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The Impact of the Ketogenic Diet on Selected Components of Metabolic Syndrome: A Narrative Review of Literature Published between 2020 and 2026

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Abstract

Background: Metabolic syndrome is a major public health concern associated with an increased risk of cardiovascular events. The ketogenic diet has gained increasing attention as a potential nutritional strategy in the management of metabolic syndrome.

Aim: This narrative review aimed to analyze publications from 2020 to 2026 to assess the impact of the ketogenic diet on selected components of metabolic syndrome.

Materials and Methods: A literature search was conducted in PubMed and Google Scholar databases. Original studies, systematic reviews, meta-analyses and randomized controlled trials published in English and in Polish between 2020 and 2026 were included. The search strategy used combinations of keywords related to the ketogenic diet, metabolic syndrome, obesity, type 2 diabetes, dyslipidemia and hypertension.

Results: The available evidence does not allow for an unequivocal assessment of the effect of the ketogenic diet on selected parameters of metabolic syndrome. Short-term benefits were observed in body weight reduction, glycemic control and some aspects of lipid metabolism. However, no significant effect was consistently demonstrated for blood pressure reduction or lowering of low-density lipoprotein cholesterol. Some studies even suggested potentially adverse effects on lipid metabolism in specific patient subgroups.

Conclusions: The ketogenic diet may serve as an adjunctive strategy in the management of metabolic syndrome, particularly regarding body weight reduction, glycemic control and lowering triglyceride levels. Nevertheless, further studies are required to evaluate its long-term effects and safety.

Keywords: Ketogenic diet; metabolic syndrome; type 2 diabetes mellitus; abdominal obesity; hypertension; dyslipidemia

1. Introduction

The ketogenic diet (KD) is currently one of the most popular dietary models. According to Google Trends data, in 2020 it was among the most frequently searched dietary patterns, alongside vegetarianism, veganism and the gluten-free diet (1). The ketogenic diet, also referred to as the keto diet, involves a marked restriction of dietary carbohydrates and a high intake of fats. Simple and complex carbohydrates are substantially reduced, usually to <50 g per day (2). In the classical ketogenic diet, fats may account for as much as 90% of total energy intake. Modified versions allow for a less restrictive approach and a greater proportion of energy derived from protein. The principle of this high-fat, low-carbohydrate dietary pattern is to shift metabolism from glucose-based energy production toward the use of fatty acids and ketone bodies as the primary energy source. Although the diet was initially recommended for patients with epilepsy, its potential benefits in the treatment of metabolic disorders such as type 2 diabetes mellitus, obesity and dyslipidemia are currently being investigated (2).

For many years, obesity, hypertension, disturbances in carbohydrate metabolism and lipid abnormalities were considered separate disease entities related to lifestyle. Currently, increasing emphasis is placed on the complexity and interrelationship of these conditions. This constellation of abnormalities significantly increases cardiovascular risk (3). It is estimated that more than 20% of the population in Western countries meets the diagnostic criteria for metabolic syndrome, defined as the coexistence of cardiovascular risk factors including central obesity, impaired carbohydrate metabolism, hypertension and dyslipidemia (3,4). The aim of this study was to review the current literature on the effects of the ketogenic diet on selected components of metabolic syndrome, particularly body weight, blood pressure, carbohydrate metabolism and lipid metabolism.

2. Materials and Methods

In June 2026, a literature review was conducted using the PubMed and Google Scholar databases. Publications concerning the effects of the ketogenic diet on parameters of metabolic syndrome and published between 2020 and 2026 were analyzed. The following keywords were used in the search process: "ketogenic diet", "diabetes mellitus type 2", "abdominal obesity", "dyslipidemia", "hypertension", and "metabolic syndrome". The logical operators AND and OR were applied. Polish-language equivalents of these terms were also included.

Publications in Polish and English were considered, including observational studies, clinical studies, randomized controlled trials, review articles, systematic reviews, and meta-analyses. Conference reports, case reports, publications unavailable in full text, and articles published in languages other than Polish and English were excluded. Narrative publications concerning the mechanisms of action of the ketogenic diet and the definition of metabolic syndrome were also considered. Information on selected studies is summarized in Table 1. Due to space limitations only studies of clinical and methodological relevance were included in the table.

Author, year	Study type	Number of participants of studies analyzed	Duration	Main findings
Wang et al. (2024)	Meta-analysis of RCTs	n = 1278 participants, 27 studies	4-96 weeks	↓body weight, ↑HDL, ↓TG, no effect on WC and SBP, ↓glucose in blood, ↓DBP
Chong et al. (2022)	Meta-analysis of RCTs	n = 611 participants, 8 studies	3-24 months (mostly up to 12 months)	↓body weight, ↓WC, ↓HbA1c, ↓TG, ↑HDL
Parry-Strong et al. (2022)	Systemic review and meta-analysis	n = 606 participants, 8 studies	3-24 months (mostly up to 12 months)	↓/ no effect on HbA1c, no effect on body weight
Patikorn et al. (2023)	Umbrella review	17 meta-analyses	8-36 weeks	↓TG, ↓LDL, ↓body weight, ↓ HbA1c, ↑total cholesterol
RCT - randomized controlled trial; HDL - high-density lipoprotein; LDL - low-density lipoprotein; TG - triglycerides; SBP - systolic blood pressure; DBP - diastolic blood pressure; WC - waist circumference; HbA1c - glycated hemoglobin.				

Table 1. Results of selected publications included in the review.

3. Results

3.1. Abdominal obesity

Central obesity is one of the main components of metabolic syndrome and an independent risk factor for cardiovascular events. It is most commonly assessed on the basis of waist circumference (WC), hip circumference (HC) and the waist-to-hip ratio (WHR). In the twenty-first century, a substantial increase in obesity prevalence has been observed, particularly in Western societies. According to WHO data, nearly 16% of the global population had a BMI >=30 kg/m² in 2023 (5). The analyzed studies suggest that the ketogenic diet may favorably affect anthropometric parameters associated with abdominal obesity, although not all studies confirm its superiority over other dietary models.

A meta-analysis published in 2024, including 27 randomized clinical trials with 1,278 participants, showed that the use of KD was associated with a significant reduction in body weight and BMI. The mean difference in body weight between the control group and the ketogenic diet group was -2.59 kg (95% CI: -3.90 to -1.28; I^2: 87.4%), and the mean difference

in BMI was -1.59 kg/m² (95% CI: -2.32 to -0.86; I^2: 84.5%). An important limitation in the interpretation of these findings is the high heterogeneity of the results. Notably, the authors did not confirm a statistically significant effect of the intervention on waist circumference reduction (6).

Similarly, a 4-week interventional study conducted in 24 Chinese women aged 19-25 years with overweight or obesity demonstrated a beneficial effect of the ketogenic diet on weight loss (-2.9 ± 2.1 kg) and BMI (-1.1 ± 0.7 kg/m²). In contrast to the previously discussed meta-analysis, the intervention also significantly reduced waist circumference (-4.0 ± 3.2 cm), hip circumference (-2.5 ± 2.3 cm), and WHR (-0.02 ± 0.03). However, the study was limited by its small sample size, short follow-up period and lack of a control group (7)

A 12-week interventional study involving 98 women (64 with obesity, 23 with overweight, and 11 with normal body weight) aged 20 to 50 years reported similar findings. Significant reductions in body weight, BMI, WC, HC and WHR were observed in all groups ($p < 0.001$). The greatest reduction in body weight was found in the obesity group, from 97.99 ± 11.48 kg to 81.94 ± 10.95 kg. Limitations of this study included the lack of menstrual cycle monitoring and the absence of a control group (8).

Overall, the available evidence suggests that the ketogenic diet may contribute to reductions in body weight and anthropometric parameters associated with obesity, such as waist circumference, hip circumference, and WHR. However, it should be emphasized that most available studies have a short follow-up period and some of the favorable effects may result from the calorie deficit accompanying the diet. In addition, heterogeneity across studies limits a clear assessment of the effectiveness of KD in reducing abdominal obesity.

3.2. Disturbances in carbohydrate metabolism

Disturbances in carbohydrate metabolism are a key component of metabolic syndrome and include insulin resistance, impaired fasting glucose, and type 2 diabetes mellitus. Clinical assessment of glycemic control includes fasting blood glucose concentration, glycated hemoglobin (HbA1c) and insulin resistance indices such as HOMA-IR. Disturbances in carbohydrate metabolism constitute an important risk factor for vascular events and are associated with poorer outcomes in patients after ischemic stroke (9).

A 2022 meta-analysis of eight randomized clinical trials involving patients with overweight/obesity and type 2 diabetes mellitus showed that the ketogenic diet was associated with a significant reduction in HbA1c (SMD: -0.38; 95% CI: -0.61 to -0.16; $I^2 = 27\%$). No significant changes were observed in fasting glucose ($p = 0.74$), fasting insulin ($p = 0.07$), or HOMA-IR ($p = 0.14$). The authors also emphasized the beneficial effect on body weight reduction. A limitation of this study was the absence of participants from East Asian countries, with most data derived from Caucasian populations (10).

A 2020 meta-analysis including 13 publications from six countries also confirmed a beneficial effect on carbohydrate metabolism. The ketogenic diet significantly reduced fasting blood glucose (-1.29 mmol/L; 95% CI: -1.78 to -0.79) and HbA1c (-1.07%; 95% CI: -1.37 to -0.78), which, according to the authors, may facilitate the achievement of therapeutic goals in diabetes management. As in previous studies, a favorable effect on body weight reduction was also observed (11).

In contrast, a 2022 meta-analysis including eight studies and 606 participants with overweight or obesity, aged 51-66 years, with prediabetes or type 2 diabetes mellitus did not demonstrate a clearly beneficial effect of a very low-carbohydrate diet on carbohydrate metabolism parameters. In two of the analyzed studies, HbA1c decreased by 0.65% after 12 months compared with the control diet (95% CI: -0.99 to -0.31; $p < 0.01$), whereas in four studies the decrease was 0.01% (95% CI: -0.22 to 0.25; $p = 0.91$). No significant heterogeneity was found in either analysis. No significant favorable effect of the ketogenic diet was observed on body weight, fasting glucose, HOMA-IR, waist circumference, body fat content or most lipid parameters (12).

These findings suggest that the ketogenic diet may improve glycemic control, particularly by lowering HbA1c and fasting glucose in the short term. However, the number of studies assessing the use of KD for longer than 12 months remains limited. The lack of long-term data makes it difficult to draw firm conclusions regarding its effectiveness in patients with disturbances in carbohydrate metabolism.

3.3. Hypertension

Due to population aging and the increasing prevalence of obesity and sedentary lifestyle, hypertension remains one of the most important public health challenges worldwide. This contributes to the burden of diseases associated with uncontrolled blood pressure, including cardiovascular events, kidney failure and retinal disease. Blood pressure assessment includes systolic blood pressure (SBP) and diastolic blood pressure (DBP). Hypertension is a multifactorial condition influenced by both genetic and environmental factors, such as obesity, smoking, excessive sodium intake, insufficient dietary potassium intake, physical inactivity and alcohol consumption (13,14).

Despite numerous studies suggesting beneficial effects of the ketogenic diet on body weight reduction and improvement in carbohydrate metabolism, findings regarding its effect on blood pressure remain inconclusive. A 2023 meta-analysis including 23 randomized controlled trials with a total of 1,664 patients found no significant effect of the ketogenic diet on systolic blood pressure (WMD: -0.87 mmHg; 95% CI: -2.05 to 0.31; $p = 0.148$) or diastolic blood pressure (WMD: -0.11 mmHg; 95% CI: -1.14 to 0.93; $p = 0.843$). No significant differences were found according to country, study duration (4 to 96 weeks), sex, health status or dietary fat content. Only for DBP did the authors note that significant heterogeneity ($I^2 = 42.9\%$; $p = 0.014$) might depend on the percentage of fat in the diet (15).

Although KD may exert beneficial effects on carbohydrate and lipid metabolism, these do not necessarily translate into a clear improvement in blood pressure control. A meta-analysis including 27 studies, seven of which addressed hypertension, found that significant blood pressure reduction was observed only for DBP (MD: -1.41 mmHg; 95% CI: -2.57 to -0.26; I^2: 49.1%), whereas the reduction in SBP was not statistically significant (MD: -1.27 mmHg; 95% CI: -2.89 to 0.35; I^2: 25.6%) (6). However, a DBP decrease of 1.41 mmHg is of limited clinical relevance.

The findings regarding the effect of the ketogenic diet on SBP and DBP reduction are therefore inconclusive. An umbrella review including 17 meta-analyses suggested a possible effect of KD on parameters of metabolic syndrome, including hypertension; however, the authors

emphasized numerous limitations of the available evidence, including low methodological quality, a median study duration of only 13 weeks and the fact that the included populations mainly represented selected patient groups, such as those with overweight or obesity, type 2 diabetes mellitus, metabolic syndrome or cancer (16).

Thus, the currently available evidence does not provide sufficient support for a significant, direct effect of the ketogenic diet on blood pressure values. The slight reduction in DBP observed in some studies may result primarily from accompanying weight loss and improvements in metabolic parameters. The small number of long-term studies precludes a definitive assessment of the effectiveness of the ketogenic diet in the treatment of hypertension.

3.4. Lipid metabolism disorders

Disorders of lipid metabolism (dyslipidemia) include abnormalities in triglycerides (TG), low-density lipoproteins (LDL), and high-density lipoproteins (HDL). They constitute one of the diagnostic criteria for metabolic syndrome. Dyslipidemia substantially increases the risk of cardiovascular disease, particularly atherosclerotic disease and ischemic events resulting from plaque formation. Of greatest clinical relevance is the reduction of LDL cholesterol concentration (17).

Available publications do not provide unequivocal findings regarding the effect of the ketogenic diet on lipid metabolism. Both the meta-analysis by Wang et al. (2024) and the umbrella review by Patikorn et al. (2023) demonstrated an increase in HDL (Wang: MD: 0.16 mmol/L; 95% CI: 0.09 to 0.23; I^2: 86.7%) and a decrease in TG (Wang: MD: -0.20 mmol/L; 95% CI: -0.29 to -0.11; I^2: 72.2%). The effect of KD on LDL remains unclear, as no statistically significant difference in serum LDL concentration after the intervention was demonstrated. The analyzed studies also had several limitations, including high heterogeneity, short overall duration of follow-up, and modest quality of the available evidence (6,16).

However, some studies indicate that LDL may increase in patients following a ketogenic diet. A review published in 2026, including two meta-analyses, demonstrated an increase in LDL concentration among normal-weight adults following KD, ranging from 41.40 to 41.76 mg/dL over 3 to 6 weeks. The data were characterized by high heterogeneity, with LDL changes ranging from 18 to 70 mg/dL. Interestingly, no LDL increase was observed in patients with overweight or class I obesity, whereas in patients with class II obesity or higher, LDL was reduced by 6.7 mg/dL. The authors emphasized that although KD may have potentially favorable effects on body weight, glycemia, and triglyceride concentration, there is currently insufficient evidence to consider the observed increase in LDL clinically irrelevant. Particular attention was drawn to the especially vulnerable subgroup of lean and physically active patients, referred to as lean mass hyper-responders, in whom marked elevations in LDL, high HDL levels and low TG levels are observed. The authors stressed the need for further studies to assess the cardiovascular implications of this lipid profile (18).

The available evidence suggests that KD may improve selected lipid parameters, especially by increasing HDL concentration and reducing TG levels. Its effect on LDL remains inconclusive and requires further investigation. Some publications even suggest an adverse effect on LDL concentration, particularly in patients with normal BMI and high physical activity levels.

A definitive assessment is additionally hampered by the short duration of follow-up and high heterogeneity of the studied populations. The effects of KD on individual components of metabolic syndrome are summarized in Table 2.

Parameter	**Effect of KD**	**Main limitations of assessment**
Body weight	Beneficial	Short follow-up, small study groups
Waist circumference	Probably Beneficial	Short follow-up, small study groups
HbA1c	Beneficial	Short follow-up
HOMA-IR	No clear effect	Limited number of studies
SBP	No clear effect	Limited number of studies
DBP	Slightly beneficial	Limited clinical relevance
TG	Beneficial	Short follow-up, high heterogeneity
HDL	Beneficial	Short follow-up, high heterogeneity
LDL	No clear effect	Possible LDL increase in some patients
KD - ketogenic diet; HbA1c - glycated hemoglobin; HOMA-IR - Homeostatic Model Assessment for Insulin Resistance; SBP - systolic blood pressure; DBP - diastolic blood pressure; TG - triglycerides; HDL - high-density lipoprotein; LDL - low-density lipoprotein.		

Table 2. Impact of ketogenic diet on individual components of metabolic syndrome

4. Discussion

Based on the currently available evidence, it is difficult to conclude unequivocally that the ketogenic diet has an overall beneficial effect on the health of patients with metabolic syndrome. Available data suggest that KD may positively influence selected components of metabolic syndrome, particularly through body weight reduction, improved glycemic control, lower triglyceride levels, and higher serum HDL concentration. However, its impact on blood pressure and serum LDL concentration remains unclear. Similar conclusions were reached by Nojek et al. (2024), who reported that ketogenic diet may contribute to weight reduction, improved glycemic control and favorable changes in selected lipid parameters in patient with metabolic disorder (21).

The available studies are characterized by substantial heterogeneity. Different authors applied different definitions of the ketogenic diet, with varying macronutrient proportions (19). An important limitation is the relatively short duration of follow-up, which in most studies did not

exceed 12 months, although follow-up length varied considerably across studies. In addition, many studies included small cohorts. The data were derived from populations in different regions of the world, which may also have influenced the observed outcomes. Adherence to the prescribed diet varied across patients, as did physical activity levels. Participants also differed in baseline health status, with many studies involving populations with overweight/obesity and type 2 diabetes mellitus.

Under standard dietary conditions, glucose is the principal energy substrate. Depending on metabolic demand, it is either directed into glycolysis and subsequent ATP-generating pathways or stored via glycogenesis. In KD, carbohydrate intake is insufficient to maintain these pathways as the predominant source of energy, and ketone bodies become the major alternative fuel. Low glucose availability leads to decreased insulin concentrations, which in turn stimulates triglyceride breakdown by lipase into free fatty acids (FFAs) and glycerol. FFAs are transported to the liver, the primary site of ketone body production. There, in the mitochondrial matrix of hepatocytes, beta-oxidation generates acetyl-CoA. Under normal conditions, acetyl-CoA would enter the Krebs cycle; however, because oxaloacetate is depleted through hepatic gluconeogenesis, acetyl-CoA is instead utilized for ketogenesis, including the formation of beta-hydroxybutyrate. Ketone bodies are subsequently used in the Krebs cycle within target tissues, mainly the heart, brain, and skeletal muscles, to generate energy required for normal functioning. Excess ketones are excreted in the urine, whereas acetone, a volatile by-product, is eliminated through the lungs. In addition, ketone bodies exert anti-inflammatory effects and act as signaling molecules (20). The ketogenic diet may also reduce appetite. Patients in ketosis do not appear to experience an increase in appetite despite body weight loss, which may be related to suppression of ghrelin secretion in individuals following KD (19).

The reduction in body weight observed in patients following KD may therefore depend on multiple mechanisms, including lower insulin levels, enhanced lipolysis, increased satiety, and modulation of inflammatory processes. Improvements in glycemic control and selected lipid parameters may also result from the biochemical mechanisms described above. Nevertheless, it should be emphasized that some of the observed benefits may also reflect intentional energy restriction and subsequent weight loss, rather than ketosis itself. It should also be noted that the quality and type of consumed fats, particularly the proportion of unsaturated and saturated fats, may substantially affect the studied parameters.

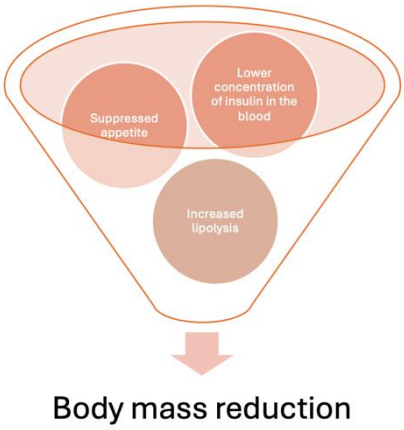

Figure 1. Proposed mechanisms by which the ketogenic diet may contribute to body weight reduction. Authors' own elaboration based on Roekenes, Martins (2021) and Malinowska, Żendzian-Piotrowska (2024).

Because the ketogenic diet is relatively restrictive, it carries a risk of nutritional deficiencies. Reduced carbohydrate intake may lower dietary fiber consumption, which in turn may contribute to constipation. Studies also suggest a potentially unfavorable effect of KD on LDL cholesterol concentration, especially in patients with normal BMI and high physical activity levels. Therefore, the choice of dietary model should take into account the individual characteristics of the patient, coexisting diseases, and the feasibility of long-term adherence. Patients should remain under regular medical supervision because of interindividual variability in response to KD. The main potential benefits and limitations are presented in Table 3.

Further studies, particularly long-term investigations, are needed to clarify the effects of KD on human health. Standardization of the definition of the ketogenic diet and a more precise classification of other low-carbohydrate diets are required. Future research should determine the effect of different types of dietary fat on specific anthropometric and biochemical parameters. Studies should also address outcomes such as mortality, hospitalization frequency, and the incidence of major adverse cardiovascular events (MACE). It is equally important to determine the effects of KD in individuals with normal body weight.

Potential benefits	**Potential risks**
Body mass reduction	Risk of nutritional deficiencies
Improved glycemic control	Low fiber intake
Reduced TG concentration	Constipation
Increased HDL concentration	Increased LDL concentration in some patients
Reduced appetite	Difficulty maintaining long-term adherence
KD - ketogenic diet; TG - triglycerides; HDL - high-density lipoprotein; LDL - low-density lipoprotein.	

Table 3. Potential benefits and risks associated with the ketogenic diet.

5. Conclusions

In summary, a definitive assessment of the effectiveness of the ketogenic diet on specific parameters of metabolic syndrome, or more broadly on overall health, is not currently possible. The ketogenic diet may represent a useful adjunctive tool in the management of selected components of metabolic syndrome. Several studies provide evidence of improvement in certain health parameters, particularly body weight reduction, glycemic control, and lower blood triglyceride levels. However, no significant effect has been consistently demonstrated for SBP, DBP or LDL. Interpretation of the findings is limited by the lack of long-term studies, high heterogeneity of the available data, and insufficient evidence regarding the effects of KD on cardiovascular events, hospitalization, or mortality. Further well-designed, methodologically robust studies are needed to define the role of the ketogenic diet in the management of metabolic syndrome.

Disclosure

Author Contributions:
Conceptualization: K.A.J., M.F.; Methodology: A.K., O.K., A.K.S.; Investigation: K.A.J., M.F., A.R.Ż., M.C-S.,J.Ł., K.J, K.B.; Data Curation: K.B., O.K; Writing –original draft: Introduction and Materials and Methods: K.J., A.K.; Obesity: K.A.J., J.Ł.; Hypertension: A.K.S.; Carbohydrate metabolism: A.K.Ż., M.C-S.; Lipid metabolism: K.B, O.K.; Results synthesis and Conclusions: K.A.J., M.F.; Writing –Review & Editing: K.A.J., A.K, M.F., A.K.S., A.R.Ż., M.C-S., J.Ł., K.J., K.B., O.K.

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During the preparation of this manuscript, the authors used ChatGPT and Claude for the linguistic refinement. These tools were used to support translation, improve language quality, enhance the clarity and consistency of the manuscript. The Authors have reviewed and edited the content as necessary and take full responsibility for the accuracy, integrity and scientific content of the publication.

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