Case Report

Tumor Lysis Syndrome Occurring After Transarterial Embolization in a 70-year-old Man with a Hepatocellular Carcinoma Ruptured in a Motor Vehicle Accident

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1. Introduction

A ruptured hepatocellular carcinoma (HCC) is an acute emergency and surgical intervention for this condition is often associated with high mortality. Transcatheter arterial embolization (TAE) is an alternative to surgery for arresting tumor bleeding and helps to stabilize patients. Tumor lysis syndrome (TLS) is an oncological emergency and a potentially lethal complication in patients treated for cancer. It most commonly occurs after chemotherapy for high-grade, rapidly proliferating treatment-sensitive tumors, such as Burkitt lymphoma and acute lymphocytic leukemia. Without prompt and proper management, the deterioration caused by TLS can result in death. Early identification of this critical condition reduces the risk of fatal complications.

We present here the case of a 70-year-old man with an HCC that was ruptured in a motor vehicle accident. The patient received TAE in the emergency department (ED). Extreme electrolyte abnormalities and anuria occurred on the 2nd day after the TAE procedure.

2. Case report

Emergency medical technicians brought a 70-year-old man to our ED after he had been injured in a motor vehicle accident while out walking in the morning. He reported severe abdominal pain and we noted distention of his abdomen in the primary survey during emergency management. His vital signs were as follows: body temperature, 36.1°C; pulse rate, 74 beats/minute; respiration rate, 20 breaths/minute; blood pressure, 150/80 mmHg; and Glasgow Coma Scale score, 15. A focused assessment with sonography for trauma revealed an accumulation of fluid over Morrison's pouch and splenic fossa. We immediately arranged contrast-enhanced abdominal and pelvic computed tomography scans, which revealed scattered heterogeneous mass lesions on the bilateral lobes of the liver and moderate intraperitoneal fluid collection with contrast medium extravasation over the splenic fossa (Fig. 1). The patient was a carrier for hepatitis B. The laboratory test results are shown in Table 1. His hemoglobin level was low and serum
Laboratory data.

<table>
<thead>
<tr>
<th>Variable</th>
<th>On admission</th>
<th>Day 1</th>
<th>Day 3</th>
<th>Reference range, adults</th>
</tr>
</thead>
<tbody>
<tr>
<td>White blood cell count /mm³</td>
<td>8000</td>
<td>16,700</td>
<td>4500–11,000</td>
<td></td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>8.4</td>
<td>9.1</td>
<td>14.4–16.0</td>
<td></td>
</tr>
<tr>
<td>Platelets (10⁹/µL)</td>
<td>342</td>
<td>225</td>
<td>150–400</td>
<td></td>
</tr>
<tr>
<td>International normalized ratio</td>
<td>1.02</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serum lactate dehydrogenase (U/L)</td>
<td>228,000</td>
<td>225–135</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serum total bilirubin (mg/dL)</td>
<td>1.2</td>
<td>0.3–1.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood urea nitrogen (mg/dL)</td>
<td>33</td>
<td>54</td>
<td>6–20</td>
<td></td>
</tr>
<tr>
<td>Blood creatinine (mg/dL)</td>
<td>2.0</td>
<td>3.5</td>
<td>0.7–1.2</td>
<td></td>
</tr>
<tr>
<td>Serum sodium (mmol/L)</td>
<td>138.6</td>
<td>145.1</td>
<td>136–145</td>
<td></td>
</tr>
<tr>
<td>Serum potassium (mmol/L)</td>
<td>4.2</td>
<td>7.4</td>
<td>3.5–5.1</td>
<td></td>
</tr>
<tr>
<td>Serum phosphate (mg/dL)</td>
<td>12.9</td>
<td>6.6</td>
<td>2.7–4.5</td>
<td></td>
</tr>
<tr>
<td>Serum uric acid (mg/dL)</td>
<td>15.5</td>
<td>5.2</td>
<td>3.4–7.0</td>
<td></td>
</tr>
<tr>
<td>α-Fetoprotein (ng/mL)</td>
<td>31.56</td>
<td>1.09–8.04</td>
<td></td>
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</tr>
</tbody>
</table>

potassium (4.2 mmol/L) was within the reference range. With respect to renal function, blood urea nitrogen (33 mg/dL), and blood creatinine (2.0 mg/dL) were moderately elevated. Based on the laboratory data and imaging findings, the clinical diagnosis was a ruptured HCC. We consulted a general surgeon for a second opinion, but surgery did not seem to be the best choice of treatment and we therefore performed TAE in the ED. Oliguria and conscious disturbance occurred 1 day after the TAE was completed, with acute kidney injury shown by elevated blood creatinine levels (3.5 mg/dL). Initially, we regarded these developments as side-effects of our contrast media, but the patient’s condition did not improve after fluid resuscitation. In addition, hyperkalemia (7.4 mmol/L), hyperphosphatemia (12.9 mg/dL), and hyperuricemia (15.5 mg/dL) were later noted. Suspecting TLS, we immediately administered urine alkalization and oral allopurinol combined with hemodialysis. The patient’s renal function stabilized after 2 days of aggressive treatment, during which time his electrolyte levels reached the reference range for plasma potassium (3.9 mmol/L), phosphate (6.6 mg/dL), and uric acid (5.2 mg/dL). However, acute pulmonary edema and dyspnea developed on the 4th day after admission. After we had explained the patient’s condition and the required endotracheal intubation to his family, they refused to allow the procedure and signed a do-not-resuscitate order. Eventually, the patient died from respiratory failure.

3. Discussion

HCC is the fifth most common type of cancer and the third leading cause of global cancer deaths. In Taiwan, malignant carcinoma and liver cancer were the first and second most common causes of death, respectively, in 2010. Encountering a ruptured hepatic tumor is common in emergency situations; in the Far East, the rupture rate is as high as 10% and the rupture rate in Hong Kong is approximately 9.7%19–12. If an HCC ruptures, emergency TAE is effective for hemostasis in hemodynamically unstable patients13.

In a retrospective review of 850 patients who underwent 2300 transcatheter arterial chemoembolization (TACE) or TAE procedures for hepatic tumors, complications were encountered in 4.4% of all patients14. The most common complication was iatrogenic dissection of a celiac artery and its branches; the second most common complication was intrahepatic biloma formation15. However, TLS was rarely considered as a complication of TAE, except in a number of cases where patients underwent TACE for HCC14,16,17.

TLS was first reported in 1929 in patients with chronic leukemia. TLS typically occurs infrequently in patients with solid tumors, but is commonly seen in patients with lung carcinoma, breast carcinoma16, and hematopoietic disorders (e.g., leukemia and high-grade lymphoma).15 TLS is characterized as a ruptured membrane of a cell with rich intracellular contents that spill out into the vascular space. The release of these components can cause severe metabolic alterations, including hyperkalemia, hyperphosphatemia, hyperuricemia, and hypocalcemia. Hyperkalemia and hyperphosphatemia directly result from rapid cell lysis. Nucleic acid purines, which are also released after cell breakdown, are ultimately metabolized to uric acid by hepatic xanthine oxidase. This conversion leads to hyperuricemia. Hypocalcemia results from acute hyperphosphatemia with the subsequent precipitation of calcium phosphate. Uric acid nephropathy is the major cause of acute kidney injury. Another cause of acute kidney injury is acute nephrocalcinosis from the precipitation of calcium phosphate crystals. When TLS develops, the standard treatment is to clear the high plasma levels of potassium, uric acid, and phosphorus, to correct acidosis, and to prevent acute renal failure. Furthermore, frequent monitoring of electrolytes, urine pH, and fluid intake and output are required during the acute phase of TLS.
In this case, the patient sustained a ruptured HCC unexpectedly after a motor vehicle accident; TAE was therefore suggested and performed immediately. Nevertheless, TLS developed. We undertook the necessary emergency management procedures and the electrolyte abnormalities were corrected. Unfortunately, the patient died from pulmonary edema and acute respiratory failure. However, we believe that the early recognition of TLS contributes to the management of this critical condition.

Albeit rare, TLS can occur after the TAE procedure for a ruptured HCC in adults. TLS can be treated effectively when detected early\(^3\). If this condition is encountered in the ED, close monitoring of electrolytes and urine output after TAE is recommended. When TLS develops, effective treatment must be undertaken immediately, including adequate hydration, urine alkalization, oral medication (allopurinol or urate oxidate\(^24\)), or hemodialysis.

References