CASE REPORT

Medical Therapy Induced Regression of Plaque in a Female Patient with ASCVD

Siming Guan, MD, Yanqing Zhang, MD, Bin Wang, MD and Wei Li, MD

Summary

The initiation and progression of atherosclerotic cardiovascular disease (ASCVD) has always been associated with a series of risk factors. Evidences of statin therapy from randomized clinical trials are abundant, whereas discussions regarding patients with ASCVD without evidence-based risk factors are rare. Here, we describe a case of a 58-year-old woman who was diagnosed with ASCVD with none of these evidence-based risk factors. After four years of medical interventions, including atorvastatin, the patient recovered completely from severe chest pain with significant regression of atherosclerotic plaques in coronary arteries.

Key words: Atorvastatin

Clinical benefits of lipid lowering with statins are well established for patients with ASCVD. Data obtained from intravascular ultrasounds (IVUS) have shown that statin intervention may halt or even reverse the progression of coronary atherosclerosis.\(^1\) Although statins\(^2\) effects on reducing cardiovascular risk and stabilizing or even reversing coronary atherosclerotic plaque were strongly proven, poor compliance with these drugs is still an important clinical problem.\(^2\) This report describes a case of a patient with ASCVD who recovered significantly from severe coronary stenosis with long-term high-intensity atorvastatin treatment.

Case Report

A 58-year-old female patient presented with a 6-month history of intermittent exertional chest pain when performing her daily activities and required absolute rest for relief. The patient had no family history of ASCVD and had no history of smoking, hypertension, diabetes, or dyslipidemia. Her oral medications prior to admission included 100 mg aspirin once daily and 23.75 mg metoprolol succinate once daily.

Coronary computed tomographic angiography (CTA) was performed, and the results suggested stenosis of the culprit proximal left anterior descending (LAD) artery by 90% and right coronary artery (RCA) stenosis by 60%. Although Prof. Guan Siming had advised stent implantation, the patient did not prefer this recommended interventional therapy. Then, conservative strategies were prescribed. The patient was initiated with 60 mg atorvastatin daily after being diagnosed with ASCVD. Therefore, her oral medications after this admission included 60 mg atorvastatin once daily, 100 mg aspirin once daily, 23.75 mg metoprolol succinate once daily, and 0.5 mg nitroglycerin sublingual tablets as needed for angina episodes.

Upon admission, the patient’s height and weight on admission were 151 centimeters and 54 kilograms, respectively. Vital signs on admission included a blood pressure of 110/70 mmHg, body temperature of 36.8°C, pulse rate of 104 beats per minute, respiratory rate 15-20 breaths per minute, a pulse oxygen saturation of 97%-98% on room air, normal serum glucose level (4.7 mmol/L), and HbA1c level (6.0%). The initial creatine kinase (CK) was 119 U/L, which increased to 149 U/L after 7 days and decreased to 107 U/L after 5 weeks. The initial total bilirubin was 14.9 μmol/L, which increased to 21.8 μmol/L after 7 days and decreased to 14.6 μmol/L after 5 weeks. Although higher than normal limits, the values of CK and total bilirubin did not reach the double upper limits and reverted to normal ranges 5 weeks later. These results suggested that the patient was completely tolerant of daily administration of 60 mg atorvastatin.

After the initiation of atorvastatin therapy, coronary CTA was performed after one, two, and four years. The initial luminal narrowing of proximal LAD was 90%, which reduced to 70-80% within one year and further reduced to 20-30% within four years (Figure 1 and Table I). Additionally, the initial luminal narrowing of RCA was 60%, which reduced to 30-40% within one year and further reduced to 10-20% within four years (Figure 1 and Table I). The patient’s lipid profile on the date of first admission included a total cholesterol of 153 mg/dL, triglycerides of 68 mg/dL, high-density lipoprotein cholesterol (HDL-C) of 43 mg/dL, and a low-density lipoprotein cholesterol (LDL-C) of 98 mg/dL. During four years of...
follow-up, the levels of LDL-C ranged from 39 mg/dL to 106 mg/dL and HDL-C from 42 mg/dL to 72 mg/dL (Table II).

Considering the alleviation of anginal symptoms, newly detected regression of coronary atherosclerotic plaque, and the low LDL-C level, Prof. Guan Siming advised the patient to decrease the dose of atorvastatin to 40 mg one year later. Four years later, the dose of atorvastatin was decreased to 20 mg because of rare occurrences of chest pain, further regression of coronary atherosclerotic plaque, and the consistently low LDL-C level. Currently, the patient’s oral medications after this admission included 20 mg atorvastatin once daily, 100 mg aspirin once daily, and 23.75 mg metoprolol succinate once daily (Figure 2).

### Discussion

Atherosclerosis is the primary cause of ASCVD, cerebral thrombosis, and peripheral vascular disease. A primary strategy in these efforts recommended to reduce atherosclerotic cardiovascular risk has been statin therapy. Recently, Keraliya, et al. reported a case of a patient with hyperlipidemia and a family history of coronary artery disease whose severe narrowing of the proximal left circumflex coronary artery regressed with four-year medical therapy. In this paper, we discuss the case of a female with severe coronary stenosis whose atherosclerotic plaque regressed with long-term, high-intensity atorvastatin intervention. The patient was not a cigarette consumer and did not have a history of obesity, hypertension, diabetes, or dyslipidemia.

During the past three decades, an impressive development in the battle against atherosclerotic diseases has been witnessed with the use of statins, which seem to retard or even halt the progression of the atherosclerotic process. A series of clinical trials to determine the effects of statins on coronary atherosclerotic plaques was conducted. In these studies, intravascular ultrasounds IVUS was used to obtain precise and reproducible serial measurements of atherosclerotic plaques in the coronary arteries. The percent atheroma volume (PAV) or total atheroma volume was calculated as the primary efficacy end point. For ex-

### Table I. Evaluation of Coronary Atherosclerosis During Follow-Up

<table>
<thead>
<tr>
<th>Time</th>
<th>LAD (eccentric, non-calcified)</th>
<th>RCA (eccentric, non-calcified)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Length (mm)</td>
<td>Narrowing (%)</td>
</tr>
<tr>
<td>June 2012</td>
<td>12</td>
<td>90</td>
</tr>
<tr>
<td>June 2013</td>
<td>10</td>
<td>70-80</td>
</tr>
<tr>
<td>May 2014</td>
<td>6</td>
<td>60-70</td>
</tr>
<tr>
<td>May 2016</td>
<td>-</td>
<td>20-30</td>
</tr>
</tbody>
</table>

LAD indicates left anterior descending and RCA, right coronary artery.

### Table II. Changes in the Dose of Atorvastatin and Serum Lipids Panel during Follow-Up

<table>
<thead>
<tr>
<th>Time points</th>
<th>June 2012</th>
<th>June 2013</th>
<th>May 2014</th>
<th>May 2016</th>
</tr>
</thead>
<tbody>
<tr>
<td>dose (mg/day)</td>
<td>60</td>
<td>40</td>
<td>30</td>
<td>20</td>
</tr>
<tr>
<td>TC (mg/dL)</td>
<td>153</td>
<td>127</td>
<td>141</td>
<td>144</td>
</tr>
<tr>
<td>HDL-c (mg/dL)</td>
<td>43</td>
<td>49</td>
<td>58</td>
<td>66</td>
</tr>
<tr>
<td>LDL-c (mg/dL)</td>
<td>98</td>
<td>46</td>
<td>52</td>
<td>58</td>
</tr>
<tr>
<td>TG (mg/dL)</td>
<td>68</td>
<td>115</td>
<td>91</td>
<td>103</td>
</tr>
</tbody>
</table>

TC indicates total cholesterol; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; and TG, triglycerides.

Figure 1. Coronary computed tomographic angiography results at four different time points before or after atorvastatin intervention. White arrows indicate proximal LAD stenosis; black arrows indicate RCA stenosis. LAD indicates left anterior descending and RCA, right coronary artery.
ample, in the SATURN study, which was designed and performed to assess the effects of two intensive statins (atorvastatin and rosuvastatin) on coronary atherosclerotic plaque regression, the primary efficacy endpoint, PAV, decreased by 0.99% with 80 mg atorvastatin and by 1.22% with 40 mg rosuvastatin after 104 weeks of therapy. For the regression of atherosclerotic plaque, the effective rates of these two agents were similar: 63.2% with atorvastatin and 68.5% with rosuvastatin. For patients with acute coronary syndrome (ACS), the extent of PAV regression seems more significant than for patients with stable coronary diseases. The degree of change in PAV was 17.5% for total patients with 4 mg pitavastatin or 20 mg atorvastatin in the JAPAN-ACS study. Recently, IVUS data from the STABLE study demonstrated that rosuvastatin treatment could change plaque composition (decrease in percent necrotic core volume and thin-cap fibroatheroma rate) and reduce plaque volume in nonculprit coronary lesions. For other types of atherosclerotic diseases, including cerebrovascular disease and peripheral artery disease, statin therapy has been associated with decreased incidence of stroke and improved amputation-free survival. Statins have many other roles beside lowering cholesterol, such as anti-inflammatory effects, immunomodulatory effects, and increasing the release of vascular endothelial nitric oxide. Collectively, compliance with statin therapy is of great importance for major events prevention and atherosclerotic plaque regression.

In the present case, coronary atherosclerotic stenosis was defined by performing coronary CTA. Then, the patient was treated with medical prescriptions, including atorvastatin, metoprolol, aspirin, and sublingual tablets of nitroglycerin as needed for angina episodes. Over time, her symptoms resolved completely. Four years later, repeat coronary CTA revealed a distinct reduction of stenosis in the proximal LAD and in the RCA. Additionally, the patient did not have any evidence-based risk factors. In conclusion, for chronic stable patients with ASCVD lacking risk factors, coronary atherosclerotic plaque can regress with medical therapy, even when severe stenosis is present.

Disclosures

Conflicts of interest: The authors have no conflicts of interest to disclose.

References