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Case Report

Patient Receiving Statin Therapy Achieves an Age of 100 Years: Report of a Case

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Abstract

Patient receiving Statin Therapy Achieves an Age of 100 Years.

Introduction: Treatment of dyslipidemia in the very old patients is, for some, an area of some controversy. This Case Report describes an elderly gentleman aged 71 years, who was found to have dyslipidemia and hypertension. Additionally, he was a cigarette smoker. His dyslipidemia was treated, as was his hypertension. He stopped smoking cigarettes. He has now achieved the age of 100 years, still taking his medications.

Materials and Methods: Case Report with review of medical records

Results: The patient was treated with statins and calcium channel blockers. He quit smoking cigarettes. His initial statin was lovastatin and the statin was upgraded to eventual rosuvastatin in 2003. He has been maintained on rosuvastatin since then. The lipid results are given in the table and show that the target goal was achieved once the high-intensity statins were initiated. At age 97 years he developed congestive heart failure, which has been treated.

Conclusions: This case partners with a previously reported case of a 102 year old lady who was still taking her statins some 25 years after she sustained an acute myocardial infarction while seeing another physician. The author concludes that statin therapy need not be stopped just because a patient achieves age 75 years. (The author has numerous patients aged 75 years and older who continue to take statin therapy without adverse reactions and continue to do well.).

Keywords: *Patient Receiving; Therapy Achieves*

Introduction

The internet is rife with commentaries about the adverse effects-usually myalgias-of statins. Patients are afraid to take statins because of these perceived adverse reactions. This situation may/may not be related to the anti-cholesterol crusade, which still exists, even among physicians. There can be little doubt that dyslipidemia is at the root of atherothrombotic disease (ATD) [1-10]. Indeed, the author has presented data to show that there is a rank order to ATD risk factors with respect to their importance: cigarette smoking, dyslipidemia, and hypertension, with some contribution by the very high blood sugar levels of uncontrolled diabetes [11]. The purpose of this report is to show that people may safely take statins even into very old age and thus delay the onset of clinical ATD.

Report of a case

The patient herein reported was 71 years old when he presented for evaluation in 1991. His blood pressure was not elevated, but he did smoke cigarettes. His initial lipid profile was abnormal, as was a confirmatory test. Therapy was initiated and the results of a representative sample of his lipid profiles, initially and on therapy, are given in the Table. The columns to the left are the various lipid profiles, accompanied by two-hour postprandial (pp)blood sugar levels when available. The comments to the right show the various therapies provided the patient. HDL refers to high-density lipoprotein; LDL, to low-density lipoprotein, and TG refers to triglycerides. AAA means abdominal aortic aneurysm and CHF means congestive heart failure. In addition to treating the patient's dyslipidemia, the author instructed him to cease smoking cigarettes, which he did. The comment "free" refers to a promotion by a local pharmacy wherein atorvastatin was given at no charge to the patient.

Date	Total Cholesterol	HDL Cholesterol	LDL Cholesterol	TG	2 hr pp glucose	
22 January 91	265	38	198	147	106	
7 February 91	262	35	207	100		
						Lovastatin 20 mg, stop smoking
25 July 91	158	35	99	119		
1 October 91	166	40	100	130		
31 March 92	157	36	95	130		
29 October 92	173	42	108	117		
						Stop Lovastatin; Add Pravastatin 10 mg, Niacin
23 October 93	180	34	122	120		
						Stop Pravastatin + Niacin; Add Simvastatin 20 mg
17 Mar 94	142	32	80	150		
18 April 95	161	24	100	184		
						Continue Simvastatin; Add Niacin
26 September 95	137	30	84	115		
11 April 96	131	31	76	119		
						↓Simvastatin 10 mg; Continue Niacin
22 April 97	141	33	88	102		
						Switch to Atorvastatin 5 mg
11 June 99	139	26	89	121		
						↑Atorvastatin 20 mg
5 October 99	119	23	70	132		
28 June 01	110	19	68	110		
16 July 02	120	19	74	137	154	
2 December 03	126	15	81	149	137	Switch to Rosuvastatin 10 mg
22 July 04	104	16	70	89		
1 June 06	110	23	60	133		
5 June 08	116	24	66	130	119	
11 November 11	107	23	65	103		
						Switch to Atorvastatin 20 mg (free)
7 May 13	114	17	63	168		
20 April 15	105	24	57	117	124	
						13 April 15 AAA on CAT
						16 April 16 CHF
29 September 17	100	23	54	113	190	
11 September 18	98	13	41	221	275	
12 March 19	124	18	61	226	292	
						Continue Atorvastatin; Pioglitazone→detemir
18 August 20	104	8	71	126	498	

Table 1

Here it must be mentioned that in 1999, at least in the author's local hospital laboratory, a change in the measurement of HDL-cholesterol (HDL-c) occurred, in which the older precipitation method of HDL-c measurement was changed to the newer enzymatic method. Unfortunately, the two methodologies do not give the same results, the newer methodology giving an HDL-c value on the order of 10 mg/dl (0.25 mmoles/L) higher than would have been obtained had the precipitation method been used. Since LDL-cholesterol (LDL-c) is not usually measured but rather calculated according to the Friedewald formula (12), this means that the LDl-c calculated by the newer HDL-c methodology will be on the order of 10 mg/dl (0.25 mmoles/L) lower. All HDL-c and LDL-c values give in the table are either measured by thee precipitation method or by converting the enzymatic HDL-c to its equivalent using the precipitation method. Hence, the 198 mg/dl (5.0 mmoles/L) value for LDL-c on the initial test would be equivalent to 188 mg/dl (4.75 mmoles/L) using the enzymatic methodology, and the 99 mg/dl (2.5 mmoles/L) would similarly be equivalent to 89 mg/dl (1.2.25 mmoles/L). The primary prevention goal of 99 mg/dl or lower was achieved permanently in 1995.

In 2015 the patient was found to have an AAA as an incidental finding of CAT scan of his abdomen and later that same year he was found to be in CHF. The AAA is being followed and the CHF has been successfully treated.

Discussion

The course of therapy of the patient's dyslipidemia has been presented. He tolerated his various therapies quite well and is still alive and living in the community, albeit at his children's home. He did develop clinical ATD, but in the author's long experience, everyone who lives long enough and does not die of something else will eventually develop some form of ATD.

The author has previously reported the case of a female patient who sustained an acute myocardial infarction at age 75 years while under the care of another physician. She underwent coronary artery bypass surgery of three arteries and did well for a few years, when her chest pains recurred. When her physician did nothing to help her, she came to the author's practice. Her dyslipidemia was treated and she was given nifedipine for her chest pain and aspirin as an anti-platelet agent [13]. At age 104 years, she stopped her statin for reasons never really explained. A few months later she developed mental symptoms, stopped eating, and shortly thereafter died. This case report is intended to partner with the previously reported case.

The afore mentioned lady's therapy was guided by a ratio between LDL-c and HDL-c, which is the author's usual practice. In this case, the patient's therapy was guided by LDL-c levels, since his HDL-c was very low and since a very low HDL-c will always give a high ratio, even when ATD risk is low. The author makes this point for readers who wish to compare the two cases.

Conclusions

The author reports this case to show that stain therapy can be safely used even in the very old patients to stave off clinical ATD. This report is presented to show what is possible when dyslipidemia is treated even in very old age.

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