

## The Skin and MUSCULOSKELETAL System

## PHARMACOLOGY

DOCTOR: Omar shaheen

SLIDES SHEET SLIDE: 1

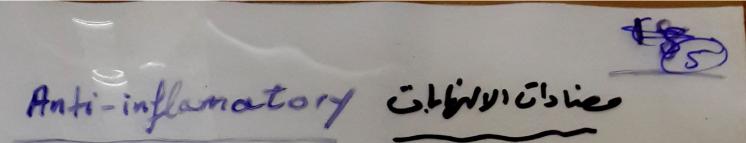
By Mohamed F. Abu Alia

Anti Brinflamatory Drugs Injury = -> Cause inflamation: Natural response to injury pretective response Types of inflamation ... I - Exogenous - Caurel by owide forors: a - physical m b- chemical cods, baser di; II - Indogenous = C-microbide . inappropriat activation of immune system. e.g. Rhematisd as thostis. : - pain, fever etc. - paliative # Characteristics - tissue repair . -> Maling - destroy invoding organism - functional disability = stop or delay inflamativy process (slaway) inflamatury process.

Inflamation -> complete -> inflamatory process subject smmune system cand differen non-sylfam injury -> inflamation progress -> pain continue healing -> disability continue cann it differtiate selfimmune system injury > and non solf arthritis :-- Reumatoid Egrovium (nourishment) Syrevial cauty Inflamatory attack: WBC activation. T-lymphocytes: (immunes) B- Symphocytes 3.34 heumatoid failse manageres + macrophages (inflamatory main ) 1 secret e autoantibodies -> maintain prosuffamatory cytokine : La invator strong the tor inflamation ----ip rogressive tissue injury no leutein .

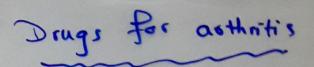
3 Effects of aytokines released histamine local effects Kinnins + prostaglandines systemic effects in joints noothelial cell Infiltration side effect Cartilage 2 chandrocytes : cells that maintain 1 poduction of appress heart proteolytre TASYMES - collagenases + - metalloposteringses degeneration of bleak down cartilage lung joint space narrowing 3-osteoclast; Tactivity liver 1- focal bone -> pai C-reactive proteins erostows produción -> reloose de mineralization masker of inflamation around Sornts relarse of proteolytic < nzyn Joint damage 7-spain , collage nese - Joint crosions poliability metalloproteinase decrease quality of life.

pharmacological interferance F = Creduce, modulate inflamatory process by - antiinflamaterry - agentls Creduce or modute inflamatory mot objective process -> by -> -> immunosuppressive - agonts 3 analgosics : clearth of modetic par 3 halting er at least slowing the mogression of disease



- Saligylates - paracetamol

- Non - selective Cox inhibitor NSAID; • Azapropazone
.Diclofenac
.Etodolac
· Fenbuten . Fenoprofen
Fenoprofen Skubiprofen Ibn profen Ketoprofen Meloxicam
Meloxicam
·Nabrezen
· solindac Solective (OK 2 inhibitor NSAIDS
. coleconib (celebren)
. rofecorch (viorx)



- Abatacept
- Adalimumab
- Anakinra
- Chloroquine
- Etenercept
- -Gold salts
- Infliximab
- leflunomide
- Methotrexate
- -D-Penicillamine - Rituximab

e

Prostaglandins

-6-

The reflects -> mechanism of action for NISAIDS 2-side effects - Job NISAIDS

can be explained and and understood Mough their inhibitory action of synthesis of prostaglanding synthesis.

Prostaglandines = Structure: 20 Carbon atoms in their Structure = Grosanoids - Eicosa = 20 carbons in showing atom + agelie ring Structure. belong te a group of compounds called = Autacoids: "pharmacologically active "compounds Characteurstics:- + formed locally by the tissues 6-act locally in these tissues (local hormonos) - produced by many tissues. (differ from harmons that (differ from harmons that preduced by specific organs) reduced by specific organs) apidly metabelized to inactive products don't circulate in the label at site d-