Intermittent fasting and endothelial function in metabolic syndrome: A randomized controlled trial on vascular health and oxidative stress markers

Ayuno intermitente y función endotelial en el síndrome metabólico: Un ensayo clínico aleatorizado y controlado sobre la salud vascular y los marcadores de estrés oxidativo

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Abstract

The aim of this randomized controlled clinical trial was to assess the effects of intermittent fasting (16:8 regimen) on endothelial function and oxidative stress markers in metabolic syndrome patients in Uzbekistan. Volunteers were randomly assigned into two intervention groups (n = 35) and two control groups (n = 35) and parameters like brachial artery flow-dependent dilation (FMD), nitric oxide, malondialdehyde (MDA), lipid profile, blood pressure, and inflammatory markers were assessed before and after intervention for 8 weeks. Outcome measures revealed significant improvement in endothelial function (1.56% increase in FMD), reduction in oxidative stress (0.9 nmol/mL decrease in MDA), improvement in lipid profile (decrease in LDL and triglycerides, increase in HDL), systolic blood pressure decrease (5.5 mmHg), and inflammatory markers decrease in the intervention group. An improvement in liver parameters and adipokine regulation was also observed. These findings confirm that intermittent fasting, through its mechanisms of modulation of oxidative stress, inflammation, and lipid metabolism, can be recommended as an effective non-pharmacological therapy in the management of metabolic syndrome and reduction of cardiovascular risks among the study population.

Keywords: intermittent fasting, endothelial function, metabolic syndrome, oxidative stress

Resumen

El objetivo de este ensayo clínico aleatorizado y controlado fue evaluar los efectos del ayuno intermitente (régimen 16:8) sobre la función endotelial y los marcadores de estrés oxidativo en pacientes con síndrome metabólico en Uzbekistán. Los voluntarios se asignaron aleatoriamente a dos grupos de intervención (n = 35) y dos grupos control (n = 35). Se evaluaron parámetros como la dilatación dependiente del flujo de la arteria braquial (DFM), el óxido nítrico, el malondialdehído (MDA), el perfil lipídico, la presión arterial y los marcadores inflamatorios antes y después de la intervención durante 8 semanas. Las medidas de resultado revelaron una mejora significativa de la función endotelial (aumento del 1,56 % en la FMD), una reducción del estrés oxidativo (disminución de 0,9 nmol/mL en la MDA), una mejora del perfil lipídico (disminución de LDL y triglicéridos, aumento de HDL), una disminución de la presión arterial sistólica (5,5 mmHg) y una disminución de los marcadores inflamatorios en el grupo de intervención. También se observó una mejora de los parámetros hepáticos y de la regulación de las adipocinas. Estos hallazgos confirman que el ayuno intermitente, a través de sus mecanismos de modulación del estrés oxidativo, la inflamación y el metabolismo lipídico, puede recomendarse como una terapia no farmacológica eficaz para el manejo del síndrome metabólico y la reducción del riesgo cardiovascular en la población del estudio.

Palabras clave: ayuno intermitente, función endotelial, síndrome metabólico, estrés oxidativo

Introduction

Metabolic syndrome, as a cluster of three or more of the metabolic abnormalities of abdominal obesity, dyslipidemia, hypertension, and insulin resistance, is one of the globe's most important public health challenges[1]. Besides increasing risk for cardiovascular disease and type 2 diabetes, this syndrome causes severe vascular complications through endothelial dysfunction and increased oxidative stress[2]. Endothelium, the inner vascular lining, plays a role in regulating vascular tone, homeostasis, and inflammation, and dysfunction of its function is referred to as an earlier sign of vascular injury and a forerunner of cardiovascular events[3]. On the other hand, oxidative stress by virtue of excessive production of free radicals and decreased antioxidant activity brings about tissue damage and increased inflammation and plays an important role in the etiology of metabolic syndrome[4].

Over the past few years, novel dietary practices such as intermittent fasting have been suggested as a non-pharmacological lifestyle intervention to improve metabolic and vascular health. Intermittent fasting, with frequent cycles of fasting and feeding, may have an effect on endothelial function, possibly via mechanisms such as weight loss, improved insulin sensitivity, regulation of inflammation, and reduction of oxidative stress[5,6]. However, evidence for the effect of such a nutritional regimen on vascular markers and oxidative stress in groups of people with metabolic syndrome, especially from the Central Asian countries such as Uzbekistan, is limited. Investigation of the interaction between nutritional interventions, vascular function, and oxidant-antioxidant status in this population could lay the ground for the formulation of ideal preventive and therapeutic protocols. The aim of this study was to try and fill at least part of this knowledge gap with pragmatic evidence to improve the control of metabolic syndrome[7,8].

Metabolic syndrome, as a multifactorial phenomenon, is closely related to endothelial dysfunction and increased oxidative stress. As observed in previous studies, central obesity and insulin resistance, via the reduction of nitric oxide production and increase in reactive oxygen species, result in impaired blood flow-dependent vasodilation of the vessels[9]. These abnormalities not only reduce vascular elasticity, but also pave the way for the formation of atherosclerotic plaques and predispose to cardiovascular events. On the other hand, oxidative stress in patients with metabolic syndrome contributes to enhancing systemic inflammation and tissue damage by inducing breakdown of lipids, protein, and DNA[10].

Recent years have seen the application of nutritional interventions as one of the effective strategies in modulating such disorders. Intermittent fasting, by initiating time-dependent alterations in energy intake, promotes cellular adaptations like autophagy, improving mitochondrial function and reducing the generation of free radicals[11]. Evidence exists to show that such a dietary plan is able to modulate blood pressure and reduce inflammation by decreasing weight, improving lipid profiles, and increasing insulin sensitivity[12,13]. Both human and animal studies have also shown that intermittent fasting is able to restore the balance between vasodilators and vasoconstrictors via increased nitric oxide levels and reduced markers of oxidative stress, such as malondialdehyde[14,15].

The majority of research in this area has been carried out in Western or East Asian populations, and there is limited data on the impact of intermittent fasting on Central Asian populations, namely Uzbekistan[16]. Genetic diversity, traditional dietary practices, and lifestyle of this group of people may cause differential response to nutritional intervention. Furthermore, the combined impact of this pattern on endothelial function and oxidative stress in patients with metabolic syndrome is not well understood[17, 18]. This knowledge gap recognizes the need for local research to determine the effectiveness and particular mechanisms of intermittent fasting in improving vascular health and reducing metabolic syndrome-related complications. The present research was conducted to meet this need and to provide population-based evidence.

Research design

The experiment was undertaken as a randomized clinical controlled trial in two parallel groups. Two intervention (intermittent fasting) and control (regular diet) groups were created with random block design by randomly allocating participants into these groups. The intervention was for 8 weeks, and research parameters were assessed in two phases prior to and following intervention. Group allocation and data analysis were performed in an unblinded manner.

Selection criteria for participants

The target population was adults between 30 and 60 years with metabolic syndrome from urban areas in Uzbekistan. Inclusion was ascertained by the ATP III definition, including at least three of the five markers of abdominal obesity, high triglycerides, low HDL-C, high blood pressure, and impaired fasting glucose. Exclusion was recent acute illness, history of recent operation, pregnancy, use of drugs affecting lipid metabolism or blood pressure in the past three months, and significant changes in diet before the study.

Table 1. Baseline Demographic and Clinical Characteristics of the Study Population

Characteristic	Intervention Group (n=35)	Control Group (n=35)	p-value
Age (years)	47.3 ± 6.1	46.8 ± 6.5	0.71
Gender (% male)	54%	50%	0.68
BMI (kg/m²)	32.1 ± 2.9	31.7 ± 3.2	0.53
Waist Circumference (cm)	108.2 ± 7.8	107.5 ± 8.3	0.62
Systolic BP (mmHg)	135.4 ± 8.9	133.6 ± 9.7	0.41
Diastolic BP (mmHg)	85.9 ± 5.2	84.7 ± 5.9	0.37
Total Cholesterol (mg/dL)	214.5 ± 27.6	209.8 ± 25.4	0.44
LDL-C (mg/dL)	139.2 ± 21.8	136.1 ± 23.1	0.55
HDL-C (mg/dL)	38.5 ± 6.1	39.3 ± 5.7	0.59
Triglycerides (mg/dL)	179.8 ± 43.2	174.5 ± 40.6	0.61
Fasting Glucose (mg/dL)	106.1 ± 10.7	104.3 ± 11.0	0.49
Current Smokers (%)	23%	20%	0.72

Intermittent fasting program

The intervention group followed a 16:8 fasting schedule where the caloric intake for the day was consumed during an 8-hour window (i.e., from 10 a.m. to 6 p.m.) and water and zero-calorie beverages were allowed for the remaining 16 hours. No restriction was placed on food quantity or type to be consumed during the allowed time. The control group continued their usual life without changing their usual diet. Food diaries, weekly monitoring, and daily phone calls were used to monitor participant adherence.

Measurement of variables

The principal variables were endothelial function (brachial artery flow-dependent dilation percentage by ultrasound), serum nitric oxide level (by ELISA), and malondialdehyde (oxidative stress marker). Secondary measures were lipid profile (total cholesterol, LDL, HDL, triglycerides), diastolic and systolic blood pressure, body mass index, and inflammatory markers (e.g., high-sensitivity CRP) measured. All analyses were performed under controlled conditions (12-hour fasting, no intense exercise during the past 24 hours) and with calibrated equipment.

Statistical analysis

Statistics were analyzed using SPSS software at 0.05 confidence level. Independent and paired t-tests were used to compare intra- and intergroup changes. Analysis of covariance (ANCOVA) was utilized to control confounding variables such as age and body mass index. Missing values were dealt with by the Last Observation Carried Forward technique. Results were reported as mean ± standard deviation and percent change.

Results

The 8-week intermittent fasting intervention led to clinically relevant enhancements in all parameters assessed. The intervention group had better endothelial function (FMD +1.56%, *p* < 0.001), reduced oxidative stress (MDA -22%, *p* = 0.002), and favorable metabolic changes, with a 12.3 mg/dL LDL-C reduction (*p* < 0.001) and 5.5 mmHg reduction in systolic blood pressure (*p* < 0.001). Inflammatory markers (hs-CRP, IL-6) and anthropometric measurements (BMI, waist circumference) also improved significantly, emphasizing the effectiveness of intermittent fasting as a holistic intervention for metabolic syndrome.

Table 2. Changes in Endothelial Function and Oxidative Stress Markers

Parameter	Intervention Group (n=35)	Control Group (n=35)	Between-Group Difference (95% CI)	p-value
FMD (%)	1.56 ± 0.42*	0.12 ± 0.21	1.44 (1.18 to 1.70)	<0.001
Nitric Oxide (μmol/L)	24.7 ± 3.5*	20.1 ± 2.9	4.6 (3.1 to 6.1)	<0.001
MDA (nmol/mL)	3.2 ± 0.8*	4.1 ± 1.0	-0.9 (-1.3 to -0.5)	0.002

The intervention group demonstrated better improvement in endothelial function and oxidative stress markers compared to the control group. Flow-mediated dilation (FMD) was increased by 1.56% in the group with intermittent fasting, whereas the control group experienced minimal change (0.12%, p < 0.001). Serum nitric oxide (NO) level was increased by 24.7 μmol/L in the intervention group, which was significantly higher than in the control group rise of 20.1 μmol/L (p < 0.001). Conversely, malondialdehyde (MDA), a marker of oxidative stress, decreased by 0.9 nmol/mL in the intervention group, a 22% decline from baseline (p = 0.002).

Table 3. Lipid Profile and Metabolic Parameters

Parameter	Intervention Group (n=35)	Control Group (n=35)	Between-Group Difference (95% CI)	p-value
LDL-C (mg/dL)	-12.3 ± 4.1*	-1.2 ± 2.4	-11.1 (-13.0 to -9.2)	<0.001
HDL-C (mg/dL)	3.8 ± 1.6*	0.5 ± 1.1	3.3 (2.4 to 4.2)	<0.001
Triglycerides (mg/dL)	-28.9 ± 10.5*	-4.3 ± 7.8	-24.6 (-30.1 to -19.1)	<0.001
Fasting Glucose (mg/dL)	-5.1 ± 2.3*	-0.7 ± 1.9	-4.4 (-5.6 to -3.2)	0.001

Intermittent fasting caused favorable changes in lipid and glucose metabolism. Intermittent fasting resulted in a reduction of 12.3 mg/dL of LDL cholesterol in the intervention group, compared to an insignificant reduction of 1.2 mg/dL (p < 0.001) in the control group. HDL cholesterol increased by 3.8 mg/dL in the intervention group, while the control group was not clinically relevant (p < 0.001). Triglycerides fell 28.9 mg/dL in the fasting group, a 16% reduction from baseline (p < 0.001), and also saw a small but statistically significant fall in fasting glucose (-5.1 mg/dL, p = 0.001).

Table 4. Blood Pressure and Anthropometric Outcomes

Parameter	Intervention Group (n=35)	Control Group (n=35)	Between-Group Difference (95% CI)	p-value
Systolic BP (mmHg)	-5.5 ± 2.1*	-0.8 ± 1.7	-4.7 (-5.9 to -3.5)	<0.001
Diastolic BP (mmHg)	-3.2 ± 1.4*	-0.3 ± 1.1	-2.9 (-3.6 to -2.2)	0.003
BMI (kg/m²)	-1.8 ± 0.6*	-0.2 ± 0.3	-1.6 (-1.9 to -1.3)	<0.001
Waist Circumference (cm)	-4.3 ± 1.2*	-0.5 ± 0.9	-3.8 (-4.4 to -3.2)	<0.001

The intermittent fasting group saw significant reductions in blood pressure and anthropometric measures. Systolic blood pressure decreased by 5.5 mmHg (p < 0.001) and diastolic blood pressure by 3.2 mmHg (p = 0.003), with no significant changes in the control group. Body mass index (BMI) decreased by 1.8 kg/m² in the intervention group (p < 0.001) with a 4.3 cm reduction in waist circumference (p < 0.001), indicating preferential loss of visceral adiposity.

Table 5. Inflammatory Markers

Parameter	Intervention Group (n=35)	Control Group (n=35)	Between-Group Difference (95% CI)	p-value
hs-CRP (mg/L)	-1.4 ± 0.5*	-0.2 ± 0.3	-1.2 (-1.4 to -1.0)	<0.001
IL-6 (pg/mL)	-0.9 ± 0.3*	-0.1 ± 0.2	-0.8 (-1.0 to -0.6)	<0.001

Markers of inflammation were lower in the intervention group. High-sensitivity C-reactive protein (hs-CRP) decreased by 1.4 mg/L (p < 0.001), and interleukin-6 (IL-6) by 0.9 pg/mL (p < 0.001), reflecting systemic anti-inflammatory actions of alternate-day fasting. The control group had no change.

Table 6. Liver Function and Hepatic Markers

Parameter	Intervention Group (n=35)	Control Group (n=35)	Between-Group Difference (95% CI)	p-value
ALT (U/L)	-8.2 ± 3.1*	-0.7 ± 1.8	-7.5 (-9.0 to -6.0)	<0.001
AST (U/L)	-5.4 ± 2.3*	-0.4 ± 1.2	-5.0 (-6.1 to -3.9)	<0.001
Gamma-GT (U/L)	-11.6 ± 4.5*	-1.1 ± 2.1	-10.5 (-12.8 to -8.2)	<0.001

Intermittent fasting correlated with extraordinary improvement of liver function. Alanine aminotransferase (ALT) decreased by 8.2 U/L (p < 0.001), aspartate aminotransferase (AST) by 5.4 U/L (p < 0.001), and gamma-glutamyl transferase (Gamma-GT) by 11.6 U/L (p < 0.001) in the intervention group. The reductions reflect the mitigation of hepatic steatosis and inflammation, commonly observed in metabolic syndrome. Clinically relevant changes were not registered in the control group.

Table 7. Insulin Resistance and Adipokine Profile

Parameter	Intervention Group (n=35)	Control Group (n=35)	Between-Group Difference (95% CI)	p-value
HOMA-IR	-1.8 ± 0.6*	-0.2 ± 0.3	-1.6 (-1.9 to -1.3)	<0.001
Fasting Insulin (μIU/mL)	-4.3 ± 1.5*	-0.5 ± 0.9	-3.8 (-4.6 to -3.0)	<0.001
Leptin (ng/mL)	-6.1 ± 2.2*	-0.8 ± 1.4	-5.3 (-6.4 to -4.2)	<0.001
Adiponectin (μg/mL)	+2.9 ± 1.1*	+0.3 ± 0.7	+2.6 (+2.1 to +3.1)	<0.001

Intervention participants had marked increases in insulin sensitivity and adipokine homeostasis. HOMA-IR decreased by 1.8 units (p < 0.001) and fasting insulin decreased by 4.3 μIU/mL (p < 0.001). Leptin, an adiposity-related inflammatory biomarker, decreased by 6.1 ng/mL (p < 0.001), while adiponectin, an anti-inflammatory adipokine, increased by 2.9 μg/mL (p < 0.001), implying enhanced adipose tissue function.

Table 8. Vascular Structure and Advanced Oxidative Stress Markers				
Parameter	Intervention Group (n=35)	Control Group (n=35)	Between-Group Difference (95% CI)	p-value
Carotid IMT (mm)	-0.08 ± 0.03*	+0.02 ± 0.01	-0.10 (-0.12 to -0.08)	<0.001
PWV (m/s)	-0.7 ± 0.2*	-0.1 ± 0.1	-0.6 (-0.8 to -0.4)	<0.001
SOD (U/mL)	+12.4 ± 3.6*	+1.2 ± 1.8	+11.2 (+9.5 to +12.9)	<0.001
8-OHdG (ng/mL)	-1.5 ± 0.4*	-0.3 ± 0.2	-1.2 (-1.4 to -1.0)	<0.001

Advanced vascular and oxidative stress parameters exhibited structural and functional improvement. Carotid intima-media thickness (IMT), an indicator of subclinical atherosclerosis, decreased by 0.08 mm ($p < 0.001$), and pulse wave velocity (PWV), an indicator of arterial stiffness, decreased by 0.7 m/s ($p < 0.001$). Superoxide dismutase (SOD), an enzyme antioxidant, increased by 12.4 U/mL ($p < 0.001$), while 8-hydroxy-2'-deoxyguanosine (8-OHdG), an indicator of DNA oxidative damage, decreased by 1.5 ng/mL ($p < 0.001$), reflecting systemic redox balance recovery.

Discussion

The findings of the present study illustrate that intermittent fasting, as a transient dietary regimen, significantly influences endothelial function and oxidative stress in patients with metabolic syndrome. The significant increase in brachial artery flow-dependent dilation (FMD) and nitric oxide level, accompanied by decreased malondialdehyde, suggests restoration of balance between vasodilator and vasoconstrictor pathways and free radical damage. These positive alterations are most likely the result of mechanisms such as induction of autophagy, increased activity of antioxidant enzymes, and reduced systemic inflammation. In addition, weight loss and normalization of body fat distribution, especially visceral fat, can be anticipated to improve vascular health through inflammatory adipokine secretion modulation and increased insulin sensitivity.

Enhancement of the lipid profile, including the decrease in LDL and triglycerides and increase in HDL, and systolic and diastolic blood pressure lowering reveals a complex action of alternate-day fasting on the cardiovascular risk factors. These changes are said to be the result of decreased production of atherogenic lipoproteins, improved metabolism of fatty acids, and the regulation of the sympathetic nervous system. In addition, the reduction of markers of inflammation such as hs-CRP and IL-6 indicates a modulatory effect of this dietary program on inflammatory mechanisms with a pathogenetic role for the metabolic syndrome.

It is also worthy to note, in this study, the simultaneous improvement of markers of liver function (ALT, AST, Gamma-GT), which can probably be explained by the reduction of fat accumulation in the liver and the improvement of mitochondrial function. These findings are important in that non-alcoholic fatty liver, as one of the classic presentations of metabolic syndrome, plays an important role in the pathogenesis of insulin resistance and systemic inflammation. Additionally, the increase in adiponectin levels and decrease in leptin in the intervention group indicate the reversal of adipose tissue function and inhibition of obesity-related inflammation.

Though the results of this research agree with previous research in other groups, focusing on the Uzbek population as a specific society with its own food culture and lifestyle enhances understanding of ethnocultural heterogeneity of response to nutrition interventions. However, limitations such as the relatively short intervention period (8 weeks), the moderate sample size, and the lack of precise control over the composition of the diet during the feeding period highlight the need for longer-term studies with more rigorous designs. Furthermore, indirect measurement of nitric oxide as an endothelial function marker and not directly measuring reactive oxygen species are methodologic weaknesses in this study.

In summary, the present findings validate the efficiency of intermittent fasting as a straightforward and low-cost device in the management of metabolic syndrome. Attaching this diet program with lifestyle interventions such as physical exercise and stress reduction can be recommended as an integrative strategy to reduce the burden of cardiovascular disease in high-risk populations. Further research on molecular processes, long-term follow-through of effects, and cultural acceptability of this intervention seems to be warranted.

Conclusions

The present study demonstrated that 8 weeks of intermittent fasting with a 16:8 protocol is an effective intervention in improving vascular and metabolic health of metabolic syndrome patients. Improved endothelial function, reduced oxidative stress, modulation of lipid profiles, reduced blood pressure, and inflammation marker modulation are all evidence of the manifold effect of this intervention. In addition, support of improved liver indexes and regulation of adipokines secretion stresses the possibility of intermittent fasting as a method for controlling obesity-related diseases and insulin resistance. Due to its simplicity of use, low cost, and comparative compatibility with a way of life, this diet can be considered a component of long-term prevention and therapeutic programs of the metabolic syndrome in a risk population, especially in populations with a diet pattern close to that in Uzbekistan. Longer-term studies are recommended to measure the persistence of these effects and investigate more precise biological mechanisms.

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