polymerization, colchicine **prevents phagocytosis, reducing** the inflammatory response to the deposited urate crystals. Additionally, it **diminishes polymorphonuclear leukocyte phagocytosis of crystals, blocking the cellular response** to these deposits. This, in turn, **reduces inflammation, cytokine release, and the signs and symptoms** of inflammatory arthritis associated with gout.

Crystal-induced inflammation



- Colchicine function by preventing the synoviocytes from engulfing the urate crystals preventing cytokines release and the influx of PMNs and preventing the cascade of events that lead to the propagation of inflammation.

PMN is critical component of crystal-induced inflammation

Colchicine - indications

Dose

Indication

high → *treatment of acute gouty arthritis*

low → *prevention of recurrent gouty arthritis*

Maintenance therapy → to prevent the further flare-ups or attacks of gout

Can either be used at a high dose or a low dose.

Colchicine - toxicity

The side effects of Colchicine :

- gastrointestinal (nausea, vomiting, cramping, diarrhea, abdominal pain).
- hematologic (agranulocytosis, aplastic anemia, thrombocytopenia)
- muscular weakness

Colchicine prevents the polymerization of microtubules which are very important for the formation of the mitotic spindles meaning that in highly replicating cells Colchicine can affect the replication process of these cells and that is **why it affects the blood forming cells (platelet, RBCs, WBCs).**

adverse effects dose-related & more common when patient has renal or hepatic disease

Gout - colchicine therapy

- more useful for daily prophylaxis (low dose)
 - ✓ prevents recurrent attacks
 - ✓ colchicine 0.6 mg qd – **bid** → **Two times a day**
- Recently , there is declining use of it in acute gout (high dose) **Replaced by NSAIDs as the first line drugs for those acute conditions.**

Predisposing cause of gout

Hyperuricemia - mechanisms

Which happens either because :

excessive
production

inadequate
excretion



hyperuricemia

One way to address this problem is either by blocking the production or enhancing the excretion and the net result will be net reduction of the total body pool of uric acid.

We have certain drugs that we call **urate-lowering drugs** and they help in the prevention of arthritis tophi and kidney stones by lowering the total body pool of uric acid.

Urate-lowering drugs

block
production

enhance
excretion



net reduction in total body pool of
uric acid

Gout - urate-lowering therapy

- prevents arthritis, tophi & stones by lowering total body pool of uric acid
- **not** indicated after first attack.
- Initiation of therapy can worsen or bring on acute gouty arthritis.
- no role to play in managing acute gout.

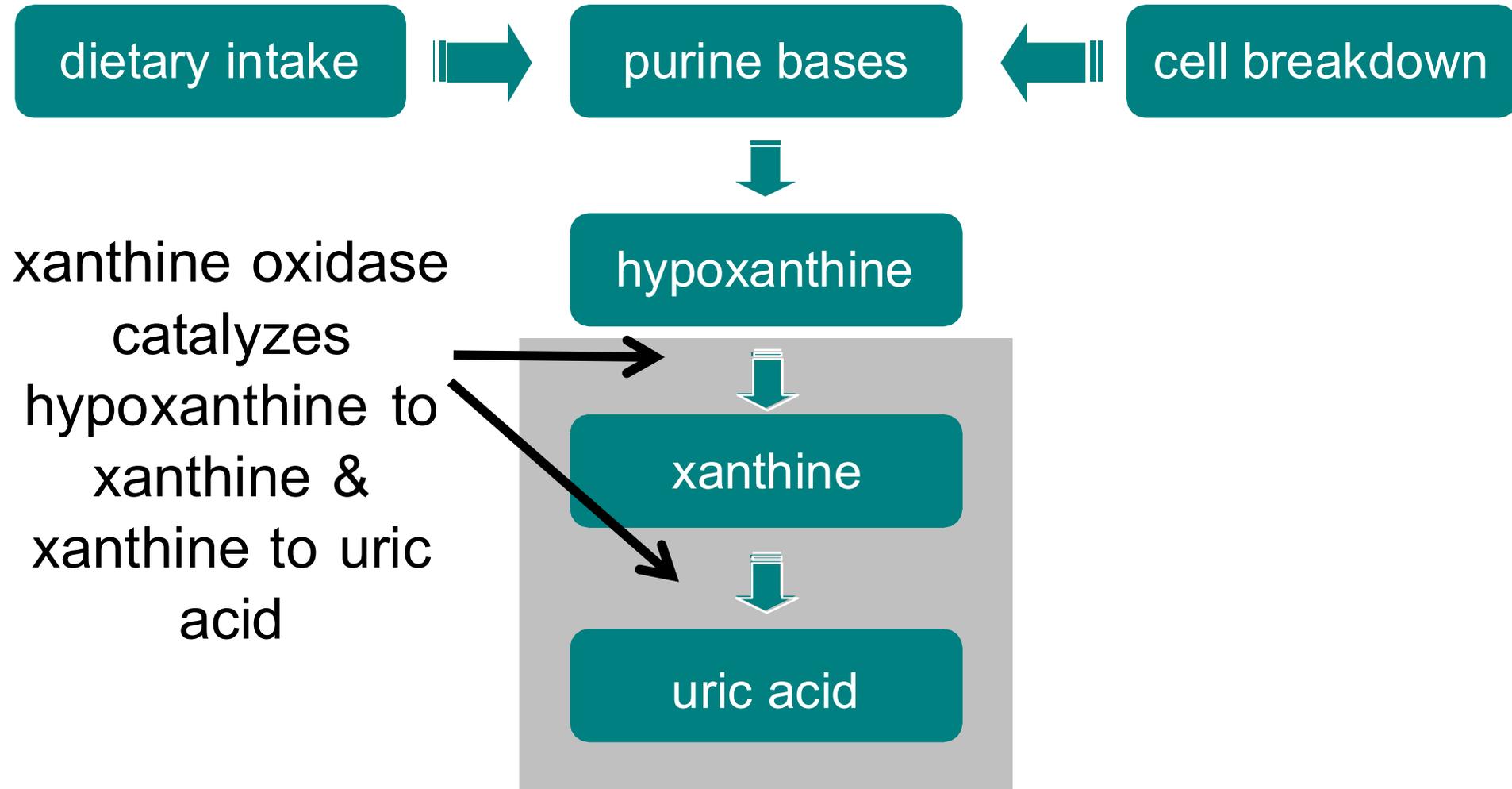
Not indicated immediately but they might worsen the attack if they were used after the first attack.

so, we must wait for some time before administering these drugs and this is because when we use these urate lowering drugs this would result in **urate crystal being shed from the cartilages** of the joints into **the joint space** which results in flare up of the acute inflammation.

Drug therapy of gout

*Drugs That Block
Production of Uric Acid*

Uric acid metabolism



Allopurinol (Zyloprim™)

- **Inhibitor** of xanthine oxidase
- effectively **blocks** formation of uric acid
- how supplied - **100 mg & 300 mg tablets (orally)**.
- **pregnancy category C** →

C

Risk cannot be ruled out: Human studies are lacking, and animal studies are either positive for fetal risk or lacking as well. However, potential benefits may justify potential risk.

We can use this drug if the **benefits outweigh the risks** → it would **interfere** with the **synthesis of uric acid** in the infants affecting the purine metabolism meaning it can cause **some risks to the fetus**.

- allopurinol



Allopurinol – usage Indications

- **management of hyperuricemia of gout**
- **management of hyperuricemia associated with chemotherapy**
- **prevention of recurrent calcium oxalate kidney stones**

Allopurinol - common reactions

- diarrhea, nausea, abnormal liver tests.
- acute attacks of gout.
- Rash.

After the initiation of allopurinol we'll have **mobilization of urate crystals** from their attached sides in the joint to the joint spaces and this results in acute changes in the levels of the uric acid serum levels which can predispose a gout attack.

Additional effects :

- Necrotizing vasculitis.
- Bone marrow suppression.
- Rarely, aplastic anemia.
- Hepatic toxicity & interstitial nephritis have been reported.
- Allergic skin reactions → pruritus or rash → into maculopapular lesions which happens in 3% of the patients.
- Some patients also develop exfoliation of the skin called exfoliative dermatitis.
- In rare cases allopurinol can become bound to the lens resulting in cataract.

Allopurinol - serious reactions

Steven Johnson Syndrome

- fever, rash, toxic epidermal necrolysis.
- hepatotoxicity, marrow suppression.
- Vasculitis.
- drug interactions (ampicillin, thiazides, mercaptopurine, azathioprine).
- Death.

Stevens-Johnson syndrome

➤ We'll have :

- target skin lesions
 - mucous membrane erosions
 - epidermal necrosis with skin detachment
- ✓ Very rare and happens in less than 2% of the patients



Some patients will develop :

Allopurinol hypersensitivity

- extremely serious problem
- prompt recognition required *Must recognize early.*
- first sign usually skin rash *Can develop to cause :*
- more common with impaired renal function.
- progression to toxic epidermal necrolysis & death.

Another drug from the same family :

Febuxostat

- recently approved by FDA In 2009 for treating gout.
- **oral xanthine oxidase inhibitor.**
- structurally chemically distinct from allopurinol. But the same mechanism of action
- 94% of patients reached urate < 6.0 mg/dl.
- minimal adverse events. Can cause reduction of urate levels below 6 mg/dl (the level we aim for).

Compared to allopurinol, it can cause diarrhea, headache and nausea.

Also, it seems more well tolerated in patients who have sensitivity or intolerance to allopurinol (great alternative).

The newest urate lowering therapy:

PEGLOTICASE

Pig → the animal

PEG → chemical compound

The addition of PEG conjugation is to increase the half life of the drug and diminish or lower the immune response for this enzyme that is not coming from a human source so it will decrease the antigenicity of the enzyme/protein.

- recently approved by FDA 2010.
- PEG-conjugate of recombinant porcine Uricase. Covalently bounded to methoxy polyethylene glycol
- treatment-resistant gout.
- uricase speeds resolution of tophi.
- It's an IV administered drug that works fast (within 24-72 hour) to reach its peak concentration in the body for days (6-13 days) Usually clearance is by antibody response _ the importance of adding PEG is to minimize antibody response in the body.

Humans do not possess the enzyme that is necessary for the breakdown of uric acid (uricase) while other mammals have it, so this drug is the recombinant form of the enzyme that is present in pigs (porcine).

PEGLOTICASE

- Pig → the animal.
- PEG → chemical compound.
- recently approved by FDA 2010.
- PEG-conjugate of **recombinant porcine uricase**.
- treatment-resistant gout.
- uricase speeds resolution of tophi.

PEGLOTICASE

- The newest urate lowering therapy..
- Used for refractory chronic gout (refractory means it doesn't respond to other medications).
- **One concern** → usage of this drug in patients with Glucose-6-phosphate dehydrogenase deficiency due to the formation of hydrogen peroxidase (Although the term “hydrogen peroxidase” was used in the lecture, it is probable that “hydrogen peroxide” was the intended term”) by the enzyme(uricase). Therefore this drug must be avoided in these patients.
- **Adverse effects** associated with it :
 - **Infusion reaction + flare-ups of gout** especially during the first 3 months of treatment.
 - ❑ **Other side effects** :
 - Nephrolithiasis (kidney stones).
 - Arthralgia.
 - Muscle pain and spasms.
 - Headache, Anemia and Nausea.
 - ❑ **less frequent** side effects :
 - Respiratory tract infection.
 - Peripheral edema.
 - UTI.
 - Diarrhea.

Drug therapy of gout

*Drugs That Enhance
Excretion of Uric Acid*

Uricosuric therapy

- probenecid
- blocks tubular reabsorption of uric acid
- enhances urine uric acid excretion
- increases urine uric acid level
- decreases serum uric acid level

Uricosuric therapy

- Mainly used in patients who have **tophaceous gout** or patients who have **frequent gouty attacks**.
- Moderately effective.
- increases risk of nephrolithiasis.
- not used in patients with renal disease.
- frequent, but mild, side effects.
- ✓ While in patients who **secrete large amounts of uric acid** or patients with **renal disease these agents will not be used**.

Uricosuric therapy

- contra-indications :
 - ✓ history of nephrolithiasis **Kidney stones.**
 - ✓ elevated urine uric acid level.
 - ✓ existing renal disease.
- less effective in elderly patients **As they have deteriorated kidney function.**
- **Mild side effects are associated with such drugs like GI irritation.**

Choosing a urate-lowering drug

excessive
production

inadequate
excretion

xanthine oxidase
inhibitor

Such as allopurinol



uricosuric agent

Such as probenecid



hyperuricemia

Drug therapy of gout

Case Presentation

Case presentation

- 55 y/o male
- 12 hours “pain in my big toe & ankle”
- went to bed last night feeling fine
- felt as if had broken toe this morning
- PMH of similar problems in right ankle & left wrist

Gout - acute arthritis

acute synovitis,
ankle & first MTP
joints

MTP = metatarsophalangeal joint

The metatarsophalangeal articulations are the joints between the metatarsal bones of the foot and the proximal bones



Gout - acute bursitis

acute olecranon
bursitis



Bursitis is inflammation of the fluid-filled sac (bursa) that lies between a tendon and skin, or between a tendon and bone

Case presentation - therapy

1) Start the patient on :

NSAID

steroid

Either are used as first line treatment

4) If there were any flare ups we can give NSAIDs again

NSAID

2) Before we end NSAIDs we start :

colchicine (low-dose)

Used as maintenance therapy to prevent further or any future attacks

Not recommended due to its side effects

3)

Can't start it immediately after an acute attack so we wait for a period of time while the patient is still on NSAIDs and Colchicine and then we can use it and stop the NSAIDs

allopurinol

Maintenance therapy maintain low levels of uric acid in the blood and after the attack is subsided we keep the patient on Colchicine and Allopurinol.

days 1-10

days 11-365

days 365+

Interleukin 1 receptor antagonist

- Used when in the patient is not responding to NSAIDs nor colchicine.

Example:

Anakinra
Canakinumab
Rilonacept

These are drugs used for treatment of rheumatoid arthritis and currently being investigated for gout . They target IL-1 pathway thus they would suppress the inflammation.

Glucocorticoids

We can use them for acute gouty arthritis

Prednisone

- Oral
- Intra-articular *inside the joints*
- Subcutaneous



Depend on the **degree of the acute attack** and the **degree of pain and inflammation** in the patient

Choose the correct answer to test yourself in the previous lecture (NSAIDs2)!



How to treat rosacea?

- A) Metronidazole.
- B) Azoleic Acid (Topical).
- C) Both (Metronidazole & Azoleic Acid) can be used.

رسالة من الفريق العلمي (الجزء ١)

الصحة النفسية

نحن نعاني من عدة مشاكل عويصة:

الأولى: اعتبار موضوع الأمراض النفسية عارًا وأشبه بالعيب، وهو أمر غير صحيح. الأمراض النفسية مثلها مثل الأمراض الجسدية، بل وبعض الأطباء يعتبرها أخطر. فقد يلاحظ الطبيب عليك تغييرًا في لون عينيك إلى الأصفر فيستدل على وجود علة في جهاز ما في جسمك، أما في حالة الأمراض النفسية، فيتطلب الأمر بناء الثقة بين الطبيب والمريض حتى يخبره المريض بمشكلته، وهو قد يكون أصعب من تشخيص الأمراض العادية.

أما الثانية والثالثة والرابعة فيمكن تلخيص الرد عليها بآية واحدة من القرآن:
"وَابْيَضَّتْ عَيْنَاهُ مِنَ الْحُزْنِ فَهُوَ كَظِيمٌ".

الثانية: المشهور والمتداول أن المؤمن لا يعاني من الاكتئاب أو التوتر أو أي صعوبات نفسية.
الصحيح: هذه الآية تذكر الحزن الشديد الذي أصاب سيدنا يعقوب... فهل يمكن لأحد أن يشكك بإيمانه؟

الثالثة: الاضطرابات النفسية شيء بسيط... لا تعطي الموضوع أكبر من حجمه وارمه وراء ظهرك وكأن شيئًا لم يكن.
الصحيح: في هذه الآية، يبين الله لنا أن الاضطراب النفسي قد يكون شديدًا لدرجة يؤثر فيها على فسيولوجية الجسم، وهذا ما حدث مع سيدنا يعقوب بفقدانه لبصره من شدة الحزن. لا أحد يمكن أن يشكك بخطورة فقدان البصر، فلماذا لا نعطي الصحة النفسية نفس الأهمية؟

رسالة من الفريق العلمي (الجزء ٢)

الصحة النفسية

الرابعة: العلاج النفسي والتحدث للآخرين غير مفيد، أو بمعنى آخر: "استرسل يا زلمة وفكك من الهبل!" الصحيح: "فهو كظيم" تعني كبت الحزن وعدم التحدث مع الآخرين، مما قد يزيد من تفاقم الحالة. وهذا يدل على أن التحدث مع الآخرين وطلب العلاج النفسي قد يخفف من وطأة الاضطراب.

الصحة النفسية مهمة جدًا، وهي موازية في الأهمية للصحة الجسدية، فلا تتردد في طلب المساعدة. وطلبك للعلاج النفسي لا يعني أنك غير مؤمن.

في الختام، وضعت في الصورة أسفل المنشور الرقم الساخن JCPA Hotlines يقدمون دعمًا مجانيًا وسريًا على مدار الساعة وطوال أيام الأسبوع، عبر الهاتف والرسائل النصية، لمن يعانون من مشاكل نفسية أو وساوس انتحارية.

الله يحفظكم ويحفظ أحبائكم



For any feedback, scan the code or click on it.



Corrections from previous versions:

| Versions | Slide # and Place of Error | Before Correction | After Correction |
|----------|----------------------------|-------------------|------------------|
| V0 → V1 | | | |
| V1 → V2 | | | |