

## Dyslipidemia Is a Well-Known Cause of Atherothrombotic Disease (ATD)

**William E. Feeman**

640 South Wintergarden, Bowling Green, Ohio 43402

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**\*Corresponding author:** William E. Feeman, 640 South Wintergarden, Bowling Green, Ohio 43402.

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### Case Report

**Introduction:** Dyslipidemia is a well-known cause of atherothrombotic disease (ATD). The author presents an unusual case of a woman with severe dyslipidemia who was unable to tolerate any dyslipidemic medication combination for any length of time over the last 25 years and yet has a normal coronary artery CAT scan done for chest pain, which is presumed to be micro vascular angina.

### Materials and Methods: Chart Review.

Results: A sampling of the patient's lipid testing is supplied in Table I.

**Table I**

Date	CT	HDL-c	LDL-c	TG	CRF
6 April 1996	288	32	211	225	0.85
20 April 1996	253	25	163	324	0.85
27 Oct 2004	319	29	239	256	0.88
19 April 2007	263	27	169	337	0.84
9 June 2008	297	27	--	691	--
18 Aug 2011	333	23	214	478	0.89
16 Nov 2016	364	26	--	624	--
13 Sept 2019	331	24	--	603	--
5 Oct 2021	362	24	275	316	0.91

CT Means Total Cholesterol

HDL-c Means High Density Lipoprotein Cholesterol

LDL-c Means Low Density Lipoprotein Cholesterol

TG Means Triglycerides

CRF Means Cholesterol Retention Fraction

Only the abnormal values are included since any ameliorating effects of any dyslipidemic therapy were only transitory due to the patient's inability to tolerate any therapeutic regimen for long. The patient tried all of the statins, ezetimibe, resins, niacin, fibrates, PCSK-9 inhibitors, lomitapide, mipromersin, and eicosapent ethyl—in various combinations, but all to no avail. Partial ileal bypass and apheresis were offered but declined by her insurance company. The patient smoked cigarettes for a short period of time as a young adult but quit smoking and has not resumed the habit. She is not diabetic, nor is she hypertensive. She does have hypothyroidism, which is under excellent control on medication. The CRF is an abbreviation for the Cholesterol Retention Fraction, which is the author's choice for determination of dyslipidemia. In the Table, CT stands for total cholesterol, LDL-c stands for low-density lipoprotein cholesterol, HDL-c stands for high-density lipoprotein cholesterol, TG stands for triglycerides, and the CRF is defined as  $(LDL-c \text{ minus } HDL-c) / LDL-c$ .

**Discussion:**

This lady has had severe dyslipidemia for at least 25 years, except for brief periods when she was able to tolerate the various therapeutic regimens. Of interest is that she has an on-going allergic diathesis, with allergies to multiple medications, severe asthma, and even eosinophilic gastroenteritis and esophagitis. She takes multiple medications for her allergies, including prednisone. She has also had a recent diagnosis of scleroderma and is being treated with mycophenol. She has also developed a seizure disorder and is being treated with Depo-Provera.

Because of chest pain, relieved by nitroglycerine, the patient, in 2017, underwent a treadmill test which was totally normal and then had a coronary artery CAT scan which showed “no significant coronary artery stenosis” and was read out as no evidence of coronary artery stenosis. The author therefore considered the patient to have microvascular angina.

The patient’s father had severe dyslipidemia, an acute coronary/cerebral event, and died. Her mother has severe dyslipidemia, but is still alive, though she has a history of angina and asymptomatic carotid stenosis. One of her daughters has dyslipidemia and the other has borderline dyslipidemia. This indicates that the patient’s dyslipidemia is inherited and that she has had dyslipidemia essentially since birth. She is now 53 years old.

The author reports this patient’s case, not to dispute the basis of cholesterol in contributing to ATD, but rather to try to understand why she has no significant coronary artery stenosis. Could the allergic diathesis with hyper-eosinophilia be the cause? The coronary artery CAT scan does not reveal any coronary abnormalities, so it would appear that there is no intimal hyperplasia. Jan Boren, MD, has advanced the hypothesis that without intimal hyperplasia there can be no plaque formation, at least in mice, at the 2021 virtual scientific symposium of the European Atherosclerosis Society, on 31 May 2021. Is there something in her medical history that blocks intimal hyperplasia? Does she have a gene pool that blocks intimal hyperplasia?

**Conclusions**

The author reports this unusual case in an attempt to understand why this patient, with a strong family history of dyslipidemia and ATD, does not herself have significant coronary plaque. He would be interested in hearing any comments.

**Conflicts of Interest:**

No conflicts of interest

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