Ministry of Science and Higher Education of the RussianFederation
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Interstate Educational Organization of Higher Education Kyrgyz-Russian Slavic University named after the first President of the Russian Federation B.N. Yeltsin

FUND OF ASSESSMENT TOOLS (FAT) for the Discipline "ENDOCRINOLOGY"

Level of Higher Education: SPECIALIST

Field of Study:
Code: 31.05.01 – RF, 560001 – KR
Name: General Medicine (for international students) Qualification: Physician
Total workload: 2 credit units (60 hours)
Course, semesters: 3th year, 6th semester
Year of commencement of training: 2026
Duration of education: 6 years

The Fund of Assessment Tools is designed to control students' knowledge in the field of study (specialty) PHYSICIAN in the discipline " **ENDOCRINOLOGY** "

The Fund of Assessment Tools was reviewed and approved at the meeting of the department of **Family medicine & Therapy**

Protocol No. 1 dated 29.08.2026

Head of Department

Family medicine & Therapy _  _Suranova G.Zh

Executors

Candidate of Medical Sciences, Associate Professor _  _SuranovaG.Zh.

1. COMPETENCIES FORMED THROUGH THE DISCIPLINE

OPK-5: Ability to assess morphofunctional, physiological states and pathological processes

Capable of assessing morphofunctional, physiological states and pathological processes in the human body to solve professional tasks.

Level	Know	Be Able To	Master	Assessment Tools
Level 1	Main morphofunctional and physiological states of the human body; mechanisms of endocrine gland function in health	Explain the essence of main morphofunctional, physiological states in the context of endocrine pathology	Skills in determining normal morphofunctional states and recognising deviations in endocrine diseases	Block A: Tests on physiology and pathophysiology of endocrine system; oral questioning
Level 2	Pathological processes in the human body in endocrine diseases (diabetic, thyrotoxic, adrenal, pituitary pathology); pathophysiology of endocrine syndromes	Reveal the essence of main pathological processes; relate morphofunctional changes to clinical manifestations of endocrine diseases	Skills in identifying main pathological processes in endocrine organs and their systemic effects	Block B: Situational cases — pathophysiological analysis of endocrine syndromes
Level 3	Morphological changes in endocrine organs in main diseases; pathophysiological mechanisms of complications	Apply knowledge of morphofunctional changes to clinical decision-making in endocrine patients	Skills in comprehensive pathophysiological analysis of endocrine diseases for diagnosis formulation	Block C: Practice tasks; Block D: Certification — complex pathophysiological cases

OPK-7: Ability to prescribe treatment and monitor its efficacy and safety

Capable of prescribing treatment and monitoring its effectiveness and safety in patients with endocrine diseases.

Level	Know	Be Able To	Master	Assessment Tools
Level 1	Aetiology, pathogenesis, clinical picture of main endocrine diseases; methods of patient management in outpatient and day-hospital settings	Compare different types and methods of treatment for patients with endocrine nosological forms; develop a treatment plan	Skills in analysing various types of treatment in patients with endocrine diseases (DM, thyroid, adrenal, pituitary pathology)	Block A: Tests on treatment principles; oral questioning on drug groups
Level 2	Pharmacological groups used in endocrinology (insulin types, OHAs, thionamides, glucocorticoids, SSAs, dopamine agonists); their interactions and contraindications	Select appropriate treatment based on disease stage; monitor efficacy (HbA1c, TFTs, cortisol) and safety (glucose, CBC, liver function)	Skills in treatment monitoring: target attainment, side effect recognition, therapy adjustment	Block B: Situational cases on treatment selection and monitoring

Level 3	Modern principles of treatment of endocrine diseases within nosological forms; criteria for treatment efficacy and safety	Prescribe drug and non-drug therapy; assess treatment outcomes; adjust therapy based on monitoring results	Skills in prescribing, monitoring and adjusting therapy for all major endocrine diseases	Block C: Prescription writing; Block D: Complex therapy planning cases

PC-4: Readiness to collect and analyse patient complaints, history, examination and investigation results

Ready to collect and analyse patient complaints, medical history, examination findings, laboratory, instrumental and other investigation results for diagnosis recognition.

Level	Know	Be Able To	Master	Assessment Tools
Level 1	Methods and means of collecting and analysing patient complaints and medical history in endocrinology; indications and contraindications for additional investigations	Collect and analyse patient complaints and medical history in patients with endocrine diseases	Skills in collecting and analysing patient complaints and medical history; interpreting common functional diagnostic methods in endocrine pathology	Block A: Tests on history-taking and examination; oral questioning
Level 2	Laboratory investigations in endocrinology: hormone assays (TSH, Free T4/T3, cortisol, IGF-1, GH, prolactin, sex hormones, PTH), glucose metabolism tests (HbA1c, OGTT), autoantibodies	Interpret results of laboratory and instrumental investigations in endocrine diseases; formulate a diagnostic algorithm	Skills in interpreting laboratory data (hormonal panels, glucose metabolism tests) and instrumental findings (thyroid ultrasound, MRI pituitary, DXA)	Block B: Lab interpretation tasks; situational cases with investigation results
Level 3	Comprehensive diagnostic evaluation in endocrinology; differential diagnostic criteria for endocrine syndromes	Integrate all clinical, laboratory and instrumental data into a unified diagnostic picture; formulate clinical diagnosis	Skills in complex clinical analysis — collecting, systematising and interpreting all diagnostic information for final diagnosis	Block C: Complex clinical assessment; Block D: Certification — full diagnostic cases

PC-5: Ability to identify pathological conditions, symptoms, syndromes and nosological forms according to ICD-10

Capable of identifying main pathological conditions, symptoms, syndromes of diseases, nosological forms in patients in accordance with ICD-10.

Level	Know	Be Able To	Master	Assessment Tools
Level 1	Main types and methods of treatment of patients with endocrine nosological forms; ICD-10 classification of endocrine diseases (Chapter IV, E00–E90)	Collect and analyse patient complaints and medical history; identify main symptoms and syndromes in endocrine diseases	Skills in collecting and analysing patient complaints; interpreting common diagnostic methods for endocrine pathology	Block A: Tests on ICD-10 classification; syndrome identification tasks
Level 2	Differential diagnostic criteria for main endocrine syndromes; classification of DM, thyroid, adrenal and pituitary diseases	Identify and formulate clinical and syndromic diagnosis; apply ICD-10 codes to endocrine conditions	Skills in interpreting functional diagnostic results; skills in formulating diagnosis with correct ICD-10 coding	Block B: Situational cases — diagnosis formulation with ICD-10; differential diagnostic tasks
Level 3	Complex endocrine nosological forms; overlapping syndromes; complications classification under ICD-10	Formulate a complete structured clinical diagnosis including nosological form, severity, stage, complications with ICD-10 code	Skills in differential diagnosis of complex endocrine presentations; complete clinical diagnosis with ICD-10	Block C: Complex diagnostic cases; Block D: Certification — full diagnosis formulation

PC-7: Ability to determine management tactics for patients with various nosological forms

Capable of determining management tactics for patients with endocrine nosological forms.

Level	Know	Be Able To	Master	Assessment Tools
Level 1	Aetiology, pathogenesis, clinical presentation of main endocrine diseases; principles of outpatient and inpatient management	Reveal the meaning of management tactics for patients with endocrine diseases; identify the appropriate management setting (outpatient vs inpatient vs emergency)	Skills in presenting and analysing aetiology, pathogenesis of endocrine diseases to determine management approach	Block A: Tests on management principles; oral questioning
Level 2	Management algorithms for main endocrine diseases; indications for hospitalisation; monitoring parameters and target values	Determine management tactics depending on disease severity, complications, patient age; plan follow-up	Skills in management algorithm application; dispensary observation planning for endocrine patients	Block B: Situational cases on management planning

Level 3	Complex management in endocrine diseases with complications; multidisciplinary approach	Plan comprehensive management including drug therapy, lifestyle modification, patient education, referral, dispensary observation	Skills in complex patient management; skills in patient education planning (diabetes school, thyroid disease management)	Block C: Complex management cases; Block D: Full management plan scenarios

PC-8: Readiness to manage and treat patients in outpatient and day-hospital settings

Ready to manage and treat patients with endocrine nosological forms in outpatient and day-hospital settings.

Level	Know	Be Able To	Master	Assessment Tools
Level 1	Aetiology, pathogenesis, clinical presentation of main endocrine diseases with various nosological forms; outpatient management principles	Correctly identify endocrine disease; apply outpatient management protocol	Skills in analysing various types of treatment for patients with different endocrine nosological forms	Block A: Tests on outpatient management; oral questioning on drug prescribing
Level 2	Drug prescribing in endocrinology: doses, routes, monitoring parameters; contraindications; outpatient follow-up schedules	Prescribe appropriate treatment for stable endocrine conditions; adjust doses based on monitoring; plan outpatient follow-up	Skills in prescription writing for endocrine drugs; outpatient monitoring protocol implementation	Block B: Prescription writing tasks; outpatient management cases
Level 3	Complex outpatient management in endocrinology; patient self-management and education principles	Manage complex endocrine patients in outpatient setting; coordinate with specialists (ophthalmologist, nephrologist, cardiologist, neurologist)	Skills in comprehensive outpatient management; patient education skills (diabetes school, thyroid patient counselling)	Block C: Outpatient management scenarios; Block D: Certification

PC-9: Readiness to provide primary healthcare for acute and chronic endocrine conditions

Ready to provide primary health care for sudden acute diseases, exacerbations of chronic endocrine conditions not accompanied by threat to life.

Level	Know	Be Able To	Master	Assessment Tools
Level 1	Clinical manifestations of acute and chronic endocrine diseases; early warning signs of decompensation	Link symptoms, examination data and investigation results into a unified clinical picture; make correct diagnosis	Skills in identifying signs of an acute endocrine disease or exacerbation of a chronic condition	Block A: Tests on acute presentations; oral questioning on emergency signs

Level 2	Diagnostic criteria for endocrine crises (DKA, HHS, hypoglycaemic coma, thyrotoxic crisis, adrenal crisis, hypercalcaemic crisis); first-line management	Rapidly assess severity of acute endocrine condition; initiate appropriate first-line management; decide on hospitalisation need	Skills in rapid clinical assessment of endocrine emergency; first-line management skills	Block B: Emergency assessment and management cases
Level 3	Full emergency management protocols for major endocrine crises; criteria for emergency hospitalisation vs ambulatory management	Provide complete primary care including emergency stabilisation, documentation, referral for acute endocrine conditions	Skills in complete emergency management of acute endocrine conditions; resuscitation principles in endocrine emergencies	Block C: Emergency management scenarios; Block D: Certification — acute endocrine crises

PC-11: Readiness to participate in emergency medical care for conditions requiring urgent intervention

Ready to participate in emergency medical care for conditions requiring urgent medical intervention in endocrine diseases.

Level	Know	Be Able To	Master	Assessment Tools
Level 1	Algorithm of emergency medical care in endocrinology; basic diagnostic and therapeutic measures for first aid in endocrine emergencies (DKA, HHS, hypoglycaemic coma, adrenal crisis, thyrotoxic crisis)	Select individual type of care based on clinical situation: first aid, ambulance, hospitalisation	Set of resuscitation measures for acute disorders in endocrine emergencies; modern methods of resuscitation and intensive care	Block A: Tests on emergency algorithms; oral questioning
Level 2	Emergency protocols for each major endocrine crisis; drug doses and routes in emergency setting; monitoring parameters during resuscitation	Initiate emergency treatment protocol for specific endocrine crisis; communicate with emergency services; stabilise patient before transfer	Skills in emergency drug administration (IV insulin, IV glucose, IV hydrocortisone, IV fluids) in endocrine crises	Block B: Emergency scenario simulation cases
Level 3	Comprehensive emergency management including post-crisis stabilisation; prevention of recurring crises; transition from	Provide complete emergency management from first aid through resuscitation to stabilisation and referral	Skills in full resuscitation sequence in endocrine emergencies; documentation of emergency care	Block C: Complex emergency management; Block D: Full emergency scenario certification

	emergency to routine care			

PC-14: Ability to maintain medical documentation

Capable of maintaining medical documentation in endocrinology practice.

Level	Know	Be Able To	Master	Assessment Tools
Level 1	List and characteristics of accounting and reporting medical documentation in endocrinology organisations; types of medical records	Conduct medical and statistical analysis of health indicators; fill out medical history, outpatient card, discharge epicrisis	Work skills and methods for accounting and reporting documentation in endocrinology medical institutions	Block A: Tests on medical documentation; practical documentation tasks
Level 2	Structure of endocrinological medical records; prescription writing rules for endocrine drugs (insulin, thionamides, glucocorticoids); referral forms	Write prescriptions for endocrinological drugs; complete referral forms and endocrinologist conclusions; maintain dispensary records	Skills in completing all types of endocrinological documentation: medical history, prescriptions, referrals, dispensary observation cards	Block B: Documentation tasks; prescription writing exercises
Level 3	Regulatory requirements for medical documentation in endocrinology; electronic health records; reporting requirements	Maintain complete documentation for complex endocrine patients including all required forms; ensure regulatory compliance	Skills in complete medical documentation for all common endocrine conditions; proficiency in electronic documentation	Block C: Case history writing and defense; Block D: Certification — documentation assessment

2. LEARNING OUTCOMES OF THE DISCIPLINE

2.1. KNOW:

- Main morphofunctional, physiological states and pathological processes in the human body in the context of endocrine diseases
- Aetiology, pathogenesis, clinical picture of main endocrine diseases with various nosological forms; methods of patient management in outpatient and day-hospital settings
- Methods and means of collecting and analysing patient complaints, medical history; indications and contraindications for additional clinical and paraclinical investigations
- Main types and methods of treatment for patients with endocrine nosological forms
- Aetiology, pathogenesis, clinical presentation of main endocrine diseases
- Clinical manifestations of acute and chronic endocrine diseases
- Algorithm of emergency medical care; basic diagnostic and therapeutic measures for first aid in endocrine emergencies
- List and characteristics of accounting and reporting medical documentation in endocrinology organisations

2.2. BE ABLE TO:

- Explain the essence of main morphofunctional, physiological states and pathological processes in endocrine diseases
- Compare different types and methods of treatment; develop a treatment plan for patients with endocrine diseases
- Collect and analyse patient complaints and medical history in endocrine pathology
- Reveal the meaning of determining management tactics for patients with various endocrine diseases
- Correctly identify endocrine disease; apply appropriate management protocol
- Link symptoms, examination data and investigation results into a unified clinical picture; make correct diagnosis
- Select individual type of care based on clinical situation: first aid, ambulance, hospitalisation
- Conduct medical and statistical analysis of health indicators; maintain medical documentation

2.3. MASTER:

- Skills in determining main morphofunctional, physiological states and pathological processes in endocrine diseases
- Skills in analysing various types of treatment for patients with endocrine nosological forms
- Skills in collecting and analysing patient complaints and history; interpreting results of common functional diagnostic methods in endocrine pathology
- Skills in presenting and analysing aetiology, pathogenesis of endocrine diseases to make a diagnosis
- Skills in identifying signs of acute endocrine disease or exacerbation of chronic condition
- Set of resuscitation measures for acute disorders; knowledge of modern methods of resuscitation in endocrine emergencies
- Work skills and methods for accounting and reporting documentation in medical institutions

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3. STRUCTURE OF ASSESSMENT TOOL BLOCKS

Block	Content	Competencies	Semester
Block A	MCQ tests on pathophysiology, clinical manifestations, classification, ICD-10, drug groups, documentation; oral questioning on history-taking and examination	OPK-5 (L1), OPK-7 (L1), PC-4 (L1), PC-5 (L1), PC-7 (L1), PC-8 (L1), PC-9 (L1), PC-11 (L1), PC-14 (L1)	6
Block B	Situational clinical cases: diagnosis formulation, treatment planning, emergency management, lab interpretation, documentation tasks; patient supervision reports	OPK-5 (L1–L2), OPK-7 (L1–L2), PC-4 (L1–L2), PC-5 (L1–L2), PC-7 (L1–L2), PC-8 (L1–L2), PC-9 (L1–L2), PC-11 (L1–L2), PC-14 (L1–L2)	6
Block C	Practice-oriented tasks: patient examination, clinical diagnosis with ICD-10, prescription writing, case history writing and defense; emergency scenario simulation	All competencies L2–L3 (OPK-5, OPK-7, PC-4, PC-5, PC-7, PC-8, PC-9, PC-11, PC-14)	6
Block D	Certification questions: comprehensive clinical analysis, differential diagnosis, emergency management, drug prescribing, case history defense, practical skills	All competencies, all levels (OPK-5, OPK-7, PC-4, PC-5, PC-7, PC-8, PC-9, PC-11, PC-14)	6 (Credit+Grade)

4. DISTRIBUTION BY SEMESTER

Semester	Control Type	Blocks Used	Competencies
6 (3.2)	Credit with Grade	Blocks A, B, C, D	OPK-5, OPK-7, PC-4, PC-5, PC-7, PC-8, PC-9, PC-11, PC-14 — all levels

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5. TECHNOLOGY MAP OF THE DISCIPLINE

Semester 6 (3.2) — 17 weeks | Credit with Grade

Module	Topic	Control Type	Form of Control	Min	Max	Week
M1	BC-1: Introduction to Endocrinology. DM type 1 and type 2: classification, aetiology, pathogenesis, clinic, diagnosis	Current	Frontal questioning, testing, clinical case analysis, attendance	2	4	3
M1		Midterm	Oral/written questioning, situational case, patient supervision	6	10	
M2	BC-2: Treatment of DM. Insulin therapy. OHAs. Self-monitoring. Bread unit calculation. Patient education	Current	Frontal questioning, testing, prescription writing, attendance	2	4	5
M2		Midterm	Oral/written questioning, prescription writing task	6	10	
M3	BC-3: Complications of DM. Diabetic retinopathy, nephropathy, neuropathy, foot. Macro-vascular complications	Current	Frontal questioning, testing, practical skills, attendance	2	4	8
M3		Midterm BC-1 (Boundary Control No. 1)	Oral/written questioning, situational case, lab interpretation	6	10	
M4	BC-4: Acute complications of DM. DKA. Hypoglycaemic coma. HHS. Lactic acidosis. Emergency management algorithms	Current	Frontal questioning, testing, emergency simulation, attendance	2	4	10
M4		Midterm	Oral/written questioning, emergency case	6	10	

M5	BC-5: Thyroid diseases. Thyrotoxicosis syndrome. Hypothyroidism syndrome. Goitre. Thyroiditis. Iodine deficiency diseases	Current	Frontal questioning, testing, practical skills, attendance	2	4	12
M5		Midterm	Oral/written questioning, situational case	6	10	
M6	BC-6: Adrenal gland diseases. Hypocorticism. Addison's disease. Acute adrenal insufficiency. Hypercorticism. Cushing syndrome	Current	Frontal questioning, testing, lab interpretation, attendance	2	4	14
M6		Midterm BC-2 (Boundary Control No. 2)	Oral/written questioning, differential diagnosis case	6	10	
M7	BC-7: Hypothalamic-pituitary diseases. Acromegaly. Diabetes insipidus. Hypopituitarism. Hyperprolactinaemia	Current	Frontal questioning, testing, MRI/hormone panel interpretation, attendance	2	4	15
M7		Midterm	Oral/written questioning, situational case	6	10	
M8	BC-8: Obesity. Metabolic syndrome. PCOS. Dispensary observation. Final test. Case history defense	Current	Frontal questioning, testing, case history defense, attendance	2	4	17
M8		Midterm (Final — Credit with Grade)	Oral/written questioning, case history defense, prescription writing, practical skills	6	10	
TOTAL				**40**	**70**	
Midterm Control	Credit with Grade			**20**	**30**	
Semester Rating				**60**	**100**	

6. PATIENT SUPERVISION AND CASE HISTORY

6.1. Supervision Scheme (Curation Scheme)

Each student receives one patient with endocrine pathology for supervision. The student conducts an interview and physical examination in accordance with the scheme below, reviews laboratory and instrumental results, and fills in the medical history.

- Passport details: full name, age, sex, date of admission, ward, case number
- Complaints: primarily those related to endocrine disease causing hospitalisation, then other complaints
- Disease history: onset, course, past treatment and investigations, reasons for hospitalisation
- Life history (brief): past diseases, heredity (family history of endocrine disease), social and occupational history, lifestyle
- Objective examination data: general examination, vital signs, endocrine system clinical assessment (thyroid palpation, BMI, body habitus, skin changes, oedema, signs of hormonal excess/deficiency)
- Laboratory and instrumental data: hormone panels, glucose metabolism tests, imaging results — interpretation
- Clinical diagnosis with ICD-10 code and justification
- Treatment plan with drug prescriptions

6.2. Structure of Educational Case History

Section	Content	Requirements	Points
Title Page	Full name, age, sex, diagnosis, date of admission, ward	Correct filling of all requisites	5
Complaints	Main endocrine complaints; secondary complaints	Complete sequential presentation; onset and duration clarified	10
Disease History	Development of endocrine disease from onset to admission	Chronological sequence; link to risk factors and triggers	15
Life History	Past diseases, heredity, social history, lifestyle	Risk factor assessment; family endocrine history	10
Objective Status	Full physical examination including endocrine system assessment	Systematic examination: general, endocrine, by organ systems	20
Clinical Diagnosis	Structured diagnosis with complications; ICD-10 code	Logical justification; nosological form + severity + complications + ICD code	20
Examination Plan	Laboratory and instrumental methods with justification	Correspondence to diagnosis; cost-effective approach	10
Treatment Plan	Drug and non-drug therapy; correct doses; monitoring plan	Evidence-based; appropriate to disease stage; drug prescriptions correctly written	10

6.3. Defense Criteria

- Completeness and accuracy of history collection (25%)
- Quality and correctness of physical examination findings (25%)
- Correctness of clinical diagnosis formulation with ICD-10 code (25%)
- Justification of examination and treatment plan with correct prescription writing (25%)

7. STUDENT'S INDEPENDENT WORK

Activity	Time/week	Notes
Study of lecture notes on the day of the lecture	10–15 min	Immediate repetition after lecture
Repetition of notes before the next lecture	10–15 min	Active reproduction of main provisions
Study of theoretical material from textbooks	1 hour	Main and additional literature
Preparation for practical classes and patient supervision	2 hours	Key concepts; case preparation
TOTAL	**3 h 30 min**	**Regular daily work**

Topics for Independent Work:

- 1. Self-control and training in type 1 and type 2 diabetes. Calculation of insulin therapy. Bread unit calculation.
- 2. Diabetes mellitus and pregnancy: features of management and risks for mother and fetus.
- 3. Modern technologies in DM treatment: insulin pumps, CGM systems, closed-loop systems.
- 4. Diabetic neuropathy. Diabetic foot syndrome: classification and prevention.
- 5. Psychological aspects of DM: compliance, patient burnout, motivational counselling.
- 6. Thyroid diseases: thyroiditis — Hashimoto, subacute, postpartum.
- 7. Hyperparathyroidism as part of endocrine syndromes.
- 8. Hypoparathyroidism: diagnosis and treatment.
- 9. Diseases of the hypothalamic-pituitary region: growth disorders, somatotropic insufficiency.
- 10. Hyperprolactinaemia: differential diagnosis and treatment.
- 11. Obesity and metabolic syndrome: role of insulin resistance, complications, treatment.
- 12. Polycystic ovary syndrome (PCOS): pathogenesis, clinical features, modern treatment.

8. TYPICAL ASSESSMENT TASKS WITH ANSWERS

All clinical cases require: (1) clinical diagnosis with ICD-10 code, (2) structured examination plan, (3) treatment plan with drug prescriptions. Competencies assessed in each section are indicated.

CONTROL SECTION No. 1

Section 1: DM Classification, Aetiology, Pathogenesis, Diagnosis

Competencies: OPK-5 (L1), PC-4 (L1–L2), PC-5 (L1), PC-14 (L1)

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

1. What is the WHO 2020 classification of diabetes mellitus?
2. Describe the aetiology and pathogenesis of type 1 DM.
3. Describe the aetiology and pathogenesis of type 2 DM.
4. What are the diagnostic criteria for DM (fasting glucose, OGTT, HbA1c)?
5. What is MODY and how does it differ from type 1 and type 2 DM?
6. What are the criteria for pre-diabetes (IGT, IFG)?

MCQ Tests:

Q1. Male, 17 years, 3-week history of polyuria, polydipsia, weight loss 8 kg, fatigue. No family history of DM. BMI 18 kg/m². Glucose random 22 mmol/L. Anti-GAD antibodies positive. C-peptide 0.06 nmol/L (norm 0.27–1.27). Which diagnosis and ICD-10 code?

A) Type 2 DM — E11
B) Type 1 DM, autoimmune — E10
C) MODY — E13
D) Secondary diabetes — E13
E) Gestational diabetes — O24

CORRECT ANSWER: B) Type 1 DM, autoimmune — E10
Young patient + acute onset + weight loss + absolute insulin deficiency (C-peptide 0.06, undetectable) + autoimmune marker (anti-GAD positive) + no obesity = Type 1 DM, autoimmune. Anti-GAD antibodies confirm immune-mediated beta-cell destruction. ICD-10: E10 (Insulin-dependent DM). Competencies: PC-5 (L1) — ICD-10 identification; PC-4 (L2) — history and lab integration.

Q2. OGTT result: fasting glucose 5.7 mmol/L, 2-hour post-load glucose 9.2 mmol/L. HbA1c 5.8%. Which diagnosis?

A) Normal glucose tolerance
B) Impaired fasting glucose (IFG)
C) Impaired glucose tolerance (IGT) — pre-diabetes
D) Type 2 DM
E) Type 1 DM, latent (LADA)

CORRECT ANSWER: C) Impaired glucose tolerance (IGT) — pre-diabetes
OGTT 2-h glucose 7.8–11.0 mmol/L = impaired glucose tolerance (pre-diabetes). Fasting glucose 5.7 is borderline (IFG = 6.1–6.9 by WHO, 5.6–6.9 by ADA). HbA1c 5.8% (5.7–6.4 = pre-diabetes by ADA). IGT represents high-risk state: ~5–10% annual conversion rate to DM2. Lifestyle intervention reduces progression by 58% (DPP trial).

Q3. Patient on prednisolone 40 mg/day for 3 months (autoimmune hepatitis). Fasting glucose 8.2 mmol/L, HbA1c 7.6%. Which most appropriate ICD-10 code?

A) E10 — Type 1 DM
B) E11 — Type 2 DM
C) E09 — Drug or chemical induced diabetes mellitus
D) E13 — Other specified DM

E) E08 — DM due to underlying condition

CORRECT ANSWER: C) E09 — Drug or chemical induced diabetes mellitus
Glucocorticoid-induced DM = E09 (Drug or chemical induced DM). Glucocorticoids cause insulin resistance + relative insulin deficiency by increasing hepatic gluconeogenesis, reducing glucose uptake in muscle, directly impairing beta-cell secretion. Pattern: predominantly post-prandial hyperglycaemia. Management: insulin (preferred), OHAs less effective for steroid-induced DM.

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient A., 24 years, admitted with 4-week history of polyuria (5–7 L/day), polydipsia, weight loss 9 kg, fatigue, nausea. BMI 17.8 kg/m². BP 110/70 mmHg. Skin turgor reduced. Breathing slightly deep. Glucose 19.4 mmol/L, HbA1c 11.2%, C-peptide 0.05 nmol/L, anti-GAD positive, anti-IA-2 positive. Urinalysis: glucose 4+, ketones 2+.

Questions:

7. Formulate the clinical diagnosis with ICD-10 code and justify. (5 points)
8. Which additional examinations are required? (5 points)
9. Compose a treatment plan with drug prescriptions. (10 points)

ANSWERS:

1. Clinical Diagnosis: Type 1 Diabetes Mellitus (autoimmune), newly diagnosed, severe, decompensated. Complication: Diabetic ketosis (ketonuria 2+, not yet full DKA — pH not stated but assess). ICD-10: E10.1 (Type 1 DM with ketoacidosis, if pH < 7.30) or E10.9 (if no ketoacidosis). Justification: young patient + acute onset + weight loss + absolute insulin deficiency (C-peptide 0.05 nmol/L, undetectable) + autoimmune markers (anti-GAD+, anti-IA-2+) + severe hyperglycaemia (19.4 mmol/L) + HbA1c 11.2% (chronic). Ketonuria 2+ + deep breathing → rule out DKA: check pH, HCO3, blood ketones urgently. Competencies: PC-5 (L1–L2), PC-4 (L1–L2).

2. Additional examinations: URGENT — arterial blood gas (pH, pCO2, HCO3 — rule out ketoacidosis); blood ketones (beta-hydroxybutyrate); electrolytes (Na+, K+, Cl−); renal function (urea, creatinine). ROUTINE — lipid profile; thyroid function (TSH — autoimmune association); coeliac antibodies (anti-tTG — autoimmune cluster); urine albumin/creatinine ratio (nephropathy baseline); ophthalmological assessment (retinopathy baseline). Competencies: PC-4 (L2), OPK-5 (L2).

3. Treatment Plan: (1) IF KETOSIS ONLY (no acidosis): IV 0.9% NaCl 500 mL/h × 2 h, then oral rehydration. (2) INSULIN THERAPY — basal-bolus regimen: • Basal: glargine (Lantus) 0.2 U/kg/day at 22:00 (e.g., 3.5 U) • Bolus: aspart (NovoRapid) before each meal by carbohydrate counting: 1 U per 10 g carbohydrates • Correction dose: 1 U per 3 mmol/L above 8 mmol/L target. (3) TARGETS: Fasting glucose 4–7 mmol/L; post-meal < 10 mmol/L; HbA1c < 7.0%. (4) DIET: Carbohydrate counting using bread units (1 BU = 10–12 g carbohydrates); regular meals 3 main + 2–3 snacks; avoid concentrated sweets. (5) SELF-MONITORING: Blood glucose 4–6 times/day (before meals and 2 hours post-meal); glucose diary. (6) PATIENT EDUCATION: Diabetes school programme — hypoglycaemia recognition and treatment; sick-day rules; injection technique; target glucose levels. (7) KETOSIS MANAGEMENT: Monitor ketones until clear; ensure adequate insulin dose. Prescription example: Insulin glargine (Lantus) 100 U/mL solution — inject 3.5 U subcutaneously at 22:00 daily. Competencies: OPK-7 (L2–L3), PC-8 (L1–L2), PC-14 (L1–L2).

CONTROL SECTION No. 2

Section 2: Treatment of DM. Insulin Therapy. OHAs. Patient Education

Competencies: OPK-7 (L1–L2), PC-7 (L1–L2), PC-8 (L1–L2), PC-14 (L1–L2)

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

10. Classify oral hypoglycaemic agents (OHAs) by mechanism of action.
11. What are the principles of insulin therapy in type 1 DM? Types of insulin by duration.
12. What are the indications for insulin therapy in type 2 DM?
13. What is a bread unit (BU) and how is the meal bolus insulin dose calculated?
14. What are the principles of SGLT-2 inhibitors — mechanism, benefits, contraindications?
15. What is the self-monitoring algorithm for DM patients?

MCQ Tests:

Q1. Patient with Type 2 DM, BMI 33 kg/m², eGFR 72 mL/min, HbA1c 8.8% on metformin 2000 mg/day maximum dose. No cardiovascular disease. Which is the preferred add-on agent?

A) Glibenclamide (sulphonylurea)
B) Dapagliflozin (SGLT-2 inhibitor)
C) Pioglitazone (thiazolidinedione)
D) Acarbose (alpha-glucosidase inhibitor)
E) Add basal insulin immediately

CORRECT ANSWER: B) Dapagliflozin (SGLT-2 inhibitor)
SGLT-2 inhibitors are preferred add-on to metformin in ADA/EASD 2022 guidelines when: (1) eGFR ≥ 45 (72 — suitable); (2) no established CVD (SGLT-2 preferred for CVD too, but GLP-1 agonist is alternative); (3) BMI 33 — weight reduction benefit; (4) HbA1c reduction 0.5–1.0%. Additional benefits: BP reduction, renal protection (DAPA-CKD). Sulphonylurea: weight gain + hypoglycaemia risk — second choice. Insulin: premature at this stage.

Q2. Patient eats lunch with 90 g carbohydrates. Carbohydrate-to-insulin ratio = 1:10. Pre-lunch glucose 11.5 mmol/L. Correction factor: 1 U reduces glucose by 3 mmol/L. Target glucose 6 mmol/L. Total lunch insulin dose?

A) 9 units
B) 11 units
C) 7 units
D) 13 units
E) 15 units

CORRECT ANSWER: B) 11 units
Calculation: (1) Meal dose: 90 g ÷ 10 = 9 units. (2) Correction dose: (current 11.5 − target 6.0) ÷ 3 = 5.5 ÷ 3 = 1.8 ≈ 2 units. (3) Total = 9 + 2 = 11 units. This is standard carbohydrate counting + correction dose calculation. Competency: PC-8 (L2) — prescribing insulin dosing; PC-14 (L2) — prescription documentation.

Q3. Type 2 DM patient develops trembling, sweating, palpitations, hunger. Glucose 2.4 mmol/L. Patient is conscious and cooperative. First action?

A) IV 40% glucose 40 mL
B) Glucagon 1 mg IM
C) 15–20 g fast-acting oral carbohydrates (rule of 15)
D) IV 5% glucose infusion

E) Call ambulance

CORRECT ANSWER: C) 15–20 g fast-acting oral carbohydrates (rule of 15)
Mild–moderate hypoglycaemia (glucose < 3.9 mmol/L) + conscious + able to swallow = ORAL carbohydrates. Rule of 15: 15–20 g fast-acting carbs (3–4 glucose tablets, 150 mL fruit juice, 3 tsp sugar). Recheck after 15 min. If still < 4 mmol/L — repeat. IV glucose is for unconscious patients. Glucagon for unconscious/unable to swallow. Competency: PC-9 (L1), PC-11 (L1).

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient K., 58 years, Type 2 DM for 6 years. Current therapy: metformin 2000 mg/day + glibenclamide 10 mg/day. HbA1c 9.6%, fasting glucose 12.4 mmol/L, postprandial 17.2 mmol/L. BMI 31 kg/m², eGFR 58 mL/min/1.73m², microalbuminuria 68 mg/g. BP 148/90 mmHg. One episode of hypoglycaemia last week.

Questions:

16. Assess compensation and identify problems with current therapy. (5 points)
17. Propose therapy intensification with justification. (5 points)
18. Write a complete treatment plan including non-pharmacological measures and monitoring. (10 points)

ANSWERS:

1. Assessment: HbA1c 9.6% — severely decompensated (target ≤ 7.0–7.5%). Fasting 12.4, postprandial 17.2 — significantly above targets. Problems with current therapy: (a) Glibenclamide contraindicated with eGFR < 60 (risk of prolonged hypoglycaemia — confirmed by recent episode); (b) Maximum dose of both drugs without target achievement. Action required: stop glibenclamide, intensify with insulin and/or SGLT-2. Competencies: OPK-7 (L1–L2), PC-7 (L1–L2).

2. Proposed intensification: (a) STOP glibenclamide (eGFR 58 < 60 — contraindicated; hypoglycaemia risk); (b) ADD basal insulin: glargine 0.1–0.2 U/kg/day (e.g., 6–12 U at 22:00), titrate by 2 U every 3 days if fasting glucose > 7 mmol/L; (c) CONTINUE metformin 2000 mg (eGFR 58 — dose reduce to 1000 mg if approaching 45 mL/min); (d) ADD RAAS inhibitor: ACE inhibitor (enalapril 5–10 mg) or ARB — for microalbuminuria + hypertension + DM; target BP < 130/80. Alternative to insulin: if patient declines — switch to DPP-4 inhibitor (dose-adjusted for eGFR 58, e.g., sitagliptin 50 mg/day). Competencies: PC-8 (L2), PC-14 (L2).

3. Treatment plan: PHARMACOLOGICAL: (1) Glargine insulin 8 U SC at 22:00 daily — titrate +2 U every 3 days if fasting glucose > 7 mmol/L; (2) Metformin 1000 mg twice daily with meals; (3) Enalapril 5 mg once daily (microalbuminuria + hypertension). NON-PHARMACOLOGICAL: (1) Diet: low-GI foods; reduce refined carbohydrates; caloric restriction 500 kcal/day deficit; limit sodium to < 5 g/day; (2) Physical activity: 150 min/week moderate aerobic; (3) Weight reduction target: 5–10% body weight. MONITORING: Self-monitoring blood glucose: fasting + post-dinner daily; HbA1c every 3 months until target, then every 6 months; eGFR + urine albumin every 6 months (CKD monitoring); BP at every visit; ophthalmological examination annually; foot examination at every visit. PATIENT EDUCATION: Hypoglycaemia recognition (now on insulin!); sick-day rules; insulin injection technique; glucose diary. Competencies: OPK-7 (L3), PC-7 (L3), PC-8 (L3), PC-14 (L1–L2).

CONTROL SECTION No. 3

Section 3: Complications of DM

Competencies: OPK-5 (L2), PC-4 (L1–L2), PC-5 (L2), PC-7 (L1–L2)

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

19. Classify microvascular complications of DM and their common pathophysiological mechanism.
20. Describe diabetic nephropathy classification and monitoring markers.
21. Describe diabetic retinopathy classification (non-proliferative vs proliferative).
22. Describe diabetic peripheral neuropathy: distal symmetric polyneuropathy — diagnosis and treatment.
23. Describe diabetic foot syndrome: Wagner classification and management principles.
24. What are the macrovascular complications of DM and their prevention?

MCQ Tests:

Q1. Patient with Type 2 DM (10 years). Urinary albumin/creatinine ratio 380 mg/g on two occasions. eGFR 52 mL/min. BP 162/96 mmHg. Not on ACE inhibitor. Which is the most important intervention?

A) Start diuretic only
B) Start ACE inhibitor or ARB + blood pressure control
C) Add SGLT-2 inhibitor only
D) Restrict protein to 0.6 g/kg/day only
E) Refer for dialysis planning

CORRECT ANSWER: B) Start ACE inhibitor or ARB + blood pressure control
Macroalbuminuria (>300 mg/g) + DM = mandatory RAAS blockade with ACE inhibitor or ARB — proven nephroprotection (reduces proteinuria and GFR decline). BP target < 130/80 mmHg in DM with nephropathy. SGLT-2 is additional (DAPA-CKD showed benefit even without DM), but not primary. Protein restriction 0.8 g/kg/day (not 0.6 — too restrictive for most). Dialysis is premature at eGFR 52. Competency: OPK-7 (L1–L2), PC-7 (L1).

Q2. Fundoscopy in Type 1 DM patient (15 years): new vessel formation on the disc (NVD) + pre-retinal haemorrhage. What stage and urgent action?

A) Moderate NPDR — tighten glycaemic control only
B) Severe NPDR — plan laser next 3 months
C) Proliferative DR — urgent panretinal laser photocoagulation or anti-VEGF
D) Early NPDR — annual review sufficient
E) Normal — reassure

CORRECT ANSWER: C) Proliferative DR — urgent panretinal laser photocoagulation or anti-VEGF
New vessel formation (NVD = neovascularisation at disc) + pre-retinal haemorrhage = PROLIFERATIVE diabetic retinopathy (PDR) — highest risk stage. Requires URGENT ophthalmological intervention: panretinal laser photocoagulation (PRP) and/or intravitreal anti-VEGF (ranibizumab, bevacizumab). Without treatment: vitreous haemorrhage and traction retinal detachment risk within months. Competency: PC-7 (L1–L2), OPK-5 (L2).

Q3. Patient with DM, absent ankle reflexes, absent 10-g monofilament sensation plantar surface great toe bilaterally. Feet: callus over metatarsal heads, small superficial ulcer on plantar surface right foot 1st toe, not infected. Wagner grade?

A) Wagner Grade 0 (high-risk foot, no ulcer)

B) Wagner Grade 1 (superficial ulcer, no infection, no ischaemia)
C) Wagner Grade 2 (deep ulcer to tendon/joint)
D) Wagner Grade 3 (deep ulcer + osteomyelitis)
E) Wagner Grade 4 (gangrene localised)

CORRECT ANSWER: B) Wagner Grade 1 (superficial ulcer, no infection, no ischaemia)
Wagner Grade 1: superficial ulcer limited to dermis, no infection, no ischaemia. Grade 0: no ulcer (high-risk foot). Grade 2: deep ulcer to tendon/capsule/bone. Grade 3: deep ulcer + infection/osteomyelitis. Grade 4: localised gangrene. Management of Grade 1: wound debridement, offloading (total contact cast or therapeutic footwear), glycaemic control, wound dressings. Competency: PC-5 (L2), OPK-5 (L2).

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient R., 52 years, Type 2 DM for 14 years, HbA1c 10.2%. Complaints: burning pain and numbness in feet (worse at night), impotence, intermittent episodes of diarrhoea. BMI 30 kg/m². BP 166/98 mmHg. eGFR 41 mL/min. Albuminuria 520 mg/g. Fundoscopy: multiple dot/blot haemorrhages, hard exudates — no neovascularisation. Monofilament: absent bilaterally below ankles. Achilles reflexes: absent.

Questions:

25. Identify all complications and formulate a structured diagnosis with ICD-10 codes. (5 points)
26. Which urgent examinations are needed? (5 points)
27. Compose a comprehensive management plan. (10 points)

ANSWERS:

1. Complications and Diagnosis: Type 2 DM, severely decompensated (HbA1c 10.2%). Complications: (a) Diabetic nephropathy CKD Stage G3b A3 (eGFR 41 + macroalbuminuria 520 mg/g) — ICD-10: E11.65; (b) Distal symmetric sensorimotor polyneuropathy (absent monofilament, absent reflexes, burning pain/numbness) — ICD-10: E11.40; (c) Autonomic neuropathy — GI (diarrhoea) + erectile dysfunction — ICD-10: E11.43; (d) Non-proliferative diabetic retinopathy, moderate–severe (dot/blot haemorrhages, hard exudates, no NV) — ICD-10: E11.31. Competencies: PC-5 (L2), OPK-5 (L2).

2. Urgent examinations: ABI (ankle-brachial index) — peripheral arterial disease (macrovascular); nerve conduction studies (NCS/EMG) — quantify neuropathy degree; ECG + echocardiography (silent MI common in DM neuropathy); complete blood count (anaemia of CKD); potassium (CKD Stage G3b — hyperkalaemia risk with RAAS blockers); ophthalmological fluorescein angiography (more precise retinopathy staging); 24-hour BP monitoring (hypertension pattern in CKD). Competencies: PC-4 (L2), OPK-5 (L2).

3. Management plan: GLYCAEMIC: Intensify with basal insulin (glargine); discontinue metformin (eGFR < 45); avoid SGLT-2 (eGFR < 45); use DPP-4 inhibitor dose-adjusted (sitagliptin 25 mg/day for eGFR 30–45) + insulin. Target HbA1c 7.5–8.0% (less stringent — CKD, complications). RENAL: ACE inhibitor or ARB — mandatory for macroalbuminuria (eGFR 41 — monitor K+ carefully); target BP < 130/80; avoid nephrotoxic drugs (NSAIDs, contrast agents without preparation); low-protein diet 0.8 g/kg/day. NEUROPATHY: Pregabalin 75–150 mg 2× daily or duloxetine 60 mg/day for neuropathic pain; alpha-lipoic acid 600 mg/day IV course; glycaemic control is primary. RETINOPATHY: Urgent ophthalmological consultation; laser photocoagulation if progression to severe NPDR; intravitreal anti-VEGF if macular oedema. ERECTILE DYSFUNCTION: PDE-5 inhibitor (sildenafil 50 mg prn) after cardiovascular exclusion. CARDIOVASCULAR RISK: Statin therapy (all DM + CKD = very high CV risk); aspirin 100 mg only if established CVD. FOOT CARE: Daily inspection; therapeutic footwear; podiatry referral. Competencies: OPK-7 (L3), PC-7 (L3), PC-8 (L3).

CONTROL SECTION No. 4

Section 4: Acute Complications of DM. Emergency Management

Competencies: PC-9 (L1–L3), PC-11 (L1–L3), OPK-7 (L2–L3)

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

28. Describe DKA pathogenesis, diagnostic criteria (mild/moderate/severe), emergency algorithm.
29. Describe hypoglycaemic coma: aetiology, clinical picture, emergency management.
30. Describe hyperosmolar hyperglycaemic state (HHS): diagnostic criteria and treatment.
31. What is lactic acidosis in DM: causes, diagnosis, treatment?
32. What is the differential diagnosis of comatose states in DM?
33. What are the emergency doses of insulin, glucose, and hydrocortisone in endocrine crises?

MCQ Tests:

Q1. Type 1 DM patient. Glucose 28 mmol/L, pH 7.08, HCO3 5 mmol/L, ketonuria 4+, blood ketones 6.2 mmol/L (norm < 0.6), K+ 5.2 mmol/L. Unconscious. Which DKA severity and first drug to administer?

A) Mild DKA — start oral glucose
B) Severe DKA — 0.9% NaCl IV first, then insulin infusion
C) Severe DKA — start insulin immediately without fluids
D) HHS — normal saline 0.45%
E) Lactic acidosis — sodium bicarbonate IV

CORRECT ANSWER: B) Severe DKA — 0.9% NaCl IV first, then insulin infusion
Severe DKA criteria: pH < 7.10 (7.08), HCO3 < 10 (5), unconscious. CRITICAL RULE: FLUIDS BEFORE INSULIN. Give 0.9% NaCl 1000 mL in first hour to expand volume, then start fixed-rate IV insulin infusion 0.1 U/kg/h. Starting insulin without volume replacement → precipitous fluid shift → cardiovascular collapse. K+ 5.2: do NOT add K+ initially — falls during treatment. Add K+ when K+ < 5.0 mmol/L. Competency: PC-11 (L1–L2).

Q2. Paramedic finds unconscious DM patient. No glucometer available. Blood glucose unknown. Which drug is given empirically while awaiting glucometer?

A) Insulin 4 U IV
B) IV 40% glucose 40 mL
C) Glucagon 1 mg IM
D) IV 0.9% NaCl 500 mL
E) Hydrocortisone 100 mg IV

CORRECT ANSWER: B) IV 40% glucose 40 mL
Empirical IV glucose 40% (40 mL = 16 g glucose) is safe in BOTH hypoglycaemia (curative) and hyperglycaemic coma (the extra glucose causes no significant harm at this dose). Glucagon IM is also acceptable if IV access unavailable. This is the prehospital protocol when glucose level is unknown. Insulin would be catastrophic if the patient is already hypoglycaemic. Competency: PC-11 (L1–L2), PC-9 (L2).

Q3. Patient with Type 2 DM, 72 years. Glucose 52 mmol/L, osmolality 348 mOsm/kg, pH 7.34, no ketonuria, creatinine 248 µmol/L. Confused. What is the fluid of choice for initial resuscitation?

A) 0.9% NaCl 1000 mL in first hour
B) 0.45% NaCl 1000 mL in first hour
C) 5% glucose 1000 mL
D) Ringer's lactate 500 mL

E) Colloid (gelatin) 500 mL

CORRECT ANSWER: A) 0.9% NaCl 1000 mL in first hour
HHS (glucose > 33 + osmolality > 320 + no significant acidosis): initial resuscitation = 0.9% NaCl — even though patient is hypertonic, isotonic saline is safer to avoid too-rapid osmolality reduction (risk of cerebral oedema). After 1–2 L of 0.9% NaCl, switch to 0.45% NaCl to reduce osmolality gradually. Target glucose reduction ≤ 3–4 mmol/L/h. Low-dose insulin infusion (0.05 U/kg/h) — start only after adequate volume replacement. Competency: PC-9 (L2), PC-11 (L2).

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient E., 20 years, Type 1 DM. Brought unconscious by parents. History: vomiting for 36 hours, could not eat or take insulin, drank large amounts of water. Examination: Kussmaul breathing, acetone breath, dry mucous membranes, skin turgor severely reduced. BP 85/50 mmHg. Pulse 128 bpm, weak. Glucose 34 mmol/L. Blood gas: pH 6.98, HCO3 4 mmol/L, pCO2 14 mmHg. Na+ 128 mmol/L, K+ 6.1 mmol/L. Ketonuria 4+, blood ketones 8.4 mmol/L.

Questions:

34. Establish diagnosis with severity. Identify life-threatening features. (5 points)
35. Describe the immediate step-by-step emergency algorithm (first 2 hours). (5 points)
36. Compose a complete 24-hour treatment protocol. (10 points)

ANSWERS:

1. Diagnosis: DKA, SEVERE (pH 6.98 < 7.0, HCO3 4 < 10, unconscious). Life-threatening features: (a) pH 6.98 — extreme acidosis (myocardial depression risk, vasodilation, arrhythmia risk); (b) BP 85/50 — hypovolaemic shock (severe dehydration); (c) K+ 6.1 — hyperkalaemia (but will fall dangerously during treatment); (d) Unconscious — airway protection required. Precipitant: insulin omission + vomiting + no sick-day rule follow. ICD-10: E10.1. Competency: PC-9 (L2), PC-11 (L1–L2).

2. Immediate emergency algorithm (first 2 hours): MINUTE 0–5: Airway — position + nasogastric tube (unconscious + vomiting = aspiration risk); call for ICU support. IV ACCESS — two wide-bore cannulae (ante-cubital). BLOOD SAMPLES — blood gas, glucose, electrolytes, ketones, CBC, culture. MONITOR — continuous cardiac monitoring (arrhythmia from K+/pH), SpO2, urine output (catheter). MINUTE 5–60 (HOUR 1): FLUIDS — 0.9% NaCl 1000 mL IV in first hour (shock resuscitation: may need 2000 mL in first 30 min if BP < 90); INSULIN — do NOT start until fluids begun; if K+ < 3.5 — correct K+ FIRST before insulin. HOUR 1–2: INSULIN — fixed-rate IV infusion: 0.1 U/kg/h (= 2 U/h for 20 kg, calculate for actual weight); FLUIDS — continue 0.9% NaCl 500 mL/h. POTASSIUM — K+ 6.1: do NOT add K+ initially; reassess at 1 hour; add when K+ < 5.0 → 20–40 mmol/h (monitor ECG). Competency: PC-11 (L2–L3).

3. 24-hour treatment protocol: FLUIDS: Hour 1: 1000 mL 0.9% NaCl (faster if shock). Hours 2–4: 500 mL/h 0.9% NaCl. Hours 4–8: 250 mL/h 0.9% NaCl. When glucose ≤ 14 mmol/L: switch to 10% glucose + 0.45% NaCl (to provide glucose for continued insulin infusion without hypoglycaemia). Total fluid target: 4–6 L in 24 h. INSULIN: 0.1 U/kg/h fixed-rate IV (= 6–8 U/h for 60–80 kg). Target glucose reduction: 3–4 mmol/L/h. Continue insulin infusion until: glucose < 12 + pH > 7.3 + HCO3 > 18 + ketones < 0.6 + patient eating. Switch to SC insulin 30–60 min before stopping infusion. POTASSIUM: K+ 6.1 → monitor hourly; do NOT add initially. When K+ < 5.0: add 20–40 mmol/h. When K+ < 3.5: stop insulin, correct K+ urgently. Target K+: 4.0–5.0 throughout. BICARBONATE: pH 6.98 — consider NaHCO3 IF pH < 6.9 persistent despite fluid resuscitation (controversial; 100 mmol over 30 min if given). MONITORING: Glucose hourly; electrolytes (Na+, K+, HCO3) every 2 hours; blood gas every 4 hours; urine output hourly; ECG continuous; vital signs 15-min then 30-min then hourly as stable. RESOLUTION criteria: glucose < 12 + pH > 7.3 + HCO3 > 18 + ketones < 0.6 + clinically improving. POST-CRISIS: Identify precipitant; sick-day rule education; adjust long-term insulin; ensure patient and family education before discharge. Competency: PC-11 (L3), PC-9 (L3), OPK-7 (L3).

CONTROL SECTION No. 5

Section 5: Thyroid Diseases. Thyrotoxicosis and Hypothyroidism Syndromes

Competencies: OPK-5 (L1–L2), PC-4 (L1–L2), PC-5 (L1–L2), PC-7 (L1–L2), PC-8 (L1–L2)

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

1. Classify thyroid diseases. Describe aetiology of thyrotoxicosis.
2. Describe Graves' disease: clinical picture, diagnosis, treatment options.
3. Describe hypothyroidism: classification, clinical picture, diagnosis (TSH, Free T4).
4. Describe iodine deficiency diseases: endemic goitre, prevention.
5. Describe thyroiditis: Hashimoto's, subacute de Quervain, postpartum.
6. What is subclinical hypothyroidism and when should it be treated?

MCQ Tests:

Q1. Female, 30 years, Graves' disease on carbimazole 30 mg/day for 8 months. Now euthyroid (Free T4 14 pmol/L, TSH 1.2). TRAb still elevated 6.8 IU/L (norm < 1.75). Patient wants definitive treatment. Which is most appropriate?

A) Continue carbimazole indefinitely
B) Stop carbimazole — TRAb is elevated but TSH is normal
C) Radioactive iodine (I-131) or thyroidectomy — TRAb elevated = high relapse risk
D) Add beta-blocker only
E) Switch to propylthiouracil

CORRECT ANSWER: C) Radioactive iodine (I-131) or thyroidectomy — TRAb elevated = high relapse risk

Persistent elevated TRAb after 8 months of antithyroid therapy = high relapse probability on drug withdrawal (>60% relapse rate). European Thyroid Guidelines recommend consideration of definitive therapy (RAI or surgery) when TRAb remains elevated at the end of planned antithyroid drug course (typically 12–18 months total). Euthyroid on drugs does not predict remission — TRAb is the predictor. Competency: OPK-7 (L2), PC-7 (L2).

Q2. TSH 18 mIU/L, Free T4 6.1 pmol/L (norm 11–22). Patient: 45-year-old male, fatigue, weight gain 6 kg, cold intolerance, dry skin, slow speech, bradycardia 52 bpm, elevated cholesterol. Which diagnosis and ICD-10?

A) Subclinical hypothyroidism — E02
B) Primary overt hypothyroidism — E03.9
C) Secondary hypothyroidism — E03.1
D) Sick euthyroid syndrome — E07.8
E) Hypothyroidism due to radioiodine — E89.0

CORRECT ANSWER: B) Primary overt hypothyroidism — E03.9

Primary overt hypothyroidism: TSH elevated (18, norm < 4) + Free T4 low (6.1, norm 11–22) = confirmed primary thyroid failure. Clinical picture matches: fatigue, weight gain, cold intolerance, dry skin, bradycardia, elevated cholesterol (all reversible with levothyroxine). Secondary hypothyroidism = low/normal TSH + low T4 (pituitary failure). Subclinical = elevated TSH + normal T4. ICD-10: E03.9. Competency: PC-5 (L1–L2).

Q3. Pregnant woman, 10 weeks. TSH 6.8 mIU/L (pregnancy norm < 2.5 in first trimester), Free T4 9.8 pmol/L. Anti-TPO antibodies positive. No prior thyroid disease. Which action?

A) No treatment — TSH often elevated in pregnancy

B) Start levothyroxine — hypothyroidism in pregnancy requires treatment
C) Start carbimazole
D) Recheck TSH after delivery
E) Thyroid ultrasound and wait

CORRECT ANSWER: B) Start levothyroxine — hypothyroidism in pregnancy requires treatment
Overt hypothyroidism in pregnancy (TSH > trimester-specific upper limit + low Free T4) requires IMMEDIATE levothyroxine treatment. Untreated: impaired fetal neurological development, miscarriage, preterm birth, pre-eclampsia. TSH target in pregnancy: < 2.5 mIU/L (first trimester), < 3.0 mIU/L (second/third). Anti-TPO+ confirms autoimmune origin (Hashimoto's). Starting dose: 1.6–2.0 µg/kg/day. Competency: PC-9 (L1), OPK-7 (L2).

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient M., 26 years (female). 5-month history: palpitations (HR 116 bpm), weight loss 11 kg, excessive sweating, irritability, hand tremor, exophthalmos (proptosis 3 mm bilateral). Thyroid: diffusely enlarged, soft, bruit. Free T4 72 pmol/L, TSH < 0.01, TRAb 22.4 IU/L. ECG: sinus tachycardia. Anti-TPO 840 IU/mL.

Questions:

7. Formulate the clinical diagnosis with ICD-10. (5 points)
8. List additional examinations and justify. (5 points)
9. Compose a treatment plan including drug prescriptions and monitoring. (10 points)

ANSWERS:

1. Diagnosis: Diffuse Toxic Goitre (Graves' Disease), Grade II, severe thyrotoxicosis. Graves' ophthalmopathy (bilateral proptosis). ICD-10: E05.0 (Thyrotoxicosis with diffuse goitre). Justification: thyrotoxicosis syndrome (tachycardia 116, weight loss 11 kg, tremor, sweating, irritability) + diffuse goitre with bruit + TRAb 22.4 (pathognomonic marker for Graves') + Free T4 72 (markedly elevated) + TSH undetectable + bilateral proptosis (Graves' ophthalmopathy). Competency: PC-5 (L1–L2), OPK-5 (L1–L2).

2. Additional examinations: Thyroid ultrasound + Doppler (goitre volume measurement; Graves' pattern: markedly increased vascularity 'thyroid inferno'); anti-TPO (already known: 840 — confirms autoimmune); anti-thyroglobulin antibodies (complete autoimmune profile); CBC (baseline before thionamide — agranulocytosis monitoring); liver function tests (baseline — thionamides hepatotoxic); echocardiography (tachycardia 116 + thyrotoxicosis → assess for high-output heart failure, pulmonary hypertension); ophthalmological examination (VISA/CAS score for GO severity and activity); bone densitometry (thyrotoxicosis causes bone loss). Competency: PC-4 (L1–L2), OPK-5 (L2).

3. Treatment plan: ANTITHYROID: Carbimazole 40 mg/day in 2 divided doses (or methimazole 40 mg/day). Titrate: check Free T4 + TSH every 4–6 weeks; reduce dose when euthyroid (target Free T4 mid-normal). PRESCRIPTION: Carbimazole 20 mg tablets — take 1 tablet twice daily with meals. Continue 12–18 months total. MONITORING FOR SAFETY: CBC at every visit (sore throat/fever = STOP immediately, check CBC for agranulocytosis); liver function monthly (hepatotoxicity); TFTs every 4–6 weeks. SYMPTOM CONTROL: Propranolol 40 mg 3 times daily (tachycardia, tremor, sweating) — continue until euthyroid, then wean. PRESCRIPTION: Propranolol 40 mg tablets — take 1 tablet 3 times daily. DEFINITIVE TREATMENT PLAN: After 12–18 months: assess TRAb. If TRAb elevated = high relapse risk → refer for radioactive iodine (I-131) or thyroidectomy. OPHTHALMOPATHY: Ophthalmological consultation; selenium supplementation (selenium 200 µg/day — mild active GO); if moderate-severe: systemic steroids (methylprednisolone IV course); achieve euthyroid state urgently (improves ophthalmopathy). NOTE: Achieve euthyroid before considering RAI — RAI may worsen GO (steroid cover required if RAI chosen). Competency: OPK-7 (L2–L3), PC-8 (L2–L3), PC-14 (L2).

CONTROL SECTION No. 6

Section 6: Adrenal Gland Diseases. Hypocorticism and Hypercorticism

Competencies: OPK-5 (L1–L2), PC-4 (L1–L2), PC-5 (L1–L2), PC-9 (L2), PC-11 (L2)

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

10. Describe chronic adrenal insufficiency (Addison's disease): aetiology, pathogenesis, diagnosis.
11. What are the differences between primary and secondary adrenal insufficiency?
12. Describe acute adrenal crisis: triggers, clinical picture, emergency management.
13. Describe Cushing's syndrome: aetiology, classification, clinical picture.
14. What are the diagnostic steps for confirming hypercorticism?
15. What is the differential diagnosis between Cushing's disease and Cushing's syndrome?

MCQ Tests:

Q1. Patient taking prednisolone 20 mg/day for 8 months undergoes elective surgery under general anaesthesia. No additional steroids given. Post-operative: refractory hypotension, nausea, abdominal pain, confusion. What is the diagnosis and immediate treatment?

A) Post-operative hypovolaemia — IV fluids only
B) Adrenal crisis from HPA suppression — hydrocortisone 100 mg IV STAT
C) Anaphylaxis — adrenaline 0.5 mg IM
D) Pulmonary embolism — anticoagulation
E) Septic shock — broad-spectrum antibiotics

CORRECT ANSWER: B) Adrenal crisis from HPA suppression — hydrocortisone 100 mg IV STAT
Surgical stress + 8 months on prednisolone 20 mg/day = certain HPA axis suppression. Failure to give perioperative steroid cover + surgical stress → adrenal crisis. Treatment: hydrocortisone 100 mg IV STAT + 100 mg every 6–8 hours + 0.9% NaCl + 5% glucose. Sick-day rule violated: should have received double dose + IV hydrocortisone for major surgery. Competency: PC-11 (L1–L2), PC-9 (L2).

Q2. 24-hour urinary free cortisol (UFC): 4.8 × upper limit of normal. Low-dose DST (1 mg overnight): cortisol 102 nmol/L (norm < 50). ACTH 12 pg/mL (norm 10–46). Which form of hypercorticism?

A) Cushing's disease (ACTH-secreting pituitary adenoma)
B) Ectopic ACTH syndrome
C) Adrenal-dependent Cushing's syndrome (ACTH-independent)
D) Pseudo-Cushing's syndrome
E) Subclinical hypercorticism

CORRECT ANSWER: C) Adrenal-dependent Cushing's syndrome (ACTH-independent)
UFC ×4.8 ULN = confirmed hypercorticism. LDDST non-suppressed (102 > 50) = confirms autonomous cortisol production. ACTH 12 pg/mL = LOW-NORMAL (suppressed by autonomous cortisol) = ACTH-INDEPENDENT = adrenal source (adenoma, carcinoma, bilateral hyperplasia). If ACTH were > 20, pituitary or ectopic source would be considered. Next step: adrenal CT to find adrenal adenoma/carcinoma. Competency: PC-5 (L1–L2), OPK-5 (L2).

Q3. Patient with Addison's disease on hydrocortisone 15 mg AM + 5 mg PM + fludrocortisone 0.1 mg/day. Develops gastroenteritis with vomiting — cannot take oral medications. BP 95/60, K+ 5.6, Na+ 130. What is the correct sick-day management?

A) Double oral dose when vomiting stops

B) Hydrocortisone 100 mg IM/IV STAT + IV 0.9% NaCl — hospitalise
C) Fludrocortisone increase only
D) Oral rehydration solutions only
E) Wait 24 hours before seeking help

CORRECT ANSWER: B) Hydrocortisone 100 mg IM/IV STAT + IV 0.9% NaCl — hospitalise
Sick-day rule: if vomiting prevents oral medication absorption → inject hydrocortisone (patient should carry emergency kit: hydrocortisone 100 mg IM). Hospital: hydrocortisone 100 mg IV STAT + continuous IV fluids (0.9% NaCl ± 5% glucose). During acute illness, mineralocorticoid (fludrocortisone) is less critical as large hydrocortisone doses have some mineralocorticoid activity. Competency: PC-9 (L2), PC-11 (L1–L2).

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient T., 36 years (female). 2-year history: weight gain primarily abdominal (buffalo hump, moon face), purple striae on abdomen, proximal muscle weakness (difficulty climbing stairs), easy bruising, amenorrhoea for 8 months, depressive mood. BP 172/104 mmHg. Fasting glucose 10.2 mmol/L. 24h UFC 6.2 × ULN. LDDST non-suppressed (cortisol 124 nmol/L). ACTH 78 pg/mL. MRI pituitary: 7 mm adenoma.

Questions:

16. Formulate the clinical diagnosis with ICD-10 code. (5 points)
17. List additional confirmatory examinations. (5 points)
18. Compose a comprehensive treatment plan. (10 points)

ANSWERS:

1. Diagnosis: Cushing's Disease (ACTH-secreting pituitary microadenoma), active phase. Complications: steroid diabetes mellitus (fasting glucose 10.2 mmol/L); arterial hypertension; myopathy; osteoporosis risk (check BMD); menstrual dysfunction (amenorrhoea); depressive syndrome. ICD-10: E24.0 (Pituitary-dependent Cushing's disease). Justification: clinical hypercorticism syndrome (central obesity, purple striae, proximal myopathy, bruising, moon face, buffalo hump) + biochemical confirmation (UFC ×6.2, LDDST non-suppressed) + ACTH 78 pg/mL (elevated = ACTH-dependent) + pituitary adenoma 7 mm on MRI. Competency: PC-5 (L1–L2), OPK-5 (L2).

2. Confirmatory examinations: High-dose DST (2 mg ×8 doses) — Cushing's disease typically suppresses > 50%; ectopic ACTH does not; inferior petrosal sinus sampling (IPSS) — gold standard for confirming pituitary source and lateralisation (ACTH gradient > 2:1 central:peripheral = pituitary confirmed); CRH stimulation test (exaggerated ACTH response in pituitary disease, blunted in ectopic); visual field testing (compressive effect); bone densitometry (osteoporosis — complication of hypercorticism); coagulation profile (hypercoagulability — DVT/PE risk); echocardiography (hypertension + hypercorticism = cardiac risk). Competency: PC-4 (L2), OPK-5 (L2).

3. Treatment plan: DEFINITIVE: Transsphenoidal surgical resection of pituitary adenoma — first-line for Cushing's disease. Remission criteria: post-operative cortisol < 50 nmol/L. PRE-SURGICAL PREPARATION: Steroidogenesis inhibitor — metyrapone (500 mg 3 times/day, titrate to normalise cortisol) to reduce perioperative complications. POST-SURGICAL: Hydrocortisone replacement (20–30 mg/day in 2–3 doses) until HPA axis recovery (6–24 months); cortisol stimulation test at 6–12 months. IF SURGERY FAILS: Repeat transsphenoidal surgery; stereotactic radiosurgery (Gamma Knife — 3–5 year delay to effect); bilateral adrenalectomy (curative but lifelong adrenal insufficiency + Nelson's syndrome risk — MRI pituitary monitoring essential). COMORBIDITIES: Antihypertensive (target < 130/80); metformin or insulin for steroid diabetes; calcium 1000 mg + vitamin D 800 IU + bisphosphonate for osteoporosis; antidepressant for mood disorder; anticoagulation assessment for VTE risk; contraception counselling (amenorrhoea will resolve with remission). MONITORING: MRI pituitary 3 months post-op then annually; HbA1c every 3 months; BP at every visit; BMD annually. Competency: OPK-7 (L3), PC-7 (L3), PC-8 (L3).

CONTROL SECTION No. 7

Section 7: Hypothalamic-Pituitary Diseases. Acromegaly. Diabetes Insipidus. Hypopituitarism. Hyperprolactinaemia

Competencies: OPK-5 (L1–L2), PC-4 (L1–L2), PC-5 (L1–L2), PC-7 (L1–L2)

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

19. Describe acromegaly: pathogenesis, clinical picture, diagnostic criteria (IGF-1, GH OGTT test).
20. Describe central diabetes insipidus: pathogenesis, diagnosis, desmopressin test.
21. What is hypopituitarism? Describe clinical features of each axis deficiency.
22. Describe hyperprolactinaemia: causes (physiological, pharmacological, tumour), clinical picture, treatment.
23. What are the indications for surgery in pituitary adenomas?
24. How are pituitary incidentalomas managed?

MCQ Tests:

Q1. Patient with acromegaly — post transsphenoidal surgery. 3 months later: IGF-1 1.4 × ULN, random GH 3.2 µg/L. GH after OGTT nadir 1.8 µg/L (norm < 1 µg/L). What does this indicate?

A) Complete remission — no further treatment needed
B) Biochemical partial remission — continue monitoring only
C) Persistent/residual disease — additional treatment required
D) GH measurement error — repeat in 6 months
E) Normal post-operative findings

CORRECT ANSWER: C) Persistent/residual disease — additional treatment required
Acromegaly REMISSION criteria (post-surgery): IGF-1 normal for age AND GH < 1 µg/L on OGTT nadir. This patient: IGF-1 still elevated (1.4 × ULN) + GH OGTT nadir 1.8 (> 1 µg/L) = PERSISTENT DISEASE. Treatment options: somatostatin analogues (octreotide LAR or lanreotide), GH receptor antagonist (pegvisomant), repeat surgery, or stereotactic radiosurgery. Competency: PC-5 (L2), PC-7 (L1–L2).

Q2. Female, 28 years, 14 months of secondary amenorrhoea, galactorrhoea bilateral, headaches. Prolactin 285 µg/L (norm < 20). MRI pituitary: 18 mm adenoma compressing optic chiasm. Visual field testing: bitemporal hemianopia. Which management?

A) Cabergoline alone — reduce tumour size first
B) Urgent transsphenoidal surgery
C) Cabergoline + visual field monitoring — surgery if vision deteriorates despite treatment
D) Bromocriptine preferred in macroadenoma
E) Radiotherapy first-line

CORRECT ANSWER: A) Cabergoline alone — reduce tumour size first
Even with optic chiasm compression, MEDICAL treatment (cabergoline) is first-line for macroprolactinoma. Cabergoline causes significant tumour shrinkage in 80% of macroprolactinomas — often relieving chiasm compression within weeks. Surgery is reserved for: drug intolerance or resistance; acute haemorrhage (pituitary apoplexy); persistent severe visual loss despite adequate dopamine agonist treatment. Cabergoline 0.5 mg twice weekly, increase to effect. Competency: OPK-7 (L2), PC-7 (L2).

Q3. Patient, 55 years, craniopharyngioma removed 2 years ago. Now: fatigue, loss of libido, erectile dysfunction, cold intolerance, weight gain, polyuria 6 L/day, polydipsia. Which axes are likely deficient?

A) Prolactin excess only

B) Gonadotrophin + GH deficiency only
C) Multiple anterior pituitary axes + posterior pituitary (ADH deficiency)
D) Thyroid axis only
E) Adrenal axis only

CORRECT ANSWER: C) Multiple anterior pituitary axes + posterior pituitary (ADH deficiency)
Craniopharyngioma surgery commonly causes PANHYPOPITUITARISM: (1) gonadotrophin deficiency — erectile dysfunction, loss of libido; (2) thyroid axis deficiency — cold intolerance, weight gain; (3) cortisol deficiency — fatigue (life-threatening if unrecognised); (4) GH deficiency (likely); (5) posterior pituitary — ADH deficiency → diabetes insipidus (polyuria 6 L/day). All axes must be systematically assessed post-craniopharyngioma. Cortisol must be replaced first (priority — life-threatening). Competency: PC-5 (L2), OPK-5 (L2).

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient G., 46 years, 8-year gradual coarsening of facial features, increased hand and foot size (shoe size increased by 2), frontal bossing, macroglossia. Diagnosed with hypertension (BP 158/96) and type 2 DM 2 years ago. Snoring and daytime somnolence (suspected OSA). IGF-1 × 4.2 ULN. GH nadir after 75 g OGTT: 7.4 μg/L (not suppressed). MRI pituitary: 2.1 cm macroadenoma, no optic chiasm contact.

Questions:

25. Formulate clinical diagnosis with ICD-10 and list all identified complications. (5 points)
26. List additional examinations with justification. (5 points)
27. Compose a complete treatment and monitoring plan. (10 points)

ANSWERS:

1. Diagnosis: Acromegaly (GH-secreting pituitary macroadenoma, 2.1 cm), active disease. Complications: secondary arterial hypertension; secondary DM type 2 (GH — insulin antagonist); acromegalic arthropathy (joint pains from osteophyte formation); macroglossia; suspected obstructive sleep apnoea syndrome. ICD-10: E22.0 (Acromegaly and pituitary gigantism). Justification: 8-year history of progressive acral growth + IGF-1 ×4.2 ULN + GH not suppressed after OGTT (7.4 μg/L, norm < 1) + pituitary macroadenoma 2.1 cm. Active = both IGF-1 elevated + GH not suppressed. Competency: PC-5 (L1–L2), OPK-5 (L2).

2. Additional examinations: Visual fields (Goldmann/Humphrey) — macroadenoma proximity to chiasm; echocardiography — acromegalic cardiomyopathy (concentric LV hypertrophy + diastolic dysfunction common); polysomnography — quantify OSA (present in 60% of acromegaly); colonoscopy — increased colorectal adenoma/cancer risk; bone densitometry — osteoporosis; complete pituitary function testing — IGF-1, LH, FSH, testosterone (males), TSH, Free T4, cortisol/ACTH stimulation test, prolactin (GH adenomas sometimes co-secrete prolactin — check); calcium and phosphorus (GH causes hypercalciuria). Competency: PC-4 (L1–L2).

3. Treatment plan: FIRST-LINE: Transsphenoidal surgical resection — cure rate 80–90% for microadenoma, 40–60% for macroadenoma. Pre-operative somatostatin analogue (octreotide LAR 20 mg monthly × 3 months) to reduce GH levels, tumour vascularity, and surgical morbidity. REMISSION CRITERIA: Post-op IGF-1 normal + GH OGTT nadir < 1 μg/L. MEDICAL THERAPY (if surgery incomplete/not curative): Somatostatin analogues: octreotide LAR 20–30 mg IM monthly or lanreotide autogel 90–120 mg SC every 28 days; GH receptor antagonist: pegvisomant 10–30 mg SC daily — normalises IGF-1 in >90% (does not reduce tumour size). RADIOTHERAPY: Gamma Knife stereotactic radiosurgery if residual disease after surgery + medical therapy inadequate; effect delayed 5–10 years; risk of hypopituitarism. COMPLICATIONS MANAGEMENT: Antihypertensive therapy (target < 130/80); metformin or insulin for DM (GH is insulin-antagonistic — insulin often required); CPAP for OSA; orthopaedic/rheumatological care for arthropathy; colorectal screening colonoscopy every 3–5 years. MONITORING: IGF-1 + GH every 6 months; MRI pituitary annually; visual fields if chiasm proximity; pituitary function annually (hypopituitarism risk especially post-surgery/radiotherapy). Competency: OPK-7 (L3), PC-7 (L3), PC-8 (L3).

CONTROL SECTION No. 8

Section 8: Obesity. Metabolic Syndrome. PCOS. Dispensary Observation

Competencies: OPK-5 (L1–L2), OPK-7 (L1–L2), PC-4 (L1–L2), PC-5 (L1–L2), PC-7 (L1–L2), PC-8 (L1), PC-14 (L1–L2)

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

28. Classify obesity (WHO BMI classification). Describe pathogenesis and role of insulin resistance.
29. What are the IDF 2006 diagnostic criteria for metabolic syndrome?
30. Describe clinical complications of obesity: cardiovascular, orthopaedic, oncological, endocrine.
31. What are the principles of drug treatment of obesity (orlistat, GLP-1 agonists)?
32. Describe PCOS: Rotterdam criteria, clinical picture, hyperandrogenaemia.
33. What is the dispensary observation schedule for DM, hypothyroidism, and Addison's disease patients?

MCQ Tests:

Q1. Female, 36 years, BMI 38.2 kg/m², waist 110 cm. Fasting glucose 6.2 mmol/L, TG 3.1 mmol/L, HDL 0.82 mmol/L, BP 146/94 mmHg. Fasting insulin 32 µU/mL, HOMA-IR 8.9. Which primary diagnosis and ICD-10?

A) Type 2 DM — E11
B) Obesity Grade III with metabolic syndrome — E66.09 + E88.81
C) Metabolic syndrome without obesity — E88.81
D) Pre-diabetes without metabolic syndrome — R73.0
E) Hypothyroidism — E03.9

CORRECT ANSWER: B) Obesity Grade III with metabolic syndrome — E66.09 + E88.81
Obesity Grade III: BMI ≥ 40 — not quite (38.2), so actually Grade II (35–39.9 BMI). IDF metabolic syndrome: abdominal obesity (waist female > 80 cm — 110 cm present) + ≥2: TG ≥ 1.7 (3.1 ✓), HDL < 1.29 female (0.82 ✓), BP ≥ 130/85 (146/94 ✓), fasting glucose ≥ 5.6 (6.2 ✓) — all 4 additional criteria met. HOMA-IR 8.9 confirms severe insulin resistance (norm < 2.5). Primary diagnosis: Obesity Grade II + metabolic syndrome. Competency: PC-5 (L1).

Q2. Patient with PCOS, BMI 27, oligomenorrhoea, hirsutism. Wants contraception but also to treat hirsutism and regulate cycle. No plans for pregnancy in next 2 years. Which is the preferred first-line treatment?

A) Metformin alone
B) Letrozole for ovulation induction
C) Combined oral contraceptive pill with anti-androgenic progestogen (e.g., drospirenone + EE)
D) Spironolactone alone
E) Clomiphene citrate

CORRECT ANSWER: C) Combined oral contraceptive pill with anti-androgenic progestogen (e.g., drospirenone + EE)
For PCOS patient NOT wanting pregnancy: COC with anti-androgenic progestogen (drospirenone, chlormadinone, cyproterone) is first-line — it: (1) regulates menstrual cycle; (2) reduces androgen production (LH suppression + SHBG increase); (3) treats hirsutism and acne; (4) provides contraception. Letrozole/clomiphene: for ovulation induction when pregnancy desired. Metformin: insulin resistance correction, can combine with COC. Competency: PC-7 (L2), OPK-7 (L2).

Q3. Patient with Type 2 DM, stable on metformin + sitagliptin, HbA1c 6.8% for 18 months. No complications. Appropriate dispensary monitoring frequency: how often should HbA1c be checked?

A) Every month
B) Every 3 months
C) Every 6 months
D) Once per year
E) Only when symptoms occur

CORRECT ANSWER: C) Every 6 months
ADA Standards of Care 2020: HbA1c every 6 months in STABLE patients with therapy unchanged and consistently at target. HbA1c every 3 months: recent therapy change, HbA1c above target, or pregnancy. Annual monitoring in stable DM: lipid profile, urine albumin/creatinine ratio, ophthalmological examination, monofilament foot examination, renal function (eGFR). Competency: PC-14 (L1–L2), PC-7 (L2).

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient N., 32 years (female). Primary infertility (2 years trying). Oligomenorrhoea (6–8 cycles/year). Hirsutism (Ferriman-Gallwey score 16, face/abdomen/thighs). Acne. BMI 32.4 kg/m², waist 98 cm. Fasting glucose 6.0 mmol/L, insulin 35 μU/mL (HOMA-IR 9.4). Total testosterone 4.1 nmol/L (norm < 2.5). LH 18 IU/L, FSH 5.8 IU/L (ratio 3.1). Pelvic US: bilateral ovarian enlargement, 16–18 small follicles/ovary, echogenic stroma. Husband: normal semen analysis.

Questions:

34. Formulate diagnosis with ICD-10. (5 points)
35. List additional examinations. (5 points)
36. Compose a comprehensive management plan — both infertility and metabolic components. (10 points)

ANSWERS:

1. Diagnosis: Polycystic Ovary Syndrome (PCOS), phenotype A (all 3 Rotterdam criteria met). Complicated by: severe insulin resistance (HOMA-IR 9.4); hyperandrogenaemia (testosterone 4.1 nmol/L); primary infertility (anovulatory); obesity Grade I (BMI 32.4); pre-diabetes (fasting glucose 6.0). ICD-10: E28.2 (PCOS). Rotterdam criteria met: (1) oligoanovulation — 6–8 cycles/year; (2) hyperandrogenism — testosterone elevated + hirsutism FG 16 + acne; (3) polycystic ovaries — ≥12 follicles/ovary on US. Competency: PC-5 (L1–L2).

2. Additional examinations: OGTT with insulin (0, 30, 60, 120 min) — assess IGT degree and insulin resistance curve; 17-OH progesterone (exclude non-classic congenital adrenal hyperplasia — can mimic PCOS); DHEAS + androstenedione (adrenal vs ovarian androgen source); anti-Müllerian hormone (AMH) — ovarian reserve for fertility planning; prolactin (exclude hyperprolactinaemia as cause of oligomenorrhoea); TSH + Free T4 (thyroid disease can cause menstrual irregularity); endometrial ultrasound (anovulation = endometrial hyperplasia risk); partner semen analysis confirmed normal — noted. Competency: PC-4 (L2).

3. Management plan: LIFESTYLE (mandatory first — most impactful): 5–10% weight reduction dramatically restores ovulation in 30–50% of obese PCOS. Diet: low-GI foods; caloric deficit 500 kcal/day; limit saturated fats. Physical activity: 150 min/week aerobic + resistance training. INSULIN RESISTANCE: Metformin 500 mg 3 times/day (titrate over 4 weeks) — reduces insulin resistance, decreases androgen production, can restore ovulation, reduces miscarriage risk if pregnancy achieved. Continue throughout fertility treatment and into pregnancy (reduces GDM risk). PRE-DIABETES: OGTT annually; lifestyle modification; consider metformin (already indicated for PCOS). INFERTILITY (anovulatory, primary): First-line ovulation induction: Letrozole 2.5–5 mg days 3–7 (aromatase inhibitor — superior to clomiphene in obese PCOS: NEJM 2014). Cycle monitoring: transvaginal US follicle tracking from day 10; hCG trigger when follicle ≥ 18 mm. Second-line: clomiphene + metformin (if letrozole unavailable). Third-line: FSH injection gonadotropin stimulation (with careful monitoring — OHSS risk). IVF: if ≥3 failed OI cycles. HYPERANDROGENAEMIA/HIRSUTISM: Concurrent anti-androgenic therapy not appropriate during fertility treatment (teratogenic). After successful pregnancy and contraception needed: COC with drospirenone or cyproterone. Spironolactone (during contraception only). DISPENSARY OBSERVATION: Every 6 months: anthropometrics, HOMA-IR, testosterone, HbA1c/OGTT; annually: endometrial assessment (anovulation risk), ophthalmological (if DM develops). Competency: OPK-7 (L3), PC-7 (L3), PC-8 (L3), PC-14 (L1–L2).

9. RECOMMENDED LITERATURE

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