

Practical Solutions

Development of Symptomatic Cardiovascular Disease after Self-Reported Adherence to the Atkins Diet

TED D. BARNETT, MD; NEAL D. BARNARD, MD; TIM L. RADAK, DrPH, RD

The Atkins Diet, developed by the late Robert Atkins, MD, was first published in book form in 1972 (1). It limits dietary carbohydrate to 20 g daily in the early or “induction” phase, with liberalized carbohydrate restriction in later phases (1,2). Analysis of a 1-day menu in the 1999 edition of *Dr Atkins’ New Diet Revolution* (2) revealed that it contains 110 g total fat (45% of energy), 38 g saturated fat (16% of energy), and 834 mg cholesterol.

Low-carbohydrate diets are popular among patients seeking to lose weight. In published studies, cholesterol levels have increased in approximately one third of individuals using low-carbohydrate diets, prompting medical organizations to question possible adverse health effects (3,4). Several studies have demonstrated significant elevations of cholesterol levels among many low-carbohydrate dieters (5-9) and two cardiac deaths have been reported, one due to coronary artery disease and the other apparently due to arrhythmia (7,10).

CASE REPORT

An independent Institutional Review Board approved the writing of this case report. In November 2000, the patient was a 51-year-old, healthy, physically active nonsmoker who avoided alcohol, had normal sexual function, and had no evidence of heart disease, hypertension, or diabetes. An older sister had experienced a myocardial infarction at age 62. The patient’s past medical history was unremarkable. His total cholesterol was 146 mg/dL (3.78 mmol/L), low-density lipoprotein (LDL) cholesterol was 85 mg/dL (2.20 mmol/L), high-density lipoprotein cholesterol was 53 mg/dL (1.37 mmol/L), and triglycerides were 42 mg/dL (0.47 mmol/L) (Table).

T. D. Barnett is senior radiologist, Department of Diagnostic Imaging, Unity Hospital, Rochester, NY. N. D. Barnard is adjunct associate professor of Medicine, George Washington University School of Medicine and Health Sciences, and president, Washington Center for Clinical Research, Washington, DC. T. L. Radak is assistant professor & graduate dietetic internship director, Department of Family and Consumer Science, Appalachian State University, Boone, NC.

Address correspondence to: Ted D. Barnett, MD, 117 Lyons Road, Rush, NY 14543. E-mail: drveggie@aol.com

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In December 2000, he underwent computed tomographic colonography because routine colonoscopy did not visualize the entire colon. At that time the patient accepted an offer to also undergo computed tomographic coronary artery calcium scoring. The scan yielded a calcium score of zero, indicating that there was no measurable plaque in his coronary arteries.

Because his weight had gradually increased from his usual 63.6 kg (body mass index [BMI]=21.3) to 67.1 kg (BMI=22.6), he decided to begin following the Atkins diet (2). In May 2001, without professional advice or supervision and following the book’s recommendations closely (but without keeping a food diary), he began the “induction phase,” restricting carbohydrate intake to less than 20 g per day for the first 6 months. After that, having reached his desired weight, he consumed 50 to 100 g of carbohydrate per day during the “maintenance phase” of the diet.

In June 2001, 1 month into the diet, his weight had decreased to 64.9 kg (BMI=21.8) and laboratory tests done as part of an annual check-up demonstrated a sharp increase in total and LDL cholesterol (Table). Despite these marked lipid changes, he remained on the diet, believing it to be safe.

In early 2003, he began to take sildenafil (Viagra, Pfizer, New York, NY) for erectile dysfunction. On October 22, 2003, he presented to the emergency department with exertional chest pain. His blood pressure was 116/84 mm Hg with a regular heart rate of 64 and weight of 63.6 kg (BMI=21.3). His cholesterol levels had decreased modestly whereas his triglycerides had increased markedly from the values obtained 1 month after initiating the diet (Table). The electrocardiogram was normal. A radionuclide sestamibi scan of the heart demonstrated moderate anteroapical ischemia without evidence of infarction. An echocardiogram at rest demonstrated normal cardiac dimensions with mild anteroapical hypokinesia.

On October 27, 2003, cardiac catheterization demonstrated a critical stenosis proximally in the first diagonal branch of the left anterior descending coronary artery, which was treated with a drug-eluting stent. There were also mild stenoses of the left main and right coronary arteries, and mild anterior hypokinesia with an ejection fraction of 50% (Figure). He was discharged on October 28, 2003, having discontinued his low-carbohydrate diet and started a low-fat diet with increased consumption of whole grains, beans, vegetables, and nuts; inclusion of vegetarian entrees; modest consumption of lean meats; and minimal consumption of refined carbohydrates.

He was started on a drug regimen of atenolol (Tenormin,

Table. Laboratory values and clinical parameters before, during, and after the use of a low-carbohydrate Atkins diet					
	November 2000: 6 months before diet started	May 2001: Low-carbohydrate diet started	June 2001: 1 month on diet	October 2003: 29 months on diet	December 2003: 2 months after coronary stent placed and diet stopped
Erectile dysfunction	No	No	No	Yes	No
Chest pain	No	No	No	Yes	No
Total cholesterol (mg/dL) ^a	146		230	209	146
LDL (mg/dL) ^b	85		154	127	81
HDL (mg/dL) ^c	53		65	53	52
Triglycerides (mg/dL) ^d	42		56	144	65
Weight (kg)	63.6	67.1	64.9	63.6	61.7
Body mass index ^e	21.3	22.6	21.8	21.3	20.7

^aTo convert mg/dL HDL, LDL, and total cholesterol to mmol/L, multiply mg/dL by 0.026. To convert mmol/L HDL, LDL, and total cholesterol to mg/dL, multiply mmol/L by 38.7. Cholesterol of 193 mg/dL=5.00 mmol/L.
^bLDL=low-density lipoprotein.
^cHDL=high-density lipoprotein.
^dTo convert mg/dL triglycerides to mmol/L, multiply mg/dL by 0.0113. To convert mmol/L triglycerides to mg/dL, multiply mmol/L by 88.6. Triglycerides of 159 mg/dL=1.80 mmol/L.
^eCalculated as kg/m².

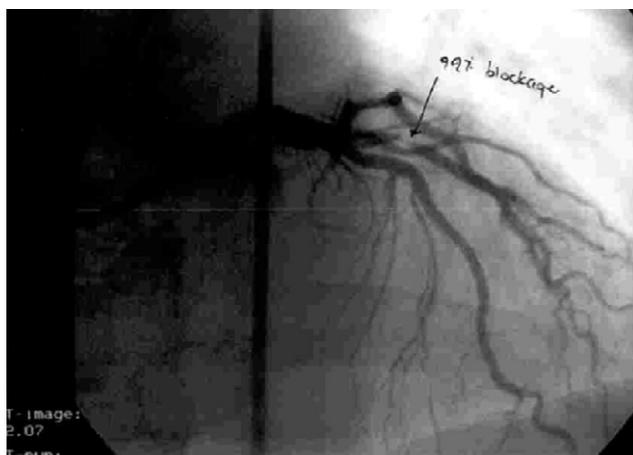


Figure. Angiogram done 29 months after initiation of a low-carbohydrate Atkins diet, revealing a 99% stenosis in the proximal portion of the first diagonal branch of the left anterior descending coronary artery.

AstraZeneca, Wilmington, DE), 25 mg daily; omeprazole (Prilosec, AstraZeneca), 20 mg daily; clopidogrel bisulfate (Plavix, Bristol-Meyers Squibb, New York, NY, and Sanofi-Aventis, Bridgewater, NJ), 75 mg daily; simvastatin (Zocor, Merck, Whitehouse Station, NJ), 40 mg daily; and aspirin, 81 mg daily. In the ensuing week, he experienced muscle cramping and simvastatin was discontinued. Aside from this 1-week period, there was no use of cholesterol-lowering medication.

Two months after discharge, the patient weighed 61.7 kg (BMI=20.7). Total cholesterol was 146 mg/dL (3.78 mmol/L), LDL cholesterol was 81 mg/dL (2.10 mmol/L), high-density lipoprotein cholesterol was 52 mg/dL (1.35 mmol/L), and triglycerides were 65 mg/dL (0.73 mmol/L). He was advised to continue all of his medications except atenolol. He did not have chest pain or erectile dysfunction and had discontinued sildenafil.

DISCUSSION

LDL cholesterol typically decreases as body weight decreases, independent of weight-loss method (11-13). Low-carbohydrate diets have proven the exception to this general rule.

Most studies of low-carbohydrate diets have not reported the proportion of individuals whose blood lipids worsened. Among those that have, approximately one third or more of individuals on low-carbohydrate diets have experienced an increase in LDL cholesterol. In a 2002 study of overweight or obese men and women placed on a low-carbohydrate diet for 6 months, LDL cholesterol increased in 12 of 41 subjects by a mean of 18 mg/dL±15 mg/dL (range, 4 to 53 mg/dL) (0.47 mmol/L±0.39 mmol/L; range, 0.10 to 1.37 mmol/L) (5). In a 2004 study, 13 of 44 low-carbohydrate dieters experienced a more than 10% increase in LDL cholesterol during a 24-week intervention (6). In a 2002 study of 12 normal-weight men following a low-carbohydrate diet for 6 weeks, seven had increases in total cholesterol ranging from 1% to 60% and increases in LDL ranging from 2% to 70% (8). These studies indicate that roughly one in three individuals on low-carbohydrate diets experience a significant increase in total or LDL cholesterol.

The very high cholesterol and saturated fat contents of typical low-carbohydrate diets are sufficient to account for their sometimes adverse effects on blood lipids. However, other aspects of these diets may lead to adverse cardiovascular effects. Methylglyoxal is a glycation product with numerous cytotoxic effects and is recognized for its ability to cause blood vessel and tissue damage. Precursors of methylglyoxal are formed during ketosis. Significant increases in methylglyoxal were reported in otherwise healthy subjects on a low-carbohydrate diet compared with a control group (14). In addition, cardiac arrhythmias have also been reported in low-carbohydrate dieters (9,10).

Other reports describe serious adverse cardiac effects, including some deaths, associated with the use of low-

carbohydrate, high-saturated fat, high-protein diets (7,10,15). Two large cohort studies recently reported significant increases in both total and cardiovascular mortality related to low-carbohydrate, high-protein intakes (16,17).

Erectile dysfunction is strongly associated with cardiovascular disease (18,19), and as with this patient, can precede the development of symptomatic coronary artery disease. Although the resolution of chest pain can be attributable to the placement of the coronary artery stent, the resolution of his erectile dysfunction supports the possibility of a diet-related effect.

This case report should be interpreted with appropriate caution. The diet was implemented without the use of a diet record. Also, cardiovascular disease risk is influenced by factors other than diet. The presence of heart disease in a sibling suggests the possibility of a genetic predisposition to cardiovascular disease. The computed tomographic coronary artery calcium scoring test, done before diet initiation, would not have detected the presence of noncalcified plaque. Nonetheless, the deterioration of plasma lipid concentrations, clinical status, and radiographic findings after starting the diet, as well as the resolution of these problems after stopping the diet, are noteworthy.

CONCLUSION

This case of a previously healthy man who experienced deteriorating plasma lipids, erectile dysfunction, and coronary atherosclerosis while on a low-carbohydrate Atkins diet, and whose symptoms and abnormal lipid profile resolved after cessation of the diet, adds to the body of evidence suggesting that some individuals experience increased cardiovascular risk related to such diets.

STATEMENT OF POTENTIAL CONFLICT OF INTEREST:

No potential conflict of interest was reported by the authors.

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