Extreme hypercholesterolaemia and hypertriglyceridaemia in a man newly diagnosed with type 2 diabetes mellitus

Ashraf Saleh

CASE
A Caucasian man aged 46 years, working as an oil rig area coordinator in remote South Australia, presented with incidental hyperglycaemia. He was sent to an onsite nurse because of lethargy and anorexia over the previous few weeks. The nurse noted an elevated blood sugar level on capillary blood sampling (reading ‘high’ on the glucometer) and sent him to the nearest regional centre for further investigations. He had not undertaken a medical examination in >10 years.

He noticed increasing shortness of breath on exertion, and polydipsia and polyuria progressing over the previous month. He had not had any chest pain, dyspnoea, orthopnoea, palpitations or abdominal symptoms. His family history was unknown, as he had been adopted as a child. He had no previous medical or surgical history and did not take any regular medications. He drank 2–3 standard alcoholic beverages per night on average, occasionally consuming up to six alcoholic beverages in one night.

He was centrally obese, with a blood pressure of 160/98 mmHg and a resting pulse rate of 104 beats per minute. Jugular venous pressure was undetectable, and he had no peripheral or sacral oedema. He had good peripheral pulses and an intact neurological system. His chest was clear, but there was tenderness in the epigastrium. There was no abdominal mass. His vision was 6/5 bilaterally. Urinalysis showed 3+ protein and 3+ glucose but no leukocytes, nitrites or blood. He had an initial non-fasting blood sugar level of 18.9 mmol/L and was sent for formal blood testing.

ANSWER 1
Metabolic syndrome is defined as a concomitant cluster of risk factors including elevated waist circumference (population group–specific), triglycerides ≥1.7 mmol/L, high-density lipoproteins (HDL) <1.0 mmol/L in men and <1.3 mmol/L in women, blood pressure ≥130/85 mmHg and fasting glucose >5.5 mmol/L.¹ The 2006 Bettering the Evaluation and Care of Health study revealed that the prevalence of metabolic syndrome in the Australian population was 15.6%.² There is a significantly increased risk of macrovascular and microvascular disease seen in people with metabolic syndrome,³ with no apparent symptomatology.

ANSWER 2
A review of available data on cardiovascular comorbidities, including diabetes, suggests a stratified analysis of cardiovascular risk and treatment based on that degree of risk.⁴ Some studies recommend moderate-dose statin therapy for patients with dyslipidaemia aged ≥40 years for primary prevention even without diabetes.⁵⁻⁷ More aggressive therapy is considered on a case-by-case basis that incorporates atherosclerotic cardiovascular disease risk factors. The evidence for benefit is strong for patients with diabetes aged 40–75 years, an age group well represented in statin trials.⁸,⁹

CASE CONTINUED
Table 1 displays the patient’s initial and five-year follow-up blood test results. The initial blood specimen taken, shown in Figure 1, indicated he had severe dyslipidaemia. He had moderate albuminuria (259 mg/L) in a first morning sample, with an albumin:creatinine ratio of 35. His electrolytes, serum proteins and renal, liver and thyroid function tests were all normal. His tests were positive for the apolipoprotein E gene, which does not predispose him to hypertriglyceridaemia but may be associated with reduced fatty acid oxidation, contributing to the accumulation of tissue and plasma lipids.¹⁰

He was sent for urgent cardiovascular workup and management of his severe dysglycaemia in a tertiary referral hospital.
by a physician. He had a computed tomography coronary angiogram that identified an occluded right coronary artery and a coronary angiogram showing a chronic complex right coronary artery occlusion, a stenotic small obtuse marginal branch and preserved left ventricular function. A subsequent myocardial perfusion scan showed no inducible ischaemia, suggesting the right coronary artery was well collateralised; therefore, no percutaneous intervention was deemed necessary. He was put on atenolol, aspirin, insulin glargine, insulin aspart, metformin, fenofibrate, rosuvastatin and ramipril.

He had no coronary events for the following five years on this treatment regimen. His biochemical parameters at this time point, comparing them to his initial results, are shown in Table 1. Despite anthropometric measures not significantly improving because of a sedentary lifestyle, his biochemical profile had gained significant headway in establishing a legacy effect to preventing the endpoints of myocardial infarction and stroke.

**QUESTION 3**
How should patients with severe dyslipidaemia be managed while awaiting definitive cardiovascular treatment?

**ANSWER 3**
Low-density lipoproteins (LDL) >4.0 mmol/L or total cholesterol >7.5 mmol/L translate to an innately high cardiovascular event risk according to the Guidelines for the management of absolute cardiovascular risk. This warrants immediate active risk management treatments including simultaneous lipid lowering and blood pressure lowering pharmacotherapy unless contraindicated. The target for blood pressure is ≤140/90 mmHg unless the patient has diabetes or is exhibiting albuminuria; in this case, ≤130/80 mmHg is recommended. Lipids should be brought down with statins as the first-line treatment, to a total cholesterol of <4.0 mmol/L and LDL of <2.0 mmol/L.

**QUESTION 4**
How prevalent is dyslipidaemia in patients with type 2 diabetes mellitus (T2DM)?

**ANSWER 4**
Dyslipidaemia occurs frequently in T2DM; a recent study in China indicates a prevalence of 67%, and in a US study, over 70% of patients with diabetes did not meet goal lipid levels.

**QUESTION 5**
What is the clinical relevance of the dyslipidaemia in T2DM?

**ANSWER 5**
Patients with severe dysglycaemia and dyslipidaemia have significant risks of cardiovascular morbidity and mortality unless these modifiable risk factors are aggressively managed. Primary care physicians should note that cardiovascular risk factors are often insidious, and a high index of suspicion needs to be raised in at-risk patient groups.

---

**Table 1. Biochemistry profile of the case patient, prior to treatment and five years post-treatment for extreme dyslipidaemia**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Year 2013</th>
<th>Year 2018</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>18.3</td>
<td>5.4</td>
<td>3.9–5.5</td>
</tr>
<tr>
<td>HDL (mmol/L)</td>
<td>*</td>
<td>0.9</td>
<td>0.9–1.5</td>
</tr>
<tr>
<td>LDL (mmol/L)</td>
<td>*</td>
<td>2.5</td>
<td>0.0–4.0</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>53.4</td>
<td>4.4</td>
<td>0.6–2.0</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>11.2</td>
<td>6.1</td>
<td>&lt;6.5</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>37.0</td>
<td>35.5</td>
<td>20–25</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>113</td>
<td>110</td>
<td>&lt;90</td>
</tr>
<tr>
<td>Five-year CVD risk (%)*</td>
<td>&gt;15</td>
<td>6</td>
<td>–</td>
</tr>
</tbody>
</table>

*HDL and LDL were not able to be assayed on this specimen because of profound lipidaemia

---

**Figure 1.** Photograph of the initial fasting blood specimen taken from the patient following centrifugation by the referring laboratory.
Acknowledgements
The author wishes to thank the People First Health Group for the privilege of accessing patient information from their medical database.

References