

# Lipid Levels in Anorexia Nervosa

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**Abstract: Objective:** *Anorexia nervosa is a chronic disorder characterized by the patient's refusal to maintain body weight and a myriad of medical complications. Two frequently cited and poorly understood complications are hypercholesterolemia and hypercarotenemia. We therefore measured fasting cholesterol and beta-carotene levels in a cohort of moderately severe anorectics.* **Method:** *All subjects were female and met DSM-IV criteria for anorexia nervosa. Lipid profiles and beta-carotene levels were determined in 23 female anorexics.* **Results:** *The mean total cholesterol for this group of anorectics was 179.3 mg/dl. Low-density lipoprotein values (LDL) ranged from 47 to 173, with a mean of 104.1 mg/dl. The mean high-density lipoprotein value (HDL) was 51.5 mg/dl. Beta-carotene levels were all normal.* **Discussion:** *We conclude that total and LDL cholesterol in anorexia are well within the range of normal, and HDL levels are favorably high. Further, hypercarotenemia was not associated with anorexia nervosa.* © 1998 by John Wiley & Sons, Inc. *Int J Eat Disord* 24: 217–221, 1998.

**Key words:** beta-carotene; cholesterol; hypercholesterolemia and hypercarotenemia

## INTRODUCTION

The medical complications of anorexia nervosa are myriad. They range from life-threatening cardiovascular complications to endocrine axis disturbances including amenorrhea and premature advanced osteoporosis (Mehler, 1996). Two frequently cited and poorly understood complications of anorexia nervosa are hypercholesterolemia and hypercarotenemia (Comerci, 1990). Most of the literature in this regard is somewhat dated (Nestel, 1974; Mordiasini, Klose, & Greten, 1978; Mira, Stewart, Vizzaard, & Abraham, 1987). The cholesterol elevation is reported to be in the low-density lipoprotein (LDL) fraction with normal high-density (HDL) and very-low-density lipoprotein (VLDL) levels. Recently, data have been published which suggest that the hyperlipidemia is strictly an acute phase finding. Results tended to be quite normal in the chronic period of anorexia (Sanchez-Muniz & Marcus, 1991). We, too, have been underwhelmed by the lipid abnormalities of anorexia, especially with regard to the low-density fraction. We, therefore,

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measured fasting cholesterol and beta-carotene levels in a cohort of moderately severe anorectics.

## METHODS

The 23 female patients whose ages ranged from 16 to 44 years old, with a mean age of 25.86 ( $\pm 2.06$  years), all met criteria for anorexia nervosa as outlined in the 4th ed. of the *Diagnostic and statistical manual of mental disorders* (DSM-IV; American Psychiatric Association, 1994). Body mass index (BMI) ranged from a minimum of 11.31 to 19.50, with a mean of 16.35 (Table 1). Seventeen of the subjects were amenorrheic. The remainder were taking oral contraceptives. Blood samples were drawn after 12 hr of fasting. Total cholesterol was determined by the Paramax cholesterol reagent formulation based on a modification of the method of Allain, Poon, Chan, Richmond, & Fu, 1974. HDL cholesterol was measured by pretreating serum with phosphotungstic acid and then running the cholesterol. LDL cholesterol was calculated from the measured cholesterol and HDL results by the formula:  $LDL = (\text{total cholesterol} - \text{HDL}) - VLDL$  (Friedwald, Levy, & Fredrickson, 1972). Triglyceride determinations are performed directly on serum using the Paramax triglyceride reagent formulations based on a modification of the procedures of Stavropoulis and Crouch (1974). Beta-carotene levels were determined by Coming Clinical Laboratories using serum and a high-performance liquid chromatography method.

Pearson's correlation coefficients were used to look at laboratory values for the total

Table 1. Characteristics of anorexia nervosa patients

OBS	ID	Age (yr)	Ht (in.)	Wt (lb)	AMEN	BMI	TC (mg/dl)	LDL (mg/dl)	HDL (mg/dl)	Carotene (mg/dl)	TRIG (mg/dl)
1	JD	27	66	116	Y	18.7395	158	93	52	18.0	65
2	EH	19	67	92	Y	14.4220	167	96	47	31.0	120
3	SM	16	65	93	Y	15.4898	112	47	48	23.0	85
4	RF	37	66	70	N	11.3083	140	90	29	19.0	105
5	CH	36	66	114	N	18.4164	165	90	48	21.0	135
6	CK	20	66	119	Y	19.2242	169	87	59	39.0	115
7	SW	44	63	85	N	15.0705	225	136	80	27.0	49
8	WH	19	62	77	Y	14.0960	218	132	44	7.0	220
9	CV	19	70	120	Y	17.2335	153	94	38	72.0	105
10	MS	38	68	121	Y	18.4143	194	98	60	82.0	182
11	SF	21	71	132	N	18.4266	168	97	51	24.0	100
12	MM	19	63	110	N	19.5029	179	102	42	28.0	134
13	RJ	19	68	90	Y	13.6966	264	160	32	561.0	349
14	AB	19	66	112	Y	18.0933	194	103	76	49.0	77
15	HA	18	64	88	Y	15.1186	246	173	43	75.0	148
16	MC	18	65	90	Y	14.9901	187	102	56	36.0	144
17	TS	22	68	115	Y	17.5012	140	80	41	31.0	100
18	CS	18	67	99	Y	15.5194	181	112	55	25.0	68
19	MO	19	64	90	Y	15.4622	245	146	72	66.0	132
20	LL	44	61	88	Y	16.6422	159	93	37	12.2	141
21	PS	37	67	103	N	16.1464	133	70	53	21.0	48
22	JM	43	63	90	Y	15.9569	188	107	66	17.0	78
23	HH	23	69	113	Y	16.7020	140	88	55	27.0	88

Note: OBS = observation; AMEN = amenorrhea; BMI = body mass index; TC = total cholesterol; LDL = low-density lipoprotein; HDL = high-density lipoprotein; TRIG = triglyceride.

group and for the subgroup of amenorrheics and those with normal periods. The statistical program SAS was used to calculate all univariate analysis. A two-sample *t* test was used to assess differences in mean values between the two groups.

## RESULTS

The mean total cholesterol for this group of 23 moderately severe anorectics was 179.3 mg/dl with a range of 112–264 mg/dl. LDL values ranged from 47 to 173, with a mean of 104.1 mg/dl. The mean HDL value for this group was 51.5 mg/dl, with a range of 32–80. Beta-carotene levels averaged 57.0 mg/dl (normal values 8–120). All patients had normal serum albumin levels (see Table 1).

Pearson's correlation coefficient revealed that there was a trend towards an inverse relationship between total and LDL cholesterol and body weight which did not reach statistical significance (Table 2). However, higher cholesterol values were associated with higher beta-carotene levels (Table 2). Subgroup analysis demonstrated an identical pattern for those women who were amenorrheic (see Table 3).

## DISCUSSION

Anorexia nervosa affects multiple different systems of the body. One laboratory abnormality frequently cited is hypercholesterolemia which might have clinical importance in light of anorexia's reported mortality rate of 4–30% (Isner, Roberts, Heymsfield, & Yaeger, 1985). Our results indicate that cholesterol elevations are probably not a plausible reason to explain this mortality rate. The mean total and LDL cholesterol values were well within the range of normal, as defined by the recently published National Cholesterol Education Panel (NCEP II) guidelines (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 1993). These guidelines emphasize coronary heart disease risk factors as guides to the type and intensity of cholesterol-lowering therapy. In this regard, the panel advocates a more conservative approach to hyperlipidemia management for young females, unless LDL cholesterol levels are very high (>220 mg/dl) or multiple other risk factors are also present.

Further, the HDL cholesterol is in a range which is considered protective against coronary artery disease and is a large reason why the total cholesterol seem high. Although one may opine that given their weight, these cholesterol levels are still higher than one would expect, the fact is that anorexia is different than other forms of malnutrition

Table 2. Relationship of cholesterol, weight, and beta-carotene

	Pearson Correlation Coefficients		
	Weight	Body Mass Index	Carotene
Total cholesterol	-0.31	-0.23	0.53
	0.13	0.28	0.01
Low-density lipoprotein	-0.38	-0.33	0.48
	0.07	0.12	0.01
High-density lipoprotein	0.13	0.25	-0.27
	0.53	0.23	0.19

Table 3. Relationship of cholesterol and weight in amenorrheic women

	Weight	Body Mass Index	Carotene
Total cholesterol	-0.53	-0.49	0.61
	0.05	0.08	0.02
Low-density lipoprotein	-0.50	-0.50	0.52
	0.07	0.08	0.05

because the serum albumin is invariably normal (Williams, 1979). Essential amino acids are not low in anorexia nervosa, in contradistinction to kwashiorkor or the malnutrition of alcoholism.

In addition, although the mode of death in severe anorexia nervosa is usually through a cardiac cause, there is currently no evidence that it is atherosclerotic in origin. There is, however, evidence from necropsy and clinical findings that sudden death in anorexia may result from ventricular tachyarrhythmias related to Q-T interval prolongation (Isner et al., 1985). Notwithstanding this clinical correlation, our data do demonstrate a trend towards an inverse relationship between cholesterol and body weight and a direct relationship between cholesterol and carotene levels. The true implication of these findings is not known despite the tantalizing possibilities which are worthy of further evaluation.

Interestingly, the literature is also replete with statements about a connection between anorexia nervosa and hypercarotenemia, given the yellowish hue found in the skin of patients with anorexia. Some studies suggest that dietary intake plays a major role (Bhanji & Mattingly, 1981), while others contend that the carotenemia of anorexia is a metabolic aberration which is not diet driven (Erdman & Celentano, 1993). Rather, they suggest that it may involve a thyroid hormone interaction. Yet, the vast majority of our patients had normal serum carotene levels. The basis of this discrepancy is not clear at this time.

In summary, we did not find abnormally high LDL cholesterol values in our series of patients with anorexia nervosa. The important clinical message should be that the majority of these patients should not be considered for an intervention to lower their lipid levels. Rather, use of estrogen replacement therapy should be encouraged for this population, who is frequently amenorrheic, in order to militate against any cardiac risk which is yet to be defined and especially against the known risk for premature osteoporosis which has clearly been defined (Andersen, Woodward, & LaFrance, 1995). Perhaps there is an important clue in the aforementioned inverse relationship between body weight and cholesterol value. Currently, additional studies are needed to better define any meaningful relationships which might exist.

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