Ministry of education and science of Ukraine
V. N. Karazin Kharkiv National University

MANAGEMENT OF A PATIENT WITH METABOLIC SYNDROME

Methodical recommendations
for the preparation of students of higher education in the 6th year of study
in the discipline «Internal Medicine»

Kharkiv – 2023

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B26

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The methodological recommendations show the main aspects of management of patients with metabolic syndrome.

For 6th year students to prepare for practical exercises in the discipline «Internal Medicine with Infectious Diseases and Phthisiatry»

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CONTENT

LIST OF CONDITIONAL ABBREVIATIONS

AH	arterial hypertension
GLP-1	glucagon-like peptide-1
BP	blood pressure
WHO	World Health Organization
ECG	electrocardiography
FM	fat mass
BMI	body mass index
IR	insulin resistance
CHD	coronary heart disease
MS	metabolic syndrome
BM	body mass
OGTT	oral glucose tolerance test
WC	waist circumference
HC	hip circumference
CVD	cardiovascular disease
HDL	high density lipoproteins
DM	diabetes mellitus
BS	blood sugar
IDF	International diabetes federation
HbA1c	glycated hemoglobin
HOMA-IR	Homeostasis Model Assessment of Insulin Resistance

1. BASIC KNOWLEDGES, SKILLS NECESSARY FOR STUDYING THE TOPIC

Names of previous disciplines	Acquired skills
Foreign language	Be able to work with foreign sources to obtain up-to-date data on methods of diagnosis and treatment of endocrine diseases
Medical informatics and statistics	Apply modern computer programs and be able to work with them, to be able to use statistical methods of processing the results of clinical trials, analyze research results, be able to evaluate and interpret the results of clinical trials presented in information sources, to be able to work with electronic databases
Human anatomy. Normal physiology. Histology, cytology and embryology	Know the normal structure, functions and regulation of the endocrine system, understand and determine the relationship of its structure and function with other organs and systems of the human body
Pathomorphology. Pathophysiology	Know typical pathogenetic processes: mechanisms of development, compensatory reactions of an organism, development of interrelations which have the character of «cause-effect» Know the pathomorphological changes of endocrine glands that lead to the corresponding functional disorders. Describe and schematically depict the mechanism of development of typical pathological syndromes in endocrine diseases, substantiate pathogenetic approaches to drug therapy

Pharmacology	Be able to navigate the range of drugs. Know the mechanism of action of drugs, their pharmacodynamics, indications and contraindications to their use. Know the features of clinical pharmacology of drugs used in the treatment of endocrine diseases, the features of the pharmacological action of these drugs in different categories of patients. Make a reasonable choice of individual drugs and treatment regimens, optimize treatment regimens, evaluate the effectiveness and safety of pharmacotherapy taking into account the individual characteristics of the patient, the presence of comorbidities in accordance with the principles of evidence-based medicine, modern national and international protocols.
Propaedeutics of internal medicine	Conduct a physical examination of patients, analyze the results of basic laboratory and instrumental research methods. Identify the leading symptoms and syndromes in endocrine patients. Be able to make a differential diagnosis, substantiate and formulate a diagnosis on the basis of physical examination and data of additional methods

1.1. The student must know

– Definition of the concept of metabolic syndrome (MS).

– Risk factors of MS.

– Mechanisms of development and progression of hormonal and metabolic disorders in case of MS.

– Etiology and pathogenesis of MS.

– Clinical picture of MS.

– Diagnostic criteria of MS.

– Indications for conducting and basics of interpretation of the results of hormonal studies and tests.

– Methods of the treatment of MS.

1.2. The student must be able to

– Identify risk factors for MS.

– Diagnose the MS.

– Determine the presence and severity of multiple organ complications of MS.
– Analyze the results of hormonal investigations and functional tests.
– Carry out differential diagnosis of MS with other pathologies.
– Make a treatment plan for MS and its complications.
– Interact with related specialists (surgeon, ophthalmologist) at the stage of making a complete diagnosis, choosing of the treatment method and tactics and following up of a patient with MS.

2. CONTENT OF THE TOPIC

2.1. Introduction

MS is currently one of the actual problems in terms of its medical and social significance, high frequency and continuing increase in prevalence, as well as the predominant development in people of working age.

MS is a strong risk factor for type 2 diabetes mellitus (DM) and cardiovascular diseases (CVD) associated with cardiovascular and total mortality. Thus, among patients with MS the risk of coronary heart disease (CHD) is 3–4 times higher, mortality from coronary heart disease, stroke is 3 times higher than in people without MS, mortality from other causes is 2 times more common than in patients without metabolic disorders.

The prevalence of the disease ranges from 20 to 40 %. The disease most often affects people aged from 35 to 65 years, mostly male patients. In women, the risk of developing the syndrome after menopause increases 5 times.

That is, the concept of MS is to identify patients at high cardiovascular risk in whom lifestyle modification and the use of adequate treatment measures can significantly improve health outcomes and prevent the onset and progression of both atherosclerosis and type 2 DM. These diseases are inextricably linked to an increase in morbidity and mortality in any population.

Synonyms of MS: in the literature MS is called syndrome X, hormonal MS, metabolic vascular syndrome, metabolic threesyndrome, polymetabolic

syndrome, «abundance» syndrome, «fatal quartet», «fatal sextet». According to the main pathogenetic factors, the most promising is the term «insulin resistance (IR) syndrome».

2.2. The concept of metabolic syndrome, risk groups and causes of the development

Metabolic syndrome – is a complex of pathologically interrelated metabolic and clinical disorders with a typical increase in visceral fat mass, decreased peripheral tissue sensitivity to insulin and compensatory hyperinsulinemia, which lead to disorders of carbohydrate, lipid, purine metabolism and arterial hypertension (AH).

Adipose tissue is an active endocrine organ that plays a key role in the development of obesity and MS. Adipocytes are able to produce the hormones adipokines – adiponectin, leptin, resistin, tumor necrosis factor alpha, visfatin, ghrelin and others.

Adiponectin has antidiabetic, antiatherogenic, anti-inflammatory properties, has a direct effect on reproductive tissues. The concentration of adiponectin in the serum is inversely proportional to the mass of fat. The level of adiponectin correlates with tissue sensitivity to insulin, i.e. hypoadiponectinemia can lead to the IR and DM.

Leptin (the so-called hunger hormone) controls the amount of adipose tissue, reducing or increasing appetite. Excreted into the blood from adipose tissue, leptin interacts with specific receptors, suppressing appetite and eating excess food. If the receptors stop responding to leptin (so-called leptin resistance), excess weight and obesity develop. When leptin levels due to overeating are constantly high, the body gradually stops responding to normal hormone levels. In this situation, a person does not feel hunger and satiety correctly, as a result of which he cannot lose weight.

Resistin is an insulin antagonist. There is evidence that the level of circulating resistin can be considered as a prognostic marker of obesity, impaired tissue sensitivity to insulin and type 2 DM.

Factors contributing to the development of MS include:

– genetic predisposition (formation of MS is genetically determined, it is known the gene for insulin receptors, which is localized on chromosome 19, more than 50 mutations of this gene have been described);

– nutritional disorders (unbalanced diet with a predominance of fatty foods in the diet, in particular overeating of animal fats containing saturated fatty acids; under such conditions, the development and progression of obesity occurs if the mass of fat consumed exceeds the body's ability for its oxidation);

– inactive lifestyle, «sedentary» work, lack of physiological exercise (it is the second from the most important factors after overeating, in the presence of which the slowing of lipolysis and utilization of triglycerides in muscle and adipose tissue takes place, reducing of translocation of glucose transporters in the muscles develops, which leads to the development of IR and obesity);

– prolonged, uncontrolled or poorly controlled hypertension (causes deterioration of peripheral blood circulation, which leads to decreased tissue sensitivity to insulin and, as a consequence, to hyperinsulinemia);

– nervous tension, stress, intense worries (lead to endocrine disorders and overeating);

– hormonal imbalance:

- in women, high or at the upper limit of normal testosterone levels, or its moderate increase in functional ovarian hyperandrogenia (polycystic ovary syndrome) in reproductive age and a decrease in estrogen-producing ovarian function in peri- and postmenopause lead to the accumulation of visceral adipose tissue, increase of IR, accumulation of atherogenic lipids due to the effects of testosterone in the female body;

- androgen deficiency in men (a decrease in testosterone, which primarily develops with age, with dyslipidemia, IR, obesity and AH, which are parts of MS and cardiovascular diseases; low testosterone level is not only associated with the above conditions, but also leads to their development and progression);
- low socioeconomic status.

Thus, the risk group for MS includes patients with the following diseases:

– AH;
– type 2 DM (or pre-diabetes);
– overweight and obesity;
– CHD, peripheral vascular disease,
– cerebrovascular diseases associated with atherosclerosis;
– direct relatives with hyperlipidemia and / or obesity, and / or DM, and / or CHD;
– postmenopausal period in women;
– other diseases (polycystic ovary syndrome in women, erectile dysfunction in men, uric acid metabolism disorders - hyperuricemia or gout).

Among the ***mechanisms of the MS development*** are considered:

- IR and hyperinsulinemia;
- activation of the sympathic-adrenal system;
- activation of the renin-angiotensin-aldosterone system;
- violation of adipocytokine synthesis by adipose tissue;
- leptin resistance;
- endothelial dysfunction;
- pathological adipose tissue ectopia (intra-abdominal, perivascular, epicardial, intrahepatic).

The interconnection of possible mechanisms of MS development is presented in Fig. 1.

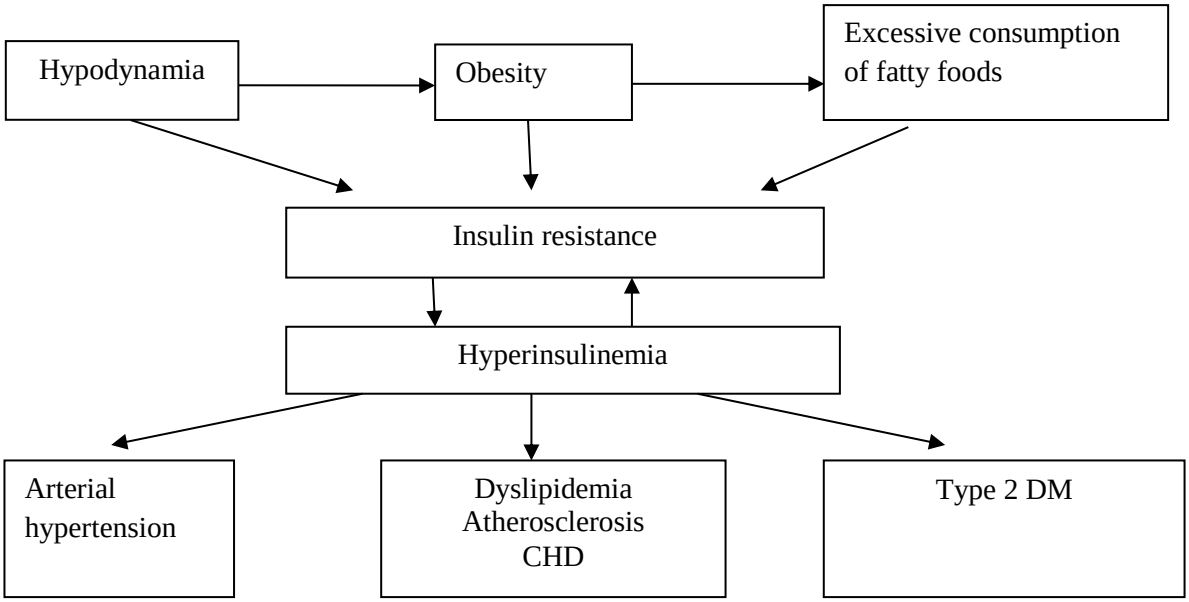


Fig. 1. Interconnection of mechanisms of MS development

IR together with visceral obesity leads to numerous pathological conditions, the main of which are presented in Fig. 2.

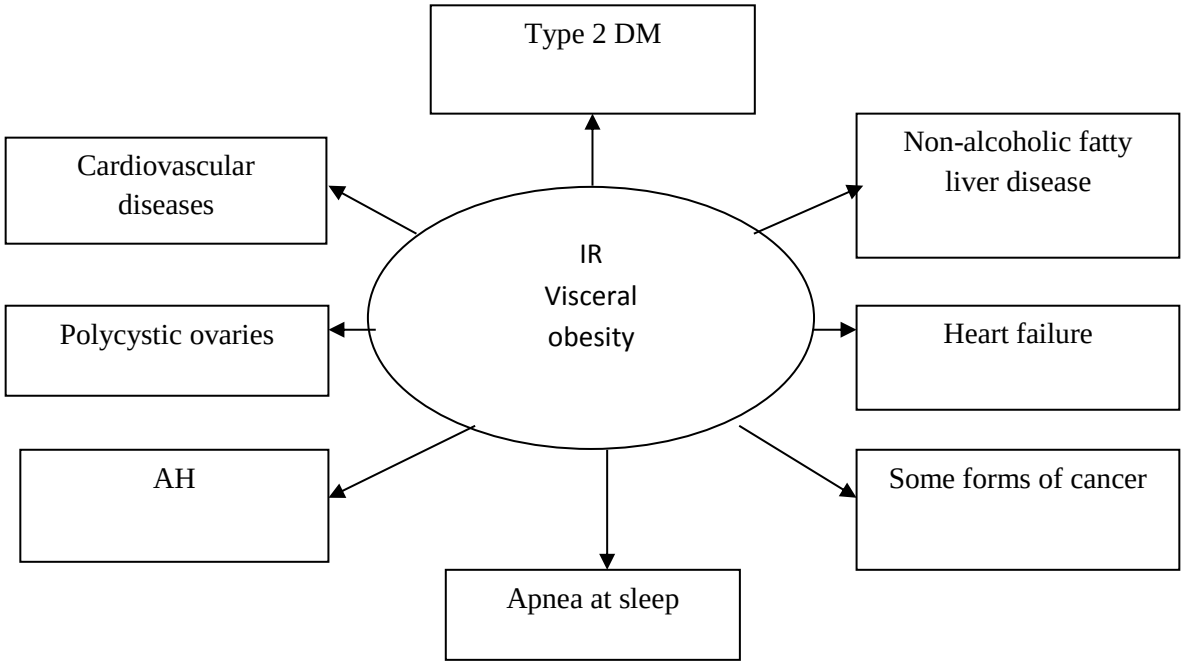


Fig. 2. Pathological conditions due to IR and visceral obesity.

The following clinical manifestations may also occur in MS:

- hyperglycemia;
- atherogenic dyslipidemia;

– chronic subclinical inflammation (increased levels of C-reactive protein and other proinflammatory cytokines);
– disorders of hemostasis system: hypercoagulation due to increased fibrinogen concentration and decreased blood fibrinolytic activity - hypofibrinolysis,
– early atherosclerosis / CHD;
– left ventricular hypertrophy with diastolic dysfunction;
– hyperuricemia and gout,
– albuminuria;
– osteoporosis;
– hyperandrogenism and polycystic ovary syndrome, etc.

2.3. Diagnosis of the metabolic syndrome

The MS criteria have been repeatedly revised by numerous reputable expert organizations. The most modern criteria are presented in the table 1.

Table 1

MS criteria according to the International Diabetes Federation (IDF)

Indicator	Value
Waist circumference (WC)	> 94 cm for men and > 80 cm for women
Triglycerides	> 1.7 mmol/l (> 150 mg/dL) or treatment of previously diagnosed hypertriglyceridemia
High density lipoproteins (LDH)	< 1,03 mmol/l (<40 mg/dL) for men <1.29 mmol/l (<50 mg/dL) for women or treatment of previously diagnosed dyslipidemia
Blood pressure (BP)	Systolic > 130 or diastolic > 85 mm Hg, or treatment of previously diagnosed hypertension
Fasting blood glucose	> 6,1 mmol/l (> 110 mg/dL) or previously diagnosed type 2 DM. If the fasting blood glucose is higher than 6.1 mmol/l, it is recommended to perform an oral glucose tolerance test (OGTT)

If a patient has at least three of these symptoms, he or she is considered to have MS.

Examination of the patient begins with the patient's complaints, which are typical for the components of MS (headaches, dizziness, dry mouth, joint pain,

etc.). Particular attention should be paid to detailing complaints: how the patient tolerates exercise, whether there is daytime drowsiness, snoring at night, what he relates to these symptoms.

In the anamnesis of the disease, it is advisable to indicate how long the patient has gained excess weight, increased blood pressure, what appeared first, if an increased blood sugar (BS) was determined before.

Life history should include questions about heredity (whether the patient's close relatives have DM, CVD, or someone who is overweight), what is the patient's level of physical activity, include nutritional assessment (whether eating disorders, nutrient imbalance are detected).

On physical examination, anthropometric measurements of height, body mass (BM), WC and hip circumference (HC) are mandatory; calculation of body mass index (BMI) and the WC/HC ratio, the percentage of fat mass (FM) in the body, the thickness of subcutaneous fat in the abdomen, shoulder measurement.

BMI is calculated by the formula:

$$\text{BMI} = \frac{\text{m}}{h^2},$$

where m – BM in kilograms, h – height in meters.

BMI is measured in kg/m^2. For example, human weight is 106 kg, height is 168 cm. Therefore, BMI in this case is calculated as follows: BMI = 106: $(1.68)^2$ = 37.55 kg/m^2.

BMI is used to determine if a patient is overweight and to determine the degree of obesity. BMI from 18.5 to 24.9 kg/m^2 is considered normal. The classification of obesity by BMI is given in the table 2.

Table 2

Classification of obesity

BMI	Degree of obesity	Risk of comorbidities
25,0–29,9	Excess body weight	Increased
30,0–34,9	Grade 1 obesity	High
35,0–39,9	Grade 2 obesity	Very high
≥ 40,0	Grade 3 obesity (severe obesity)	Extremely high

The percentage of FM in the body can be determined using special equipment or calculated by the formula:

FM% = 29,47335 + (0,20125×BM) + (0,13456×HC) – (0,16368× height),

where the value of BM is given in kg, the value of HC and height - in cm.

Table 3

Characteristics of the FM% based on gender (WHO, 2010 yr.)

Gender	Insufficient	Optimal	High	Very high
Women	< 10 %	10–19,9 %	20–24,9 %	> 25 %
Men	< 20 %	20–29,9 %	30–34,9 %	> 35 %

The key diagnostic criterion for MS is WC. If a patient, even in the absence of obesity (by BMI), has an WC > 94 cm for men and > 80 cm for women, he should be considered as having MS. This is due to the fact that WC determines the visceral type of subcutaneous fat distribution, which is associated with an increased risk of AH, IR, DM, dyslipidemia, CHD.

Laboratory examination includes:

- complete blood test;
- urinalysis (primarily to detect albuminuria);
- urine test (daily or single dose) for albuminuria;
- BS, glycated hemoglobin (HbA1c), OGTT (according to the indications);
- blood lipid spectrum (HDL, low and very low density lipoproteins);
- serum uric acid (a marker of the purine metabolism state);
- coagulogram (in MS blood clotting indicators increase);
- hormonal profile: insulin, cortisol, thyroid hormones (according to the indications), prolactin (according to the indications), aldosterone-renin ratio (in the case of diagnostic search for the cause of AH).
- calculation of the IR index (HOMA-IR – Homeostasis Model Assessment of Insulin Resistance) by the formula:

$$\text{HOMA-IR} = \frac{\text{glucose level (mmol/l)} \times \text{insulin level (}\mu IU/\text{ml)}}{22,5}$$

Normally, the HOMA index does not exceed 2,7;

• biomarkers of adipose tissue: leptin (its excess enhances IR, increases the risk of cardiovascular complications), adiponectin, ghrelin;

• indicators of liver status (alanine aminotransferase, aspartate aminotransferase);

• indicators of pro-inflammatory status: C-reactive protein, pro-inflammatory cytokines (tumor necrosis factor - alpha, interleukin-6).

One of the main clinical manifestations of MS is a violation of carbohydrate metabolism, for the diagnosis of which the appropriate indicators are used (table 4).

Table 4

Diagnostic criteria for diabetes mellitus and other glycemic disorders

	Norm	Impaired glucose tolerance	DM	Impaired fasting glycemia
fasting BS	< 5,6 mmol/l (100 mg/dl)	< 7,0 mmol/l (126 mg/dl)	≥ 7,0 mmol/l (126 mg/dl)	5,6–6,9 mmol/l (100–125 мг/дл)
BS in 2 hours in the dynamic of OGTT	< 7,8 mmol/l (140 mg/dl)	7,8–11,0 mmol/l (140–200 mg/dl)	≥ 11,1 mmol/l (200 mg/dl)	< 7,8 mmol/l (140 mg/dl)
HbA1c	< 5,7 %	5,7–6,4 %	≥ 6,5 %	<6,4 %
Additional criteria			Presence of the symptoms of diabetes, random glycemia ≥ 11.1 mmol/l (200 mg/dL)	

Instrumental examination

– electrocardiography (ECG), 24-hour ECG monitoring;

- 24-hour BP monitoring;
- echocardiography;
- cardiorespiratory sleep examination (performed at night in special laboratories to detect obstructive sleep apnea syndrome);
- bioimpedancemetry (allows to determine body composition, i.e. fluid content, fat, lean and muscle BM; to determine which component is the main in the patient's overweight);
- ultrasound examination of the abdominal organs, in particular the liver.

Due to the overweight, it is advisable to estimate the daily caloric intake of the patient based on his physical activity.

When diagnosing MS, it is necessary to take into account the increased risk of some forms of cancer development:

- of the liver,
- of the pancreas,
- of the uterus,
- of the breast,
- of the bladder,
- colorectal cancer.

2.4. Differential diagnosis of metabolic syndrome

Isolated alimentary obesity is the result of an imbalance between calories consumed and expended and, as a rule, is not accompanied by comorbid pathology typical for MS.

The combination of abdominal obesity, AH, carbohydrate metabolism disorders also occurs in patients with Cushing's syndrome (hypercortisolism), that requires differential diagnosis (table 5).

If Cushing's syndrome is suspected, a hormonal examination (cortisol, adrenocorticotropic hormone), dexamethasone functional tests (according to

indications), and instrumental examination (magnetic resonance or computed tomography to rule out tumor or hyperplasia) should be performed.

Table 5

Differential diagnosis of MS and Cushing's syndrome

Clinical sign	MS	Hypercortisolism
Stretch marks	No or white	Yes, pink or purple (catabolic effect of cortisol)
Bruises	No	Yes (catabolic effect of cortisol)
Thinning of the skin	No	Yes (catabolic effect of cortisol)
Proximal myopathy	No	Yes (catabolic effect of cortisol)
Osteoporosis	May be present in elderly patients	Yes (catabolic effect of cortisol)
Dorsocervical fat pad (buffalo hump)	No	Yes (catabolic effect of cortisol)
Facial hyperemia	No	Yes (catabolic effect of cortisol)
Distribution of subcutaneous fat	Central	Dysplastic (central with predominant fat deposition in the upper half of the torso with thin extremities)
Hyperandrogenism / menstrual disorders	Possible in polycystic ovary syndrome	Yes (cortisone inhibition of gonadotropin release and increased androgen secretion)

Overweight, high BP, drowsiness, and sometimes carbohydrate metabolism disorders can be symptoms of hypothyroidism. At the same time, complaints typical for this disease (chilliness, swelling of the face, hands, hair loss, severe dryness of the skin, difficulty and slowing of speech, etc.) help to suspect pathology of the thyroid gland. Finally, the determination of the level of the pituitary thyroid-stimulating hormone allows to confirm or refute the clinical hypothesis.

2.5. Treatment of metabolic syndrome

The main purpose of MS treatment is to prevent the development of acute vascular catastrophes (in particular, myocardial infarction, stroke), as well as

chronic complications inherent in the diseases included in this symptom complex.

Specific goals of MS treatment

1. Weight loss at a rate of not more than 0.5–1 kg per week.

The nearest goal is 2 kg per month.

Long-term targets – 6–10 kg in 6 months.

2. Maintaining a new, lower body weight and preventing weight re-gain.
3. Reducing the severity or elimination of the MS manifestations: normalization of the BP, compensation of carbohydrate, lipid, purine metabolism.
4. Improving the quality of life, well-being, improving efficiency.

The first step in the treatment of MS is dietary correction. There are general principles:

- the number of meals should be detected individually for each patient and can be divided into three main meals and small portions up to 6 times a day;
- it is necessary to exclude fast food, significantly reduce the consumption of high fat foods, easily digestible carbohydrates (sweets, muffins, sweet drinks);
- increase consumption of vegetables and fruits (at least 400 g of vegetables and fruits per day), sufficient quantity of cereals (porridge, whole-grain bread);
- limit salt intake to 5–-6 g per day in patients with increased BP (taking into account salt containing in products);
- never starve.

It is essential to correct eating behavior that is impaired due to serotonin deficiencies in the brain systems that regulate appetite.

At the same time with proper nutrition, it is extremely important to maintain adequate physical exertion. It is necessary to do at least 30 minutes of exercise a day.

Medical treatment for MS

1. Medication-assisted treatment of obesity (when non-medication measures are ineffective - decrease in body weight is less than 5% of the output level during 3 months of treatment, when there is a high degree of obesity, when a quick weight reduction is needed due to comorbid pathology, etc.).

In 2020, the US Food and Drug Administration approved 5 medications for the treatment of obesity:

- orlistat;
- phentermine + topiramate combination;
- lorcaserin;
- naltrexone + bupropion combination;
- liraglutide.

Orlistat has a peripheral effect by inhibiting gastrointestinal lipases. They lose the ability to break down fats that come with food in the form of triglycerides, into free fatty acids that are absorbed and monoglycerides. This reduces the number of calories that enter the body. After 24–40 h, there is an increase in the concentration of fat in the fecal mass, which can cause diarrhea. The severity of this side effect depends on the amount of fat consumed, which further motivates patients to follow a diet. Orlistat is prescribed at a dose 120 mg 3 times a day during or no later than 1 hour after the main meals.

The combination of the sympathomimetic phentermine and the antiepileptic drug topiramate has a central mechanism of action, reducing appetite. The initial dose of the drug is 3.75/23 mg, then 7.5/46 mg.

Lorcaserin is an agonist of serotonin 2 C-receptors, the activation of which in the brain helps to speed up satiety even with a small amount of food consumed and blocks the feeling of hunger. Lorcaserin is prescribed at a dose 10 mg 2 times a day.

The opioid receptor antagonist naltrexone in combination with the antidepressant bupropion is effective in treating obesity also through central mechanisms. The initial dose of the combined drug is 8/90 mg, then 16/180 mg.

Liraglutide (glucagon like peptide-1 (GLP-1) receptor agonist) (Saxenda) reduces adipose tissue mass by reducing food intake and regulating appetite by enhancing gastric emptying and satiety, along with reducing hunger. The initial dose is 0.6 mg subcutaneously, the target dose is 3.0 mg daily.

2. Correction of insulin resistance.

- Biguanides (metformin) - can reduce the production of glucose by the liver, inhibit the absorption of glucose in the small intestine, increase tissue sensitivity to insulin. The dose of the drug is 500–2000 mg per day during or immediately after a meal.
- Alpha-glucosidase inhibitors (acarbose, voglibose) - cause reverse blockade of alpha-glucosidase (glucomylase, maltase) in the upper small intestine. This leads to impaired enzymatic cleavage of poly- and oligosaccharides and absorption of monosaccharides, which prevents the development of postprandial hyperglycemia and reduces insulin levels. The dose is 50–100 mg three times a day.
- Thiazolidinediones (pioglitazone) - reduce glucose production by the liver and reduce IR. The dose is 15-45 mg once a day.
- GLP-1 receptor agonist (liraglutide (victose), semaglutide) - glucose-dependently stimulate insulin synthesis and inhibit glucagon synthesis. The mechanism of blood glucose lowering also includes a slight slowing of gastric emptying. The dose of the drug is 0.6–1.8 mg once per day subcutaneously.

In the presence of type 2 DM, its treatment is carried out according to generally accepted algorithms.

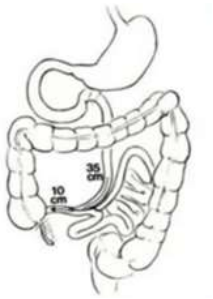
3. Correction of the lipid metabolism disorders (3-hydroxy-3-methylglutaryl-coenzyme A reductase inhibitors, fibroic acid derivatives).

4. Treatment of AH (angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, calcium channel blockers, diuretics, β-blockers, combined antihypertensive drugs).

Surgical treatment of MS (including obesity)

In severe obesity (BMI ≥ 40 kg/m^2 or BMI ≥ 35 kg/m^2 in the presence of severe comorbid pathology and ineffectiveness of drugs and non-drug treatment methods), it is advisable to consider the use of bariatric surgery. Bariatric surgery is not a cosmetic procedure, but primarily aimed at avoiding the pathological consequences of morbid obesity. Today, various techniques are used (mostly laparoscopic), which can be divided into 3 main groups.

1. Malabsorbtive interventions that reduce the absorption of nutrients in the gastrointestinal tract by "excluding" part of the gastrointestinal tract from the digestive process.



They include:

- jejunoileal shunt,
- jejunocolic shunt.

2. Gastro-restrictive operations, which reduce the patient's stomach volume, cause a feeling of rapid satiety, increase the stimulation of gastric mechanical and chemical receptors, reduce the rate of gastric emptying and modulate the level of gastric hormones that contribute to obesity. They include:

- gastric banding,
- vertical banded gastroplasty,
- sleeve gastrectomy.

Some endoscopic procedures also mimic the reduction of the stomach size by placing special temporarily inserted devices, such as intragastric balloons, transpyloric shuttles, etc.

3. Combined operations:

- gastric bypass surgery,
- biliopancreatic shunting.

Evaluation of the results of obesity therapy

1. No increase in BM in the future - a satisfactory result.
2. Reducing of BM by > 5% and keeping the weight at the same level is a good result.
3. Reducing of BM by > 10% and keeping the weight at the same level is an excellent result.

TEST TASKS FOR SELF-CONTROL

1. Select the main MS criterion:
   a. central type of obesity;
   b. obstructive sleep apnea;
   c. hepatic steatosis;
   d. osteoporosis.

2. What is the basis for the development of MS:
   a. dyslipidemia;
   b. insulin deficiency;
   c. insulin resistance;
   d. obesity.

3. Which of the diseases listed below is the part of the MS:
   a. rheumatoid arthritis;
   b. hypothyroidism;
   c. nephropathy;
   d. arterial hypertension

4. The method of laboratory diagnostics of insulin resistance is:
   a. determination of fasting blood sugar;
   b. determination of fasting blood insulin level;
   c. determination of blood lipids;
   d. determination of the HOMA-IR index

5. The drug of choice in the treatment of insulin resistance is:
   a. biguanides;
   b. sulfonylurea drugs;
   c. diuretics;
   d. β-blockers.

6. Evaluation of the excellent results of MS therapy includes:
   a. weight loss > 10 %;
   b. weight loss > 17 %;
   c. weight loss > 15 %;
   d. weight loss > 20 %.

7. A life-threatening complication of MS is:
   a. cerebral circulatory disorders;
   b. obesity;
   c. arterial hypertension;
   d. infertility.

8. What type of surgery is not related to bariatric surgery?
    a. gastric bypass surgery;
    b. sleeve gastrectomy;
    c. biliopancreatic shunting;
    d. liposuction.

9. What type of adipose tissue deposition is characteristic of MS?
    a. android (visceral);
    b. gynoid;
    c. mixed;
    d. dysplastic.

10. What is the NOMA-IR index used for?
    a. to determine insulin resistance;
    b. to determine the presence of obesity;
    c. to determine the distribution of fat mass;
    d. for the diagnosis of diabetes mellitus.

Keys: 1) a; 2) c ; 3) d; 4) d; 5) a; 6) a; 7) a; 8) d; 9) a; 10) a.

SITUATIONAL TASKS

1. The patient, a 38-year-old woman, complains of weight gain, increased appetite. Objectively: hyperstenic, BMI 33.5 kg/m^2, WC 101 cm. The WC/HC ratio is 0.95. What diagnosis will you make?
   a. 2 degree dysplastic obesity.
   b. Metabolic syndrome.
   c. 1 degree android type alimentary obesity.
   d. 2 degree gynoid type alimentary obesity.

2. A 47-year-old patient complains of overweight, fatigue, general weakness, edema of the lower extremities in the evening. Objectively: height - 160 cm, weight - 104 kg, puffy face, dry skin, pulse - 60 beats per minute, BP - 110/60 mm Hg. On examination, the fasting glucose level is 5.9 mmol/l, the level of glycated hemoglobin is 6.1%, insulin is 28.4 mIU/l, TSH is 2.3 mIU/l, and T4 is 14.1 mIU/l. What medications are needed?
   a. Mercazolyl.
   b. Metformin.
   c. Levothyroxine.
   d. Prednisolone.

3. A 38-year-old man underwent surgery for diffuse toxic goiter. After surgery, the level of fasting capillary blood glucose is 4.3 mmol/l, the level of thyroid hormones is normal. 2 months after surgery, weight gain of about 10 kg. At the repeated inspection the level of glucose - 5,1 mmol/l, the level of glycated hemoglobin - 5,1%, TSH - 12,3 mIU/l, T4 - 7,1 mIU/l. What disease do you think of?
   a. Metabolic syndrome
   b. Recurrence of diffuse toxic goiter
   c. Impaired glucose tolerance
   d. Postoperative hypothyroidism

4. A 55-year-old woman with 3 degree obesity and AH hyperlipidemia due to LDL, decreased HDL and hyperinsulinemia revealed. Diagnosed with metabolic syndrome. What is the main pathogenetic mechanism of this syndrome?
   a. Hyperlipidemia.
   b. Hyperinsulinemia and insulin resistance.
   c. Decreased LDL concentration.
   d. Hyperuricemia.

5. Patient M., 38 years old, complains of shortness of breath while walking, palpitations, swelling of the extremities in the evening. Height - 164 cm, body

weight - 104 kg. Objectively: the patient is overweight. Heart tones are weakened, tachycardia up to 90 beats per minute. The menstrual cycle is not disturbed, blood sugar - 5.6 mmol/l, functional tests with adrenocorticotropic hormone are not changed. No pathology was detected on the radiograph of the Turkish saddle. What disease should you think about?

a. Climax.
b. Pituitary obesity.
c. Alimentary obesity.
d. Diabetes mellitus.

Keys: 1) c; 2) b; 3) d; 4) b; 5) c

RECOMMENDED LITERATURE

1. Dwarfism: Medical and Psychosocial Aspects of Profound Short Stature. Betti M, Aelson. 2015. – 368 p.
2. Endocrinology and metabolism /Ed. by Pinchera. – London: McGraw Hill Int., 2016. – 811 p.
3. Handbook of Physiology. Section 7: Endocrine system. Volume III: Endocrine regulation of Water and electrolyte balance. / Ed. by J.C. S. Fray. – Oxford University press, 2014. – 750 p. 28
4. Textbook of endocrine physiology / Ed. by J.E. Griffin, S.R. Ojeda. – 4-th ed. –Oxford University press, 2009. – 490 p.

Навчальне видання

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ВЕДЕННЯ ХВОРОГО З МЕТАБОЛІЧНИМ СИНДРОМОМ

Методичні рекомендації
для підготовки здобувачів вищої освіти 6-го року навчання з дисципліни
«Внутрішня медицина» (англ. мовою)