

International Journal of Research in AYUSH and Pharmaceutical Sciences

Review Article

REVIEW ON NON-ALCOHOLIC FATTY LIVER DISEASE AND ITS HOMOEOPATHIC TREATMENT

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Keywords: Fatty Liver, Homoeopathy, Nux vomica, Podophyllum, Chelidonium, Lycopodium

ABSTRACT

Non-alcoholic fatty liver disease (NAFLD) is the commonest liver disorder in western industrialized countries (prevalence≈20%), NAFLD represents increase fat in hepatocytes (steatosis) visualized, e.g. on ultrasound that cannot be attributed to other causes (most commonly alcohol so consider NAFLD if Drink male <18u/wk, female <9u). If inflammation is also present (increase LFT, typically increase ALT) = non-alcoholic steatohepatitis (NASH). Rule out other causes of liver disease and check for associated metabolic disorders (obesity, dyslipidemia, diabetes and hypertension). Progression to cirrhosis may occur—biopsy or elastography may be needed. Risk factors for progression are older age; obesity; DM; NASH. Control risk factors; including obesity (bariatric surgery helps). Address cardiovascular risk (commonest cause of death). Avoid alcohol consumption. No drug is of proven Benefit, though vitamin E may improve histology in fibrosis (eg400 iu/d-higher doses Associated with excess mortality). Monitor for complications (NASH, cirrhosis, DM). If cirrhotic, screen for HCC with ultrasound± AFP twice-yearly. There is no standardized treatment for fatty liver. Treating the underlying cause can easily reverse the abnormal changes in the liver, provided, it is early in the disease. Homoeopathic remedies like Arsenic Album, Nux Vomica, Chelidonium, Cardus m, Apocynum, Lycopodium, Sepia, Phosphorous, Digitalis, Bryonia, Helleborus Niger, Ferrum Met, kali Carb, Iris V, Natrum Carb and many other medicines are very helpful in the treatment of fatty liver symptoms.

INTRODUCTION

Fatty liver is the accumulation of triglycerides and other fats in the liver cells. The amount of fatty acid in the liver depends on the balance between the processes of delivery and removal. In some patients, fatty liver may be accompanied by hepatic inflammation and liver cell death (steatohepatitis).^[1]

Potential pathophysiologic mechanisms for fatty liver include the following:

- Decreased mitochondrial fatty acid beta-oxidation.
- Increased endogenous fatty acid synthesis or enhanced delivery of fatty acids to the liver

- Deficient incorporation or export of triglycerides as very low-density lipoprotein (VLDL).

Tripodi et al reported that in nonalcoholic fatty liver disease (NAFLD), a procoagulant imbalance progresses from steatosis to metabolic cirrhosis, which may be caused by an increase in factor VIII and a reduction of protein C. The investigators speculated that this imbalance could play a role in the risk for cardiovascular disease and liver fibrosis, conditions commonly associated with NAFLD.^[2]

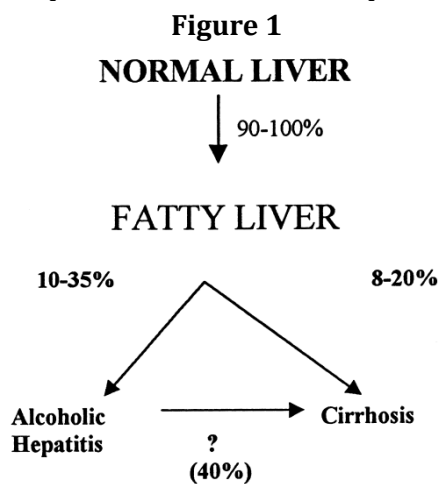
Serum leptin, a cytokine-type peptide hormone mainly produced by adipocytes, may play

an important role in the pathogenesis of steatosis. Steatosis occurs with decreased leptin action, whether due to leptin deficiency or resistance. In patients with alcoholic liver disease, the serum leptin level appears to be independently correlated with the grade of steatosis. [3]

The condition most commonly associated with fatty liver disease is metabolic syndrome. This includes conditions such as type II diabetes, obesity, and hypertriglyceridemia.

Other factors, such as drugs (eg, amiodarone, tamoxifen, methotrexate), alcohol, metabolic abnormalities (eg, galactosemia, glycogen storage diseases, homocystinuria, and tyrosinemia), nutritional status (eg, overnutrition, severe malnutrition, total parenteral nutrition [TPN], or starvation diet), or other health problems (eg, celiac sprue and Wilson disease) may contribute to fatty liver disease.

It has been estimated, as shown in Figure 1, that although 90–100% of heavy drinkers show evidence of fatty liver, only 10–35% develop alcoholic hepatitis and 8–20% develop cirrhosis. [4]



Progression of alcoholic liver disease in heavy drinkers

Several risk factors may influence the development of advanced ALD, including the following:

- Minimum amounts of alcohol intake associated with an increased risk of ALD range from 40 to 80 g/day for 10-12 years; safe limits for alcohol use are not clearly defined. [5]
- Genetics play a role in alcohol consumption and alcoholism; early data suggested a genetic predisposition to the development of ALD, mostly related to differences in major hepatic enzymes involved in the metabolism of alcohol (e.g., alcohol dehydrogenase [ADH], acetaldehyde dehydrogenase [ALDH], and the cytochrome P-450 system [CYP4502E1]). [6]

- Several studies demonstrate a high prevalence of hepatitis C virus (HCV) antibody in patients with ALD, as well as iron overload
- Obesity and dietary habits have been implicated in individual susceptibility to ALD

Stages of Nonalcoholic Fatty Liver Disease (NAFLD) [7]

NAFLD develops in 4 main stages.

Most people will only ever develop the first stage, usually without realizing it.

In a small number of cases, it can progress and eventually lead to liver damage if not detected and managed.

The main stages of NAFLD are:

1. **Simple Fatty Liver (Steatosis)** – a largely harmless build-up of fat in the liver cells that may only be diagnosed during tests carried out for another reason
2. **Non-Alcoholic Steatohepatitis (NASH)** – a more serious form of NAFLD, where the liver has become inflamed; this is estimated to affect up to 5% of the UK population.
3. **Fibrosis** – where persistent inflammation causes scar tissue around the liver and nearby blood vessels, but the liver is still able to function normally.
4. **Cirrhosis** – the most severe stage, occurring after years of inflammation, where the liver shrinks and becomes scarred and lumpy; this damage is permanent and can lead to liver failure (where your liver stops working properly) and liver cancer.

CLINICAL PRESENTATION

Fatty liver occurs commonly after the ingestion of a moderate or large amount of alcohol, even for a short period of time. Alcohol-induced steatosis usually is asymptomatic. Severe fatty infiltration of the liver can result in symptoms of malaise, weakness, anorexia, nausea, and abdominal discomfort. Jaundice is present in 15% of patients admitted to the hospital.

A thorough clinical history, especially with regard to the amount of alcohol consumption, is essential for determining the role of alcohol in the etiology of abnormal liver test results. History obtained from family members may reveal past alcohol-related problems. No specific test is available to rule out drug-related toxicity, but a good review of all concurrent and recent medications, including over-the-counter medications and alternative treatments, is valuable in evaluating the possible causes of abnormal liver test results.

The 2010 American Association for the Study of Liver Diseases (AASLD) practice guideline for alcoholic liver disease (ALD) recommends the following for diagnosis:^[8]

- If alcohol abuse or excess is suspected from discussion of alcohol use with the patient, screen the patient for alcohol abuse using a structured questionnaire such as the Alcohol Use Disorders Identification Test (AUDIT).
- If the patient's history or a screening test indicates alcohol abuse, use laboratory testing to verify the diagnosis of ALD and rule out other considerations.
- If ALD is confirmed, test for other alcohol-related organ damage.

Most patients with nonalcoholic fatty liver disease (NAFLD) are asymptomatic. However, if questioned, more than 50% of patients with fatty liver or nonalcoholic steatohepatitis (NASH) report persistent fatigue, malaise, or upper abdominal discomfort.

Symptoms of liver disease, such as ascites, edema, and jaundice, may arise in patients with cirrhosis due to progressive NASH. Laboratory abnormalities during blood donations or life insurance physical examinations often reveal elevated alanine aminotransferase (ALT) levels and ultimately lead to the diagnosis of fatty liver disease.

Physical Examination

Alcoholic fatty liver may be present in the absence of any abnormalities noted on the physical examination. Hepatomegaly is common in patients who are hospitalized, occurring in over 70% of persons with steatosis proven on biopsy. Portal hypertension is rare in alcoholic steatosis. Extrahepatic effects, such as skeletal muscle wasting, cardiomyopathy, pancreatitis, or peripheral neuropathy, may be present.

Hepatomegaly is also common with NAFLD. Splenomegaly and stigmata of portal hypertension (eg, ascites, edema, spider angiomas, varices, gynecomastia, and menstrual disorders) may occur in patients with cirrhosis. Patients with drug-induced fatty liver may present with rapid fulminant liver failure.^[9]

Complications

Continued alcohol consumption may result in a more advanced form of liver disease, either alcoholic hepatitis or cirrhosis. In a study from Denmark, using a population-based National Registry, investigators noted an increased mortality and an increased cancer risk, particularly liver

cancer, among patients discharged with a diagnosis of alcoholic fatty liver.^[10]

In patients with nonalcoholic fatty liver, steatohepatitis may progress to cirrhosis, with complications that include variceal bleeding, ascites, encephalopathy, and liver failure. The rate of progression appears to be worse if more than 1 liver disease (eg, ALD or chronic viral hepatitis) is present. Uncontrolled diabetes and hypertriglyceridemia also appear to predict worse fibrosis.^[11-14] The rate of formation of hepatocellular carcinoma appears to be the same as with other forms of liver disease,^[15] although NAFLD appears to increase the risk for hepatocellular carcinoma in patients whose livers are not cirrhotic.^[16] NAFLD also appears to be a strong and independent risk factor for prediabetes in the general adult population.^[17]

Differential Diagnosis

The differential diagnosis is broad and includes the following conditions:

▪ Alcoholic Hepatitis	▪ Hepatitis E
▪ Alcoholism	▪ Hepatitis, Viral
▪ Alpha1-Antitrypsin Deficiency	▪ Hyperthyroidism
▪ Autoimmune Hepatitis	▪ Hypothyroidism
▪ Celiac Sprue	▪ Isoniazid Hepatotoxicity
▪ Cirrhosis	▪ Malabsorption
▪ Drug-Induced Hepatotoxicity	▪ Primary Biliary Cirrhosis
▪ Hemochromatosis	▪ Primary Sclerosing Cholangitis
▪ Hepatitis A	▪ Protein-Losing Enteropathy
▪ Hepatitis B	▪ Vitamin A Toxicity
▪ Hepatitis C	▪ Wilson Disease
▪ Hepatitis D	

Steatosis can be observed on histology in the following conditions:

- I. Alcohol excess
- II. Starvation
- III. Total parenteral nutrition (TPN)
- IV. Nonalcoholic steatohepatitis (NASH) – A diagnosis of NASH can be established only when alcohol excess (>10 g/day) can be excluded.
- V. Drug-induced liver disease (eg, disease caused by valproic acid, tetracycline, antiviral agents)

such as zidovudine, amiodarone, perhexiline maleate, methotrexate, corticosteroids, or estrogens).

- VI. Acute fatty liver of pregnancy^[18]– This can occur during pregnancy and likely results from maternal-fetal interactions related to genetic abnormalities in mitochondrial beta-oxidation of fatty acids
- VII. Metabolic liver disease and other inborn errors of metabolism
- VIII. Reye syndrome

LABORATORY STUDIES

Fasting insulin and glucose levels will alert the clinician to potential glucose intolerance and may lead to more effective therapies.

An increase in the levels of glutamyltranspeptidase (GGT) may be related to alcohol use, but this study lacks specificity and sensitivity, and as many as 70% of people who abuse alcohol have normal values.

In rare cases, patients with alcoholic steatosis have severe cholestasis. Ballard et al described 5 patients with alcoholic steatosis presenting with jaundice.^[19] Liver biopsy results in all 5 showed severe steatosis and marked cholestasis with little hepatic fibrosis. Hepatic failure characterized by progressive encephalopathy and coagulopathy developed and led to death in 2 patients. In a large cooperative study of ALD conducted by the Department of Veterans Affairs (VA), histologic cholestasis was observed in only 19% of patients with alcoholic steatosis.

Macrocytosis (increased mean cell volume) is common in patients with alcoholic liver disease (ALD), with a low sensitivity (27-52%) and a high specificity (85-91%). Serum carbohydrate-deficient transferrin (CDT) is a specific and sensitive test for alcoholism in patients whose alcohol intake exceeds 60 g/day.

Hypertriglyceridemia, steatosis, and hemolysis (Zieve syndrome) may be associated with alcohol abuse. Hyperlipidemia may be present in nonalcoholic fatty liver disease (NAFLD). Increased triglycerides are common in children and in patients with metabolic syndrome.

The alkaline phosphatase (ALP) level can be elevated in some patients with nonalcoholic steatohepatitis (NASH). Usually, it is less than twice to 3 times normal.

Abnormal levels of aminotransferases (ie, aspartate aminotransferase [AST] and alanine aminotransferase [ALT]) and bilirubin are found in

about one third of hospitalized patients with alcohol-induced steatosis. In such patients, elevated bilirubin levels largely result from an increase in the indirect reacting fraction and may reflect alcohol-associated hemolysis. AST levels are usually higher than ALT. The absolute values of serum AST and ALT are almost always less than 500 IU/L.

An elevated AST or ALT level may be the only abnormality in patients with fatty liver; these levels may be elevated as much as 10-fold. However, AST and ALT levels may be normal in some patients with fatty liver or NASH. In the absence of cirrhosis, an AST-to-ALT ratio greater than 2 suggests alcohol use, whereas a ratio of less than 1 may occur in patients with NASH.

Viral serologies for hepatitis C should be ordered to identify or exclude viral infection. In addition, iron levels and total iron-binding capacity (TIBC) should be measured, and abnormal results from liver function tests should be evaluated as indicated.

Elevations in serum ferritin or iron, decreased transferrin saturation, or both may occur in patients with NASH. Although iron overload occurs in a small proportion of patients with NASH, these patients have more severe disease. Evidence exists that a serum ferritin greater than 1.5 times the upper limit of the normal range in patients with NAFLD is associated with a higher NAFLD activity score (and thus, NASH) and with advanced hepatic fibrosis.^[20] An iron index score may be ordered on a liver biopsy specimen to evaluate for phlebotomy. Hemochromatosis gene testing is recommended when the ferritin is significantly elevated. Simply eliminating dietary iron has been shown to improve fatty liver.

Autoimmune markers, such as antinuclear antibody (ANA) and anti-smooth muscle antibody (ASMA), are often slightly elevated in NASH. Positive antibodies are associated with more severe fibrosis levels. In the appropriate clinical setting, serum protein electrophoresis (SPEP) and anti-liver-kidney antibody may lead to a diagnosis of autoimmune liver disease.

Often, a clinical picture of obesity, hypertriglyceridemia, and elevated transaminases is enough to allow the clinician to conclude that a patient has NASH. However, underlying alcohol or other drug ingestion, as well as smoldering autoimmune disease or hemochromatosis, must be ruled out. Referral to a hepatologist with or without liver biopsy may help in staging and prognosis.

Serum beta-trophin level may have potential as a new marker for noninvasive

evaluation of NAFLD and liver fibrosis, according to a study by Cengiz et al.^[21] In their cohort of 69 patients with NAFLD and 69 healthy control subjects, serum beta-trophin levels were lower in the NAFLD group; those with mild fibrosis had elevated serum beta-trophin levels compared to those with significant fibrosis. In multivariate and ROC (receiver operating characteristic) analyses, serum beta-trophin was, respectively, an independent predictor of significant fibrosis and was statistically significant in identifying significant fibrosis.^[21]

In a separate study, Abdel-Razik et al proposed mean platelet volume and the neutrophil-lymphocyte ratio as novel inexpensive and simple markers of inflammation to predict fibrosis in patients with NAFLD as well as to predict the presence of NASH. The investigators noted that patients with NASH had elevated levels of mean platelet volume and neutrophil-lymphocyte ratio compared to those without NASH, as well as in patients with advanced fibrosis compared to those with early fibrosis.^[22]

Ultrasonography, CT, and MRI

Noninvasive studies such as Ultrasonography (US), computed tomography (CT), and magnetic resonance imaging (MRI) are useful in helping to establish a diagnosis of steatosis, as well as in finding evidence for portal hypertension; these imaging tests are also helpful in ruling out biliary dilation from other disorders (e.g., Choledocholithiasis) in patients with a cholestatic pattern of liver test result abnormalities.

However, these modalities can neither define the cause of steatosis nor reliably distinguish between benign steatosis and steatohepatitis. Benign steatosis may be focal or diffuse, whereas steatohepatitis is usually diffuse.

In patients with alcoholic steatosis, the liver appears diffusely echogenic on US. In patients with nonalcoholic fatty liver disease (NAFLD), the liver is hyperechogenic or bright. Steatosis is detected only when substantial ($\geq 30\%$) fatty change is present. Studies in patients who are about to undergo gastric bypass surgery indicate that US has a 93% predictive value for NAFLD, with an accuracy of 76%. Patients with steatosis on US have a higher incidence of coronary artery disease and should undergo cardiac evaluation if suspicious symptoms are present.^[23]

The mean CT (Hounsfield unit) count is lower in the liver than in the spleen. CT scans may be used to monitor the course of the disease on successive scans. Focal fatty lesions may be

identified by dual-energy CT scans that demonstrate increased attenuation with increasing energy and no change in normal.

MRI may be useful for excluding fatty infiltration. Phase-contrast imaging correlates with the quantitative assessment of fatty infiltration across the entire range of liver disease. Loss of intensity on T1-weighted images may be useful in identifying focal fat.

Liver Biopsy and Histopathologic Examination

Liver biopsy and histopathologic examination are important components of the diagnostic evaluation in patients with suspected alcoholic liver disease (ALD). They are the most sensitive and specific means of evaluating the degree of liver cell injury and hepatic fibrosis. Several reasons justify obtaining a liver biopsy in patients with ALD, including the following:

- Confirming the diagnosis
- Excluding other unsuspected causes of liver disease
- Assessing the extent of liver damage
- Defining the prognosis

In making the decision on whether to perform a biopsy, it is important to consider the strength of the clinical diagnosis and the role that the biopsy findings would have in guiding therapeutic options. For patients who are unlikely to receive specific treatments or who have conditions that make a biopsy unsafe, the 2010 ALD guideline recommends including procedure risk in the biopsy decision.⁸

A liver biopsy and histopathologic examination are required to establish the diagnosis of nonalcoholic fatty liver disease (NAFLD). The diagnosis should be considered in all patients with unexplained elevations in serum aminotransferases (eg, with findings negative for viral markers or autoantibodies or with no history of alcohol use). The Brunt classification is the standard used to report NAFLD and nonalcoholic steatohepatitis (NASH) biopsy specimens.^[24]

HISTOLOGIC FINDINGS

Histologically, fatty liver is characterized by fat accumulation, which is most prominent in the pericentral (centrilobular) zone. Macrovesicular steatosis is the rule; hepatocytes containing 1 or more large fat droplets displace the nucleus to an eccentric position. Occasional lipid release from rupture of distended hepatocytes may produce a mild localized inflammatory response (lipogranulomas) composed predominantly of macrophages and occasional lymphocytes.

Although infiltration of liver with inflammatory cells typically is not prominent in patients with steatosis alone, in some instances, fibrosis around terminal venules (i.e., perivenular fibrosis) or hepatocytes (ie, pericellular fibrosis) has been noted. Early changes observed with the electron microscope include accumulation of membrane-bound fat droplets, proliferation of smooth endoplasmic reticulum, and gradual distortion of mitochondria. Microvesicular steatosis also is being recognized with increasing frequency.

Alcoholic foamy degeneration (microvesicular fatty change) was the term used by Uchida et al to describe a clinical syndrome in people with chronic alcoholism.^[25] The syndrome is characterized by jaundice and hyperlipidemia and is associated with striking microvesicular steatosis and abundant giant mitochondria observed on liver biopsy.

Specific histologic findings in NAFLD or NASH include the following:

- Steatosis, which usually is macrovesicular but may be microvesicular or mixed
- Inflammatory infiltrates consisting of mixed neutrophilic and mononuclear cells, usually without portal infiltrates (in contrast to hepatitis C)
- Ballooning degeneration
- Fibrosis

The first 3 findings are used to calculate the NAFLD activity score, which is determined on a scale of 0 to 8. The stage of disease is determined by the NAFLD activity score and the amount of fibrosis present.

HOMOEOPATHIC TREATMENT

There are following remedies which are helpful in the treatment of fatty liver symptoms:

Bryonia ^[26,27]

When there are stitching pains in the right hypochondriac region, Bryonia is the first remedy to be thought of, though for these pains we have other remedies, such as Chelidonium and Kali carbonicum. Under Bryonia the liver is swollen, congested and inflamed; the pains in the hypochondriac region are worse from any motion, and better from lying on the right side, which lessens the motion of the parts when breathing. It is one of the chief remedies for jaundice brought on by a fit of anger.

Chelidonium ^[28-30]

Chelidonium is distinguished by the character of the stools. Bryonia is pre-eminently a gastro-hepatic remedy, and has pain in right

shoulder, giddiness, skin and eyes slightly yellow. Hughes says it hardly reaches true hepatitis.

The liver symptoms of Chelidonium are very prominent. There is soreness and stitching pains in the region of the liver, but the keynote for this drug in hepatic diseases is a pain under the angle of the right shoulder blade, which may extend to the chest, stomach, or hypochondrium; there is swelling of the liver, chilliness, fever, jaundice, yellow coated tongue, bitter taste and a craving for acids and sour things, such as pickles and vinegar.

Mercurius. ^[31,26]

This remedy has much sensitiveness and dull pain in the region of the liver; the patient cannot lie on the right side. The liver is enlarged. The skin and conjunctiva are jaundiced. The stools are either clay-colored from absence of bile, or yellowish-green bilious stools passed with a great deal of tenesmus. There is a yellowish white coated tongue which takes the imprint of the teeth and there is a foetid breath, loss of appetite and depression of spirits.

Podophyllum ^[32,33]

The principal use of Podophyllum is in liver affections. Primarily, it induces a large flow of bile, and, secondarily, great torpidity, followed by jaundice. It is indicated in torpid or chronically congested liver, when diarrhea is present. The liver is swollen and sensitive, the face and eyes are yellow and there is a bad taste in the mouth. The tongue is coated white or yellow and the bile may form gall stones.

Digitalis. ^[26,31]

When jaundice arises from cardiac diseases, Digitalis may be the remedy. There is no retention of bile, nor obstruction of the ducts, but the jaundice is due to the fact that the liver does not take from the blood the elements which go to form bile. There is present drowsiness, bitter taste, soreness, enlargement and bruised feeling in the region of the liver.

Myricacerifera ^[34]

Myrica is an important liver remedy. There is first despondency and also jaundice due to imperfect formation of bile in the liver, and not to any obstruction, comparing here with Digitalis. There is dull headache, worse in the morning, the eyes have a dingy, dirty, yellowish hue, the tongue is coated yellow.

Nux vomica ^[35,36]

In liver affections occurring in those who have indulged to excess in alcoholic liquors, highly seasoned food, quinine, or in those who have

abused themselves with purgatives, Nux is the first remedy to be thought of. The liver is swollen hard and sensitive to the touch and pressure of clothing is uncomfortable. The first remedy in cirrhosis of the liver. Colic may be present.

Lycopodium^[37]

Lycopodium acts powerfully on the liver. The region of the liver is sensitive to the touch, and there is a feeling of tension in it, a feeling as if a cord were tied about the waist. Cirrhosis. The pains are dull and aching instead of sharp and lancinating, as under Chelidonium. Fullness in the stomach after eating a small quantity.

Carduus marianus^[31]

This remedy is indicated in jaundice with dull headache, bitter taste, white tongue with red edges, nausea and vomiting of a greenish fluid. There is an uncomfortable fullness in the region of the liver, the stools are bilious and the urine golden yellow; there is sensitiveness in the epigastrium and right hypochondrium. Burnett regards a dark brownish patch over the lower part of the sternum as a useful hint for Carduus, and in such cases he observes that both the liver and heart are at fault. The presence of "liver spot seems to be a special indication for the remedy.

Sulphur^[36,26]

Sulphur is suitable to chronic affections of the liver; it increases the flow of bile and there is much pain and soreness in the liver. Sulphur often completes the cure commenced by Nux. Liver complaints from abuse of mercury will oftentimes call for Sulphur. If the stools are colorless and if much jaundice or ascites be present Sulphur is contra-indicated. Lachesis, however, has jaundice, as do all snake poisons, and is useful in the enlarged livers of drunkards, with tenderness on pressure and throbbing in the right side.

Phosphorus^[38]

Phosphorus is homoeopathic to fatty degeneration of the liver, with well-marked soreness and jaundice. The stools are grayish white. Cirrhosis and atrophy may also call for Phosphorus. The jaundice is indicative of organic diseases, and the remedy is a useful one in malignant diseases of the liver. Digitalis has also been recommended in acute yellow atrophy. Jaundice accompanying pneumonia may also call for Phosphorus.

Taraxacum^[39-41]

This is a decided liver remedy, and the indications are a mapped tongue and a bitter taste in the mouth, chilliness after eating, pain and soreness in the region of the liver and bilious diarrhea. Kali bichromicum also has a mapped

tongue. Yucca filamentosa has a pain going from the upper region of the liver to the back and a bad taste in the mouth. The stools are loose and bilious, accompanied with much flatus.

CONCLUSION

Homeopathy is one of the most popular holistic systems of medicine. The selection of remedy is based upon the theory of individualization and symptoms similarity by using holistic approach. This is the only way through which a state of complete health can be regained by removing all the sign and symptoms from which the patient is suffering. The aim of homeopathy is not only to treat fatty liver symptoms but to address its underlying cause and individual susceptibility. As far as therapeutic medication is concerned, several remedies are available to treat fatty liver symptoms that can be selected on the basis of cause, sensations and modalities of the complaints.

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Cite this article as:

Sayyad Roohullah, Zahid Khan. Review on Non-Alcoholic Fatty Liver Disease and its Homoeopathic Treatment. International Journal of Research in AYUSH and Pharmaceutical Sciences, 2017;1(6):184-192.

Source of support: Nil, Conflict of interest: None Declared

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