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Case Report

Type II Hyperlipoproteinemia in a Woman with Initially Normal Low-Density Lipoprotein Cholesterol

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Abstract

Introduction: This case reports the lipid history of a lady with Type IIA Hyperlipoproteinemia (T-IIA HLP), who lipids were normal on initial testing in childhood but whose lipids over the ensuing years deteriorated, reaching very high levels of low-density lipoproteinemia cholesterol (LDL-c).

Methods: Chart review.

Results: The patient was found on incidental testing to have a normal level of LDL-c at age 12 years. A subsequent lipid panel was performed at age 15 years and LDL-c was found to have risen dramatically. Her lipid history is given in the table.

Conclusion: T-IIA HLP may not be detectable initially in childhood. Serial testing however can detect T-IIA HLP as the patient grows older. Serial testing is therefore recommended.

Keywords: *Type IIA Hyperlipoproteinemia (T-IIA HLP); Low-Density Lipoproteinemia Cholesterol (LDL-c); Familial Hypercholesterolemia (FH); High-Density Lipoprotein Cholesterol (HDL-c)*

Introduction

This case report is stimulated by the recent report by Orringer and Grant of a young lady (aged 23 years of age) who had genetic markers for Type II Hyperlipoproteinemia (T-II HLP) but a normal low-density lipoprotein cholesterol (LDL-c) [1]. The genetic testing was done because of a strong family history of Familial Hypercholesterolemia (FH). (The author prefers the term T-II HLP to FH because the former term limits the disorder to isolated elevations of LDL-c, whereas the latter term could have high total serum cholesterols (CT) related to high LDL-c, sitosterol, very high triglycerides, and/or markedly elevated high-density lipoprotein cholesterol (HDL-c) levels. In this report the author describes the case of an adolescent (aged 12 years of age at time of initial lipid determination) who had a normal LDL-c, but who over the next two decades developed a markedly high LDL-c level on serial testing.

Materials and Methods

This report is based on chart review.

Case Report and Results

The patient was found to have slightly elevated CT (211 mg/dl or 5.5 mmoles/L) on incidental testing at age 12 years. She subsequently underwent full lipid testing with the results as listed in the table. She had a further lipid evaluation a few years later and was found to have an elevated LDL-c. At this point, she underwent serial lipid testing, with the results listed in the table. Because she was in her childbearing phase of life, she was not offered pharmacological therapy. By May of 2001, she had decided to not have any more children and statin therapy was initiated, with the results given in the table. Lipid studies on her family members (mother and siblings) are not available to the author, but her father does not have T-II HLP. Many years after the patient's initial testing, her grandmother fell and sustained a hip fracture. Evaluation at that time revealed that she had sustained an occult myocardial infarction at some time in the past and was said to have an elevated CT. Her children do not have an elevated LDL-c, and their lipids are being followed.

Date	Total Cholesterol	HDL-c	LDL-c (mg/dl)	LDL-c (mmoles/L)	TG	RX
25 Oct 79	181	52	116	3.0	64	
17 Oct 81	222	55	161	4.2	29	
26 May 82	186	60	93	2.4	163	
22 March 88	204	47	148	3.8	54	
19 Dec 88	211	40	158	4.1	63	
28 Dec 89	220	52	154	4.0	68	
27 Oct 90	217	43	164	4.2	49	
30 Sept 92	229	52	166	4.3	56	
6 July 99	262	44	203	5.2	75	
2 May 01	286	45	227	5.9	70	
31 Dec 01	154	38	101	2.6	74	Lipitor 40 mg
8 July 03	296	42	241	6.2	63	Ran out of Lipitor 40 mg
7 May 05	159	41	107	2.8	55	Switch to Crestor 20 mg
30 Aug 07	255	34	202	5.2	94	Ran out of Crestor
28 May 09	178	39	124	3.2	76	
3 May 10	147	32	97	2.5	92	
18 Aug 14	185	39	128	3.3	92	
16 Feb 17	161	49	100	2.6	62	↑ Crestor to 40 mg
11 Dec 17	165	44	108	2.8	64	

Table: *Cholesterol test results over time.*
LDL-c: Means Low-Density Lipoprotein Cholesterol; HDL-c: Means High-Density Lipoprotein Cholesterol; TG: Means Triglycerides.

Here it should be noted that the HDL-c levels were measured by the precipitation methodology. This methodology was used by the auto-analyzers at the local laboratory until early May of 1999, where after the enzymatic methodology was utilized. These two differing methodologies give differing results, the latter method giving a result on the order of 10 mg/dl (0.25 mmoles/L) higher than the former method. LDL-c is not measured, but rather calculated by the Friedewald formula [2]. Hence the LDL-c level using the enzymatic method

will be on the order of 10 mg/dl (0.25 mmoles/L) lower than then would have been obtained, had the precipitation method been used. All lipids presented in the table are based on the precipitation method of HDL-c determination or are corrected from the enzymatic method values to the equivalent values that would have been obtained had the precipitation method been used.

Discussion

This patient had serial testing over the years, as indicated in the table. Genetic testing was not available at the time of her initial testing and was not ordered subsequently. Serial lipids were followed. When her LDL-c became severe and she expressed no desire to have more children, statin therapy was initiated on 2 May 2001, with the results given in the table. The patient now lives out of state, but is alive and well to the best of the author's knowledge. She continues on statin therapy.

In this report, the author the author presents the case of a lady who initially had normal LDL-c levels, but who subsequently developed T-II HLP. This case illustrates the value of serial testing.

Since the only reason to treat dyslipidemia is the prevention of atherothrombotic disease (ATD), or if extant, then prevention of future ATD events, it is important to know the timing of the development of the dyslipidemia. The presence and severity of plaque depend upon the degree of the dyslipidemia and the duration of time during which the dyslipidemia has been operative. The intensity of therapy depends upon the degree of severity of the dyslipidemia and the length of time the dyslipidemia has been operative. (Of course the presence/absence of the other ATD risk factors, such as cigarette smoking, hypertension and diabetes, will also influence ATD risk, but are not germaine to this case report. This has been discussed elsewhere [3,4]). Early detection of the dyslipidemia permits the treating physician to estimate the presence/absence of plaque and hence the intensity with which the patient must be treated. It is widely assumed that it is easier to prevent plaque than to stabilize/regress it.

The finding of a normal LDL-c in childhood/adolescence does not preclude a marked elevation later in life. The well-known rise of LDL-c in the pre-, peri-, and post-menopausal times of female life is more gradual in nature. This case is entirely different and hence is the subject of this report.

Conclusion

The presence of a normal LDL-c in childhood/adolescence does not preclude a more marked LDL-c elevation within a relatively few years. Serial testing is therefore warranted. Such testing is valuable if an abnormality is demonstrated, but a normal result allows the physician treating his/her patient later in life to be able to approximate when the dyslipidemia began and hence to adjust his/her therapy. Similar cases from other physicians should be reported to determine how frequent cases such as this case occur.

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