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Reviews of non-alcoholic fatty liver disease and its treatment with homoeopathic mother tinctures

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Abstract

Non-alcoholic fatty liver disease (NAFLD) is the most prevalent liver disorder in western industrialized countries, with a prevalence of approximately 20%. NAFLD is characterized by an increase in fat in hepatocytes (steatosis) that is visible on ultrasound and cannot be attributed to other causes. The most common cause of NAFLD is alcohol, so it is important to consider NAFLD if the male consumes less than 18 units per week and the female consumes less than 9 units per week. Non-alcoholic steatohepatitis (NASH) is diagnosed when inflammation is present (increased LFT, typically ALT). Check for associated metabolic disorders (obesity, dyslipidemia, diabetes, and hypertension) and eliminate other causes of liver disease. The development of cirrhosis may necessitate the use of elastography or biopsy. Obesity, diabetes mellitus, NASH, and advanced age are all risk factors for progression. Manage risk factors, such as obesity, with the assistance of bariatric surgery. Address cardiovascular risk (the most prevalent cause of mortality). Refrain from consuming alcohol. Ultrasound± AFP is performed twice annually. Fatty liver does not have a standardized treatment. The abnormal changes in the liver can be readily reversed by treating the underlying cause, provided that the disease is detected early. Arsenic Album, Nux Vomica, Chelidonium, Cardus m, Apocynum, Lycopodium, Sepia, Phosphorous, Digitalis, Bryonia, Helleborus Niger, Ferrum Met, Kali Carb, Iris V, Natrum Carb, and numerous other homoeopathic remedies are highly beneficial in the treatment of fatty liver symptoms.

Keywords: Fatty liver, homoeopathy, mother tincture, podophyllum, chelidonium, carduus marianus

Introduction

The accumulation of triglycerides and other lipids in the liver cells is referred to as fatty liver. The quantity of fatty acids in the liver is contingent upon the equilibrium between the processes of delivery and elimination. Hepatic inflammation and liver cell death (steatohepatitis) may accompany fatty liver in certain patients [1]. Decreased mitochondrial fatty acid betaoxidation and increased endogenous fatty acid synthesis or enhanced delivery of fatty acids to the liver are potential pathophysiologic mechanisms for fatty liver. Inadequate incorporation or export of triglycerides as very low-density lipoprotein (VLDL). Tripodi et al. reported that a procoagulant imbalance in nonalcoholic fatty liver disease (NAFLD) can progress from steatosis to metabolic cirrhosis. This imbalance may be caused by a decrease in protein C and an increase in factor VIII. The investigators hypothesized that this imbalance could contribute to the risk of cardiovascular disease and liver fibrosis, which are frequently associated with NAFLD [2].Metabolic syndrome is the most prevalent condition associated with fatty liver disease. This encompasses conditions such as hypertriglyceridemia, obesity, and type II diabetes. Fatty liver disease may be exacerbated by a variety of factors, including drugs (e.g., amiodarone, tamoxifen, methotrexate), alcohol, metabolic abnormalities (e.g., galactosemia, glycogen storage diseases, homocystinuria, and tyrosinemia), nutritional status (e.g., overnutrition, severe malnutrition, total parenteral nutrition [TPN], or starvation diet), or other health conditions (e.g., celiac disease and Wilson disease). It has been estimated that, despite the fact that 90–100% of frequent consumers exhibit evidence of obese liver, only 10–35% develop alcoholic hepatitis and 8–20% develop cirrhosis, as illustrated in Figure 1. Three The development of advanced ALD may be influenced by a variety of risk factors, such as The safe limits for alcohol use are not clearly defined, and the minimum quantities of alcohol intake associated with an increased risk of ALD range from 40 to 80 g/day for 10-12 years [4].Alcoholism and alcohol consumption are both influenced by genetics. Initial research indicated that there was a genetic predisposition to the development of ALD, primarily due to variations in the major hepatic enzymes involved in the metabolism of alcohol, such as alcohol dehydrogenase [ADH], acetaldehyde dehydrogenase [ALDH], and the cytochrome P-450 system [CYP4502E1]. Five.

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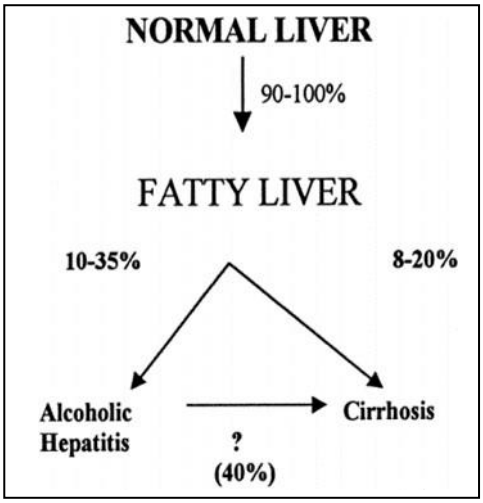

Fig 1: Progression of alcoholic liver disease in heavydrinkers

Stages of Nonalcoholic Fatty Liver Disease (NAFLD)
[6].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 largelyharmless build- up of fat in the liver cells thatmay only be diagnosed during tests carried outfor another reason.

2. Non-Alcoholic Steatohepatitis (NASH): amore 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 is a common occurrence following the consumption of a moderate or large quantity of alcohol, even for a brief period. Alcohol-induced steatosis is typically asymptomatic. Symptoms of malaise, lethargy, anorexia, vertigo, and abdominal distress may be the result of severe fatty infiltration of the liver. In 15% of patients who are admitted to the hospital, jaundice is observed.
It is imperative to obtain a comprehensive clinical history, particularly in relation to the quantity of alcohol consumed, in order to ascertain the role of alcohol in the etiology of aberrant liver test results. Past alcohol-related issues may be disclosed through family history. A comprehensive review of all concurrent and recent medications, including over-the-counter medications and alternative treatments, is useful in assessing the potential causes of aberrant liver test results, although no specific test is available to rule out drug-related toxicity [7].

The majority of patients with nonalcoholic fatty liver disease (NAFLD) are asymptomatic. However, if, questioned, more than 50% of patients with fattyliver 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.

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 followingconditions

- Alcohol excess.
- Starvation.
- Total parenteral nutrition (TPN).
- Nonalcoholic steatohepatitis (NASH) – A diagnosis of NASH can be established only when alcohol excess(>10 g/day) can be excluded.
- Drug-induced liver disease (eg, disease caused by valproic acid, tetracycline, antiviral agentssuch as zidovudine, amiodarone, perhexiline maleate, methotrexate, corticosteroids, or estrogens).
- Acute fatty liver of pregnancy [8]. This can occur during pregnancy and likely results from maternal-fetal interactions related to genetic abnormalities in mitochondrial beta-oxidation of fatty acids.
- Metabolic liver disease and other inborn errors of metabolism.
- Reye syndrome.

Laboratory Studies [9].

Blood tests

- Complete blood count
- Liver enzyme and liver function tests
- Tests for chronic viral hepatitis (hepatitis A, hepatitis Cand others)
- Celiac disease screening test
- Fasting blood sugar
- Hemoglobin A1C, which shows how stable your bloodsugar is
- Lipid profile, which measures blood fats, such as cholesterol and triglycerides

Imaging procedures

Imaging procedures used to diagnose NAFLD include:

- **Abdominal ultrasound,** which is often the initial test

  when liver disease is suspected.
- **Computerized tomography (CT) scanning or magnetic resonance imaging (MRI)** of the abdomen. These techniques lack the ability to distinguish NASH from NAFLD, but still may be used.
- **Transient elastography,** an enhanced form of ultrasound that measures the stiffness of your liver. Liver stiffness indicates fibrosis or scarring.
- **Magnetic resonance elastography,** works by combining MRI imaging with sound waves to create a visual map (elastogram) showing the stiffness of body tissues.

Histologic Findings

Histologically, fatty liver is characterized byfat 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 amild localized inflammatory response (lipogranulomas) composed predominantly of macrophages and occasional lymphocytes. Although infiltration of liver with inflammatory cells typically is not prominent inpatients with steatosis alone, in some instances, fibrosis around terminal venules (i.e., perivenularfibrosis) or hepatocytes (ie, pericellular fibrosis) has been noted. Early changes observed with the electron microscope include accumulation of membrane-bound fat droplets, proliferation ofsmooth endoplasmic reticulum, and gradual distortion of mitochondria. Micro vesicular steatosis also is being recognized with increasing frequency.Alcoholic foamy degeneration (micro vesicular fatty change) was the term used by Uchida *et al*. to describe a clinical syndrome inpeople with chronic alcoholism. [10] The syndrome is characterized by jaundice and hyperlipidemia andis associated with striking microvesicular steatosisand abundant giant mitochondria observed on liverbiopsy.

Specific histologic findings in NAFLD orNASH include the following

- Steatosis, which usually is macrovesicular butmay be microvesicular or mixed.
- Inflammatory infiltrates consisting of mixedneutrophilic and mononuclear cells, usually without portal infiltrates(in contrast tohepatitis 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 mother tinctures for fatty liver treatmentBryonia Alba

Liver region swollen, sore, tensive. Burning pain, stitches; worse pressure, coughing, breathing. Inflammation of the liver. Pains in the live, mostly shooting, tensive or burning. Tractive pains in the hypochondrium, extending to the stomach and the back, in the morning and after dinne, sometimes with vomiting.

Chelidonium Majus

A prominent liver remedy, covering many of the direct reflex symptoms of diseased conditions of that organ. Jaundice due to hepatic and gall bladder obstruction. Liver enlarged [11]. Stitches in liver and spleen. Shooting stitching through liver to back, crampy pain inner angle of scapula.

Right (and left) hypochondrium and scrobiculuss cordis leense and painful on pressure [12]. Constant pain under the lower and iner angle of right scapula. Hepatic diseases; jaundice, pain in right shoulder [13].

Carduus marianus

The action of this drug is centered in the liver and portal system causing soreness, pain, jaundice. Hemorrhages, especially connected with hepatic disease. Dropsical conditions depending on liver disease and when due to pelvic congestion and hepatic disease. Gallstone disease with enlarged liver. Pain in region of liver. Left lobe very sensitive. Hyperaemia of liver, with jaundice [11]. Liver region sensitive to pressure. Pressure, tension and stitches inliver on lying on left side. Swelling, sensitiveness and induration of left lobe of liver, causing by compression respiratory embrassment and caugh with thick expectoration. Liver disease affecting lungs and causing hemoptysis [12].

Ceanothus Americanus

Aneamic patients where liver and spleen are at fault. Pain in liver and back [11]. Immediately after dinner, dull pain in region of liver. Full feeling in region of liver. Pain in liver worse lying on right side [12].

Chelone Glabra

A remedy in liver affections with pain or soreness of the leftlobe of the liver and extending downwards. Dyspepsia with hepatic torpor. Jaundice [11]. Pain or soreness of the left lobe of the liver and extending downwards. Chelone acts in a linebetween the hilus of the liver and fundus of the uterus.Debility from loss of tone of digestive organs or liver or from exhausting diseases [12].

China officinalis

Pain right hypochondrium. Liver and spleen swollen and enlarged. Jaundice [11]. Shooting and pressive pains in the hepatic region, especially when is touched. Hardness and swelling of the liver [12].

Chionanthus Virginica

A prominent liver remedy. Sore; enlarged liver, jaundiceand constipation. Hepatic region tender jaundice with arrest of menses [11]. Uneasy sore feeling in region of right hypochondrium, extending to left iliac region. Uneasy sensations in region of spleen and liver. Hypochondrium of liver. Obstructions of liver in malarious districts. Sorenessin region of liver, quick weak pulse, stools undigested and showing entire absence of bile, urine almost black. Chronic cases of jaundice. Jaundice recurring every summer [12].

Podophyllum Peltatum

Is especially adapted to persons of bilious temperament. It affects chiefly the duodenum, small intestines, liver and rectum. Torpidity of the liver; portal engorgement with a tendency to haemorrhoids, hypogastric pain, fullness of superficial veins, jaundice. Liver region painful, better rubbing part [11]. Fulness in right hypochondrium, with flatulence, pain and soreness. Twisting in right hypochondrium with burning. Stitches in hypochondria, worse while eating. Pain region of liver with inclination to rub the part with the. Excessive secretion of bile, great irritability of liver. Hepatitis with costiveness, tenderness and pain in region of liver [12].

Conclusion

Homeopathy is one of the most widely used holistic medical systems. The holistic approach is employed to determine the appropriate remedy, which is based on the theory of individualization and the similarity of symptoms. This is the sole method by which a patient can regain a state of complete health by eliminating all of the signs and symptoms that are causing them to suffer. Homeopathy is designed to not only alleviate the symptoms of fatty liver but also to address the underlying cause and individual susceptibility. In terms of therapeutic medication, there are numerous options available to alleviate symptoms of fatty liver disease. These remedies can be chosen based on the cause, symptoms, and modalities of the complaints.

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