



## VISCERAL FATTENING: PATHOGENESIS, CLINICAL SIGNIFICANCE, AND SCIENTIFIC AND PRACTICAL FOUNDATIONS OF ITS REDUCTION.

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Article history:	Abstract:
<b>Received:</b> 20 <sup>th</sup> February 2026 <b>Accepted:</b> 14 <sup>th</sup> March 2026	This review article analyzes the pathogenetic mechanisms of visceral fat accumulation, its clinical significance, and scientifically based methods for reducing it. The relationship between visceral fat and insulin resistance, chronic low-intensity inflammation, dyslipidemia, arterial hypertension, non-alcoholic fatty liver disease, and cardiovascular diseases is highlighted. The significance of waist circumference and other anthropometric indicators for assessing visceral adiposity, as well as the role of diet, aerobic and strength exercises, sleep, and stress management in reducing visceral fat, were also summarized.
<b>Keywords:</b> Visceral fat, abdominal obesity, insulin resistance, adipokines, inflammation, metabolic syndrome, NAFLD, prevention.	

**INTRODUCTION.** Overweight has become one of the most pressing global medical and social problems of the 21st century. Of particular clinical importance is not only the total volume of adipose tissue but also its distribution in the body, particularly the increase in the amount of visceral fat located around internal organs [1-4]. Visceral fat, unlike subcutaneous fat, has high metabolic and endocrine activity, actively secretes adipokines, inflammation mediators, and free fatty acids [5, 8, 9]. Therefore, visceral adiposity is considered in modern clinical medicine as the central pathogenetic link of cardiometabolic risk [1, 3, 4].

Numerous studies have shown a correlation between visceral fat and insulin resistance, dyslipidemia, hypertension, non-alcoholic fatty liver disease, and atherosclerotic complications [3, 6, 7, 25, 27, 30]. At the same time, in practice, focusing solely on the body mass index is insufficient, as some people, even with relatively normal BMI, may have a high level of visceral fat [10-12]. In this regard, assessing the accumulation of visceral fat, understanding its consequences, and generalizing scientifically based approaches to its reduction remain relevant.

**THE PURPOSE OF THE STUDY.** The purpose of this review article is to analyze the modern pathogenetic mechanisms of visceral fat accumulation, assess its clinical significance, and summarize scientifically based methods for its reduction.

**MATERIALS AND METHODS OF RESEARCH.** The article is an overview. Foreign and international scientific publications published in 1999-2025 were analyzed. The analysis included systematic reviews, meta-analyses, clinical studies, position statements, and consensus documents. When selecting sources, priority was given to the following areas: visceral adiposity and insulin resistance, adipokines and inflammation, cardiovascular risk, NAFLD, anthropometric assessment, physical activity, diet therapy, sleep, and stress factors.

During the analysis, the scientific significance of the publications, their direct connection to the topic, and the possibilities of practical application were taken into account. For the review article, special attention was paid to ensuring that the sources covered various areas: fundamental pathogenesis, clinical complications, diagnostic approaches, as well as preventive and therapeutic measures.

**OBTAINED RESULTS AND DISCUSSION.** The analysis results show that the accumulation of visceral fat is a multifactorial and gradually developing process. Excessive calorie intake, especially when consuming refined carbohydrates and products with high energy density, combined with a sedentary lifestyle, leads to a positive energy balance. As a result, triglycerides begin to accumulate in the body, primarily in intra-abdominal depots [1-4, 13-16]. However, from a clinical perspective, the greatest risk is not related to the total amount of fat, but rather to the predominance of visceral adipose tissue [1, 2].

As the hypertrophy of visceral adipocytes increases in the tissue, relative hypoxia develops. This condition intensifies macrophage infiltration and activates the pro-inflammatory signaling pathways [5, 24]. As a result, the secretion of TNF- $\alpha$ , IL-6, and other mediators increases, forming a chronic low-intensity inflammation [4, 5]. This condition deepens metabolic disorders and further increases the pathogenetic significance of visceral obesity.

One of the most important pathogenetic consequences of visceral fat is insulin resistance. Excessive flow of free fatty acids into the liver through the portal bloodstream reduces the liver's sensitivity to insulin, increases gluconeogenesis, and disrupts lipid metabolism [3, 6, 7]. This creates an excessive functional load on  $\beta$ -cells and forms prerequisites for the development of type 2 diabetes mellitus [3, 4, 6]. Simultaneously, a decrease in adiponektin levels and an increase in leptin resistance lead to adipokine imbalance, further worsening insulin sensitivity [8, 9, 26].

The negative impact of visceral adiposity on the cardiovascular system is also significant. It is associated with dyslipidemia, endothelial dysfunction, arterial hypertension, and atherosclerotic changes, increasing the risk of coronary heart disease and stroke [1, 2, 30]. For this reason, some authors propose to consider the waist circumference in clinical practice as a "vital indicator" [12]. Waist circumference, waist-to-height ratio, and visceral adiposity index are simple, accessible, and clinically beneficial diagnostic indicators [10-12].

The connection between visceral fat and liver pathology is also significant. Non-alcoholic fatty liver disease is closely related to visceral obesity, insulin resistance, and metabolic syndrome. Free fatty acids and inflammatory signals coming from the visceral depots can lead to liver steatosis, and subsequently, to the development of steatohepatitis [7, 25, 27, 28]. In this regard, reducing visceral fat is important not only for reducing cardiovascular risk but also for preventing liver diseases.

Psycho-emotional stress and neuroendocrine factors also affect the accumulation of visceral fat. Prolonged increased cortisol secretion is associated with the central fat deposition type [18, 29]. Chronic stress activates the hypothalamic-pituitary-adrenal axis, altering appetite, insulin sensitivity, and fat tissue distribution. Therefore, it is advisable to consider visceral obesity not only as a problem of nutrition or lack of physical activity, but also as a biopsychosocial problem.

Lack of sleep is also related to abdominal obesity. Short sleep duration changes the balance of leptin and grelin hormones, increases appetite, and can lead to excessive calorie intake [19, 20]. For this reason, sleep hygiene is considered an important supplementary component of visceral fat reduction programs.

Diet therapy is crucial for reducing visceral fat. Calorie-deficient diets, especially those based on limiting refined carbohydrates and optimizing the proportion of protein and dietary fiber, can contribute to the reduction of abdominal and visceral fat [13-16]. A number of studies show that low-carbohydrate diets effectively reduce visceral fat in the short and medium term [13-15]. However, in long-term practice, it is important to consider the sustainability of the diet, its individual tolerance, and the overall metabolic state [16].

Data on physical exercises indicate that aerobic exercises are particularly effective in reducing visceral fat [17, 21, 22]. Strength training is important for maintaining muscle mass and maintaining basic energy expenditure, while aerobic exercises can have a stronger effect on reducing visceral and even liver fat [17, 22]. The best results are typically achieved through a comprehensive individualized approach that combines diet, aerobic and strength loads, sleep hygiene, and stress management [21-23, 30].

Thus, the accumulation of visceral fat is not just an aesthetic problem, but one of the pathogenetic bases of many non-communicable diseases. Its early diagnosis, assessment of risk factors, and the application of scientifically based preventive and therapeutic measures are of great importance for clinical practice.

**CONCLUSION.** Accumulation of visceral fat is one of the most important markers and pathogenetic factors of cardiometabolic risk. It creates prerequisites for the development of insulin resistance, chronic low-intensity inflammation, adipokine imbalance, arterial hypertension, non-alcoholic fatty liver disease, and atherosclerotic processes. The waist circumference and nearby anthropometric indicators are among the simple, accessible, and clinically beneficial methods for early assessment of visceral adiposity.

To reduce visceral fat, the most appropriate strategy is a comprehensive approach that includes rational and stable nutrition, regular aerobic and strength exercises, sleep hygiene, and stress management. This approach not only improves body composition but also helps reduce the overall cardiometabolic risk. Therefore, early detection of visceral fat and strengthening of medical and social measures for its prevention remain highly relevant.

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