CASE REPORT: FATTY LIVER DISEASE

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ABSTRACT

Fatty liver disease, encompassing both alcoholic and non-alcoholic etiologies, is one of the most common chronic liver conditions worldwide. This case report presents a middle-aged adult with non-alcoholic fatty liver disease (NAFLD), detailing clinical presentation, diagnostic evaluation, and management approach. The report highlights the importance of early detection, lifestyle intervention, and long-term monitoring to prevent progression to non-alcoholic steatohepatitis (NASH), fibrosis, or cirrhosis. Through discussion of this case, key considerations in diagnostic workup and evidence-based management strategies are explored. Fatty liver disease (now often called MASLD - Metabolic dysfunction-associated steatotic liver disease) is when excess fat builds up in the liver, often without symptoms in early stages, but can progress to inflammation (MASH/NASH) and liver damage, linked to obesity, diabetes, and alcohol use. Treatment focuses on lifestyle changes like weight loss, healthy diet, exercise, and avoiding alcohol, with potential to reverse damage

Key words: Fatty liver disease, non-alcoholic fatty liver disease, alcoholic fatty liver disease, Lifestyle

INTRODUCTION

Fatty liver disease refers to abnormal fat accumulation within hepatocytes. It is broadly classified into alcoholic fatty liver disease (AFLD) and non-alcoholic fatty liver disease (NAFLD). NAFLD is strongly associated with features of metabolic syndrome including obesity, insulin resistance, type 2 diabetes, dyslipidemia, and hypertension. NAFLD has become a leading cause of chronic liver disease globally due to rising rates of obesity and sedentary lifestyle. NAFLD encompasses a spectrum ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), which includes hepatocyte injury and inflammation. If untreated, NASH may advance to significant fibrosis, cirrhosis, and even hepatocellular carcinoma. However, the condition is reversible in early stages, making timely diagnosis essential. This case illustrates the typical clinical course of NAFLD and emphasizes the importance of lifestyle interventions in treatment.

Types

- **MASLD (formerly NAFLD):** Fatty liver not caused by heavy alcohol use, often linked to metabolic issues.
- **Alcoholic Fatty Liver Disease:** Due to heavy alcohol consumption.
- **MASH (formerly NASH):** A more serious form of MASLD with inflammation and liver cell damage

FUNCTIONAL ANATOMY OF LIVER AND BILIARY SYSTEM

Liver is a dual organ having both secretary and excretory functions. It is the largest gland in the body, weighing about 1.5 kg in man. It is located in the upper and right side of the abdominal cavity, immediately beneath diaphragm.

Liver

Hepatic Lobes

Liver is made up of many lobes called hepatic lobes (Fig. 40.1). Each lobe consists of many lobules called hepatic lobules.

Hepatic Lobules

Hepatic lobule is the structural and functional unit of liver. There are about 50,000 to 100,000 lobules in the liver. The lobule is a honeycomb-like structure and it is made up of liver cells called hepatocytes.

Hepatocytes and Hepatic Plates

Hepatocytes are arranged in columns, which form the hepatic plates. Each plate is made up of two columns of cells. In between the two columns of each plate lies a bile canaliculus . In between the neighboring plates, a blood space called sinusoid is present. Sinusoid is lined by the endothelial cells. In between the endothelial cells some special macrophages called Kupffer cells are present.

Portal Triads

Each lobule is surrounded by many portal triads. Each portal triad consists of three vessels:

1. A branch of hepatic artery

2. A branch of portal vein
3. A tributary of bile duct.

BLOOD SUPPLY TO LIVER

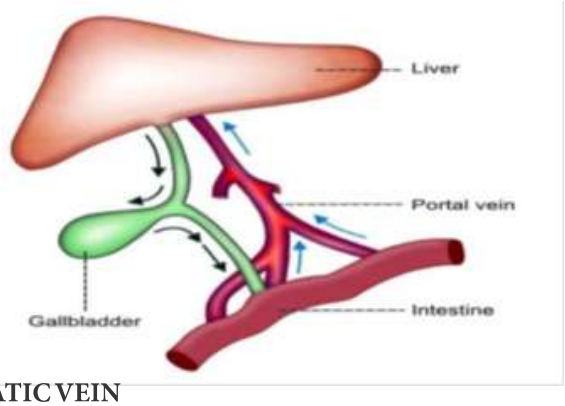
Liver receives maximum blood supply of about 1,500 mL/minute. It receives blood from two sources, namely the hepatic artery and portal vein .

HEPATIC ARTERY

Hepatic artery arises directly from aorta and supplies oxygenated blood to liver. After entering the liver, the hepatic artery divides into many branches. Each branch enters a portal triad.

PORTAL VEIN

Portal vein is formed by superior mesenteric vein and splenic vein. It brings deoxygenated blood from stomach, intestine, spleen and pancreas. Portal blood is rich in monosaccharides and amino acids. It also contains bile salts, bilirubin, urobilinogen and GI hormones. However, the oxygen content is less in portal blood. Flow of blood from intestine to liver through portal vein is known as entero hepatic circulation (Fig.). The blood from hepatic artery mixes with blood from portal vein in hepatic sinusoids. Hepatic cells obtain oxygen and nutrients from the sinusoid.



HEPATIC VEIN

Substances synthesized by hepatic cells, waste products and carbon dioxide are discharged into sinusoids. Sinusoids drain them into central vein of the lobule. Central veins from many lobules unite toform bigger veins, which ultimately form hepatic veins (right and left) which open into inferior vena cava.

FUNCTIONS OF BILE

Most of the functions of bile are due to the bile salts.

1. DIGESTIVE FUNCTION

Refer functions of bile salts.

2. ABSORPTIVE FUNCTIONS

Refer functions of bile salts.

3. EXCRETORY FUNCTIONS

Bile pigments are the major excretory products of the bile. Other substances excreted in bile are: i. Heavy metals like copper and iron ii. Some bacteria like typhoid bacteria iii. Some toxins iv. Cholesterol v. Lecithin vi. Alkaline phosphatase.

4. LAXATIVE ACTION

Bile salts act as laxatives

5. ANTISEPTIC ACTION

Bile inhibits the growth of certain bacteria in the lumen of intestine by its natural detergent action.

6. CHOLERETIC ACTION

7. MAINTENANCE OF pH IN GASTROINTESTINAL TRACT

As bile is highly alkaline, it neutralizes the acid chyme which enters the intestine from stomach. Thus, an optimum pH is maintained for the action of digestive enzymes.

8. PREVENTION OF GALLSTONE FORMATION

9. LUBRICATION FUNCTION

The mucin in bile acts as a lubricant for the chyme in intestine.

10. CHOLAGOGUE ACTION

Bile salts act as cholagogues

FUNCTIONS OF LIVER

Liver is the largest gland and one of the vital organs of the body. It performs many vital metabolic and homeostatic functions, which are summarized below.

1. METABOLIC FUNCTION

Liver is the organ where maximum metabolic reactions such as metabolism of carbohydrates, proteins, fats, vitamins and many hormones are carried out.

2. STORAGE FUNCTION

Many substances like glycogen, amino acids, iron, folic acid and vitamins A, B12 and D are stored in liver.

3. SYNTHETIC FUNCTION

Liver produces glucose by gluconeogenesis. Itsynthesizes all the plasma proteins and other proteins (except immunoglobulins) such as clotting factors, complement factors and hormonebinding proteins. It also synthesizes steroids, somatomedin and heparin.

4. SECRETION OF BILE

Liver secretes bile which contains bile salts, bile pigments,

cholesterol, fatty acids and lecithin. The functions of bile are mainly due to bile salts. Bile salts are required for digestion and absorption of fats in the intestine. Bile helps to carry away waste products and breakdown fats, which are excreted through feces or urine.

5. EXCRETORY FUNCTION

Liver excretes cholesterol, bile pigments, heavy metals (like lead, arsenic and bismuth), toxins, bacteria and virus (like that of yellow fever) through bile.

6. HEAT PRODUCTION

Enormous amount of heat is produced in the liver because of metabolic reactions. Liver is the organ where maximum heat is produced.

7. HEMOPOIETIC FUNCTION

In fetus (hepatic stage), liver produces the blood cells . It stores vitamin B12 necessary for erythropoiesis and iron necessary for synthesis 256 Section 4 t Digestive Systemof hemoglobin. Liver produces thrombopoietin that promotes production of thrombocytes.

8. HEMOLYTIC FUNCTION

The senile RBCs after a lifespan of 120 days are destroyed by reticuloendothelial cells (Kupffer cells) of liver.

9. INACTIVATION OF HORMONES AND DRUGS

Liver catabolizes the hormones such as growth hormone, parathormone, cortisol, insulin, glucagon and estrogen. It also inactivates the drugs, particularly the fatsoluble drugs. The fatsoluble drugs are converted into watersoluble substances, which are excreted through bile or urine.

10.DEFENSIVE AND DETOXIFICATION FUNCTIONS

Reticuloendothelial cells (Kupffer cells) of the liver play an important role in the defense of the body. Liver is also involved in the detoxification of the foreign bodies.

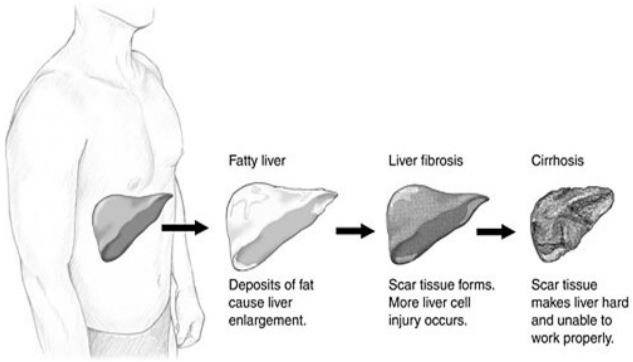
- Metabolic liver disease is a group of disorders that affect the liver's ability to process and metabolize nutrients, leading to the accumulation of harmful substances in the body. These disorders can be inherited or acquired and may result in liver damage, impaired function, or even liver failure. Risk factors Family history of fatty liver disease or obesity.
- Growth hormone deficiency, which means the body doesn't make enough hormones to grow.
- High cholesterol.
- High levels of triglycerides in the blood.
- Insulin resistance.
- Metabolic syndrome.

Obesity, especially when fat is centered in the waist

Case

A 47-year-old male presented to the outpatient clinic with complaints of persistent fatigue and intermittent right upper quadrant discomfort over the past six months. His medical history included poorly controlled type 2 diabetes mellitus diagnosed eight years earlier, obesity with a BMI of 32 kg/m², and dyslipidemia. He denied alcohol consumption and had no history of viral hepatitis, medication overuse, or prior liver disease.

Physical examination revealed central obesity but was otherwise unremarkable, with no jaundice, hepatomegaly, or stigmata of chronic liver disease. Laboratory investigations showed mildly elevated ALT (72 U/L) and AST (58 U/L), with normal bilirubin and synthetic function markers. HbA1c was 8.4%, indicating poor glycemic control. An abdominal ultrasound demonstrated increased hepatic echogenicity consistent with fatty infiltration. Further evaluation with FibroScan showed mild steatosis without significant fibrosis. After excluding secondary causes, the patient was diagnosed with non-alcoholic fatty liver disease. The patient was counseled on lifestyle modifications including weight reduction through diet and exercise, improved glycemic control, and reduction of saturated fats. A target of 7–10% body weight loss was recommended. He was advised to increase physical activity to at least 150 minutes of moderate-intensity exercise weekly. Follow-up was scheduled at 12 weeks.



Symptoms

Often silent, but can include:

- Fatigue, weakness
- Pain or discomfort in the upper right abdomen
- Later stages: Jaundice (yellow skin/eyes), swollen abdomen/legs, itchy skin, mental confusion.

Causes & Risk Factors

- Obesity, excess weight.
- Type 2 diabetes, insulin resistance, high cholesterol, high blood pressure (metabolic syndrome).
- Heavy alcohol use.
- Poor diet (high sugar, saturated fats, processed foods).
- Certain medications, PCOS, underactive thyroid.

Diagnosis

- Blood tests (liver enzymes).
- Imaging (Ultrasound, CT, MRI).
- Liver biopsy (to differentiate NAFL from NASH).

Treatment & Management

- **Weight Loss:** Losing 5-10% of body weight.
- **Diet:** Healthy, balanced diet (e.g., Mediterranean diet).
- **Exercise:** Aim for 150 mins/week.
- **Avoid Alcohol:** Especially if alcoholic fatty liver disease.
- **Control Conditions:** Manage diabetes, blood pressure, cholesterol.
- **No Specific Drugs:** Treatment focuses on lifestyle.

Prevention

To reduce your risk of MASLD:

- **Eat a healthy diet.** Eat a diet that's rich in fruits, vegetables, whole grains and healthy fats. One such diet is the Mediterranean diet.
- **Limit alcohol, simple sugars and portion sizes.** Avoid sugary drinks such as soda, sports drinks, juices and sweet tea. Avoid or limit alcohol, which can damage the liver.
- **Keep a healthy weight.** If you are overweight or have obesity, work with your healthcare team to gradually lose weight. If you are at a healthy weight, work to keep it by eating a healthy diet and exercising.
- **Exercise.** Be active most days of the week. Get an OK from your healthcare team first if you haven't been exercising regularly.

Complications

Fatty liver can develop into hepatic fibrosis, cirrhosis or liver cancer. For people affected by NAFLD, the 10-year survival rate was about 80%. The rate of progression of fibrosis is estimated to be one per 7 years in NASH and one per 14 years in NAFLD, with an increasing speed. There is a strong relationship between these pathologies and metabolic illnesses (diabetes type II, metabolic syndrome). These pathologies can also affect non-obese people, who are then at a higher risk

Less than 10% of people with cirrhotic alcoholic FLD will develop hepatocellular carcinoma, the most common type of primary liver cancer in adults, but up to 45% people with NASH without cirrhosis can develop hepatocellular carcinoma.

The condition is also associated with other diseases that influence fat metabolism.

DISCUSSION

NAFLD is considered a hepatic manifestation of metabolic syndrome and is now recognized as a major global health burden. Insulin resistance plays a central role in the pathogenesis of NAFLD, promoting lipolysis, increased free fatty acid delivery to the liver, and de novo lipogenesis. Diagnosis requires evidence of hepatic steatosis, exclusion of significant alcohol intake, and ruling out secondary causes such as viral hepatitis, medications (e.g., corticosteroids, methotrexate), or genetic disorders.

Ultrasound remains the most widely used initial imaging modality due to accessibility and cost-effectiveness, although it has limited sensitivity in early disease. FibroScan and MRI-based techniques offer improved detection of fibrosis and fat quantification.

Management primarily focuses on lifestyle modification, particularly weight loss. A reduction of at least 5% of body weight improves steatosis, while 10% or greater may reverse inflammation and fibrosis. Dietary changes such as adopting a Mediterranean-style diet have shown benefit. Physical activity also independently improves hepatic fat content. Pharmacologic options remain limited; however, medications targeting metabolic dysfunction—such as GLP-1 receptor agonists for diabetes—have shown promise in improving liver histology. Vitamin E may be considered in non-diabetic patients with biopsy-proven NASH. Regular

monitoring is essential due to the risk of progression.

CONCLUSION

This case underscores the growing prevalence of NAFLD and the importance of recognizing patients at high risk. Early diagnosis allows for effective intervention, primarily through lifestyle modification. Weight reduction, improved glycemic control, and regular monitoring remain the cornerstone of therapy. Greater awareness and proactive screening in individuals with metabolic risk factors can reduce long-term complications and improve outcomes.

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