Hyperalphalipoproteinemia in Epileptic patient – Cardiovascular Protection from Carbamazepine Use?
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Background

High density lipoprotein is a plasma lipid-protein complex composed of lipids and alphalipoproteins (apolipoproteins A-I and A-II). It is involved in reverse transport of cholesterol from peripheral tissue to liver, allowing cholesterol degradation.

HDL cholesterol (HDL-C) levels are inversely associated with cardiovascular risk. HDL-C levels can be elevated due to genetic causes or drugs. We present a case where significantly raised HDL levels caused some concern at the outset.

Case

A 66-year-old female was referred to lipid clinic for elevated cholesterol. She had a background of epilepsy, which was well controlled on carbamazepine.

There were no peripheral stigmata of hyperlipidaemia. She had no family history of premature coronary artery disease and the calculated cardiovascular risk was low.

Case Progress

Separate cardiology review for chest pain resulted in coronary angiogram and transthoracic echocardiogram. These confirmed normal coronary arteries and preserved ventricular function. Abnormal liver function triggered Hepatology investigations and Fibrosan®. The cause was deemed to be carbamazepine.

Repeat lipid profile revealed similar picture. Lipid-lowering treatment was not started especially in the setting of liver dysfunction.

Discussion

Hyperalphalipoproteinemia (HALP) is a condition where HDL-C levels are elevated raising total cholesterol values. LDL-cholesterol may be normal or elevated.

Peripheral stigmata of hyperlipidaemia are usually absent. HALP can result from primary causes due to familial genetic defects or can be secondary to drugs, alcohol, or primary biliary cirrhosis. Most patients are incidentally diagnosed. Medical therapy is rarely required.

Carbamazepine is well documented to cause elevated HDL-C, attributed to its enzyme inducing effect leading to increased hepatic synthesis of alphalipoproteins.

Conclusion

Careful clinical and biochemical evaluation is essential in patients presenting with lipid abnormalities.

Statin therapy is not a panacea for all patients with hypercholesterolaemia.

Table 1: Fasting lipid profile

<table>
<thead>
<tr>
<th>Fasting Results</th>
<th>Result</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Cholesterol</td>
<td>9.7 mmol/l</td>
<td>3.5-5.0</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>2.1 mmol/l</td>
<td>0.8-1.8</td>
</tr>
<tr>
<td>HDL</td>
<td>3.5 mmol</td>
<td>1.0-2.10</td>
</tr>
<tr>
<td>LDL</td>
<td>5.2 mmol/l</td>
<td>1.5-4.0</td>
</tr>
<tr>
<td>TC: HDL Ratio</td>
<td>2.8 mmol/l</td>
<td>0-4.5</td>
</tr>
<tr>
<td>Apolipoprotein A-I</td>
<td>3.65 g/L</td>
<td>1.25 – 2.15</td>
</tr>
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