A CASE REPORT ON ISCHEMIC HEPATITIS – SHOCK LIVER

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ABSTRACT

Ischemic hepatitis or shock liver is defined as an extensive hepatocellular necrosis associated with a decrease in hepatic perfusion caused by inadequate blood or oxygen supply. Ischemic hepatitis refers to diffuse hepatic injury secondary to acute hypoperfusion. Herewith we report 19 years old male with a fracture shaft of Right femur following road traffic accident, corrected by open reduction & internal fixation. Intraoperatively patient developed hypotension and then post-operative wound infection leading to ischemic hepatitis.

KEY WORDS: Ischemic hepatitis – Shock liver.
INTRODUCTION

Ischemic hepatitis is a condition which damages whole of the liver. The cause can be due to inadequate blood or oxygen supply\(^1\). Unlike, Hepatitis which is the inflammation of liver, in ischemic hepatitis, the cells are damaged and cell death (necrosis) occurs\(^2\). Though there is liver cell death, it is still termed Ischemic hepatitis, because as in viral and other types of hepatitis, aminotransferases leak from damaged liver cells. In ischemic hepatitis centrilobular necrosis occurs which has been documented in seven cases by autopsy\(^1\). Symptoms and clinical presentation tend to reflect the underlying state of shock, but can sometimes also include nausea, vomiting, anorexia, malaise and right upper quadrant pain\(^2\). Ischemic hepatitis has a characteristic pattern of liver function tests. Aspartate and alanine aminotransferases rise rapidly, peaking more than 10 times the base line value within 1-3 days with an early massive rise of Lactate dehydrogenase\(^3\). Bilirubin and Alkaline phosphatase are usually only mildly elevated\(^4\).

CASE REPORT

HISTORY & EXAMINATION

A 19 yr male was admitted in Sree Balaji Medical College and Hospital (Ortho department) with complaints of pain and swelling in the right thigh following a road traffic accident. X-ray showed fracture shaft of right femur. He was planned for Open Reduction and Internal Fixation with Plates and Screws. After clinical laboratory investigations work up the patient was taken up for surgery on the day of admission. Intraoperatively patient developed hypotension which was managed by the operating surgeon. Post operatively patient had pus discharge from the operated site and developed wound infection which was managed appropriately \(^5\).

INVESTIGATIONS

<table>
<thead>
<tr>
<th>INVESTIGATIONS</th>
<th>DAY1</th>
<th>DAY2</th>
<th>DAY3</th>
<th>DAY4</th>
<th>DAY7</th>
<th>DAY10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb (gm%)</td>
<td>12.6</td>
<td>11.5</td>
<td>8.2</td>
<td>6.6</td>
<td>7.8</td>
<td>9.5</td>
</tr>
<tr>
<td>SGOT (IU)</td>
<td>40</td>
<td>56</td>
<td>1654</td>
<td>2302</td>
<td>750</td>
<td>120</td>
</tr>
<tr>
<td>SGPT (IU)</td>
<td>35</td>
<td>45</td>
<td>1075</td>
<td>1740</td>
<td>684</td>
<td>98</td>
</tr>
<tr>
<td>ALP (IU)</td>
<td>120</td>
<td>130</td>
<td>221</td>
<td>1900</td>
<td>264</td>
<td>124</td>
</tr>
<tr>
<td>WBC</td>
<td>6000</td>
<td>7000</td>
<td>13700</td>
<td>10600</td>
<td>9000</td>
<td>8000</td>
</tr>
</tbody>
</table>

Hb- Hemoglobin, SGOT- serum glutamate oxaloacetate transaminase, SGPT-Serum glutamate pyruvate transaminase, ALP—Alkaline phosphatase, WBC—white blood cell.

First post operative day: 
LDH= 7379 IU 
GTT = 300 IU 
Sr.bilirubin (total) =7.2mg/dl, Indirect=2.9mg/dl. 
Direct= 4.3mg/dl 
PERIPHERAL SMEAR = Microcytic hypochromic anemia

DISCUSSION

There are many causes of Ischemic hepatitis like heart failure, respiratory failure, shock, & massive bleeding \(^6\). Any severe infection could also lead to increased demand for oxygen and thus contribute to Ischemic hepatitis \(^7\). In our case, following surgery and wound infection patient showed high levels of the liver transaminase enzymes with marked elevation of Lactate dehydrogenase –characteristic pattern of ischemic hepatitis \(^8\). Hepatitis was also confirmed by ultrasound –which showed hepatomegaly, pericholecystic edema. The enzymes leak into the blood stream from the damaged hepatocytes as a result the lab values are increased. With appropriate treatment by the clinician, the aminotransferases and lactate dehydrogenase level came down to upper baseline 300 U/L.
CONCLUSION

In our case, the patient was clinically diagnosed to have sepsis and there was also massive bleeding due to fracture. Both massive bleeding and sepsis secondary to wound infection could have contributed to ischemic hepatitis.

REFERENCES