FARMAKOTERAPI OBAT SISTEM PENCERNAAN II dan ENDOKRIN

Penulis: Dr. dr. Fathiyah Safithri, M.Kes

I. Tujuan Belajar

Diharapkan mahasiswa mampu :

1. Memahami jenis-jenis insulin dan mampu menganalisis indikasi, kontraindikasi, efek samping, interaksi dan cara penggunaan insulin
2. Memahami jenis-jenis OAD dan mampu menganalisis indikasi, kontraindikasi, efek samping, interaksi dan cara penggunaannya
3. Memahami jenis-jenis obat hipolipidemi dan mampu menganalisis indikasi, kontraindikasi, efek samping, interaksi dan cara penggunaannya
4. Memahami jenis-jenis obat yang digunakan pada kasus GERD dan mampu menganalisis indikasi, kontraindikasi, efek samping, interaksi dan cara penggunaannya

II. *Prerequisite knowledge*

Sebelum melakukan praktikum mahasiswa harus memahami :

1. Biokimia metabolisme karbohidrat dan lemak
2. Patofisiologi DM tipe 2, Hiperlipid, GERD
3. Farmakodinamik dan farmakokinetik dasar

III. Kegiatan Pembelajaran

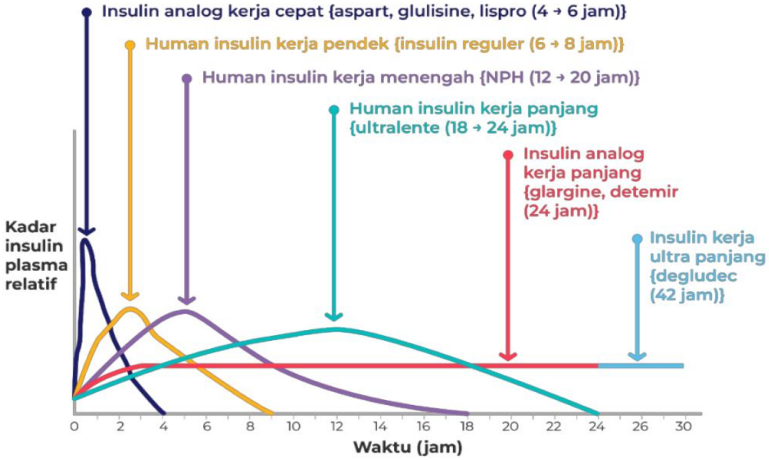
Pembelajaran dilakukan dalam tahapan sebagai berikut:

Tahapan pembelajaran	Lama	Metode	Pelaksana/ Penanggung Jawab
Pre tes	10 menit	Test	Dosen
Presentasi Pembahasan Modul dan diskusi	80 menit	Presentasi Tanya Jawab	Mahasiswa
Feed back dan resume	10	Ceramah	Dosen

IV. Sumber belajar

FARMAKOTERAPI INSULIN

Penggolongan Insulin

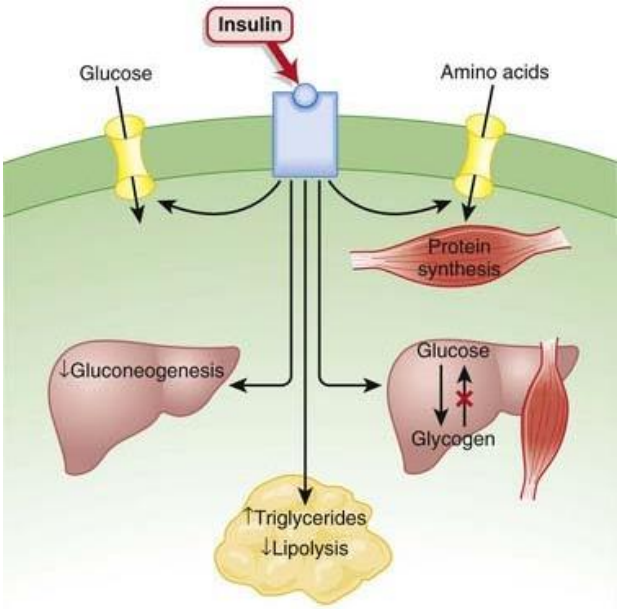


Gambar 1. Penggolongan insulin berdasar onset dan durasi (Perkeni, 2021)

Fungsi Insulin	Jenis Insulin	Profil Farmakokinetik	Awitan (Onset)	Puncak Efek	Lama Kerja	Kemasan
Basal	*Human Insulin*	Kerja Menengah • Humulin® N • Insulatard® • Insuman® Basal	1,5 → 4 jam	4 → 10 jam	5 → 12 jam	Vial *Penfill* Vial
	Analog	Kerja Panjang • Glargine (Lantus®) • Detemir (Levemir®)	1 →3 jam	Hampir tanpa puncak	12 → 24 jam	Pen/Vial 100 U/mL Pen 100 U/mL
		Kerja Ultra-Panjang • Degludec (Tresiba®)	30 → 60 menit	Hampir tanpa puncak	Sampai 48 jam	Pen
		• Glargine U300 (Lantus® XR)	1 → 3 jam	Tanpa puncak	>24 jam	Pen 300 U/mL
	Biosimiler Analog	Kerja Panjang • Glargine (Basaglar ®)	1 → 2 jam	Tanpa puncak	24 jam	*Vial catridge disposible* *Penfill catridge* 100 U/mL
		• Glargline (Ezelin ®)	1 → 2 jam	Tanpa puncak	24 jam	Pen/flexpen 100 U/mL
Prandial	*Human Insulin* (Reguler)	Kerja Pendek • Humulin R® • Actrapid ® • Insuman® • Sansulin®	30 → 45 menit	2 → 4 jam	6 → 8 jam	Vial *Penfill*
	Analog	Kerja Cepat • Lispro (Humalog®) • Aspart (Novorapid®) • Glulisin (Apidra®)	5 → 15 menit	1 → 2 jam	4 → 6 jam	*Vial/pen* *Flexpen* *Vial/pen*
Premixed	*Human Insulin*	• Humulin® 30/70 (30% regular, 70% NPH*)	30 → 60 menit	3 → 12 jam	14 – 24 jam	Vial 30/70 *Penfill*
		• Mixtard® 30/70 (30% regular, 70% NPH*)				
	Analog	• Humalog® Mix 25/75 (25% lispro, 75% protamin lispro) • Humalog® Mix 50/50 (50% protamin lispro, 50% lispro) • Novomix® 30/70 (30% aspart, 70% protamin aspart)	15 → 30 menit	1 → 4 jam	4 → 6 jam	Vial 10 mL; Pen 3 mL Penfill/flexpen
		• *Co-formulation Degludec-Aspart* : Ryzodeg® 70/30 atau iDegAsp (70% degludec, 30% aspart)	9 → 14 menit	72 → 80 menit	24 jam	Prefilled pen : 3 mL; 100 U/mL
Fixed-Ratio Combination **(Insulin Basal dan GLP-1 RA)**		Glargline/Lixisenatide (iGlarLixi) → Soliqua ®	Segera saat makan besar	Tanpa puncak	24 jam	*Pre-filled pen* : ▪ Soliqua® 10 – 40 (mengandung 100 unit Glargine/mL + 50 mcg Lixisenatide/mL *solution for injection*) ▪ Soliqua® 30 – 60 (mengandung 100 unit Glargine/mL + 33 mcg Lixisenatide/mL *solution for injection*)
		Degludex/Liraglutide (iDegLira) → Victoza ® dan Xultophy ® 100/3.6	Segera saat makan atau tanpa makan	Tanpa puncak	24 jam	*Pre-filled pen* : Xultrophy® 100/3.6 (mengandung 1 unit Degludec + 0.036 mg Liraglutide)

Keterangan : GLP-1 RA (*Glucagon like peptide-1 receptor agonist*), NPH (*Neutral Protamine Hagedorn*)

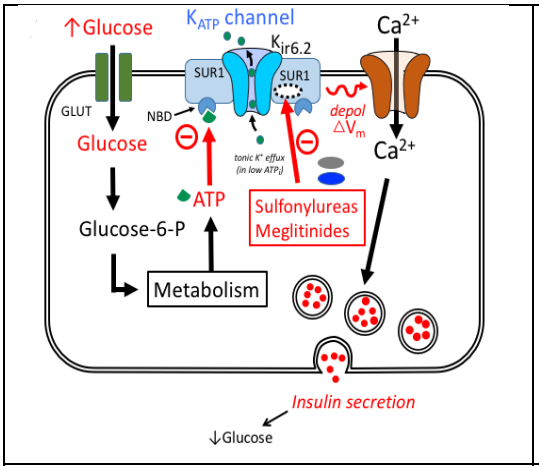
Gambar 2. Profil Kinetik Masing-masing Insulin (Perkeni, 2021)



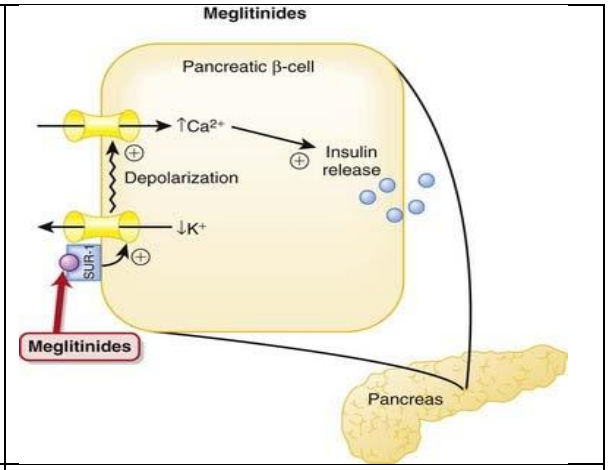
Gambar 3. Farmakodinami Insulin (clinical gate.com)

FARMAKOTERAPI ORAL ANTIDIABETIK

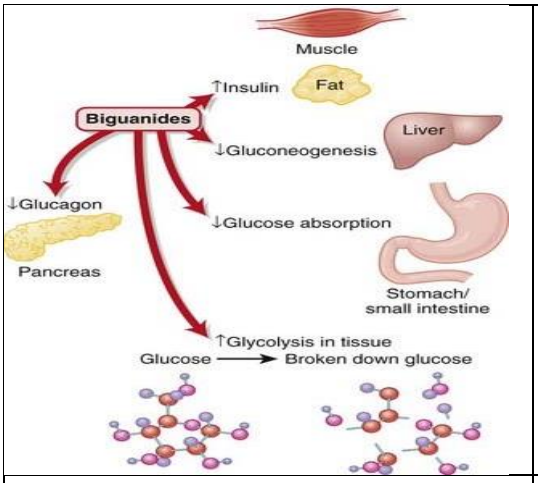
Farmakodinamik OAD

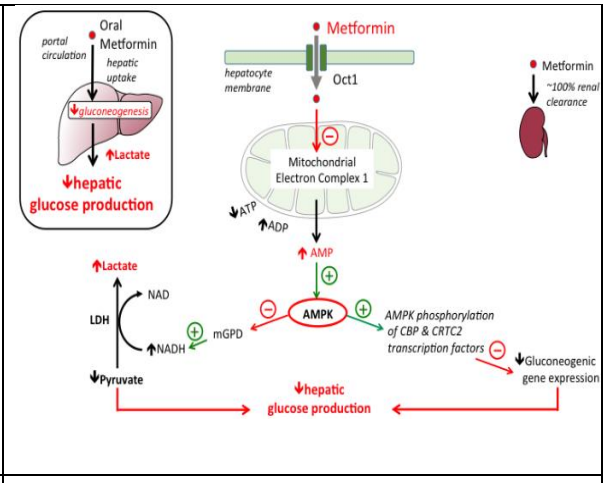


A. Sulfonilurea

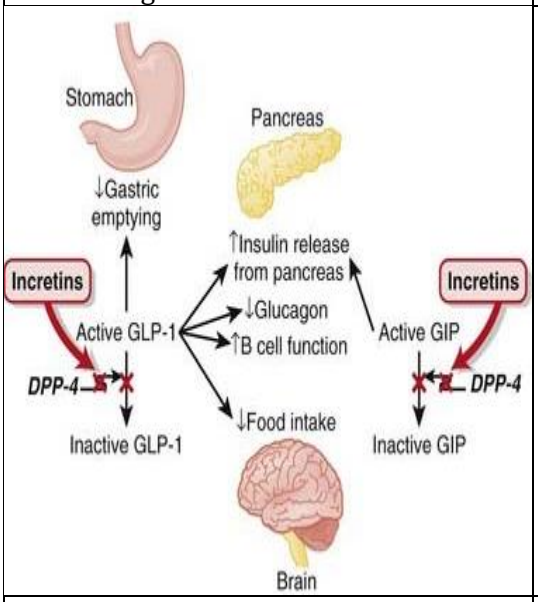


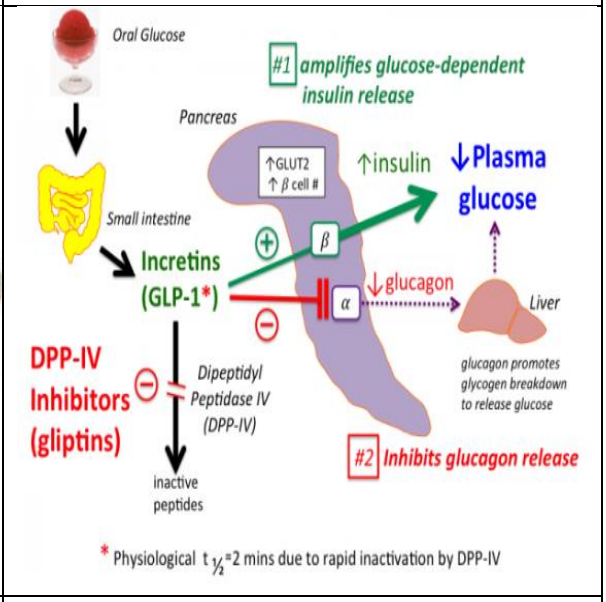
B. Meglitinide



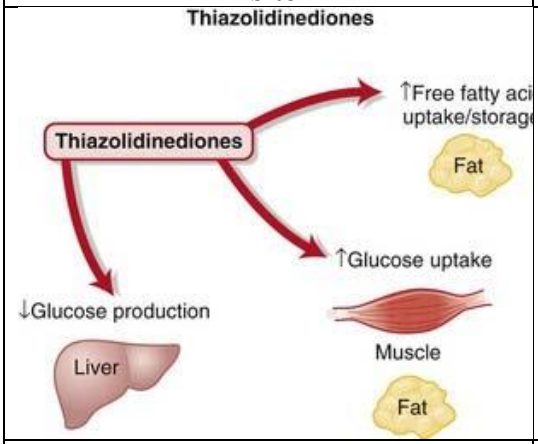


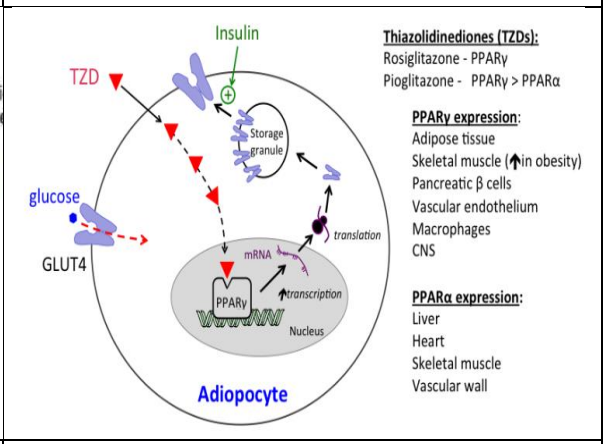
C. Biguanid



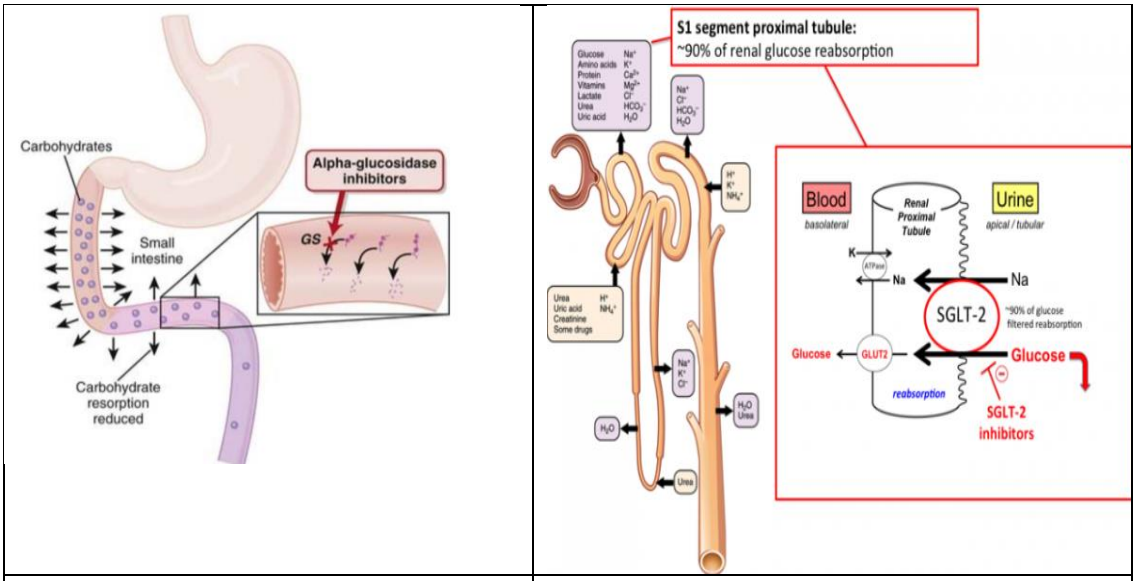


D. DPP-4 inhibitor





E. Thiazolidinedion



F. Alfa Glikosidase inhibitor | G. SGLT2 inhibitor

Gambar 4. Farmakodinami OAD

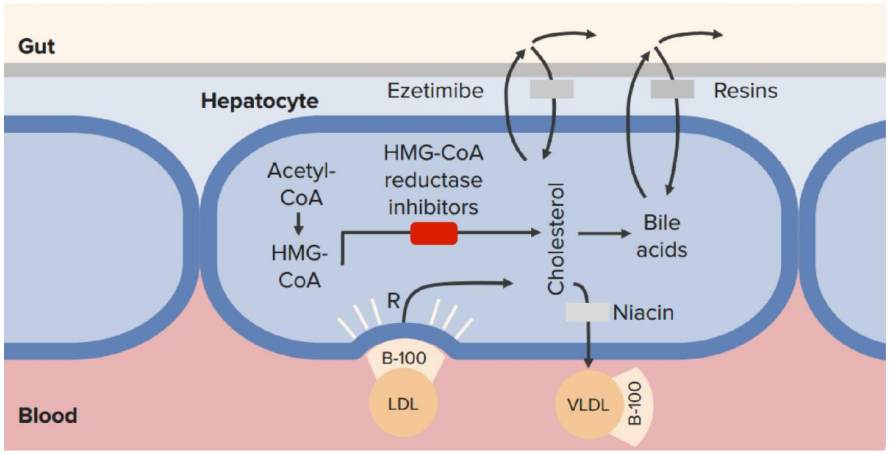
Profil Kinetik OAD

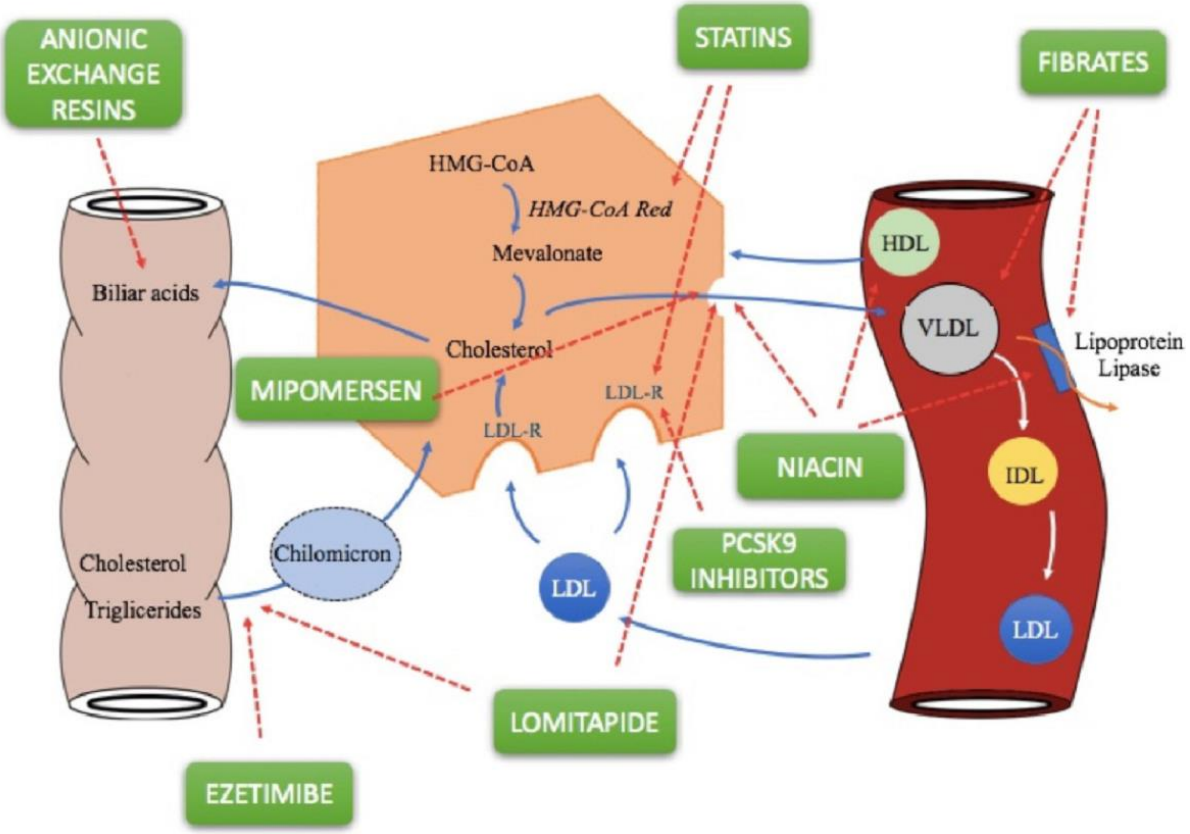
Subclass	Mechanism of Action	Effects	Clinical Applications	Pharmacokinetics, Toxicities, Interactions
INSULINS				
• Rapid-acting: Lispro, aspart, glulisine • Short-acting: Regular • Intermediate-acting: NPH • Long-acting: Detemir, glargine	Activate insulin receptor	Reduce circulating glucose • promote glucose transport and oxidation; glycogen, lipid, protein synthesis; and regulation of gene expression	Type 1 and type 2 diabetes	Parenteral (SC or IV) • duration varies (see text) • *Toxicity:* Hypoglycemia, weight gain, lipodystrophy (rare)
SULFONYLUREAS				
• Glipizide • Glyburide • Glimepiride	Insulin secretagogues: Close K^+ channels in beta cells • increase insulin release	In patients with functioning beta cells, reduce circulating glucose • increase glycogen, fat, and protein formation • gene regulation	Type 2 diabetes	Orally active • duration 10–24 h • *Toxicity:* Hypoglycemia, weight gain
• *Tolazamide, tolbutamide, chlorpropamide: Older sulfonylureas, lower potency, greater toxicity; rarely used*				
GLITINIDES				
• Repaglinide	Insulin secretagogue: Similar to sulfonylureas with some overlap in binding sites	In patients with functioning beta cells, reduces circulating glucose • increases glycogen, fat, and protein formation • gene regulation	Type 2 diabetes	Oral • very fast onset of action • duration 5–8 h • *Toxicity:* Hypoglycemia
• Nateglinide	Insulin secretagogue: Similar to sulfonylureas with some overlap in binding sites	In patients with functioning beta cells, reduces circulating glucose • increases glycogen, fat, and protein formation • gene regulation	Type 2 diabetes	Oral • very fast onset and short duration (< 4 h) • *Toxicity:* Hypoglycemia
BIGUANIDES				
• Metformin	Obscure: Reduced hepatic and renal gluconeogenesis	Decreased endogenous glucose production	Type 2 diabetes	Oral • maximal plasma concentration in 2–3 h • *Toxicity:* Gastrointestinal symptoms, lactic acidosis (rare) • cannot use if impaired renal/hepatic function • congestive heart failure (CHF), hypoxic/acidotic states, alcoholism

ALPHA-GLUCOSIDASE INHIBITORS				
• Acarbose, miglitol	Inhibit intestinal α-glucosidases	Reduce conversion of starch and disaccharides to monosaccharides • reduce postprandial hyperglycemia	Type 2 diabetes	Oral • rapid onset • *Toxicity:* Gastrointestinal symptoms • cannot use if impaired renal/hepatic function, intestinal disorders
THIAZOLIDINEDIONES				
• Pioglitazone	Regulates gene expression by binding to PPAR-γ and PPAR-α	Reduces insulin resistance	Type 2 diabetes	Oral • long-acting (> 24 h) • *Toxicity:* Fluid retention, edema, anemia, weight gain, macular edema, bone fractures in women • cannot use if CHF, hepatic disease
• Rosiglitazone	Regulates gene expression by binding to PPAR-γ	Reduces insulin resistance	Type 2 diabetes	Oral • long-acting (> 24 h) • *Toxicity:* Fluid retention, edema, anemia, weight gain, macular edema, bone fractures in women • cannot use if CHF, hepatic disease • may worsen heart disease
GLUCAGON-LIKE POLYPEPTIDE-1 (GLP-1) RECEPTOR AGONISTS				
• Exenatide	Analog of GLP-1: Binds to GLP-1 receptors	Reduces post-meal glucose excursions: Increases glucose-mediated insulin release, lowers glucagon levels, slows gastric emptying, decreases appetite	Type 2 diabetes	Parenteral (SC) • half-life ~2.4 h • *Toxicity:* Nausea, headache, vomiting, anorexia, mild weight loss, pancreatitis
• *Liraglutide: Similar to exenatide; duration up to 24 h; immune reactions, possible thyroid carcinoma risk*				
DIPEPTIDYL PEPTIDASE-4 (DPP-4) INHIBITORS				
• Sitagliptin	DPP-4 inhibitor: Blocks degradation of GLP-1, raises circulating GLP-1 levels	Reduces post-meal glucose excursions: Increases glucose-mediated insulin release, lowers glucagon levels, slows gastric emptying, decreases appetite	Type 2 diabetes	Oral • half-life ~12 h • 24-h duration of action • *Toxicity:* Rhinitis, upper respiratory infections, headaches, pancreatitis, rare allergic reactions
• *Saxagliptin, linagliptin: Similar to sitagliptin; longer duration of action*				
AMYLIN ANALOG				
• Pramlintide	Analog of amylin: Binds to amylin receptors	Reduces post-meal glucose excursions: Lowers glucagon levels, slows gastric emptying, decreases appetite	Type 1 and type 2 diabetes	Parenteral (SC) • rapid onset • half-life ~ 48 min • *Toxicity:* Nausea, anorexia, hypoglycemia, headache
BILE ACID SEQUESTRANT				
Colesevelam hydrochloride	Bile acid binder	Lowers glucose through unknown mechanisms	Type 2 diabetes	Oral • 24-h duration of action • *Toxicity:* Constipation, indigestion, flatulence

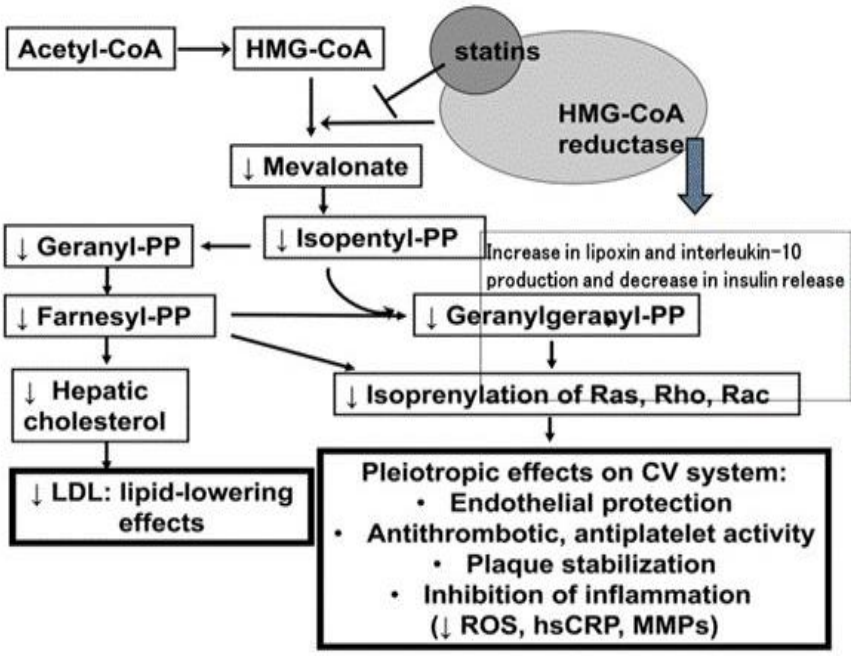
Gambar 5. Profil Kinetik OAD

FARMAKOTERAPI OBAT HIPOLIPIDEMI

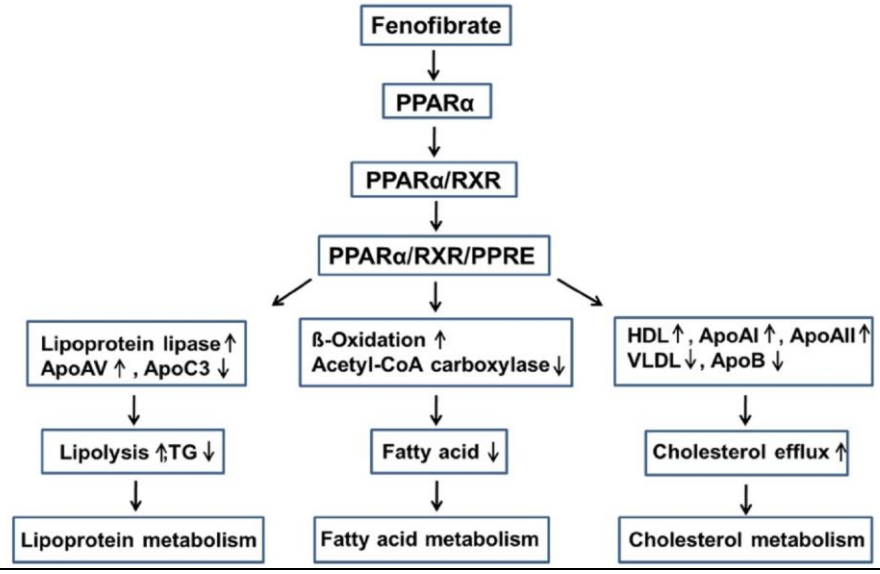




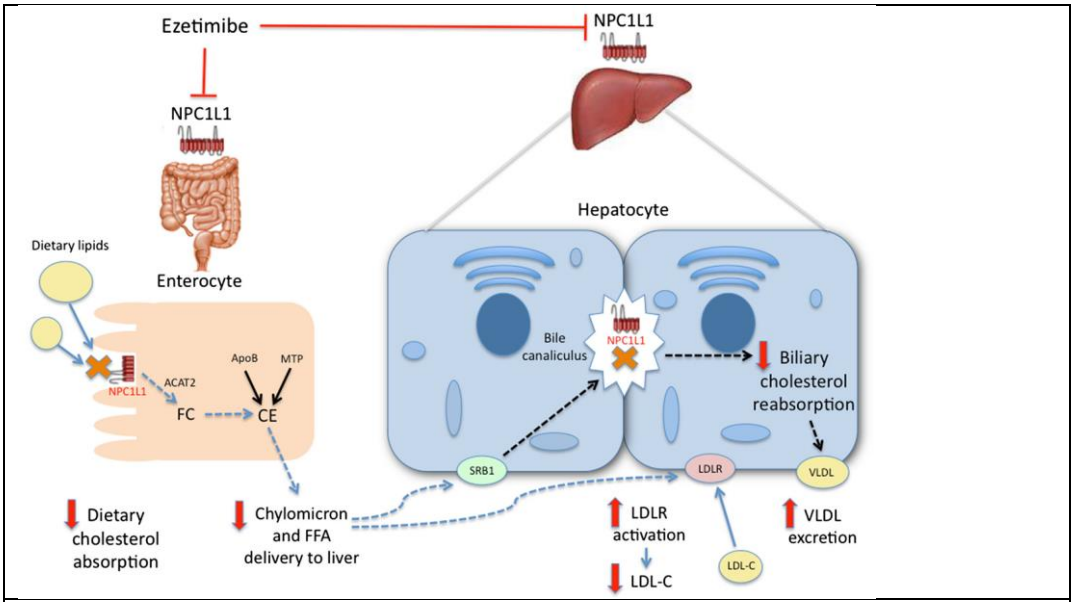
Gambar 1. Target Kerja Obat Hipolipidemia (Zodda, 2018)



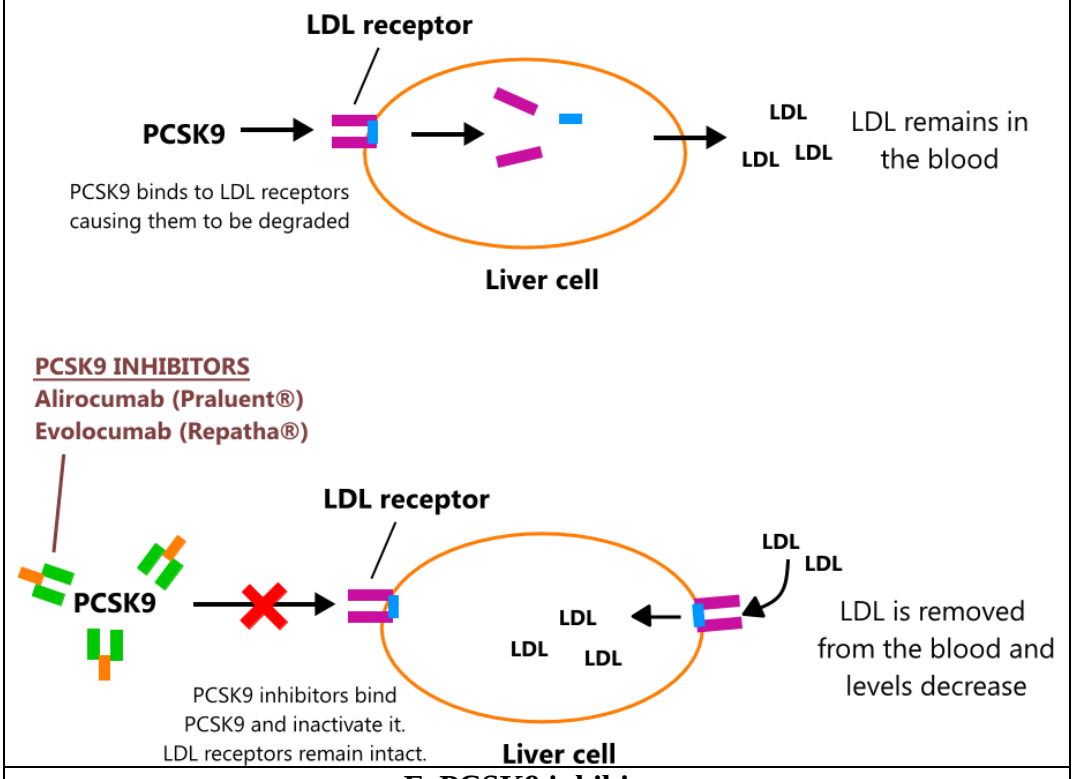
A. Statin



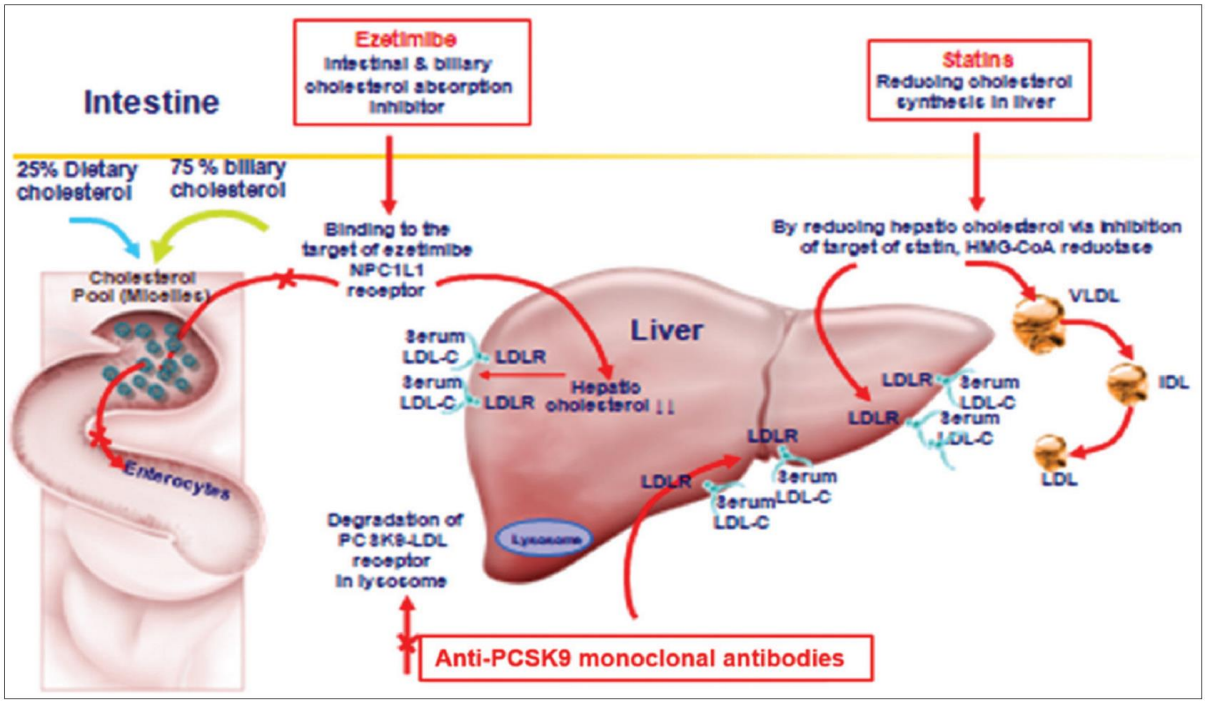
B. Fibrat



C. Ezetimibe



F. PCSK9 inhibitor



Subclass	Mechanism of Action	Effects	Clinical Applications	Pharmacokinetics, Toxicities, Interactions
STATINS				
• Atorvastatin, simvastatin, rosuvastatin, pitavastatin	Inhibit HMG-CoA reductase	Reduce cholesterol synthesis and up-regulate low-density lipoprotein (LDL) receptors on hepatocytes • modest reduction in triglycerides	Atherosclerotic vascular disease (primary and secondary prevention) • acute coronary syndromes	Oral • duration 12–24 h • *Toxicity:* Myopathy, hepatic dysfunction • *Interactions:* CYP-dependent metabolism (3A4, 2C9) interacts with CYP inhibitors
• *Fluvastatin, pravastatin, lovastatin: Similar but somewhat less efficacious*				
FIBRATES				
• Fenofibrate, gemfibrozil	Peroxisome proliferator-activated receptor-alpha (PPAR-α) agonists	Decrease secretion of very-low-density lipoproteins (VLDL) • increase lipoprotein lipase activity • increase high-density lipoproteins (HDL)	Hypertriglyceridemia, low HDL	Oral • duration 3–24 h • *Toxicity:* Myopathy, hepatic dysfunction
BILE ACID SEQUESTRANTS				
• Colestipol	Binds bile acids in gut • prevents reabsorption • increases cholesterol catabolism • up-regulates LDL receptors	Decreases LDL	Elevated LDL, digitalis toxicity, pruritus	Oral • taken with meals • not absorbed • *Toxicity:* Constipation, bloating • interferes with absorption of some drugs and vitamins
• *Cholestyramine, colesevelam: Similar to colestipol*				
STEROL ABSORPTION INHIBITOR				
• Ezetimibe	Blocks sterol transporter NPC1L1 in intestine brush border	Inhibits reabsorption of cholesterol excreted in bile • decreases LDL and phytosterols	Elevated LDL, phytosterolemia	Oral • duration 24 h • *Toxicity:* Low incidence of hepatic dysfunction, myositis
NIACIN				
	Decreases catabolism of apo AI • reduces VLDL secretion from liver	Increases HDL • decreases lipoprotein(a) [Lp(a)], LDL, and triglycerides	Low HDL • elevated VLDL, LDL, Lp(a)	Oral • large doses • *Toxicity:* Gastric irritation, flushing, low incidence of hepatic toxicity • may reduce glucose tolerance
• *Extended-release niacin: Similar to regular niacin*				
• *Sustained-release niacin (not the same as extended-release product): Should be avoided*				

FARMAKOTERAPI OBAT SISTEM GIT & HEPATOBILIER

Subclass	Mechanism of Action	Effects	Clinical Applications	Pharmacokinetics, Toxicities, Interactions
ANTIEMETIC DRUGS				
• Ondansetron, other 5-HT_3 antagonists	5-HT_3 blockade in gut and CNS with shorter duration of binding than alosetron	Extremely effective in preventing chemotherapy-induced and postoperative nausea and vomiting	First-line agents in cancer chemotherapy; also useful for postop emesis	Usually given IV but orally active in prophylaxis • 4–9 h duration of action • very low toxicity but may slow colonic transit
• Aprepitant	NK_1-receptor blocker in CNS	Interferes with vomiting reflex • no effect on 5-HT, dopamine, or steroid receptors	Effective in reducing both early and delayed emesis in cancer chemotherapy	Given orally • IV fosaprepitant available • fatigue, dizziness, diarrhea • CYP interactions
• *Corticosteroids: Mechanism not known but useful in antiemetic IV cocktails*				
• *Antimuscarinics (scopolamine): Effective in emesis due to motion sickness; not other types*				
• *Antihistaminics: Moderate efficacy in motion sickness and chemotherapy-induced emesis*				
• *Phenothiazines: Act primarily through block of D_2 and muscarinic receptors*				
• *Cannabinoids: Dronabinol is available for use in chemotherapy-induced nausea and vomiting, but is associated with CNS marijuana effects*				
DRUGS USED IN INFLAMMATORY BOWEL DISEASE (IBD)				
• 5-Aminosalicylates, eg, mesalamine in many formulations • Sulfasalazine	Mechanism uncertain • may be inhibition of eicosanoid inflammatory mediators	Topical therapeutic action • systemic absorption may cause toxicity	Mild to moderately severe Crohn's disease and ulcerative colitis	Sulfasalazine causes sulfonamide toxicity and may cause GI upset, myalgias, arthralgias, myelosuppression • other aminosalicylates much less toxic
• Purine analogs and antimetabolites, eg, 6-mercaptopurine, methotrexate	Mechanism uncertain • may promote apoptosis of immune cells • Methotrexate blocks dihydrofolate reductase	Generalized suppression of immune processes	Moderately severe to severe Crohn's disease and ulcerative colitis	GI upset, mucositis • myelosuppression • purine analogs may cause hepatotoxicity, but rare with methotrexate at the low doses used
• Anti-TNF antibodies, eg, infliximab, others	Bind tumor necrosis factor and prevent it from binding to its receptors	Suppression of several aspects of immune function, especially T_H1 lymphocytes	Infliximab: Moderately severe to severe Crohn's disease and ulcerative colitis • others approved in Crohn's disease	Infusion reactions • reactivation of latent tuberculosis • increased risk of dangerous systemic fungal and bacterial infections
• *Corticosteroids: Generalized anti-inflammatory effect; see Chapter 39*				
DRUGS FOR IRRITABLE BOWEL SYNDROME (IBS)				
• Alosetron	5-HT_3 antagonist of high potency and duration of binding	Reduces smooth muscle activity in gut	Approved for severe diarrhea-predominant IBS in women	Rare but serious constipation • ischemic colitis • infarction
• *Anticholinergics: Nonselective action on gut activity, usually associated with typical antimuscarinic toxicity*				
• *Chloride channel activator: Lubiprostone (see above); useful in constipation-predominant IBS in women*				
PANCREATIC SUPPLEMENTS				
• Pancrelipase	Replacement enzymes from animal pancreatic extracts	Improves digestion of dietary fat, protein, and carbohydrate	Pancreatic insufficiency due to cystic fibrosis, pancreatitis, pancreatectomy	Taken with every meal • may increase incidence of gout
• *Pancreatin: Similar pancreatic extracts but much lower potency; rarely used*				
BILE ACID THERAPY FOR GALLSTONES				
• Ursodiol	Reduces cholesterol secretion into bile	Dissolves gallstones	Gallstones in patients refusing or not eligible for surgery	May cause diarrhea
DRUGS USED TO TREAT VARICEAL HEMORRHAGE				
• Octreotide	Somatostatin analog • mechanism not certain	May alter portal blood flow and variceal pressures	Patients with bleeding varices or at high risk of repeat bleeding	Reduced endocrine and exocrine pancreatic activity • other endocrine abnormalities • GI upset
• *β Blockers: Reduce cardiac output and splanchnic blood flow; see Chapter 10*				

V. TUGAS MAHASISWA

Diskusikan kasus di bawah ini dalam kelompok

a. Jawaban kasus ditulis dalam **format LAPORAN (word)** berupa:
   - Judul ditulis **LAPORAN PRAKTIKUM FARMAKOLOGI** → (enter) judul **BLOK**
   - Format penulisan: Times New Roman, font uk 12, rata kanan kiri, penulisan rapi, warna font hitam, spasi 1,5
   - jawaban **TIDAK *COPY PASTE* ANTAR KELOMPOK**

- Pembuatan Tabel tidak *screen shoot* dari referensi, melainkan membuat sendiri, kecuali gambar
- Jika referensi ddidapat dari jurnal asing, harap menerjemahkan dahulu ke Bahasa indonesia
- Penjelasan lengkap pada word **HARUS MENCANTUMKAN REFERENSI TERBARU DAN LAYAK** di setiap jawaban

b. Menyiapkan presentasi dalam **bentuk ppt**, berupa **jawaban singkat/ringkas** (bukan *copy paste* dari poin a
c. Sumber referensi (jurnal) dari jawaban poin a **diberi identitas** (jurnal tersebut untuk menjawab kasus yang mana)
d. Poin a,b, dan c dimasukkan google drive dan dikirimkan lewat **email farmako fkumm**

I. FARMAKOTERAPI OBAT DIABETES MELLITUS DAN OBAT DISPEPSIA

KASUS 1

Seorang pria, 53 tahun datang dengan keluhan sering kesemutan di kedua kaki. Pasien juga mengeluh cepat haus, cepat lapar, banyak BAK, dan penurunan berat badan (BB sebelumya: 85kg). Keluhan disertai ulu hati terasa tidak nyaman dan kadang mual. Riwayat minum obat metformin. Riwayat merokok (+). Pasien pernah didiagnosis DM 10 tahun yang lalu namun tidak rutin minum obat dan kontrol ke dokter. Pemeriksaan fisik didapatkan TD 145/90 mmHg, RR 22 kali/menit, HR 88 kali/menit, suhu afebris, BB = 72 kg, TB = 170 cm. Tes neurologis didapatkan *gloves and stocking* (+). Pemeriksaan laboratorium didapatkan GDP 130 mg/dL (N<100-125 mg/dL), HbA1c: 8% (N<6%), dan kolesterol total 250 mg/dL (N<200 mg/dL). Satu minggu setelahnya pasien dicek tes toleransi glukosa oral dan didapatkan gula darah postprandial 250 mg/dL (N<200 mg/dL).

Tugas:

1. Dokter menyimpulkan pasien menderita DM tipe II dan merencanakan pemberian OAD untuk pasien.
   a. Golongan OAD mana yang tepat untuk kasus tersebut? Jelaskan dengan membuat TABEL perbandingan masing-masing OAD tersebut termasuk cantumkan jenis obat, dosis, farmakodinamik, kelebihan, dan kekurangan sesuai kasus (jelaskan berdasarkan referensi terbaru dan lampirkan jurnalnya)!
   b. Apakah pasien sudah memerlukan injeksi insulin? Jelaskan alasannya? (sertakan referensi)
   c. Bagaimana tatalaksannya bila terjadi hipoglikemi? Jelaskan dengan membuat ALUR tatalaksana berdasarkan referensi terbaru dan lampirkan jurnalnya!
2. Apakah pasien perlu mendapatkan obat anti dislipidemia? Jika perlu sebutkan obat anti dislipidemia yang tepat diberikan untuk pasien tersebut (termasuk farmakodinamik, dosis, cara pemakaian, indikasi, dan efek samping) (Jelaskan dengan membuat TABEL perbandingan masing-masing obat anti dislipidemia tersebut berdasarkan referensi terbaru dan lampirkan jurnalnya)!
3. Sebagai terapi dislipidemia, Dokter memutuskan untuk memberikan obat golongan statin. Jelaskan efek pleiotropik obat golongan statin! (Sertakan referensi terbaru dan lampirkan jurnalnya).

II. FARMAKOTERAPI OBAT SISTEM HEPATOBILIER DAN ANTI DISLIPIDEMIA

KASUS 2

Seorang wanita, 48 tahun, sering mengalami nyeri perut di kanan atas setelah makan. Nyeri makin sering terjadi ketika ia mengkonsumsi makanan berlemak. Kadang-kadang juga disertai mual dan muntah. Ia sudah mencoba mengobatinya sendiri dengan obat-obat gastritis, namun hanya berkurang sedikit. Pasien juga sering mengkonsumsi makan makanan berlemak, bersantan, dan makanan cepat saji. Pemeriksaan fisik didapatkan TD 165/100 mmHg, RR 22 kali/menit, HR 88 kali/menit, suhu afebris, BB = 85 kg, TB = 167 cm. Pemeriksaan laboratorium didapatkan kolesterol total 350 mg/dL (N<200mg/dL), LDL 168 mg/dL (N<100 mg/dL), HDL 30 mg/dL (N=35-80 mg/dL) dan Trigliserida 162 mg/dL (N<150 mg/dL). Hasil pemeriksaan USG menunjukkan ada beberapa batu kecil di kandung empedunya. Penderita menolak operasi, dan dokter memberikan obat berupa Ursodiol.

Tugas :

1. Dokter memberikan obat dispepsia untuk menurunkan keluhan pasien. Apakah obat dispepsia yang tepat untuk pasien tersebut? Dan bedakan masing-masing dalam bentuk tabel (jelaskan dengan membuat TABEL sertakan berdasarkan referensi terbaru dan lampirkan jurnalnya)
2. Perlukah penderita diberikan obat antivomiting? Jelaskan alasannya!
3. Jelaskan penggolongan obat antivomiting dan bedakan mekanisme kerja, indikasi, KI, efek samping serta bagaimana penggunaannya! (jelaskan dengan membuat TABEL sertakan berdasarkan referensi terbaru dan lampirkan jurnalnya)
4. Apa tujuan dokter memberikan Ursodiol dan bagaimana cara penggunaannya? (jelaskan berdasarkan referensi terbaru dan lampirkan jurnalnya)

VI. RUBRIK PENILAIAN

Penilaian Pre-Test

PENILAIAN PRE-TEST PRAKTIKUM					
			RUBRIK PENILAIAN		
NO	MATERI	BOBOT	0	1	JUMLAH
			tidak menjawab atau jawaban salah	Jawaban benar	
	NILAI				(jumlah jawaban benar : jumlah soal) x 100%

Penilaian Ujian Praktikum

PENILAIAN UJIAN PRAKTIKUM FARMAKOLOGI BLOK NMS 1					
			RUBRIK PENILAIAN		JUMLAH
NO	MATERI	BOBOT	0	1	

	TOTAL	10			(jumlah jawaban benar : jumlah soal) x 100%

Penilaian Laporan Dan Diskusi

		60	70	80
1	Kebenaran dan kelengkapan jawaban			
2	Jawaban berdasarkan Referensi yang benar			
3	Keaktifan kelompok dalam diskusi			

Nilai Akhir Praktikum Farmakologi Blok Neoplasma = 10%
PRETEST + 20% LAPORAN + 70% MCQ

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