Pharma final

RU FA

Sheets + dr.Abdel-Motaal Fouda videos

Prototype →
يعبر عن مجموعه من الادويه حيث ان باقي الادويه لي بنفس المجموعه غالبا لها نفس المواصفات

Coparison of analgesic

Feature	Narocotic	Non narocotic (NSAID)- mainly perphral
efficacy	Strong	Weak
Prototype	Morphine	**ASPRINE =**acetyl salicylic acid
Pain relieved	Any type	Musculoskeletal
Site of action	CNS	PNS
Mechanism	Opioid receptor	**PG synthesis : inhibiting** the **cyclooxygenase (COX)** enzymes that catalyse the first step in prostanoid (PG) biosynthesis
Danger –side effect	Tolerance and dependence	G.I irritation تهيج by inhibiting **PGE2**
Anti-inflamm	NOOO	**YESSS**
Antipyretic	NOO	**YESSS :** 1- asprine decrease the production on PGE2 مسؤول اساسي عن رفع الحراره in the brain 2- asprine effect on hypothalamus to not response to IL-1 مسؤول ايضا عن رفع الحراره 3- cutaneous vasodilatation → increase sweating
Antiplatelets	NO	**YESSSSSSSS**

الفرق بين الاسبرين وباقي الادويه من عائله ال NSAIDs

- **Aspirin** can cause **irreversible** inactivation of COX-1 and COX-2. while the other NSAIDs are all reversible.

- ASPIRIN **does not** touch the **heart**

Aspirin (NSAISs) effects

analgesics	antipyretic	Respiratory action	Gastro-intestinal	platelets (antithrombotic)	kidneys
- used to management of **low** and **moderate** pain intensity - Pain specially that arise from musculoskeletal disorders - Inhibition PGE2 →decrease pain sensation.	- Aspirin has no effect on normal body temperature - It's antipyretic ONLY NOT hypothyrmic By → inhibition PGE2→peripheral vasodilation +sweating	-Does **not** do **METABOlIC (HCo3) ALKALOSIS** -**therapeutic doses**→ increases alveolar ventilation→ causing **uncoupling of oxidative phosphorylation** → elevated CO2 and increased respiration. -**Higher doses**→ hyper-ventilation and **respiratory alkalosis** - **toxic levels**→ cause central respiratory paralysis which leads to **acidosis** (metabolic acidosis)	**-PGE2** inhibition → increased gastric acid secretion and diminished mucus protection. - have problem with increase acid secretion, to solve the problem give **proton pump inhibitors (PPI)** these drugs inhibits the gastric and duodenal ulcers **PPI** ex: E… L… O… اسم امي لانسو ^_^	- **low doses 75-81 mg daily** of aspirin can **irreversibly** inhibit **thromboxane** (TXA2) production in platelets -lack of thromboxane **persists** for the **lifetime of the platelet (7days)** مريض ياخذ اسبرين ممنوع اجراء اي عمليه له الا بعد7 ايام واذا كانت عمليه طارئه نقوم بنقل الدم او packed platelets لو كان اي نوع اخر من الدواء غير اسبرين عادي يعمل العمليه بعد يوم او كم ساعه **other NSAIDs are all reversible.**	Inhibition the **PGE2** and **PGI2** (prostacyclin) → decrease in renal blood flow→**kidney injury** Inhibition the PGE2 and PGI2 →retention of sodium and water→and may cause **edema** and **hyper-kalaemia** **Interstitial nephritis** can also occur with **all** NSAID 

Therapeutic uses of aspirin

Analgesic → **low** and **moderate** pain only
Ex: analgesia include **headache**, . ألم العضالت**myalgia** and , ألم المفاصل**arthralgia**
*any kind of pain related to musculoskeletal system

antipyretic

Anti-inflammatory

Antithrombotic → **LOW** dose (75-81mg) / يعطى بشروط معينه فقط وهي
:
1- used **prophylactically** to reduce the risk of recurring **transient ischemic attacks** (**TIAs**) and **stroke** or death
2- **elderly** patient who have **diabetes +history of CV diseases or hypertension**
3- **acute myocardial infarction or angina**.

Keratolytic NOT THE FUNCTION OF ASPIRNE

****Salicylic acid** is used topically to **treat** **corns** اللحم مسمار and **warts** الثآليل او السنطbecause it has keratolytic activity.

مش من استخدامات Keratolytic الاسبرررين

Because asprine is **acetyl** salicylic acid

Pharmacokinetics of aspirin

Administration and distribution

oral administration → un-ionized salicylates are passively absorbed from the stomach and the small intestine

Rectal absorption → given as suppository تحاميل → which is slow and unreliable, but it is a useful route for administration to vomiting children. BUT Salicylates must be avoided in children and teenagers (<15 years old or <12 sometimes) with varicella (chickenpox) or influenza → cause REYE'S Syndrome → FATAL

المحبب اكثر والمستخدم اكثر عند الاطفال هو :

in children we use paracetamol

الجدول لي بصفحه 4 اعتبر اي شيئ ضار ومش منيح هو adverse effect ورح ينحفظ معك هاد الجدول بسهوله

• Adverse effects

• **Gastrointestinal** → 1- epigastric distress 2- nausea 3- vomiting 4- microscopic GI bleeding

• Why → because asprine IONIZED and TRAPPED in mucosal cells

• **Blood** → inhibition of platelet aggregation and a prolonged bleeding time (1 week).

• **Respiration** → in **toxic** dose **central respiratory paralysis** which leads to acidosis (metabolic acidosis)
• Other word → salicylates cause respiratory depression and a combination of
uncompensated respiratory and **metabolic** acidosis

• **Metabolic processes** → in **toxic** dose → **uncouple oxidative phosphorylation→ hyperthermia** الحاله التي يقوم بها الاسبرين برفع درجه حراره المصاب
• **Hypersensitivity**→ Symptoms :

- Urticaria
- Bronchoconstriction
- Angioedema
- Fatal anaphylactic shock is rare

• **Reye's syndrome** →**children** and **teenagers** (<15 years old or <12 sometimes) with **varicella** (chickenpox) or **influenza** → cause **REYE'S Syndrome** → **FATAL**

Drug interactions

Aspirin can displace **anticoagulants** (**warfarin**), and **antiepileptic** (**Phenytoin** and **Valproic**) from plasma proteins leading to **increase their plasma concentrations**.
Contraindication : Ketorolac + **asprine** → increased risk of GI bleeding and platelet aggregation inhibition
In pregnancy → Aspirin is classifieds pregnancy category **C risk** during Trimesters 1 and 2 يعني من الشهر **ال3 – للشهر ال6** من فتره الحمل **category D** during Trimester 3 **الشهر التاسع (9)** in women who has experienced **miscarriage** because of the **rejection** of the **fetus** because of the **formation** of **antibodies** against him → **use aspirin** in **low doses** in the **first trimester** to increase the circulation to the fetus.
aspirin should be **avoided** during **pregnancy** and while **breastfeeding**.

Toxicity

mild form called **salicylism** : symptoms →
Nausea / vomiting /**marked hyperventilation**من الاعراض الاولى سرعه تنفس/
headache /mental Confusion/dizziness /tinnitus طنين الاذن

Treatment → serious cases =**intravenous** administration of **fluid**, **dialysis**
→ correct the **acid-base**

Propionic acid derivatives
They are NSAIDs other than aspirin

Examples → Ibu**profen →** safest for children , **naproxen**, feno**profen**, keto**profen** , flurbi**profen**

Therapeutic uses → anti-inflammatory, analgesic, and antipyretic activity

Administration → well absorbed on **oral** administration and are almost totally **bound** to serum **albumin**.

Side effects → GI irritation (most common + dyspepsia and bleeding)
central nervous system → headache + tinnitus + dizziness
Respiratory → with **asthmatic** patient cause **bronchoconstriction** and **increase bronchial secretions**
****ibuprofen and naproxen: contraindication**
-category **C** and **D** risk on **pregnant** women from the **3rd trimester**
-**Increase** the risk of cardiovascular **thrombotic** event, **MI** and **stroke**.
-Increase risk of GI bleeding
-**Ibuprofen** taken **with food or with water** to avoid GI irritation

- drugs are **reversible** inhibitors of the cyclooxygenases
- their **GI effects** are generally **less intense** than those of aspirin
- undergo **hepatic** metabolism and are **excreted** by the **kidney**

Acetic acid derivatives:

Example → indomethacin→ more potent than aspirin **, sulindac, Etodolac**

Therapeutic uses → anti-inflammatory / analgesic / antipyretic activity
indomethacin → treatment of **acute gouty** arthritis and **ankylosing spondylitis**

Side effects →same but less severe than, those of the other NSAIDs نفسهم لي بصفحه 10 او 7 الاثنين نفس الشي تقريبا

- **These drugs are cyclooxygenases inhibitors**
- **Indomethacin the most toxic**

Oxicam derivatives

Example → Piroxicam , meloxicam

Therapeutic uses → treat **rheumatoid arthritis(RA)**, **ankylosing spondylitis**, and **osteoarthritis**

long half-lives → once-daily administration
metabolites are **renally excreted** in the urine

MELOXICAN INHIBITE COX2 MOREE THAN COX1 (selective for COX-2)

MELOXICAN→ at **low** to **moderate** doses shows **less GI irritation** than piroxicam and other NSAIDs

Fenamates

Example →Mefenamic acid

كل شي زيها زي باقي ال NSAIDs من ناحيه الاستخدام وmechanism of action ...الخ

side effect → diarrhea which can be severe, and they are **associated** with **inflammation of the bowel.**
***Cases of haemolytic anaemia have been associated with these agents. **

Heteroaryl acetic acids

Example → Diclofenac (Voltaren) , tolmetin , ketorolac

Therapeutic uses → RA, osteoarthritis , inflammatory conditions of the joints

Administration of Diclofenac sodium :orally 50mg after food
I.M. injection 75mg

Side effects → Diclofenac sodium category C risk in pregnancy
Diclofenac **sodium** cause hypersensitivity

Diclofenac is **more potent** than indomethacin or naproxen
excretion for the drug → **kidney**
Diclofenac **potassium** is prompt release ينتشر اسرعand has **quicker** onset whereas the **Diclofenac sodium** is delayed release

Diclofenac **sodium → Metabolism: liver / excretion: urine**

Contraindication → asthmatic patients & patient with history of peptic ulcer

Selective COX-2 inhibitor

Example → Celecoxib , Meloxicam , Rofecoxib

celecoxib is still available and is used in **chronic inflammatory** conditions such as
Rheumatoid arthritis

side effects → thromboembolic effects (platelet aggregation)

more selective for COX-2

Rofecoxibبطل يستخدم وتم سحبه من الصيدليات لانه مرتبط مع
deaths related to thromboembolic effects

Acetaminophen (Paracetamol)

الاسم التجاري Panadol

Therapeutic uses → analgesic , antipyretic , **choice for children with viral infections or chickenpox**

doesn't have anti-inflammatory property

***does not affect platelet function or increase blood clotting time**

Absorbed → GI tract

significant first-pass metabolism → luminal cells of the **intestine + hepatocytes**

Excerted → urine

Adverse effects → Renal tubular necrosis +hypoglycaemic coma are very **rare**

Large doses →1- cause Hepatic necrosis → trx by **acetylcysteine (before 8hs up to 12hs of the overdose)** → remove toxic radicals

→ 2- Renal tubular necrosis

normal therapeutic doses →free of any significant adverse effects

Acetaminophen inhibits prostaglandin synthesis in the **CNS so it** has less effect on cyclooxygenase in peripheral tissues

الباراسيتامول بديل ممتاز للاسبرين للاشخاص لي عندهم مشاكل بالجهاز الهضمي او
those in whom prolongation of bleeding time would be a disadvantage OR dont require the anti- inflammatory action

acetaminophen is safe drug but if given in high doses it shows toxicity → accumulation of the toxic metabolite in liver

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بعد ما خلصت محاضره
الفارما
– اهلين شو اخباركم.. امي صبح؟

Rheumatoid arthritis

Immunological disease +usually **associated** with **Chronic synovial inflammation**

Effects on many kinds of cells →

1- **OSTEOCLASTS** : leading to bone **RESORPTION** and **EROSION** تاكل

2-synoviocytes (TNF-α stimulate it) : **more joint inflammation** → pain and swelling of the joint

3- **chondrocytes (TNF-**α stimulate it) → **cartilage degradation**& joint space **narrowing**

Most important **cytokines** that are **responsible for inflammation & joint destruction** :
TNR-α
IL-1, -6,-17, -8
prostaglandins

Drugs:
NSAIDs
DMARDs→
- Reduce swelling and inflammation
- Relief pain
- Improve function of the joint
-reduce EROSIONS (reduce radiographic progression)
Glucocorticoids

Synthetic disease modified antirheumatic drugs (SDMARDs)

Effect may take 6 weeks to 6 months to become clinically evident

Example :

Methotrexate (MTX)

Sulphasalazine

Chloroquine , hydroxchloroquine

Leflunomide

Cyclophosphamide

Cyclosporine

Mycophenolate

	Methotrexate	**Sulphasalazine**	**Chloroquine +hydroxychloroquine**
Mechanism of acrion (MAIN)	Inhibition **AICAR transformylase** and **Thymidylate synthetase**	Metabolized to : -**Sulphapyridine** (**active** when treating RH) -5-aminosalicylic acid HELP IN **REMOVING TOXIC free radicals** from the body **Suppression t cell** Inhibition b cell proliferation Reduction in IL-1,6,12 and TNF-a	Unknown but it's effect : -suppression t.lymphocyte -Inhibition leukocyte chemotaxis -stabilization of lysosomal ENZ -Inhibition DNA and RNA synthesis -trapping free radical
Pharmaco-kinetics	eliminated → kidney Absorb → orally 70%		
Half life	6-9 hours		
Administrat-ion	oral		
Adverse eff	-hepatotoxicity -bone marrow suppression -dyspepsia , oral ulcer -Pneumonitis -teratogenicity	- igA and igM decreased - Dyspepsia - Rashes - Bone marrow suppression	Ocular toxicity **IRREVERSIBLE retinal toxicity and corneal deposits** Dyspepsia Nausea Vomiting Abdominal pain Rashes Nightmares
Monitor	CBC FBC ALT – liver function Creatinine -kidney		Ophthalmological evaluation every 6 months or 12 months
معلومات مهمه	Give the patient **FOLIC ACID** **- First line to treat RH**		Treatment **MILD** RH Takes at **least 1month** to see effect

	Leflunomide	**Cyclophosphsmids**
Mechanism of acrion (MAIN)	Competitive inhibitor of **DIHYDROOROTATE Hydrogenase** (**rate limiting enzyme** in the de novo synthesis of **pyramids**) -reduces lymphocyte proliferation -IL-10 Receptor mRNA **increase** -IL-8 recp mRNA **decrease** -TNF-a dependent nuclear factor kappa B (NF-kB) **reduce**	The active metabolite is **PHOSPHORAMIDE MUSTARD** Which **cross links the DNA to** prevent cell replication → suppresses t and b cells function about 30-40%
Pharmacokinetics	Elimination → hepatic	
Half life	4-28 days	
Administration	orally	Orally **not used I.V for treatment of RH **
Adverse effects	Hepatotoxicity → increase liver enzyme Bone marrow suppression Diarrhea → 25% patient Rashes + mild alopecia Weight gain Hypertension	In toxic dose : Nausea – vomiting – loss appetite or weight – abdominal pain – hair loss – change skin color – sores – diarrhea – change in color of fingernails or toenails ** **toxic occur in rapid growing tissues****
Monitor		
معلومات مهمه	Action of drug seen within 1 month **AVOID PREGNANCY UP TO 2 YEARS**	

	Cyclosporine	Mycophenolate Mofetil (MMF)
Mechanism of acrion (MAIN)	Metabolized by **CYTOCHROME P-450 A3** enzyme system	Hydrolyzed **to MYCOPHENOLIC acid** (**active metabolite**) → **immune suppressive moiety** **REVERSIBLE** inhibitor of enzyme **INOSINE MONOPHOSPHATE DEHYDROGENASE** → **anti proliferative** effects on T and B cells
Pharmacokinetics		
Half life		
Administration	Orally IV Inhalation Ophthalmic solution	Orally IV
Adverse effects	**Nephrotoxicity** Hypertension Hyperglycemia Liver dysfunction Hyperkalemia Altered mental status – seizures Hirsutism Lymphoma and (Kaposi's sarcoma – skin cancer) **due to induction of TGF-B**	كل شي يخطر ببالكم ^_^ Most important → **Primarily** neutropenia **REVERSIBLE** myelosuppression Increased incidence of infection Rarely malignancy اذا بدكم تشوفو الباقي موجودين بشيت 6 صفحه 8
Monitor	Close monitoring of the druge level to prevent drug–drug interaction Cyclosporine level Serum electrolytes Renal function Hepatic function Blood pressure Serum cholesterol	Plasma levels because it can cause **mylosuppression** and **neutropenia**
معلومات مهمه	**Can use it** with human organ **transplantation** and suppress graft versus host disease after **hematopoietic stem cell transplantation** also in selected **autoimmune disorders** → RH+ uveitis + psoriasis and asthma	Derived from PENICILLIUM GLAUCUS Efective in → renal disease due to systemic lupus erythematosus **Vasculitis and wanger** (sever RH)

- To treat RH using 2 to 3 DMARDs at a time to work better

- Common DMARDs combinations :

Using 3 drugs → **METHOTREXATE** + **SULPHASALAZINE** +hydroxychloroquine

Using 2 drugs → **METHOTREXATE + SULPHASALAZINE**

or

→ **METHOTREXATE +**leflunomide

or

→ **METHOTREXATE + hydroxychloroquine**

Biological agent biological therapy

Complex protein molecules

Examples →

Monoclonal antibodies targeting **TNF** → inflix**imab** and adalim**umab**

Soluble receptor decoy for TNF → etanercept → consists two soluble **TNF-a p75 receptor moieties** linked to the **Fc portion of human IgG1**

Receptor antagonist for IL-1 → anakinra

Monoclonal antibody to CD 20 → ritu**ximab**

UMAB→ part of this antibody comes from the human gene → humanized monoclonal antibody
IMAB or XIMAB → part of this antibody comes from the another organism → chimeric antibody

	INFLIXIMAB and adalimumab	Abatacept Modified antibody fusion protein
Mechanism of acrion	**Reducing** the **effects** of **TNF-a** by binding this antibody to the receptor of TNF-a on the surface of number of cells → preventing the cascade of events that would lead to the propagation of inflammation	Contains an endogenous ligand CTLA-4 that binds to CD80 and CD86 on the surface of cell → inhibiting the binding of CD28 and preventing the activation of T cells
Adverse effects	-Increase susceptibility of infection -Demyelinating disorder (neurological problems) -Increase chance of malignancy or tumor formation -Worsening congestive heart failure CHF	-Slight increase in risk of infection in the upper respiratory tract NOT recommended -**Abatacept + TNF-a antagonists** increased the risk of serious infection -Related to infusion : hypersensitivity and anaphylaxis -Increase risk of development of lymphomas **not other malignancies**
معلومات مهمه	- They are IMMUNOSUPPRESSIVE agent	

وضعي حاليا :

Gout

- type of **complex ARTHRITIS**
- _happen to one or more joints, the big toe is the most common
- happened because an **inflammatory reaction** in that side+ **metabolic disease**
- **DUE TO HYPERURICEMIA** → crystal deposition → bind with receptors on synoviocytes → synoviocytes engulfed the crystal → cytokines released → bring the attraction of **PMNs** → inflammation process

الالتهاب الحاد لي صار كان بسبب تدخل ال **pmns** لانها انفجرت واطلقت السايتوكاينز لي بداخلها

- **Main characteristics**

1- sudden onset

2-affect mainly middle-aged males

3-distal joints like in the big toe

4-Intense inflammation

NO ASPRINE WITH GOUT

5-recurrent episodes at any time

6-influenced by diet (الناس لي بياكلو بروتينات من مصادر حيوانيه بشكل مبالغ فيه)

7- appearance of bony erosions on X-ray

- Symptoms :

1- pain (sever) due to IL-1

2- swollen joint

3- redness

Diagnosis→ **hyperuricemia** and bony erosions on **X-ray(**you cans see cysts and joint destruction)

Chronic tophaceous gout

- chronic form of gout
- Characteristics **→ nodular masses**
  → localized deposit of monosodium urate crystals.
- very painful inflammatory process
- manifestations of gout that need pharmacological treatment:

- **uric acid in blood stream**
- Arthritis
- Tophi
- Nephrolithiasis (kidney stone) – nephropathy

Treatment by uricase (pegloticase)

- The treatment course includes the drug+ rest+ analgesia + time.

Treatment gout by NSAIDs

Anti inflammatory + pain killer

	Indomethacin	Colchicine
mechanism of action	inhibits urate crystal phagocytosis occurs also in most NSAIDs, **except for aspirin**	inhibition **microtubule** polymerization by binding to tubulin → obstructs the mitosis of cell and movement
Administration	four times daily, usually for(5- 7) days	
Adverse effects		****more common when patient has renal or hepatic disease.**** • Related to gastrointestinal (nausea, vomiting, cramping, diarrhea, abdominal pain) • muscular weakness • Related to hematologic (agranulocytosis, aplastic anemia, thrombocytopenia)
معلومات مهمه	used in the initial treatment of gout	**not** an **analgesic** +s **not** affect renal excretion of uric acid+**not** alter plasma solubility of uric acid+neither raises nor lowers serum uric acid

لما اتذكر انو ضايلي 3 صفحات فارما واختمها

Drugs That Block Production of Uric Acid

	Allopurinol (Zyloprim)	**Febuxostat**	**PEGLOTICASE**
mechanism of action	effectively blocks formation of uric acid *inhibitor of xanthine oxidase	**xanthine oxidase inhibitor**	recombinant mammalian **uricase** inhibitor that covalently binds to **methoxy polyethylene glycol MPEG**
Administration	orally		IV and **works fast** within the first **(24-72) hours** +stays in the body for (**6-13) days**
Adverse effects	*diarrhea, nausea, abnormal liver tests • acute attacks of gout •rash and allergic skin reaction • necrotizing fasciitis • Bone marrow suppression ***Very rarely** aplastic anemia and cataracts • Hepatic toxicity • Interstitial nephritis • allergic skin → start with rash and develops to MACULOPAPULAR **steven Johnson syndrome or epidermal necrolysis**.	• Diarrhea • headache • Nausea	➢ Infusion reaction ➢ Flare up of the gout, especially During the **first 3 months of treatment)** ➢ Nephrolithiasis (kidney stones) ➢ Arthralgia-Muscle pain and spam-Headache-Anemia-Nausea ➢ Peripheral edema-Diarrhea- Urinary tract infection ➢ **respiratory tract infection يحصل بشكل متكرر**
معلومات مهمه	**drug interactions** (ampicillin, thiazides, mercaptopurine, azathioprine)	minimal adverse effects comparing to allopurinol -who have sensitivity or to allopurinol, it's a **good alternative for allopurinol**	the addition of polyethylene glycol is **decreasing the antigenicity** for **uricase** enzyme ** **avoided with patients who suffer from glucose-6-phosphate dehydrogenase deficiency** **

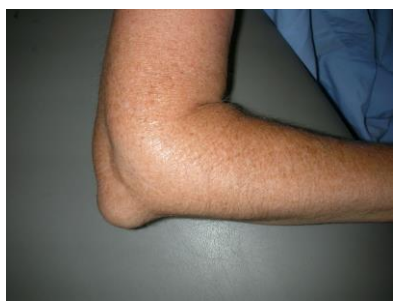
- **- usage indications of ALLOPURINOL:**
- **Management** of **hyperuricemia** of gout and **hyperruricemia** associated with chemotherapy
- **Prevention** of recurrent calcium oxalate kidney **stones**

Drugs That Enhance Excretion of Uric Acid

	Uricosuric therapy like probenecid
mechanism of action	**block tubular reabsorption of uric acid** +enhance urine uric acid excretion
contraindication	❑ not used in patients with **renal disease** ❑ patients with history of **nephrolithiasis** and who **excrete huge amount of uric acid** ❑ less effective in **elderly** patients because they usually have **deteriorated kidney function**.
Adverse effects	**mild** like GI irritation
معلومات مهمه	mainly used for **tophaceous** gout and **frequent gout attack** treatment

كمشه معلومات مهمه

خلص تعيطوش .. هيكم خلصتو



- **excessive** production of uric acid → use **xanthine oxidase inhibitor** as allopurinol or Febuxostat
- **inadequate** excretion--> use **uricosuric agent** such as probenecid

- acute olecranon bursitis → inflammation of the fluid-filled sac (bursa) that lies between a tendon and skin, or between a tendon and bone

- Treatment **start** with **NSAID** also we can also give the patient **steroid** and this last for about (**1-10 days)** before stop NSAID give **low doses of COLCHICINE** (**don't give ALLOPURINE at the first attack**) after that **wait a period** and then **stop NSAID** and **give allopurinol After** the **attack is subsided maintain** the patient on **colchicine and allopurinol(**colchicine isn't recommended so can give the patient NSAID in that period)

- when patient doesn't response to NSAID and colchicine→ give **IL-1receptor antagonis**t such as (Anakinra, Canakinumab Rilonacept) . Used to treat rheumatoid arthritis and currently for gout to inhibiting the inflammation
- **Glucocorticotiods-**-> like Prednisone, used during acute gouty arthritis attack. Can be administered orally, Intra-articular and Subcutaneous, this depends on the degree of pain, inflammation and the degree of acute attack.

شكلي بس خلصت فارما واحاول اتذكر لي درسته

اسبرينوباراسيتاميتوفيل الاعراض الجانبيه امراه حامل بالشهر 15 في معدتها