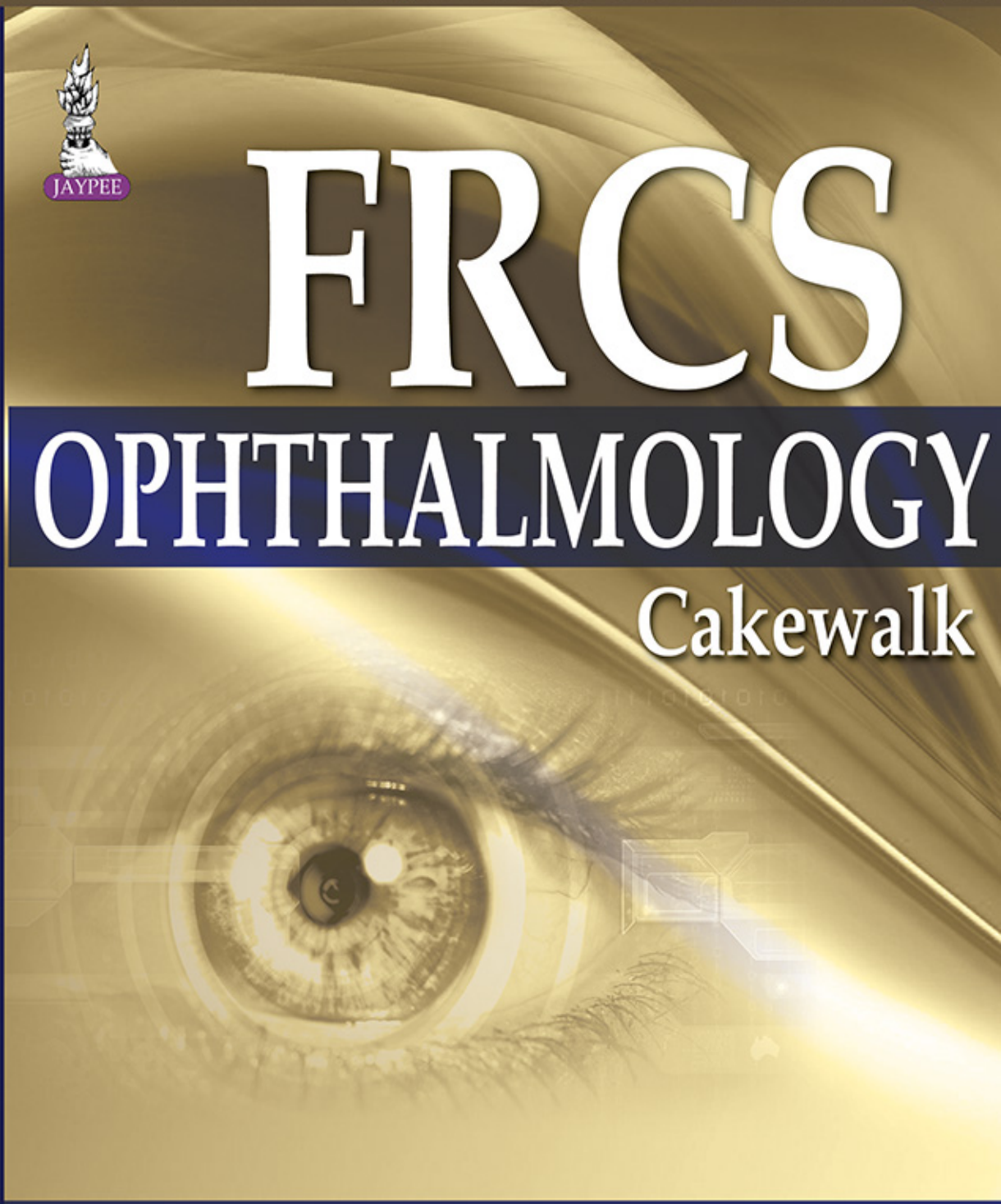

FRCS

OPHTHALMOLOGY

Cakewalk

Mamta Mittal

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Chapter 4

Ocular Pharmacology

Main Topics

4.1 Basics Pharmacology
4.2 Anti-infective Drugs
4.3 Antiglaucoma Medication
4.4 Immunosuppressives
4.5 Mydriatics and Cycloplegics
4.6 Anti-VEGFS and Vascular Endothelial Growth Factors
4.7 Botulinium Toxin
4.8 Tissue Adhesives
4.9 Ocular Viscoelastic Drugs (OVD)
4.10 Vitamins and Eye
4.11 Interferons in Ophthalmology
4.12 NSAIDS
4.13 Mast Cell Stabilizers
4.14 Ocular Toxicity Systemic Drugs

4 .1 BASICS PHARMACOLOGY

- **Concentration:**
  - 1% solution = 1 g/100 ml = 10 mg/ml
  - Example
  - How much atropine is there in 5 ml of 2% solution?
  - Answer 2% = 2 g/100 ml = 20 mg/ml
    - 1 ml has 20 mg of atropine, so 5 ml has 100 mg of atropine.
- **Barriers to topical medication:**
  - Properties of different corneal layers
  - **Epithelium:** Hydrophobic
  - **Stroma:** Hydrophilic
  - **Endothelium:** Hydrophobic
  - **Tight junctions of epithelium, endothelium:** Hinders hydrophilic medicines
  - **Stroma:** Hinders passage of lipophilic medicines
  - Systemic Medicines must pass through blood ocular, blood retinal barrier to reach the eye.
- **Methods of increasing absorption:**
  - **Add surfactant:** Disrupts epithelial intergrity, e.g. local anesthetics, benzalkonium chloride

- Close eyelid after instilling drops
- Punctal compression
- Increase frequency of drops.

- **Drug delivery systems:**
  - Drops, suspensions, ointments
  - Prodrugs, e.g. nevanac
  - Sustained release gels, e.g. pilocarpine
  - **Inserts:** Pilocarpine (ocusert), dexamethasone (ozudrex)
  - **Collagen shields:** Bandage contact lens presoaked in drug.

4.2 ANTI-INFECTIVE DRUGS

ANTIBIOTICS

Drugs	Mechanism	Spectrum	Indications	Adverse Effect
Inhibitors of Intermediary Metabolism (Static)				
Sulfonamide	Inhibit Folic Acid Synthesis	Gp, Gn, Toxoplasma, Chlamydia, Actinomycosis	Blepharitis, conjunc tivitis, toxoplasmosis	BM Suppression SJ syndrome, transient myopia
Trimethoprim (usually with Sulfmathoxazole-Bactrim)	Blocks next step in folate metabolism	Gp, Gn, toxo	Conjunctivitis	

Inhibitors of Cell Wall Synthesis

BETA–LACTAMS (CIDAL)				
Penicillins (PCN)G injectable		Strepto, Staphylo, Neisseria, Trepomeme	Syphilis, Throat Infection Pneumonia	Anaphylaxis Diarrhoea
Amino PCN • Ampicillin • Amoxicillin		Gp (except Staph), Enterobactere	Preseptal cellulitis	
Carboxy PCN • Carbenicillin • Ticarcellin		Gp, Gn, Pseudomonas, Enterobacterie, anaerobes		
Ureido PCN • Piperacillin • Mezlocillin • Azlocillin		Gp, Gn, Anaerobes (most active against Pseudomonas)	Hospital acquired infections (Not MRSA) Infections in neutropenic patients	

Cont...

Cont...

BETA–LACTAMS (CIDAL)				
Beta-lactamase Inhibitor Augmentin (Amox+Clavulanate) Flucloxacillin Temocillin	Increase spectrum of activity-due to beta-lactam inhibitor clavulanic acid in penicillin resistant bacteria	Resistant Staph Resistant Gn bacteria	Pneumonia Cellulitis Cellulitis Osteomyelitis Septicemea URI LRI	Anaphylaxis Diarrhoea

Cephalosporins- more resistant to beta-lactamase, cross react with PCN allergic patients			
1st Generation- oral • Cefazolin • Cefaclor	Gp, some Gn	Cefazolin for keratitis, endophthalmitis	Hypersensitivity
2nd Generation • Cefamandole • Cefoxitin • Cefuroxime	Better Gn, less Gp, anaerobes (most active against Heamophilus)		Hypersensitivity colitis
3rd Generation • Cefotaxime • Ceftazidime • Ceftriaxone • Moxalactam	Even better Gn, Pseudomonas, less Gp		Hypersensitivity colitis
Carbapenems For B-lactal- Mipenem, mase Doripenem resistant	All Gp, Gn, anaerobes (Not MRSA)	Hospital acquired infections (Not MRSA)	

Non Beta Lactamase (CIDAL)				
Polymyxin B	Simple peptide Disrupt cell membrane	Gn	Conjunctivitis	
Bacitracin (with Polymyxin)	Polypeptide	Gp, Nisseriae, Haemophilus, Actinomycosis,	Conjunctivitis	
Vancomycin Teicoplanin	Glycopeptide	Gp (inc MRSA) Clostridium difficile bacillus	Keratitis, endophthalmitis Surgical prophylaxis for MRSA	Systemic drugs- ototoxicity, nephrotoxicity

INHIBITORS OF PROTEIN SYNTHESIS				
Drugs	**Mechanism**	**Spectrum**	**Indications**	**Adverse Effect**
Aminoglycoside (Cidal) Gentamycin Tobramycin Amikacin Neomycin	Inhibit 30S ribosome	Gn, some Gp Tobra-better for Pseudomonas Amikacin-best for Pseudomonas, Neomycin-best for Acanthamoeba	Keratitis, Conjunctivitis	Systemic drugs-ototoxicity, nephrotoxicity
Tetracyclines (Static) Doxy, Minocyclines	Inhibit 30S ribosome Anti-inflammatory, anticollagenolytic activity	Gp, Gn, (esp Rickettesia, Mycoplasma, Chlamydia Brucella)	Prophylaxis and tt of ophthalmia neonatorum, rosacea, mebominitis, sclera melting	GI upset, phototoxic dermatitis tooth discolouration Nephr/hepatotoxic
Macrolides (Static) Erythromycin Azithromycin Clarithromycin	Inhibit 50S ribosome	Gp, few Gn, Chlamydia, Mycoplasma	Blepharitis, conjuctivitis	GI upset
Chloram-Phenicol (Static)	Inhibit 50S ribosome	Gp, Gn, anaerobes, Chla-mydia, Mycoplasma		Aplastic anemia BM suppression (reversible)
Clindamycin (Static)	Inhibit 50S ribosome	Gp, anaerobes toxoplasma	Toxoplasmosis	Pseudomembranous colitis (t/t-vancomycin)

QUINOLONES				
Drugs	**Mechanism**	**Spectrum**	**Indications**	**Adverse Effect**
Fluoroquinolones (Cidal)	• Inhibit DNA gyrase	• Aerobic Gn and some Gp • Chlamydia, Mycobacteria	• Conjuncti-vitis, • Keratitis, surgical prophylaxis	• GI upset, carti-lage damage in kids
2nd Gen Ciprofloxacin Ofloxacin Norfloxacin			• Ofloxacin-UTI, RTI, gonococcal infections	
3rd Gen Levofloxacin				
4th Gen Gatifloxacin Moxifloxacin Besifloxacin		• 4th Gen-against resistant bacterias and atypical mycobacteria		

ANTIVIRALS (STATIC) NUCLEOTIDE ANALOGUES

Drugs	Mechanism	Spectrum	Indications	Adverse Effect
TOPICAL Idoxuridine Vidarabine Triflurothymidine Ganciclovir Acyclovir	Inhibits genetic replication of virus in host cells		HSV keratitis	
Systemic Acyclovir Valacyclovir Famciclovir			HSV, HZV infections treatment/ prophylaxis	GI upset Nephro/ neurotoxic in high doses
Ganciclovir Foscarnet			CMV retinitis	

ANTIFUNGALS–Disrupt Cell Membranes

DRUG	Polyenes	Azoles		Pyrimidine	Echinocandins
Mechanism	• Bind to ergosterol, damage cell membrane • Poor corneal penetration • Broad spectrum	• Inhibit ergosterol synthesis • Broad spectrum		• Converted to 5-FU-disrupt DNA synthesis	• Disturb integrity of fungal cell wall
Preparations	• Amphotericin-for candida- first choice • Natamycin-Filamentous fungi	**Imidazoles** • Miconazole 1% • Ketaconazole • Clotrimazole	• Triazoles • 2 % drop, oral 200mg/day • Fluconazole itraconazole • Voriconazole	Fluocytocine	Capsofungin micafungin
Uses	• Fungal Endophthalmitis (in drug abuse, hyperalimentation • Fungal keratitis (first line treatment)	• **Voriconazole:** Best antifungal, good corneal penetration • **Itraconazole:** Aspergillus Candida • **Fluconazole:** Candida		• Most fungi are resistant ex cryptococcus, some chlamydia	• Refractory • Aspergillosis, candidemia, systemic mycosis
Side effects	• Fever hypotension headache, anemia nephrotoxic	• Hepatotoxic • Gynecomastia	• GI upset, headache rash	• Myelosuppression, nausea, vomit, diarrhoea • Nephro/hepatotoxic	• Hepatotoxic

4.3 ANTIGLAUCOMA MEDICATION

Common Questions Asked

- Classify antiglaucoma drugs. Which one to prefer and in what situations?
- Complications of beta blockers and miotics.
- Antiglaucoma medication – details with advantage and side effects.
- Management protocols.

Drug Classification	Side Effects	Contraindication
Beta-adrenergic antagonist **Nonselective β1 and β2** • Timolol • Timoptol 0.25%, 0.5% b.d. • Timoptol-LA 0.25%, 0.5 OD • Levobunolol 0.5% BD • Carteolol 1%, 2% BD Selective β1 antagonist • Betaxolol 0.5% BD	• Ocular toxicity • Bronchospasm (β2 blockade) • Bradycardia (β1 blockade) • CHF • Depression, anxiety • Impotence • Nocturnal hypotension • (so avoid at bed time) • Exacerbate MG	• COPD • CHF • Asthma • Diabetes • Bradycardia • Hypotension • Greater than first degree heart block
Selective alpha-adrenergic agonists **Brimonidine** 0.2% b.d. **Apraclonidine** 0.5%, 1% (tachyphlaxis–so no long term use)	• Allergic conjuctivitis • Xerostomia, • Drowsiness, fatigue headache	• MAO inhibitor therapy • Infanta and children <3 yrs (cross BBB) • Adrenergic sensitivity
Parasympathomimetic agents • Pilocarpine drops 1%, 2%, 3%, 4% QID as monotherapy • Pilocarpine gel bed time	• Increased myopia • Cataract • Brow ache • Corneal toxicity • Miosis	• Neovascular glaucoma • Uveitic glaucoma • Malignant glaucoma • Predisposition to RD
Carbonic anhydrase inhibitors **Topical** • **Dorzolamide** (Trusopt) 2% t.i.d • **Brinzolamide** (Azopt) 1% b.d. or tid **Oral** • Acetazolamide Tablets 250 mg • Methazolamide Tablets 50 mg	**Topical** • Metallic taste • Periocular dermatitis • Corneal epithelial toxicity • Choroidal detachment **Oral** • SJ syndrome • Malaise, anorexia, GI upset • Paraesthesiae • Renal calculi, • Hypokalemia • Aplastic anaemia- Dose-related • Thrombocytopenia-Idiosyncratic	• Sulfonamide allergy • Kidney stones • Aplastic anaemia • Thrombocytopenia • Sickle cell disease • Decompensated corneal endothelium

Contd...

Contd...

Drug Classification	Side Effects	Contraindication
Prostaglandin analogues: • **Latanoprost** 0.005% OD • **Travoprost** 0.004% OD • **Bimatoprost** 0.03% OD • **Tafluprost** 0.0015% OD	**Ocular** • Conjuctival hyperemia • Hypertrichosis • Periocular pigmentation • Anterior uveitis • Herpes infection activation • CME **Systemic** • Precipitation of migraine • Flu like symptoms • Muscle joint /back pain • Skin rashes	• Active macular edema • Active uveitis • History of herpetic keratitis • Pregnancy- terato-genic

- **Treatment goals**
  - **Target Pressure:** Therapy should maintain the IOP at or below the target level
  - **Monitoring** of the optic nerve and visual fields. In the event of further damage the target IOP is reset at a lower level.
- **Principles of treatment**
  - **Choice of drug:** Drug is chosen with maximum potency, less frequency of instillation, fewest side effects.
  - Initial treatment is usually with one drug, usually a beta-blocker or prostaglandin analogue.
- **Follow-up after 4 weeks**
  - A fall in IOP of >4 mm Hg is usually considered significant, subsequent assessment is after 2 months and at 3–4-monthly intervals thereafter.
  - If the response is unsatisfactory the initial drug is withdrawn and another substituted.
- **Follow-up after another 4 weeks**
  - If the response is still unsatisfactory yet another drug is added or a combined preparation substituted
  - Annual perimetry, gonioscopy, if optic disc is stable.

Why Beta-blockers are Contraindicated in Diabetes?

Because they may mask the symptoms of hypoglycemia.

What is the Mechanism of Action of CAI ? How do You Prevent Complications?

- Inhibits carbonic anhydrase enzme and blocks aqueous production
- Consider pretreatment blood counts, monitor blood potassium levels, avoid in c/o decompensated corneas, sulfa allergy and blood dyscrasias to prevent complications.

What is the Mechanism of Action of Beta Adrenergic Antagonists?

β1 and β2 receptors are present on nonpigmented ciliary epithelium. Receptor blockade reduces aqueous humor production.

What is the Mechanism of Action of Pilocarpine?

Reduces IOP by contacting ciliary muscle and pulling scleral spur. This tightens TM, increasing the outflow of aqueous humour.

How do You Prevent Side-effects of Antiglaucoma Medications?

Advise lacrimal punctum occlusion and tight eyelid closure for 3 min after instilling eyedrops.

Name Some Hyperosmotic Agents? When are They Used? Mechanism of Action? What are the Limitations?

- **Oral:** Glycerin 50 % 1–1.5 g/kg bw with lemon juice and ice
- **Systemic:** Mannitol 20 % 1–2 g/kg bw IV over 30 min
- **Indications:** Short-term or emergency treatment of high IOP, e.g. acute ACG, pupillary block or malignant glaucoma
- **Mechanism of action:** Reduce vitreous volume by creating osmotic gradient between blood and vitreous (so larger dose and rapid administration is important for greater reduction in IOP)
- **Limitations:** Limited effectiveness and duration of action (because blood ocular barrier is disrupted causing rebound elevation in IOP)
- **Complications:** Headache, urinary frequency and urgency, CHF, renal impairment, lethargy, hypersensitivity reactions, hyperkalemia, subdural/ subarachnoid hemorrhages.

4.4 IMMUNOSUPPRESSIVES

Drugs	Mechanism of Action	Main Indications
Hormones • Corticosteroids	**Inhibits phospholipase A2** (conversion of phospholipids to arachidonic acid) leading to decreased prostaglandins and leukotrienes	**First line therapy** to control acute ocular inflammations
Antimetabolites • Azathioprine • Methotrexate • Mycophenolate	• Purine analogues • Folate antagonists • Purine analogues	• Thyroid eye disease, behcets • Sarcoidosis and JIA • Contraindicated in children VKH syndrome
Immune modulators • Cyclosporine • Tacrolimus	• Fungal product inhibits T-cells activation, block IL2	• Behcets, VKH, sympathetic ophthalmia, birdshot retinopathy

Cont...

Cont...

Drugs	Mechanism of Action	Main Indications
Alkylating agents • Cyclophospha-mide	Nitrogen mustard derivative	Wegener's granulomatosis, Mooren's ulcer and ocular cicatricial pemphigoid
Biological blockers Tumour necrosis factor α • Inflixamab • Etanercept • Interferon alpha **IL-2 receptor antagonists**	• Remicade • Enbrel Daclizumab	• Vasculitis • Optic neuritis
Antibiotics: • Mitomycin C (MMC)		Pterygium, trabeculectomy
Pyrimidine analogues: • 5-fluorouracil (5-FU)		Pterygium, trabeculectomy

COMMON VIVA QUESTIONS

Ocular indications of immunosuppressive therapy

- **Cornea:**
  - Peripheral ulcerative keratitis
  - Mooren's ulcer
- **Ocular surface diseases** (ocular cicatricial pemphigoid, Stevens Johnson's syndrome)
- **Necrotizing Scleritis with systemic association.**
- **Uveitis:**
  - Bechet's disease
  - Serpiginous choroiditis
  - Vogt Koyanagi Harada
  - Sympathetic ophthalmia
  - Birdshot chorioretinopathy.
- **Orbit:**
  - Thyroid eye disease
  - Inflammatory orbital disease (pseudotumor).
- **Retinitis / vasculitis**
- **Optic neuritis**
- **When do you use oral steroids in uveitis?**
  - Intermediate uveitis unresponsive to posterior sub-Tenon injections.
  - Sight-threatening posterior or panuveitis, particularly with bilateral involvement.
  - Anterior uveitis resistant to topical therapy.

- **When do you use antimetabolites in uveitis?**
  - **Sight-threatening uveitis,** which is usually bilateral, noninfectious, reversible and has failed to respond to adequate steroid therapy
  - **Steroid-sparing therapy** in patients with intolerable side-effects from systemic steroids.
- **In what situations you avoid corticosteroids?**
  - Poorly controlled diabetes is a relative contraindication.
  - Peptic ulceration.
  - Osteoporosis.
  - Active systemic infection. (TB, herpes)
  - Psychosis on previous exposure to steroids.
- **What are the complications of steroid therapy?**
  - **Ocular:**
    - Cataract (posterior subcapsular type)
    - Glaucoma
    - Exacerbation of infection (bacterial)
    - Keratitis, fungal keratitis, HSV).
  - **Systemic:**
    - Cardiac complications (arrhythmias, heart)
    - Ulcer (gastric ulcer)
    - Hypertension, hirsutism
    - Ischemic necrosis of femur and osteoporosis
    - Neutropenia and infection
    - Growth problems in children
    - Psychosis.
- **How can you avoid the side effects of corticosteroids?**
  - **Prevent osteoporosis:** Calcium supplements, biphosphonates, etc. if treating with > 0.5 mg of oral prednisolone per day for > 3 months. Perform bone density scan
  - Prevent GI side effects by adding H2 blockers or PPP blockers
- **How do you withdraw steroids?**
  - According to Consensus Panel on immunosuppression for ocular disease.

Short course of any dose for <10 days	**No tapering required**
Over 40 mg/d	Reduce 10 mg/d every 1–2 weeks
40-20 mg/d	Reduce 5 mg/d every 1–2 weeks
20-10 mg/d	Reduce 2.5 mg/d every 1–2 weeks
10-0 mg/d	Reduce 1–2.5 mg/d every 1–4 weeks

- **What are the cautions you take with immunosuppressives?**
  - **Pretreatment:** Rule out TB
  - Patient education
  - **Precautions:** Avoid conception during treatment:
    - Urgent medical review if any infections like sore throat
  - **Regular blood tests:** FBC, LFT, check BP, sugar

- **What are the topical uses of cyclosporine?**
  Necrotizing scleritis, Sjogrens syndrome, ligneous conjunctivitis, atopic keratoconjuctivitis.

Describe the Main Side-effects of Immunosuppressive Therapy

Antimetabolites	
• Azathioprine	Bone marrow suppression, GI upset, sec malignancy, alopecia
• Methotrexate	Hepatotoxicity, bone marrow suppression, GI upset
• Mycophenolate	Bone marrow suppression, GI upset, sec malignancy
Immune modulators	
• Cyclosporine	Nephrotoxicity, HT, hepatotoxicity, gingival hyperplasia, hypertrichosis
• Tacrolimus	Nephrotoxicity, HT, neurotoxicity, hepatotoxicity, hyperglycemea
Alkylating agents Cyclophosphamide	Bone marrow suppression, GI upset, hemorrhagic cystitis
Biological blockers	
• Inflixamab	Serum sickness, TB reactivation
• Etanercept	TB reactivation, hypersensitivity reactions
• Interferon alpha	Leukopenia, depression, nephro and hepato toxicity, flu like symptoms, TB reactivation

QUESTIONS TO PRACTICE

- Complications of systemic steroids and immunosuppressants
- Which is worse immunosuppressive or steroids?
- Indications for oral corticosteroids in ophthalmology
- Contraindications of immunosuppressives.

4.5 MYDRIATICS AND CYCLOPLEGICS

COMMON VIVA QUESTIONS

How Many Mydriatic Preparations You Know of?

- **Short-acting:**
  - **Tropicamide** (0.5% and 1%) has duration of 6 hours.
  - **Cyclopentolate** (0.5% and 1%) has duration of 24 hours.
  - **Phenylephrine** (2.5% and 10%) has duration of 3 hours but no cycloplegic effects.
- **Long-acting:**
  - Homatropine 2% has duration of up to 2 days.
  - Atropine 1% is the most powerful cycloplegic and mydriatic with duration of up to 2 weeks.

Mydriatric Recovery Period

- Mydriatic recovery in normal eyes is as follows:
- Atropine (7–10 days), scopolamine (3–7 days), homatropine (1–3 days), cyclopentolate (1 day)
- And tropicamide (6 hours).

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Congratulations ! You have taken the first step on the path of your success in FRCS ophthalmology exam.
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