Flood Syndrome: Rare Case Report of Ruptured Umbilical Hernia Expressing Ascitic Fluid in Liver Cirrhosis.

Mr Lava Krishna Kannappa, MBBS, MRCS, Dr Mohsin Hussein, MBBS, Dr May Hnin Lwin Ko, MBBS, Mr Yahya Salama, FRCS.

Department of Surgery
Kettering general Hospital.
United Kingdom.

Abstract
Flood syndrome, Eponym for rupture of umbilical hernia in cirrhotic patients is a rare complication. Flood syndrome occurs due to sudden increase in intra-abdominal pressure in chronic liver cirrhosis patients with as cites.

Case Presentation:
We present a 65 year old Caucasian man with chronic liver disease, who presented to the emergency department with sudden onset of abdominal pain and increase in swelling of the pre-existing umbilical hernia with associated erythematous changes over the skin of umbilical hernia resulting in discharge of yellow coloured liquid.

Conclusion:
Ruptured umbilical hernia with exudation of ascetic fluid (Flood syndrome) associated with skin ulceration or necrosis should be identified immediately. Patient should be resuscitated initially with albumin, I.V antibiotics and have surgical repair with post-operative treatment under gastroenterologist for ascites.

Key words:

Introduction:
Flood syndrome is a rare complication of long-standing ascites and end-stage liver disease. The syndrome is named for the sudden rush of ascetic fluid that accompanies spontaneous rupture of an umbilical hernia. We present a case of flood syndrome in 64 year old man with Liver cirrhosis presented with sudden increase in size of umbilical hernia with associated skin changes with resultant rupture. Prompt recognition of ulceration and necrosis with rupture needs surgical management.

Case Presentation:
64 year old male presented to emergency department with history of generalised abdominal pain and distension initially for the first 2 days with spontaneous leakage of around 5 pints of yellow coloured fluid from the umbilical hernia during the night. The umbilical hernia increased in size associated with sudden discolouration of skin 2 days prior to rupture. He was known to have umbilical hernia since the last one year before the rupture. Past history included alcoholic
liver disease (Chronic liver disease class 3) and umbilical hernia. He did not have any allergies and was on no medications. Patient smoked 8 to 10 cigarettes per day and had left alcohol few months ago. His family history included grandfather diagnosed of bowel cancer. The patient Lives with family, walks independently in home but uses mobility scooter for travelling outside.

On Examination, The patient presented with vitals of pulse of 85/mt, BP 105/55, Respiratory Rate - 14, SpO2 94-95 on air and Temperature of 37.3. Abdomen was Soft. There was exudation of yellow coloured liquid on increased abdominal pressure like sitting up on bed. The blood results showed Hb 128, WBC 8.4, Platelets 94, Na 123, K 3.9, CRP 29, Amylase 291, Bilirubin 107, ALT 31, and Albumin - 27. Child-Pugh score was calculated based on Total Bilirubin, Albumin, INR, As cites and Encephalopathy and was estimated at 10 and 55% mortality at one year. Patient has two ultrasound scans since 2015 and his blood results were as in Table 1. Ultrasound abdomen done on 20/05/2015 showed the liver is echo bright and has an irregular outline consistent with cirrhotic change. There is a moderate amount of as cites present. The portal vein is patent with a normal direction of flow. Second ultrasound abdomen done on 08/03/2016 showed the liver is bright in echo texture and coarse with patent portal vein demonstrates ante grade flow.

The patient was treated with Albumin to compensate for loss of ascetic fluid, I. Antibiotics after discussion with the gastroenterologist and was booked for urgent theatre. The patient underwent primary repair with Non-Absorbable sutures without a mesh and a Robinsons drain inserted into the abdomen. Skin was closed with absorbable sutures. Patient had an uneventful recovery and was transferred under gastroenterologists for further management. Patient had further treatment under the gastroenterologist for cirrhosis and did not develop complications at four weeks of follow up.

<table>
<thead>
<tr>
<th>Date</th>
<th>Sodium (mmol/L)</th>
<th>Creatinine (μmol/L)</th>
<th>Bilirubin (μmol/L)</th>
<th>ALT (U/L)</th>
<th>ALP (U/L)</th>
<th>Albumin (g/L)</th>
<th>Total protein (g/L)</th>
<th>INR</th>
<th>PT Test (Sec)</th>
<th>CRP</th>
</tr>
</thead>
<tbody>
<tr>
<td>09/05/2018</td>
<td>123</td>
<td>63</td>
<td>107</td>
<td>31</td>
<td>101</td>
<td>27</td>
<td>65</td>
<td>1.5</td>
<td>18.2</td>
<td>39</td>
</tr>
<tr>
<td>23/02/2018</td>
<td>129</td>
<td>46</td>
<td>30</td>
<td>18</td>
<td>143</td>
<td>36</td>
<td>74</td>
<td>----</td>
<td>15.1</td>
<td>----</td>
</tr>
<tr>
<td>15/02/2018</td>
<td>128</td>
<td>50</td>
<td>17</td>
<td>17</td>
<td>107</td>
<td>42</td>
<td>72</td>
<td>----</td>
<td>12.9</td>
<td>----</td>
</tr>
</tbody>
</table>
Table 1: Blood results

<p>| | | | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>23/10/2015</td>
<td>138</td>
<td>62</td>
<td>20</td>
<td>13</td>
<td>79</td>
<td>35</td>
<td>70</td>
<td>1.5</td>
</tr>
</tbody>
</table>

Table 1: Blood results

Discussion:

Ruptured umbilical hernia with exudation of ascetic fluid (Flood syndrome) in cirrhotic patients was first described in 1901. Umbilical hernia is frequently seen in patients with advanced liver disease. The risk for patients with liver cirrhosis and ascites developing an umbilical hernia is estimated at 20%. The factors that contribute to the development of an umbilical hernia in these patients have been attributed to increased intra-abdominal pressure from the ascites formation, weakness of the abdominal fascia and muscle wasting as a result of poor nutritional status, and the dilated umbilical vein enlarging the pre-existent supra-umbilical facial opening in patients with portal hypertension and also due to the transmission of portal pressure via the reanalyzed umbilical vein to the umbilicus. The presence of discolouration, ulceration or a rapid increase in size of the umbilical hernia signals impending rupture. Rupture may follow a sudden increase in intra-abdominal pressure with coughing, vomiting, straining, or rising from a seated position.

The optimal management of umbilical hernia in cirrhotic patient is not clear. There are always questions about whether conservative management of wait and watch policy or early surgical option is better. Conservative management can be complicated by incarceration or spontaneous rupture from necrosis of overlying skin, forcing an emergency repair in patients who are at greater risk of complications after such operations than after elective operation. Two studies found in the literature have evaluated the results of management of umbilical hernias in patients with concurrent ascites and liver cirrhosis in order to define optimal hernia treatment.
studies concluded that, Conservative management of umbilical hernias in patients with liver cirrhosis and as cites leads to a high rate of incarcerations with subsequent hernia repair in an emergency setting, whereas elective repair can be performed with less morbidity and is therefore advocated in particular the association of as cites with an umbilical hernia should prompt surgical correction. On the other hand, Small cohort study of eleven patients with ruptured umbilical hernia also concluded that successful primary repair of ruptured umbilical hernias in cirrhotic patients is feasible after meticulous optimisation and satisfactory control of the ascites.

References:


