Homeopathic Management of Non-Alcoholic Fatty Liver Disease

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Abstract- Non-alcoholic fatty liver disease (NAFLD) represents a growing clinical and public health concern owing to its strong association with metabolic dysfunction and its potential for progressive hepatic injury. This narrative review aims to synthesize current biomedical knowledge of NAFLD with homoeopathic clinical perspectives. A critical appraisal of relevant literature was undertaken to summarize evidence related to disease epidemiology, etiological factors, underlying pathophysiological mechanisms, clinical manifestations, diagnostic evaluation, complications, and established management approaches. Particular emphasis is placed on the contributory roles of insulin resistance, disordered lipid metabolism, and chronic inflammatory pathways in disease progression. In addition to conventional lifestyle-based interventions, the review discusses commonly indicated homoeopathic medicines selected on characteristic hepatic symptomatology and supported by repertorial references from standard homoeopathic repertories. By presenting an integrative overview, this article seeks to enhance understanding of NAFLD and support rational, individualized homoeopathic prescribing as part of a holistic management approach.

Index Terms **- Non-alcoholic fatty liver disease, NAFLD, Metabolic dysfunction, Insulin resistance, Homoeopathy, Repertorial analysis.**

I. INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is a rapidly emerging global health concern, characterized by excessive fat accumulation in the liver in individuals who consume little to no alcohol. Once considered rare, its prevalence among both adults and adolescents has risen sharply in recent years, making it one of the most common chronic hepatic disorders, particularly in developing nations. NAFLD is closely associated with a range of metabolic conditions including obesity, type 2 diabetes, dyslipidaemia, and hypertension highlighting its strong link with the growing burden of metabolic syndrome.[1] It represents a broad pathological spectrum, beginning with simple steatosis and extending to the more severe form, steatohepatitis. About 10–20% of affected individuals progress to non-alcoholic steatohepatitis (NASH), a stage marked by inflammation and hepatocellular injury that can ultimately lead to fibrosis, cirrhosis, and even hepatocellular carcinoma. As the incidence of these metabolic disorders increases worldwide, NAFLD has swiftly become one of the fastest-growing chronic liver diseases, underscoring the urgent need for early recognition, prevention, and effective management.

II. EPIDEMIOLOGY

A recent systematic review and meta-analysis documented a significant global rise in NAFLD prevalence, increasing from 25.3% in 1990–2006 to 38.0% in 2016–2019, with notable regional variations highest in Latin America (44.37%), followed by the Middle East and North Africa (36.53%), South Asia (33.83%), Southeast Asia (33.07%), North America (31.20%), East Asia (29.71%), the Asia-Pacific region (28.02%), and Western Europe (25.10%).[2] Within South Asia, India exhibits a particularly wide prevalence range (9–53%) [3,4], influenced by differences in diagnostic methods and the predominance of hospital-based studies. Urban populations consistently demonstrate higher NAFLD rates than rural communities, while high-risk groups such as individuals with type 2 diabetes by 40–80% [5], prediabetes, obesity by 30–90%, and metabolic syndrome show markedly elevated prevalence, reaching up to 56.5%. [6,7] Recent reports of increasing NAFLD among obese Indian children further highlight the escalating public health burden of this disease.

III. AETIOLOGY

Micro vesicular [8] (small fat droplets in hepatocytes)	Macro vesicular [8] (large fat droplets in hepatocytes)
• Acute fatty liver of pregnancy • Reye's syndrome • Jamaican vomiting sickness • Drugs (valproate, tetracycline) • Hepatotoxins (e.g. phosphorus, petrochemicals)	• Alcohol • Diabetes mellitus • Obesity • Lipodystrophy • Dysbetalipoproteinemias • Protein-energy malnutrition • Starvation • Prolonged parenteral nutrition • Jejunoileal bypass • Rapid weight loss • Inflammatory bowel disease • Drugs (methotrexate, vitamin A, glucocorticoids)

IV. PATHOPHYSIOLOGY

It is primarily a consequence of metabolic disturbances, especially insulin resistance, which alters lipid handling in the liver. The abnormal accumulation of triglycerides within hepatocytes is termed *fatty liver* or *hepatic steatosis.* [9] This occurs when fat influx and synthesis exceed the liver's ability to oxidize or export these lipids. Typically, macro-vesicular steatosis predominates, characterized by large fat vacuoles displacing the hepatocyte nucleus.

The pathogenesis progresses through three interconnected pathological steps: [10,11]

Step 1- Steatosis – Fat Accumulation: Insulin resistance increases free fatty acid delivery to the liver, enhances de novo lipogenesis, and impairs mitochondrial fatty acid oxidation. This results in excessive triglyceride storage within hepatocytes. Liver function remains largely preserved and the condition is reversible at this stage.

Step 2 - Steatohepatitis – Inflammation and Hepatocyte Injury: Persistently elevated fat causes oxidative stress and lipid peroxidation, damaging liver cells. Pro-inflammatory cytokines are released, and hepatocyte ballooning occurs, defining the transition to NASH (Non-Alcoholic Steatohepatitis). This represents active liver injury.

Step 3- Fibrosis and Cirrhosis – Scarring and Dysfunction**:** Chronic inflammation activates hepatic stellate cells, leading to scar tissue formation around hepatocytes and vascular structures.

With continued progression, fibrosis advances to cirrhosis, where liver architecture becomes distorted and function declines significantly, increasing the risk of portal hypertension, hepatic failure, and hepatocellular carcinoma (HCC).

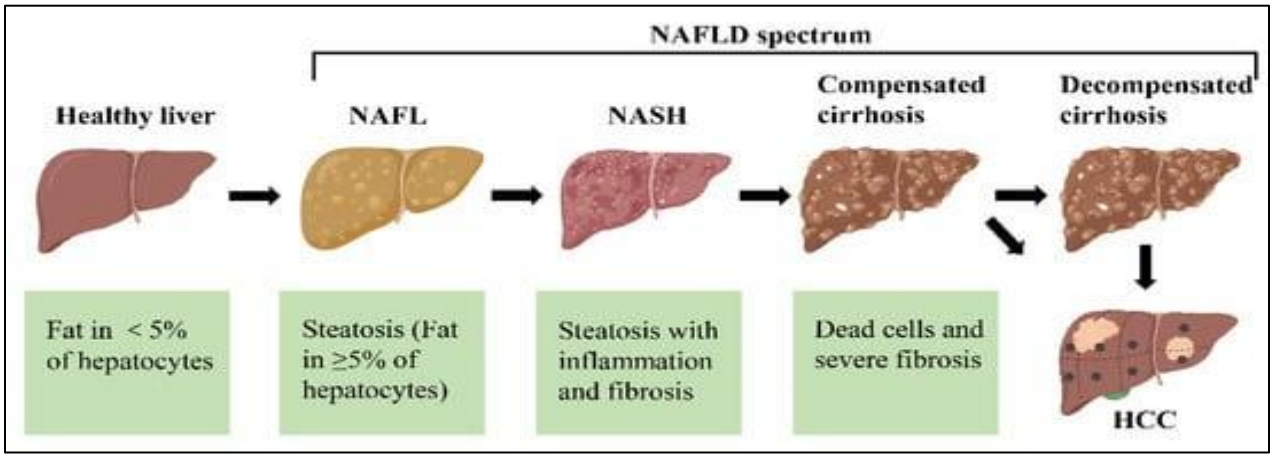

Figure 1 NAFLD advancing through several clinical stages.[11]

V. CLINICAL FEATURES

The symptoms associated with fatty liver disease vary depending on its severity and underlying cause. Many individuals remain asymptomatic, and the condition is often detected incidentally during routine investigations. On examination, the liver may appear enlarged, firm, and usually non-tender, although mild discomfort can occasionally be present. Rapid fat accumulation (e.g., due to toxins or over nutrition) may cause noticeable liver tenderness from stretching of Glisson's capsule.[12]

Although mild fatty liver rarely causes noticeable symptoms, some patients may experience vague and non-specific complaints, including: [13]

- Persistent fatigue or malaise
- A sense of abdominal fullness or heaviness, especially in the right upper abdomen
- Occasional discomfort or dull pain over the liver

If the disease progresses untreated, it may lead to cirrhosis and liver failure. In advanced stages, symptoms become more pronounced and may include: [13,14]

- Jaundice with dark urine
- Unintentional weight loss
- Nausea or diarrhoea
- Loss of appetite
- Ascites causing abdominal distension

VI. COMPLICATIONS [15]

- Progression to Non-Alcoholic Steatohepatitis (NASH)
- Fibrosis and Cirrhosis leading to architectural distortion of the liver
- Major cirrhosis-related complications include Variceal bleeding, Ascites, Hepatic encephalopathy, Liver failure.
- In Hepatocellular carcinoma (HCC) Risk increased in both alcoholic and non-alcoholic fatty liver. Can occur even without cirrhosis in NAFLD
- Higher mortality and increased cancer risk in alcoholic fatty liver disease
- Faster progression when combined with other liver insults (e.g., alcohol, chronic viral hepatitis)
- Metabolic risk factors worsen fibrosis (e.g., uncontrolled diabetes, hypertriglyceridemia)
- Increased cardiovascular risk due to metabolic syndrome association
- Higher likelihood of prediabetes and type 2 diabetes in NAFLD.

VII. EVALUATION

- Complete blood count (CBC)
- Liver function tests, including ALT and AST (may be mildly elevated or normal)
- Fasting blood glucose and HbA1c for assessing long-term glucose control.
- Lipid profile to evaluate cholesterol and triglycerides.
- Screening for viral hepatitis and celiac disease when indicated.
- Gold Standard tests are Liver biopsy – definitive method to confirm NAFLD and determine the extent of inflammation and fibrosis.

Imaging Studies:

- Abdominal ultrasonography – first-line tool for detecting liver fat; most effective in moderate to severe steatosis.
- CT scan or MRI – provide detailed images but cannot distinguish NASH from simple steatosis.
- Magnetic resonance elastography (MRE) – combines MRI and sound waves to create a map of tissue stiffness.

VIII. MANAGEMENT

- Lifestyle modification is the first-line and most effective approach for managing Non-Alcoholic Fatty Liver Disease (NAFLD). Evidence supports that sustainable changes in diet, physical activity, and metabolic control can significantly reduce hepatic steatosis and prevent progression to NASH and fibrosis.
- Weight Reduction: It is the strongest predictor of improvement in NAFLD. A reduction of 7–10% of current body weight leads to significant improvement in liver fat and inflammation. Gradual weight loss (0.5–1.5 kg per week) is recommended to avoid worsening hepatic injury. [16]
- Dietary Modifications: Adopting a balanced, calorie-appropriate diet with emphasis on: Green leafy and cruciferous vegetables (spinach, broccoli, kale, cabbage) for antioxidant and anti-inflammatory benefits. High-fibre foods,

especially whole grains and oatmeal to reduce triglycerides and support gut health. Plant proteins such as soybeans, legumes, and chickpeas for better metabolic balance. Healthy fats including Omega-3 fatty acids from fish, flaxseeds, and walnuts.

- Foods to Avoid or Limit include: Alcohol (even moderate intake worsens NAFLD), Added sugars and high-fructose corn syrup, Sugary beverages and carbonated drinks, Fried foods and processed snacks, Red and processed meats, Refined carbohydrates (white bread, pasta, pastries, pizza), Excessive fruit juices, due to high fructose load.
- Physical Activity: Regular exercise improves insulin sensitivity and reduces hepatic fat independent of weight loss. Recommendations are at least 150–300 minutes/week of moderate-intensity aerobic exercise. Muscle-strengthening activities 2–3 times per week.
- Management of Metabolic Risk Factors: Control of associated metabolic disorders is crucial. Diabetes – improves hepatic fat and inflammation. Hypertension and dyslipidaemia; reduces cardiovascular risk, which is a major cause of mortality in NAFLD.
- Screening and Follow-Up: High-risk individuals are those who are obese, diabetic, or have metabolic syndrome. They should undergo periodic liver function assessment and imaging to monitor disease progression.

IX. HOMOEOPATHIC THERAPEUTICS [17,18,19,20]

1. **Bryonia alba** is indicated in liver conditions where the right upper abdomen feels enlarged, tense, and sore to the touch. Pain is typically sharp, stitching, or burning in nature, and is aggravated by movement such as coughing, deep breathing, or applying pressure. Hepatic inflammation is often present. Discomfort may extend toward the stomach or back, particularly noticeable in the morning or following meals, and may be accompanied by nausea or vomiting.
2. Chelidonium majus – It is a prominent hepatic remedy. It is characterized by soreness and stitching pains in the hepatic region, with its keynote being a pain under the angle of the right shoulder blade, often radiating to the chest, stomach, or hypochondrium. Associated features include liver swelling, chilliness, fever, jaundice, yellow-coated tongue, bitter taste, and a notable craving for acids and sour things such as pickles and vinegar. These well-defined symptoms make Chelidonium a reliable remedy in various hepatic disorders.
3. Cardus marianus – It acts chiefly on the liver and portal system, producing soreness, pain, fullness, and jaundice, with moist skin and marked hyperaemia of the liver. It is indicated in dropsical conditions arising from liver disease, pelvic congestion related to hepatic disorders, an enlarged liver, and even gallstone disease. Patients may experience dull headache, bitter taste, white-coated tongue with red edges, nausea, and vomiting of greenish fluid, along with bilious stools and golden-yellow urine. There is notable sensitiveness in the epigastrium and right hypochondrium, while the left lobe of the liver may be extremely tender, leading to pressure, tension, and stitching pains especially worse when lying on the left side. Swelling, induration, and sensitivity of the left hepatic lobe may also cause respiratory discomfort and thick expectoration, linking hepatic pathology with lung involvement, including haemoptysis. A distinctive clinical clue is a dark brownish “liver spot” over the lower sternum, suggesting combined liver and cardiac involvement and serving as a valuable indication for Carduus marianus.
4. Ceanothus americanus – It is suited to anaemic patients in whom the liver and spleen are primarily affected. There is pain in the liver and back, with a dull ache in the hepatic region immediately after meals and a persistent sense of fullness in that area. The liver pain is notably aggravated when lying on the right side.
5. Chelone glabra is particularly useful in liver disorders characterized by tenderness or discomfort in the left lobe, often radiating downward. It is indicated in cases of sluggish liver function with digestive upset and may also aid in conditions associated with jaundice.
6. Chionanthus virginica is useful in liver disorders characterized by a sore, uncomfortable sensation in the right upper abdomen, sometimes spreading toward the left lower abdominal region. Patients

may also feel discomfort around the spleen. It is often indicated in liver congestion seen in individuals from malarial areas. Typical signs include marked tenderness over the liver, a rapid yet weak pulse, pale and undigested stools lacking bile, and dark-colored urine. It has been especially noted in chronic jaundice cases that tend to recur seasonally, particularly during summer.

7. China officinalis is suited to liver disorders with marked discomfort in the right hypochondrium. The liver and spleen may be palpably enlarged, often presenting with jaundice. The hepatic region is sensitive, with shooting or pressure-like pain that becomes worse on touch. Firmness and swelling of the liver are characteristic clinical indicators for this remedy.
8. Podophyllum peltatum – It is primarily indicated in liver affections marked by torpidity or chronic congestion, especially when accompanied by diarrhoea. It first stimulates an excessive flow of bile and later leads to marked liver sluggishness with jaundice. Patients often show a swollen, sensitive liver, yellowing of the face and eyes, a bad taste in the mouth, and a white or yellow-coated tongue, with a tendency toward gall-stone formation. Suited to those of a bilious temperament, the remedy chiefly acts on the duodenum, small intestines, liver, and rectum. Characteristic features include portal engorgement, hypogastric discomfort, fullness of superficial veins, and right hypochondrial fullness with flatulence, pain, and burning or twisting sensations, aggravated by eating. The hepatic pain is often relieved by pressure or massage.
9. Leptandra virginica primarily targets the liver, especially conditions involving hepatic congestion and portal system disturbances that lead to chronic digestive complaints. Patients may experience a persistent dull ache in the right upper abdomen, often radiating toward the back near the gallbladder region. Abdominal colic accompanied by marked gurgling can be present. A key feature guiding its use is the presence of dark, tar-like stools.
10. Myrica cerifera is a key remedy for liver dysfunction marked by a persistent aching pain just beneath the ribs. Individuals needing this remedy often present with a heavily coated yellow-white tongue, unpleasant bitter taste, foul breath, and reduced appetite. The skin may appear jaundiced, and urine becomes dark in color. Excessive drowsiness, extreme fatigue, and thick, sticky salivary secretions are common. Pain may radiate to the area beneath the shoulder blades, further confirming the liver as the primary organ involved.

X. REPERTORIAL EXTRACTS

FROM KENT REPERTORY [21]

[1] ABDOMEN -FATTY degeneration of liver: Chel., lyco., lyss., *merc., phos.*

[2] ABDOMEN –LIVER and region of: Abies-c, abrot., Acon., *aesc., agar*., all-c., aloe, *alum.am-m*., *ant-t., apoc., arg-n., arn., ars*.,ars-i., asaf., aur.,aur-m., *bapt*., bar-c., Bell., Berb., Bry., Bufo, Calc.,*calc-f*., Calc-p., camph., Carb-s., *carb-v,* Card-m*., cham.,* Chel.,*chin., cimx*., cinnb., clem., *cocc., colch., coll., coloc, con.,* Corn*., croc., crot-c., crot-h., cupr.,dig*., dros., dulc., ferr*., fl-ac*.,gels., *graph.,* grind., *hep*., *hydr*., Iod., *iris, kali-b*., Kali-c., *kali-s.,* Lach, *laur*., Lept., Lyc., mag-c., Mag-m., Merc., merc-c., mur-ac., nat-a., nat-c, *nat-m*., Nat-s., Nit-ac., Nux-m., Nux-v., petr., *ph-ac.,* Phos., plat., *plb.,* Podo., *prun., psor*., puls., ran-b., ran-s., rhus-t., ruta, sabad., *sang.*, sel., Sep., sil., spig., Sulph., sul-ac., tab., verat., *zinc.*

[3] ABDOMEN – ENLARGED – Liver: ars., aur-m., Bry., Calc., card-m., chel., Chin., chin-a., chin, con, Dig, ferr., ferr-i., ferr-p., iod., kali-c, kali-s, lach, lact., laur., Lyc., Mag-m., merc., phos, podo., ptel., nat- M., Nat-s., nit-a, nux-v., Sulph.

[4] ABDOMEN – FULLNESS – Hypochondria – right: Aesc, aloe, chel, eup-per., kali-c., nat-m, podo, sang, Thuj.

[5] ABDOMEN – INFLAMMATION- LIVER – Acon, Apis., Ars., Bell, Bry., calc, camph, cham., card-m., Chel, chin, hep, hippoz., kali- c., Lach., Lyc., merc, nat-a, nat-c., Nat-m, nit-s., Nux-v., phos., phyt, Podo, psor., puls.

FROM CLARKE REPERTORY [22]

1. Liver – ABCESS of – Med., Rap., Rhs., Ther.
2. Liver – AFFECTIONS of – Aesc*, Am.m*., *As. i.,* Au. M., Ca. ar., Ca. fl., Crb.s., Crd. m., *Chel.,* Evm., Ev.a., Fag., Gel., *Hep., Hdr.,* Iod., *Iris.,*

K.ca., Kis., Lach., Lct.v., Lau., *Lpt., Mag.m*., Mlr., Mn.s., *Mr.sol.,* My.c., Ost., Phyt., Pch., Pb., Pod., Plp., Pso., Rap., Sac.o., Sel., Stil., Su.x., Trx., Thl., Up., Vic.

3. Liver – DERANGEMENTS of – *Co.c., Lyc*., Sul. [10]
4. Liver – DISEASES of – Bo.la., chne., *Chi.*, Chio., Chist., K.i., Ol.j., Pho.
5. Liver – DISORDERS of – Ab.c., Ac.. l., *Ber., Bry*., chm.u., Cob., Crt.h., *Dio.*, Evo., Fe.pi., *Nux*, Ther.
6. Liver-FATTY- Pi.x.[10]
7. Liver- PAIN in. GRIPING- Sch.
8. Liver- SORENESS- E.pf., snc

FROM BOERICKE REPERTORY [23]

1. LIVER – Affections in general – Abies c.; Aesc.; *Aloe; Am.m;* Ars iod; *Astacus*; Aur.m; *Aur.m.n*; *Berb.v;* Brassica*; Bry*.; *Calc c.; Cardus m.; Ceanoth; Cham.; Chel*.; Chelone.; Chenop.; Chionanth.; Cholest.; *Cinch.;* Cob.; Con.; Corn.c.; Croc.; Crot.; Diosc.; Dolichos.; *Eup. Perf.;* Euonym.; *Ferr.picr.; Hep.;* Hydr.; Iod.; Iodof.; *Iris.; Kali.c.;* Kali iod.; Lach.; Lept.; *Lyc.; Mag.m.;* Mang.s.; Marrub.; *Merc.s.; Myr.; Nat.s.; Nux v.; Phos*.; Pichi.;Plumb.; *Pod.; Ptel*.; Puls.; Querc.; Raph.; Selen.; *Sep*.; Stellar.; *Sul.; Tarax*.; Thlaspi; Uran.; Vanad.; Veron.
2. LIVER - Fatty degeneration – Aur.m.; Chel.; Kali bich.; Phlorid.; *Phos.; Picr. ac.;* Vanad.
3. ABDOMEN – Liver, CONGESTION (hyperemia, fullness, torpidity): Aesc., Aloe, Berb, Bry, Card-m., Cham., Carb-v, Chel, Chin., Hep, Hydr, Iris, Dios., Kali-bi, Kali-c., Kali-m., Lach., Lyc., Merc., Mag.m, Merc-d, Nat-s, Nux-v., Phos., Podo., Ptel., Sep.
4. ABDOMEN – Liver, Enlargement (hypertrophy): Aesc., Agar., Ars., Calc-ar., Card-m., Chel Chin, Chinin-ar., Chion, Coloc., Con., Dig., Ferr-ar., Ferr-i., Glyc., Graph., Iod., Kali-c., Mag-m., Mang-act., Merc., Merc-d, Nat-s., Nur-v, Podo., Sec., Sel., Stel., Tarax, Vip., Zinc.
5. ABDOMEN – Liver, Induration: Abies-c., Ars., Aur., Chin., Con., Fl-ac., Graph., Lyc., Mag- m., Merc., Nux-v., Sil., Tarax., Zinc.
6. ABDOMEN – Liver, Inflammation (perihepatitis, hepatitis): Acon, Act-sp., Ars, Aur, Bry, Cham., Chel, Corn., Hep., Iod., Kali-i., Lach., Merc., Merc-d., Nat-s., Phos., Psor., Sil, Stel., Sulph.

FROM SYNTHESIS REPERTORY [24]

1. ABDOMEN - FATTY DEGENERATION of liver- aur. Calc. Calc-f. Chel. Germ-met. Kali-bi. Kali-s. Lac-d. *Lyc.* Lyss. Mang. Mang-act. Merc. Phlor. Phos. Pic-ac. Vanad.
2. ABDOMEN – ENLARGED – Liver – chronic - Chol. Mang
3. ABDOMEN - INFLAMMATION – Liver – Chronic- Arn. Aur. Bell. Carc. Card-m. Corn. Crot-h. Lach. Lact. Lyc. Mag-m. Merc. Nat-c. Nat-m. Nat-s. Nit-ac. Nux-v. Phos. Phyt. Podo. Psor. Ptel. Ran-s. Sel. Sil. Sulph. Tub.
4. ABDOMEN – STIFFNESS – Liver - sensation of nat-m. phys.
5. ABDOMEN – ABSCESS – Liver- Ars. Bell. Bold. Bry. Bufo chinin-ar. Eberth. Fl-ac. Hep. Kali-c. Lach. Lyc. Med. Merc. Merc-c. Nux-v. Phos. Puls. Pyrog. Raph. Rhus-t. Ruta sep. Sil. Ther. Vip. Yers.
6. ABDOMEN – DISTENTION - Liver - Arn. Bry. Calc. Cham. Chel. Iod. Kali-c. Lyc. Merc. Merc-c. Nit-ac. Phos. Sep. Sil. Sulph. Thuj.
7. ABDOMEN - FULLNESS, sensation of – liver - Acon. Aesc. Aloe apoc. Arg-n. Bell. Berb. Bry. Cean. Chel. Ferr. Gels. Kali-c. Kreos. Lach. Lept. Mang. Myric. Nat-m. Nux-m. Nux-v. Phos. Podo. Ptel. Sang. Sep. Sulph. Thuj.

FROM PHATAK REPERTORY [25]

1. Liver (including right hypochondria):*Aesc*, Aloe, Am-c, Am-m, Ars, Aur, Bar-c, Bell, *Berb, Bry,* Card-m, Chel, Chin, Cocc, Colch, Dios, Gels, Hydr, Iris, Kali-c, Lach, Lept, Lyc, *Mag-m, Merc, Nat-s,* Nit-ac, Nux-m, Nux-v, Phos, *Podo,* Rheum, Sang, *Sep,* Sulph
2. Liver (including right hypochondria): Heavy: Bry, Mag-m, Nat-s, Ph-ac, Ptel
3. Liver (including right hypochondria): Inflammation: Acon, Ars, Bell, Chel, Lyc, *Mag-m,* Nat-s, Nux-v

FROM MURPHY REPERTORY [26]

1. LIVER - DISTENSION, liver region-arn., *bry.,* chel., kali-i., lyc., *merc*., nit-ac., phos., sep., *sil*.
2. LIVER - ENLARGED, liver absin., aesc., agar., aloe, am-m., ant-t., *ars*., ars-i., aur., aur-ar., aur-i.,

aur-m., aur-s., bac., bar-m., *bry.*, bufo, *calc., calc-ar.,* calc-sil., *carb-v.,* carc., CARD-M., CHEL., CHIN., chin-ar., *chion.*, cocc., coloc., *con., crot-h.,* dig., eup-per., *ferr*., ferr-ar., *ferr-i*., ferr-p., *fl-ac.,* foll., glyc., graph., *hep., hippoz*., hydr., *iod.,* kali- br., *kali-c.,* kali-m., kali-s., lach., lact., *laun*, LYC., mag-c., MAG-M., mag-p., mang-acet.,menis., *merc., merc-d.,* merc-i-r., mur-ac., naja, *nat-m.,* NAT-S., *nit-ac., nux-m.,* NUX-V., *phos.*, phyt., plb., *podo.*, ptel., sanic., sec., sel., senn., sep., sil., *stel*., sul-i., *sulph.,* tab., tarax., thuj., *tub.,* urt-u., vip., *zinc*., zinc-p.

3. LIVER - FATTY degeneration, liver- alco., am-van., ant-c., ant-t., arg-n., ars., aur., bism., cadm-s., *calc*., calc-f., carb-ac., carb-v., carbn-chl., *card-m*., chel., chlf., fl-ac., hippoz., iodof., kali-bi., kali-c., kali-chl., kali-s., lac-d., *lyc.,* lyss., mag-m., mang., *merc.,* nat-m., nux-v., phlor., PHOS., *pic-ac.,* sul-ac., sulph., thal., vanad.
4. LIVER - INFLAMMATION, liver – ACON., *act-sp.,* adlu., aesc., alco., aloe, alum., am-c., ambr., anag., anan., ant-c., *ant-t*., apis, arn., ARS., ars-i., asaf., astac., *aur.,* aur-m., bapt., BELL., bol., bry., cael., *calc*., calc-f., *camph*., cann-s., canth., *caps*., CARC., CARD-M., *cham.*, CHEL., chelo., chim., *chin*., cic., cocc., coloc., *corn.,* crot-h., cub., cupr., cupr- acet., dig., dol., eup-per., euph-c., fl-ac., *flor-p.,* graph., grin., guat., *hep., hippoz., hydr.,* ign., iod., kali-ar., kali-bi., *kali-c*., kali-chl., kali-i., kali-n., kali-p., *lach., laur*., lept., LYC., mag-c., *mag-m.,* mand., mang., mang-m., mang-s., med., menis., *merc*., merc-c., merc-d., morg-g., nat-ar., nat-c., *nat-m.,* NAT-S., *nit-ac.,* nit-m-ac., nux-m., NUX-V., ost., petr., *ph-ac.,* PHOS., phyt., plb., *podo., psor., ptel.,* puls., ran-b., ran-s., rob., sang., sarr., SCIRR., sec., sel., sep., sil., solin., *stann.,* staph., stel., sulfa., *sulph.,* tab., tell., verat-v., vip., vip-a., wies
5. LIVER - INDURATION, liver, (see Hard)- abies-c., ammc., anag., ARS., *aur., chel.,* CHIN., cinnam., *con.,* DIG., GRAPH., *fl-ac*., lact., laur., lyc., MAG-M., merc., nux-v., *phos.,* podo., RAT., *sil., sulph., tarax,* zinc.
6. LIVER - STIFFNESS, sensation - nat-m., phys.
7. LIVER - SCLEROSIS, liver - aur.

XI. CONCLUSION

Non-alcoholic fatty liver disease represents a significant and growing global health burden, largely reflecting the widespread increase in metabolic disorders and sedentary lifestyles. Its insidious onset, frequent lack of early symptoms, and potential to progress to advanced liver disease make NAFLD a condition of considerable clinical importance. Understanding the complex interplay between metabolic dysregulation, insulin resistance, lipid accumulation, oxidative stress, and chronic inflammation is essential for effective disease prevention and management. Although lifestyle modification and metabolic risk control remain the cornerstone of conventional management, long-term adherence and individualized response continue to pose challenges. This underscores the need for complementary approaches that address not only hepatic pathology but also the patient's overall metabolic and constitutional state. The homoeopathic system of medicine, with its emphasis on individualized prescribing based on characteristic symptomatology, offers a holistic framework that may complement standard therapeutic strategies. The remedies and repertorial references discussed in this review demonstrate a consistent therapeutic focus on hepatic congestion, fatty degeneration, inflammation, and functional disturbance of the liver. When applied judiciously, homoeopathic medicines may aid in symptom relief, support hepatic function, and contribute to overall metabolic balance, particularly in early and uncomplicated stages of NAFLD. In conclusion, an integrative approach that combines early diagnosis, sustained lifestyle intervention, metabolic risk factor optimization, and individualized homoeopathic management holds promise in addressing the multifaceted nature of NAFLD. Further methodologically robust clinical studies are required to validate integrative treatment outcomes and to define the role of homoeopathy within evidence-based strategies aimed at reducing disease progression, complications, and long-term morbidity.

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XIII. CONFLICTS OF INTEREST

The author has no conflicts of interest to disclose.

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