Anaemia in Well Compensated Portal Hypertension Secondary to Alcoholism Induced Liver Cirrhosis

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Abstract

Background: This study was conducted at Index Medical College, Indore with an aim to study the Anaemia in will Compensated portal Hypertension Secondary to Alcoholism induced Liver Cirrhosis. Result: 2 patients were there who had vitamin B12/folic acid deficiency (macrocytic hypochromic) while rest all were iron deficiency thus microcytic hypochromic. Hemoglobin range= 2.4gm% - 12.8gm%, With mode=7.2gm%, With mean=7.128+5.3gm%.

Conclusion: All alcoholics who drinks moderate amount of alcohol (more than 3 drinks per day or more than 7 per week) develops iron deficiency anemia due to decrease absorption from stomach and git (alteration in git epithelium, chronic gastritis, increase git congestion and improper diet).Other being alcohol induced liver cirrohsis following decrease storage and hypersplenism due to portal hypertension.

Keywords: Anaemia, Hypertension, Alcoholism & Liver Cirrhosis.

Study Designed: Observational Study.

INTRODUCTION

Alcoholic liver cirrhosis is a term that includes the liver manifestation of alcohol overconsumption including fatty liver, alcoholic hepatitis and chronic hepatitis with liver fibrosis or cirrhosis.

Cirrhosis involves loss of lives cells and irreversible scarring of liver Leading to portal hypertension and splenomegaly. Is alcohol consumption affects iron absorption from stomach and duodenum?

METABOLISM OF ALCOHOL AND ASSOCIATED LIVER DAMAGE:-
Anemia Is Due To-- 1. Alcohol Induced Decrease Absorption of Iron from Git. 
2. Alcoholic Liver Cirrhosis Followed By Portal Hypertension Followed By Hypersplenism.

Splenic Enlargement History
Splenic enlargement can be caused by vascular engorgement or cellular infiltration. & Splenomegaly/hypersplenism seen in portosplenel vein(forming by joining of splenic vein and mesenteric vein) hypertension, leads to combination of neutropenia, thrombocytopenia and anemia[3].

The spleen has intrigued physicians and philosophers since ancient times. The spleen has been assigned mysterious powers but its association with destruction of blood cells was not elucidated until the turn of twentieth century.the exaggerated and unfounded worry about somatic complaints often reflected by the sense of pain in the spleen(left hypochondrium) led to the term hypochondriac. In 1899, Chauffard? Proposed that increased splenic activity causes hemolysis. This proposal provide the impetus for therapeutic spenectomy, which was performed first in 1910 by Sutherland and Burghard in a patient with splenic anemia (hemolytic anemia)[4].

The designation hypersplenism refers to exaggeration of the spleen’s normal filtration and phagocytic functions. The disorder can occur primarily by enlargement of spleen from vascular congestion, histiophagocytic hyperplasia, cellular infiltration, or secondarily by the inability of physically abnormal red cells, such as sickle cells or antibody coated cells, such as immune thrombocytopenia purpura. Hypersplenism usually is associated with triad of splenomegaly, blood cytopenia, and compensatory marrow hyperplasia.

Structural and Functional Organisation
The normal adult spleen weighs 135 +30 gram and has a blood flow of approximately 4 to 5 percent of cardiac out [put. The spleen’s principal structure is organised around an arborizing array of arterioles that branch and narrow until they terminate in either-
- The stroma of cords, forming the open circulation or
- The sinuses, the closed circulation of the spleen. The cordal elements include histiocytes, antigen presenting cells, pericytes, fibroblasts, and other cells necessary to maintain the discontinuous basal lamina that separates cords from lumen. Lymphatic tissue is inconspicuous and found in T cell rich zones in the periariteliolymphoid sheaths.

Blood cells must pass clusters of macrophages to enter the sinuoids. A principal function of the spleen is serving as a filter retaining defective blood cells and foreign particles in the bed of phagocytic cells. This function is accomplished by diverting part of the splenic blood supply into red pulp, where the blood slowly percolates through the nonendothelialized mesh studied with macrophages. The blood then reenters the circulation through narrow slits measuring 1 to 3 um, in the endothelium of the venous sinuses. The bulk of blood is rapidly channeled through no specialized vessels that link the arterioles with the venous sinuses. The blood is not filtered or modified in many animals, such as dogs and horses, the red pulp is a reservoir for red cells and splenic contraction provides there cell volume with a functionally important boost. In humuns, however, the splenic capsule is poorly contractile, and the spleen does not store red cells to any significant degree. On the other hand, a large fraction of circulating neutrophil pool is marginated in the spleen and about one third of platelets normally are sequestered at any time [5].

Pathophysiology
Filtration and elimination In this circumstances, cytopenias of varying severity may ensue. In the case of removal of antibody coated cells, the spleen itself produce antieccl antibodies, especially antplatelet antibodies. Thus the spleen contributes to immunizing the cells via its immune function and removing them through the Fc recognition function of large macrophage population.

Splenomegaly increases the proportion of blood channeled through the red pulp,causing
inappropriate hypersplenic sequestration of normal and abnormal blood cells. Spleen enlargement may result from expansion of the red pulp compartment in any red cell sequestration process.

The increased size of the filtering bed is more pronounced when the splenomegaly is caused by congestion (as in portal hypertension) rather than when it is caused by cellular infiltration, may be associated with severe hypersplenic sequestration of normal cells [6].

Splenomegaly increases the vascular surface area and thereby the margined neutrophil pool. However, sequestrated white cells and platelets survive in the spleen and may be available when increased demand requires neutrophils or platelets. Red cell, on the other hand, often is destroyed prematurely in the red pulp. Anemia in patient with splenomegaly has been considered the result of dilution of red cells in an expanded plasma volume. However expansion, as measured by radiolabelled albumin or fibronogen, results more from an increase in the splenic pool protein rather than an increase in circulating plasma volume.

The increased blood flow from an enlarged spleen expands the splenic and portal veins. A significant increase in portal venous pressure may occur when hepatic vessel compliance is deceased, as in cirrhosis or myelofibrosis. This process initiates a vicious cycle in which portal hypertension contributes to splenomegaly, which in turn increases portal pressure as a result of increased arterial flow in response to organ enlargement [7].

**MATERIALS AND METHODOLOGY**

Total cases included in study=150
- Patients with brief history of alcohol intake
- Ultrasound whole abdomen for establishing portal hypertension, liver cirrhosis and splenomegaly.

**Laboratory Features**
Characteristic triad of hypersplenism is -
- Splenomegaly
- Blood cytopenia
- Hyperplasia of corresponding lineage in the marrow.

Pancytopenia is a common finding in patients with hepatic cirrhosis and portal hypertension. However why some patient with cirrhosis develop marked cytopenia and others do not is not clear. About one third of patients with cirrhosis develop severe hypersplenism viz platelets less than 70000/ul and neutrophil less than 2000/ul.

**Clinical Features**
- Slight to moderate enlargement of the spleen usually does not produce local symptoms.
- Findings suggestive of impaired splenic function like presence of Howell-jolly bodies and erythrocyte pits.

**Exclusion Criteria in Study**
- Patients Suffering From Chronic Hepatitis B/C/D.
- Storage Disorder Leading To Cirrhosis Like Copper, Leulemias Etc
- Hemoglobinopathies Like Sherocytosis
- Hemolytic Disorders

**Inclusion Criteria**
30 - 60 YEARS ALCOHOLIC / COUNTRY LIQUOR (Homemade - called mahuva) >100 ML/DAY FOR MORE THAN 10 YEARS. Total cases included in study=150

**TABLE-1: HEMOGLOBIN**

<table>
<thead>
<tr>
<th>HEMOGLOBIN RANGE (GM%)</th>
<th>NUMBER OF PATIENTS</th>
<th>COMMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 to 4</td>
<td>16</td>
<td>Need urgent blood transfusion 5 to 6 units in 1 or 2 units per day.RCC/WHOLE BLOOD/PRP as &amp; when needed</td>
</tr>
<tr>
<td>4.1 to 6</td>
<td>39</td>
<td>Urgent blood RCC/PRP transfusion 3 to 4 units</td>
</tr>
<tr>
<td>6.1 to 8</td>
<td>45</td>
<td>1 to 2 units RCC (two were macrocytic)</td>
</tr>
<tr>
<td>8.1 to 10</td>
<td>29</td>
<td>BT not needed</td>
</tr>
<tr>
<td>10.1 to 12</td>
<td>15</td>
<td>BT not needed</td>
</tr>
<tr>
<td>12.1 to 14</td>
<td>6</td>
<td>BT not needed</td>
</tr>
</tbody>
</table>

Hemoglobin range= 2.4gm% -12.8gm%  
With mode= 7.2gm%  
With mean=7.128+5.3gm%  
2 patients were there who had vitamin B12/folic acid deficiency (macrocytic hypochromic) while rest all were iron deficiency thus microcytic hypochromic.
TABLE-2: MCV

<table>
<thead>
<tr>
<th>MCV (fl)</th>
<th>Number of patients</th>
<th>comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;40</td>
<td>1</td>
<td>Severe microcytic anemia</td>
</tr>
<tr>
<td>41 to 60</td>
<td>27</td>
<td>Severe microcytic anemia</td>
</tr>
<tr>
<td>61 to 80</td>
<td>86</td>
<td>Moderate microcytic anemia</td>
</tr>
<tr>
<td>81 to 100</td>
<td>34</td>
<td>Normal</td>
</tr>
<tr>
<td>101 to 120</td>
<td>1</td>
<td>Macrocytic anemia</td>
</tr>
<tr>
<td>121 to 140</td>
<td>2</td>
<td>Macrocytic anemia</td>
</tr>
</tbody>
</table>

Normal range=74 -96 fl  
Lowest =39.5 fl  
Highest =127.1 fl  
With mode = 71.4  
Mean=71.45 +46.9

TABLE-3: MCH

<table>
<thead>
<tr>
<th>MCH (pg)</th>
<th>NUMBER OF PATIENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 to 20</td>
<td>42</td>
</tr>
<tr>
<td>20 to 30</td>
<td>91</td>
</tr>
<tr>
<td>30 to 40</td>
<td>17</td>
</tr>
</tbody>
</table>

Normal range=27 -32 pg  
Lowest =11.30 pg  
Highest =36.5 pg  
Mode=22.1  
Mean=22.83+12

TABLE-4: MCHC

<table>
<thead>
<tr>
<th>MCHC (%)</th>
<th>NUMBER OF PATIENTS</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 to 30</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>31 to 40</td>
<td>108</td>
<td>NORMAL</td>
</tr>
<tr>
<td>41 and above</td>
<td>2</td>
<td>Hyperchromic</td>
</tr>
</tbody>
</table>

Normal range = 30-35%  
Lowest= 22.9%  
Highest =42.6%  
Mode =32.2  
Mean =32.046 +9.95

CONCLUSION

All alcoholics who drinks moderate amount of alcohol (more than 3 drinks per day or more than 7 per week)-develops iron deficiency anemia due to decrease absorption from stomach and gut (alteration in gut epithelium, chronic gastritis, increase gut congestion and improper diet). Other being alcohol induced liver cirrhosis following decrease storage and hypersplenism due to portal hypertension.

REFERENCES