Effect of mini low carbohydrate diet and eating speed in obese adults on fasting blood glucose

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ABSTRACT

Background: Obesity is an issue with public health that affects people worldwide and raises morbidity and mortality rates. In addition to dietary factors, non-nutritional factors like meal frequency and duration also contribute to the increased risk of obesity. This study aims to evaluate the duration of meals that will be compared between obese subjects who receive a mini low carbohydrate diet and those who do not.

Methods: Used a cross-sectional, quasi-experimental design in this study. Seven days of testing on 70 obese subjects who met the inclusion criteria for this study were conducted in November at the Palangka Raya city health center. Using a stopwatch to time the length of each meal in seconds and fasting blood glucose tests. Paired T-test and Independent Sample T-test are used in statistical analysis, while Wilcoxon and Mann-Whitney tests are used in the non-parametric analysis. Data were analyzed using SPSS version 20.0 for Windows.

Results: Blood glucose levels and eating speed did not correlate significantly in the mini low carbohydrate diet (M-LCD) group (p>0.05), but they correlated significantly in the control group (CG) (p<0.05). There was a significant difference in fasting blood glucose of the M-LCD and CG groups (p<0.05).

Conclusion: There was no correlation between meal speed and fasting glucose levels in the M-LCD group, but there was a significant difference between the M-LCD and CG groups’ fasting blood glucose levels.

Keywords: Fasting blood glucose, obesity, speed of eating, low carbohydrate diet


INTRODUCTION

Obesity is a global public health issue that raises morbidity and mortality rates. An increase in the prevalence of obesity will increase diabetes-related mortality. The prevalence of obesity among adults 18 and older has increased 1.5 times globally since 2000.1

In 2016, the global prevalence of obese adults aged 18 and older was 13.1%. Indonesia will see a continued rise in obesity, making it a health issue that must be considered. Obesity prevalence in adults over the age of 18 increased from 14.8% in 2013 to 21.8% in 2018.2,3

Food intake and obesity disease are closely related, and an imbalance between food intake and energy expenditure significantly impacts the condition. Exercise and food selection are crucial for maintaining an appropriate energy balance.4 Obesity and cardiometabolic diseases are highly susceptible to excessive food intake.5,6

In addition to dietary intake factors, non-nutritional factors like meal frequency and duration also contribute to the increased risk of obesity and cardiovascular disease.7,8 Waist circumference, obesity, and body mass index (BMI) can all be affected by dietary changes.9 As a result, obesity significantly increases the risk of developing diabetes and cardiovascular diseases.10,11

Eating fast is associated with an increased risk of obesity-related diseases.12 According to the findings, fasting more extended periods at lunch and dinner increased the risk of developing dyslipidemia in obese diseases.13 A higher risk of obesity is also linked to recent behavioral changes in lifestyle, mealtime, fast food, and meal duration.14 Eating fast increases the risk of developing diabetes-related eating patterns.15 Eating fast increases the risk of blood glucose elevation.16 Consequently, eating speed raises the possibility of type 2 diabetes.17 Eating fast consumption-related energy intake can lead to long-term body weight

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gain, insulin resistance, and metabolism problems. Therefore, controlling eating speed is crucial for preventing type 2 diabetes.

A low-carbohydrate diet (LCD) may increase energy expenditure for weight loss. According to some studies, LCD can help people lose weight and improve their glycemic control. LCD reduces blood sugar levels, enhances glucose tolerance, and boosts insulin sensitivity. Furthermore, LCDs can maintain or enhance average lipid profiles, lower the risk of cardiovascular disease, and more.

Based on those mentioned above, this study aims to compare the fasting blood glucose levels of obese subjects on the M-LCD and obese subjects who are not on a diet.

METHODS

This study used cross-sectional and quasi-experimental research designs in this study. Subjects were chosen based on the inclusion criteria, which included being over 18, having a BMI of at least 24.9 kg/m², and residing near a health center in Palangka Raya, Central Kalimantan. Purposive sampling was used to collect subjects. Based on the prevalence of obesity, which is higher in urban areas than rural areas and more prevalent in older age groups, the first stage acquired eight health centers. In the second stage, purposive sampling is done by dividing the subjects evenly in each health center. Then the subjects will be randomized between the control group (CG) and the mini-low carbohydrate diet (M-LCD) intervention using the Simple Random Sampling method.

The inclusion criteria for taking subjects are male and female adults aged >18 years, have a BMI ≥ 24.9 kg/m² (Obesity), do not have a critical illness (Critical Illness), and are willing to follow the research intervention. Exclusion criteria for the study were BMI < 24.9 kg/m²; having a critical illness such as coronary heart disease, heart failure, severe renal failure, hepatic cirrhosis, stroke, and cancer known from the interview before the study; obese adults who are pregnant; breastfeeding; drinking alcohol; anti-obesity drugs and drugs that can affect lipid profiles. Primary data taken in this study were meal duration and fasting blood glucose. This study used instruments of characteristics of obese adults and meal duration obtained through interviews.

For seven days, M-LCD was administered three times (in the morning, noon, and evening) with a caloric requirement of 40% carbohydrate, 30-35% fat, and 25%-30% protein. Calculate the subject’s nutritional needs using the Nutritional Adequacy Rate (AKG). This value shows the average need for certain nutrients that must be met daily with specific characteristics, including age, gender, level of physical activity, and physiological conditions. The nutritional needs of the subjects in this study used AKG and a calorie reduction of 500 kcal. Calorie reduction can lead to significant weight loss. The average nutritional requirement according to the AKG is 2,458.5 kcal after deducting 500 kcal to 1,958.2 kcal, 76.2-gm fat, 122.3-gm protein, and 195.8-gm carbohydrate.

A questionnaire was used as a guide to conduct the interviews. Eating speed used a stopwatch to time each meal, and a spectrophotometer was used to measure fasting blood glucose. Based on BMI calculations, the subject assessed nutritional status. According to Asia Pacific criteria, nutritional status is classified as follows based on BMI: underweight at 18.5 kg/m², optimal nutritional status at 18.5-22.9 kg/m², overweight at 23-24.9 kg/m², obesity grade I at 25-29.9 kg/m², and obesity grade II at 30 kg/m², and residing near a health center (Obesity), do not have a critical illness (Critical Illness), and are willing to follow the research intervention. Exclusion (Critical Illness), and are willing to follow the research intervention. Exclusion criteria for the study were BMI < 24.9 kg/m²; having a critical illness such as coronary heart disease, heart failure, severe renal failure, hepatic cirrhosis, stroke, and cancer known from the interview before the study; obese adults who are pregnant; breastfeeding; drinking alcohol; anti-obesity drugs and drugs that can affect lipid profiles. Primary data taken in this study were meal duration and fasting blood glucose. This study used instruments of characteristics of obese adults and meal duration obtained through interviews.

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The relationship between eating speed and fasting blood glucose was examined using Paired T-tests, and the difference between M-LCD and CG was examined using Independent T-tests. The Wilcoxon test or Mann-Whitney test analyzed data that data did not usually distribute. The p-value significance limit was set at <0.05. All data analysis was performed using SPSS version 20.0 for Windows.

RESULTS

Subject characteristics

This study involved up to 70 obese adults over 18, 35 receiving M-LCD treatment and 35 receiving no treatment in the CG group. Thirty-eight subjects (54.3%) were between 21 and 30 years, the age range for most research subjects. In this study, the subjects had a majority of undergraduate degrees (51.4%), followed by high school diplomas (44.3%), and the least number of postgraduate degrees (4.3%). Males comprised 64.3% of the subjects, compared to females (35.7%). When compared to eating slowly (>10 minutes), obese adults’ average eating speed was under 10 minutes (68.6%). According to the M-LCD group and CG group characteristics of eating duration, in the control group, eating fast lasted 20 subjects (57.2%), and eating slow lasted 15 subjects (42.8%). In contrast, in the intervention group, eating fast lasted 28 subjects (80%) and eating slow lasted seven subjects (20%) (Table 1).

According to the findings of statistical tests, there was no significant difference between the fasting blood glucose levels of the CG group before and after the intervention (p<0.05). At the same time, there was a significant difference between the fasting blood glucose levels of the M-LCD group before and after the intervention (p>0.05). Before the intervention, there was no significant difference in fasting blood glucose between the M-LCD and CG groups (p>0.05). Still, after the intervention, there was a significant difference between the M-LCD group and the control group (p<0.05) (Table 2).

There was no significant correlation between eating speed and fasting blood glucose in obese adults, according to the statistical test results in the M-LCD group (p>0.05). The M-LCD group had a very weak correlation between eating speed and fasting blood sugar (r=0.157). According to the CG group, fasting blood glucose levels and meal duration were significantly correlated (p=0.009). The longer the eating speed to finish a meal, the more it will lower fasting blood glucose levels. Still, the relationship between the two in the CG group is very weak (r= -0.330), with a negative relationship direction (Table 3).

DISCUSSION

Seventy obese adults were split into two groups for this study. The M-LCD group received a low-carbohydrate mini diet consisting of 40% carbohydrates, 30-
35% fat, and 25-30% protein, while the CG group received no intervention (26). The findings demonstrated a significant difference in fasting blood glucose levels between the M-LCD and CG groups before and after the intervention (p<0.05). Before the intervention, there was no difference in the fasting blood glucose levels between the M-LCD and CG groups (p>0.05), but there was one after the intervention (p<0.05). These findings show that the M-LCD diet can lower fasting blood sugar levels.29–31

Due to its ability to initiate glycogenolysis and gluconeogenesis, M-LCD can lower blood sugar levels. When the blood’s glucose level becomes insufficient, glycogenolysis kicks in to keep the blood’s glucose level stable by releasing the glucose stored in glycogen.32 Additionally, the gluconeogenesis process will produce glucose from non-carbohydrate sources using lactate, pyruvate, glycerol, and amino acids as precursors for glucose formation if the glycogenolysis process cannot meet the blood’s glucose requirements or if the blood’s glycogen stores are depleted.33–34

As a result, blood levels of glucose, low-density lipoprotein (LDL), and triglycerides will drop. M-LCD may be a helpful method for controlling the metabolism of diseases associated with metabolic syndrome. Additionally, M-LCD can lower body weight, BMI, and waist circumference.35

In addition to its beneficial effects, LCD can impair brain activity, resulting in lightheadedness and unconsciousness.33 According to some studies, this LCD’s ability to reduce body fat mass is only temporary.36–39 Further research has revealed that long-term LCD may raise morbidity at 12 months and above.40 However, some research indicates that LCD’s long-term effects may lower the risk of cardiovascular disease.41 Research from randomized control trials (RCTs) indicates LCD is safe for long-term treatment of type 2 diabetes mellitus.33 Further research is required to determine the safety of LCDs because some of these studies have not resolved the pros and cons of LCD use, particularly over the long term.

This study compared the M-LCD and CG groups to fasting blood glucose and eating speed. The findings demonstrated that while there was no relationship between eating speed and fasting blood glucose in the M-LCD group, there was a relationship between these two variables in the CG group. Therefore, a controlled diet and rapid eating can lower mortality and morbidity, particularly in obese adults. The eating speed in the CG group had a significant relationship with fasting blood glucose because the stomach takes about 20 minutes to signal satiety to the brain. When someone eats quickly, they tend not to feel full and tend to overeat.42 In line with other research, eating fast will increase energy intake,

Table 1. Subject characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>MLCD Group (n=35)</th>
<th>Control Group (n=35)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (Years), n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18-20</td>
<td>5 (14.2)</td>
<td>4 (11.4)</td>
</tr>
<tr>
<td>21-30</td>
<td>24 (68.6)</td>
<td>14 (40.0)</td>
</tr>
<tr>
<td>31-40</td>
<td>4 (11.4)</td>
<td>11 (31.4)</td>
</tr>
<tr>
<td>41-50</td>
<td>1 (2.9)</td>
<td>4 (11.4)</td>
</tr>
<tr>
<td>50-60</td>
<td>1 (2.9)</td>
<td>1 (2.9)</td>
</tr>
<tr>
<td>&gt;60</td>
<td>0 (0.0)</td>
<td>1 (2.9)</td>
</tr>
<tr>
<td>Education, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uneducation</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Primary School</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Junior High School</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Senior High School</td>
<td>18 (51.4)</td>
<td>13 (37.1)</td>
</tr>
<tr>
<td>Undergraduate</td>
<td>14 (40.0)</td>
<td>22 (62.9)</td>
</tr>
<tr>
<td>Postgraduate</td>
<td>3 (8.6)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Gender, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>22 (62.9)</td>
<td>23 (65.7)</td>
</tr>
<tr>
<td>Female</td>
<td>13 (37.1)</td>
<td>12 (34.3)</td>
</tr>
<tr>
<td>Eating Speed (minutes), n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fast (&lt;10)</td>
<td>28 (80.0)</td>
<td>20 (57.2)</td>
</tr>
<tr>
<td>Slow (≥10)</td>
<td>7 (20.0)</td>
<td>15 (42.8)</td>
</tr>
<tr>
<td>Fasting Glucose (mg/dl), n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 100</td>
<td>6 (17.1)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>100-125</td>
<td>16 (45.8)</td>
<td>4 (11.4)</td>
</tr>
<tr>
<td>≥ 126</td>
<td>13 (37.1)</td>
<td>31 (88.6)</td>
</tr>
</tbody>
</table>

MLCD: Mini Low Carbohydrate Diet

Table 2. Characteristics of Fasting Glucose Before and After Intervention

<table>
<thead>
<tr>
<th>Group</th>
<th>Fasting Glucose (mg/dl)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before Intervention</td>
<td>After Intervention</td>
</tr>
<tr>
<td>M-LCD</td>
<td>157.17 ± 62.32</td>
<td>126.77 ± 37.30</td>
</tr>
<tr>
<td>CG</td>
<td>175.37 ± 71.68</td>
<td>207.26 ± 69.98</td>
</tr>
<tr>
<td>p</td>
<td>0.350b</td>
<td>0.000r</td>
</tr>
</tbody>
</table>

M-LCD = Mini Low Carbohydrate Diet; CG = Control Group; *Wilcoxon; bMann-Whitney; +Statistically significant if p-value less than 0.05

Table 3. Relationship between eating speed and fasting blood glucose

<table>
<thead>
<tr>
<th>Group</th>
<th>Eating Speed</th>
<th>r</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>M-LCD (mg/dl)</td>
<td>Fast (&lt;10 minutes)</td>
<td>119.71±37.30</td>
<td>0.157</td>
</tr>
<tr>
<td>CG (mg/dl)</td>
<td>Slow (≥10 minutes)</td>
<td>231.55±80.18</td>
<td>-0.330</td>
</tr>
</tbody>
</table>

M-LCD = Mini Low Carbohydrate Diet; CG = Control Group; r = Coefficient Correlation; *Spearman Correlation test; +Statistically significant if p-value less than 0.05
leading to weight gain, insulin resistance, and worsening metabolism in the long run.\textsuperscript{15-45} Several studies have shown that eating fast increases the risk of type 2 diabetes.\textsuperscript{15,41-45} The duration of eating fast is also associated with the risk of impaired glucose tolerance.\textsuperscript{46} Also, eating fast leads to reduced mastication, which may increase the risk of hyperglycemia.\textsuperscript{47,48}

Decreased secretion of YY and glucagon-like peptide-1 (GLP-1) with rapid eating can lead to hyperglycemia.\textsuperscript{49} In contrast, a slow meal duration can increase the secretion of peptide YY and GLP-1.\textsuperscript{49,50}

Eating fast causes a delayed feeling of satiety, leading to overeating. A previous study reported that slow meal duration could reduce ghrelin secretion.\textsuperscript{51} These results suggest that eating fast can cause hormonal changes that can delay feelings of fullness, thus triggering overeating. In addition, the M-LCD diet group did not have a significant relationship with fasting blood glucose, as LCD can reduce hunger and improve mood.\textsuperscript{32,33}

This study’s limitation is that the subjects’ eating speed was subjectively self-reported, so researchers cannot exclude subject reporting bias. Second, the short duration of the intervention is for a week, so the causal relationship must still be proven with long-term intervention. Third, during the intervention, the researcher provided a food record form to the subject to record any food eaten outside of the intervention provided. Time constraints prevented researchers from fully controlling the subject’s daily food intake and more frequent home visits. Fourth, physical activity was measured using the Physical Activity Ratio (PAR), and it was found that the average physical activity category of the study subjects was sedentary or light activity.

**CONCLUSION**

Eating speed and fasting blood glucose levels did not correlate in the M-LCD group, while they did in the CG group. However, the M-LCD and CG groups’ fasting blood glucose levels differed significantly. This result requires additional research with long-term intervention to determine whether eating speed is advantageous for preventing metabolic diseases.

**CONFLICT OF INTEREST**

The author reports no conflicts of interest in this work.

**ETHICAL APPROVAL**

This study has been declared ethically sound by the Research Ethics Committee of the Faculty of Medicine, Sebelas Maret University, with the ethical feasibility number No: 109/UN27.06.11/KEP/EC/2022.

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**AUTHOR CONTRIBUTIONS**

All authors equally contribute to the study from the conceptual framework, data acquisition, and data analysis until reporting the study results through publication.

**REFERENCES**


