Management of Very Severe Hyperlipidaemias: A rare case report

Case Report

ABSTRACT

Hyperlipidaemia and hypertriglyceridemia (HTG) is an independent risk factor for accelerated cardiovascular disease and acute pancreatitis. Our patient presented with very severe hyperlipidaemias and we successfully managed with using heparin and insulin. Heparin and insulin regime appear to be safe, effective and inexpensive first line therapy for inpatient management of severe hyperlipidaemias.

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Citation of the Article

I. INTRODUCTION

An elevated plasma lipid is a common finding in clinical practice. Hyperlipidaemia is characterized by elevation in one or more lipids including Cholesterol, triglycerides, LDL, VLDL or reduced HDL levels. Hyperlipidaemia is one of the major risk factors for cardiovascular disease. Risk of pancreatitis is high when triglyceride levels are above 10mmol/l.

II. CASE REPORT

58 year old gentleman was electively admitted from diabetic day care after he was found having severe hypercholesterolaemia and hypertriglyceridaemia. He has past medical history of type 2 Diabetes for more than 20 years. Hypertension, Dyslipidaemia, Peripheral vascular disease, right below knee amputation, Ischemic heart disease and CABG. There was no family history of familial hyperlipidaemias and he denies any history of smoking or alcohol abuse.

Routine bloods biochemistry for lipid profile was requested, which could not be processed as sample was severely lipaemic. Triglycerides level was 65.4mmol/l (normal 0-1.9mmol/l) and cholesterol was 17mmol/l (normal 0-5.2mmol/l). HDL was 0.53mmol/l (normal 0.9-1.4 mmol/l) and LDL was 0.3mmol/l (normal 3.36-4.11 mmol/l). Previous cholesterol level was 3.7mmol/l and triglyceride level was 5.3mmol/l about a year back. His diabetes was also uncontrolled as evidenced by HbA1c of 92mmol/mol.

Possible causes for very high lipids level in our patient were uncontrolled DM (HbA1c 92mmol/mol), Diet with high fat and carbohydrates content, positive energy intake, and insufficient physical activity.

Patient was at imminent risk of having acute pancreatitis and long term risk of accelerated CV disease. Therefore he was admitted and treated with intravenous insulin and fluids (NaCl vs. dextrose).along with full dose subcutaneous LMWH (Enoxaparin 1mg/kg BD) was also given. He was continued with his regular lipid lowering agents Atorvastatin was increased from 20 to 80mg daily and commenced on Gemfibrozil (600mg BD), Omega-3 Fatty acids. He was encouraged to have low fat/calorie diet.

On 2nd day of admission cholesterol dropped to 11.3mmol/l. On 3rd day cholesterol was 10.3mmol/l and triglycerides were 28.4mmol/l. IV insulin and fluids infusion as well as LMWH was continued for 4 days until triglycerides level came below 20mmol/l. Later IV insulin was switched to routine subcut insulin regime.

Table 1: Daily progress of Lipid profile while patient was on insulin, fluids and LMWH regime.

<table>
<thead>
<tr>
<th>Days</th>
<th>One</th>
<th>Two</th>
<th>Three</th>
<th>Four</th>
<th>Five</th>
<th>Six</th>
<th>Seven</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>17</td>
<td>14.6</td>
<td>11</td>
<td>10.3</td>
<td>8.7</td>
<td>6.7</td>
<td>5</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>65.4</td>
<td>58.6</td>
<td></td>
<td>28.4</td>
<td>21.2</td>
<td>17</td>
<td>13.1</td>
</tr>
<tr>
<td>HDL</td>
<td>0.53</td>
<td>0.5</td>
<td>0.48</td>
<td>0.51</td>
<td>0.48</td>
<td>0.54</td>
<td></td>
</tr>
<tr>
<td>LDL</td>
<td>0.3</td>
<td>0.3</td>
<td>0.9</td>
<td>1.3</td>
<td>1.3</td>
<td>1.3</td>
<td></td>
</tr>
</tbody>
</table>

III. DISCUSSION

Although no large studies have been conducted for safety and efficacy of heparin and insulin infusion, yet multiple case reports have been published for the treatment of severe hypertriglyceridemia. Mechanism of action is through activation of Lipoprotein lipase (LPL).Insulin promotes synthesis and activation of LPL thus leading to accelerated chylomicron degradation. LPL is bound to capillary endothelium by heparin sulphate proteoglycan chains. Heparin has stronger affinity for binding sites than heparin sulphate thus releasing LPL-Heparin complexes from endothelium to plasma. LPL metabolizes lipoproteins and lowers serum triglyceride levels. Insulin also Inhibits hormone-sensitive lipase in adipocytes.[1]

Both Unfractionated heparin (UFH) and LMWH have been used to treat Hypertriglyceridemia. Insulin has to be used as continuous infusion. Heparin and insulin appear to be safe, effective and inexpensive first line therapy in the management of severe HTG and HTG associated pancreatitis [2]. Bleeding risk need to be considered. No complications happened in our patient.

Patients with elevated lipids should be evaluated for secondary causes like excessive alcohol intake, untreated diabetes, endocrine conditions, renal or liver disease, pregnancy, autoimmune disorders, and use of certain medications(e.g.,thiazides, beta blockers, estrogen, isotretinoin,corticosteroids, antiretroviral protease inhibitors, immunosuppressants, antipsychotics) and treated accordingly.[3]

Mild to moderate hypertriglyceridemia should be treated with lifestyle modification and if fails, pharmacologic therapy. Severe HTG should be treated with medications to prevent the complications. Oral option include Statins, Fibrates, Ezetemibe, Niacin and omega-3 fatty acids. In very severe HTG, insulin and LMWH (as in our case) or therapeutic plasma exchange may be required.[4]

IV. CONCLUSION

Hypertriglyceridemia is associated with coronary artery disease and acute pancreatitis. In severe hypertriglyceridaemia (SHTG, triglycerides > 1,000 mg/dL or 11.3mmol/l), rapid lowering of plasma triglycerides (TG) is imperative. Treatment regimes include nutritional intervention, the use of lipid lowering drugs, therapeutic apheresis and heparin and insulin infusion. Heparin and insulin infusion treatment is indicated in medical emergencies to prevent hypertriglyceridemic pancreatitis. However, it is necessary to study more patients in order to identify the ideal treatment regimes for diabetic patients and to assess their efficacy and safety.

V. CONFLICT OF INTEREST

The authors confirm that there are no conflicts of interest.
REFERENCES


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