



POSTPARTUM WELLNESS: RECOGNIZING AND MANAGING FATTY LIVER POTENTIAL

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ABSTRACT

Postpartum women are at risk of developing fatty liver due to hormonal changes, increased insulin resistance, and metabolic shifts. This abstract reviews strategies for recognizing and managing potential fatty liver in the postpartum period. Recognizing symptoms such as fatigue, abdominal pain, and jaundice is crucial for early detection. Lifestyle modifications including dietary changes, exercise, and weight management are essential for prevention and management. Healthcare providers play a pivotal role in educating women about the risks and implementing appropriate interventions to optimize postpartum wellness and prevent long-term complications associated with fatty liver disease.

Keywords: Postpartum, Fatty Liver

INTRODUCTION

The postpartum period, characterized by significant physiological changes and hormonal fluctuations, presents a vulnerable time for women's health. Among the various health concerns during this phase, the risk of developing fatty liver disease has gained attention due to its potential long-term implications. Fatty liver disease, encompassing non-alcoholic fatty liver disease (NAFLD) and its progressive form, non-alcoholic steatohepatitis (NASH), can manifest or worsen postpartum due to metabolic alterations and hormonal shifts. Understanding the factors contributing to postpartum fatty liver development and implementing effective management strategies are essential to safeguard maternal health and prevent complications. This introduction provides an overview of the importance of recognizing and managing potential fatty liver disease in the postpartum period, highlighting the need for proactive healthcare interventions and patient education to promote optimal postpartum wellness.

DEFINITION

Fatty liver disease, also known as hepatic steatosis, is a condition characterized by the accumulation of fat within the liver cells. This excessive accumulation of fat can impair liver function and lead to inflammation, potentially progressing to more severe conditions such as non-alcoholic fatty liver disease (NAFLD) or non-alcoholic steatohepatitis (NASH). NAFLD refers to a spectrum of liver conditions ranging from simple fatty liver to inflammation and liver cell damage (NASH), which can eventually lead to fibrosis, cirrhosis, or liver cancer. Fatty liver disease can occur due to various factors including obesity, insulin resistance, metabolic syndrome, excessive alcohol consumption, certain medications, and rapid weight loss. Early recognition and management are crucial to prevent the progression of fatty liver disease and mitigate its associated risks.

INCIDENCE

The incidence of fatty liver disease in the postpartum period is a growing concern, reflecting broader trends of increasing rates of non-alcoholic fatty liver disease (NAFLD) globally. While specific incidence rates during the postpartum period may vary depending on factors such as demographic characteristics and predisposing conditions, studies suggest that the risk of developing fatty liver increases following childbirth. The hormonal changes, metabolic shifts, and potential weight gain during pregnancy contribute to this heightened risk. Although precise

postpartum incidence rates of fatty liver disease may not be readily available, healthcare providers should remain vigilant in recognizing symptoms and risk factors to implement timely interventions and support strategies for women during this vulnerable period.

CLASSIFICATION

1. Non-alcoholic fatty liver disease (NAFLD) can further be classified based on histological features:

a. Non-alcoholic fatty liver (NAFL): Presence of fat in liver cells without significant inflammation or liver cell damage.

- NAFL represents the initial stage of NAFLD and is characterized by the accumulation of fat in the liver cells (steatosis) without significant inflammation or liver cell damage.

- Individuals with NAFL may have elevated liver enzymes, indicating liver injury, but typically do not exhibit symptoms of liver disease.

b. Non-alcoholic steatohepatitis (NASH): Presence of liver inflammation and liver cell damage along with fat accumulation in liver cells.

NASH is a more severe form of NAFLD characterized by liver inflammation and liver cell damage (hepatocyte injury) in addition to fat accumulation.

- NASH is associated with a higher risk of progression to advanced liver fibrosis, cirrhosis, and liver-related complications such as hepatocellular carcinoma (liver cancer).

c. Fibrosis: Presence of scar tissue in the liver, indicating progression of liver damage.

- Fibrosis refers to the development of scar tissue in the liver as a result of ongoing liver injury and inflammation.

- Liver fibrosis can progress over time and is an important predictor of liver-related morbidity and mortality in individuals with NAFLD.

d. Cirrhosis: Advanced stage of liver fibrosis characterized by extensive scarring, nodules, and impaired liver function.

- Cirrhosis represents the most advanced stage of liver fibrosis, characterized by extensive scarring, nodular regeneration, and impaired liver function.

- Individuals with cirrhosis are at increased risk of liver failure, portal hypertension, variceal bleeding, hepatic encephalopathy, and hepatocellular carcinoma.

ETIOLOGICAL FACTORS

The development and progression of fatty liver disease, particularly non-alcoholic fatty liver disease (NAFLD), are influenced by various etiological factors. Here are some key contributors:

1. **Obesity:** Excess body weight, particularly abdominal obesity, is strongly associated with NAFLD. Adipose tissue dysfunction and increased release of free fatty acids contribute to fat accumulation in the liver.
2. **Insulin Resistance and Metabolic Syndrome:** Insulin resistance, a hallmark of type 2 diabetes and metabolic syndrome, promotes lipolysis in adipose tissue and hepatic de novo lipogenesis, leading to hepatic fat accumulation.
3. **Dietary Factors:** A high intake of saturated fats, fructose, and refined carbohydrates, along with a low intake of fibre, antioxidants, and omega-3 fatty acids, may contribute to NAFLD development.
4. **Physical Inactivity:** A sedentary lifestyle and lack of regular physical activity are risk factors for NAFLD. Exercise has beneficial effects on insulin sensitivity, lipid metabolism, and hepatic fat content.
5. **Genetic Predisposition:** Genetic factors play a role in determining susceptibility to NAFLD. Variants in genes involved in lipid metabolism, insulin signalling, and inflammation may influence disease risk.
6. **Gut Microbiota Dysbiosis:** Alterations in the composition and function of the gut microbiota can contribute to NAFLD pathogenesis through effects on energy metabolism, gut barrier integrity, and inflammation.
7. **Medications:** Certain medications, such as corticosteroids, tamoxifen, methotrexate, and some antiretroviral drugs, can induce or exacerbate fatty liver disease.
8. **Hormonal Factors:** Hormonal changes, such as those occurring during pregnancy or menopause, may influence hepatic lipid metabolism and contribute to the development of fatty liver.
9. **Sleep Disorders:** Obstructive sleep apnoea and other sleep disorders are associated with insulin resistance, dyslipidemia, and NAFLD.
10. **Environmental Factors:** Environmental pollutants, including endocrine-disrupting chemicals and air pollution, have been implicated in NAFLD pathogenesis.

RISK FACTORS

1. **Obesity:** Excess body weight, particularly abdominal obesity, is a significant risk factor for fatty liver disease. Accumulation of fat in the liver is closely associated with obesity and metabolic syndrome.
2. **Insulin Resistance:** Insulin resistance, a condition in which cells do not respond effectively to insulin, is a key risk factor for non-alcoholic fatty liver disease (NAFLD). It leads to increased production of glucose by the liver and promotes fat accumulation in the liver.
3. **Type 2 Diabetes:** People with type 2 diabetes are at a higher risk of developing NAFLD due to insulin resistance and dysregulation of lipid metabolism.
4. **Metabolic Syndrome:** Metabolic syndrome, characterized by a combination of obesity, high blood pressure, high blood sugar, and abnormal lipid levels, significantly increases the risk of fatty liver disease.
5. **High Blood Lipid Levels:** Elevated levels of triglycerides and low-density lipoprotein (LDL) cholesterol, combined with low levels of high-density lipoprotein (HDL) cholesterol, are associated with an increased risk of NAFLD.
6. **Poor Dietary Habits:** Diets high in saturated fats, refined carbohydrates, and added sugars contribute to the development of fatty liver disease. High fructose consumption, in particular, has been linked to hepatic fat accumulation.
7. **Sedentary Lifestyle:** Lack of regular physical activity and sedentary behaviour are risk factors for NAFLD. Exercise helps improve insulin sensitivity, promote weight loss, and reduce liver fat.

PATHOPHYSIOLOGY

The pathophysiology of fatty liver disease, particularly non-alcoholic fatty liver disease (NAFLD), involves complex interactions between metabolic, genetic, environmental, and inflammatory factors.

1. **Fat Accumulation:** The hallmark feature of fatty liver disease is the accumulation of triglycerides within hepatocytes (liver cells). This excessive fat accumulation can result from increased delivery of free fatty acids to the liver from adipose tissue, increased de novo lipogenesis (the synthesis of fatty acids within the liver), and impaired fatty acid oxidation.
2. **Insulin Resistance:** Insulin resistance plays a central role in the development of NAFLD. It leads to hyperinsulinemia (high insulin levels), which promotes lipogenesis and inhibits fatty acid oxidation in the liver. Insulin resistance also contributes to dysregulated glucose

metabolism, leading to increased hepatic glucose production and further exacerbating liver fat accumulation.

3. **Oxidative Stress and Lipotoxicity:** Excess accumulation of fat in the liver can lead to mitochondrial dysfunction and increased production of reactive oxygen species (ROS), resulting in oxidative stress. Lipotoxicity, caused by the accumulation of toxic lipid intermediates such as ceramides and diacylglycerols, contributes to hepatocyte injury and inflammation.
4. **Inflammation:** Hepatic inflammation is a key feature of non-alcoholic steatohepatitis (NASH), the more severe form of NAFLD. Inflammatory cytokines, such as tumour necrosis factor-alpha (TNF- α) and interleukin-6 (IL-6), are released by activated immune cells and contribute to hepatocyte injury, fibrosis, and disease progression.
5. **Hepatic Stellate Cell Activation and Fibrosis:** Persistent liver injury and inflammation lead to the activation of hepatic stellate cells, which are responsible for producing extracellular matrix components such as collagen. This results in the deposition of scar tissue (fibrosis) in the liver parenchyma, impairing liver function and increasing the risk of cirrhosis and liver failure.
6. **Genetic and Epigenetic Factors:** Genetic variants and epigenetic modifications influence individual susceptibility to NAFLD and disease progression. Variants in genes involved in lipid metabolism, insulin signalling, inflammation, and fibrogenesis can impact the risk of developing fatty liver disease and its severity.
7. **Gut-Liver Axis:** Dysbiosis of the gut microbiota and increased intestinal permeability contribute to the pathogenesis of NAFLD through the release of bacterial products, such as lipopolysaccharides (LPS), into the portal circulation. This triggers inflammatory responses in the liver and promotes disease progression.
8. **Environmental Factors:** Environmental factors, including dietary habits, physical activity levels, exposure to endocrine-disrupting chemicals, and socioeconomic status, interact with genetic and metabolic factors to influence the development and progression of fatty liver disease.

CLINICAL MANIFESTATION

1. **Asymptomatic:** In the early stages, fatty liver disease may be asymptomatic and often discovered incidentally during routine health check-ups or imaging studies for unrelated conditions.
2. **Fatigue:** Fatigue is a common symptom reported by individuals with fatty liver disease, which may be attributed to underlying metabolic abnormalities and inflammation.
3. **Abdominal Discomfort:** Some individuals may experience vague discomfort or pain in the upper right abdomen, especially in the region of the liver.
4. **Hepatomegaly:** Enlargement of the liver (hepatomegaly) may be detected on physical examination in some cases, particularly in individuals with more advanced disease.
5. **Elevated Liver Enzymes:** Elevated levels of liver enzymes, such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST), are commonly observed in blood tests and may indicate hepatocellular injury and inflammation.
6. **Jaundice:** In rare cases, advanced fatty liver disease or NASH may progress to liver failure, leading to jaundice (yellowing of the skin and eyes), dark urine, and clay-coloured stools.

COMPLICATIONS

1. **Liver Fibrosis and Cirrhosis:** Persistent inflammation and liver injury can result in the accumulation of scar tissue (fibrosis) in the liver. Over time, progressive fibrosis can lead to cirrhosis, a condition characterized by extensive scarring, nodular regeneration, and impaired liver function.
2. **Portal Hypertension:** Cirrhosis can cause increased pressure in the portal vein (portal hypertension), leading to complications such as ascites (fluid accumulation in the abdominal cavity), varices (enlarged and swollen blood vessels in the oesophagus or stomach), splenomegaly (enlargement of the spleen), and portosystemic shunts.
3. **Hepatic Decompensation:** Advanced cirrhosis may result in hepatic decompensation, where the liver can no longer perform its essential functions adequately. Decompensation may manifest as jaundice, hepatic encephalopathy (altered mental status due to liver dysfunction), coagulopathy (bleeding disorders), and hepatorenal syndrome (kidney dysfunction).
4. **Hepatocellular Carcinoma (HCC):** Individuals with advanced fibrosis/cirrhosis due to fatty liver disease are at increased risk of developing hepatocellular carcinoma, the most common

type of primary liver cancer. HCC often presents at an advanced stage and has a poor prognosis.

5. **Cardiovascular Disease:** Fatty liver disease is associated with an increased risk of cardiovascular events, including coronary artery disease, stroke, and heart failure. Metabolic abnormalities such as obesity, insulin resistance, dyslipidemia, and hypertension contribute to the heightened cardiovascular risk.
6. **Type 2 Diabetes:** Fatty liver disease and insulin resistance are closely intertwined, with each condition exacerbating the other. Individuals with fatty liver disease are at increased risk of developing type 2 diabetes, which further promotes liver fat accumulation and inflammation.

RISK FACTORS

1. **Obesity:** Excess body weight, particularly abdominal obesity, is a major risk factor for fatty liver disease. Fat accumulation in the liver is closely linked to obesity and metabolic syndrome.
2. **Insulin Resistance:** Insulin resistance, a condition in which cells do not respond effectively to insulin, is strongly associated with fatty liver disease. It leads to increased production of glucose by the liver and promotes fat accumulation.
3. **Type 2 Diabetes:** Individuals with type 2 diabetes are at a higher risk of developing fatty liver disease due to insulin resistance and dysregulated glucose metabolism.
4. **Metabolic Syndrome:** Metabolic syndrome, characterized by a combination of obesity, high blood pressure, high blood sugar, and abnormal lipid levels, significantly increases the risk of fatty liver disease.
5. **High Blood Lipid Levels:** Elevated levels of triglycerides and low-density lipoprotein (LDL) cholesterol, combined with low levels of high-density lipoprotein (HDL) cholesterol, are associated with an increased risk of fatty liver disease.
6. **Poor Dietary Habits:** Diets high in saturated fats, refined carbohydrates, and added sugars contribute to the development of fatty liver disease. High fructose consumption, in particular, has been linked to hepatic fat accumulation.
7. **Sedentary Lifestyle:** Lack of regular physical activity and sedentary behaviour are risk factors for fatty liver disease. Exercise helps improve insulin sensitivity, promote weight loss, and reduce liver fat.

DIAGNOSTIC EVALUATION

1. Clinical Assessment:

- A comprehensive medical history and physical examination are conducted to assess risk factors, symptoms, and signs suggestive of fatty liver disease, such as abdominal discomfort, hepatomegaly, or evidence of metabolic syndrome.

2. Laboratory Tests:

- Liver Function Tests (LFTs): Blood tests, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and gamma-glutamyl transferase (GGT), are performed to assess liver function and detect hepatocellular injury and inflammation.
- Lipid Profile: Measurement of serum lipid levels, including total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides, helps assess cardiovascular risk and metabolic abnormalities associated with fatty liver disease.
- Fasting Blood Glucose and Hemoglobin A1c (HbA1c): Evaluation of blood glucose levels and HbA1c helps identify individuals with insulin resistance and diabetes, which are common comorbidities of fatty liver disease.
- Other Tests: Additional tests may include markers of inflammation (e.g., C-reactive protein), markers of fibrosis (e.g., FIB-4 index, FibroTest), and screening for viral hepatitis (e.g., hepatitis B surface antigen, hepatitis C antibody).

3. Imaging Studies:

- Ultrasonography: Ultrasonography is often the initial imaging modality used to evaluate fatty liver disease due to its accessibility, safety, and cost-effectiveness. Ultrasonography can detect hepatic steatosis, hepatomegaly, and signs of advanced liver disease.
- Computed Tomography (CT) Scan and Magnetic Resonance Imaging (MRI): CT and MRI scans provide more detailed imaging of the liver and can help quantify liver fat content and assess for complications such as liver fibrosis and cirrhosis.
- Transient Elastography (FibroScan): Transient elastography measures liver stiffness as a surrogate marker of fibrosis. It is a non-invasive method used to assess liver fibrosis and monitor disease progression over time.

4. Liver Biopsy:

- Liver biopsy may be considered in selected cases, particularly when the diagnosis is uncertain or when there is suspicion of advanced liver disease or NASH. Liver biopsy provides histological evaluation of liver tissue, allowing for grading of steatosis, assessment of inflammation, fibrosis staging, and exclusion of other liver diseases.

5. Non-Invasive Biomarkers:

- Various non-invasive biomarkers and scoring systems have been developed to assess liver fibrosis and predict the presence of advanced fibrosis or cirrhosis in patients with fatty liver disease. Examples include the Fibrosis-4 (FIB-4) index, NAFLD Fibrosis Score (NFS), Enhanced Liver Fibrosis (ELF) test, and FibroTest. These biomarkers incorporate combinations of clinical parameters and laboratory tests to estimate the degree of liver fibrosis without the need for liver biopsy.

Medical Management

1. Lifestyle Modifications:

- **Weight Loss:** Achieving and maintaining a healthy weight through a combination of caloric restriction, dietary modifications, and regular physical activity is the cornerstone of fatty liver disease management. Gradual weight loss of 5-10% of body weight over 6-12 months has been shown to improve liver enzymes, hepatic steatosis, and insulin sensitivity.
- **Diet:** Emphasizing a balanced diet rich in fruits, vegetables, whole grains, lean proteins, and healthy fats while reducing intake of saturated fats, refined carbohydrates, added sugars, and processed foods can help improve metabolic health and reduce liver fat accumulation.
- **Exercise:** Regular aerobic exercise, resistance training, or a combination of both can improve insulin sensitivity, promote weight loss, and reduce liver fat content. Aim for at least 150 minutes of moderate-intensity aerobic exercise or 75 minutes of vigorous-intensity exercise per week, along with muscle-strengthening activities on two or more days per week.
- **Alcohol Abstinence:** For individuals with alcoholic fatty liver disease (AFLD), abstinence from alcohol is essential to prevent further liver damage and disease progression.

2. Pharmacological Interventions:

- **Insulin Sensitizers:** Insulin sensitizing medications such as metformin and thiazolidinediones (e.g., pioglitazone) may be considered in individuals with fatty liver disease and insulin resistance or type 2 diabetes. These medications can improve insulin sensitivity, reduce hepatic fat content, and have modest effects on liver enzymes and histology.

- Lipid-Lowering Agents: Statins and fibrates may be used to manage dyslipidemia and reduce cardiovascular risk in individuals with fatty liver disease. Statins are generally safe and effective in patients with NAFLD/NASH, while fibrates may be considered in those with elevated triglycerides.
 - Antioxidants: Vitamin E, an antioxidant with anti-inflammatory properties, has been shown to improve liver histology in some individuals with NASH. However, its use should be carefully considered due to potential safety concerns, particularly in certain patient populations.
 - Novel Therapies: Various investigational medications targeting pathways involved in hepatic steatosis, inflammation, fibrosis, and metabolic dysfunction are being evaluated in clinical trials for the treatment of NASH and advanced fibrosis.
3. Treatment of Metabolic Comorbidities:
- Management of type 2 diabetes, hypertension, dyslipidemia, and other metabolic conditions is essential to reduce cardiovascular risk and improve overall metabolic health in individuals with fatty liver disease. Lifestyle interventions, pharmacotherapy, and targeted management strategies should be tailored to individual patient needs and risk profiles.
4. Regular Monitoring and Follow-Up:
- Regular monitoring of liver enzymes, liver function tests, metabolic parameters, and disease progression through imaging studies and non-invasive biomarkers is important for assessing treatment response, detecting complications, and guiding further management decisions.
5. Screening for Hepatocellular Carcinoma:
- Individuals with advanced fibrosis or cirrhosis due to fatty liver disease are at increased risk of hepatocellular carcinoma (HCC). Therefore, regular surveillance with abdominal ultrasound or other imaging modalities is recommended for early detection of HCC in high-risk populations.

SURGICAL MANAGEMENT

1. Liver Transplantation:

- Liver transplantation is indicated for individuals with advanced liver disease (cirrhosis) and liver failure due to complications of NASH, including decompensated cirrhosis, refractory

ascites, hepatic encephalopathy, hepatorenal syndrome, or hepatocellular carcinoma (HCC) within Milan criteria.

- Liver transplantation offers the best chance for long-term survival and improved quality of life in individuals with end-stage liver disease secondary to fatty liver disease.
- The availability of donor organs, careful patient selection, and appropriate timing of transplantation are critical considerations in the management of advanced fatty liver disease.

2. Bariatric Surgery:

- Bariatric surgery may be considered in obese individuals with fatty liver disease, particularly those with morbid obesity (BMI ≥ 40 kg/m²) or obesity-related comorbidities such as type 2 diabetes, hypertension, or obstructive sleep apnea.
- Bariatric surgery can lead to significant weight loss, improvement in metabolic parameters, resolution of non-alcoholic fatty liver disease (NAFLD), and regression of fibrosis in some patients.
- Common types of bariatric surgery include gastric bypass surgery, sleeve gastrectomy, and adjustable gastric banding. The choice of procedure depends on individual patient characteristics, preferences, and risk factors.

3. Hepatic Resection:

- Hepatic resection (partial liver resection) may be considered in selected patients with localized liver tumours, including hepatocellular carcinoma (HCC), arising from fatty liver disease.
- Resection of liver tumours in the setting of fatty liver disease requires careful preoperative evaluation, including assessment of liver function, tumour characteristics, and extent of liver fibrosis, to minimize the risk of postoperative complications and ensure adequate liver remnant function.

4. Experimental Therapies:

- Various experimental therapies and surgical interventions targeting specific aspects of fatty liver disease, such as hepatic steatosis, inflammation, fibrosis, and metabolic dysfunction, are being investigated in clinical trials.
- Examples include metabolic surgeries (e.g., duodenal-jejunal bypass liner), minimally invasive procedures (e.g., radiofrequency ablation, transarterial chemoembolization), and emerging pharmacological therapies targeting novel pathways involved in liver disease pathogenesis.

5. Combined Liver-Kidney Transplantation:

- In individuals with advanced fibrosis or cirrhosis and concomitant renal dysfunction (e.g., hepatorenal syndrome), combined liver-kidney transplantation may be indicated to address both liver and kidney failure.
- Careful patient selection and multidisciplinary evaluation are essential to determine the optimal timing and type of surgical intervention for individuals with fatty liver disease.

NURSING MANAGEMENT

1. Assessment and Monitoring
2. Education and Counselling
3. Nutritional Support
4. Medication Management
5. Psychosocial Support
6. Health Promotion and Disease Prevention

PREVENTION

1. Maintain a Healthy Weight:
 - Aim for a body mass index (BMI) within the normal range (18.5-24.9 kg/m²) by adopting a balanced diet and regular physical activity.
 - Avoid rapid weight gain or loss, as fluctuations in weight can contribute to liver fat accumulation.
2. Follow a Balanced Diet:
 - Consume a diet rich in fruits, vegetables, whole grains, lean proteins, and healthy fats while limiting intake of saturated fats, trans fats, refined carbohydrates, and added sugars.
 - Choose foods with a low glycemic index to help regulate blood sugar levels and prevent insulin resistance.
3. Engage in Regular Physical Activity:
 - Aim for at least 150 minutes of moderate-intensity aerobic exercise or 75 minutes of vigorous-intensity exercise per week, as recommended by guidelines from health organizations.
 - Incorporate strength training exercises at least two days per week to improve muscle mass and metabolic health.

4. Limit Alcohol Consumption:

- If you drink alcohol, do so in moderation. Limit intake to no more than one drink per day for women and two drinks per day for men, as recommended by health guidelines.
- Avoid binge drinking and heavy alcohol consumption, as excessive alcohol intake can contribute to alcoholic fatty liver disease (AFLD).

5. Manage Underlying Medical Conditions:

- Control underlying medical conditions such as obesity, type 2 diabetes, hypertension, and dyslipidemia through lifestyle modifications, medication adherence, and regular monitoring.
- Work closely with healthcare providers to optimize the management of metabolic risk factors and prevent progression to fatty liver disease.

6. Avoid Exposure to Hepatotoxic Substances:

- Minimize exposure to hepatotoxic substances such as certain medications, industrial chemicals, and environmental toxins that can contribute to liver injury and inflammation.
- Follow healthcare provider recommendations for medication use and avoid self-medication or overuse of potentially harmful substances.

7. Vaccination Against Hepatitis Viruses:

- Get vaccinated against hepatitis A and hepatitis B viruses to prevent viral hepatitis infections, which can cause liver inflammation and contribute to fatty liver disease.
- Follow national vaccination guidelines and recommendations for hepatitis immunization, particularly for high- risk populations.

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