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Fatty liver diseases and Unani system of medicine

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ABSTRACT

Fatty liver, also acknowledged as fatty liver disease (FLD) or hepatic steatosis, is a reversible condition wherein large vacuoles of triglyceride fat accumulate in liver cells via the process of steatosis (i.e., abnormal retention of lipids within a cell). It's normal to have some fat in the liver but in the case of fatty liver, more than 5-10 % of liver weight is fat. It now has become key medical condition and this importance mainly results from its ability to progress to cirrhosis and liver failure. It is generally classified as Alcoholic fatty liver and Non-Alcoholic Fatty Liver. Unani Scholars were also aware of the significant role of the liver in the normal functioning of the body and its mizaj (Temperament) is documented as hot and moist. Due to erratic dietary habits, excessive alcohol consumption, excess intake of fatty food, its mizaj altered to barid (cold) or haar (hot), which is not acceptable to liver , thereby, allowing the accretion of morbid matter in the form of fat (Tashhamul Kabid) which affects the normal functioning of liver. In spite, of incredible advancement in mainstream medicine, the role of pharmacotherapy in FLD remains investigational and it is not recommended for routine clinical practice. The present review highlighted the treasure of Unani classical text about Tashhamul Kabid which closely resembles FLD for efficient management of FLD.

Keywords: Fatty liver Disease, Fatty liver Unani Medicine, Tashhamul Kabid.

INTRODUCTION

Fatty liver, also acknowledged as fatty liver disease (FLD) or hepatic steatosis, is a reversible condition wherein large vacuoles of triglyceride fat accumulate in liver cells via the process of steatosis (i.e. abnormal retention of lipids within a cell). It's normal to have some fat in the liver but in case of fatty liver more than 5-10 % of liver weight is fat. Fatty liver is a reversible condition that can be resolved with changes in life style. It often has no symptoms and typically does not cause permanent damage (Walker et al, 2014).

It has become key medical condition and this importance is mainly resulted from its ability to progress to cirrhosis and liver failure. The liver is an important organ in the metabolism (handling) of fat.

The liver makes and supplies fat to other parts of the body. It also removes fat from the blood that has been released by other tissues in the body, for example, by fat cells, or absorbed from the food we eat (Kumar et al., 2005). In fatty liver disease, the handling of fat by liver cells is disturbed. Increased amounts of fat are removed from the blood and/or are produced by liver cells, and not enough is disposed of or exported by the cells (Kumar et al., 2005). As a result, fat accumulates in the liver. This leads to interruption in normal functioning of liver. The liver commonly repairs itself by rebuilding new liver cells when the old ones are damaged but when there is repeated damage, permanent scarring takes place called cirrhosis (Kumar et al., 2005).

Unani Concept

Fatty liver may be literally termed as Tashhamul Kabid. There is no specific depiction of fatty liver in Unani literature, but features described by the eminent physicians under the heading of Amraze Jigar (Liver disorders) due to Su'mizaj haar (impaired hot temperament) and Su'mizaj barid (impaired cold temperament) in various Unani text resemble with sign and symptoms of fatty liver (Ibn Sina, 2010; Ibn Rushd, 1987; Razi, 2000).

Thus, Su'mizaj haar kabid can be correlated to the present concept of AFL and Su'mizaj barid kabid to NAFLD.

According to scholars, kabid (Liver) is said to be one of the fundamental organ responsible for the metabolic activities mainly synthesis of Akhlaat (Ibn Sina, 2010; Ibn Rushd, 1987; Razi, 2000; Ibn Zuhr, 1986). The normal mizaj (Temperament) of liver is hot and moist (Ibn Rushd, 1987; Tabri 1997; Khan, 2011; Jurjani, 2010). Due to erratic dietary habits ,excessive fat consumption etc its mizaj is altered to barid, which is incompatible to the liver ,thereby allowing the accretion of morbid matter in the form of fat which disturbs the normal functioning of liver resulting in Su'mizaj barid kabid. Sometimes, the temperament of liver may be altered to haar due to excessive intake of hot foods,drinks, drugs, alcohol etc. Which interrupts the normal functioning of liver resulting in Su'mizaj haar kabid (Ibn Sina, 2010; Khan, 2011; Jurjani, 2010; Shah, 2007; Tabri, 2010).

Epidemiology

- Most cases of fatty liver are detected in people between ages 40 and 60.
- The prevalence of FLD in the general population ranges from 10% to 24% in various countries (Angulo, 2002).
- There is high prevalence of ALD in India and 50% cases of cirrhosis in India is due to alcohol abuse (Longo et al., 2012; Kelley et al., 1989; Kumar et al., 2009).
- NAFLD is thought to be the commonest liver disorder in western countries, affecting about a third of US population (Browing et al., 2014).
- The estimated prevalence of NAFLD in United States and Europe ranges from 14-20% (Longo et al., 2012) while in Asia it is around 12-24% (Chitturi et al., 2007). However, the condition is observed in up to 60-90% of obese people and up to 50% of Diabetes type II 9 (Walker et al., 2014).
- Fatty liver disease is the most common cause of liver disease in children. Almost 10% of children may have NAFLD, due in large part to an alarming increase in childhood obesity (Ovchinsky and Lavine, 2012). Fatty liver disease affects almost 3% of children and 22–53% of obese children. Fatty liver disease can be originated in children as young as four years of age. The chances of developing NAFLD increases with age, thus is more common in adolescents. Furthermore, more boys present the disease than girls (2:1) (Loomba et al., 2009).

What are the types of fatty liver: It is generally classified as Alcoholic fatty liver and Non-Alcoholic Fatty Liver (Kumar et al., 2005; Crowley, 2007; Goldman and Ausiello, 2008).

Alcoholic fatty liver: It is the liver manifestation of overconsumption of alcohol. Alcoholic fatty liver is the earliest phase of alcohol-related liver disease. Heavy drinking damages the liver, and the liver cannot break down fats as a result. If damage continues it leads to more severe form called alcoholic steatohepatitis. It is the major cause of liver disease in Western countries (O'shea et al., 2010). Of all chronic heavy drinkers, only 15–20% develops hepatitis or cirrhosis, which can occur concomitantly or in succession (Menon et al., 2001).

The mechanism behind this is not completely understood. 80% of alcohol passes through the liver to be detoxified. Chronic use of alcohol results in the discharge of pro-inflammatory cytokines (TNF-alpha, Interleukin 6 [IL6] and Interleukin 8[IL8]), oxidative stress, lipid peroxidation, and acetaldehyde toxicity. These factors cause inflammation, apoptosis and eventually fibrosis of liver cells (Longstreth et al., 2009).

The risk factors for ALD

- **Quantity of alcohol taken**: Consumption of 60–80g per day (about 75–100 mL/day) for 20 years or more in men, or 20 g/day (about 25 mL/day) for women significantly increases the risk of hepatitis and fibrosis by 7 to 47%m (O'shea et al., 2010; Mandayam et al., 2004).
- **Pattern of drinking:** Drinking outside of meal times increases up to 3 times the risk of alcoholic liver disease (Menon et al., 2001).
- **Gender:** Women are twice as susceptible to alcohol-related liver disease. (The lesser amount of alcohol dehydrogenase secreted

in the gut, higher proportion of body fat in women, and changes in fat absorption due to the menstrual cycle may explain this phenomenon (Menon et al., 2001).

- **Hepatitis C infection**: A concomitant hepatitis C infection significantly accelerates the process of liver injury (Menon et al., 2001).
- **Genetic factors:** Genetic factors predispose to alcoholism and to alcoholic liver disease. Both monozygotic twins are more likely to be alcoholics and to develop liver cirrhosis than both dizygotic twins. Polymorphisms in the enzymes involved in the metabolism of alcohol, such as ADH, ALDH, CYP4502E1, mitochondrial dysfunction, and cytokine polymorphism may partly explain this genetic component. Though, no specific polymorphisms have presently been firmly linked to alcoholic liver disease (concordance rates for alcohol related cirrhosis are three times higher in momozygotic twins than in dizygotic twins) (O'shea et al., 2010; Mandayam et al., 2004).
- **Iron overload (**Hemochromatosis**).**
- **Diet**: Malnutrition, particularly vitamin A and E deficiencies, can worsen alcohol-induced liver damage by preventing regeneration of hepatocytes. This is particularly a concern as alcoholics are usually malnourished because of a poor diet, anorexia, and encephalopathy (Menon et al., 2001)
- **Obesity**

According to our literature following are the causes of Su'mizaj haar

- Intake of hot foods and drinks.
- Medications.
- Putrefaction.
- Vigrous physical and mental exercise.
- Occupation which produces heat, anger, mild worry (Ibn Sina, 2010).

Pathogenesis

Fatty change, or steatosis is the accumulation of fatty acids in liver cells. Alcoholism causes development of large fatty globules (macro vesicular steatosis) all through the liver and can start to occur after a few days of heavy drinking (Inaba et al., 2004). Alcohol is metabolized by alcohol dehydrogenase (ADH) intoacetaldehyde, then additionally metabolized by aldehyde dehydrogenase (ALDH) into acetic acid, which is ultimately oxidized into carbon dioxide (CO_2) and water (H_2O) (Goldman and Ausiello, 2008). This process generates NADH, and increases the NADH/NAD+ ratio. An elevated NADH concentration provokes fatty acid synthesis while a diminished NAD level results in decreased fatty acid oxidation. Subsequently, the higher levels of fatty acids signal the liver cells to compound it to glycerol to form triglycerides. These triglycerides accumulate, resulting in fatty liver (NIAAA, 1993).

Alcoholic hepatitis

Alcoholic hepatitis is characterized by the inflammation of hepatocytes. Between 10% and 35% of heavy drinkers exhibit alcoholic hepatitis (NIAAA, 1993).

Cirrhosis

Cirrhosis is a late stage of serious liver disease marked
by inflammation (swelling), fibrosis (cellular hardening) and damaged membranes inhibiting detoxification of chemicals in the body, ending in scarring and necrosis (cell death). Between 10% to 20% of heavy drinkers will produce cirrhosis of the liver (NIAAA, 1993). Acetaldehyde may be responsible for alcohol-induced fibrosis by stimulating collagen deposition by hepatic stellate cells. Symptoms include jaundice (yellowing), liver enlargement, and pain and tenderness from the structural changes in damaged liver architecture. Without total abstinence from alcohol use, cirrhosis will finally lead to liver failure. Late complications of cirrhosis or liver failure include portal hypertension (high blood pressure in the portal vein due to the increased flow resistance through the damaged liver), coagulation disorders (due to impaired production of coagulation factors), ascites (heavy abdominal swelling due to buildup of fluids in the tissues) and other complications, including hepatic encephalopathy and the hepatorenal syndrome (Leewen et al., 2000). Fatty change and alcoholic hepatitis with abstinence can be reversible. The later stages of fibrosis and cirrhosis tend to be irreversible, but can usually be contained with abstinence for long periods of time.

Unani concept

Alcohol disturb the mizaj of liver,as it is highly absorbable, its excess heat directly affects and increases the hararat of liver resulting in weakening of hepatic faculties (Ibn sina, 2010, tabri AHAM, 997). Futhermore, Ghaleez sharab (concentrated

wine) sweet one is capable of causing hararat and sudda in liver. Since, liver has high affinity towards alcohol so it is directly absorbed, causing congestion in the narrow canaliculi which ultimately alters its mizaj (Qamri, 2008; Akbar, YNM).

Clinical manifestation of patients with AFL and most patients with mild/moderate alcoholic hepatitis are usually asymptomatic. Some experiences vague symptoms like

- Anorexia.
- Nausea,vomiting.
- Hiccup.
- Malaise.
- Weakness.
- Abdominal pain.
- Icterus.
- Weight loss.
- Tender hepatomegaly.
- Fever.
- Excessive thirst
- Diarrhoea
- Splenomegaly (Walker, 2014; Munjal et al., 2012; Longo, 2012; Kumar et al., 2009).

Clinical features

- Less desire for thirst.
- Anorexia.
- Dyspepsia.
- Irregular bowel habits.
- Dull ache in hepatic region after food reaches to liver.
- Dull face.
- Pallor of tongue and lips (Ibn Sina, 2010; Razi, 2000; Khan, 2011; Baghdadi, 2004; Akbar, YNM).

Nonalcoholic Fatty Liver (NAFL)

NAFLD is a progressive complex of liver disease which starts with fat accumulation in the liver without excessive alcohol consumption. It is strongly associated with metabolic syndrome (obesity+insulin resistence+ dyslipidemia) (Anstee and Day, 2013). In fact it is considered as a manifestation of the metabolic syndrome. It develops when the liver has difficulty breaking down fats, which causes a buildup in the liver tissue. The cause is not related to alcohol. NAFL is diagnosed when more than 10 percent of the liver is fat (Kumar et al., 2005).

Non alcoholic fatty liver disease can be divided into isolated fatty liver in which there is only accumulation of fat, and non alcoholic steatohepatitis (NASH) in which there is fat, inflammation, and damage to liver cells. In both isolated fatty liver and NASH there is an abnormal amount of fat in the liver cells, but, in addition, in NASH there is inflammation within the liver, and, as a result, the liver cells are damaged, they die, and are replaced by scar tissue (Kumar et al., 2005).

Risk factors

Fatty liver (FL) is commonly associated with metabolic
syndrome (diabetes, hypertension, obesity,
and dyslipidemia), but can also be due to any one of many causes (Anstee and Day, 2013).

- Genetic inheritance.
- Rapid weight loss.
- Side effect of certain medications, including aspirin, steroids, tamoxifen, tetracycline etc.
- Acute fatty liver of pregnancy.
- Malnutrition.
- Inflammatory bowel disease.
- HIV, hepatitis C (especially genotype 3), alpha 1-antitrypsin deficiency (Loomba and Sanyal, 2013)

If left untreated, NASH can progress to permanent scarring of the liver and eventual liver failure. The prevalence of NASH is 2-6% in the general population. Up to 20% of adults with NASH develop cirrhosis and up to 11% may experience liver-related deaths.

Most of the patients with NAFLD are asymptomatic (Walker et al., 2014; Leeuwen et al., 2000; Munjal et al., 2012; Kumar et al., 2005; Goldman and Ausiello, 2008; Longo et al., 2012; Kelley et al., 1989). Diagnosis most often follows incidental detection of raised liver enzymes or fatty liver on ultrasound. Some experience dyspepsia, malaise, fatigue vague right upper quadrant discomfort (Walker et al., 2014; Munjal et al., 2012; Longo et al., 2012).

According to our concept causes of Su'mizaj barid are

- Excess food and drink.

- Marked reduction in food.
- Intake of cold food, drinks.
- Excessive repose leading to suppression of innate heat.
- Excessive activity leading to dispersion of innate heat.
- Undue retention of fuzlaat (morbid matter).
- Obstruction from the accumulation of fuzla.
- Occupation which produces cold.
- Excessive worry, joy, pleasure, fear and anxiety (Ibn Sina, 2010; Ibn Rushd, 1987; Shah, 2007; Nafees, YNM).

Pathogenesis

The most important factor for causing NAFD appears to be presence obesity and diabetes. Usually fat tissue were inert and they only serve the function of storage of fat but when large amount of fat is present as in case of obesity fat cells become metabolically active (actually inflamed) and starts producing and releasing some proteins and enzymes into the blood that have effect on cells throughout the body (Kumar et al., 2005). One of the many effect is to promote insulin resistance in cells, in which the cells do not respond to insulin, which is the major promoter of glucose uptake from the blood by cells. As, a result not enough sugar enters the cells, and it begins to accumulate in the blood, a state called diabetes. In addition ,to releasing hormones and proteins, fats cells also release some fat that is stored in them in the form of fatty acids. Liver cells, like other cells of body become insulin resistance, and their metabolic processes, including handling of fat become altered and liver cells starts uptake of fatty acids from the blood. Within the liver cells, fatty acids are changed into storage fat and the fat accumulates. At the same time, the ability of liver to dispose of or export the accumulated fat is reduced. So, liver itself continues to produce fat and to receive fat from the diet leading to fat accumulation in greater amount (Walker et al., 2014).

Unani concept

Due to the causes mentioned above the normal mizaj of liver is transformed to barid, thus allowing deposition of fat causing Zofe Kabid (hepatic impairement) (Ibn Sina, 2010; Ibn Rushd,1987; Razi, 2000; Ibn Zuhr, 1986; Tabri, 1997; Jurjani, 2010; Qamri, 2008). In addition, to this sudda formation in the liver due to accumulation of morbid matter disturbs the mizaj of liver resulting in weakening of hepatic faculties (Razi 2000; Ibn Sina, 2010).

Clinical features: that are illustrated in our literature includes

- Feeling of uncomfortable heat.
- Excessive thirst.
- Bitter taste.
- Anorexia.
- Vomiting.
- Diarrhoea.
- Itching and heaviness at right hypochondrium.
- Inability to sleep on the right side (Qamri 2008; Razi 2000; Khan 2011; Baghdadi 2004; Akbar, YNM).

How Is Fatty Liver Diagnosed

1. **Physical Examination**
2. **Blood Tests:** Liver enzymes are higher than normal but this doesn't confirm a diagnosis of fatty liver. Further analysis is necessary to find the cause of the inflammation
3. **Ultrasound:** The fat on your liver will show up as a white area on the ultrasound image. Other imaging studies may also be done, such as CT or MRI scans. Imaging studies can detect fat in the liver, but they cannot help to confirm any further damage.
4. **Liver Biopsy:** This is the only way to know for certain if you have fatty liver. The biopsy will also help in determining the exact cause. No medical imagery, however, is able to distinguish simple steatosis from advanced NASH.

Complications: includes Non-alcoholic steato-hepatitis (NASH), Alcoholic hepatitis, fibrosis, cirrhosis, ascitis, hepatocellular carcinoma (Walker, 2014; Munjal et al., 2012; Longo et al., 2012; Feldman et al., 1998).

Unani concept

- Zofe kabid (hepatic impairment)
- Waja ul kabid (hepatalgia)
- Su'lqinya (Anaemia)
- Istisqa (Ascitia) (Ibn Sina, 2010).

How Is Fatty Liver Treated

- In modern system of medicine the role of pharmacological therapy is continued to be investigational.
- Currently, there is no medication proven to effectively treat. If the main causes are related to obesity, diabetes and dyslipidemia. The treatment is based in lifestyle modification, weight loss, and physical activity in order to reduce the amount of fat in the liver (Sargent, 2009). Patients who are obese are advised to achieve a gradual and sustained weight loss through proper nutrition and exercise. The weight loss should be around 5-10% of body weight to reduce steatosis and above 10% to improve the inflammation in patients with NASH (Anstee, 2013).
- Patients with diabetes and high lipids in their blood have to improve their sugar control and lower lipids levels. Usually, a low fat, low calorie diet is recommended along with insulin or medications to lower blood sugar in people with diabetes (Takei, 2013).
- Fatty liver disease can also be reversed by reducing or eliminating fatty foods and foods high in sugar from your diet. Choose healthier foods like fresh fruits, vegetables, and whole grains. Replace red meats with lean animal proteins like chicken and fish (Anstee, 2013).

Can fatty liver disease be prevented?

Protecting your liver is one of the best ways to prevent fatty liver. By choosing a healthy life style, you may prevent obesity - **the number one reason for fatty liver disease**. If you choose to drink alcoholic beverages, do so in moderation. According to the Centers for Disease Control and Prevention (CDC), "moderate alcohol consumption is defined as having up to one drink per day for women and up to two drinks per day for men."

Follow your doctor's instructions, and take medications for diabetes or high cholesterol as directed. Additionally, aim for at least 30 minutes of exercise most days of the week to maintain a healthy body.

The following are some suggestions for preventing fatty liver disease (Loomba, 2013):

- Choose to lead a healthy lifestyle.
- If you are overweight, strive for a gradual and sustained weight loss.
- Eat a well-balanced diet that is low in saturated fats and high in fibre.
- Minimize sugar consumption, reduce the intake of fried food
- Introduce exercise into your routine, at least four times a week. You can enjoy walking, swimming, gardening, stretching.
- Avoid alcohol.

Treatment offered by Unani system of Medicine

Usool-e-ilaj (Principles of management)

- First of all focus is strengthening of hepatic faculties because health is entirely dependent on normal functioning of liver (Khan, 2011; Jurjani 2010).
- According to Ibn sina all the measures which are part of ilaj bil zid (counteractive treatment) if adopted may restore tabai mizaj of liver (Ibn Sina 2010; Baghdad 2004).
- Simple heat/cold producing measures has to be adoptedfor normalizing the deranged mizaj by suppressing or stimulating innate heat through diet,drugs possessing Mubarrid (Refrigerant), Musakkin (Calorifacient) property besides Muattir (Aromatic), Muhallil (anti-inflammatory), Mufatteh sudad (Deobstruent), Muqawwi (tonic) and Mushtahi (Appetizer) properties. (Ibn Sina, 2010; Tabri 1997; Khan 2011; Baghdadi 2004; Ibn Baitar 2000; Said 1997).
- It is recommended to use either purgative or diuretics depending on the site of pathology in liver. If it is on concave side (Inferior surface) purgative and light muhallilat are adviced such as Bekh kasni, Mako khushk. If it on convex side (Superior surface) of liver, diuretics are advocated such as Sikanjabeen, Aab kasni, Aab mako,Aab anarain (Ibn Rushd 1987; Razi 2000; Baghdadi 2004; Akbar YNM; Abbas, 2010).

Ilaj (treatment): It comprises of three components

1. **Ilaj bil Ghiza (Dietotherapy)**
    - Diet play an important role in the management of fatty liver as erratic dietary habit is one of the major causative factor.

- Both starvation and excessive food intake leads to Su'mizaj barid, hence balanced food intake is adviced.
- Avoid oily, fatty, spicy fried and hardly digestible food.
- Light and easily digestable diet is recommended for patients with liver affections such as small bird soup, chicken soup, pulses, sagodana kheer (*Metroxylan Sago* gruel), Daliya (Wheat gruel), Kishneez (*Coriandrum sativum*,Linn.), Pudina (*Mentha piperita*) etc. (Ibn Sina 2010; Razi 2000; Khan 2011; Jurjani 2010).

2. Ilaj bit Tadbeer (Regeminal therapy)

- Riyazat (excercise) in the form of brisk running is highly recommended as it reduces body mass (Qamri, 2008).
- Dalak (Massage) with cold or hot oils over hepatic region e.g. Roghan Afsanteen, Roghan Baboona etc.
- Zimad over hepatic region with Zimad jalinoos, Zimad sumbul tib etc. (Razi 2000; Akbar YNM; Said, 1997; Abbas, 2010; Arzani 1998).
- Hamman (Steam bath) are also adviced (Ibn Sina 2010; Tabri 1997; Khan 2011; Jurjani 2010; Baghdadi, 2004)

3. Ilaj bid Dawa (Pharmacotherapy)

- Jalinoos an eminenient scholar has advocated a Majoon for liver disorders comprises of Zafran (4.5 gm), Maweez munaqqa (4.5 gm), Charita sheerin (9 gm), Muqil (11.25 gm), Daarcini (4.5 gm), Saleekha (2.25 gm), Balchad (13.5 gm), Murmukhi (15 gm), hab-darkht batm (9 gm), Shahed (60 gm).
- Pills (3.5-4.5 gm) made up of Mastagi (10.5 gm), Balchad (10.5 gm), Asaroon (10.5 gm), Luk magsool (10.5 gm), Gul e surkh (14 gm), Zeera (14 gm), Anisoon (7 gm), Usar e ghafis (3.5 gm), Afsanteen (3.5 gm), Rewand chini (3.5 gm), Zafran (3 gm) are advised in case of hepatic pain due to Su'mizaj barid.
- For relieving pain Zimadat (ointment) for local application are very effective, which help in altering mizaj along with their analgesic property. So, for this purpose Radeaat (Divergents) and Muhallilat (Anti-inflammatory) are used in ointment such as ood, Zafran (Ibn Sina 2010; Razi 2000; Akbar YNM).
- Different compound drugs mentioned in classical Unani text for liver ailments are Dawaul Kurkum, Dawaul Luk, Dawaai Asanasia, Dawai khubsul hadid, Qurs afsanteen, Qurs Qust, Qurs Rewand, Qurs Luk, Sikanjabeen, Jawarish Jalinoos, Majoon Dabeedulward, Dawaul misk moatadil, Khameera marwarid, Majoon Afsanteen, Zimad jalinoos, Zimad sumbul tib, Roghan afsanteen (Ibn Sina 2010; Razi 2000; Said 1997; Abbas Almajoosi 2010; Arzani 1998).

Preventive measures documented in our literature are

- Avoid intake of food before previous meal getting digest.
- Sudden intake of cold water on empty stomach or after bath, coitus or after exercise.
- All types of oily substances.
- Avoid high calorie food (Ibn Sina 2010; Khan 2011; Jurjani 2010).

What Is the Long-Term Outlook for Fatty Liver

Fortunately, many cases of fatty liver don't develop into liver disease. The liver can repair itself, so if you take the necessary steps to treat high cholesterol, diabetes, or obesity, you can reverse your fatty liver. If you're a heavy drinker, stopping drinking may heal your liver completely.

CONCLUSION

Despite of incredible advancement in Modern field, the role of pharmacotherapy in Fatty liver remains investigational and is not recommended for routine clinical practices. In contrast, the approach by Unani scholars in addition to an array of single and compound formulations amply testifies that this disease can managed successfully which is feasible,

cost effective and free from adverse effects. The present review highlighted the treasure of Unani classical literature about Tashhamul Kabid, which closely resembles Fatty Liver Disease (FLD). Hence, time has reached that these regimens must be explored for it efficacy on scientific parameter.

REFERENCES

Abbas Almajoosi AB.Kamilussana.Vol.2 (part-2). New Delhi, India: CCRUM; 2010.

Akbar Arzani. Tibbe Akbar. Vol. 2. Deoband: Faisal publications, YNM.

Angulo P. Non0alcoholic fatty liver disease. New England Journal of Medicine. 2002; 346 (16): 1221-31.

Anstee QM, Day CP. The genetics of NAFLD. Nature Reviews Gastroenterology & Hepatology. 2013; 10(11): 645-55.

Baghdadi IH. Kitabul Mukhtarat Fit Tib. Vol 3. New Delhi, India: CCRUM; 2004.

Browning JD, Szczepaniak LS, dobbins R et al. Prevelance of hepatic steatosis in an urban population in the United states: impact of ethnicity. Hepatology. 2004; 40: 1387-95.

Chitturi S, Farrell GC, Hashimoto E, Saibar T, Lau GK, Sollano JD. Non-alcoholic fatty liver disease in the Asia Pacific region: definitions and overview of proposed guidelines. Journal of Gastroenterology and Hepatology. 2007; 22(6): 778-87.

Crowley LV. An introduction to human disease-pathology and pathophysiology correlations. 7th ed. USA: Bartlett Publishers; 2007.

Feldman M, Scharschmidt BF, Sleisenger MH. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. 6th ed. Vol. 2. New Delhi, India: WB Saunders Company; 1998.

Goldman,l and Ausiello D. Cecil medicine 24th ed. Philidelphia: Saunders Elseveir; 2008.

Ibn Baitar. Aljamiul mufradat al adviya wal agziya.Vol. 1. New Delhi, India: CCRUM; 2000.

Ibn Rushd. Kitabul Kulliyat (Urdu translation by CCRUM) New Delhi, India: CCRUM; 1987.

Ibn Sina. Al Qanoon Fit Tib. (Urdu translation by Ghulam Hussain Kantoori). Vol 3. New Delhi, India; Idara Kitabulshifa; 2010.

Ibn Zuhr AM. Kitabul Taiseer. New Delhi, India: CCRUM; 1986.

Jurjani I. Zakheera Khwarzam Shahi Vol. 6. New Delhi, India: Idara Kitabul Shifa; 2010.

Kelley WN et al. Text book of Internal medicine. Vol. 1. USA: JB Lippincott Company; 1989.

Khan MA. Akseer Azam (Urdu Translation by Kabeeruddin M) New Delhi India: Idara Kitabul Shifa; 2011.

Kumar V, Abbas AK, Fausto N. Robbins and Cotran Pathologic Basis of Disease. 7th ed. New Delhi, India: Saunders Elseveir; 2005.

Leewen DJV, Reeders JWAG, Ariyama J. Imaging in hepatobiliary and pancreatic diseases: A practical clinical approach. China: WB Saunders; 2000.

Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J. Harrison's Principles of Internal Medicine. 18th ed USA: McGrew Hill; 2012.

Loomba R, Sanyal AJ. The Global NAFLD Epidemic. Nature Reviews Gastroenterology & Hepatology. 2013; 10(11): 686-90.

Loomba R, Sirlin CB, Schwimmer JB, Lavine JE. Advances in pediatric nonalcoholic fatty liver disease. Hepatology. 2009;50(4):1282-93.

Mandayam S, Jamal MM, Morgan TR. "Epidemiology of alcoholic liver disease". Seminars in Liver Disease. 2004; 24 (3): 217–32.

Menon KV, Gores GJ, Shah VH. "Pathogenesis, diagnosis, and treatment of alcoholic liver disease". Mayo Clinic Proceedings. 2001;76 (10): 1021–9.

Munjal YP et al. API Text book of medicine.9th ed. India: API; 2012.

Naees AB. Kulliyat Nafisi (Urdu translation by Kabeeruddin) New Delhi, India: Idara Kitabulshifa; YNM.

O'Shea RS, Dasarathy S, Mc Cullough AJ. Alcoholic liver disease: AASLD Practice Guidelines. Hepatology. 2010; 51 (1): 307–28.

Ovchinsky N, Lavine JE. A critical appraisal of advances in pediatric nonalcoholic Fatty liver disease. Seminars in Liver Disease. 2012;32(4):317-24.

Qamri AMH. Ghina Muna Ma Tarjuma minhajul Ilaj .1st ed. (Urdu translation) New delhi, India; CCRUM; 2008.

Razi Z. Kitab Al Hawi.Vol.7 New Delhi, India: CCRUM; 2000.

Said M. Hamdard pharmacopoeia of Eastern medicien. New Delhi, India: Sri Satguru Publications; 1997.

Sargent S. Liver diseases: an essential guide for nurses and health care professionals. 1st ed. USA: Wiley Blackwell; 2009.

Shah MH. The General Princilples of Aviceena's Canon of Medicine. New Cdelhi: Idara Kitabul Shifa; 2007.

Tabri AHAM. Moalijate Bukhratia.Vol.3. New Delhi, India: CCRUM; 1997.

Tabri AHR. Firdousul hikmat (translated by H. Md Awwal Shah) New Delhi, India: Idara Kitabul Shifa: 2010.

Walker BR, Colladge NR, Raltson SH, Penman ID. Davidson's Principles and Practice of Medicine. 22nd ed. China: Elsevier; 2014.