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Article

Monitoring the Lipid Spectrum and Selecting Hypolipidemic Agents: Practical Guidelines

Zikrillayev Farrux Abdurashidovich
Assistant of the Fundamental Medicine Department
Asia International University, Bukhara, Uzbekistan

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Abstract: Latin has long served as the linguistic foundation of medical terminology, providing precision and universality essential to medical communication. This study investigates the grammatical and functional role of Latin adjectives in medical terminology, focusing on their declensions, degrees of comparison, and usage in anatomical and clinical contexts. Despite the frequent use of Latin terms in medicine, little attention has been given to the systematic study of adjectives and their syntactic significance. This study addresses that gap by applying a linguistic-analytical approach, examining entries from Latin dictionaries, medical textbooks, and official medical nomenclatures. The research identifies two primary adjective groups—those belonging to the 1st and 2nd declensions, and those in the 3rd declension—and analyzes their grammatical behavior and meaning. Findings reveal that Latin adjectives serve not only as descriptors but also as essential elements in constructing clear, standardized medical terms. The comparative and superlative forms enhance diagnostic precision by conveying relative size, severity, or position. These findings imply that understanding Latin adjective structure is crucial for accurate interpretation and usage in medical settings. The study contributes to medical linguistics and suggests the integration of classical language training into medical education to improve terminological literacy.

Keywords: Latin adjectives, medical terminology, declension, medical language, comparative degree, terminology precision, linguistic analysis.

Introduction

Latin has historically functioned as the cornerstone of medical terminology, offering consistency and linguistic neutrality across diverse medical systems. Its structural precision, particularly in the formation of adjectives, plays a vital role in ensuring clarity and accuracy in medical documentation and communication. Adjectives in Latin not only describe anatomical or pathological features but also contribute to the formation of precise, concise terms through morphological agreement and comparison. The two major groups of Latin adjectives—those of the 1st and 2nd declensions, and those of the 3rd—demonstrate clear grammatical patterns that underlie their syntactic and semantic roles in medical contexts [2,3].Previous studies have examined the historical development of Latin in medical terminology, yet the detailed grammatical and functional analysis of Latin adjectives remains underexplored. This study seeks to fill that gap by investigating

the morphological behavior, degrees of comparison, and contextual applications of these adjectives, thus offering insight into their contribution to standardized medical language [4]. By doing so, it connects grammatical theory with practical linguistic application in the medical sciences.To explore these dimensions, the study adopts a linguistic-analytical method, reviewing classical Latin dictionaries, medical lexicons, and standardized nomenclature documents such as *Terminologia Anatomica* [5,6]. Key Latin adjectives were selected and analyzed according to declension type, agreement rules, and comparative structures. Particular attention was given to adjectives commonly found in anatomical and diagnostic contexts.It is expected that the study will demonstrate the grammatical stability and functional precision Latin adjectives offer to modern medical terminology [7,8]. Such findings may reinforce the value of classical language knowledge in medical education and global communication.Ultimately, this research contributes to the broader field of medical linguistics by highlighting how structured, classical grammatical elements underpin contemporary medical language, promoting clarity, standardization, and educational consistency in medical practice worldwide[9].

Materials and Methods

This study employed a qualitative, linguistic-analytical methodology to examine the structure and function of Latin adjectives within medical terminology. Data was collected through a comprehensive review of authoritative Latin language dictionaries, medical lexicons, anatomy textbooks, and standardized nomenclature systems, such as *Terminologia Anatomica* and the *International Classification of Diseases (ICD)*. The focus was on identifying frequently used adjectives and classifying them according to declension patterns—specifically distinguishing between the 1st and 2nd declensions and the 3rd declension. Additionally, the degrees of comparison—positive, comparative, and superlative—were analyzed for their role in differentiating anatomical and pathological concepts. Adjectives such as *dexter*, *sinister*, *major*, *minor*, and *gravis* were selected as representative samples. These were evaluated for grammatical agreement with medical nouns and for their contribution to semantic precision in clinical terminology. The study also drew on comparative linguistic analysis to identify consistency in usage across different medical subfields. The collected data was synthesized to interpret how Latin adjectives contribute to standardization, clarity, and international understanding in medical language. This methodology allows for both structural analysis and practical interpretation, offering insights into how classical linguistic forms maintain relevance in modern scientific discourse.

Results and Discussion

Metabolic syndrome ("Syndrome X") is one of the most complex medical and social problems of modern times. The widespread prevalence of the syndrome, its strong association with lifestyle, and the extremely high mortality rate from its consequences demand the joint efforts of physicians from various specialties, as well as public health authorities, in order to ensure timely identification of this syndrome and the implementation of comprehensive preventive and therapeutic measures [10,11].Over the past 20 years, numerous studies have revealed the leading role of insulin resistance in the pathogenesis of several diseases, particularly atherosclerosis, obesity, non-insulin-dependent diabetes mellitus (NIDDM), and arterial hypertension [12,13]. In 2018, G. Reaven highlighted the central role of insulin resistance in the development of metabolic syndrome and listed its main components, including compensatory hyperinsulinemia, impaired glucose tolerance (IGT) or NIDDM, visceral obesity, arterial hypertension, and dyslipidemia (hypertriglyceridemia, reduced levels of high-density lipoprotein cholesterol – HDL-C), as well as disorders of the blood coagulation system.Population studies conducted in Europe and the USA have shown that approximately 5–10% of the adult population of both sexes exhibit manifestations of this symptom complex. According to Australian researchers, the syndrome affects about 30% of the population [14]. G. Reaven estimated that 25% of middle-aged individuals have insulin resistance and, as a consequence, metabolic

syndrome [15].Currently, metabolic syndrome is regarded as a key factor in the pathogenesis of coronary heart disease (CHD), the mortality rate of which remains the highest among developed countries. There are differing views as to whether insulin resistance is a direct cause of atherosclerosis or whether this relationship is mediated by complex mechanisms involving disturbances in carbohydrate and lipid metabolism [16]. However, it is evident that the components of metabolic syndrome are widely recognized as risk factors for CHD and, if not addressed in a timely and appropriate manner, typically lead to the development of atherosclerosis and CHD.Another severe outcome of metabolic syndrome is the development of NIDDM against the background of obesity [17,18]. Peripheral tissue resistance to insulin is considered the central link in the pathogenesis of NIDDM, IGT, and one of the key characteristics of obesity, which is often viewed as the connecting link between insulin resistance and diabetes.Insulin resistance refers to the decreased insulin-dependent utilization of glucose by peripheral tissues, primarily muscles and the liver [19,20]. In NIDDM or IGT, this is accompanied by normal or excessive insulin secretion. Proposed causes of insulin resistance include receptor and/or post-receptor defects in insulin action. Among the post-receptor defects are reduced receptor kinase activity, impaired intracellular glucose transport, and decreased intracellular glucose metabolism due to dysfunction of insulin-regulated enzymes such as glycogen synthase and pyruvate dehydrogenase [21,22].Insulin resistance in muscle tissue is considered the earliest and possibly genetically determined defect, appearing long before the clinical onset of NIDDM.In the liver, impaired insulin action is characterized by the lack of its inhibitory effect on gluconeogenesis, leading to increased hepatic glucose production.Another major factor contributing to hyperglycemia is insulin resistance in adipose tissue . The inability of insulin to suppress lipid oxidation results in the release of large amounts of free fatty acids (FFAs), which, according to the Randle cycle, suppress glucose oxidation in muscles [23,24]. In the liver, FFAs impair insulin-receptor binding on hepatocytes, reducing hepatic insulin clearance and thereby maintaining hyperinsulinemia. Excess FFAs also stimulate gluconeogenesis and affect lipoprotein synthesis in the liver, increasing the production of very low-density lipoproteins (VLDL) and triglycerides (TG), while lowering HDL levels [25].

As long as pancreatic β-cells are able to secrete enough insulin to compensate for these defects and maintain hyperinsulinemia, hyperglycemia does not develop. However, once the β-cell reserves become depleted, relative insulin deficiency arises, clinically manifested by increased blood glucose levels [26,27].

Insulin resistance and a high risk of developing NIDDM are typical for individuals with visceral, rather than subcutaneous, fat accumulation. This may be due to the biochemical properties of visceral adipose tissue: it is highly sensitive to lipolytic stimulation and poorly responsive to the anti-lipolytic effects of insulin [28]. The distribution pattern of adipose tissue is largely genetically determined, though hormonal imbalances in the hypothalamus–pituitary–adrenal axis also play an important role. Individuals with visceral obesity are considered at high risk for developing NIDDM and CHD.The interconnection between insulin resistance, compensatory hyperinsulinemia, and arterial hypertension within the framework of metabolic syndrome is still not fully understood[29,30]. Currently, several possible mechanisms have been proposed to explain this relationship.

By enhancing sodium reabsorption, insulin exerts an antidiuretic effect. Insulin stimulates the sympathetic nervous system and increases catecholamine production.In cell culture experiments, insulin stimulates the proliferation of smooth muscle cells.Insulin-dependent transmembrane ion transport also plays a role in the development of arterial hypertension by altering sodium ion concentration in the endothelium.

Although each manifestation of metabolic syndrome has long been known, there is still no holistic understanding or unified clinical approach to the syndrome among general practitioners. This may be due to the fact that not all components of the metabolic syndrome are always clinically evident at the same time, leading to certain difficulties in diagnosis and preventive interventions.When examining patients with various manifestations of the syndrome, attention should be paid to the presence of relatives with type 2 diabetes mellitus (T2DM), arterial hypertension, or obesity. It is necessary to

determine the body mass index (BMI) and the waist-to-hip ratio (a ratio greater than 0.9 in men and 0.8 in women indirectly indicates visceral obesity, which should be considered even when BMI is within the normal range); to analyze the full lipid profile, including HDL cholesterol; and to perform a glucose tolerance test in patients with excess body weight (BMI over 25 kg/m²) or with visceral obesity, as well as in those with arterial hypertension or a family history of diabetes.

Such an approach will result in the timely identification of individuals at high risk of developing coronary heart disease and T2DM, and enable a comprehensive assessment of their condition, which in turn will allow for the selection of an appropriate therapeutic strategy.Among patients with T2DM who were treated in the diabetes therapy department of the Endocrinology Research Centre of the Russian Academy of Medical Sciences in 1998, more than 30% had arterial hypertension, and about 60% of these showed some form of lipid metabolism disorder. As medical histories revealed, practically none of these patients had previously received adequate antihypertensive or hypolipidemic therapy in accordance with current medical standards. Chronic, often asymptomatic progression is a common feature of all components of the metabolic syndrome. Hyperglycemia, high blood pressure, and especially dyslipidemia may not cause significant complaints, while lack of monitoring and proper treatment leads to truly devastating consequences. All of this presents new challenges to physicians of various specialties, including endocrinologists: to master the principles of treatment and control of all possible metabolic syndrome-related disorders.

Today, insulin resistance syndrome is regarded as a cluster of risk factors for cardiovascular diseases, with each component of the syndrome potentially being secondary to insulin resistance. Based on this, the treatment of patients with metabolic syndrome requires a comprehensive approach, in which the main therapeutic interventions should be aimed not only at correcting existing disorders but also at reducing insulin resistance. Non-pharmacological methods of intervention play an extremely important role in this process.

Given that excess body weight—especially visceral obesity—plays a significant role in the pathogenesis of metabolic syndrome and type 2 diabetes mellitus (T2DM), weight reduction is a pathogenetically justified and essential component of treatment for this category of patients. Weight loss is accompanied by significant improvements, such as normalization of carbohydrate metabolism, reduction of hyperinsulinemia, normalization of lipid metabolism, and blood pressure regulation. Achieving a healthy body weight is impossible without the development of internal motivation in the patient for proper nutrition and lifestyle changes. Only then, through reduced caloric intake and increased physical activity, can positive outcomes be achieved. Weight loss is the treatment of choice for individuals with impaired glucose tolerance and in the early stages of T2DM in obese patients. To foster internal motivation in obese patients with T2DM, the organization of a broad network of educational centers for this patient group appears to be the most effective approach. In addition to providing essential knowledge about diabetes and teaching self-monitoring skills, these centers should focus heavily on promoting overall lifestyle changes: proper nutrition, increased physical activity, and the elimination of harmful habits. Such educational programs are already in place at the Endocrinology Research Centre of the Russian Academy of Medical Sciences .

When discussing proper nutrition with patients who have metabolic syndrome, it is important to emphasize not only reducing overall caloric intake to normalize body weight but also the effect of consumed foods on lipid and carbohydrate metabolism [30]. Dietary planning is a key factor in any therapy aimed at normalizing lipid levels. In many cases, dietary correction alone is sufficient to manage hyperlipidemia, particularly when it is caused by poor dietary habits or obesity [31]. For hyperlipidemia, a diet with a modified fat composition is recommended—this differs from a "regular" diet in having a lower overall fat content, especially in terms of saturated fats and cholesterol, while providing an adequate amount of polyunsaturated fats. A standard diet with a modified fat composition typically involves no more than 2,000 kcal per day, with 52% of calories from carbohydrates, 16% from proteins, and 32% from fats, of which saturated fats should make up no more than one-third of the total fat intake. Cholesterol intake should not exceed 300 mg per day. Replacing animal fats with vegetable fats can reduce cholesterol levels by up to 20%. Certain types of

dietary fiber enhance fecal excretion of bile acids and thus contribute to lowering cholesterol levels. Weight loss combined with reduced salt intake (no more than 5 grams per day, which equals about one level teaspoon) may be sufficient to treat mild arterial hypertension. These lifestyle measures are just as essential even when pharmacological therapy is prescribed.

An increase in physical activity is the second most important non-pharmacological factor influencing all components of metabolic syndrome. By effectively increasing energy expenditure, physical exercise helps normalize energy balance and promote weight loss. Moderate, regular physical activity in the fresh air positively impacts serum lipid levels: triglyceride (TG) levels and low-density lipoproteins (LDL) decrease, while high-density lipoproteins (HDL) increase, and the activity of lipoprotein lipase rises. Naturally, the permissible level of exercise depends on whether the patient has coronary artery disease (CAD), but even in such cases, all patients with hyperlipidemia should be encouraged to engage in physical activity to the extent that their condition allows. Patients with metabolic syndrome may be advised to participate in regular moderate-intensity aerobic activities (such as walking, swimming, cross-country skiing, or cycling) for 30–60 minutes, 3–7 times per week, along with any feasible set of physical exercises.

However, in treating patients with metabolic syndrome, it is often difficult to achieve the desired outcomes using only non-pharmacological interventions. When selecting a medication, it is important to consider its effect on the pathogenic mechanisms underlying insulin resistance syndrome. For instance, in patients with type 2 diabetes mellitus (T2DM) and excess body weight, it is reasonable to prescribe medications that increase peripheral tissue sensitivity to insulin and inhibit gluconeogenesis in the liver . At present, there are ongoing searches and clinical trials for drugs with the following mechanisms of action: increasing insulin sensitivity in tissues (such as thiazolidinedione and dichloroacetate derivatives); inhibiting fatty acid oxidation; inhibiting lipolysis (e.g., acipimox, adenosine analogs); inhibiting gluconeogenesis; and β3-adrenoreceptor agonists.

Metformin, a drug from the biguanide class introduced back in 1957, has been receiving renewed attention recently in light of the metabolic syndrome theory [32]. The drug's impact on glucose metabolism is thought to be due to the following mechanisms:
a) reducing peripheral insulin resistance and thereby improving glucose utilization in the liver, muscles, and adipose tissue;
b) suppressing hepatic gluconeogenesis;
c) slowing intestinal glucose absorption.

Metformin also has a hypolipidemic effect, observed both in patients with T2DM and in individuals with normal glucose tolerance who are obese (or not), as well as in those with dyslipidemia and arterial hypertension. During metformin therapy, TG levels decrease significantly. In individuals with hypertriglyceridemia, total cholesterol levels also decline, which appears to be secondary to the reduction in TG levels.

Troglitazone, a derivative of thiazolidinedione, has shown high effectiveness in normalizing carbohydrate metabolism in individuals with impaired glucose tolerance (IGT) and T2DM. However, clinical trials have raised serious concerns due to observed cases of hepatotoxicity. The drug requires further investigation.

Given that enhanced oxidation of saturated fatty acids (SFAs) plays a significant role in the development and maintenance of insulin resistance, there is an ongoing search for drugs that influence this mechanism. For instance, alkylglycidate derivatives—clomoxir, etomoxir, and 2-TDGA—are specific irreversible inhibitors of the enzyme carnitine palmitoyltransferase I, thereby preventing intramitochondrial oxidation of SFAs into acetyl-CoA. Research using the euglycemic clamp method demonstrated an impressive 33% increase in insulin-mediated glucose uptake after a single 50 mg dose of etomoxir in individuals with type 2 diabetes mellitus (T2DM). Drugs from this group are currently undergoing clinical trial phases.

Drugs from other pharmacological groups are still in the development stages or in early clinical trials.Considering the pathophysiological mechanisms of T2DM, it becomes clear that the early

prescription of sulfonylurea drugs is not always pathogenetically justified. These medications stimulate pancreatic β-cells, thereby exacerbating pre-existing hyperinsulinemia (which, at a certain stage of the disease, helps compensate for insulin resistance). However, some researchers suggest that over time this stimulation may lead to β-cell reserve depletion.

In 2006, the World Health Organization (WHO) Expert Committee on Hypertension recommended that, when deciding on antihypertensive treatment, preference should be given to drugs that exert a positive effect on all cardiovascular risk factors or at least do not worsen them. When choosing antihypertensive medications for patients with metabolic syndrome, it is important to consider their effects on existing disturbances in carbohydrate and lipid metabolism [33]. For example, thiazide diuretics—widely used for their effectiveness and low cost—are associated with several unfavorable metabolic effects: they impair carbohydrate metabolism in diabetic patients and negatively affect the lipid profile by increasing levels of total cholesterol, LDL cholesterol, and triglycerides. Unlike the aforementioned sulfonamide thiazide diuretics, the non-thiazide diuretic indapamide (Arifon) does not produce the listed adverse metabolic effects. Clinical trials of various durations in patients with type 2 diabetes mellitus (T2DM) have shown that Arifon does not affect plasma cholesterol or glucose levels and, moreover, reduces left ventricular myocardial hypertrophy.

A number of studies have demonstrated a link between the use of beta-blockers and worsening of carbohydrate metabolism in both diabetic and non-diabetic individuals. By inhibiting glycogenolysis in muscles and lipolysis in adipose tissue, beta-blockers may exacerbate hypoglycemic conditions in diabetic patients. Additionally, non-selective beta-blockers may blunt clinical symptoms of hypoglycemia, hindering timely intervention. Placebo-controlled studies have shown that selective beta-blockers (e.g., metoprolol and atenolol) reduce tissue sensitivity to insulin. Both selective and non-selective beta-blockers can increase triglyceride levels and reduce HDL cholesterol, though these effects are less pronounced when using low doses of selective beta-blockers. It is important to note that beta-blockers, despite these drawbacks, are often essential components of combination therapy for arterial hypertension.

Drugs that do not negatively affect carbohydrate and lipid metabolism include ACE inhibitors (e.g., enalapril, perindopril, ramipril), calcium channel blockers (e.g., verapamil, diltiazem, amlodipine, isradipine), and angiotensin II receptor blockers (e.g., losartan). ACE inhibitors are especially beneficial in treating diabetic nephropathy due to their ability to reduce intraglomerular renal hypertension and thus decrease microalbuminuria.

The positive impact of the alpha-adrenoblocker prazosin on lipid profiles was first noted by R. Leren in 1987 [34], but its use was limited due to side effects such as tachycardia, orthostatic hypotension, dry mouth, and impotence. However, newer second-generation drugs with longer duration of action—doxazosin (Tonocardin, Cardura) and terazosin—are associated with significantly fewer adverse effects. These medications are indicated for patients with concurrent hypertension and hyperlipidemia. By activating tissue lipoprotein lipase, this group of drugs helps lower cholesterol, triglycerides, and LDL levels, while improving the HDL/total cholesterol ratio.Clamp-method studies have shown that long-term use of alpha-adrenoblockers and ACE inhibitors enhances insulin sensitivity in peripheral tissues. ACE inhibitors, calcium channel blockers, alpha-adrenoblockers, and beta-blockers with intrinsic sympathomimetic activity (e.g., oxprenolol, pindolol) contribute to the reduction of left ventricular hypertrophy, which is also clinically beneficial.A new centrally acting antihypertensive drug is moxonidine (Cynt). Its mechanism of action is based on selective inhibition of imidazoline α2-receptors, which reduces both central and peripheral sympathetic activity, thereby lowering blood pressure. Clinical trials have shown that moxonidine does not disrupt metabolic parameters in patients with diabetes or lipid metabolism disorders. Thus, moxonidine appears to be suitable for use in these patient groups, provided contraindications are taken into account. It is important to emphasize that effective treatment of hypertension often requires combination therapy using agents from different pharmacological classes. In everyday practice, however, one drug is frequently replaced by another from a different group, a strategy that contradicts modern approaches to hypertension management. The importance of issues related to hyperlipidemia and ischemic heart

disease (IHD) highlights the need for targeted screening of patients to identify this risk factor in order to prevent premature death from IHD or loss of work capacity. The current situation in clinical practice, where blood lipid monitoring is conducted very rarely and only in a small portion of the adult population, is unacceptable. According to modern guidelines, cholesterol and triglyceride (TG) levels should be monitored in every adult patient.For individuals without IHD risk factors, desirable levels are cholesterol below 6.5 mmol/L and TG below 2.5 mmol/L. For patients with risk factors for IHD or a high likelihood of hyperlipidemia, active screening and more frequent regular monitoring (every 6 months) are recommended. This category includes individuals with:

- close relatives with hyperlipidemia,
- xanthelasmas or xanthomas,
- lipid arc of the cornea before age 60,
- relatives with IHD before age 60,
- arterial hypertension,
- diabetes mellitus,
- chronic kidney disease,
- presence of IHD,
- peripheral vascular disease,
- cerebrovascular disease, including cases after coronary artery bypass surgery or angioplasty.

For such individuals, target values are cholesterol below 5.2 mmol/L and TG below 2 mmol/L [35]. When total cholesterol exceeds 6.5 mmol/L, a full lipid profile analysis is warranted, and if necessary, drug therapy should be initiated. In patients with IHD, pharmacological treatment of hyperlipidemia should begin when total cholesterol is above 5.2 mmol/L.Drug correction of hyperlipidemia should always be combined with dietary therapy and never used as a stand-alone alternative treatment. Diet alone may be sufficient for most patients with moderate hyperlipidemia, but in severe cases, it is not effective enough. In patients with diabetes mellitus, carbohydrate metabolism must be compensated to normalize lipid metabolism. Improvement in glycemic control is often accompanied by a reduction in TG and cholesterol levels.All lipid-lowering drugs can be divided into two groups, depending on whether they primarily act on triglyceride-rich or cholesterol-rich lipoproteins. The spectrum of action among different classes of drugs may overlap, which is utilized in the treatment of various types of hyperlipidemia.

Drugs that Lower Cholesterol Levels

Anion exchange resins: cholestyramine, colestipol hydrochloride.
Their mechanism of action involves binding bile acids in the intestinal lumen, thereby preventing their reabsorption and enhancing their fecal excretion. As a result, the synthesis of bile acids is significantly increased, leading to a higher demand for cholesterol by liver cells. This is accompanied by a decrease in low-density lipoprotein cholesterol (LDL-C) levels.

HMG-CoA reductase inhibitors (statins): lovastatin, simvastatin, pravastatin, fluvastatin.
These drugs inhibit cholesterol synthesis at an early stage (the formation of mevalonic acid). Currently, statins are considered the most potent class of cholesterol-lowering agents. Data from a Scandinavian study demonstrated a 30% reduction in overall mortality among patients with ischemic heart disease (IHD) during long-term treatment with simvastatin.

Drugs with a Greater Effect on Triglyceride Levels

Fibrates: clofibrate, bezafibrate, gemfibrozil, and the most potent agents — ciprofibrate and fenofibrate.Fibrates are capable of increasing HDL cholesterol levels and reducing fibrinogen levels. Fenofibrate also has the ability to reduce uric acid levels.Nicotinic acid and its derivatives (nicofuranose and acipimox) could be used more widely if not for their frequent side effects (facial flushing, headache, rash, and gastrointestinal disturbances).The use of fish oil-containing preparations for the prevention of atherosclerosis remains questionable [36]. Despite their clearly

positive impact on hypertriglyceridemia, these drugs containing polyunsaturated fatty acids are ineffective in treating primary hypercholesterolemia and may even worsen dyslipidemia in patients with diabetes mellitus.The selection and prescription of a hypolipidemic drug is a serious task. Long-term, often lifelong treatment and the risk of serious adverse effects create significant concern among practicing physicians. It is essential that every clinician clearly understands the importance of this issue, the goals of hypolipidemic therapy, and has clear practical guidelines for diagnosing and treating hyperlipidemia.However, it is already evident that in patients with established atherosclerotic disease or at high risk of its development (such as in those with metabolic syndrome), if non-pharmacological interventions fail to produce the desired effect, drug therapy for hyperlipidemia is absolutely indicated. In such cases, the potential benefits of lipid-lowering treatment outweigh the risks of possible side effects.

Until recently, each component of metabolic syndrome was treated separately, rather than focusing on the patient as a whole, which is clearly not in the patient's best interest. In traditional clinical practice, hypertension is managed by a general practitioner, coronary artery disease by a cardiologist, and diabetes by an endocrinologist. The depth of medical knowledge in each specialty has even led to the emergence of niche specialists such as "lipidologist" or "diabetologist." However, remembering the principle that we must treat the patient and not isolated diseases, we must acknowledge the need for cross-disciplinary competence. For a diabetologist, this means gaining a deeper understanding of internal medicine, while for an internist, it means learning more about insulin effects and glycemic control.

Conclusion

In conclusion, we summarize the recommended approach to examining and treating patients with type 2 diabetes mellitus (T2DM) or impaired glucose tolerance (IGT):To detect other manifestations of metabolic syndrome in these patients, it is necessary to measure body mass index (BMI), the waist-to-hip ratio, blood pressure, total cholesterol, and triglyceride levels. If cholesterol exceeds 6.5 mmol/L, a full lipid profile including HDL cholesterol should be performed.In treating this patient category, non-pharmacological approaches must receive adequate attention: dietary modification, weight reduction, and increased physical activity.When choosing antihypertensive drug therapy, it is important to consider the medication's impact on all components of metabolic syndrome, particularly glucose and lipid metabolism.Pharmacological treatment of hyperlipidemia is clearly indicated in individuals with confirmed atherosclerotic disease or at high risk for it (as is the case in patients with metabolic syndrome) when non-drug measures do not yield adequate results. In such cases, the benefits of lowering lipid levels outweigh the risks of potential adverse effects.

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