

## ALCOHOLIC HEPATITIS - CONCEPT AND MANAGEMENT IN UNANI MEDICINE: A REVIEW

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#### ABSTRACT

Alcoholic hepatitis is a much more severe lesion than alcoholic fatty liver and is often a precursor to the development of cirrhosis. Excessive alcohol consumption is the third leading preventable cause of death in the world. It is associated with many short term and long term liver damage. The association between alcohol intake and alcoholic liver diseases are well known documented. Alcohol consumption exceeding 60-80 gm/ day in men and 20 gm/day in women increases the risk of liver disease. If we trace back the genesis of this problem we came to know that the ancient Unani scholars were well acquainted with the basic concept of this disorder. Warm KabidHarSafravi as described in literatures are supposed to be caused due to excessive alcohol consumption. The symptom and signs of alcoholic hepatitis are abdominal pain, anorexia, low grade fever, nausea, vomiting, enlarged liver, yellow coloured urine, breathlessness et depending upon the site of involvement. The full picture of a florid, acute alcoholic hepatitis is relatively rare. There are all gradations of severity. The hepatitis may be separate or can be combined with an established cirrhosis. Despite the advancement in modern pharmacotherapy, the figure in terms of remission of disease, withdrawal symptoms and adverse side effects grossly suggest the limitation in its management.

KEY WORDS: Warm kabidharsafravi; alcoholic hepatitis; Unani;IlajbilGhiza; Ilajbiltadabeer; Ilajbildawa.

## INTRODUCTION

Alcoholic hepatitis is one of the clinical manifestations of chronic alcohol abuse. Alcoholic liver diseases are characterised histologically by fatty changes, degeneration and necrosis of hepatocytes, and an inflammatory infiltrate of neutrophils.<sup>1</sup> Almost all patients have fibrosis and they may have cirrhosis of liver.<sup>1</sup> The pathology of alcoholic liver disease comprises three major lesions, with the injury rarely existing in a pure form: (1) fatty liver, (2) alcoholic hepatitis, and (3) cirrhosis. Fatty liver is present in >90% of binge and chronic drinkers. A much smaller percentage of heavy drinkers will progress to alcoholic hepatitis, thought to be a precursor to cirrhosis.<sup>2,3</sup>

**Defination of Alcohol:** Alcohol is a type of drink prepared by the process of fermentation (khumr) and is commonly known as Nabeez.<sup>4</sup> Alcohol has been one of the most commonly used chemical substances for intoxication by man since time immemorial. Originally alcohol is powder.<sup>5</sup> Alcohol, word used in general was derived from Arabic literatures. Arabic chemists also used this term in sense of al-kuhul meaning 'the kohl' (powder for the eyes), which later came to mean 'finely divided spirit' by Latin Scientist. They also used this to refer to other substances that were obtained by distillation.<sup>5</sup> It is produced naturally by interaction of yeast and fruit juices which are rich in sugar contents e.g. grapes, honey, dates etc.<sup>4</sup>Alcoholic beverages were manufactured by three different process i.e.(1)Fermentation (khumr), (2)Brewing, (3)Distillation (AmalTaqteer).<sup>6</sup> The natural sugars in fruit juices, get fermented by making its contact with yeast, and ethanol is produced until

the alcohol content of beverages reaches about 12%. At 12% concentration of ethanol, yeast is killed and fermentation stops. Wines produced by fermentation average an alcohol content of about 12%. The process of brewing, by which beer is made, is similar to fermentation that is carried out at a higher temperature then wines. More higher concentration as in "Spirits", are obtained by the process of distillation. All the alcoholic beverages usually having the same chemical compound known as ethyl alcohol or ethanol. It is a hydrocarbon with one hydrogen atom replaced by hydroxyl group(OH) group. Different types of alcohol, there preparations and effects are described in detail in ancient texts. White wine, which has been fermented with a piece of cake or bread, and is diluted with water couple of hours before use, is useful for hot temperament people. They do not cause headache and their moistening effect relieves headache caused by irritation of stomach.<sup>7,8</sup> Old red wine suits persons of cold and phlegmatic temperaments better. Superior the aroma and flavour of the wine, the more beneficial it would be as drink. Alcohol assists the diffusion of nutrient throughout the body. It makes phlegm thin and aids the elimination of bile through urine and other excretions.<sup>9</sup> Old wine is more a medicine than a food. Fresh wines upset the liver and produces diarrhoea and flatulence.<sup>7</sup> The best wine is the one which is neither too old nor too fresh. It may be white or red but should be clear.<sup>9</sup>It should taste neither sweet nor sour and should have a pleasant smell. Maghsoolis a special drink prepared by boiling three parts of grape juice with one pert of water until one third remains. In childhood, drinking alcohol is like adding fuel to fire. Elderly persons may be allowed to drink within the limit of tolerance, but young should be moderate.9,10

### EPIDEMIOLOGY

Alcohol is one of the most common drugs of abuse, In developed countries alcohol is one of the ten leading causes of disease and injury. Worldwide, alcohol causes 3.27 per cent of deaths (1.8 million) and 4 per cent of 'disability adjusted life years' lost (DALYS) (58.3 million). In developed countries, alcohol is responsible for 9.2 per cent of the disease burden. Alcohol causes nearly 10 percent of all ill-health and premature deaths in Europe. The World Health Organization's Global Burden of Disease Study finds that alcohol is the third most important risk factor, after smoking and raised blood pressure, for ill-health and premature death. Alcohol is more important than high cholesterol levels and overweight, three times more important than diabetes and five times more important than asthma.<sup>11</sup>

This level of alcohol-related death, disease and disability is much higher in men than women, it ranges from 8 - 18% for males and 2 - 4% for females. Beside the direct effects of intoxication and addiction, worldwide alcohol is estimated to cause 20-30% of cancer of the oesophagus, liver cancer, cirrhosis of the liver, epilepsy, homicide and motor vehicle accidents.<sup>12</sup>

For all types of alcohol-related harm, including cancers, cardiovascular diseases and cirrhosis of the liver, the more an individual drinks, the greater the risk of harm. The annual risk of death from alcohol related cancers (mouth, gullet, throat and liver) increases from 14 per 100,000 for non-drinking middle-aged men to 50 per 100,000 at 4 or more drinks (4 glasses of wine) a day. The risk of breast cancer by age 80 years increases from 88 per 1000 non-drinking women to 133 per 1000 at 6 drinks (a bottle of wine) a day. <sup>11,12</sup>

#### INCIDENCE

Chronic alcohol consumption has a variety of adverse effects. Of great impact, however, are the three distinctive, albeit overlapping, forms of alcoholic liver disease: (1) hepatic steatosis (fatty liver), (2) alcoholic hepatitis, and (3) cirrhosis, collectively referred to as alcoholic liver disease (ALD).<sup>13,14</sup> 90% to 100% of heavy drinkers develop fatty liver (steatosis), and of those, 10% to 35% develop alcoholic hepatitis. However, only 8% to 20% of chronic alcoholics develop cirrhosis. Steatosis and alcoholic hepatitis may develop independently, and thus, they do not necessarily represent a continuum of changes. Out of these, fatty liver and alcoholic hepatitis are somewhat reversible as the result of abstinence and proper management, but once cirrhosis develops, it can't be reversed. Cirrhosis is the end stage of liver diseases. <sup>2,13,14</sup>

#### PATHOGENESIS Direct toxic effects of alcohol

**Sue mizaj** of liver: Alcohol disturbed the mizaj of the liver, as it is highly absorbable, so it readily get absorbed to the liver and its excess heat effects directly to the liver and increases the hararat of the liver.<sup>15,16,</sup>

**Sudda** formation: Ghaleezsharab and especially sweet one, is capable of creating sudda in the liver. Because the liver has its affinity toward the sweet alcohol and alcohol itself is highly absorbable. So it directly absorbed to the liver prior to full digestion and the canules of liver is also very constricted. So this heavy matter block the fine canules.<sup>17,18,19</sup>

**TaffarrukIttesal:** Alcohol is responsible for the diarrhoea, because of its water content which makes intestine wet and relaxed. Due to the diarrhoea the food is not properly digested and the amount which have to reach liver ultimately decreases in amount. Hence making the faculties of liver week. Also alcohol produces tension in liver by producing riyah in it. That leads to taffarrukittesal (hepatic cell injury). All the above pathology is responsible for the **zoosantariyakabidi**. <sup>5,16,17,20</sup>

Alcohol, acetaldehyde and their metabolic products all play a role in promoting liver damage; the precise contribution of each is difficult to define. <sup>21,22,23</sup>

**Quantity and pattern of drinking:** It is one of the main risk factor that should always be estimated, as it helps in making the diagnosis. Drinking pattern vary from person to person and in different regions of the world. The amount of alcohol in the blood stream is called as **Blood Alcohol Concentration** (**BAC**).<sup>1,2,14</sup> This unit is called milligrams percent (literally "milligrams per hundred") and is abbreviated mg%. Thus 120 mg% means 120 mg of ethanol per 100 ml of blood.<sup>24</sup>

The threshold for developing alcoholic liver disease in men is an intake of >60-80 gm/d of alcohol for 10 years, while women are at increased risk for developing similar degrees of liver injury by consuming 20–40 gm/d. Ingestion of 160 g/d is associated with 25–fold increased risk of developing alcoholic cirrhosis.<sup>14,25</sup> Through normal metabolism, the average person's body is able to decrease BAC by .015-.017 per hour. For a 160 pound (72 kg) male, that is roughly the equivalent of one half ounce of ethanol per hour (14 gm.). The following drinks according to their concentration gives same effects.<sup>3,13</sup>

- 10 ounces of beer (5% ethanol) (280 gm)
- 4 ounces of table wine (12% ethanol) (113 gm)
- 2.5 ounces of fortified wine (20% ethanol) (70 gm)
- 1 ounce of 100 proof liquor (50% ethanol) (28 gm)

Gender-dependent differences result from poorly understood effects of estrogen and the metabolism of alcohol. Social, immunologic, and heritable factors have all been postulated to play a part in the development of the pathogenic process.<sup>2,3,13</sup>

### CLINICAL MANIFESTATION

The spectrum of ALD ranges from asymptomatic hepatomegaly to hepatocellular failure from alcoholic hepatitis or end-stage cirrhosis.<sup>6,26</sup> The clinical manifestations of alcoholic fatty liver are subtle and characteristically detected as a consequence of the patient's visit for a seemingly unrelated matter.<sup>25</sup> Previously unsuspected hepatomegaly is often the only clinical finding. Patients with alcoholic hepatitis usually complain of : Anorexia, Nausea, Vomiting, Hiccups, Malaise, Weakness, Abdominal pain, Icterus, Weight loss, Tender, hepatomegaly, Fever, Excessive thirst, Diarrhea, Encephalopathy, Ascites and Splenomegaly<sup>7,26</sup>

### **UNANI DIAGNOSTIC TOOLS**

**Nabz**(examination of pulse), **Qaroora** (naked eye examination of urine) and **Baraaz** (naked eye examination of stool) are the unique diagnostic tools of this system.

**Nabz:** Nabzmauji, azeem, sariee, mutawatir along with flushing of face and redness of tongue indicates that it is Damvi warm. And if nabz is sulb, sagheer, sariee, mutawatir and minsharialong with other symptoms of warm kabidhar indicates that it is Safravi warm.<sup>10,17,18,27</sup>

**Qaroora:** Yellow coloured urine Baulnari, is the sign of warm kabid haar.  $^{18,27}$ 

**Baraaz:**Yellow and slight orange colour stool.<sup>8,10,15</sup> Stool is soft in consistency if warm kabidis accompanied by zofekabid<sup>27</sup>

## PROGNOSIS

The most important prognostic factor is the patient's ability to stop drinking alcohol. General health and life expectancy are improved when this occurs, irrespective of the form of alcoholic liver disease.<sup>3</sup>

As mentioned in classic text, it is stated that if there is epistaxis from right nostril and inflammation is on convex side, then there is a hope. And also if excessive micturition along with excess sweat and inflammation is on concave side then also it is sign of good prognosis.<sup>17</sup> Alcoholic hepatitis has a significantly worse prognosis. About one-third of patients die in the acute episode, particularly those with hepatic encephalopathy or a prothrombin time sufficiently prolonged to exclude a percutaneous liver biopsy. Cirrhosis, if not already present, will occur if drinking continues. Patients with acute alcoholic hepatitis often deteriorate during the first 1-3 weeks in hospital. Even if they abstain, it may take up to 6 months for jaundice to resolve. In patients presenting with jaundice who subsequently abstain, the 3- and 5-year survival is 70%. In contrast, those who continue to drink have 3- and 5-year survival rates of 60% and 34% respectively.<sup>12</sup> Critically ill patients with alcoholic hepatitis have short-term (30 day) mortality rates >50%. Severe alcoholic hepatitis is heralded by coagulopathy (prothrombin time >5 s), anemia, serum albumin concentrations <25 g/L (2.5 mg/dL), serum bilirubin levels > 137 mol/L (8 mg/dL), renal failure, and ascites. A Discriminant function calculated as 4.6 x [prothrombin time control (seconds)] + serum bilirubin (mg/dL) can identify patients with a poor prognosis (discriminant function>32)  $^{2,3,25}$ (discriminant function>32).

# USOOLE ILAJ( PRINCIPLE OF MANAGEMENT)

Tracing through the ancient literature, now it is quite clear that alcoholic hepatitis is better interpreted as warm kabidhaarsafravi. The line of management of warm kabidhaarsafravi can be used in alcoholic hepatits. Constipation is avoided in the warm kabid but excess of purgation also controlled, as it may be lethal.<sup>17,18</sup> As the condition of patients allows first do the fasadof basaleeq or haft indam. Use of radeaat (repellents), mulattif (demulcent) and mufattah (deobstruent) drugs is to be prescribed.<sup>4,10,19,28</sup> Because radeaat prevents the matter to become viscous and hard and muhallil drugs along with qabizat (astringents) and atrivat(volatile aromatic) helps in dissolution of morbid matter and also act as liver tonic.<sup>7,8,29</sup> This line of management should be followed both in oral and local formulations for warm kabid.<sup>19</sup> In the initial stage of diseases use of sharbat, ghiza and tila e radeatis preferable but along with them at least a small amount of muhallil should be increased.<sup>10,18</sup> It is used in this way because radae drugs causes constriction of canules of liver which hinders the passage of morbid matter outside that already get accumulated in the liver. So it can be a cause of conversion of 7827this warm kabid into warm sulb (hard ).

IbnSarabiyun stated that drugs used for warm kabid must be of moderate potency neither very hot nor cold, as cold drugs will cause ascites and hot drugs leads to excessive weakness and in both situations treatment will become difficult.<sup>17</sup>

Use of muhallil durgs, can leads to weakness of the faculties of liver, so to overcome this we should add some qabiz(astringent)

and atriyat(aromatics) which act as liver tonic.<sup>1</sup>. As the diseases progress, radeat should be tapered down and muhallilat should be increased. At last when disease is in its final stage only a small amount of astringent and repellent are to be given and mostly treatment should be consist of muhallil drugs.<sup>18,28</sup> If the inflammation is on concave side then mushil(purgatives) are used. But avoid very high purgatives and if it is on convex side than prefer using mudirrat (diuretics).<sup>27,29</sup> According to Jaleenoos, until the inflammation is on prodromal period, the use of diuretics and purgatives are avoided. When the inflammation comes to the period of dissolution and the sign of nuzj is observed then use mulayyanat (light laxatives).<sup>15,20</sup>

## ILAJ:

In Unani system of medicine, treatment of any diseases is comprised of three different forms:

**IlajbilGhiza(Dietotherapy):** It is obvious that nutrition plays important part in management of alcoholic liver disease. As there is always associated malnutrition with it. It may be due to the fact that this affects the Quwwat (faculties) of liver and ultimately disrupts there functions. So, different type of nutritious diet should be recommended especially of protein calorie type, as it improves nitrogen balance, may improve liver function and may decrease hepatic fat accumulation.<sup>30</sup>

**Recommended diet:** Light and easily digestible diet should be prescribed for this. Use of mutton soup, chicken soup, pulses such as moong dal arhar dal etc., sagodanankikheer, aabnakhud, Daliya, palak, pudina proves to be beneficial. Maul shaer is also better for these conditions.<sup>31,32</sup>

**Restricted diet:** The first thing to leave is alcohol consumption. Alcohol abstinence is first line of treatment for this. Use of high caloric and fatty food items are avoided. Use of beaf meat, egg, butter, milk, garammasalehjaat, lahsun, pyaz, , ladyfinger, potato, mash kidaal etc. are better not given.<sup>31,32</sup>

## Ilajbiltadabeer (Regiminal Therapy)

In case of warm jigarhaar, if patients conditions allow and other factors are suitable then first do the fasad (venesection) of baasaliq (basilic vein) of the right side.<sup>18</sup> After venesection give the patients sikanjabeenshakri along with aab lablab and amlatas to relieve the patients.<sup>13,14,15</sup> Huqna(enema) of low potency can be used. Enema can be prepared by laxatives drugs like unnab, sapistan, banafsha, neelofar, khatmi and roghangul.<sup>27,28</sup>

## Ilajbildawa(Pharmaco Therapy)

After that according to position of inflammation, purgatives and diuretics can be used. If the inflammation is on concave side then use of diuretics are avoided and purgatives and light muhallil such as TukhmKasni, BekhKasni, MakoKhushk, all given as khisanda and when fever comes down mixing of sugar and given in joshanda (decoction) is beneficial.<sup>18,27</sup>

If the inflammation is on convex side of liver, then use of diuretics of cold in nature are used. For this purpose AabAnar, Sikanjabeen are quite effective.<sup>4,10</sup> Also AabKasni, Aabanbusalab, Sharbatanarain, Sikanjabeensada, Parsiyaonshan, Asalalsoosetc can be given.<sup>15</sup> Use of purgatives are avoided but if constipation is severe than light acting laxatives like Luabbahdana, luabasapghol etc can be given.<sup>17,18</sup> Locally zimadaat (ointment) are effective in relieving pain as well as inflammation. For the purpose Radeaat and muhallilat are used in ointment such as sandal, zarward, aradjo, all along with Aabkishneez and roghangul mixed and applied locally.<sup>18,19,33</sup> Use of akleelulmalik, afsanteen, and zaafran are advised in active stages, and in last stage of diseases only muqawwiand

muhallil are used in ointment, such as ood, zaafran etc.<sup>28,29</sup> If there is diarrhoea along with inflammation, then tukhmhamas, tabasheer, revandchini can be given.<sup>29</sup> Rub rebas, rub zarishk, and rub anar can have the same effect.<sup>18,19</sup> All the drugs and diet which have astringent effect such as guava, apple, etc. are not indicated because it leads to constriction of passage between liver and gall bladder. Due to which bile get collected in the liver and help in increase of inflamation.<sup>20</sup>

Along with all these things it should be cleared that liver has its tendency to dissolute easily so when using muhallil it should be kept in mind that those muhallilat which have also laza (irritant) property should never be given.<sup>18,19,33</sup> For this use of Malulshaer (barley water) is beneficial as it have no irritant activity. Maulasal (honey water) can also be used but due to its sweetness it can cause sudda. So better to give maalshaer to the patients in all stages of diseases.<sup>16,18,19</sup>

Most common drugs that are useful in this condition are as: Kasni, mako, adrakraevand, kishneez, afsanteen, aneesoon, tukhmkassos, bekhkarafas, barggulab, snadalain, bekhazkhar, gaozban, tukhmkhyarain, bartang, gulneelofar, aabananas, aab lemon, ushna, anarain, bekhbadyan, tukhmkharpaza, oodshirin ,ood tursh.<sup>34,35</sup> Most effective compound drugs as mentioned in classic text are: Sikanjabeen, Dawaul Kirkum, Jawarishjalinoos, Majun Reavand, Majun Dabidulward, Dawaul Miskmoatdil, Khameeramarwarid, Qurs Zarishk, Qurs Ghafis, MajunJuntiyana, Majungul, Majun Kalkalanj, Majun Afsanteen, Zimad Jalinoos, Zimad Sunbuluttib, Roghan Afsanteen, Dawae khabsulhadid.<sup>36,37</sup>

### CONCLUSION

It is evident from the above described subjects that the Unani system of medicines has properly mentioned the every aspect of alcohol related entity. So regarding treatment of alcohol related diseases and its complications Unani drugs are quite effective and have better prognosis. Using the natural way of healing we can have a better scene of treating alcoholic liver diseases.

#### REFERENCES

- Ghosh Amit K. Mayo clinic Internal Medicine Review. 8<sup>th</sup> ed. Florence. Mayo Clinic Scientific Press; 2008:302-303.
- Kasper DL, Braunwald E, Hauser SL, Fauci AS, Longo L, Jameson L. Harrison's Principle of Internal Medicine. 17<sup>th</sup> ed. Vol. 2nd. New Delhi: McGraw Hill; 2008: 1969-1971.
- Hunter AAJ, Colledge RN, Boon AN, Chilvers RE, Christopher H. Davidson's Principle and Practice of Medicine. 20<sup>th</sup>ed. New Delhi: Churchill Livingstone; 2004: 954-957
- Majusi AIA. KamilusSanaa. Vol-2. (Urdu translation by Kantoori GH) New Delhi: IdaraKitabushShifa; 2010: 442-448.
- Anonymous. History of alcohol: Report by WHO, SEARO; 2003:7-10
- Rosenthal MD, Glew RH. Medical biochemistry:Human Metabolism In Health And Disease. 1<sup>st</sup> ed. USA. John Wiley &Sons, Inc.;2009: 191-198
- Tabri AM. MolaejatBuqratiyah. (Urdu translation by CCRUM). Vol- 3<sup>rd</sup>. New Delhi: Ministry of Health and Family Welfare; 1997: 280-299.
- Ismail Jurjani, ZakheeraKhawarzamShahi. (Urdu translation by Khan HH). Vol-2<sup>nd</sup>. Part 6<sup>th</sup> : New Delhi: IdaraKitabushShifa; 2010: 385-395.
- Shah Mazhar H. The General Principles of Avicenna's Canon of Medicine. New Delhi. IdaraKitab-Ul-Shifa. 2007: 321-325.

- 10. Blocker JS et. al.:Alcohol and Temperance in History. An International Encyclopedia. 2003: 1-8.
- 11. IAS Factsheet- Alcohol and health; Institue of Alcohol Studies; Cambridge;2010; 1-18
- Alcohol & substances abuses; WHO Globus Status Report on Alcohol; 2004;27-32
- Kumar P, Clark M. Kumar and Clark's Clinical Medicine. 7<sup>th</sup>ed. New Delhi. Saunders Elseviers;2009; 358-359
- DavidHumes H. Kellys Textbook of Internal Medicine. 4<sup>th</sup> ed. USA: Lippincott Williams & Wilkins; 2000:984-996
- 15. Arzani Akbar. Tibe Akbar (Urdu translation by Hussain M). Deoband: Faisal Publications; YNM: 739-740.
- DawoodAntaki. TazkirahOolilAlbab (Arabic).Vol-3. New Delhi: CCRUM, Ministry of Health and Family Welfare; 2010: 279, 334-338.
- Qamri AMH. GhinaMuna Ma TarjumaMinhajulllaj. 1<sup>st</sup>ed. (Urdu translation by CCRUM). New Delhi: Ministry of Health and Family Welfare; Govt. of India; 2008: 252-263.
- Khan Azam. Ramozazam, Vol-II, CCRUM. New Delhi: Ministry of Health and Family Welfare; Govt. of India; 2006: 12.
- Qarshi HMH. Jamiulhikmat. New Delhi: IdaraKitab-Us-Shifa; 2011:801-807.
- Khan MA. AkseerAzam; Vol-2; (Urdu translation by Kabeeruddin M): New Delhi. Aijaz Publishing House; 2003:848-865.
- Goldman L, Ausiello D. Cecil medicine. 24<sup>th</sup> ed. Philadelphia: Saunders Elseviers; 2008: 996-998.
- Lawrence M. Tierney, Stephen J. McPhee, Maxine A.Current Medical Diagnosis & Treatment. 49<sup>th</sup> ed. USA: McGraw Hill. 2010: 614-617.
- 23. Kumar, Abbas, Fausto, Mitchell. Robins Basic Pathology. 8<sup>th</sup>ed. New Delhi: Saunders Elseviers: 2007; 904-907.
- 24. Padilla FJB, Elizondo GV, Vazquez MAM, Garza HJM. Pentoxifylline and Prednisolone in severe alcoholic hepatitis. Annals of hepatology. 2009; 8(4); 402-404.
- 25. Siddharth N. Shah. API Textbook of Medicine. 8<sup>th</sup> ed. Vol-1. Mumbai: The Association of Physicians of India; 2008: 693-696.
- Chauhan A, Sahu RL, Bainola PK. Trends Changes in Alcoholic Consumption and its Impacts on Indian Society. Researcher. 2012; 4(3): 52-57.
- 27. Khan A, Haziq. 1<sup>st</sup>ed. New Delhi. Jasim Book Depot:1983: 304-310.
- Razi ABZ.Alhavi Fit Tib. Vol-7 ;New Delhi: CCRUM.: Ministry of Health and Family Welfare; Govt. of India:2000; 47-64.
- 29. Nafees AB, Kulliyatnafisi. (Urdu translation of Nafeesi by Kabeeruddin M). New Delhi: IdaraKitabushShifa; 398-415.
- Griffith CM, Schenker S. The Role of nutritional therapy in alcoholic liver disease. Alcohol Research and Health; 2006; 29(4): 296-306.
- George AS, Bauman A, Johnston A, Farrell G, Chey T. Effect of life style intervention in patients with abnormal liver enzymes and metabolic risk factors. Journal of Gastroentrology and Hepatology; 2008; 24(2009): 399-407.
- 32. Miller WR. Alcohol and its effect on behavior. World Health Forum; 1994: 15:229-231.
- Baghdadi IbnHubal. KitabulMukhtaratFilTibb. (Urdu translation by CCRUM). Vol-II,III New Delhi: Ministry of H & FW, Govt. of India; 2005: 330, 279-280.
- Ghani N. KhazainulAdvia. New Delhi: IdaraKitabulShifa; 2010:202,293,325,750,1357.
- 35. IbnBaitar. Al JamiulMufradatulAdviawalAghzia. (Urdu translation CCRUM). New Delhi: Ministry of Health and Family Welfare, Govt. of India; Vol-I,II, III, IV; 2000: 144,197,275,139-144, 265, 389,412.

- Ahsan Ali Hkm. QarabadeenEhsani. CCRUM. New Delhi: Ministry of Health and Family Welfare; Govt. of India; 2006: 134.
- Kabeeruddin M. Alqarabadeen. CCRUM. New Delhi: Ministry of Health and Family Welfare; Govt. of India; 2006:1162-1163.

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