Terlipressin Induced Cardiac Arrest: A Case Report

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Abstract

Terlipressin is a vasoconstrictor with effect on vascular smooth muscles in the portal and splanchnic circulation, thereby decreasing blood pressure in esophagus varices. Therefore, terlipressin is recommended as first-line of pharmacological therapy in patients with bleeding esophagus varices. Terlipressin has been reported in a few cases to induce prolonged QT interval causing cardiac arrhythmias, mostly Torsade de Pointes. In this case report, a 34-year-old woman admitted with upper gastrointestinal bleeding were treated with terlipressin and subsequently had cardiac arrest. Monitoring during cardiac arrest showed ventricular fibrillation. After return of circulation, laboratory results showed hypokalemia and hypomagnesemia. Other likely causes to cardiac arrest were ruled out by clinical examination, chest x-ray and echocardiography. ECG showed prolonged QT interval. The administration of terlipressin, the hypokalemia and the hypomagnesemia may all be either cause or a contribution to ventricular fibrillation. In conclusion, this case report illustrates the importance of measurement of electrolytes upon admission along with observation of the patient during and after administration of terlipressin.

Keywords: Liver cirrhosis, Glypressin, Torsade de point, Arrhythmia, Esophagus varices

Introduction

Increased portal venous pressure causes esophagus varices, mostly due to liver cirrhosis. Esophagus varices have a potential risk of bleeding and an acute esophageal bleeding is associated with a 20-50% mortality [1]. The guideline of World Gastroenterology Organization for treatment of bleeding esophagus varices has terlipressin-infusion as first-line of pharmacological therapy followed by gastroscopic banding of the bleeding site during the first 12 hours of hospitalization [2]. Terlipressin has been showed to reduce mortality, rebleeding and failure of hemostasis [3]. However, only a few case reports have been published about the serious side effect of terlipressin infusion resulting in cardiac arrhythmia (Table 1).

Terlipressin

The prodrug terlipressin used to treat esophagus varices is cleaved enzymatic to lysinvasopressin, a synthetic anti diarrheic hormone. Approximately, 30 min after intravenous injection of terlipressin, lysinvasopressin can be detected in plasma and maximum concentration occurs after 1-2 hours. Terlipressin has a plasma half-life of 40 min and is 99% metabolized by peptidases. The effect of terlipressin is primarily vasoconstrictive on vascular smooth muscles in the portal and splanchnic circulation, which is mediated by V1a receptors. This causes a decrease in esophageal venous blood pressure and thus the clinically required effect - hemostasis. However, terlipressin can increase systemic mean arterial pressure, which is also mediated by V1a receptors. The increased pressure can trigger a decrease in heart rate mediated by glomus caroticum. This bradycardic effect can further be amplified by all drugs known to cause bradycardia [4-8].

Case Presentation

A 34-years-old unmedicated female known with anorexia, DM: Diabetes, HT: hypertension, Hep C: hepatitis C, TdP: Torsade de Pointes, VT: Ventricular tachycardia.

Table 1: Cases of terlipressin-induced arrhythmias.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Male</th>
<th>Male</th>
<th>Male</th>
<th>Male</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Terlipressin dose</td>
<td>1 mg</td>
<td>1 mg every 6 hour</td>
<td>1 mg every 8 hour</td>
<td>1 mg every 6 hour</td>
</tr>
<tr>
<td>Admitting diagnosis</td>
<td>Bleeding esophageal varices</td>
<td>Bleeding esophageal varices</td>
<td>Weakness and ascites</td>
<td>Coffee-ground emesis</td>
</tr>
<tr>
<td>Comorbidity</td>
<td>DM, HT, Hep C, liver cirrhosis and kidney disease</td>
<td>Alcoholic</td>
<td>Liver cirrhosis and kidney failure</td>
<td>Alcoholic, chronic pancreatitis, liver cirrhosis</td>
</tr>
<tr>
<td>Country</td>
<td>Taiwan</td>
<td>India</td>
<td>Germany</td>
<td>Czech Republic</td>
</tr>
<tr>
<td>Potassium levels</td>
<td>4.22 mEq/L</td>
<td>3.4 mEq/L</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Magnesium levels</td>
<td>Unknown</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Cardiac arrhythmia</td>
<td>TdP same day as initial treatment</td>
<td>VT same day as initial treatment</td>
<td>TdP</td>
<td>TdP after 6 doses of treatment</td>
</tr>
</tbody>
</table>

Table 1: Cases of terlipressin-induced arrhythmias.

was without any pathology. Terlipressin was discontinued.

At the time of admission, the patient was slightly hypokalemic with a potassium of 3.3 mmol/L (reference 3.5-4.6 mmol/L). Supplements were administered. On the day of cardiac arrest, potassium had decreased to 2.1 mmol/L. Post-ROSC, the patient had hypomagnesemia on 0.64 mmol/L (reference 0.70-1.10 mmol/L) and supplements were given. Terlipressin was discontinued. The patient was transferred to intensive care unit without any new arrhythmias but died two weeks later due to hepatic encephalopathy.

Discussion

Terlipressin is known to induce QT prolongation. This may be enhanced by electrolyte abnormalities and by medication known to prolong QT interval. This case report illustrates the importance of measurement of electrolytes at the time of admission along with observation of patient during and after administration of terlipressin. The administration of terlipressin, the hypokalemia and hypomagnesemia might all be either cause or a contributor to ventricular fibrillation. In high-risk patients, cardiac monitoring should be considered.

Conflicts of Interest

None declared.

Source of Funding

None declared.

References


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