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PHARMACOLOGY

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- Helpful during revision.



MS Umamageswari

Forewords
TP Kalaniti
V Kumaran

JAYPEE

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MBBS Examination 2006

ANSWER ALL QUESTIONS

I. Essay questions (10 Marks each)

1. a. Classify thyroid inhibitors.
   b. Describe in detail the mechanism of action and the adverse effects of thyroid hormone synthesis inhibitors.
   c. What are their advantages and disadvantages?
   d. What is the role of radioactive iodine in thyroid disorders?
2. a. Classify anti-parkinsonian drugs.
   b. Discuss the actions and adverse effects of L-dopa.
   c. What are the benefits obtained by combining L-dopa with decarboxylase inhibitors?
   d. Write two drugs inducing Parkinsonism and how do you treat drug-induced parkinsonism.
3. a. Describe the pathophysiology of congestive cardiac failure.
   b. How will you manage a case of acute heart failure?
   c. What is the treatment of digitalis toxicity?
4. a. Classify skeletal muscle relaxants.
   b. Describe in detail about the mechanism of action and therapeutic uses of succinyl choline.
   c. Write about the adverse effects of succinylcholine apnea.
5. a. Classify diuretics.
   b. Describe in detail the mechanism of action of loop diuretics and their adverse effects.
   c. What are the therapeutic effects of Spironolactone?
6. a. Describe the various drugs used in the treatment of tuberculosis.
   b. What do you understand by DOTS?
   c. How do you manage a patient of MDR tuberculosis?
7. a. Classify corticosteroids according to their duration of action.
   b. Describe in detail the mechanism of action and therapeutic uses of corticosteroids.
   c. Adverse effects and contraindication of corticosteroids.
   d. Mention the names of two glucocorticoids antagonist.
8. a. Write the chemical classification of antimalarials.
   b. Describe in detail the mechanism of action and adverse effect of chloroquine.
   c. Write about therapeutic uses of chloroquine.
   d. How will you treat a case of chloroquine-resistant *P. falciparum* malaria?

II. Short notes (4 Marks each)

1. Bacterial resistance.
2. Allopurinol.
3. Management of chloroquine-resistant malaria.
4. Selective estrogen receptor modulators (SERM).
5. Anti-*Helicobacter pylori* drugs.
6. Uses and contraindications of purgatives.
7. Hormone replacement therapy.
8. Anti-HIV agents.

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9. Dihydrofolate reductase inhibitors in therapy.
10. Tocolytic agents.
11. Enoxaparin.
12. Phase I reactions in biotransformation.
13. Therapeutic uses of Atropine.
14. K^+ channel openers.
15. Pantaprazole.
16. Iron preparations.
17. Broad spectrum penicillins.
18. Toxicity of aminoglycoside antibiotics
19. Praziquantel.
20. Methotrexate – leucovorin rescue.
21. Tacrolimus.
22. Injectable contraceptive.

I. ESSAY QUESTIONS

1. a. Classify thyroid inhibitors.
b. Describe in detail the mechanism of action and the adverse effects of thyroid-hormone synthesis inhibitors.
c. What are their advantages and disadvantages?
d. What is the role of radioactive iodine in thyroid disorders?

a. Classify thyroid inhibitors.

Classification of Thyroid Inhibitors

- *Inhibit hormone synthesis (Anti-thyroid drugs):*
  - Propylthiouracil, methimazole, carbimazole
- *Inhibit iodide trapping:*
  - Thiocyanates, perchlorates
- *Inhibit hormone release:*
  - Iodine, iodides
- *Destroy thyroid tissue:*
  - Radioactive iodine

b. Describe in detail the mechanism of action and the adverse effects of thyroid-hormone synthesis inhibitors.

Mechanism of Action of Thyroid-hormone Synthesis Inhibitor

- Thyroid hormone synthesis inhibitors bind to thyroid peroxidase and inhibit oxidation of iodide/iodotyrosyl residues. So they inhibit:
  - Iodination of tyrosine residues in thyroglobulin
  - Coupling of iodotyrosine residues and T3, T4 formation
  - They cause depletion of thyroid colloid and also blood T3/T4 level
  - Prophylphiouracil, also inhibits peripheral conversion of T4 to more active T3.

c. What are their advantages and disadvantages?

Advantages of Antithyroid Drugs

- Well tolerated and attain euthyroidism rapidly. Euthyroid level can be maintained by maintenance dose
- Produces reversible type of hypothyroidism.
- Do not aggravate exophthalmos
- Can be used in children and young patients
- Low doses of propylthiouracil can be used even in pregnant and nursing mothers where the radioactive iodine is contra-indicated.

Disadvantages of Antithyroid Drugs

- Prolonged treatment, even lifelong treatment is required
- Higher recurrence rate
- Less effective than radioactive therapy and surgery
- In congestive cardiac failure patients, these drugs produce slower action
- Not practicable in uncooperative patient.

d. What is the role of radioactive iodine in thyroid disorders?

Role of Radioactive Iodine in Thyroid Disorder

- Radioactive iodine (^{131}I) is used in hyperthyroidism due to Graves' disease or toxic nodular goiter
  It causes destruction of thyroid follicles and inhibits the replication of thyroidal follicle without affecting the surrounding tissues
- Partial ablation of thyroid is achieved and the cure is permanent
- But it may cause hypothyroidism and thyroid carcinoma in later life (rare)

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- It cannot be used in children and pregnancy.

2. a. Classify anti-parkinsonian drugs.
b. Discuss the actions and adverse effects of L-dopa.
c. What are the benefits obtained by combining L-dopa with decarboxylase inhibitors?
d. Write two drugs inducing parkinsonism, and how do you treat drug-induced parkinsonism.

a. Classify anti-parkinsonian drugs.

Classification of Anti-parkinsonian Drugs

Drugs Affecting Brain Dopaminergic System

- *Dopamine precursor:* Levodopa
- *Peripheral decarboxylase inhibitors:* Carbidopa, Benserazide
- *Dopaminergic agonists:* Bromocriptine, Ropinirole, Pramipexole
- *MAO-B inhibitor:* Selegiline, Rasagiline
- *COMT inhibitors:* Entacapone, Tolcapone
- *Dopamine facilitator:* Amantadine.

Drugs Affecting Brain Cholinergic System

- *Central anticholinergics:* Trihexyphenidyl, Benztropine, Procyclidine, Biperiden
- *Antihistaminics:* Orphenadrine, Promethazine.

b. Discuss the actions and adverse effects of L-dopa.

Levodopa

Mechanism of Action

- It is a prodrug. Crosses blood-brain barrier and gets converted into dopamine in striatal dopaminergic neurons by decarboxylase enzyme
- About 97–99% of levodopa is metabolized peripherally by peripheral decarboxylase enzyme.

Pharmacological Actions

Central nervous system

- It causes improvement in parkinsonism patient
- Hypotonia, rigidity resolves first followed by tremor
- Posture, gait, handwriting, speech, facial expression, mood, self-care are normalized slowly
- In some patients it causes excitement to frank psychosis
- It does NOT improve dementia.

Cardiovascular system

- Peripherally converted dopamine causes tachycardia
- No increase in BP. It causes Hypotension due to its central action.

Chemoreceptor triggered zone

Peripherally converted dopamine stimulates chemoreceptor triggered zone and causes vomiting.

Endocrine

- It inhibits prolactin release
- It increases GH release.

Adverse Effects

- *Wearing-off effect:* 'End of dose' deterioration which gradually develops into 'on-off' effect
- *'All or none' response:* Patient feels alternatively well and disabled
- Dyskinesia
- Psychotomimetic effect
- *Nausea, vomiting:* Due to stimulation of CTZ
- Cardiac arrhythmia and postural hypotension.

c. What are the benefits obtained by combining L-dopa with decarboxylase inhibitors?

- Levodopa is combined with peripheral decarboxylase inhibitor carbidopa
- Carbidopa inhibits only the peripheral metabolism of levodopa to dopamine not in the CNS, because carbidopa does not crosses blood brain barrier.

Benefits Obtained by Combining L-dopa with Decarboxylase Inhibitors

- Carbidopa prolongs plasma t ½ of levodopa
- Dose of levodopa can be reduced to $1/4^{th}$
- Decreases systemic concentration of dopamine → adverse effects like nausea, vomiting are reduced

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- Therapeutic concentration of levodopa can be attained easily
- Pyridoxine reversal of L-dopa does not occur
- Minimizes 'on and off' effect
- More improvement → also improves patients adequately responding to levodopa
  Dose: 1:10 to 1:4 (Carbidopa : L-dopa)

d. Write two drugs inducing Parkinsonism and how do you treat drug induced Parkinsonism

Refer 2005-Q.No: II-18

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3. a. Describe the pathophysiology of congestive cardiac failure.
b. How will you manage a case of acute heart failure?
c. What is the treatment of digitalis toxicity?

---

a. Describe the pathophysiology of congestive cardiac failure.

Congestive cardiac failure means a condition in which heart is unable to pump sufficient amount of blood to meet the metabolic demand of the body at all time.
It may be: Acute heart failure/chronic heart failure.

Etiology

- Pressure overload: Seen in hypertension and in stenosis of cardiac valves
- Volume overload: Mitral/aortic valve regurgitation
- Loss of cardiac muscle: Seen in MI
- Impairment of cardiac contraction: Myocarditis
- Restriction of cardiac filling: Constrictive pericarditis
- Other factors worsen cardiac failure: Fever, anemia, thyrotoxicosis, hypoxia, acidosis, arrhythmia.

Pathophysiology of Heart Failure

- Loss of function of cardiac muscle leads to compensatory increase in hemodynamic and neurohumoral function (peripheral vasoconstriction, tachycardia), i.e. increased sympathetic activity.

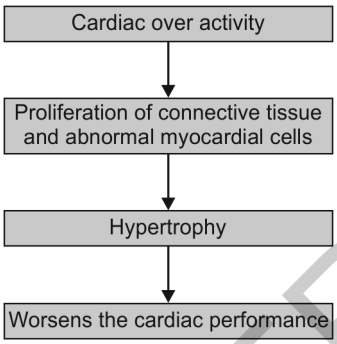

Fig. 1: Shows cardiac remodelling

- Increased preload → stimulation of rennin-angiotensin-aldosterone system → vasoconstriction → increases after load → increased cardiac performance
- Increased aldosterone secretion leads to sodium retention, increased extracellular volume, this in turn stimulates atrial natriuretic peptide → vasodilatation and natriuresis (opposing the effects of angiotensin II and aldosterone)
- Atrial distension stimulates baroreceptor → bradycardia → counteracts sympathetic stimulation
  This mechanism is called 'compensated cardiac failure'
- Ventricular remodelling is due to: (cardiac remodelling) **(Fig. 1)**.

b. How will you manage a case of acute heart failure?

Management of Acute Heart Failure

- Furosemide 40–80 mg IV repeated every 30 minutes. It causes diuresis and vasodilatation
- Morphine IV: Relives anxiety and reduces central sympathetic activity and pulmonary congestion by shifting blood from pulmonary circulation to systemic circulation
- Aminophylline IV 200–250 mg over 15 minutes: Used in patient with bronchospasm
- Sublingual nitroglycerine/IV nitroglycerine/IV sodium nitroprusside: Reduces pulmonary capillary pressure (by venodilatation). Blood pressure is monitored carefully to prevent severe hypotension

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- Digoxin IV: 0.5 mg IV over 15 min. Used in patient with atrial fibrillation/supraventricular tachycardia
- Inotropic drugs: Dopamine, dobutamine
  - Dopamine IV (3-10 μg/kg/min): Used in cardiogenic shock with myocardial infarction.

  Dose > 5 μg/kg/min: Increases afterload
  Dose at 2 μg/kg/min: Causes selective renal vasodilatation → restores diuretic response.
  - Dobutamine IV (2–8 μg/kg/min): Used in patients with myocardial infarction. It does not raise systemic vascular resistance.

c. What is the treatment of digitalis toxicity?

Stop Digoxin

- Mild toxicity (Ventricular premature beats): 5–7.5 g of oral potassium chloride
- More severe tachyarrhythmia: 20 m.mol/hour of KCl intravenously → It decreases the binding of digoxin to Na^+K^+ATPase and antagonizes digitalis induced automaticity
- For ventricular arrhythmia: Lidocaine IV 1–2 mg/kg loading dose followed by maintenance dose of 1–2 mg/kg at 20–30 minutes interval → It suppresses automaticity
- Supraventricular arrhythmia: Propranolol 10– 40 mg oral /0.5 mg IV, every 6 hours
- For AV-block: Atropine IV
- For severe toxicity: Antidigoxin antibody is used.

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4. a. Classify skeletal muscle relaxants.
b. Describe in detail about the mechanism of action and therapeutic uses of succinyl choline.
c. Write about the adverse effects of Succinyl Choline apnea.

---

a. Classify skeletal muscle relaxants.

Classification

I. Neuromuscular blocking agents
Nondepolarizing blockers (competitive)
1. Long acting: d-Tubocurarine, pancuronium, doxacurium, pipecuronium
2. Intermediate acting: Vecuronium, atracurium, cisatracurium, rocuronium, rapacuronium
3. Short acting: Mivacurium

Depolarizing blockers: Succinylcholine (suxamethonium), decamethonium
Directly-acting agents: Dantrolene sodium, Quinine
Other drugs: Aminoglycosides, tetracycline, polypeptide antibiotics.

II. Centrally-acting muscle relaxants
1. Mephenesin congeners: Mephenesin, carisoprodol, chlorzoxazone, chlormezanone, methocarbamol
2. Benzodiazepines: Diazepam
3. GABA mimetics: Baclofen, thiocolchicoside
4. Central α2 agonist: Tizanidine.

b. Describe in detail about the mechanism of action and therapeutic uses of succinylcholine.

Succinylcholine

Mechanism of Action

- Has high affinity for NM receptor than Ach
- It depolarizes the Na+ channels by acting on NM receptors and produces flaccid paralysis
- It produces Phase-I and Phase-II block.

Phase-I block

- Rapid onset
- Due to → persistent depolarization
- This phase is augmented not reversed by anticholinesterases like neostigmine

Phase-II block

- Slow onset
- Due to → desensitization of NM receptor because of continuous exposure to succinyl choline
- Phase-II is similar to non-depolarizing blocker like d-Tubocurarine
- Can be reversed by anticholinesterases.

Therapeutic Uses of Succinylcholine

- Adjuvant to general anesthetics (GA) as skeletal muscle relaxant

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  - During intubation, laryngoscopy, endoscopy, abdominal surgeries, thoracic surgeries, orthopedic manipulation.
- For assisted ventilation
- To prevent trauma and convulsion during 'electroconvulsive therapy'
- Severe cases of tetanus and status epilepticus.

c. Write about the adverse effects of succinyl choline apnea.

Succinylcholine (SCh) Apnea

- SCh is metabolized by pseudocholinesterase
- Patients with atypical pseudocholinesterase enzyme metabolizes succinylcholine slowly
- In those patients succinylcholine produces apnea for 4–6 hours (normal: 2–5 min).

Treatment

- Mechanical ventilation → supportive measure
- Fresh blood transfusion → provides pseudocholinesterase enzyme for succinylcholine metabolism.

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5. a. Classify diuretics.
b. Describe in detail the mechanism of action of loop diuretics and their adverse effects.
c. What are the therapeutic effects of Spironolactone?

---

a. Classify diuretics.

Classification

- **High ceiling (loop) diuretics.**
  - Furosemide (lasix), bumetanide, torsemide.
- **Medium efficacy diuretics.**
  Thiazides
  Hydrochlorthiazide, benzthiazide, hydroflumethiazide, bendroflumethiazide.
  Thiazide like:
  Chlorthalidone, metolazone, xipamide, indapamide, clopamide.
- **Weak/adjuvant diuretics:**
  K^+ sparing diuretics
  Spironolactone, eplerenone, triamterene and amiloride.
  Carbonic anhydrase inhibitors:
  Acetazolamide, dorzolamide.
  Osmotic diuretics:
  Mannitol, isosorbide, glycerol.

b. Describe in detail the mechanism of action of loop diuretics and their adverse effects.

Mechanism of Action of Furosemide

- Secreted in proximal tubule by OAT and inhibits Na^+-K^+-$2Cl^-$ in the thick ascending limb of loop of Henley
- Causes **greater natriuresis**
- Effective even at low glomerular filtration rates (e.g. chronic renal failure), where thiazides are ineffective.

Adverse Drug Reactions of Furosemide

- Hypokalemia, hypomagnesemia
- Acute saline depletion
- Dilutional hyponatremia
- Gastrointestinal tract (GIT) and central nervous system (CNS) disturbances
- Reversible hearing loss
- Allergic manifestation—because of sulfamoyl compound
- Hyperuricemia
- Hyperglycemia: K^+ depletion interferes with conversion of proinsulin to insulin
- Hyperlipidemia
- Hypocalcemia
- Mental disturbances
- Toxemia of pregnancy: Causes fetal death.

c. What are the therapeutic effects of Spironolactone?

Therapeutic Effects of Spironolactone

Spironolactone is the aldosterone antagonist.

- Aldosterone acts on mineralocorticoid receptors in DCT and CD → induces 'aldosterone-induced protein' and promotes Na^+ reabsorption and K^+ excretion
- Spironolactone binds with mineralocorticoid receptors, inhibits formation of aldosterone-induced protein

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  - Causes Na^+ excretion
  - Decreases K^+ excretion
- Spironolactone also increases Ca^{2+} excretion by direct action.

Therapeutic Uses

- Along with thiazide and loop diuretic: To counteract K^+ loss
- Treatment of cirrhotic edema, nephritic edema
- Hypertension: As an adjuvant to thiazide
- Treatment of primary hyperaldosteronism (Conn syndrome): Spironolactone is used
- Congestive cardiac failure: As an adjuvant drug to delay disease progression.

6. a. Describe the various drugs used in the treatment of tuberculosis.
b. What do you understand by DOTS?
c. How do you manage a patient of MDR tuberculosis?

a. Describe the various drugs used in the treatment of tuberculosis.

Classification of Antitubercular Drugs

Refer 2003-Q.No: I-6

b. What do you understand by DOTS?

- *DOTS:* Directly observed therapy short course
- In DOTS, a trained healthcare worker provides the prescribed anti-TB drugs and supervises the swallowing of the drug thrice weekly
- Consumption of every dose in initial intensive phase of therapy and at least the first dose of the week in continuation phase must be directly supervised
- It is cost-effective.

DOTS Regimen of RNTCP

Category	Patient type	Intensive phase	Maintenance phase
I	New sputum positive, New sputum negative, New extrapulmonary	2HRZE	4HR

Contd...

Contd...

Category	Patient type	Intensive phase	Maintenance phase
II	Smear positive cases previously exposed to anti-TB drugs, relapse smear positive, treatment failure smear positive, smear positive or negative recurrent extrapulmonary	2HRZES + 1HRZE	5HRE

H-600 mg, R-450 mg, Z-1500 mg, E-1200 mg, S-750 mg

c. How do you manage a patient of MDR tuberculosis?

Multidrug Resistant Tuberculosis

- Multidrug resistant tuberculosis (MDR-TB): Multidrug resistant tuberculosis is defined as resistance to both isoniazid and rifampin and may be any number of other first line drug
- Second line drugs are used for longer duration.

These drugs should have minimum 4 drugs.

- For H + R resistance: ZE + one second line drug + one fluoroquinolone + one/two injectable drugs (6 drugs) for 6–9 months in intensive phase (For example, ZE + ethionamide and cycloserine + ofloxacin + kanamycin along with pyridoxine)
- Followed by 4 drugs in continuation phase: Ethambutol + ethionamide and cycloserine + ofloxacin)
- Extensive drug resistance cases (XDR-TB): MDR-TB cases resistant to isoniazid, rifampin, fluoroquinolones and one of the injectable drug. It is difficult to treat
- XDR-TB is treated with drugs like thiacetazone, clarithromycin, clofazimine, linezolid, amoxicillin/clavulanate, imipenem/cilastatin, meropenum/clavulanate.

Newer drugs: Bedaquiline, delamanid, prothionamide

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7. a. Classify corticosteroids according to their duration of action.
b. Describe in detail the mechanism of action and therapeutic uses of corticosteroids.
c. Adverse effects and contraindication of corticosteroids.
d. Mention the names of two glucocorticoids antagonist.

a. Classify corticosteroids according to their duration of action.
Refer 2005-Q.No: I-8

b. Describe in detail the mechanism of action and therapeutic uses of corticosteroids.

Mechanism of Action of Corticosteroids

Refer 2005-Q.No: I-8

Uses of Corticosteroids

- ***Glucocorticoids:***
  Refer 2004-Q.No: I-2
- ***Mineralocorticoids:***
  – Addison's disease, salt-losing congenital adrenal hyperplasia, severe postural hypotension due to autonomic neuropathy.

c. Adverse effects and contraindication of corticosteroids.

Adverse Effects

- ***Glucocorticoids:***
  Refer 2005-Q.No: I-8
- ***Mineralocorticoid:***
  1. Sodium and water retention → edema
  2. Hypokalemia, alkalosis
  3. Rise in BP.
- **Contraindications of corticosteroids:**
  1. Peptic ulcer
  2. Diabetes mellitus
  3. Hypertension
  4. Viral and fungal infection
  5. Tuberculosis and other infection
  6. Osteoporosis
  7. Psychosis
  8. Epilepsy
  9. Congestive cardiac failure
  10. Renal failure.

d. Mention the names of two glucocorticoids antagonist.

Glucocorticoid antagonist: Mifepristone, high-dose ketoconazole.

8. a. Write the chemical classification of antimalarials.
b. Describe in detail the mechanism of action and adverse effect of chloroquine.
c. Write about therapeutic uses of chloroquine.
d. How will you treat a case of chloroquine-resistant *P. falciparum* malaria?

a. Write the chemical classification of antimalarials.

Classification

Refer 2003-Q.No: I-5

b. Describe in detail the mechanism of action and adverse effect of chloroquine.
Refer 2003-Q.No: I-5

c. Write about therapeutic uses of chloroquine.

Therapeutic Uses

In Malaria

- Treatment of *P. vivax*
- Uncomplicated *P. falciparum* malaria
- Does not have 'Teratogenic action. So, it can be used in pregnancy.

Other Uses of Chloroquine

- Extraintestinal amebiasis and giardiasis
- Rheumatoid arthritis
- Discoid lupus erythematosus
- Lepra reaction
- Photogenic reactions
- For symptomatic relief in infectious mononucleosis
- *Clonorchis sinensis* infestation.

d. How will you treat a case of chloroquine-resistant *P. falciparum* malaria?
Refer 2003-Q.No: I-5

II. SHORT NOTES

1. Bacterial resistance
Refer 2003-Q.No: I-25

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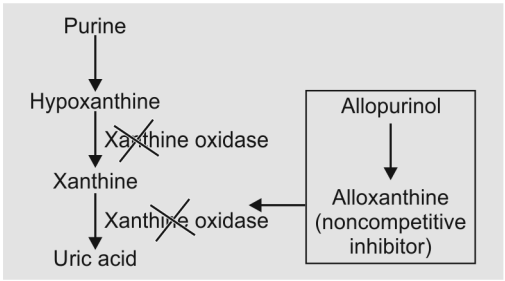

Fig. 2: Mechanism of action of allopurinol.

2. Allopurinol.

Allopurinol

Mechanism of Action

- Hypoxanthine analog → converted to alloxanthine → inhibits xanthine oxidase
- Increased hypoxanthine and xanthine level causes feedback inhibition of purine metabolism **(Fig. 2)**.

Adverse Drug Reactions

Hypersensitivity reaction, Stevens-Johnson syndrome, renal impairment, GI irritation.

Uses

Chronic gout, secondary hyperuricemia, to potentiate 6-MP action, Kala-azar.

Precaution

Should not be used in acute gout because of fluctuation of plasma level may cause recrystallization in joints. During initial therapy → acute gouty attacks are common.

3. Management of chloroquine-resistant malaria. Refer 2003-Q.No: I-5

4. Selective estrogen receptor modulators (SERM).

Drugs

Tamoxifen, Raloxifene, Ormeloxifene (Centchroman), Toremifene.

Selective estrogen receptor modulators are the drugs which has partial estrogenic activity in some tissues and antiestrogenic activity at some tissues.

Tamoxifen Citrate

Agonist: At uterus, bone, liver and pituitary.

Antagonist: At breast, blood vessels and some peripheral sites.

Pharmacological Actions

- It inhibits human breast cancer cells
- Stimulates endometrial proliferation
- Decreases gonadotropin and prolactin levels
- Improves bone density
- Decreases total LDL level
- It increases the risk of deep vein thrombosis due to its effect on clotting factor.

Uses

- Estrogen sensitive/nonsensitive breast cancer
- Metastatic breast carcinoma
- Male breast carcinoma
- Prophylaxis of breast cancer in high risk patients.

Adverse Drug Reactions

- Hot flushes, vaginal bleeding, menstrual irregularities
- Thromboembolism
- Risk of endometrial carcinoma
- Dermatitis, depression, leukopenia.

Toremifene: It is a newer congener of tamoxifen.

Raloxifene

Agonist: At bone, cardiovascular system, liver.
Antagonist: At endometrium, breast.

Pharmacological Actions

- It stimulates hot flushes.
- Improves bone mineral density and reduces the risk of vertebral fracture in postmenopausal women.
- Reduces LDL cholesterol.

Uses

Osteoporosis prevention in postmenopausal women.

Adverse Drug Reactions

- Hot flushes, leg cramps
- Increases risk of thromboembolism.

Ormeloxifene

Antagonist: At breast and uterus.

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Uses

- Dysfunctional uterine bleeding with anovulatory cycles
- Non-hormonal oral contraception.

Adverse Effects

- Fluid retention and weight gain
- Nausea, headache.

5. Anti-*Helicobacter pylori* drugs.

- *Helicobacter pylori* is the G –ve bacillus attaches to the surface of epithelium and promotes back diffusion of H^+ ions.
- It causes chronic gastritis, dyspepsia, peptic ulcer, gastric lymphoma and gastric carcinoma.

Drugs Used to Eradicate the *H. pylori*

Antimicrobials: Amoxicillin, clarithromycin, tetracycline, metronidazole, tinidazole.

Drugs Reduces Gastric Acid Secretion

- *Proton pump inhibitors:* Omeprazole, Esomeprazole, Lansoprazole, Pantoprazole, Rabeprazole
- *H_2 blockers:* Ranitidine, Famotidine, Roxatidine
- *Ulcer protectives:* Colloidal bismuth subcitrate.

Anti-*H. pylori* Regimen

One of the proton pump inhibitors with 2 or 3 antimicrobials used for 2 weeks.

Triple Drug Regimen

US-FDA Regimen

Lansoprazole 30 mg + Amoxicillin 1000 mg + Clarithromycin 500 mg → Twice daily for 2 weeks.

Other Regimen

Omeprazole 40 mg once daily + Metronidazole 400 mg three times a day + Amoxicillin 500 mg three times a day for 2 weeks.

Four Drug Regimen (Used in Eradication Failure Cases)

Colloidal bismuth subcitrate 120 mg four times daily + Tetracycline 500 mg four times daily + Metronidazole 400 mg three times daily + Omeprazole 20 mg twice daily for 2 weeks.

6. Uses and contraindications of purgatives.

Uses of Purgatives

- *Functional constipation:*
  - *Spastic constipation:* Stools are hard, rounded and stone like. Dietary fibers/bulk-forming agents are used.
- Stimulants purgatives are contraindicated
  - *Atonic constipation:* Common in geriatric cases. Bulk-forming agents/bisacodyl/senna (stimulant purgatives) are used.
- *Bedridden patients:*
  - *Prevention:* Bulk-forming agents, docusates, lactulose and liquid paraffin are used
  - *Treatment:* Bisacodyl/Senna are used.
- *To avoid straining at stool:* In patients with hernia, cardiovascular diseases, eye surgery
- *Preparation of bowel for surgery:* Before colonoscopy, abdominal X-ray
- *After anthelmintic therapy (for tapeworm):* Saline purgative or senna → used to flush out the worm
- *Food/drug poisoning:* To evacuate the unabsorbed material.

Saline Purgative are Used

- In patient with painful anal conditions such as fissure, thrombosed piles: Bulk-forming agents are used
- Patient with neuromuscular diseases: Bulk-forming agents are indicated
- Patient with hepatic encephalopathy: Lactulose is used.

Contraindications

- Undiagnosed abdominal pain
- Secondary constipation due to stricture/obstruction, hypothyroidism
- Along with drug such as opiates, sedatives, anticholinergics, oral iron, clonidine, calcium-channel blockers (constipating drugs)

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MS Umamageswari MD is Associate Professor of Pharmacology, KMCH Institute of Health Sciences and Research, Coimbatore, Tamil Nadu, India. She has contributed several articles in national and international journals and has presented several papers/posters in national fora. Her current fields of interest include medical education and medical ethics.

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