Effect of Short-Term Pritikin Diet Therapy on the Metabolic Syndrome

It is estimated that about one fourth of adults in the United States have the metabolic syndrome as defined by the National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) criteria. The high prevalence of the metabolic syndrome has considerable clinical implications, because the syndrome is an important risk factor for cardiovascular disease. Data from several studies have demonstrated that the metabolic syndrome is associated with a 3–4-fold increased risk of cardiovascular disease, cardiovascular mortality, and stroke.

Guidelines provided by the NCEP for treating the metabolic syndrome involve initial therapy with exercise and diet modification. The recommended dietary fat content is 25%–35% of total calories; greater reductions in dietary fat intake were not recommended because of the concern that a very-low-fat diet (ie, <20% of calories from fat) can decrease serum high-density lipoprotein cholesterol (HDL-C) levels and increase serum triglyceride concentrations.

Conclusions regarding the adverse effects of very-low-fat diets on serum lipids are based, however, on data from studies that compared relative fat intake as part of a eucaloric diet, which may not reflect changes in serum lipids that occur when a low-calorie diet is consumed.

The Pritikin Program (Aventura, FL) provides a unique opportunity to evaluate the clinical effects of an ad libitum, very-low-fat diet (10%–15% of total calories) and exercise program in patients who have the metabolic syndrome. This comprehensive program is offered in an inpatient setting, which enhances compliance with therapy because all meals are prepared and served on site, and daily exercise is supervised.

The purpose of the present study was to determine the effect of short-term therapy with a very-low-fat diet and daily exercise on metabolic coronary heart disease (CHD) risk factors in patients with the metabolic syndrome. We hypothesized that this therapy would improve most features of the metabolic syndrome (blood pressure, blood glucose, and serum triglyceride concentration), but decrease serum HDL-C concentration; however, the total cholesterol to HDL-C ratio would improve because of a decrease serum low-density lipoprotein cholesterol (LDL-C) concentration.

Methods

Subjects. Data were obtained by review of medical records of adult (21 years and older) clients who stayed at the Pritikin Longevity Center for 12–15 days between January 1, 2000, and December 31, 2003. The use of client charts for this study was approved by the Human Studies Committee of Washington University School of Medicine. All subjects had a comprehensive medical evaluation including a multistage symptom-limited stress test before starting the program.

Subjects were diagnosed as having the metabolic syndrome if they had ≥3 criteria as defined by the NCEP guidelines, based on history and physical examination, blood tests, and receiving medication used to treat a metabolic syndrome abnormality. A body mass index (BMI) >30 kg/m² was used as a metabolic syndrome criterion when waist circumference measurements were not available. Subjects were excluded if
They had severe uncontrolled hypertension (blood pressure ≥190/120 mm Hg), severe hypertriglyceridemia (serum triglycerides >600 mg/dL), untreated hypothyroidism, or were pregnant or lactating. Body weight; BMI; fasting blood glucose; blood pressure; and serum HDL-C, triglycerides, total cholesterol, and LDL-C concentrations were recorded at the beginning and end of 12–15 days of treatment. Serum glucose and lipid concentrations were measured at a commercial laboratory (Quest Diagnostics, Fort Lauderdale, FL). Total cholesterol, HDL-C, and triglyceride concentrations were measured with spectrophotometry (Olympus Spectrophotometer, Olympus American Inc, Melville, NY). Serum LDL-C concentration was calculated with the Friedewald formula.13 Medications used to treat clinical features of the metabolic syndrome were recorded.

**Table I. Effect of Short-Term Pritikin Therapy on Metabolic Syndrome Criteria (N=67)**

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Before Treatment, No.</th>
<th>After Treatment, No.</th>
<th>Change, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose ≥110 mg/dL</td>
<td>47</td>
<td>36*</td>
<td>−23%</td>
</tr>
<tr>
<td>Systolic BP ≥130 mm Hg</td>
<td>48</td>
<td>16*</td>
<td>−67%</td>
</tr>
<tr>
<td>Diastolic BP ≥85 mm Hg</td>
<td>22</td>
<td>5*</td>
<td>−77%</td>
</tr>
<tr>
<td>HDL-C ≤40 mg/dL (men) or ≤50 mg/dL (women)</td>
<td>50</td>
<td>51</td>
<td>2%</td>
</tr>
<tr>
<td>Triglycerides ≥150 mg/dL</td>
<td>63</td>
<td>29*</td>
<td>−54%</td>
</tr>
<tr>
<td>Metabolic syndrome</td>
<td>67</td>
<td>46*</td>
<td>−31%</td>
</tr>
<tr>
<td>No. of metabolic syndrome criteria/person, mean ± SD</td>
<td>4.3±0.7</td>
<td>3.3±1.2*</td>
<td>−23%</td>
</tr>
</tbody>
</table>

*P<.01 compared with before treatment. BP indicates blood pressure; HDL-C, high-density lipoprotein cholesterol.

Subjects also received instruction for a personalized exercise program (outdoor walking plus daily exercise classes) for a total of 45–60 minutes of aerobic exercise performed to achieve a heart rate of 70%–85% of maximal heart rate.

**Statistical Analyses.** The Student t test for paired samples was used to analyze the effect of treatment on metabolic parameters. The Wilcoxon rank sum test was used to analyze serum triglyceride concentrations and fasting blood glucose concentrations because these data were unevenly distributed.

**Results**

Of 300 client charts that were reviewed, 67 subjects (52 men and 15 women; mean age, 60±10 years) met the inclusion criteria for this study. All subjects were obese (BMI ≥30 kg/m²). Forty (60%) of the subjects had diabetes, determined by either a prescription for medication to treat hyperglycemia or a fasting blood glucose of ≥126 mg/dL. Fifty-one (76%) subjects were taking medications to treat ≥1 clinical feature of the metabolic syndrome, which included antihypertensive, hypoglycemic, and lipid-lowering agents. At the start of treatment, subjects were taking an average of 2.4±2.0 medications.

At the end of treatment, 21 (31%) subjects no longer met NCEP criteria for the metabolic syndrome (Table I). Moreover, with the exception of serum HDL-C concentration, all metabolic abnormalities of the metabolic syndrome improved with treatment. The number of medications used to treat the metabolic syndrome decreased in 16 (24%) subjects and increased in 6 (9%) subjects.

**CHD Risk Factors.** The effect of Pritikin diet therapy on metabolic CHD risk factors is shown in Table II. On average, subjects lost 3.18%±0.02% body weight. All risk factors, with the exception of HDL-C concentration, improved with treatment. Systolic and diastolic blood pressure and serum glucose and LDL-C concentrations decreased by 10%–15%; serum triglycerides decreased by 36%; serum HDL-C concentration decreased by 3%, and the total cholesterol to HDL-C ratio decreased by 17%. The improvement in CHD risk factors in response to the treatment was the same in subjects who had diabetes as in those without diabetes (data not shown). There was no association between percentage weight loss and changes in CHD risk factors.

**Discussion**

The purpose of the present study was to evaluate the clinical efficacy of short-term therapy with a very-low-fat diet and exercise on metabolic CHD risk factors in adult patients with the metabolic syndrome. Approximately 2 weeks of treatment improved most CHD risk factors (BMI, blood pressure, and serum triglyceride and LDL-C concentrations), and more than one third of subjects no longer met NCEP criteria for the metabolic syndrome. Nonetheless, the intervention caused a small decline in serum HDL-C concentration. These data demonstrate that brief treatment with a very-low-fat diet and exercise program has considerable metabolic benefits and simultaneously improves multiple CHD risk factors in patients with the metabolic syndrome.
Table II. Effect of Short-Term Pritikin Therapy on Coronary Heart Disease Risk Factors

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Before Treatment, Mean ± SD</th>
<th>After Treatment, Mean ± SD</th>
<th>Change, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index, kg/m²</td>
<td>37.0±6.61</td>
<td>35.8±6.2*</td>
<td>–3.4</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>139.8±18.6</td>
<td>119.0±15.1*</td>
<td>–14.9</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>80.5±10.4</td>
<td>73.3±8.7*</td>
<td>–8.9</td>
</tr>
<tr>
<td>Glucose, mg/dL</td>
<td>127.1±38.0</td>
<td>112.3±25.3*</td>
<td>–11.6</td>
</tr>
<tr>
<td>Triglyceride, mg/dL</td>
<td>245.2±92.2</td>
<td>157.1±57.1*</td>
<td>–35.9</td>
</tr>
<tr>
<td>HDL cholesterol, mg/dL</td>
<td>38.5±9.3</td>
<td>37.2±8.5†</td>
<td>–3.3</td>
</tr>
<tr>
<td>LDL cholesterol, mg/dL</td>
<td>118.9±44.4</td>
<td>102.6±37.4*</td>
<td>–10.9</td>
</tr>
<tr>
<td>Total cholesterol, mg/dL</td>
<td>200.8±49.2</td>
<td>171.2±41.2*</td>
<td>–14.7</td>
</tr>
<tr>
<td>Total cholesterol:HDL cholesterol ratio</td>
<td>5.3±1.1</td>
<td>4.4±1.1*</td>
<td>–17.0</td>
</tr>
</tbody>
</table>

*P<.001 compared with before treatment. †P<.05 compared with before treatment. BP indicates blood pressure; HDL, high-density lipoprotein; and LDL, low-density lipoprotein.

It is likely that several components of the intervention program contributed to the beneficial effects observed in CHD risk factors. First, the participants in this study lost a small amount of weight during the treatment period. Moderate diet-induced weight loss can decrease systolic and diastolic blood pressure, decrease serum glucose concentration, and decrease serum LDL-C and triglyceride concentrations. During early weight loss, however, serum HDL-C concentration often decreases, as observed in our subjects. Second, the therapeutic intervention included a supervised endurance exercise program, which can independently decrease serum triglyceride concentrations, decrease blood pressure, and decrease blood glucose concentration. Third, specific components of the diet may have had beneficial metabolic effects, independent of decreased energy intake and weight loss. Our subjects consumed a very low amount of fat, which can decrease serum LDL-C concentration, a low amount of sodium, which can decrease blood pressure, and a high amount of fiber, which can improve glucose homeostasis.

A weight maintenance diet that replaces dietary fat with an equal amount of calories from carbohydrate (low-fat, high-carbohydrate diet) can have adverse effects on serum lipids by increasing serum triglycerides and decreasing serum HDL-C concentrations. Our subjects experienced a decrease in serum triglyceride concentrations, despite consuming a very-low-fat, high-carbohydrate diet, which is consistent with data from previous studies that evaluated the effect of Pritikin diet therapy on serum lipids. It is likely that a low-fat diet that results in a negative energy balance and weight loss causes a decline in fasting serum triglyceride concentrations, whereas decreasing dietary fat in conjunction with a compensatory increase in calories from dietary carbohydrate causes an increase in fasting serum triglyceride concentrations. Although serum HDL-C concentrations decreased in our subjects, there was a marked decrease in total cholesterol concentrations. These changes led to a significant improvement in the ratio of total cholesterol to HDL-C, which is an established risk factor for CHD.

Consuming a very-low-fat, high-unrefined carbohydrate, and high-fiber diet in our subjects caused weight loss despite ad libitum food intake. The low energy density of this diet likely contributed to the subjects’ decrease in total energy intake and weight loss. The energy density of food is determined by the number of calories per gram of food. Therefore, at any given amount of calories, the volume of low energy density foods is greater than the volume of high energy density foods. Typically, foods that are low in fat content and high in water content, such as the Pritikin diet, are low in energy density. Data from short-term intervention studies suggest that decreasing energy density results in decreased total energy intake and weight loss.

This study has several limitations. First, this is a retrospective study and not a randomized clinical trial. Therefore, we cannot determine whether Pritikin diet therapy is better (or worse) than other low-fat, high-fiber diets. Second, the participants in this study may not be representative of other patients who have the metabolic syndrome. Our subjects were highly motivated to make healthy lifestyle changes and paid the cost of the program. Therefore, these results may not necessarily reflect the outcome that would be achieved in other subjects. Third, the duration of the intervention was approximately 2 weeks. Therefore, the results from this short-term intervention cannot determine long-term outcomes. For example, data from most lifestyle therapy interventions designed to achieve weight loss have shown that many obese persons who achieve short-term weight loss during therapy often regain much of their lost weight over time.

Conclusions

Short-term therapy with a very-low-fat, low-sodium, high-fiber diet and exercise, as implemented within the Pritikin Longevity Program, simultaneously improves most metabolic CHD risk factors and decreases the prevalence of the metabolic syndrome in patients who have the metabolic syndrome. Additional studies are needed to evaluate long-term effectiveness and compliance with this type of diet and activity program.
REFERENCES


