



Available at <https://www.iasj.net/iasj>

Iraqi Academic Scientific Journals

Journal homepage:
<https://journals.uokerbala.edu.iq/index.php/UOKJ>



Research Article

Effect of Alcohol on Some Blood and Biochemical Parameters in the Babylon Governorate

Jenan M. Al-Kawaz

Department of Biology, College of Science, University of Babylon, Iraq.

Article Info

Article history:

Received 8-06-2022

Received in revised form 7-08-2022

Accepted 26-09-2022

Available online 06-06-2022

Keywords: Alcohol ,
Blood parameters,
Biochemical enzymes.

Abstract

The study was conducted between a February to July of 2021 on 110 subjects (80 alcohol consumers and 30 non-alcohol consumers as control group) in the Babylon Province. The ages of the subjects ranged between 20 and 45 years . Blood samples have been tested to investigate the following parameters: Effect of alcohol consumption on complete blood picture (CBC), Erythrocyte sedimentation rate (ESR), serum iron, TIBC and the activity of the liver enzymes (AST and ALT). Results of this study showed significant decrease ($P < 0.05$) in Hb , RBCs , WBCs , ESR , PCV, MCV, MCH and MCHC , as compared with results of the control group. On the other hand, significant rise in the activity of AST and ALT enzymes was seen. Effect of alcohol on serum iron and TIBC of blood serum indicated that there was a significant decrease ($P < 0.05$) in serum iron with a significant increase ($P < 0.05$) in TIBC of blood serum relative to control group. Therefore, this study confirm that alcohol consumption can lead to anemia especially Iron-deficiency anemia and cause damage to the liver tissues.

Corresponding Author E-mail : sci.jinan.mhadi@uobabylon.edu.iq

Peer review under responsibility of Iraqi Academic Scientific Journal and University of Kerbala.

Introduction:

Alcohol has numerous adverse effects on the various types of blood cells and their functions. For example, heavy alcohol consumption can cause generalized suppression of blood cell production and the production of structurally abnormal blood cell precursors that cannot mature into functional cells. Alcoholics frequently have defective red blood cells that are destroyed prematurely, possibly resulting in anemia. Alcohol also interferes with the production and function of white blood cells, especially those that defend the body against invading bacteria. Consequently, alcoholics frequently suffer from bacterial infections. Finally, alcohol adversely affects the platelets and other components of the blood-clotting system. Heavy alcohol consumption thus may increase the drinkers risk of suffering a stroke (Ballard, 1997).

People who abuse alcohol are at risk for numerous alcohol-related medical complications, including those affecting the blood (i.e., the blood cells as well as proteins present in the blood plasma) and the bone marrow, where the blood cells are produced (Deitrich *et al.*, 1996). Alcohols adverse effects on the blood building, or hematopoietic, system are both direct and indirect. The direct consequences of excessive alcohol consumption include toxic effects on the bone marrow, the blood cell precursors, and mature red blood cells (RBC's), white blood cells (WBC's), and platelets. Alcohol's indirect effects include nutritional deficiencies that impair the production and function of various blood cells (Holmgren & Jones, 2010).

These direct and indirect effects of alcohol can result in serious medical problems for the drinker. For example, anemia resulting from diminished RBC production and impaired RBC metabolism and function can cause fatigue, shortness of breath, light

headedness, and even reduced mental capacity and abnormal heart beats. A decrease in the number and function of WBC's increases the drinker's risk of serious infection, and impaired platelet production and function interfere with blood clotting, leading to symptoms ranging from a simple nosebleed to bleeding in the brain (i.e., hemorrhagic stroke). Finally, alcohol induced abnormalities in the plasma proteins that are required for blood clotting can lead to the formation of blood clots (i.e., thrombosis) (Lee *et al.*, 2015). Alcohol (ethanol) is the psychoactive substance most often encountered in forensic toxicology casework, because excessive drinking and drunkenness are tightly linked to various types of criminal activity. First and foremost drunken-driving offenses, sexual assaults, domestic violence, bodily harm, and aggressive behavior in general (Scott-Ham & Burton, 2005; Holmgren & Jones, 2010).

As alcohol consumption is increased all over the world and according to the WHO was stated that about 3.3 million people die annually from alcohol consumption, alcohol effect on different organs should be studied regularly (WHO, 2018). Although there are no profound recent studies regarding the alcohol effect on hematology of human and particularly on red blood corpuscles count (RBCs), in addition to the total white blood cells count (WBCs), erythrocyte sedimentation rate (ESR) and the values of packed cell volume (PCV), the present study is focused on the possible changes of these parameters and blood criteria such as mean of corpuscle volume (MCV), mean of corpuscles haemoglobin (MCH), in addition to mean concentration of the corpuscles haemoglobin (MCHC), in alcohol consumption and compared it with control group, also this study is focused on the activity of liver enzymes AST and ALT in blood serum as well as serum iron and TIBC of blood serum (Scott-Ham & Burton, 2005).

Materials & Methods :

Patients and control groups:

This study was done on a group of prisoners in the Babylon governorate who were alcohol users, for period from February to July 2021 .This study was conducted on 110 adult males categorized into two groups : drinkers and non-drinkers.80 persons were consume alcohol (drinkers) and 30 persons were non-alcoholics(non-drinkers)taken as control ,their ages ranged between 20 and 45 years.Laboratory tests were conducted in detection the effect of alcohol on some blood parameters in health laboratories at AL- Hilla.

Blood Samples:

Venous blood was collected from each sample of the study .The blood was placed after being drawn into tubes containing an anticoagulant for the purpose of measuring physiological blood parameters. As for the other part of the blood, it was placed in labeled gel tubes for serum separation,the labeled tubes left to coagulate for about 1 hour at the room temperature and then the serum was separated by using the centrifuge 3000 cycle / 15 minutes , then the serum was transferred by pipette from the gel tube to labeled Eppendorf tubes and kept in the refrigerator until used .The serum was used to measure liver enzymes (ALT,AST), serum iron and (TIBC) .

Count of physiological blood parameters

- Estimation of leucocytes count

A blood cell counter and dilution solution (Turk's fluid) was used to calculate the total white blood cell count (Brown , 1976).

2-Mean Corpuscle Volume (MCV)

This amount represents the volume of red blood corpuscle measured by units of femtoliter (fl) and was calculated from the following equation (Shirlyn,2004):

$$MCV(fl)= \frac{Packed\ cell\ volume\ (\%)}{Red\ cell\ count\ per\ liter} \times 10$$

3- Mean Cell Haemoglobin Concentration Measurment(MCHC)

It was calculated from the following equation (Shirlyn,2004):

- Estimation of Red Blood corpuscles

1- Total red blood corpuscles

The hematology and Hynes fluid solution was used as a dilution solution for the red blood corpuscles count (Hall & Malia ,1984) .

2-Haemoglobin estimation

Haemoglobin meter and Drabkins solution were used as a dilution solution to estimate the concentration of hemoglobin in the blood sample (Hall & Malia , 1984).

3- Packed Cell Volume Measurement

Capillary tubes, microcentrifuge, and haematocrite reader were used to determine the percentage of packed cell volume (Brown , 1976) .

4 - Erythrocyte sedimentation rate measurement

The Westergreens method was used to estimate the erythrocyte sedimentation rate (Brown ,1976)

- Measurement of Red Blood Corpuscles indices

Red blood cell indices were calculated by using the values obtained for packed cell volume, hemoglobin concentration, and red blood corpuscles count (Brown ,1976)

1-Mean Corpuscular Haemoglobin (MCH)

This amount represents the rate (weight) of hemoglobin in the red corpuscle of the studied sample and was measured by units of pictograms (pg) and was calculated from the following equation (Shirlyn ,2004):

$$MCH(pg) = \frac{Haemoglobin\ in\ gm/dl}{Red\ Cell\ Count\ per\ liter} \times 10$$

MCHC

$$= (g/dl) \frac{Haemoglobin\ concentration\ (gm/dl)}{Volume\ of\ red\ cells\ (\%)} \times 100$$

-Measurement of liver enzyme activity (AST and ALT) in blood serum

Activity of the enzymes carrying the group of amine Alanine and Aspartate aminotransaminases (ALT,AST) were estimated in serum. This method is based on an estimate of the amount of pyruvate and

oxaloacetate released by their interaction with Dianitrophenyl hydrazine (Reitman & Frankel, 1957).

- Measurement of serum iron

Three test tubes were taken, classified into a whistle tube, a test tube and a standard solution tube, and put 1 ml of the first solution R1 in each of the three tubes, then put 200 µL of distilled water into the whistle tube, and 200 µL of the second solution R2 to the standard solution tube, and 200 µL of serum sample to the test tube, the tubes were mixed well and left for 3 minutes at room temperature. Then prepared three other plastic tubes and also classified into a whistle tube, test tube and tube of the standard solution and put in them 1 ml of the working reagent solution which consisting of 15 ml of R1 and 1 ml of R2 and the rest of the volumes as previously, then left the tubes for 5 minutes at room temperature after which measured optical density for each of the test solution and standard solution after calibration of device with whistle solution on a wave length 600 nanometer, and concentration of serum iron was calculated by applying the following equation:

$$\text{Serum Iron } (\mu\text{mol/L}) = \frac{(A_2 - A_1)_{\text{assay}}}{(A_2 - A_1)_{\text{standard}}} \times \text{standard concentration}$$

Results:

- Effect of alcohol on physiological blood parameters

A total of 110 subjects were recruited for this study and were categorized into two with thirty in non-alcoholic subjects group (control) and eighty in alcoholic subjects group. The effect of drinking patterns on physiological parameters is shown in table (1). The values obtained for Hb in drinkers was $(7.9129 \pm 0.14424 \text{ g/dl})$ and in non-

- Measurement of mount of total iron binding capacity

A 1 ml of the serum was placed in a plastic test tube and added to it 2 ml of iron solution R1, mixed well and then left for 10 minutes at room temperature and then added to 150 mg of precipitator which is the capsule R2 after it was mixed well by shaking way and left for 30 minutes at room temperature, Then it was mixed well by shaking method and left for 30 minutes at room temperature, then put in the centrifuge for 10 minutes, then the amount of iron in the floating part of the tube was calculated according to the previously mentioned serum iron measurement steps and the total binding capacity of iron was calculated by applying the following equation:

$$\text{TIBC} (\mu\text{mol} / \text{L}) = \text{Iron concentration measured in supernatant} \times 3$$

-Statistical analysis

The results of the study were analyzed statistically using the statistical program (Genstat) version (1995), and this analysis included the calculation (Mean $\pm$ S.E.).

drinkers was $(15.24 \pm 0.11613 \text{ g/dl})$. The count of WBC in drinkers was (6304 ± 2002) million / mm³ and in non - drinkers was (8340 ± 1826) million/mm³. The value obtained for PCV in drinkers was $(26.1290 \pm 0.46465)\%$ and in non - drinkers was $(50.3076 \pm 0.02964)\%$. The values obtained for ESR in drinkers was $(1.457 \pm 9.12 \text{ mm} / \text{h.})$ and in non-drinkers was $(2.223 \pm 5.32 \text{ mm} / \text{h.})$ As well as the count of RBC in drinkers was (4.2612 ± 0.23098) million /mm³ and in non - drinkers was (5.246 ± 0.05626) .

Table (1): Comparison of hematological parameters between non-alcoholic and alcoholic subjects.

parameters	Non-alcoholic subjects N=30	Alcoholic subjects N=80
	Mean±SE	
Hb (g /dl)	15.24±0.11613	* 7.9129±0.14424
WBC count (cells /mm ³)	8340±1826	* 6304±2002
ESR (mm/ h.)	2.223±5.32	*1.457± 9.12
PCV (L/L)%	50.3076±0.02964	*26.1290±0.46465
RBC(cells/mm ³)	5.246±0.05626	*4.2612±0.23098

*The significant values are between Non-alcoholic and alcoholic subjects at(p<0.05).HB=hemoglobin.WBC=white blood cells , ESR=erythrocyte sediment rate,RBC=Red blood corpuscles.

- Effect of alcohol on red blood corpuscles indices

The effect of drinking patterns on red blood corpuscles was shown in table (2), the values obtained for MCV in drinkers was (68.268±2.71298 fl) and in non drinkers

was(95.506±0.93847 fl).The value obtained for MCH in drinkers was (20.348±1.09201 pg) and in non drinkers was (28.96±0.27273 pg). Also the values obtained for MCHC in drinkers was (30.26±1.46) % and in non drinkers was (36.26±1.14)%.

Table (2): Comparison of red blood corpuscles indices between non-alcoholic and alcoholic subjects.

Parameters	Non-alcoholic subjects n=30	Alcoholic subjects n=80
	Mean ± SE	
MCV(fl)	95.506±0.93847	*68.268±2.71298
MCH(Pg)	28.96±0.27273	*20.348±1.09201
MCHC (%)	36.26±1.14	*30.26±1.46

*The significant values are between Non-alcoholic and alcoholic subjects at(p<0.05).MCH=Mean of corpuscles haemoglobin ,MCHC=Mean of concentration of the corpuscles haemoglobin ,MCV=Mean of corpuscle volume.

- Effect of alcohol on biochemical blood parameters

The effect of drinking alcohol on biochemical parameters is shown in Table (3).The values obtained for ALT and AST were significantly higher (p<0.05) in alcoholic subjects than the values obtained for these parameters in non-alcoholic

subjects.While the values obtained for serum iron were significantly lower (p<0.05) in alcoholic subjects than values obtained for these values in non-alcoholic subjects.And the values obtained for TIBC were significantly higher (p<0.05) in alcoholic subjects than values obtained for these values non-alcoholic subjects.

Table (3): Comparison of biochemical parameters between non-alcoholic and alcoholic subjects.

Parameters	Non-alcoholic subjects n=30	Alcoholic subjects n=80
	Mean±SE	
ALT(IU/L)	7.3±0.67	*10.7±0.81
AST (IU/L)	7.9±0.62	*12.0±0.9
Serum iron (µmol/L)	6.92±35.76	*4.43±20.89
TIBC(µmol/L)	7.79±58.43	*10.18±85.03

*The significant values are between Non-alcoholic and alcoholic subjects at(p<0.05).ALT=Alanine transaminase , AST= Aspartic transaminase,TIBC= Total iron binding capacity of serum.

Discussion:

The results of the present investigation indicate that hematological parameters are altered in individuals with alcohol intake. It is observed that anemia is more common in individuals with alcoholics. In the present study, alcoholic subjects had low hemoglobin levels, mean MCH, and MCHC which were normal among the non-alcoholic group and it decreasing among alcoholics. All alcoholic subjects have total white blood cell (WBC) count and RBC count were low as compared to non-alcoholic subjects.

Alcohol abuse is a growing epidemic, especially among men, and nowadays, it is becoming a major problem among young adults . The clinical manifestations of alcohol-induced hematologic disorders are profoundly influenced by the patient's social and economic status, and the presence or absence of other factors such as nutritional deficiency or alcoholic cirrhosis. Most of these changes result, either directly or indirectly, in anemia and when extensive liver disease is present, the patient may develop an abnormally functioning fibrinogen or other coagulation disorders, which may initiate or exacerbate bleeding .Studies had shown that even before anemia appears, approximately 90% of alcoholics have a macrocytosis (mean corpuscular volume (MCV) between 100 and 110 femtoliter (fL) Alcohol-induced macrocytosis occurs even though patients are folate and cobalamin replete and do not have liver disease. The mechanism is unknown, but it takes 2-4 months for the macrocytosis to disappear after the patient becomes abstinent. The results of the study are in concordance with that of the previous study. Earlier studies have found that prolonged and excessive consumption of alcohol through direct or indirect effect suppresses hematopoiesis in individuals with alcohol dependence leading to a decrease in RBCs and WBCs counts. The previous investigators have found an increased prevalence of anemia in individuals with alcohol consumption for a long duration. The results of the present study are in concordance with that of the earlier studies (Bjorkholm,1980; Seppa & Sillanaukee,1992 ;Das,2005).

Alcohol as well as alcohol-induced cirrhosis leads to decreased RBC production. Hypersplenism can cause premature RBC destruction. Folic acid deficiency impairs RBC production and results from decreased ingestion, decreased absorption, and abnormal metabolism of folic acid (Harold and Ballard,1997). Hypersplenism, blood loss, liver disease, folic acid deficiency, and reduced RBC production are causes of low hemoglobin levels in alcoholics (Thinnahanumai *et al.*,2012). Alcoholism has effect on blood indices, and total leukocyte count also Chalmers *et al.*,1981).Stanly and Wakwe (2003) routine hematological indices were studied in problem drinkers to help in the diagnosis of alcohol-related disorders. A look at the hemoglobin profile , especially MCV, can alert a physician if the patient is alcoholic, even when there is no anemia.

Excessive alcohol consumption can cause liver diseases including fatty liver, hepatitis and cirrhosis (Zakhani,2007;Oshea *et al.*,2010 ; Kim *et al.*,2012).Since alcohol is mainly metabolized by the liver, it is a primary site of alcohol-induced adverse health effects. Alcohol consumers had significantly higher AST and ALT activities compared to non-consumers confirming previous findings demonstrating that alcohol intake is associated with increased hepatic enzyme activities (Alatalo *et al.*,2009). Changes in liver enzymes activities are biomarkers of liver damage and are routinely assessed for diagnostic purposes and as part of physical examinations(Limdy *et al.*,2003;Wbreta and Alqahtani,2014). Abnormal activities of liver enzymes are also strong predictors of mortality associated with liver disease, cardiovascular disease, diabetes and cancer (Lee *et al.*,2008 ; Ruhi and Evenhart ,2009 ; Targher,2010 ; Kunutsor *et al.*,2014).

In the last 15 year, alcohol-related deaths have doubled. In 2005, there were 8386 deaths related to alcohol. Death rates in both sexes and all age groups are increasing. Alcohol-related deaths are twice as frequent in males (National Statistics , 2006).

Alcoholic liver disease (ALD) is conventionally divided up into three

histological types, although features of each may coexist in the same patient. The metabolism of ethanol causes the accumulation of lipid in liver cells (Lieber ,1995) This is steatosis or fatty liver estimated to occur in 90 – 100% of all heavy drinkers Largely symptom free, it resolves completely within a few months of cessation of alcohol intake (McCullough & O'Connor , 1998). Ethanol metabolism can generate reactive oxygen species and neo-antigens (Lieber , 1995) promoting inflammation. This alcoholic hepatitis (AH) occurs in 10 – 35% of heavy drinkers. It may be mild and virtually asymptomatic or it may lead to acute flare ups with all of the signs of the systemic inflammatory response syndrome(IRS) and subsequent multi-organ failure(MOF). In patients with confirmed AH on biopsy who continue to drink, 40% progress on to cirrhosis (McCullough & O'Connor , 1998).

Prolonged hepatocellular damage generates myofibroblast like cells which produce collagen resulting in fibrosis (Lieber ,1995) As hepatocytes are destroyed and liver architecture changes, hepatic function falls and increased resistance to portal blood flow produces portal hypertension. Approximately (8 – 20%) of heavy drinkers will develop cirrhosis, which may be asymptomatic. However, acute episodes of liver failure may

Conclusions:

The data presented in this study shows that alcohol consumption affects physiological

be induced by episodes of AH or complications of portal hypertension such as variceal bleeding. In patients whose first presentation is an episode of severe decompensated ALD 85 % will have AH and more than 70% will have cirrhosis (Elphick *et al.*, 2007)

The single biggest risk factor is the quantity of alcohol ingested, irrespective of what form it is taken in there is considerable debate as to what constitutes a 'safe' level of alcohol consumption, although it is evident that females are more at risk than males at any individual consumption level. Epidemiological data had initially suggested consumption of (80 g) of ethanol per day in men and (60 g) in women, for between (10 and 12) year, is necessary for alcoholic liver damage to occur. However, recently these levels have been questioned, with new figures of (40 g) per day in men and (10 – 20 g) per day in women being associated with an increased relative risk of developing liver disease. For comparison, one UK 'unit' of alcohol contains (10 – 12 g) of ethanol. Other risk factors include obesity and hepatitis C infection .Despite this, it is unknown exactly why ALD only affects a proportion of heavy drinkers, even in the presence of other risk factors (Lieber , 1995).

blood parameters, blood indices and activity of liver enzymes AST and ALT. In addition, this study indicated alcohol had an effect on serum iron and TIBC of blood serum.

References:

- Alatalo , P.;Koivisto ,H.;Puukka K;Hietsla J.Anttila P,&Boigu R.(2009).Biomarkers of liver status in heavy drinkers,moderate drinkers and abstainers alcohol .Alcohol,44:199-203
- Ballard ,H.S.(1997). The hematological complication of alcoholism.Alcohol Research and Health.National Institute on Alcohol Abuse and Alcoholism,21(1),pp.42-52.Available at:/pmc/articles/PMC6826798/?report=abstract(Accessed:21December 2020).
- Bjorkholm M.(1980).Immunological and hematological abnormalities in chronic alcoholism .J. Intern. Med.,207:197-200.
- Chalmers D.M.; Rinsler M.G.;MacDermott S.;Spicer C.C.&Levi A.J.(1981).Biochemical and haematological indicators of excessive alcohol consumption.Gut,22:992-996.
- Brown,B.A.(1976). Haematology:Principles and procedures.2th ed.,Lea and Febiger,Philadelphia.
- Das S.K.& Vasudevan D.M.(2005).Biochemical diagnosis of alcoholism.Indian J.Clin.Biochem.,20:35-42.
- Deitrich , R.A. , & Erwin ,V.G.,EDS.(1996).Pharmacological effects of ethanol on the nervous system.Boca Raton,FL:CRC Press,pp.383-408.
- Elphick D.A. ; Dube A.K. & McFarlane E.(2007). Spectrum of liver histology in presumed decompensated alcoholic liver disease. Am. J. Gastroenterol., 102: 780–8.
- Hall,R.D. and Malia R.S.(1984).Medical Laboratory Haematology.3rd ed ,Butterworths,London.
- Harold S.& Ballard M.D. (1997).Haematological complications of alcoholism.Alcohol Health Res World ,21:42-52.
- Holmgren,A. and Jones , A.W.(2010).Demographics of suicide victims in Sweden in relation to their blood-alcohol concentration and the ciecummstances and manner of death .Forensic science international ,198(1-3),pp.17-22.
- Kim H.C. ; Nam O. M. ; Jee S.H. Han K.H. ;Oh D.K.&Suh I.(2012).Normal serum aminotransferase concentration and risk of mortality from liver diseases prospective cohort study.Brit.Med. J., 36: 328-983.
- Kunutsor S. K.; Apekey T.A.; Seddoh D.&Walley J.(2014).Liver enzymes and risk of all-cause mortality in general populations a systematic review and meta-analysis . Int.J.Epidemiol., 43:187-201.
- Lee ,S.Y.Park H.J.;Best-popes C.; Jang S.& Park Y.K.(2015).The effects of ethanol on the mprphological and biochemical properties of individual human red blood cells .PLoS One :10e0145327.
- Lieber C.S.(1995). Medical disorders of alcoholism. N. Engl. J .Med., 333:1058–65.
- Limdi J.K. &Hyde ,G.M.(2003).Evaluation of abnormal liver function tests.Postgrad Med. J.,79:307-312.
- McCullough A. J. & O'Connor J.F.B.(1998). Alcoholic liver disease: proposed recommendations for the American College of Gastroenterology. Am. J. Gastroenterol., 93: 2022–36.
- National Statistics. Alcohol-related death rates almost double since(1991) . News Release, issued November 7, 2006. Available from <http://www.statistics.gov.uk/pdffdir/aldeaths1106.pdf>.
- O'Shea R.S. ; Dasarathy S. & McCullough A.J.Alcoholic.(2010).

- liver disease
.Am.J.Gastroenterol.,105:14-32 Lee
T.H. ; Kim W.R.;Benson
J.T.;Themeau T.M.&Melton
L.J.(2008).Serumaminotransferase
activity and mortality risk in a United
States community.Hepatology
,47:880-887.
20. Retiman,F.S. and
Frankel,H.N.(1957).A colorimetric
method for determination of serum
glutamic oxaloacetic and glutamic
pyruvic
aminotransaminases,Am.J.Clin.Patho.
28:56-59.
21. Ruhi C.E. &Evehart J.E.
(2009)..Elevated serum alanine
aminotransferase and gamma-
glutamyltransferase and mortality in
the United States
population.Gastroenterology
,136:477-485.
22. Scott-Ham,M. and Burton ,
F.C.(2005).Toxicological findings in
cases of alleged drug-facilitated sexual
assault in the United Kingdom over a
3-year period.Journal of clinical
forensic medicine,1294),pp.175-186.
23. Seppa K.;Sillanaukee P.& Koivula
T.(1992).Abnormalities of
hematologic parameters in heavy
drinkers and alcoholics .Alcohol Clin
Exp Res,16:117-21.
24. Shirlyn , B.S. (2004). Clinical
Laboratory Haematology, 1st ed.,
University of Texas ScienceXCenter
at San Antonio.U.S.A.
31. on liver disease .hepatology, 46:2032-
2039.
25. Stanly , P. C.;Wakwe , V.C. & Ejele ,
O.A. (2003).Haematological profile
in chronic alcoholics consumers Niger
J. Clin Pract.,6:84-88.
26. Targher G.(2010).Elevated serum
gamma-glutamyltransferase activity is
associated with increased risk of
mortality ,incident type 2 diabetes
cardiovascular events chronic kidney
disease and cancer- a review
.Clin.Chem.Lab.Med.,48:147-157.
27. Thinnahanumaih M.; Puranik
N.;Bidare C.;Kammar
K.F.Venkatakrishniah O.K. &Maitri
V.(2012).Moderate alcohol intake for
even a short duration has deleterious
effects on hematologic profile in
Indian men
.Int.J.Med.Sci.Public.Health ,1:105-
108.
28. Wbreta T.A. &Alqahtani S.A.(2014).
Evaluation of abnormal liver tests
.Med Clin.N.Am.,98:1-16.
29. World Health Organization.The World
Health Report (2018). Reducing
Risks, Promoting Healthy Life
Geneva: World Health Organization;
2018. Available
from:[http://whqlibdoc.who.
int/publication /2018/9241562072.
pdf](http://whqlibdoc.who.int/publication/2018/9241562072.pdf). [Last assessed on 2019 May 28].
30. Zakhani S. &Li
T.K.(2007).determinats of alcohol use
and abuse :impact of quantity and
frequency patterms