Successful reintroduction of statin therapy after myositis: was there another cause?

Clinical record

A 43-year-old man presented with myalgia and upper respiratory tract symptoms. He had noted weight gain of 7 kg, lethargy and cold intolerance over the previous 12 months. He had no chest pain, rash or fever.

His past history included acute myocardial infarction (AMI) 12 years earlier. Subsequently, he had been taking low-dose aspirin. He commenced simvastatin 2 years after the AMI for hypercholesterolaemia (> 7 mmol/L). His sister and father have hypercholesterolaemia and his father had an AMI before 40 years of age, suggesting heterozygous familial hypercholesterolaemia. Two years before presentation, his hypolipidaemic therapy was switched from simvastatin to atorvastatin, although he denied experiencing any side effects of the former. He reported consuming 40–50 g/day of alcohol, and that he had recently quit smoking.

Examination revealed that he was hypertensive (160/90 mmHg) and obese (body mass index, 32 kg/m²). He had marked corneal arcus, but no tendon xanthomas or xanthelasma, and no goitre. There was no muscle tenderness and power was normal. Reflexes were delayed.

The results of some of the laboratory tests performed are shown below. In addition, normal findings were recorded for full blood count, erythrocyte sedimentation rate, and C-reactive protein. Serological tests for hepatitis, and test results for protein electrophoresis, antinuclear factor, immunoglobulin and prostate-specific antigen, were all normal. There was no myoglobinuria, haematuria or proteinuria. Chest x-ray findings were normal, electrocardiography showed old Q waves, and an abdominal ultrasound showed hepatic steatosis. Apart from γ-glutamyltransferase, normal results were obtained for all other liver function tests.

Statin-induced myositis was suspected and atorvastatin was stopped. Thyroid function tests instituted after endocrine review 5 days later revealed hypothyroidism (free T₄, 1.9 pmol/L [normal range, 0.30–5.00 pmol/L]). Tests for thyroid peroxidase and thyroglobulin antibodies both gave positive results, suggesting Hashimoto’s thyroiditis. The patient was prescribed thyroxine 50 µg daily, with subsequent slow-dose titration because of ischaemic heart disease.

The Box (page 473) shows the gradual normalisation of the creatine kinase (CK) level over time. Lipid levels remained elevated despite thyroxine therapy. Simvastatin 10 mg/d was recommenced with careful monitoring. The dose was increased progressively up to 80 mg daily for persistent hyperlipidaemia. Despite full-dose statin therapy, there was no change in the findings of liver function tests and only mild asymptomatic elevation of CK levels.

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Result (normal range)</th>
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<tbody>
<tr>
<td>Creatine kinase (CK) (IU/L)</td>
<td>4890 (&lt; 240)</td>
</tr>
<tr>
<td>CK-MB index (%)</td>
<td>2.0 (&lt; 5.0)</td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>9.0 (&lt; 5.5)</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>2.7 (&lt; 2.0)</td>
</tr>
<tr>
<td>High-density lipoprotein cholesterol (mmol/L)</td>
<td>1.2 (&gt; 1.0)</td>
</tr>
<tr>
<td>Urea (mmol/L)</td>
<td>6.7 (2.3–7.6)</td>
</tr>
<tr>
<td>Creatinine (mmol/L)</td>
<td>0.197 (0.05–0.11)</td>
</tr>
<tr>
<td>γ-Glutamyltransferase (U/L)</td>
<td>196 (10–55)</td>
</tr>
</tbody>
</table>

Lessons from practice

- Hypothyroidism should be considered as a secondary cause of hypercholesterolaemia in all patients.
- Patients developing myopathy when taking statin therapy should be tested for hypothyroidism.
- It may be safe to cautiously reintroduce statin therapy in patients with myopathy, once coexisting hypothyroidism has been treated.
Statin therapy in patients with a history of myopathy, once coexisting hypothyroidism has been treated.

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Competing interests: None identified.


(Received 11 Nov 2003, accepted 19 Feb 2004)