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Medicine

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

Dr. S.M. Holkar[1], Dr. Bushra Khanam[2*], Dr. Satish Choudhary[3], Dr. Shubham Satsangi[4], Dr. Himanshu Singh[5], Dr. Rishu Bansal[6]

[1]Prof., [2]Assoc. Prof, [3,4,5,6]PG Resident, Department Of Medicine, Index Medical College Hospital & Research Centre, Indore, Madhya Pradesh, Pin- 452001, India

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***Corresponding author:** Dr. Bushra Khanam

Abstract **Original Research Article**

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

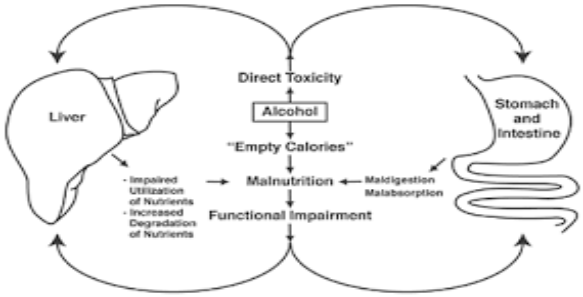
Alcoholic liver cirrhosis is a term that includes the liver menifestation of alcohol overconsumption including fatty liver, alcoholic hepatitis and chronic hepatitis with liver fibrosis or cirrhosis.

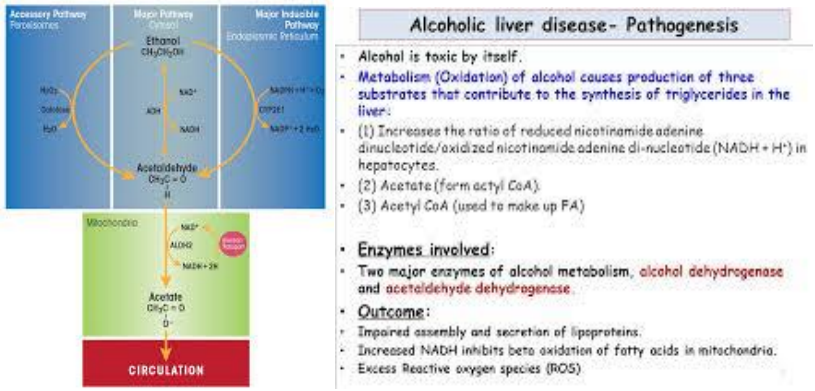
Cirrhosis involves loss of lives cells and irreversible scarring of liver Leading to portal hypertension and spenomegaly. Is alcohol ccnsumption affects iron absorption from stomach and duodenem? Probably yes since it may alters lining epithelium of GIT [1].

Alcoholism

Worldwide global consumption is 38.3% population.In India >30% population with 11% being indulged in heavy or binge drinking. Kerala at the top among all states.While we are considering the home made alcoholism called mahuva (in local language) which is much more readily available and highly consumed than above data in Indian families [2].

METABOLISM OF ALCOHOL AND ASSOCIATED LIVER DAMAGE:-





Anemia Is Due To-- 1. Alcohol Induced Decrease Absorption of Iron from Git.
2. Alcholic Liver Cirrohsis Follwed by Portal Hypertension Followed By Hyperspenism.

Splenic Enlargement History

Splenic enlargement can be caused by vascular engorgement or cellular infiltration. & Splenomegaly/hypersplenism seen in portosplenal vein(formed 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 compaints 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. Hyperspenism 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 sinusoids, 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 seperates cords from lumen. Lymphatic tissue is inconspicuous and found in T cell rich zones in the periarteriolar lymphoid sheaths.

Blood cells must pass clusters of macrophages to enter the sinusoids. 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 studed 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 reservior 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 platlets 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 anticell antibodies, especially antiplatelet 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 hyperslenic 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 therby the marginated neutrophil pool. However, sequestrated white cells and platlets survive in the spleen and may be available when increased demand requires neutrophils or platlets. Red cell, on the other hand, often is destroyed prematurly in the red pulp. Anemia in patient with splenomegaly has been considered the result of dilution 0f 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 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.
- Blood investigation including haemogram, blood group, liver function and peripheral smear of blood.
- History of dyspepsia AND upper GI Endoscopy in patient with severe epigastric pain

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 impared 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

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

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

MCV (fl)	Number of patients	comment
<40	1	Severe microcytic anemia
41 to 60	27	Severe microcytic anemia
61 to 80	86	Moderate microcytic anemia
81 to 100	34	Normal
101 to 120	1	Macrocytic anemia
121 to 140	2	Macrocytic anemia

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

MCH (pg)	NUMBER OF PATIENTS
10 to 20	42
20 to 30	91
30 to 40	17

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

TABLE-4: MCHC

MCHC (%)	NUMBER OF PATIENTS	Comment
20 to 30	40	Hypochromic
31 to 40	108	NORMAL
41 and above	2	Hyperchromic

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 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.

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