MINISTRY OF SCIENCE AND HIGHER EDUCATION OF THE RUSSIAN FEDERATION

MINISTRY OF SCIENCE, HIGHER EDUCATION AND INNOVATION OF THE KYRGYZ REPUBLIC

Kyrgyz-Russian Slavic University

named after the first President of the Russian Federation B.N. Yeltsin

FUND OF ASSESSMENT TOOLS (FAT)

Discipline: ENDOCRINOLOGY

Assigned to Department	Therapy No. 1 (Pediatrics and Dentistry)
Curriculum Code	310501_20_6 ld in.plx
Specialty (RF/KR)	31.05.01 (RF) / 560001 (KR) — General Medicine (for foreign students)
Qualification	Specialist / Physician
Form of Study	Full-time
Total Credits	2 ZET (72 hours)
Classroom Hours	54 hours (Lectures 18 h + Practical 36 h)
Independent Work	17.7 hours
Year / Semester	4th year, Semester 8 (4.2)
Teaching Weeks	16 weeks
Duration of Programme	2020–2026 academic years
Control Type	Credit with Grade — Semester 8

APPROVED:

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Developed in accordance with FSES 3++: Order of the Ministry of Education and Science of the Russian Federation dated 12.08.2020 No. 988, Specialty 31.05.01 General Medicine.

1. COMPETENCIES FORMED THROUGH THE DISCIPLINE

OPK-6: Readiness to Maintain Medical Records

Capable and ready to maintain medical documentation in endocrinology: medical history, outpatient card, discharge epicrisis, prescriptions for endocrinological drugs.

Level	Know	Be Able To	Master	Assessment Tools
Level 1	Principles of filling out medical documentation in endocrinology; structure of the medical history of a patient with endocrine pathology	Draw up a medical history, outpatient card, discharge epicrisis for patients with endocrine diseases	Primary medical documentation skills in endocrinology	Block A: Tests on medical documentation; practical documentation tasks
Level 2	Structure of the medical history of a patient with endocrine pathology; rules for issuing prescriptions for hypoglycaemic and thyroid drugs	Write prescriptions for endocrinological drugs; fill out referrals and endocrinologist conclusions	Algorithm for issuing referrals and conclusions of an endocrinologist	Block B: Situational cases with documentation tasks; prescription writing
Level 3	Rules for issuing prescriptions for hypoglycaemic and thyroid drugs; regulatory requirements of MoH KR	Complete all endocrinology documentation in compliance with current regulatory standards	Complete documentation skills for all common endocrine conditions	Block C: Case history defense; Block D: Certification questions

PC-9: Readiness to Provide Primary Healthcare for Acute and Chronic Endocrine Conditions

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

Level	Know	Be Able To	Master	Assessment Tools
Level 1	Clinical manifestations of acute and chronic endocrine diseases; emergency presentations of endocrine crises	Link symptoms, examination data and laboratory/instrumental findings into a unified clinical picture and establish diagnosis	Skills to identify signs of an acute endocrine disease or exacerbation of a chronic condition	Block A: Tests on clinical manifestations; oral questioning
Level 2	Methods of treatment and medical manipulations for endocrine pathological conditions;	Prepare patient, instruments and medications for necessary manipulations; prescribe appropriate therapy	Skills of aetiological and pathogenetic therapy in treatment of endocrine diseases depending on severity	Block B: Situational cases on treatment planning; drug calculation tasks

	pharmacological groups and drug interactions			
Level 3	General principles of treatment; knowledge of pharmacological groups; assessment of disease stage	Assess stage of endocrine disease and prescribe therapy appropriate to stage; provide emergency care for endocrine crises	Skills and methods of providing medical care for acute (diabetic ketoacidosis, thyrotoxic crisis, adrenal crisis) and chronic endocrine diseases	Block C: Practice tasks; Block D: Certification — emergency management scenarios

PC-6: Ability to Determine Pathological Conditions and Syndromes According to ICD-10

Capable of determining main pathological conditions, symptoms, syndromes in patients with endocrine diseases in accordance with ICD-10.

Level	Know	Be Able To	Master	Assessment Tools
Level 1	Aetiology, pathogenesis and classification of the most common forms of endocrine diseases (ICD-10 codes); differential diagnostic criteria for endocrine syndromes	Determine the algorithm for diagnosing an endocrine disease taking into account ICD-10; identify syndrome	Skills in formulating a clinical diagnosis of endocrine diseases	Block A: Tests on classification, ICD-10, syndromes
Level 2	Differential diagnostic criteria for endocrine syndromes; features of secondary endocrine disorders	Formulate a detailed clinical diagnosis in accordance with ICD-10; draw up laboratory and instrumental examination plan	Skills in drawing up a plan for laboratory and instrumental examination in endocrine diseases	Block B: Situational cases — differential diagnosis; examination plan composition
Level 3	Complex endocrine syndromes; overlap between endocrine conditions; complications classification	Conduct differential diagnosis of endocrine syndromes; formulate structured diagnosis with complications	Skills in differential syndromic diagnosis and structured clinical diagnosis formulation	Block C: Practice tasks; Block D: Complex diagnostic cases

2. LEARNING OUTCOMES OF THE DISCIPLINE

2.1. KNOW:

- Medical documentation in endocrinology organisations
- Pathological symptoms and syndromes in the most common endocrine diseases
- Aetiology, pathogenesis and classification of the most common forms of endocrine diseases
- Clinical picture, diagnostic criteria and complications of diseases of the endocrine system
- Modern methods of clinical, laboratory and instrumental examination of patients with endocrine pathology
- The main groups of drugs used to treat endocrine diseases
- Modern principles of treatment of diseases of the endocrine system
- Features of emergency care for endocrine diseases
- Basic principles of prevention of typical forms of endocrine diseases

2.2. BE ABLE TO:

- Use educational, scientific literature and Internet resources for professional activities
- Work with medical documentation; fill out medical histories and write prescriptions
- Conduct a survey and physical examination of the patient; assess patient condition; examine the organs of the endocrine system
- Identify the main pathological symptoms and syndromes of endocrine diseases
- Draw up a plan of laboratory and instrumental examination; interpret results
- Determine the algorithm for diagnosing an endocrine disease according to ICD-10; formulate a detailed clinical diagnosis
- Develop a treatment plan; select and prescribe drug and non-drug therapy
- Identify urgent and life-threatening conditions and provide emergency medical care for endocrine diseases
- Use methods of primary and secondary prevention of endocrine diseases based on evidence-based medicine

2.3. MASTER:

- Skills in maintaining medical records; technique of drawing up a medical history in endocrinology
- Algorithm of general clinical examination of a patient with endocrine pathology
- Algorithm of laboratory and instrumental examination in suspected endocrine pathology; interpretation of results
- Skills in making a preliminary diagnosis and forming an examination plan
- Skills in formulating a clinical diagnosis of patients with endocrine diseases in typical form
- Skills in management and treatment of patients with endocrine pathology; assessment of treatment effectiveness
- Methods of emergency care for endocrine diseases (diabetic ketoacidosis, hypoglycaemic coma, thyrotoxic crisis, adrenal crisis)
- Skills in planning prevention and treatment of the most common endocrine diseases

- **3. STRUCTURE OF ASSESSMENT TOOL BLOCKS**

Block	Content	Competencies	Semester
Block A	MCQ tests on aetiology, pathogenesis, classification, clinical manifestations, emergency conditions, ICD-10, medical documentation; oral questioning	OPK-6 (L1), PC-6 (L1–L2), PC-9 (L1): knowledge of endocrine diseases and documentation	8
Block B	Situational clinical cases: diagnosis formulation, treatment planning, emergency management, documentation tasks; patient supervision reports	OPK-6 (L1–L2), PC-6 (L1–L2), PC-9 (L1–L2): application of diagnostic and treatment algorithms	8
Block C	Practice-oriented tasks: patient examination, clinical diagnosis formulation, prescription writing, case history writing and defense	OPK-6 (L2–L3), PC-6 (L2–L3), PC-9 (L2–L3): full competency application	8
Block D	Certification questions: differential diagnosis, emergency management, treatment planning, ECG/lab interpretation, practical skills	All competencies, all levels (OPK-6, PC-6, PC-9)	8 (Credit+Grade)

4. DISTRIBUTION BY SEMESTER

Semester	Control Type	Blocks Used	Competencies
8 (4.2)	Credit with Grade	Blocks A, B, C, D	OPK-6, PC-6, PC-9 — all levels

5. TECHNOLOGY MAP OF THE DISCIPLINE

Semester 8 (4.2) — 16 weeks | Credit with Grade

Module	Topic	Control Type	Form of Control	Min	Max	Week
M1	BC-1: Introduction to Endocrinology. Diabetes Mellitus: classification, aetiology, pathogenesis, clinical picture, 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 Type 1 and Type 2 DM. Insulin therapy principles. Oral hypoglycaemic agents. Self-control. Bread unit calculation	Current	Frontal questioning, testing, prescription writing, attendance	2	4	5
M2		Midterm	Oral/written questioning, drug calculation task	6	10	

M3	BC-3: Micro- and macrovascular complications of DM. Diabetic retinopathy, nephropathy, neuropathy. Diabetic foot syndrome	Current	Frontal questioning, testing, practical skills, attendance	2	4	8
M3		Midterm BC-1 (Boundary Control No. 1)	Oral/written questioning, situational case, ECG/lab interpretation	6	10	
M4	BC-4: Acute complications of DM. Diabetic ketoacidosis. Hypoglycaemic coma. Hyperosmolar states. Lactic acidosis. Emergency management	Current	Frontal questioning, testing, emergency scenario, attendance	2	4	10
M4		Midterm	Oral/written questioning, emergency management case	6	10	
M5	BC-5: Thyroid diseases. Thyrotoxicosis syndrome. Hypothyroidism syndrome. Diffuse toxic goitre. Iodine deficiency diseases. Thyroiditis	Current	Frontal questioning, testing, practical skills, attendance	2	4	12
M5		Midterm	Oral/written questioning, situational case	6	10	
M6	BC-6: Parathyroid diseases. Adrenal diseases. Hypocorticism syndrome. Hypercorticism syndrome. Addison's disease. Cushing syndrome	Current	Frontal questioning, testing, lab interpretation, attendance	2	4	13
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, practical skills, attendance	2	4	14
M7		Midterm	Oral/written questioning, situational case	6	10	

M8	BC-8: Obesity. Metabolic syndrome. PCOS. Prevention. Dispensary observation. Final test	Current	Frontal questioning, testing, case history defense, attendance	2	4	16
M8		Midterm (Final)	Oral/written questioning, case history defense, prescription writing	6	10	
TOTAL				**40**	**70**	
Midterm Control	Credit with Grade			**20**	**30**	
Semester Rating				**60**	**100**	

6. PATIENT SUPERVISION AND CASE HISTORY SECTION

6.1. General Requirements

Each student receives one patient with endocrine pathology for supervision. The student conducts an interview and physical examination, reviews laboratory and instrumental results, and fills in the medical history according to the established scheme. The case history is the main legal and medical document and must be completed in accordance with MoH KR orders.

6.2. Supervision Scheme (Curation Scheme)

- Passport details (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, reasons for hospitalisation
- Life history (brief): past diseases, heredity, social history
- Objective examination data: summary of pathological findings by system and organ; clinical description of the endocrine system
- Laboratory and instrumental data: interpretation of available results
- Diagnosis and conclusion on supervision

6.3. Structure of Educational Case History (Diabetes Mellitus Section)

Section	Content	Requirements	Points
Title Page	Full name, age, sex, diagnosis, date of admission, ward, case number	Correct filling of all requisites	5
Complaints	Main endocrine complaints; secondary complaints	Complete sequential presentation; onset and duration	10
Disease History	Development of endocrine disease from onset to admission	Chronological sequence; link to risk factors	15

Life History	Past diseases, heredity, lifestyle, social history	Risk factor assessment; family endocrine history	10
Objective Status	Physical examination including endocrine system assessment	Systematic examination; accurate formulations	20
Clinical Diagnosis	Structured diagnosis with complications; ICD-10 code	Logical justification; nosological and syndrome components	20
Examination Plan	Laboratory and instrumental methods	Justification; correspondence to diagnosis	10
Treatment Plan	Drug and non-drug therapy; insulin/drug dosing	Evidence-based; appropriate to disease stage	10

6.4. Procedure for Defending Case History

- Student observes the patient throughout the study period at the department
- Case history is drawn up in accordance with the established form
- Case history is defended in the form of a report with presentation of clinical case
- Assessment criteria: completeness of history (25%), quality of physical examination (25%), correctness of clinical diagnosis with ICD-10 code (25%), justification of examination and treatment plan (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 and guidelines	1 hour	Main and additional literature
Preparation for practical classes and patient supervision	2 hours	Key concepts; preparation for case analysis
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, continuous glucose monitoring systems, artificial pancreas.
- 4. Diabetic neuropathy. Diabetic foot syndrome: pathogenesis, classification, treatment.
- 5. Psychological aspects of DM: compliance, patient burnout syndrome, motivational counselling.
- 6. Thyroid diseases: thyroiditis — classification, diagnosis, principles of treatment.
- 7. Hyperparathyroidism as part of endocrine syndromes.
- 8. Diseases of the hypothalamic-pituitary region: growth disorders, somatotropic insufficiency.

- 9. Hyperprolactinaemia: differential diagnosis and treatment.
- 10. Obesity and metabolic syndrome: classification, role of insulin resistance, complications.
- 11. Polycystic ovary syndrome (PCOS): pathogenesis, clinical features, modern approaches to treatment.

8. TYPICAL ASSESSMENT TASKS WITH ANSWERS

All clinical cases and MCQ tests require formulation of a structured CLINICAL DIAGNOSIS including: nosological form, aetiology/type, severity/stage, complications, concomitant diseases — and ICD-10 coding.

CONTROL SECTION No. 1

Section 1: Diabetes Mellitus — Classification, Diagnosis, Clinical Picture

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions (3–4 selected):

1. What is the WHO 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 according to WHO and ADA 2020?
5. Describe the clinical picture of type 1 and type 2 DM.
6. What is HbA1c and what are its diagnostic targets?

MCQ Tests:

Q1. Patient, 24 years, acute onset of polyuria, polydipsia, weight loss 8 kg in 3 weeks, weakness. Fasting glucose 16.2 mmol/L. C-peptide undetectable. Anti-GAD antibodies positive. Which form of DM?

A) Type 2 diabetes mellitus
B) Type 1 diabetes mellitus (autoimmune)
C) MODY (maturity-onset diabetes of the young)
D) Steroid-induced diabetes
E) Gestational diabetes

CORRECT ANSWER: B) Type 1 diabetes mellitus (autoimmune)
Young patient + acute onset + polyuria/polydipsia/weight loss (classic hyperglycaemia triad) + undetectable C-peptide (absolute insulin deficiency) + positive anti-GAD antibodies (autoimmune marker) = Type 1 DM, autoimmune form. C-peptide reflects residual beta-cell function; its absence confirms total destruction.

Q2. Fasting plasma glucose on two occasions: 7.4 mmol/L and 7.6 mmol/L. HbA1c = 6.9%. Patient is asymptomatic, overweight. Which diagnosis meets WHO 2020 criteria?

A) Impaired fasting glucose (pre-diabetes)
B) Type 2 diabetes mellitus
C) Type 1 diabetes mellitus
D) Normal carbohydrate metabolism
E) Requires OGTT before diagnosis

CORRECT ANSWER: B) Type 2 diabetes mellitus
Fasting plasma glucose ≥ 7.0 mmol/L on two occasions = DM diagnosis without need for OGTT. HbA1c ≥ 6.5% additionally confirms the diagnosis. Asymptomatic overweight patient = typical Type 2 DM presentation. Both criteria (FPG and HbA1c) independently meet WHO/ADA diagnostic threshold.

Q3. OGTT: fasting glucose 5.8 mmol/L, 2-hour post-load glucose 8.4 mmol/L. HbA1c 5.9%. Which diagnosis?

A) Normal glucose tolerance
B) Impaired fasting glucose
C) Impaired glucose tolerance (pre-diabetes)
D) Type 2 diabetes mellitus
E) Reactive hypoglycaemia

CORRECT ANSWER: C) Impaired glucose tolerance (pre-diabetes)

OGTT 2-hour glucose 7.8–11.0 mmol/L = impaired glucose tolerance (IGT). Fasting glucose 5.8 (normal < 6.1) and HbA1c 5.9% (pre-diabetes range 5.7–6.4%). IGT represents a high-risk pre-diabetic state with ~30% 10-year progression to DM.

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient D., 19 years, admitted with 3-week history of polyuria (6–7 L/day), polydipsia, weight loss 10 kg, fatigue, blurred vision. No prior medical history. BMI 18.2 kg/m². Fasting glucose 18.6 mmol/L, HbA1c 10.8%, C-peptide 0.08 nmol/L (norm 0.27–1.27), anti-GAD antibodies positive. Urinalysis: glucosuria 4+, ketonuria 2+.

Questions:

7. Formulate the clinical diagnosis with ICD-10 code. (5 points)
8. What additional examinations are required? (5 points)
9. Compose a treatment plan for this patient. (10 points)

ANSWERS:

1. Clinical diagnosis: Type 1 Diabetes Mellitus, newly diagnosed (autoimmune form). Severity: moderate–severe. Compensation: decompensated (HbA1c 10.8%). Complications: diabetic ketosis (ketonuria 2+). ICD-10: E10.9 (Type 1 DM without complications, or E10.1 with ketoacidosis if criteria met). Justification: young patient, acute onset, absolute insulin deficiency (C-peptide 0.08 nmol/L, undetectable), autoimmune markers (anti-GAD+), hyperglycaemia 18.6 mmol/L, HbA1c 10.8%.

2. Additional examinations: complete blood count (assess for infection as trigger); electrolytes (Na+, K+, bicarbonate — ketosis/acidosis assessment); arterial blood gas (pH, HCO3 — ketoacidosis severity); blood urea and creatinine (renal function baseline); lipid profile (dyslipidaemia associated with DM); thyroid function (TSH — autoimmune association with Type 1 DM); coeliac antibodies (anti-tTG — autoimmune cluster); ophthalmological examination (initial fundus assessment); urine albumin/creatinine ratio (nephropathy baseline).

3. Treatment plan: (1) Insulin therapy — intensive basal-bolus regimen: basal insulin (glargine/detemir) 0.2–0.3 U/kg/day at bedtime + rapid-acting insulin (lispro/aspart) before each meal based on carbohydrate content (1 unit per 10 g carbohydrates initially). (2) Target glycaemia: pre-meal 4–7 mmol/L, post-meal < 10 mmol/L, HbA1c < 7.0%. (3) Diet: carbohydrate counting using bread units; regular meals. (4) Self-monitoring: blood glucose 4–6 times daily. (5) Education: diabetes school programme (hypoglycaemia recognition, sick-day rules, injection technique). (6) Monitor ketones until cleared. Ketoacidosis prevention: adequate insulin, hydration. (7) Follow-up: every 3 months (HbA1c, renal function, lipids); annual ophthalmological examination.

CONTROL SECTION No. 2

Section 2: Treatment of DM. Insulin Therapy. Oral Hypoglycaemic Agents

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

10. What are the principles of insulin therapy in type 1 DM?
11. What are the indications for insulin therapy in type 2 DM?
12. Classify oral hypoglycaemic agents (OHAs) and their mechanisms of action.
13. What is a bread unit (BU) and how is it used in meal planning?
14. What is the self-monitoring algorithm for a patient with DM?
15. What are the principles of lifestyle modification in type 2 DM?

MCQ Tests:

Q1. Patient with Type 2 DM, BMI 31 kg/m², eGFR 78 mL/min/1.73m², no cardiovascular disease. HbA1c 8.2% on diet alone. Which first-line pharmacological agent?

A) Sulphonylurea (glibenclamide)
B) Metformin
C) Insulin glargine
D) SGLT-2 inhibitor (dapagliflozin)
E) DPP-4 inhibitor (sitagliptin)

CORRECT ANSWER: B) Metformin
Metformin is the first-line agent for Type 2 DM in the absence of contraindications (eGFR ≥ 45 mL/min, no lactic acidosis risk). It reduces hepatic glucose production, improves insulin sensitivity, is weight-neutral, and has the strongest evidence base for cardiovascular protection. ADA Standards 2020 recommend metformin as initial therapy.

Q2. Patient receives: breakfast 60 g carbohydrates, lunch 90 g carbohydrates, dinner 60 g carbohydrates. Carbohydrate-to-insulin ratio = 1 unit per 10 g. How many units of rapid-acting insulin are needed for lunch?

A) 6 units
B) 9 units
C) 10 units
D) 12 units
E) 15 units

CORRECT ANSWER: B) 9 units
Calculation: 90 g carbohydrates ÷ 10 g/unit = 9 units of rapid-acting insulin for lunch. This is the carbohydrate-counting method used in intensive insulin therapy. 90 g ÷ 10 = 9 BU, each BU requiring 1 unit = 9 units total. This approach allows flexible meal planning while maintaining glycaemic targets.

Q3. Patient with Type 2 DM has eGFR 28 mL/min/1.73m², HbA1c 9.1%. Which drugs are CONTRAINDICATED?

A) Insulin + SGLT-2 inhibitor
B) Metformin + SGLT-2 inhibitor
C) GLP-1 agonist + DPP-4 inhibitor
D) Insulin glargine + glulisine
E) DPP-4 inhibitor (dose-adjusted) + insulin

CORRECT ANSWER: B) Metformin + SGLT-2 inhibitor
Metformin is contraindicated when eGFR < 30 mL/min (risk of lactic acidosis). SGLT-2 inhibitors lose efficacy and safety when eGFR < 30 mL/min (insufficient glucose filtration, increased UTI/DKA risk). Both are contraindicated in severe CKD. Insulin with dose-adjusted DPP-4 inhibitor remains an option.

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient V., 52 years, Type 2 DM diagnosed 3 years ago. On metformin 2000 mg/day. HbA1c 9.4%, fasting glucose 11.2 mmol/L, postprandial 15.6 mmol/L. BMI 29 kg/m², eGFR 65 mL/min, BP 138/86 mmHg. No cardiovascular events. Urinary albumin 42 mg/g (microalbuminuria).

Questions:

16. Assess compensation of DM. Justify intensification of therapy. (5 points)
17. Which additional drug should be added? Justify the choice. (5 points)
18. Compose a comprehensive treatment plan. (10 points)

ANSWERS:

1. Assessment of compensation: HbA1c 9.4% — decompensated (target < 7.0–7.5% for age 52). Fasting glucose 11.2 mmol/L (target 4–7) and postprandial 15.6 mmol/L (target < 10) — both above targets. Therapy intensification is mandatory: dual OHA combination or transition to triple therapy.

2. Drug choice: Add an SGLT-2 inhibitor (dapagliflozin/empagliflozin). Justification: (a) eGFR 65 — within safe range (≥45); (b) microalbuminuria present — SGLT-2 inhibitors have nephroprotective effect (DAPA-CKD, EMPA-REG OUTCOME trials); (c) BMI 29 — weight reduction benefit; (d) BP 138/86 — mild antihypertensive effect. Alternative: GLP-1 receptor agonist (liraglutide) — cardiovascular and renal protection, weight loss.

3. Comprehensive treatment plan: (1) Continue metformin 2000 mg/day. (2) Add dapagliflozin 10 mg/day (or empagliflozin 10 mg/day) — nephroprotection, glucose reduction, mild BP reduction, weight reduction. (3) RAAS inhibitor: add ACE inhibitor (lisinopril 5–10 mg/day) or ARB — microalbuminuria is an indication. (4) Target BP < 130/80 mmHg. (5) Dietary modification: caloric restriction 500 kcal/day deficit; carbohydrate distribution; low sodium. (6) Physical activity: 150 min/week moderate aerobic exercise. (7) Self-monitoring: 2–3 blood glucose measurements/day. (8) HbA1c control every 3 months. (9) Annual: ophthalmological examination, monofilament foot examination, lipid profile, urine albumin/creatinine ratio.

CONTROL SECTION No. 3

Section 3: Micro- and Macrovascular Complications of DM

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

19. Classify microvascular complications of DM and their pathogenesis.
20. Describe the classification and diagnosis of diabetic retinopathy.
21. Describe the classification and diagnosis of diabetic nephropathy.
22. Describe the classification and diagnosis of diabetic neuropathy.
23. What are the features of IHD and arterial hypertension in DM?
24. Describe diabetic foot syndrome: classification and management principles.

MCQ Tests:

Q1. Patient with Type 1 DM (12 years duration). Urinary albumin/creatinine ratio 185 mg/g, eGFR 62 mL/min, BP 152/94 mmHg. Which stage of diabetic nephropathy?

A) Hyperfiltration stage
B) Stage of microalbuminuria (incipient nephropathy)
C) Stage of macroalbuminuria (overt nephropathy)
D) Stage of renal failure
E) Normal renal function

CORRECT ANSWER: C) Stage of macroalbuminuria (overt nephropathy)
Urinary albumin/creatinine ratio > 300 mg/g = macroalbuminuria = overt diabetic nephropathy (Stage 3–4 by IDF classification, CKD Stage G2–A3). eGFR 62 = CKD Stage G2 (mildly reduced). Hypertension is both a cause and consequence of nephropathy progression.

Q2. Patient with DM complains of burning pain and numbness in feet, worse at night, with relative relief on walking. Monofilament test: absent 10-g sensation bilaterally at great toe. Which complication?

A) Peripheral arterial disease
B) Distal symmetric sensorimotor diabetic neuropathy
C) Mononeuritis (focal neuropathy)
D) Charcot arthropathy
E) Plantar fasciitis

CORRECT ANSWER: B) Distal symmetric sensorimotor diabetic neuropathy
Burning pain + numbness + nocturnal worsening + relief on walking (characteristic of neuropathic pain) + absent monofilament sensation (protective sensation loss) = distal symmetric polyneuropathy (DSP), the most common form of diabetic neuropathy. Loss of 10-g monofilament sensation is a key indicator of high-risk foot.

Q3. Patient with Type 2 DM. Ophthalmological examination: multiple flame haemorrhages, hard exudates, and neovascularisation at the disc and elsewhere. Which stage of retinopathy?

A) No diabetic retinopathy
B) Mild non-proliferative diabetic retinopathy
C) Moderate non-proliferative diabetic retinopathy
D) Severe non-proliferative diabetic retinopathy
E) Proliferative diabetic retinopathy

CORRECT ANSWER: E) Proliferative diabetic retinopathy

Neovascularisation (new vessel formation at disc or elsewhere) defines PROLIFERATIVE diabetic retinopathy (PDR) — the most advanced stage with risk of vitreous haemorrhage and traction retinal detachment. This stage requires urgent panretinal laser photocoagulation (PRP) or anti-VEGF treatment.

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient B., 48 years, Type 2 DM for 11 years. HbA1c 9.8%. Complaints: burning pain and numbness in feet, impotence, diarrhoea (alternating with constipation). Examination: absent Achilles reflexes bilaterally; vibration sensation absent below knees; dry cracked skin on feet. BP 158/96 mmHg. eGFR 48 mL/min. Albuminuria 380 mg/g. Fundoscopy: multiple haemorrhages, hard exudates — no neovascularisation.

Questions:

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

ANSWERS:

1. Complications: (a) Distal symmetric sensorimotor polyneuropathy — burning pain, numbness, absent reflexes, absent vibration, dry feet; (b) Autonomic neuropathy — impotence (erectile dysfunction syndrome), gastrointestinal autonomic neuropathy (alternating diarrhoea/constipation); (c) Diabetic nephropathy — macroalbuminuria 380 mg/g + eGFR 48 (CKD Stage G3a) + hypertension; (d) Non-proliferative diabetic retinopathy (moderate–severe) — haemorrhages, exudates, no neovascularisation. Diagnosis: Type 2 DM, decompensated (HbA1c 9.8%). Complications: distal sensorimotor polyneuropathy + autonomic neuropathy (erectile, GI) + diabetic nephropathy CKD G3a A3 + non-proliferative diabetic retinopathy. ICD-10: E11.40, E11.65, E11.21.

2. Urgent examinations: ABI (ankle-brachial index) — peripheral arterial disease; nerve conduction studies (NCS/EMG) — quantify neuropathy; HbA1c (already noted); lipid profile (dyslipidaemia management); electrolytes + bicarbonate (CKD G3); 24-hour blood pressure monitoring; ophthalmological examination with fluorescein angiography — more detailed retinopathy staging; Doppler ultrasound of renal arteries — renovascular hypertension.

3. Management plan: (1) Glycaemic control: intensify therapy — add/switch to insulin (eGFR 48 limits SGLT-2 and metformin use); target HbA1c 7.5–8.0% (CKD patient, less stringent target). (2) Nephroprotection: ACE inhibitor or ARB (mandatory for macroalbuminuria + hypertension); target BP < 130/80 mmHg; low-protein diet (0.8 g/kg/day); avoid nephrotoxic agents. (3) Neuropathy treatment: pregabalin or duloxetine for neuropathic pain; alpha-lipoic acid (antioxidant); good glycaemic control slows progression. (4) Retinopathy: urgent ophthalmological consultation; laser photocoagulation if indicated. (5) Erectile dysfunction: PDE-5 inhibitor (sildenafil) after cardiovascular risk assessment. (6) Dyslipidaemia: statin therapy (all DM + CKD = high cardiovascular risk). (7) Foot care: daily inspection; therapeutic footwear; podiatry referral.

CONTROL SECTION No. 4

Section 4: Acute Complications of DM. Emergency Management

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

28. Describe the pathogenesis and clinical picture of diabetic ketoacidosis (DKA).
29. What are the diagnostic criteria for DKA (mild, moderate, severe)?
30. Describe the treatment algorithm for DKA.
31. Describe hypoglycaemia and hypoglycaemic coma: aetiology, clinical picture, emergency management.
32. Describe hyperosmolar hyperglycaemic state (HHS): diagnosis and treatment.
33. Describe lactic acidosis in DM: causes, diagnosis, treatment.

MCQ Tests:

Q1. Patient with Type 1 DM. Glucose 24.8 mmol/L, pH 7.12, bicarbonate 8 mmol/L (norm 22–26), ketonuria 4+, stupor. Which acute complication and severity?

A) Hyperosmolar hyperglycaemic state
B) Lactic acidosis
C) Diabetic ketoacidosis — mild
D) Diabetic ketoacidosis — severe
E) Hypoglycaemic coma

CORRECT ANSWER: D) Diabetic ketoacidosis — severe
DKA diagnosis: glucose > 14 mmol/L + ketonuria/ketonaemia + metabolic acidosis (pH < 7.30). SEVERE DKA: pH < 7.10 (pH 7.12 is severe) + bicarbonate < 10 mmol/L (8 mmol/L) + altered consciousness (stupor). Mild DKA: pH 7.25–7.30; Moderate: pH 7.00–7.24; Severe: pH < 7.00 or severe neurological impairment.

Q2. Patient with DM on insulin develops: trembling, sweating, palpitations, hunger. Glucose 2.6 mmol/L. Patient is conscious and can swallow. First action?

A) Intravenous 40% glucose 40–60 mL
B) Intramuscular glucagon 1 mg
C) 15–20 g of fast-acting oral carbohydrates (Rule of 15)
D) Intravenous 5% glucose infusion
E) Reduce insulin dose and observe

CORRECT ANSWER: C) 15–20 g of fast-acting oral carbohydrates (Rule of 15)
Mild–moderate hypoglycaemia in a CONSCIOUS patient who can swallow = ORAL carbohydrates FIRST. Rule of 15: give 15–20 g fast-acting carbohydrates (4 glucose tablets, 150 mL fruit juice, 3 tsp sugar), recheck glucose after 15 minutes, repeat if still < 4 mmol/L. IV glucose is for unconscious patients or those unable to swallow.

Q3. Patient with Type 2 DM. Glucose 48 mmol/L, serum osmolality 340 mOsm/kg, no significant ketonuria, pH 7.32. Confused, dehydrated. Which condition?

A) Diabetic ketoacidosis — severe
B) Hyperosmolar hyperglycaemic state (HHS)
C) Lactic acidosis
D) Type 1 DM decompensation
E) Hypoglycaemia with rebound hyperglycaemia

CORRECT ANSWER: B) Hyperosmolar hyperglycaemic state (HHS)

HHS criteria: glucose > 33 mmol/L (48 mmol/L) + effective osmolality > 320 mOsm/kg (340) + absence of significant ketonaemia/acidosis (pH 7.32, no ketonuria) + altered consciousness. Typical in elderly Type 2 DM patients. Marked dehydration (>8 L deficit typical) is the hallmark. Mortality higher than DKA.

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient T., 22 years, Type 1 DM. Brought by ambulance: unconscious, vomiting, deep Kussmaul breathing, acetone breath. Last insulin injection 2 days ago (deliberately omitted). Glucose 31.4 mmol/L. Blood gas: pH 7.06, HCO3 6 mmol/L, pCO2 18 mmHg. Ketonuria 4+. Na+ 132 mmol/L, K+ 5.8 mmol/L (before treatment).

Questions:

34. Establish diagnosis with severity. (5 points)
35. Describe the immediate emergency management algorithm. (5 points)
36. Compose a detailed treatment plan for the first 24 hours. (10 points)

ANSWERS:

1. Diagnosis: Diabetic Ketoacidosis (DKA), SEVERE. Criteria: glucose 31.4 mmol/L, pH 7.06 (< 7.10 = severe), HCO3 6 mmol/L (< 10 = severe), Kussmaul breathing (compensatory respiratory alkalosis, pCO2 18), acetone breath (ketonaemia), ketonuria 4+, unconsciousness. Precipitant: insulin omission. ICD-10: E10.1.

2. Immediate emergency management: (1) Airway — protect airway; positioning to prevent aspiration; nasogastric tube if vomiting persists. (2) IV access — two wide-bore cannulae. (3) Monitoring — continuous cardiac monitoring (hypokalaemia risk during treatment), hourly glucose, 2-hourly electrolytes. (4) Do NOT give bicarbonate routinely (pH 7.06 — bicarbonate use controversial, some guidelines: consider if pH < 6.9). (5) Treat precipitating cause (infection? — blood cultures, CXR, urine culture).

3. 24-hour treatment plan: FLUIDS: 0.9% NaCl — first hour: 1000 mL; hours 2–4: 500 mL/h; subsequent: 250 mL/h (total 4–6 L in 24 h) — aim for gradual glucose drop (3–4 mmol/L/h, not faster). When glucose reaches 14 mmol/L: switch to 5% glucose + 0.45% NaCl. INSULIN: fixed-rate IV insulin infusion 0.1 U/kg/h (minimum 6 U/h) after fluid resuscitation begins. Continue until ketonaemia/ketonuria resolves (not just glucose normalises). Switch to subcutaneous insulin when patient eating and pH > 7.3. POTASSIUM: K+ 5.8 pre-treatment — do NOT add K+ initially. K+ falls during treatment (insulin drives K+ intracellular) — reassess every 2 hours; add when K+ < 5.0: 20–40 mmol/h. Target K+ 4.0–5.0 mmol/L throughout. MONITORING: glucose hourly; electrolytes (Na+, K+, HCO3) every 2 hours; blood gas every 4 hours; urine output hourly; clinical assessment hourly. RESOLUTION criteria: glucose < 12 mmol/L + pH > 7.3 + HCO3 > 18 mmol/L + clear mentation.

CONTROL SECTION No. 5

Section 5: Thyroid Diseases. Thyrotoxicosis and Hypothyroidism Syndromes

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

37. Describe the classification of thyroid diseases.
38. Describe thyrotoxicosis syndrome: aetiology, clinical picture, diagnosis.
39. What are the treatment methods for diffuse toxic goitre (Graves' disease)?
40. Describe hypothyroidism: classification, aetiology, clinical picture, diagnosis, treatment.
41. Describe iodine deficiency diseases: classification, prevention.
42. Describe thyroiditis: classification, clinical features, treatment.

MCQ Tests:

Q1. Female, 34 years, weight loss 12 kg in 4 months, palpitations, hand tremor, excessive sweating, irritability, exophthalmos. Free T4 52 pmol/L (norm 11–22), TSH 0.01 mIU/L (norm 0.4–4.0), TRAb positive. Which diagnosis and ICD-10 code?

A) Hypothyroidism — E03.9
B) Autoimmune thyroiditis (euthyroid phase) — E06.3
C) Diffuse toxic goitre (Graves' disease) — E05.0
D) Toxic multinodular goitre — E05.2
E) Subacute thyroiditis — E06.1

CORRECT ANSWER: C) Diffuse toxic goitre (Graves' disease) — E05.0
Classic Graves' disease: (1) thyrotoxicosis symptoms (weight loss, tachycardia, tremor, hyperhidrosis, irritability); (2) exophthalmos (Graves' ophthalmopathy, TSH receptor antibodies in orbital tissue); (3) Free T4 elevated + TSH suppressed (primary hyperthyroidism); (4) TRAb positive (pathognomonic for Graves' disease). ICD-10: E05.0 — Thyrotoxicosis with diffuse goitre.

Q2. Patient, 62 years, progressive fatigue, cold intolerance, weight gain 8 kg, constipation, dry skin, slow speech. TSH 28 mIU/L (norm 0.4–4.0), Free T4 5.2 pmol/L (norm 11–22). Which diagnosis?

A) Primary hypothyroidism — E03.9
B) Secondary (central) hypothyroidism — E03.1
C) Subclinical hypothyroidism — E02
D) Sick euthyroid syndrome
E) Hyperthyroidism with high TSH

CORRECT ANSWER: A) Primary hypothyroidism — E03.9
Primary hypothyroidism: TSH markedly elevated (28 mIU/L — the pituitary compensates for low TH) + Free T4 markedly low (5.2 pmol/L) = primary thyroid gland failure. Classic clinical picture: cold intolerance, weight gain, constipation, dry skin (myxoedema), slow speech (bradylalia). Secondary hypothyroidism would have LOW TSH (pituitary failure).

Q3. Thionamide therapy (methimazole 30 mg/day) for Graves' disease, Week 6. Patient presents with sudden fever 38.8°C, severe sore throat, difficulty swallowing. Urgent test?

A) Thyroid function tests (TSH, Free T4)
B) Complete blood count (CBC) — URGENT
C) TRAb titre
D) Thyroid ultrasound
E) Urine culture

CORRECT ANSWER: B) Complete blood count (CBC) — URGENT
Sudden fever + severe sore throat in a patient on thionamide = AGRANULOCYTOSIS until proven otherwise (life-threatening side effect, incidence 0.2–0.5%). CBC must be obtained IMMEDIATELY. If neutrophil count < 0.5 × 10⁹/L = agranulocytosis — stop methimazole immediately, hospitalise, give G-CSF, broad-spectrum antibiotics. This is a medical emergency.

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient M., 28 years (female), 6-month history of palpitations (HR 118 bpm), weight loss 9 kg, hand tremor, irritability, excessive sweating. 3 months ago noticed eye prominence. Thyroid gland: diffusely enlarged (Grade II), soft, bruit on auscultation. Free T4 68 pmol/L, TSH < 0.01 mIU/L, TRAb 18.4 IU/L (norm < 1.75). ECG: sinus tachycardia 118 bpm.

Questions:

43. Formulate the clinical diagnosis with ICD-10 code. (5 points)
44. Which additional examinations are needed? (5 points)
45. Compose a treatment plan. (10 points)

ANSWERS:

1. Diagnosis: Diffuse Toxic Goitre (Graves' Disease), Grade II, with severe thyrotoxicosis. Endocrine ophthalmopathy (Graves' ophthalmopathy). ICD-10: E05.0 (Thyrotoxicosis with diffuse goitre). Justification: diffuse goitre Grade II + thyrotoxicosis syndrome (tachycardia, weight loss, tremor, hyperhidrosis, irritability) + primary hyperthyroidism (Free T4 68, TSH suppressed) + TRAb markedly positive (18.4, pathognomonic) + exophthalmos.

2. Additional examinations: thyroid ultrasound with Doppler (goitre volume, vascularity — Graves' pattern: markedly increased blood flow); thyroid scintigraphy (if diagnosis uncertain — diffuse increased uptake in Graves); CBC (baseline before thionamide, agranulocytosis monitoring); liver function tests (baseline, thionamides hepatotoxic); thyroid antibodies (anti-TPO, anti-thyroglobulin — autoimmune profile); ophthalmological examination (Graves' ophthalmopathy staging by VISA/CAS score); echocardiography (tachycardia 118 — assess for thyrotoxic cardiomyopathy); bone densitometry (hyperthyroidism causes bone loss).

3. Treatment plan: (1) THIONAMIDE: Methimazole (thiamazole) 30–40 mg/day in divided doses. Start block-and-replace after 4 weeks if indicated. Monitor TFTs every 4–6 weeks. (2) SYMPTOM CONTROL: Beta-blocker — propranolol 40 mg 3 times/day (controls tachycardia, tremor, sweating, anxiety). Continue until euthyroid state achieved. (3) MONITORING: CBC at every visit (agranulocytosis); liver function tests monthly; Free T4 + TSH every 4–6 weeks. (4) DURATION: Thionamide for 12–18 months, then attempt withdrawal and check for remission (30–50% remission rate). (5) DEFINITIVE TREATMENT (if relapse or preference): Radioactive iodine (RAI, I-131) — first-line definitive treatment; or thyroidectomy (if very large goitre, compression, patient preference). NOTE: RAI may worsen ophthalmopathy — steroid cover required. (6) OPHTHALMOPATHY: Ophthalmological consultation; selenium supplementation (mild GO); systemic steroids or decompression (moderate-severe GO). Achieve euthyroid state as rapidly as possible.

CONTROL SECTION No. 6

Section 6: Adrenal Gland Diseases. Hypocorticism and Hypercorticism Syndromes

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

46. Describe the aetiology, pathogenesis and classification of chronic adrenal insufficiency.
47. Describe the clinical picture and differential diagnosis of primary and secondary adrenal insufficiency.
48. Describe acute adrenal insufficiency (Addisonian crisis): diagnosis and emergency treatment.
49. Describe hypercorticism syndrome: aetiology, classification, clinical picture.
50. What are the diagnostic criteria for Cushing's syndrome?
51. What is the differential diagnosis of Cushing's disease vs Cushing's syndrome?

MCQ Tests:

Q1. Patient, 42 years, progressive weakness, weight loss, hyperpigmentation of skin and mucous membranes, hypotension 90/60 mmHg, nausea, salt craving. Laboratory: Na+ 128 mmol/L, K+ 5.9 mmol/L, cortisol 8 nmol/L (norm 140–690 AM), ACTH 890 pg/mL (norm 10–46). Which diagnosis?

A) Secondary adrenal insufficiency
B) Cushing's syndrome
C) Primary chronic adrenal insufficiency (Addison's disease)
D) Hypothyroidism
E) Pituitary insufficiency (hypopituitarism)

CORRECT ANSWER: C) Primary chronic adrenal insufficiency (Addison's disease)
Classic Addison's: (1) hyperpigmentation (elevated ACTH stimulates melanocytes — only in PRIMARY); (2) hypotension (aldosterone deficiency + cortisol deficiency); (3) hyponatraemia + hyperkalaemia (aldosterone deficiency); (4) very low cortisol (8 nmol/L) + very HIGH ACTH (890 pg/mL) = primary failure (intact pituitary overcompensates). Salt craving = physiological response to aldosterone deficiency. ICD-10: E27.1.

Q2. Patient with Addison's disease on hydrocortisone 20 mg/day develops severe infection (pneumonia). She vomits and cannot take oral medication. BP 80/50. Which immediate action?

A) Double oral hydrocortisone dose
B) IV hydrocortisone 100 mg bolus immediately
C) IV fludrocortisone only
D) IV dexamethasone 4 mg
E) Oral prednisolone 40 mg

CORRECT ANSWER: B) IV hydrocortisone 100 mg bolus immediately
Addisonian crisis = life-threatening emergency. Sick-day rules: if unable to take oral medications + vomiting = inject hydrocortisone 100 mg IM/IV IMMEDIATELY. Follow with hydrocortisone 100 mg IV every 6–8 hours + IV 0.9% NaCl + 5% glucose. Dexamethasone lacks mineralocorticoid activity — not appropriate for acute crisis. Fludrocortisone alone is insufficient. This is not a dose-doubling situation.

Q3. Patient, 35 years, central obesity (buffalo hump, moon face, supraclavicular fat pad), purple striae, proximal myopathy, hypertension, glucose 8.9 mmol/L fasting. 24-hour urine cortisol 4 times upper limit of normal. ACTH 85 pg/mL (norm 10–46). MRI pituitary: 6 mm microadenoma. Which form of hypercorticism?

A) Cushing's syndrome from adrenal adenoma (ACTH-independent)
B) Ectopic ACTH syndrome
C) Cushing's disease (ACTH-dependent, pituitary)

D) Iatrogenic Cushing's syndrome
E) Pseudo-Cushing's syndrome

CORRECT ANSWER: C) Cushing's disease (ACTH-dependent, pituitary)
Cushing's DISEASE = pituitary ACTH-secreting adenoma: (1) elevated cortisol (24h UFC ×4 ULN); (2) elevated ACTH (85 pg/mL — confirms ACTH-dependent); (3) MRI pituitary microadenoma (6 mm). Cushing's SYNDROME from adrenal adenoma = ACTH-independent (suppressed ACTH). Ectopic ACTH = very HIGH ACTH (typically >200 pg/mL), aggressive tumour, more severe hypokalaemia.

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient A., 38 years (female), 2-year history of weight gain (especially central), purple striae on abdomen and thighs, hirsutism, menstrual irregularity (oligomenorrhoea), proximal muscle weakness, depressive mood. BP 168/100 mmHg. Fasting glucose 9.2 mmol/L. Laboratory: 24h urinary free cortisol 5.8 × ULN; late-night salivary cortisol elevated on 2 occasions; ACTH 92 pg/mL. Low-dose dexamethasone suppression test (LDDST): cortisol not suppressed (28 nmol/L). MRI pituitary: 8 mm adenoma right side.

Questions:

52. Establish diagnosis with ICD-10 code. (5 points)
53. Which additional examinations confirm the diagnosis? (5 points)
54. Compose a treatment plan. (10 points)

ANSWERS:

1. Diagnosis: Cushing's Disease (pituitary ACTH-secreting adenoma). Moderate severity. Complications: arterial hypertension, impaired fasting glucose (steroid diabetes risk), hirsutism, menstrual dysfunction, proximal myopathy, mood disorder. ICD-10: E24.0 (Pituitary-dependent Cushing's disease). Justification: central obesity + purple striae + proximal myopathy + hypertension + hyperglycaemia (hypercorticism syndrome) + 24h UFC ×5.8 + late-night salivary cortisol elevated (loss of normal circadian rhythm) + LDDST non-suppressed + ACTH 92 (elevated = ACTH-dependent) + pituitary adenoma on MRI.

2. Confirmatory examinations: High-dose dexamethasone suppression test (HDDST: 2 mg × 8 doses) — Cushing's DISEASE typically suppresses >50%, ectopic ACTH does not; inferior petrosal sinus sampling (IPSS) — gold standard for lateralisation and confirmation of pituitary source (ACTH gradient > 2 central:peripheral confirms pituitary); CRH stimulation test (exaggerated ACTH response in pituitary disease); bone densitometry (osteoporosis assessment); cardiac evaluation (hypertensive target organ damage); complete coagulation (hypercoagulability in Cushing's).

3. Treatment plan: (1) DEFINITIVE: Transsphenoidal surgical resection of pituitary adenoma — first-line treatment for Cushing's disease. Success rate 70–90% for microadenomas. (2) PRE-SURGICAL PREPARATION: Steroidogenesis inhibitors — metyrapone (500 mg 3 times/day, titrate) or ketoconazole — reduce cortisol to safe levels before surgery. (3) POST-SURGICAL MONITORING: Cortisol measured every 6 hours after surgery; if <50 nmol/L = remission. Hydrocortisone replacement (20 mg/day) until HPA axis recovers (6–24 months). (4) IF SURGERY FAILS OR RECURS: Repeat surgery; radiotherapy (stereotactic); bilateral adrenalectomy (curative but permanent adrenal insufficiency + risk of Nelson's syndrome). (5) COMORBIDITIES: Antihypertensive therapy; metformin or insulin for steroid diabetes; calcium + vitamin D + bisphosphonate for osteoporosis; antidepressants for mood disorder. (6) FOLLOW-UP: MRI pituitary 3 months post-op; HbA1c and BP control.

CONTROL SECTION No. 7

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

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

55. Describe acromegaly: aetiology, pathogenesis, clinical picture, diagnostic criteria.
56. Describe diabetes insipidus: aetiology, clinical picture, differential diagnosis of central vs nephrogenic.
57. Describe hypopituitarism: aetiology, clinical features of each axis involvement.
58. Describe hyperprolactinaemia: aetiology, clinical picture, treatment principles.
59. What are the principles of growth hormone replacement therapy in somatotropic insufficiency?
60. Describe the main diseases of the hypothalamic-pituitary region and their diagnostic algorithms.

MCQ Tests:

Q1. Patient, 44 years, noted coarsening of facial features over 8 years (enlarged nose, lips, jaw, hands and feet), increased ring and shoe size. Glucose 10.4 mmol/L. IGF-1 4.5 × ULN. Random GH 28 µg/L (norm < 1 after OGTT). MRI pituitary: 2.4 cm macroadenoma. Which diagnosis?

A) Hyperprolactinaemia
B) Acromegaly — E22.0
C) Hypothyroidism
D) Cushing's disease
E) Gigantism

CORRECT ANSWER: B) Acromegaly — E22.0
Acromegaly (adult onset = after epiphyseal fusion): acral enlargement + coarsening of features + IGF-1 markedly elevated + GH not suppressed after OGTT (normal < 1 µg/L at nadir) + pituitary macroadenoma. IGF-1 is the best SCREENING test (reflects 24h GH secretion). Glucose intolerance/DM is a common complication (GH is insulin-antagonistic). ICD-10: E22.0.

Q2. Patient complains of polyuria 8–10 L/day, polydipsia. Urine osmolality 85 mOsm/kg (norm 50–1200). Plasma osmolality 305 mOsm/kg. After desmopressin administration: urine osmolality increases to 620 mOsm/kg. Which diagnosis?

A) Type 2 diabetes mellitus
B) Nephrogenic diabetes insipidus
C) Central diabetes insipidus
D) Primary polydipsia (psychogenic)
E) SIADH

CORRECT ANSWER: C) Central diabetes insipidus
Central DI: polyuria with very dilute urine (osmolality 85) + elevated plasma osmolality (hyperosmolality from water loss) + EXCELLENT response to desmopressin (urine osmolality ×7 increase = kidneys can concentrate when given ADH) = central (pituitary) ADH deficiency. Nephrogenic DI: minimal or no response to desmopressin. Primary polydipsia: normal urine concentration after water deprivation.

Q3. Young woman, 26 years, galactorrhoea, oligomenorrhoea, infertility for 2 years. Prolactin 142 µg/L (norm < 20). MRI pituitary: 11 mm microadenoma. Which first-line treatment?

A) Transsphenoidal surgery
B) Radiotherapy
C) Cabergoline (dopamine agonist)
D) Bromocriptine only (cabergoline not available)

E) Combined oral contraceptive pill

CORRECT ANSWER: C) Cabergoline (dopamine agonist)
Prolactinoma (prolactin-secreting pituitary adenoma) — first-line treatment is MEDICAL: cabergoline 0.5–2 mg twice weekly. Dopamine agonists normalise prolactin, restore menstrual cycles/fertility, and reduce tumour size in >80% of cases. Surgery is reserved for drug intolerance, resistance, or compressive emergencies. Cabergoline is preferred over bromocriptine (better tolerability, once or twice weekly dosing, higher efficacy).

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient G., 50 years, 10-year history of increasing hand and foot size (shoe size +3, ring size +4), coarsening facial features, macroglossia, hyperhidrosis, joint pains, snoring (suspected sleep apnoea). Glucose 11.8 mmol/L (fasting). BP 152/96 mmHg. IGF-1 ×3.5 ULN. GH during OGTT: nadir 8.6 μg/L (not suppressed). MRI pituitary: 1.8 cm macroadenoma, no optic chiasm compression.

Questions:

61. Formulate the clinical diagnosis with ICD-10 and complications. (5 points)
62. Which additional examinations are needed? (5 points)
63. Compose a treatment plan. (10 points)

ANSWERS:

1. Diagnosis: Acromegaly due to GH-secreting pituitary macroadenoma (1.8 cm), active phase. Complications: secondary diabetes mellitus (fasting glucose 11.8 mmol/L); arterial hypertension; articular syndrome (acromegalic arthropathy); macroglossia; suspected obstructive sleep apnoea syndrome. ICD-10: E22.0 (Acromegaly and pituitary gigantism). Justification: progressive acral enlargement + IGF-1 ×3.5 ULN + GH non-suppressed after OGTT (nadir 8.6, norm < 1) + pituitary macroadenoma confirmed on MRI.

2. Additional examinations: visual field testing (Goldmann/Humphrey perimetry) — assess optic chiasm proximity; echocardiography (acromegalic cardiomyopathy — biventricular hypertrophy); colonoscopy (increased colorectal cancer risk in acromegaly); polysomnography (sleep apnoea quantification); bone densitometry (osteoporosis); thyroid function + cortisol + gonadal axis (GH macroadenoma may compress other pituitary cells — assess for hypopituitarism); calcium and phosphorus (acromegaly causes hypercalciuria); prolactin (GH tumours may also secrete prolactin).

3. Treatment plan: (1) FIRST-LINE: Transsphenoidal surgical resection — preferred for macroadenoma without chiasm compression; remission criteria: GH < 1 μg/L after OGTT + normal IGF-1. (2) MEDICAL THERAPY (pre-surgery or if surgery incomplete/contraindicated): Somatostatin analogues — octreotide LAR 20–30 mg IM monthly or lanreotide autogel 90–120 mg SC monthly; reduce GH/IGF-1 in ~60% of patients; shrink tumour in ~20%. GH receptor antagonist — pegvisomant (if SSA ineffective): normalises IGF-1 in >90%. (3) RADIOTHERAPY: Stereotactic radiosurgery (Gamma Knife) — if surgery failed and medical therapy insufficient; effect delayed 5–10 years. (4) COMPLICATIONS MANAGEMENT: Metformin or insulin for DM; antihypertensive therapy; CPAP for sleep apnoea; orthopedic/rheumatological care for arthropathy; colorectal screening. (5) MONITORING: IGF-1 + GH every 6 months; MRI pituitary annually; visual fields; cardiovascular assessment.

CONTROL SECTION No. 8

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

BLOCK A — Reproductive Level | Time: 30 min

Oral Questions:

64. Describe the classification and pathogenesis of obesity. Role of insulin resistance.
65. What are the diagnostic criteria for metabolic syndrome (IDF 2006 criteria)?
66. Describe the clinical manifestations and complications of obesity.
67. What are the principles of drug and surgical treatment of obesity?
68. Describe PCOS: pathogenesis, clinical picture, modern approaches to treatment.
69. What are the principles of dispensary observation of endocrinological patients (DM, thyroid, adrenal)?

MCQ Tests:

Q1. Patient, 47 years, BMI 36.2 kg/m², waist circumference 108 cm (female). Fasting glucose 6.4 mmol/L, TG 2.8 mmol/L, HDL 0.9 mmol/L, BP 142/92 mmHg. Which diagnosis meets IDF metabolic syndrome criteria?

A) Obesity alone, no metabolic syndrome
B) Metabolic syndrome (abdominal obesity + ≥2 components)
C) Type 2 DM
D) Hypothyroidism mimicking metabolic syndrome
E) Pre-diabetes without metabolic syndrome

CORRECT ANSWER: B) Metabolic syndrome (abdominal obesity + ≥2 components)
IDF 2006 metabolic syndrome criteria: CENTRAL OBESITY (waist female > 80 cm — present at 108 cm) PLUS ≥2 of: (1) TG ≥ 1.7 mmol/L — present (2.8); (2) HDL < 1.29 mmol/L female — present (0.9); (3) BP ≥ 130/85 — present (142/92); (4) Fasting glucose ≥ 5.6 — present (6.4). ALL 4 additional components present = metabolic syndrome. BMI 36.2 = obesity Grade II (35.0–39.9 kg/m²).

Q2. Female, 24 years, hirsutism (Ferriman-Gallwey score 14), oligomenorrhoea (cycle 45–90 days), bilateral polycystic ovaries on ultrasound (>12 follicles per ovary, ovarian volume > 10 mL), LH/FSH ratio 3.2. Total testosterone elevated. Which diagnosis?

A) Hypothyroidism with menstrual irregularity
B) Hyperprolactinaemia
C) Polycystic ovary syndrome (PCOS) — E28.2
D) Congenital adrenal hyperplasia
E) Premature ovarian insufficiency

CORRECT ANSWER: C) Polycystic ovary syndrome (PCOS) — E28.2
PCOS Rotterdam criteria (2003): ≥2 of 3: (1) oligo/anovulation — present (oligomenorrhoea 45–90 day cycles); (2) clinical/biochemical hyperandrogenism — present (hirsutism FG 14, elevated testosterone); (3) polycystic ovaries on ultrasound — present (>12 follicles/ovary, volume >10 mL). All 3 criteria met. LH/FSH > 2 supports but is not a diagnostic criterion. ICD-10: E28.2.

Q3. Patient with Type 2 DM on metformin and dapagliflozin. HbA1c stable at 6.9% for 2 years. BP controlled. No complications. Appropriate dispensary follow-up frequency for HbA1c?

A) Every month
B) Every 3 months
C) Every 6 months
D) Once per year

E) Only if symptoms occur

CORRECT ANSWER: C) Every 6 months
ADA Standards 2020: HbA1c every 6 months in STABLE, well-controlled patients (HbA1c at target, no recent therapy changes). HbA1c every 3 months: unstable control, recent therapy change, HbA1c above target, or pregnancy. Annual monitoring: lipids (stable patients), urine albumin/creatinine ratio, ophthalmological examination, foot examination, renal function.

BLOCK B — Reconstructive Level | Time: 60 min

CLINICAL CASE 1

Patient N., 30 years (female), hirsutism (face, abdomen, thighs), irregular menstruation (8–10 cycles/year), infertility (1.5 years of trying), acne. BMI 29.4 kg/m². Waist 92 cm. Fasting glucose 5.9 mmol/L, insulin 28 μU/mL (norm < 20) — HOMA-IR 7.4. Total testosterone 3.8 nmol/L (norm < 2.5). LH 14.8 IU/L, FSH 5.2 IU/L (ratio 2.85). Pelvic ultrasound: both ovaries enlarged with 14–16 small follicles in periphery, echogenic stroma.

Questions:

70. Formulate the diagnosis with ICD-10. (5 points)
71. Which additional examinations are needed? (5 points)
72. Compose a comprehensive treatment plan including infertility management. (10 points)

ANSWERS:

1. Diagnosis: Polycystic Ovary Syndrome (PCOS), phenotype A (all 3 Rotterdam criteria met). Complicated by: insulin resistance (HOMA-IR 7.4 > 2.5); hyperandrogenaemia (testosterone 3.8 nmol/L); infertility (anovulatory); overweight (BMI 29.4). ICD-10: E28.2 (PCOS). Justification: (1) oligo-anovulation — 8–10 cycles/year; (2) biochemical hyperandrogenism — testosterone 3.8 + hirsutism, acne; (3) polycystic ovaries — ≥12 follicles per ovary on US. HOMA-IR 7.4 confirms significant insulin resistance.

2. Additional examinations: OGTT with insulin (75 g glucose, measure glucose and insulin at 0 and 120 min) — assess impaired glucose tolerance and insulin resistance degree; 17-OH progesterone (exclude congenital adrenal hyperplasia — non-classic form can mimic PCOS); prolactin + TSH (exclude hyperprolactinaemia and hypothyroidism as cause of menstrual irregularity); DHEAS + androstenedione (full androgen profile — adrenal vs ovarian source); lipid profile (metabolic syndrome component); pelvic ultrasound detailed measurement of ovarian volume and antral follicle count; partner semen analysis (infertility evaluation); cortisol + dexamethasone suppression (exclude Cushing's if clinically suspected).

3. Treatment plan: (1) LIFESTYLE MODIFICATION (first-line for all): 5–10% weight reduction dramatically improves PCOS (ovulation restoration in 30–50%). Diet: low glycaemic index foods; caloric restriction 500 kcal/day deficit. Physical activity: 150 min/week aerobic + resistance training. (2) INSULIN RESISTANCE: Metformin 500–1000 mg twice daily — improves insulin sensitivity, reduces androgen production, can restore ovulation. (3) HYPERANDROGENISM/HIRSUTISM: Combined oral contraceptive pill (COC) with anti-androgenic progestogen (e.g., drospirenone + ethinylestradiol) — reduces androgens, regulates cycle, treats acne and hirsutism. Spironolactone 100–200 mg/day — additional anti-androgen if COC insufficient. (4) INFERTILITY (anovulatory): First-line ovulation induction: letrozole (aromatase inhibitor) 2.5–5 mg days 3–7 (superior to clomiphene in PCOS per NEJM 2014). Second-line: gonadotropins (FSH injections) with monitoring. Third-line: IVF (with modified protocols to prevent OHSS). Metformin continues throughout. (5) DISPENSARY OBSERVATION: Annual: OGTT (high DM risk), lipid profile, BP, endometrial assessment (anovulation = endometrial hyperplasia risk). Every 6 months: anthropometrics, androgen levels, HbA1c if pre-diabetes.

9. RECOMMENDED LITERATURE

Main Literature (L1)

- L1.1. Melmed S., Auchus R.J., Goldfine A.B. et al. Williams Textbook of Endocrinology. — Elsevier, 2020.
- L1.2. Jameson J.L., De Groot L.J. Endocrinology: Adult and Pediatric. — Elsevier, 2020.
- L1.4. Gardner D.G., Shoback D.M. Greenspan's Basic & Clinical Endocrinology. — McGraw-Hill, 2020.
- L1.5. Feingold K.R. et al. Endotext [Internet]. — MDText.com, Inc., 2020.
- L1.7. American Diabetes Association. Standards of Medical Care in Diabetes — 2020. — Diabetes Care, 2020.

Additional Literature (L2)

- L2.1. American Diabetes Association. Standards of Medical Care in Diabetes — 2020: Algorithm of specialized medical care. — Diabetes Care, 2020.
- L2.2. Melmed S. et al. Williams Textbook of Endocrinology. — Elsevier, 2020.
- L2.6. Braunwald E. et al. Harrison's Principles of Internal Medicine. — McGraw-Hill, 2020.
- L2.7. Zimmerman M.B., Boelaert K. Iodine Deficiency and Thyroid Disorders. — Thyroid, 2020.

Regulatory Documentation

- ICD-10 / ICD-11 — International Classification of Diseases (Endocrine chapter: E00–E90).
- Clinical Protocols of the Ministry of Health of the Kyrgyz Republic — Endocrinology.
- Order of the Ministry of Education and Science of the Russian Federation dated 12.08.2020 No. 988 — FSES 3++ Specialty 31.05.01.
- Electronic Library of KRSU: www.lib.krsu.kg