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A Study of Haematological Manifestation in Alcoholics in Tertiary Care M.G.M. Hospital, Warangal T.S

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ABSTRACT

Alcohol consumption is known for morbidity and mortality, being a serious health hazard of the people in the world. Multiple organs can be involved like hepato biliary system, cardiovascular system, central nervous system, hemopoietic system. Many times hematological changes are left undetected and untreated which could progress to cardiac failure. **Aims & Objectives**: 1. To study the hematological changes with respect to the quantity of alcohol consumption and duration of alcohol consumption. 2. To compare the hematological changes occurring in moderate and severe alcoholics. **Materials and Methods**: A prospective cross sectinal study conducted on 75 admitted patients, 49 adult moderate alcoholics and 26 severe alcoholics to study Hematological parameters in MGM Hospital. **Results**: Among these 75 patients 60 % (45) of the alcoholics had anemia 9.2 gms% among moderate alcoholics and 9 gm% in severe alcoholics. The total WBC mean of moderate alcoholics 10160.8±4448.65. and severe alcoholics 11763±5101.30. The mean MCV in moderate alcoholics 96.6±7.77fl and severe alcoholics 104.5±11.75fl. Peripheral blood smear showed all types of anemia. Normocytic normochromic anemia 40%. microcytic hypochromic anemia 28%, macrocytic anemia 26.66% patients. Abnormal bone marrow picture was seen in 58.6% patients. Among which megaloblastic picture 16%, Erythroid hyperplasia 13.3%, Vacuolated RBC 9.33%, Sideroblasts 8% of alcoholics. Other laboratory findings are elevations of serum γ-glutamyl transferase, alkaline phosphates, and bilirubin levels. Up to 70% of patients already have cirrhosis in both alcoholics.[8] **Conclusion**: Anemia was a predominant feature among chronic alcoholics. Hematological manifestations are reversible with cessation of alcohol. Early detection of anemia among alcoholics can prevent further complications of anemia leads failure.

Keywords: Alcohol, Megaloblasts, erythroid, sideroblastic.

INTRODUCTION

The amount of alcohol capable of producing diseases, depend on variety of factors including genetic predisposition malnutrition and concomitant viral infection of the liver. Although the exact date when alcohol was produced remains elusive, the discovery of late 'Stone Age Beer Jugs' has established the fact that intentionally fermented beverages existed as early as the Neolithic period.[3] Wine has appeared in Egyptian pictographs around 4000 B.C[3]. Since prehistoric times a variety of alcoholic beverages have been used in China. Around 2000 B.C[3] the art of wine making reached the Hellenic peninsula. Alcoholic drinks were an intricate part of most civilization ranging from China and India and Western Asia to Europe. According to national council of alcoholism and drug dependence alcoholism is a primary chronic disease with a genetic psychosocial and environmental factors

influencing its developmental manifestations. It is characterized by continuous or periodic impaired control over drinking over pre occupation with the drug alcohol and distortion in thinking most notably denial [1].

According National Council of Alcoholism the adults who drank in past year 64%,[1] and the number of alcohol induced deaths excluding accidents and homicides 21,081.[1] The office for national statistic revealed that number of deaths due to alcohol was 4144 in 1991[1] have increased to 8386 by 2006[1] and alcoholic liver disease deaths 12,548[1]. Hence alcohol consumption is known for morbidity and mortality, being a serious health hazard of the people all over the world. Multiple organs can be involved like Hepato-biliary system, cardiovascular system, Central nervous system, Hemopoitic system. Many times hematological changes are left undetected and untreated which could progress to cardiac failure. Early detection and treatment of hematological changes can prevent complications and reduce the mortality; these are the basis and the need for the study.

In India alcoholic beverages appeared during the Indus Valley Civilization, In Hindu Ayurvedic texts both the beneficial and detrimental effects of alcohol have been outlined. Distilled spirits originated around 800 B.C[3] in India and China; the distillation process emerged in Europe around the eleventh century. During the middle Ages, wine was the preferred beverage and the consumption of alcohol began to spread to all parts of the world. Today it is widely consumed by people all over the world[3].

Alcohol is classified according to the relation between the carbon atoms in it. The most commonly used primary alcohols are methanol and ethanol. As methanol was formerly known as ‘wood alcohol’. Methylated spirits or ‘surgical spirits’ is a form of ethanol. Ethanol has been consumed by humans in the form of alcoholic beverages since prehistoric times. About 20% of alcohol is absorbed by the stomach and 80 % by the small intestine leading to intoxication. The human body responds to alcohol in stages. Initially, a person consuming alcohols experiences a state of euphoria, become more self-confident and appear flushed. Later, he reaches a stage of excitement and is unable to react swiftly to natural situations. This is followed by a sense of stupor where he does not respond to stimuli. Excessive consumption of alcohol can lead to a coma or death. Most are aware that drugs are a major factor in our biggest social problems: violence, crime, poverty, AIDS, family disintegration “but many do not think of alcohol as a drug at all, only as a social beverage”. More than 100,000 U.S. deaths are caused by excessive alcohol consumption each year. Direct and indirect causes of death include drunk driving, cirrhosis of the liver, falls, cancer, and stroke.[9] At least once a year, the guidelines for low risk drinking are exceeded by an estimated 74% of male drinkers and 72% of female drinkers aged 21 and older.[9] 65% of youth surveyed said that they got the alcohol they drink from family and friends.[7,9] Across people of all ages, males are four times as likely as females to be heavy drinkers.[1] Up to 70% of patients already have cirrhosis in both alcoholics.[8]More than 18% of Americans experience alcohol abuse or alcohol dependence at some time in their lives.[6]Traffic crashes are the greatest single cause of death for persons aged 16–33. About 45% of these fatalities are in alcohol-related crashes.[4]

Chronic alcohol abuse can result in a spectrum of liver injury ranging from mild fatty infiltration to cirrhosis and hepatocellular carcinoma. [7,] Alcoholic liver disease also is a major health care problem, accounting for 40% of deaths from cirrhosis and more than 30% of cases of hepatocellular carcinoma in the United States.[1,2] Comparable statistics have been reported from Europe. Increase of alcohol consumption in past two decades in India 106 % .[4] Numerous studies have shown that alcoholic liver disease develops in women after a shorter duration of drinking and with a lower daily alcohol intake than in men.[4,5] Population-based surveys have shown that men drinking 40 to 80 g of alcohol daily and women drinking 20 to 40 g daily for 10 to 12 years are at a significant risk of developing liver disease. India is one of the largest producers of alcohol in the world and there has been a steady increase in its production over the last 15 years, according to new statistics[5]. India is a dominant producer of alcohol in South-East Asia, with 65% of the total share, and contributes to around 7%, more than two-thirds of the total beverage alcohol consumption in the region is in India, according to figures in the newly-compiled.[6]

Prevalence of alcoholism is greater among monozygotic than dizygotic twins, suggesting an

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inherited defect. Different rates of alcohol elimination may be related to genetic polymorphism of enzyme systems [10] Individuals with different alcohol dehydrogenize (ADH) iso-enzymes have different alcohol elimination rates. Cytotoxic T lymphocyte interaction may play a role in the genesis or perpetuation of alcoholic liver disease. Endotoxins are increased in the blood of alcoholics[11,12] . The endotoxin releases a battery of cytokines [11,12] Cytokines IL1, IL2 and TNF-a are released from non-parenchymal cells. TNF-a can depress P450 drug metabolism, induce cell surface expression of HLA antigens and cause hepato-toxicity. Microvesicular fat represents mitochondrial damage and more active lipid synthesis by the hepatocyte. Hepatic mitochondrial DNA deletion is associated .[13] Mallory bodies are seen by haematoxylin and eosin as purplish-red intra-cytoplasmic inclusions[13]. Alcohol abuse can increase iron levels in the body, iron absorption from the food in the gastrointestinal tract may be elevated in alcoholics. The increased iron levels can cause hemochromatosis, a condition characterized by the formation of iron deposits throughout the body (e.g., in the liver, pancreas, heart, joints, and gonads), and hemochromatosis have led to liver cirrhosis are at increased risk for liver cancer.

In many alcoholic patients, blood loss (results in anemia) and subsequent iron deficiency are caused by gastrointestinal bleeding. Iron deficiency in alcoholics often is difficult to diagnose, however, because it may be masked by symptoms of other nutritional deficiencies (e.g., folic acid deficiency) or by coexisting liver disease and other alcohol-related inflammatory conditions. Blood cell precursors require folic acid and other vitamins for their continued production. The resulting deficiency in RBC's, WBC's, and platelets (i.e., pancytopenia) has numerous adverse consequences for the patient. Megaloblasts occur frequently in the bone marrow of alcoholics, they are particularly common among alcoholics with symptoms of anemia, affecting up to one-third of these patients. Significantly higher use of alcohol has been recorded among tribal, rural and lower socio- economic urban sections. A substantial portion of family income is spent on alcohol, more so in rural households, which also tend to be poor and marginalized 32 per cent urban and 24 per cent rural[5, 6].

Table 1: Alcohol Content of Various Beverages[8]

Beverage	Alcohol Content	Serving Size	Amount of Alcohol	Daily Intake Needed to Exceed Threshold for Alcoholic Liver Disease[*]	
				Men	Women
Beer	5%	12oz	13.85 g	3-6 cans	1.5-3 cans
Wine	12%	4oz	10.7 g	4-8 glasses	2-4 glasses
Fortified wine	20%	4oz	17.8 g	2-4 glasses	1-2 glasses
Hard liquor	40%	1.5oz	13.4 g	3-6 drinks	1.5-3 drinks

OBJECTIVES

1. To study the hematological changes with respect to the quantity of alcohol consumption and duration of alcohol consumption.
2. To compare the hematological changes occurring in moderate and severe alcoholics.

Material and Methods

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Study Design: Prospective cross sectional study conducted on admitted 75 patients with both moderate and severe alcoholic patients in Tertiary care M.GM Hospital, Warangal Telangana State.

Samples Size: 75 patients among them 49 adult patients who are moderate alcoholics, 26 patients who are severe alcoholics and 67 Male patients and 8 female patients.

Inclusion Criteria: All patients who are moderate alcoholics that is who consume alcohol less than 80 to 90 mg proof alcohol that is about 11 drinks per day or 80 mg of proof alcohol three or four times a week. All patients who are severe alcoholics that is who consume more than 80 to 90 mg proof alcohol daily or more than 11 drinks per day. Patients attending and admitted Mahathma Gandhi Memorial Hospital (MGMH) Warangal attached to Kakatiya Medical College, Warangal.

Exclusion Criteria: All patients who are less than 18 years, Patients with other hepatic disorders and Patients receiving hepatotoxic drugs**.**

Duration of Study: one year. July 2019 to June 2020

Method of Analysis: The data collected will be analyzed for descriptive statistical methods like frequency, distribution and association using Microsoft Excel, Worksheet and SPSS for Windows.

The following information was collected from all patients admission and

1.Demography- name, age, gender, occupation, socio-economic status, date of admission and discharge.

2. Present History- Jaundice, pain abdomen, abdominal distention bilateral pedal edema, melena, hematemesis, altered sensorium. 3. Alcoholic history- amount of alcohol taken ,number of days taken in a week, 4. Group-moderate or severe

Examination-

a.General Physical Examination- icterus, clubbing, pedal edema pallor, lymphnodes. Signs of liver cell failure like palmar erytheionma, loss of axillary hair, dupytrens contracture, parotid swelling, breast atrophy in females or testicular atrophy in males.

b.Systemic Examination- Per abdomen, Central nervous system, Respiratory System and Cardiovascular System were examined in detail by – inspection, palpation, percussion and auscultation.

Investigations- Information was collected for following investigations-

Complete blood count-Hemoglobin, red blood count, packed cell count, mean corpuscular volume, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, total count, platelet count and peripheral blood smear.

Liver function test- Total bilirubin, direct bilurubin, serum glutamate aspartate transferase, serum glutamate oxalo transferase, albumin, alkaline phosphatase.

Coagulation profile-prothrombin time and INR, **Random blood sugar**, **Renal function test**- urea , creatine, **Bone marrow examination, Ultrasound abdomen, Upper gastrointestinal endoscopy**

Cardio echography Treatment History-drugs used, dosage, duration, blood transfusion, ventilator support, gastro endoscopic interventions. **Result-** patient cured or expired.

RESULTS AND ANALYSIS

Table 2: Distribution of Age in years

Age	Alcoholic	
	No.	%
21-30	13	17.4
31-40	24	32
41-50	27	36
51-60	11	14.6
Total	75	100

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Mean ± SD	45.34 ±11.20

The table shows the maximum (68%) alcoholics are from 31 years to 50 years age group . distribution of presenting complaints shows the major complaint are 60% (45/75) from alcoholics, in addition jaundice 40%, pedal edema 28%, pain abdomen 25.%, hematemesis 20%, breathlessness 21%, altered sensorium 21% and 10.6% melena.

Table 3: Per Abdomen Findings among study alcoholic group

P/A Findings	**Number(n = 75)**	**%**
Abnormal	55	73.33
Hepatomegaly	40	53.33
Splenomegaly	5	6.66
Ascites	10	13.33
CNS Abnormal	**Number (n = 75)**	**%**
Pre Coma	5	6.66
Altered Sensorium	15	20

The table shows the maximum (73.3%) alcoholics are presenting with abnormal abdominal findings. Among them 53% hepatomegaly,13% ascitis and 6% Spleenomegaly. Under CNS abnormality symptoms 6% pre coma status and 20% altered sensorium.

Table 4 : Comparison of complete blood count in Moderate and severe alcoholics

Complete Blood Count	**Alcoholics**		**p-Value**
	Moderate	**Severe**	
Hemoglobin	9.2 ± 2.96	9.0 ± 2.99	0.776
WBC Total Count	10160.8 ± 4448.65	11763.9 ± 5101.30	0.1471
RBC	2.5 ± 0.83	1.9 ± 0.61	0.0002***
MCV	96.6 ± 7.77	104.5 ± 11.75	0.0021***
MCH	29.00 ± 5.36	27.5 ± 4.98	0.2189
MCHC	29.8 ± 4.37	24.4 ± 4.30	0.00004***
PCV	27.0 ± 7.40	28.8 ± 4.90	0.2147
Platelates Count (Lakhs)	1.9 ± 0.69	1.40 ± 0.58	0.002***

The Table shows:- 1. Hemoglobin slight reduction in both moderate and severe alcoholic. 2.Mean WBC total count is elevated.3. Mean RBC count is markedly reduced. P< 0.0002 * S 4. The Mean Corpuscular Value elevated in severe alcoholics as compare with moderate alcoholics 5. The Mean Corpuscular hemoglobin reduced in severe alcoholics as compare with moderate alcoholics 6. The Mean Corpuscular hemoglobin Concentration reduced in severe alcoholics as compare with moderate alcoholics.P<0.00004 * S 7. The Packed cell volume elevated in severe alcoholics as compare

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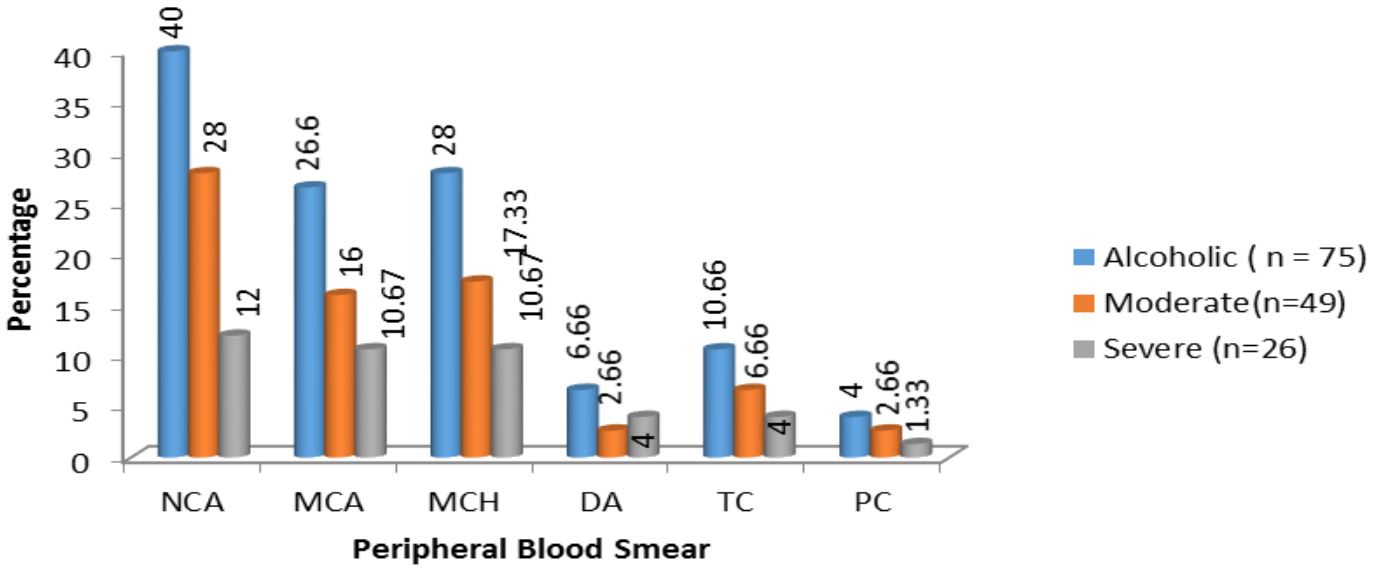
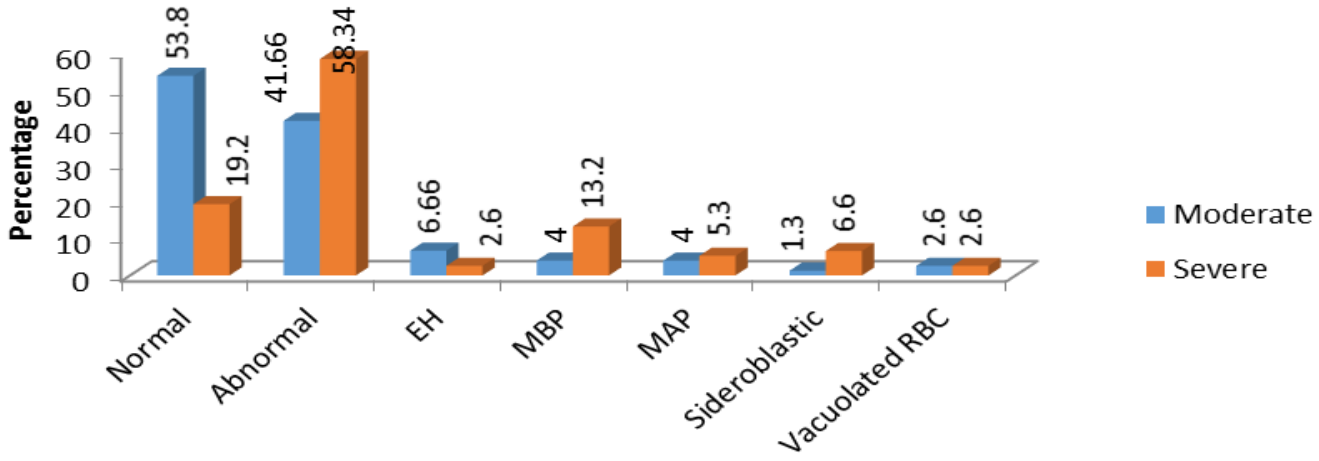
with moderate alcoholics 8. The platelet count reduced in severe alcoholics as compare with moderate alcoholics. P< 0.002 * S Graph 1 : Peripheral Blood Smear in Moderate and Severe Alcoholic Cases.



microcytic hypochromic anemia picture is more (28%) as compared with Macrocytic anemia presenting picture is 26% in study group 2. Dimorphic anemia 6.6%. 3.Thrombocytopenia 10.6% 4. Pancytopenia 4%



bone marrow shows abnormal picture 41.% in moderate alcoholics and 59% in severe alcoholics. 1. The Erythroid hyperplasia among the moderate alcoholics 6.6% and 2.6 in severe alcoholics 2. Megaloblastic anaemia presents 13.2% in severe alcoholics as compared 4% in moderate alcoholics. 3. Myeloidaplasia picture, 5.3% in severe alcoholics and 4% in moderate alcoholics. 4. Sideroblastic picture more (6.6%) in severe alcoholics and 1.3% in moderate alcoholics. 5. Vccuolated RBC seen 5.3% in moderate alcoholics and 4% in severe alcoholics.

The abnormal LFT have been found in 30/75 (40%). The total bilirubin raised in both moderate and severe alcoholics. P<0.000.

1. Direct bilirubin raised more in severe alcoholics as compared with moderate alcoholics. P <0.003.

2. The proteins levels are very low in severe alcoholics and albumin levels are very low, as compared with moderate alcoholics. 3. SGOT & SGPT is more elevated in severe alcoholics.

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ALP is significantly raised in severe alcoholics. P< 0.0006 *S

DISCUSSION

Demographic Details:

In our present study 75 alcoholic patients and their age group ranged from 20 years to 60 years, mean age being 37.3. In a similar study done by T. Oduola et al[15] in Nigeria out of 200 patients age of patients ranged from 20 years to 57 years mean age being 36.04±11.28 years.

In our study the maximum prevalence's of alcoholics were in the age group 31-50 years (51/75) 68%. There was less prevalence of alcoholism below 20years and above 60 years. This could be probably because middle age group people belonging to lower socioeconomic status . The maximum number of hematological abnormalities ie anemia was seen in age group of 31 to 60 years. In a similar study done by D. Chalmers et al[16] in 1981 the mean age group was 59.9 years.

Gender Distribution: In present study among 75 alcoholics 80% (67/75) were males and 20% (8/75) were females. This shows an increasing trend of alcohol consumption in women also. In a similar study conducted by D. Charmers et al[16] in 1981 from Harrow showed a study of 219 out of which 146 (66.66%) were men 73 (33.33) were females. In a similar study conducted by Hislop et al[17] 1983 in England male to female ratio of 2.9:1 was found. In a similar study done by T. Oduola et al[15], in Ile at Nigeria out of 200 (100%) all were men. In a study by Ray R et al 1988 from NIMHANS Bangalore all were 100% males.

Socio-Economic Status: In the present study most of the patients 72% (54/75) belonged to lower socio-economic class and 28.% (21/75) were middle socio economic class. There is increased number of alcoholics in lower socioeconomic group (labor class) are visiting our hospitals. Being poor they tend to consume low quality drink like arrack. In a similar survey done by Wilson et al in 1980 showed a high incidence in low socio-economic group.

Duration of Alcohol Intake: In our present study there were patients who consumed alcohol more than 10 years duration 62.6% (47/75) and 25% (19/75) were less than 10 years duration. In the similar study conducted by D. Chalmers et al[16] 219/373 were severe alcoholics for more than 10 years. Another similar study conducted by T.Oduola et al[15] 200 patients were studied among which 50% were moderate alcoholics consuming for less than 10 years. 50% were severe alcoholic who consumed for more than 10 years.

Haematological Manifestations Complete blood count: In the present study the mean hemoglobin was 9.2 gms% among moderate alcoholics and 8.5gm% among severe alcoholics' In study conducted by T. Oduala et al16 the hemoglobin (g/dl) was 14.5 ± 1.2 among moderate alcoholics and 14.8 ± 1.2 among severe alcoholics.

In our study the total count of white blood cells were mean of 10160.84±4448.65 in moderate alcoholics and 11763.9±5101.30 in severe alcoholics. In the similar study of T.Oduola et al[15] WCC (mm3) was 4 4516.7 ± 2825.6 among moderate alcoholics and 4733.3 ± 1400.6 among severe alcoholics.

In the present study MCV in moderate alcoholics was 96.6±7.77 fl. in severe alcoholics it was 104.5±11.75 The highest was 110.6fl. In the same study by T. Oduola et al[15] the MCV (µm3) was 84.9 ± 9.1 in moderate alcoholics and 89.7 ± 9.7 among severe alcoholics.

In our study he MCH showed 29.00±5.32 among moderate alcoholics and 27.5±4.98 among severe alcoholics. In the similar study by T. Oduola et al[15] MCH (pg/L) among moderate alcoholics was 28.4 ± 4.1 among severe alcoholics it was 28.9 ± 4.3.

In present study the MCHC was 29.8±4.37 among moderate alcoholics and 24.4±4.30 among severe alcoholics. In the same study of T. Oduola et al[15] MCHC 30.8 ± 1.8 among moderate alcoholics and 32.7 ± 0.9 among severe alcoholics.

In the present study the PCV among moderate alcoholics was 27.0±7.40 and among severe alcoholics it was 28.8±4.90. In the same study conducted by T. Oduola et al[15] PCV (%) 44.2 ± 3.7 among moderate alcoholics and 45.3 ± 3.8 among severe alcoholics.

Platelet count showed a mean of 190000±0.69 in moderate alcoholics, in severe alcoholics the plate let count was a mean of 140000 +_0.58. The lowest platelet count was 40,000. In the similar study by T.

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Oduola et al[15] the platelet count was 211733.3 ± 49906.8 among moderate alcoholics and 217966.8 ± 41736.0 among severe alcoholics. The platelet counts were above 2 lakhs normal in all groups

In our study 10% of study group had thrombocytopenia out of which mean was 40,000cells/cumm. This can be the cause of transient intravascular hemolysis associated with alcoholic liver disease.18 In other similar study by Latvala Jaana and Parkkila[14,15] thrombocytopenia was found in 41% of alcoholics. In a similar study done by Shinji Nakao, M.D.19, Mine Harada from japan in 1990 showed thrombocytopenia 5% in severe alcoholics. However haematological manifestations in T. Oduola et al[15] study showed no significant changes in occasional and moderate drinkers.

Peripheral Blood Smear: In our study moderate drinkers showed normocytic normochromic anemia in peripheral blood smear. Heavy drinkers showed 30.66% of other types anemia in the peripheral blood smear. In the similar study by T. Oduola et al[15] in severe drinkers they showed predominantly a macrocytic blood picture in peripheral blood smear.

In addition our study other types of anemia like 28% showed microcytic hypochromic, 40% normocytic normocromic anemia, 6.6% dimorphic anemia, 10.66% thrombocytopenia and 4% pancytopenia. In the similar study conducted by T. Oduola et al[15] the platelet counts were above 2 lakhs normal in all groups. In our study 10% of our study group had thrombocytopenia out of which mean was 40,000cells/cumm. In a study conducted by Latvala jaana, Parkkila[14] 144 subjects were studied. The incidence of anemia was 51% in the alcohol abusers. , ($p < 0.05$). A diverse pattern of hematological effects was observed in the alcohol abusers. In present study 60% of patients showed anemia among alcoholics .Increased mean cell volume of erythrocytes macrocytosis was seen in 60% of alcoholics $p < 0.006$. In our study 26.6% of alcoholics showed macrocytes in peripheral blood smear. In a similar study done by H. Koivisto, J. Hietala, P. Anttila[18] out of 105 alcoholics 60% showed macrocytecs in blood smear.

Bone Marrow Aspiration Study: In our study abnormalities of bone marrow shows moderate alcoholics 23 and 21 severe alcoholics. Among them Erythroid hyperplasia 10.6% in moderate alcoholics and 2.6% in severe alcoholics. In addition our study 6.6% of Megaloblastic picture seen in moderate alcoholics, 9.3%in severe alcoholics and Sideroblastic picture 1.3% in moderate alcoholics and 6.6% in severe alcoholics and vacculation RBC 5.3% and 4% in severe alcoholics. In the similar study conducted by Latvala Jaana and Parkkila[14] bone marrow study revealed vacuolization of pro-normoblasts in 24% of the alcoholic patients. Megakaryocytes in the cell periphery were also vacuolated in 20% of the alcohol abusers. The bone marrow abnormalities were related to the duration of alcohol intake.

In our study out of 44 patients who had abnormal bone marrow 30.6% were moderate alcoholics 28%were severe alcoholics. In a similar study done by J. Latvala,. Melkko[14], and O. Niemelä out of 138 consecutive adult patients undergoing bone marrow aspiration due to macrocytosis 49% were severe alcoholics and 20% were moderate alcoholics. Bone marrow aspirates from 12 alcoholic patients showed vacuolization of pro-normoblasts and the presence of ring sideroblasts were noted in 8 cases. In a similar study done by Shinji Nakao et al.[19], Mine Harada from japan in 1990 showed thrombocytopenia 5% in severe alcoholics.

In our present study liver function tests shows that abnormal LFT 30/75 (40%) of alcoholics. The total bilirubin raised in both moderate and severe alcoholics. P<0.000.*S Direct bilirubin raised more in severe alcoholics as compared with moderate alcoholics. P <0.003.*(S) The proteins levels are very low in severe alcoholics and albumin levels are very low, as compared with moderate alcoholics. SGOT & SGPT is more elevated in severe alcoholics. ALP is significantly raised in severe alcoholics. P< 0.0006 *(S). In the similar study done by "Niemela O in Biomarkers in alcoholism shows the laboratory findings elevations of serum γ glut amyl transferees, alkaline phosphatase and bilirubin levels. Up to 70% of patients with moderate to severe alcoholic hepatitis already have cirrhosis identifiable on biopsy examination at the time of diagnosis.[20] Another similar study by Menon KV, Gores GJ, Shah VH of all chronic heavy drinkers, only 15–20% develop hepatitis or cirrhosis, which can occur concomitantly or in succession .[21]

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Presenting Complaints In present study 60% (45/75) of patients presented with distension of abdomen. Next frequent presentation was with jaundice 40% (30/75), bilateral pedal edema 28% (21/75), Pain abdomen 25.3% (19/75), hematemesis 20% (15/75), melena 10.7% (8/75) breathlessness 10.7% (8/75) altered sensorium 21.3% (16/75). In a similar study done by D. Chalmers et al[16] gastrointestinal symptoms were predominant. About 60% of patients presented with duodenal ulcer and dyspepsia. 20% of patients with hematemesis. Jaundice in 20%. Altered sensorium in 10% of patients.

CONCLUSION; There also abnormality in liver function tests like, elevated serum bilirubin, lower levels of Albumin, SGOT, SGPT and also elevated ALP in chronic alcoholics. Hematological manifestations are reversible with cessation of alcohol, Early detection of anemia among alcoholics can prevent further complications of anemia like failure and reduce the mortality in lower socioeconomic group of people.

Ethical committee: Approved by ethical committee of MGMH, KMC Warangal.

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