Pharma Unit

Pharmacotherapeutics Top 10 Most Repeated Questions with Answers

According to New Syllabus ER 2020-21

2nd Year D. Pharmacy

1) Define Pharmacotherapeutics? Explain scope and objectives of Pharmacotherapeutics?

Ans.

Definition:

Pharmacotherapeutics is a branch of pharmacology that deals with the therapeutic uses and effects of drugs.

Scope of Pharmacotherapeutics

1. Enhances knowledge and skills essential for the safe use and distribution of medicines by pharmacists and nurses in hospitals to ensure patient safety.
2. Promotes a deeper understanding of the pharmacist's role in retail settings, focusing on disease management and proper medication dispensing.
3. Facilitates the comprehension of the pathophysiology of common diseases and their effective management.
4. Strengthens foundational knowledge for professionals working in diagnostic and pathology laboratories.
5. Empowers patients by improving their functional capacity while reducing healthcare costs for both individuals and society.

Objectives of Pharmacotherapeutics

1. To identify and define the patient's problem requiring treatment.
2. To formulate appropriate therapeutic plans based on accurate diagnosis.
3. To ensure patient compliance with the prescribed therapy.
4. To verify the suitability and effectiveness of the chosen treatment.
5. To minimize medical errors in the therapeutic process.
6. To maximize the therapeutic benefits while minimizing adverse effects.
7. To outline possible therapeutic approaches for effective disease management.
8. To highlight the positive outcomes of drug therapy.
9. To develop personalized treatment plans tailored to individual diagnoses.
10. To conduct ongoing assessments for continuing or discontinuing treatment based on patient progress.

2) What do you mean by evidence-based medicines? Write its importance?

Ans.

Evidence-Based Medicine (EBM)

Evidence-based medicine is a systematic approach to medical practice where doctors and other healthcare professionals use the latest and most reliable scientific evidence from clinical research to make decisions about individual patient care

Objectives of Evidence-Based Medicine

1. To utilize current knowledge by combining patient preferences and clinical expertise, aiming to standardize and enhance care processes and improve patient outcomes.
2. To recognize the need for specific information while caring for a patient.
3. To identify the most reliable evidence available to address clinical problems effectively.
4. To integrate high-quality evidence into personalized medical plans.

Importance/Significance of Evidence-Based Medicine

1. Ensures the delivery of high-quality medical care and positive clinical outcomes.
2. Provides cost-effective medical solutions.
3. Promotes consistency in treatment protocols, leading to optimal results.
4. Helps establish national standards for patient care practices.
5. Keeps healthcare professionals informed and up to date with the latest medical advancements.
6. Encourages the adoption of effective practices while eliminating harmful or ineffective ones.

Steps Involved in Practicing Evidence-Based Medicine

1. Framing Answerable Clinical Questions: Formulate precise and relevant questions to guide patient care decisions.
2. Finding the Evidence: Search for the best available clinical research and evidence.
3. Appraising the Evidence: Critically evaluate the quality, validity, and relevance of the evidence.
4. Applying the Evidence: Incorporate the evidence into patient care, aligning it with clinical expertise and patient preferences.
5. Evaluating Performance: Assess the outcomes of the applied evidence to improve future practices

3) Explain Hypertension in detail?

Ans.

Definition: Hypertension is defined as a systolic blood pressure greater than 140 mmHg and a diastolic pressure greater than 90 mmHg.

Types of Hypertensions:

1. Primary Hypertension: This is the most common type, with no specific identifiable cause. It often involves narrowing of blood vessels, increasing blood pressure.
2. Secondary Hypertension: This type has an identifiable cause, such as kidney diseases, hormonal disorders (e.g., Cushing's syndrome, pheochromocytoma), vascular conditions (e.g., renal artery disease), or pregnancy-related conditions like preeclampsia.

Etiopathogenesis

1. Primary Hypertension: Exact cause unknown. Likely influenced by genetic and environmental factors.
2. Secondary Hypertension: Caused by specific conditions like:
   a) Renal diseases: Chronic glomerulonephritis, pyelonephritis, polycystic kidneys.
   b) Endocrine disorders: Cushing's syndrome, pheochromocytoma, primary aldosteronism.
   c) Vascular lesions: Renal artery disease, coarctation of the aorta.
   d) Other causes: Obesity, stress, high salt or fatty diet, alcohol intake, thyroid dysfunction, and pregnancy-related toxemia.

Clinical Manifestations: Severe headache, Fatigue, Anxiety, nervousness, dizziness, Sweating, facial flushing, Epistaxis (nosebleeds), vomiting, Difficulty breathing, Irregular heartbeats, Sensation of pulsation in the neck or head, Vision problems, Chest pain, Nosebleeds, Giddiness, palpitations

Non-Pharmacological Management of Hypertension

1. Lifestyle modifications is the first-line treatment
2. **DASH Diet:** Rich in fruits, vegetables, and low-fat dairy.
3. Reduce sodium intake, engage in regular exercise, Maintain a healthy weight., Limit alcohol consumption.
4. Quit smoking, Practice stress management, Incorporate yoga and meditation.
5. Minimize exposure to air pollution and cold temperatures.

Pharmacological Management of Hypertension

Pharmacological management involves using medications to control blood pressure.

1. Diuretics: Increase urine output by inhibiting sodium reabsorption in the distal convoluted tubules and loop of Henle.
   Examples: Hydrochlorothiazide, chlorthalidone, furosemide.
2. Beta (β)-Blockers: Reduce heart workload by slowing the heart rate and reducing its force.
   Examples: Propranolol, atenolol.
3. Alpha (α)-Blockers: Cause peripheral vasodilation of blood vessels.
   Example: Prazosin.
4. Calcium Channel Blockers (Ca^{2+} Blockers): Block calcium movement into cells, causing vasodilation and reduced heart rate.
   Examples: Nifedipine, amlodipine.
5. ACE Inhibitors: Inhibit angiotensin-converting enzyme, reducing angiotensin-II production and preventing vasoconstriction.
   Examples: Captopril, ramipril, enalapril.
6. Vasodilators: Directly relax arterial wall muscles, preventing narrowing.
   Examples: Hydralazine, nitro-glycerine, sodium nitroprusside.

4) Write details about CHF?
Ans.

Congestive Heart Failure (CHF)

Congestive heart failure (CHF) is a chronic, progressive condition where the heart fails to pump sufficient blood to meet the body's metabolic demands due to pathological changes in the myocardium. It is a serious condition characterized by reduced pumping capacity and is also referred to as "heart failure." Heart failure means the heart's inability to supply oxygenated blood needed for the body's functions.

Types of CHF (CCF)

1. **Left-Sided Heart Failure:** It is Caused by Ischemic heart disease, Myocardial diseases, Systemic hypertension
2. **Right-Sided Heart Failure:** It is Caused by Lung diseases, Pulmonary hypertension, Myocardial diseases, Tricuspid valve rupture

Causes of CHF: Coronary artery disease, Deficiencies of thiamine, selenium, calcium, etc, Alcohol, cocaine, cannabis use, Severe anemia, obesity, Congenital heart disease, Tachyarrhythmia, Valvular heart disease, Hypertension, Rheumatic fever, Pneumonia, Diabetes, hypo/hyperthyroidism, pheochromocytoma

Etiopathogenesis of CHF

1. Causes include pressure/volume overload, muscle loss, primary muscle disease, or excessive peripheral demands (e.g., high output failure).
2. The heart muscle becomes less contractile.
3. Common triggers include coronary artery disease (e.g., myocardial infarction), hypertension, atrial fibrillation, valvular heart disease, alcohol use, infections, and cardiomyopathy.

Clinical Manifestations of CHF: Shortness of breath, Excessive fatigue and weakness, Palpitations and irregular heartbeats, Nausea and loss of appetite, swelling in legs, ankles, feet, or abdomen, Chest pain, dyspnoea, Difficulty concentrating or staying alert, Nocturia, Fluid retention and slow weight gain.

Nonpharmacological Management of CHF

1. Quit smoking and alcohol consumption.
2. Regular exercise, Dietary and fluid restrictions.
3. Control comorbid conditions like hypertension and diabetes.
4. Maintain a healthy weight and diet, Manage stress effectively.

Pharmacological Management of CHF

1. **Beta-Blockers:** Metoprolol, atenolol, bisoprolol, celiprolol.
2. **ACE Inhibitors:** Captopril, enalapril, ramipril.
3. **Angiotensin Receptor Blockers (ARBs):** Losartan, candesartan, telmisartan, valsartan, irbesartan, Olmesartan.
4. **Cardiac Glycosides:** Digoxin, digitoxin.
5. **Diuretics:** Furosemide, hydrochlorothiazide, bumetanide, spironolactone.

5) Explain PCOS?
Ans.

Polycystic Ovary Syndrome (PCOS): Polycystic ovary syndrome (PCOS) is one of the most common hormonal disorders among women of reproductive age. It is characterized by the presence of ovarian cysts, anovulation (lack of ovulation), and endocrine disturbances. The cysts are fluid-filled and contain immature eggs. PCOS is also known as **Stein-Leventhal syndrome**, named after the American gynaecologists who first described it in 1935. The condition is marked by menstrual irregularities, hyperandrogenism (excess male hormones), obesity, and polycystic ovaries.

Etiopathogenesis of PCOS

1. **Insulin Resistance and Hyperinsulinemia:** Peripheral insulin resistance leads to hyperinsulinemia, a primary factor in disrupted ovarian function and excess androgen production. This may contribute to metabolic syndrome, which includes diabetes, dyslipidemia, and coronary artery disease (CAD).
2. **Decreased Sex Hormone-Binding Globulin (SHBG):** Low levels of SHBG increase free active androgens, causing hirsutism, acne, and other signs of hyperandrogenism.
3. **Elevated Estrone:** High estrone levels stimulate ovarian stromal cell hyperplasia, and unopposed estrogen affects the endometrium, increasing the risk of abnormal uterine bleeding and endometrial cancer.
4. **Biochemical Profile of PCOS:** Increased levels of androgen, luteinizing hormone (LH), testosterone, androstenedione, prolactin, and insulin. Low to normal follicle-stimulating hormone (FSH). Low to normal estradiol, with elevated estrone. Decreased SHBG

Risk Factors

1. Age: Women of reproductive age
2. Other Conditions: Obesity, subclinical vascular disease, type 2 diabetes, dyslipidaemia, non-alcoholic steatohepatitis, and obstructive sleep apnea
3. Genetics: Family history can increase risk
4. Ethnicity/Race: Certain populations may be more predisposed

Clinical Manifestations of PCOS: Menstrual disorders (e.g., amenorrhea, menorrhagia), Irregular or absent periods, Infertility or difficulty getting pregnant, Mood changes, depression, anxiety, Sleep apnea, Depression and anxiety disorders Insulin resistance, Obesity, Acne and oily skin, Excessive hair growth (hirsutism), Scalp hair thinning (alopecia), Difficulty losing weight, Darkened skin patches (Acanthosis nigricans)

Nonpharmacological Management of PCOS: Lifestyle Modifications which includes Weight loss and regular exercise, Healthy diet with low sugar and low-carbohydrate foods, Protein-rich diet to balance blood sugar levels, Stress reduction and emotional stability, Sound sleep and supplementation with key nutrients (magnesium, chromium, inositol, berberine)

Pharmacological Management of PCOS

1. **Combination Birth Control Pills:** Pills containing estrogen and progestin regulate hormone levels, reduce androgen production, and help manage menstrual irregularities, acne, and excess hair growth. They also lower the risk of endometrial cancer.
2. **Progestin Therapy:** Progestin can regulate periods and protect against endometrial cancer but does not improve androgen levels. It is usually taken for 10-14 days over 1-2 months.
3. **Ovulation Induction Medications:**
   Clomiphene: Oral antioestrogen taken in the first part of the menstrual cycle to stimulate ovulation.
   Letrozole (Femara): Stimulates the ovaries to promote ovulation.
   Metformin: An oral medication used to improve insulin resistance and lower insulin levels.
   Gonadotropins: Hormonal injections that stimulate ovulation.

6) Explain Dysmenorrhoea?
Ans.

Dysmenorrhoea: Dysmenorrhoea refers to painful menstrual periods, commonly caused by uterine contractions. It typically starts at the onset of menstruation, and the pain is often localized in the pelvis or lower abdomen. It is also known as menstrual cramps.

Types of Dysmenorrhoea

1. **Primary Dysmenorrhoea:**
   This is recurrent lower abdominal pain that occurs shortly before or during menstruation, without any underlying pelvic pathology.
2. **Secondary Dysmenorrhoea:**
   This is recurrent lower abdominal pain caused by an underlying pelvic pathology, which is usually present before or during menstruation.

Etiopathogenesis of Dysmenorrhoea: Dysmenorrhoea is primarily attributed to excessive production of prostaglandins in the endometrium, the lining of the uterus. Prostaglandins play a significant role in causing uterine contractions during menstruation. In primary dysmenorrhoea, the excessive levels of prostaglandins lead to stronger and more painful uterine contractions. This increased prostaglandin production during an ovulatory cycle result in more intense menstrual cramps. Secondary dysmenorrhoea, on the other hand, is caused by underlying pelvic conditions such as endometriosis, where endometrial tissue grows outside the uterus; uterine fibroids, noncancerous growths in the uterine wall; adenomyosis, where endometrial tissue invades the uterine muscle; and pelvic inflammatory disease (PID), an infection usually caused by sexually transmitted bacteria. Other causes include cervical stenosis, where a narrow cervix obstructs menstrual flow, and the presence of ovarian cysts or tumours. Additionally, the use of intrauterine devices (IUDs) may also contribute to secondary dysmenorrhoea.

Clinical Manifestations of Dysmenorrhoea

1. **Primary Dysmenorrhoea:** Intermittent crampy pain in the lower abdomen, Headache, nausea, vomiting, Diarrhoea, light-headedness, fatigue, Fever, muscle cramps, Nervousness, fainting, poor sleep quality.
2. **Secondary Dysmenorrhoea:** Throbbing or cramping lower abdominal pain, Dull, continuous ache, Heavy menstrual flow, Irregular menstrual cycles, Dizziness and headaches.

Nonpharmacological Management of Dysmenorrhoea:

- Apply a heating pad or hot water bottle to the abdomen or lower back.
- Rest as needed.
- Avoid foods with caffeine, smoking, and alcohol.
- Massage the lower abdomen and back.
- Engage in yoga and relaxation or breathing exercises.
- Acupuncture and acupressure may help.

Pharmacological Management of Dysmenorrhoea

1. **Pain Relievers (NSAIDs):** Examples: Diclofenac, ibuprofen, ketoprofen, mefenamic acid, naproxen.
2. **Hormonal Birth Control Pills:** Oral contraceptives containing hormones that prevent ovulation and reduce the severity of menstrual cramps.

7) Write about psoriasis?

Ans.

Psoriasis

Psoriasis is a chronic autoimmune skin condition characterized by excessive skin cell production, leading to thick, scaly patches. It causes itching, inflammation, and redness, commonly on the knees, elbows, trunk, and scalp. The accelerated skin cell turnover results in silvery scales and red patches, which may crack and bleed.

Etiopathogenesis of Psoriasis

Psoriasis is an autoimmune disorder where T-cells mistakenly attack healthy skin cells, leading to accelerated cell turnover. Normally, skin cells renew in 28–30 days, but in psoriasis, this process occurs 6–9 times faster, resulting in thick, silvery scales and red patches. Environmental triggers include cold weather, infections (especially group A β-haemolytic streptococci), trauma (Koebner response), certain drugs like lithium and NSAIDs, as well as stress, alcohol, and smoking. Genetic factors play a significant role, with a family history often present in affected individuals. Psoriasis is associated with other conditions like psoriatic arthritis and can vary in severity, from mild patches to large, widespread plaques.

Clinical Manifestations of Psoriasis:

1. Skin Symptoms: Red to Brownish-Gray patches covered with thick, silvery scales, Dry, swollen, and inflamed patches that may crack and bleed. Raised, thickened skin areas. Pus-filled blisters may appear in severe cases. Cracked and bleeding skin in severe or advanced cases.
2. Itching and Sensation: Pain, itching, and burning sensation in the affected areas. Restricted joint motion or pain due to psoriatic arthritis.
3. Nail Changes: Pitting, small depressions on the surface of the nails. Yellow, discolored nails.
4. Scalp and Hair: Dandruff-like scales on the scalp. Hair loss in severe cases due to the impact of psoriasis on the scalp.
5. Other Affected Areas: Genital lesions in males. Arthritis (psoriatic arthritis) in some patients, leading to joint pain and swelling. Raw, sensitive, swollen skin that may become more vulnerable to infections.
6. Systemic Symptoms: Mood changes, such as depression and anxiety, due to the emotional and physical impact of the disease.

Nonpharmacological Management of Psoriasis

Management of psoriasis includes avoiding known trigger factors, maintaining a healthy lifestyle, and using moisturizing and medicated creams. Patients are advised to take daily baths, cover affected areas overnight, and apply creams or ointments to alleviate symptoms. Stress management, regular exercise, smoking cessation, and avoiding alcohol are essential. Fish oil supplements and aloe extract creams may help soothe affected skin, and maintaining a healthy body weight is also beneficial.

Pharmacological Management of Psoriasis:

A. Topical Therapies:

1. Corticosteroids - e.g., Betamethasone cream): Reduce inflammation and suppress immune responses.
2. Coal Tar (0.3–33%): Alleviates itching and reduces skin cell turnover.
3. Keratolytic (e.g., Anthralin, Urea): Help remove scales and soften thickened skin.
4. Vitamin D analogues (e.g., Calcitriol ointment): Slow skin cell growth and reduce inflammation.
5. Salicylic Acid: Used in shampoos and scalp solutions to remove scales and soothe the scalp.

6. Goeckerman Therapy: Combines coal tar treatment with light therapy.

B. Light Therapy (Phototherapy):

1. Sunlight: Natural UV light helps reduce symptoms.
2. PUVA (Psoralen plus ultraviolet A): UV light exposure after taking a medication that increases skin sensitivity.
3. Excimer Laser: Targets specific skin areas with UV light.

C. Oral or Injected Medications:

1. Steroids (e.g., Triamcinolone): Reduce inflammation and suppress immune system activity.
2. Retinoids (e.g., Acitretin): Reduce skin cell turnover and scaling.
3. Methotrexate: Suppresses immune system and reduces skin cell production.
4. Cyclophosphamide: Used in severe cases to suppress the immune system.
5. Biologics (e.g., Etanercept, Infliximab, Adalimumab, Ustekinumab): Target immune system components to reduce inflammation and abnormal skin cell growth.
6. Miscellaneous: Drugs like Thioguanine, Hydroxyurea, and Cyclosporine may be used in certain cases.

8) Describe in detail about diabetes mellitus?

Ans.

Diabetes Mellitus

Definition: A metabolic disorder caused by insulin deficiency, leading to high blood sugar levels over an extended period. It is **Commonly Known as** 'Madhu-meha' (meaning "sweet body fluid"). **Hyperglycaemia is the** common result of uncontrolled diabetes. Insulin secreted by the beta-cells of the pancreas, helps maintain normal blood glucose levels.

Types of Diabetes Mellitus:

1. **Type 1 Diabetes (IDDM or Juvenile Onset Diabetes):**
   - Low insulin production.
   - Requires daily insulin administration.
2. **Type 2 Diabetes (NIDDM or Maturity Onset Diabetes):**
   - Most common type of diabetes
   - Involves insulin resistance and beta-cell dysfunction.
   - Often associated with obesity and overweight.

Etiopathogenesis of Diabetes Mellitus: The etiopathogenesis of diabetes mellitus involves defects in insulin secretion and action, along with disturbances in carbohydrate, fat, and protein metabolism. The primary cause is the destruction of beta-cells in the pancreas, which is influenced by genetic factors, insulin resistance, autoimmunity, inflammation, and environmental factors. For Type 2 diabetes, obesity and lack of physical activity are significant risk factors.

Clinical Manifestations of Diabetes Mellitus: Fatigue and irritability, Polyuria (frequent urination), Polyphagia (excessive hunger), Weight loss, Glycosuria (glucose in urine), Hyperglycaemia, Ketoacidosis, Ketonuria (presence of ketones in urine), Slow wound healing, blurring of vision, Frequent infections, Generalized pruritus (itchy skin), Diabetic retinopathy.

Nonpharmacological Management

1. **Healthy Diet:** Focus on balanced and nutritious foods.
2. **Physical Exercise/Yoga:** Regular activity to maintain healthy glucose levels.

3. **Limit Carbohydrate Intake:** Reduce simple sugar consumption.
4. **Avoid Risk Factors:** Such as smoking, stress, and hypertension.
5. **Monitor Blood Glucose Levels:** Regular monitoring to manage diabetes.
6. **Good Hygiene Practices:** Prevent infections and promote general health.
7. **Strict Meal Planning:** Adhere to a disciplined eating schedule.

Pharmacological Management

A. **Hypoglycaemic Drugs:**
- **Sulfonylureas:** E.g., chlorpropamide, tolbutamide, glibenclamide.
- **Biguanides:** E.g., phenformin, metformin.
- **Thiazolidinediones:** E.g., pioglitazone, ciglitazone.
- **Alpha-Glucosidase Inhibitors:** E.g., acarbose, miglitol.

B. **Insulin Therapy:**
- **Rapid-Acting Insulin:** Starts working within 15 minutes, lasts 3-4 hours.
- **Short-Acting Insulin:** Starts working within 30 minutes, lasts 6-8 hours.
- **Intermediate-Acting Insulin:** Starts working within 1-2 hours, lasts 12-18 hours.
- **Long-Acting Insulin:** Starts working after a few hours, lasts 24 hours.

9) Describe Rheumatoid arthritis?

Ans.

Rheumatoid Arthritis: Rheumatoid arthritis is a chronic, systemic autoimmune disease that primarily affects the joints, causing pain, swelling, warmth, stiffness, and deformity. Commonly Affected Areas are the wrist, and hands are most affected. In rheumatoid arthritis Inflammation of the synovial membrane leads to cartilage erosion, joint swelling, and painful immobility.

Etiopathogenesis of Rheumatoid Arthritis

Clinical Manifestations of Rheumatoid Arthritis: Painful, tender, and swollen joints, Weight loss, Low-grade fever, Anorexia, Anemia, Splenomegaly (enlarged spleen), Morning stiffness, Fatigue and muscle pain (myalgia), Depression, Muscle wasting

Nonpharmacological Management of Rheumatoid Arthritis

- Regular Exercise: To improve physical performance and joint flexibility.
- Heat and Cold Therapy: To relieve pain and relax muscles.
- Breathing and Muscle Relaxation: Techniques to manage stress and pain.
- Healthy Diet and Dietary Counselling
- Occupational Therapy: To assist with daily activities.
- Recreational Activities: Swimming and other beneficial activities.
- Splints: For pain relief and joint stability.
- Surgical Treatments: Such as joint replacement.

Pharmacological Management of Rheumatoid Arthritis

1. Anti-inflammatory Drugs (NSAIDs): E.g., aspirin, ibuprofen, piroxicam, etoricoxib, diclofenac, indomethacin.
2. Corticosteroids: E.g., prednisolone, methylprednisolone, triamcinolone.
3. Antirheumatic Drugs: E.g., gold compounds, penicillamine.
4. DMARDs (Disease-Modifying Antirheumatic Drugs): E.g., methotrexate, chloroquine, sulphasalazine, infliximab, cyclophosphamide.

10) Explain GERD?

Ans.

Definition: GERD is a chronic condition where stomach contents flow back into the esophagus, causing irritation or tissue damage. The reflux is involuntary and can lead to heartburn or indigestion.

Etiopathogenesis: GERD is mainly caused by frequent acid reflux due to the dysfunction of the lower oesophageal sphincter (LES). Factors like stomach distention, delayed emptying, large hiatal hernia, or excessive stomach acid contribute to the condition.

Clinical Manifestations:

A. Esophageal (Typical): Heartburn (retrosternal pain), Chest pain, Epigastric pain, Regurgitation (usually after meals), Dysphagia (difficulty swallowing), Odynophagia (painful swallowing), Nausea, belching, early satiety, bloating, water brash (excessive salivation), and halitosis (bad breath)
B. Extraesophageal (Atypical): Chronic cough, asthma, laryngitis, Bronchospasm, recurrent pulmonary infections, Otitis media, dental erosion, Nasal discomfort and trouble sleeping

Nonpharmacological Management:

- Maintain a healthy weight, stop smoking
- Elevate the head of the bed (6-9 inches)
- Avoid lying down after meals, eat slowly and chew thoroughly
- Avoid triggers like fatty foods, alcohol, chocolate, caffeine, etc.
- Avoid tight clothing around the waist.

Pharmacological Management:

1. Antacids - e.g., aluminium hydroxide, magnesium hydroxide
2. H2 blockers - e.g., cimetidine, ranitidine, famotidine
3. Proton Pump Inhibitors (PPIs) - e.g., omeprazole, pantoprazole, rabeprazole
4. Prokinetics - e.g., domperidone, metoclopramide

Extra Questions:

i. Explain Antimicrobial resistant disease
ii. Explain about Premenstrual syndrome
iii. Explain rational use of medicine
iv. Explain Clinical manifestation, and treatment of angina, myocardial infarction, asthma, COPD
v. Explain about thyroid disorder
vi. Explain about Parkinson and Alzheimer disease, Glaucoma and conjunctivitis

All The Best For Your Exam

Very Imp Note:

- Please Read All the chapters very carefully before Pharmacotherapeutics Exam.
- These questions are only for the reference purpose.