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Diagnostic Problem of Hyperbilirubinemia in Diabetes Mellitus Patient: Case Report

Problema Diagnóstico de Hiperbilirrubinemia en paciente con Diabetes Mellitus: Reporte de Caso

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SUMMARY

This case report presents a 57-year-old woman with worsening jaundice and associated symptoms, including abdominal pain, bloating, weakness, weight loss, and malaise. Clinical examination revealed icteric sclera, pale conjunctiva, and ascites. Laboratory findings showed elevated bilirubin, decreased albumin, increased alkaline phosphatase and gamma GT, severe dyslipidemia, and poorly controlled diabetes. Imaging studies revealed hepatomegaly, ascites, and enlarged lymph nodes without biliary obstruction. A liver biopsy confirmed cirrhosis. The diagnostic approach for jaundice involves a comprehensive evaluation, including patient history, physical examination, laboratory tests, imaging studies, and liver biopsy. This case highlights the importance of considering non-alcoholic fatty liver disease (NAFLD) in patients with metabolic disorders, particularly type 2 diabetes mellitus (T2DM). NAFLD is strongly associated with T2DM, with diabetes driving the progression to cirrhosis and increasing mortality risk. Treatment for NAFLD primarily focuses on lifestyle

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changes, particularly weight reduction. For diabetic patients with NAFLD, specific diabetes medications like pioglitazone and GLP-1 receptor agonists may provide extra advantages. This case highlights the crucial role of liver biopsy in confirming diagnoses and evaluating disease severity in complex liver conditions, particularly those associated with metabolic disorders. It also emphasizes the importance of a multidisciplinary approach in managing patients with NAFLD and associated metabolic conditions to improve outcomes and prevent disease progression.

Keywords: *Hyperbilirubinemia, jaundice, jaundice in diabetic patient, hyperbilirubinemia in diabetes.*

RESUMEN

Este reporte de caso presenta a una mujer de 57 años con ictericia en empeoramiento y síntomas asociados, incluyendo dolor abdominal, distensión, debilidad, pérdida de peso y malestar. El examen clínico reveló esclerótica ictérica, conjuntiva pálida y ascitis. Los hallazgos de laboratorio mostraron bilirrubina elevada, disminución de la albúmina, aumento de la fosfatasa alcalina y gamma GT, dislipidemia severa y diabetes mal controlada. Los estudios de imagen revelaron hepatomegalia, ascitis y ganglios linfáticos agrandados sin obstrucción biliar. La biopsia hepática confirmó cirrosis. El enfoque diagnóstico para la ictericia implica una evaluación integral, que incluye la historia clínica del paciente, el examen físico, pruebas de laboratorio, estudios de imagen y biopsia hepática. Este caso destaca la importancia de considerar la enfermedad del hígado graso no alcohólico (NAFLD) en pacientes con trastornos metabólicos, particularmente la diabetes mellitus tipo 2 (T2DM). La enfermedad hepática grasa no alcohólica (NAFLD) está fuertemente asociada con la diabetes tipo 2 (T2DM), siendo la diabetes un factor que impulsa la progresión hacia la cirrosis y aumenta el riesgo de mortalidad. El tratamiento para la NAFLD se centra principalmente en cambios en el estilo de vida, particularmente en la reducción de peso. Para los pacientes diabéticos con NAFLD, medicamentos específicos para la diabetes como la pioglitazona y los agonistas del receptor GLP-1 pueden proporcionar ventajas adicionales. Este caso destaca el papel crucial de la biopsia hepática en la confirmación de diagnósticos y la evaluación de la gravedad de la enfermedad en condiciones hepáticas complejas, particularmente aquellas asociadas con trastornos metabólicos. También enfatiza la importancia de un enfoque multidisciplinario en el manejo de pacientes con NAFLD y condiciones metabólicas asociadas para mejorar los resultados y prevenir la progresión de la enfermedad.

Palabras clave: *Hiperbilirrubinemia, ictericia, ictericia en paciente diabético, hiperbilirrubinemia en diabetes.*

INTRODUCTION

Jaundice is a distinctive and striking yellow discoloration of the skin, conjunctiva, and mucous membranes resulting from the widespread deposition of bilirubin compounds. It is generally viewed as a sign of liver and bile duct disease and has a broad differential diagnosis; identifying the cause and finding the best way to treat it have been challenges for physicians for thousands of years (1).

The latest report from the International Diabetes Federation states that 10.5 % of the world's adult population suffers from diabetes. If this trend continues, one in eight adults will live with diabetes by 2045 (2). It is estimated that 462 million individuals worldwide, or 6.28 percent of the global population, suffer from type 2 diabetes mellitus (T2DM). It is the tenth and most significant cause of death globally, accounting for more than a million fatalities in 2017 (3).

NAFLD, or non-alcoholic fatty liver disease, affects a quarter of the global population. NAFLD consists of a spectrum of liver disorders, ranging from isolated hepatic steatosis to NASH or non-alcoholic steatohepatitis, advanced fibrosis, cirrhosis, hepatoma, and liver-related mortality. Obesity, insulin resistance, and type 2 diabetes mellitus are the primary risk factors for NAFLD development among the numerous variables involved in the pathophysiology of the disease (2). Obstructive sleep apnea, cardiovascular disease, and type 2 diabetes mellitus are strongly correlated with NAFLD, which is regarded as the hepatic manifestation of metabolic syndrome (1). According to a survey done between 1990 and 2019, the overall prevalence of NAFLD worldwide was assessed to be 30.05 % for the whole study period (4).

According to the most recent meta-analysis, approximately 60 % of type 2 diabetes patients worldwide have NAFLD, more than twice as common as the general population, and one-third of these individuals also have NASH (1). The

estimated global prevalence of NAFLD among patients with T2DM is 55.48 %, with a regional prevalence of 51.77 % in the United States, 56.83 % in Latin America, 67.97% in Europe, 52.04 % in East Asia, 57.87 % in South Asia, 67.29 % in West Asia, and 30.39 % in Africa. Based on the global prevalence of T2DM (8.5%), the predicted prevalence of patients with T2DM and NAFLD is 47.16 per 1 000 global population (5). By 2030, it is anticipated that an obesity-related lifestyle, an aging population, and the rising incidence of type 2 diabetes will cause significant increases in liver mortality and NASH-related hepatoma of 137 % and 178 %, respectively (2).

Case reports regarding patients with early jaundice symptoms in T2DM patients are still rare. Here, we discuss the case of a female patient with hyperbilirubinemia and type 2 diabetes mellitus (T2DM). Discussion on how to establish a diagnosis for patients.

Case Presentation

Mrs S, 57 years old, was admitted to the ER with complaints of right upper quadrant abdominal pain three days before admission. The pain was intermittent and did not radiate. She also complained of jaundice in the entire body, including her eyes, which had been present for the past six months. Additionally, she complained of an abdominal enlargement over the last three months. Since the onset of jaundice, the patient experienced itching in the entire body. No complaints of nausea or vomiting and no fever were reported. She mentioned dark-colored urine over the past month but denied pale-colored stools resembling putty. The patient's weight had decreased by approximately 20 kg in the last six months. The patient had been hospitalized in Madiun within the past six months due to complaints of jaundice. Over the same period, the patient felt weak and easily fatigued. The patient was non-alcoholic, has a history of diabetes mellitus for the past five years and was last treated with insulin; however, the patient did not use it regularly due to frequent complaints of weakness after insulin use. The patient rarely attended follow-up visits. There was no history of hypertension, kidney disease, or lung disease.

She was admitted in weak condition with a Glasgow Coma Scale (GCS) of 456. She weighed 45 kg, had a height of 150 cm, and had a Body Mass Index (BMI) of 20. Vital signs include blood pressure of 146/94 mmHg, a regular pulse of 94 beats per minute, a respiratory rate of 22 breaths per minute, an axillary temperature of 35.9°C, and oxygen saturation of 99 % on room air. Head and neck examination showed pale conjunctiva and icteric sclera. Her skin was icteric. Abdominal examination reveals signs of ascites (Figure 1).

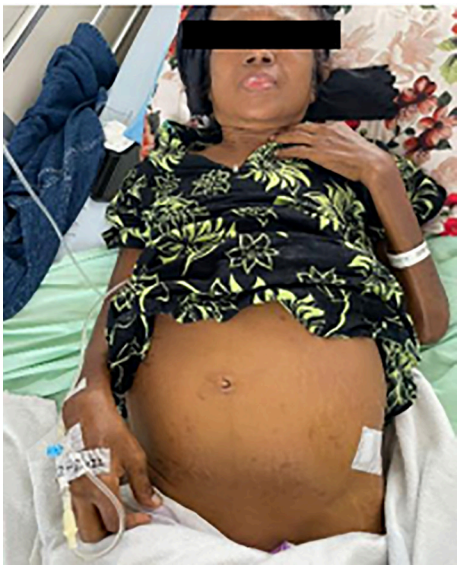

Figure 1. Clinical Appearance of the patient.

Laboratory examination showed Hb 12, haematocrit (HCT) 36 %, White Blood Counts (WBC) 13 270, platelets 351 000, direct bilirubin 10.4 mg/dL, total bilirubin 14.9 mg/dL, albumin 2.51 g/dL, ANA test 17.5, C3 123.5 mg/dL, C4 32.53 mg/dL, LDH 200 U/L, ALP 1 108 IU/L, glutamyl gamma-glutamyl transferase 1 073 U/L, AFP <2 ng/mL, Ca 19-9 442 U/mL, Ca 125 132.63 U/mL, CEA 2.03 ng/mL, non-reactive HbsAg, non-reactive HCV, non-reactive HIV, total cholesterol 658 mg/dL, triglyceride 194 mg/dL, LDL 1 108 mg/dL, HDL 20 mg/dL and HbA1C 6.2 %. Antimitochondrial

Antibody (AMA) showed negative results with positive results in Antismooth Muscle Antibody (ASMA). Fibroscan examination showed CAP 173 dB/m, spleen stiffness 51.7 kPa, and liver fibrosis with no steatosis. Magnetic Resonance Cholangiopancreatography (MRCP) showed cholecystitis, ascites, hepatomegaly, and thickening of the omentum wall. Liver biopsy exhibited fibrous connective tissue with disturbed architecture, containing bile duct proliferation and inflammatory cell infiltration, indicating liver cirrhosis. The patient was initially diagnosed with non-alcoholic fatty liver disease (NAFLD) (Figure 2).

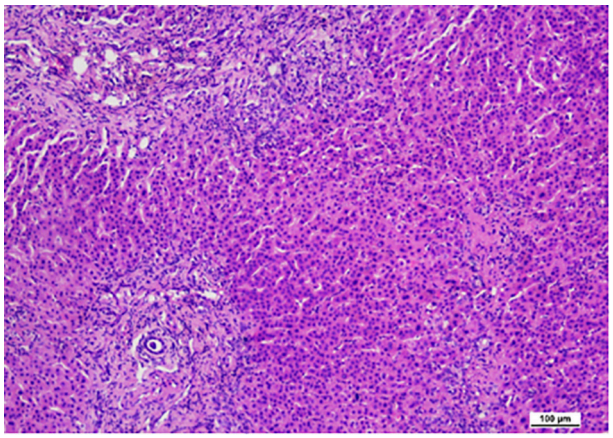

Figure 2. Histopathology of Liver.

During the admission, the patient received a subcutaneous glargine injection once daily and oral ursodeoxycholic acid, furosemide, spironolactone, and atorvastatin once daily. The patient was then discharged with improvement of clinical symptoms.

DISCUSSION

Jaundice is a distinctive and striking yellow discoloration of the skin, conjunctiva, and mucous membranes resulting from the widespread deposition of bilirubin compounds (1). Bilirubin metabolism is divided into prehepatic, intrahepatic, and posthepatic phases. The prehepatic phase begins with the formation of bilirubin, 70 %-80 % of which comes from the breakdown of mature red blood cells, while the remaining 20 %-30 % comes from other heme proteins primarily found in bone marrow and liver. The intrahepatic phase occurs when free bilirubin concentrated in liver cells undergoes conjugation with uridine diphosphate (UDP) glucuronic acid to form bilirubin diglucuronide or conjugated bilirubin or direct bilirubin. The post-hepatic phase occurs when conjugated bilirubin is excreted into the canaliculus along with other substances (6).

The general algorithm for evaluating patients with jaundice involves four basic steps: 1) patient history, thorough physical examination, and screening laboratory studies; 2) formulation of a working differential diagnosis; 3) selection of specific tests to narrow diagnostic possibilities; and 4) development of a strategy for treatment or further testing if unexpected diagnostic possibilities arise. Acute viral hepatitis is a strong diagnostic possibility when viral prodromal symptoms (such as anorexia, malaise, and myalgia) are present, particularly if there are exposure risk factors. Hereditary hyperbilirubinemia and genetic liver disease are more likely to occur in families with a history of jaundice or liver disease. A palpable abdominal mass found during a physical examination suggests that the source of the obstructive jaundice is malignant sings of portal hypertension (ascites, splenomegaly, large abdominal veins), telangiectasias, gynecomastia, and asterixis can all be indicators of the presence of cirrhosis (1).

Laboratory tests in patients with jaundice include serum total bilirubin, alkaline phosphatase, aminotransferases, complete blood count, and prothrombin time. Serum alkaline phosphatase activity originates from isoenzymes expressed on the membranes of various cell types; the hepatobiliary alkaline phosphatase isoenzyme is a membrane-bound protein localized primarily to the apical pole of hepatocytes and cholangiocytes. Biliary obstruction and intrahepatic cholestasis increase basolateral release of alkaline phosphatase, and serum alkaline phosphatase activity increases. A predominant increase in serum alkaline phosphatase activity (relative to aminotransferases) indicates biliary obstruction or intrahepatic cholestasis. However, an increase in

serum alkaline phosphatase activity (especially if aminotransferase activity is normal) may indicate the release of alkaline phosphatase isoenzymes from extrahepatic tissues. If there is diagnostic uncertainty, increased serum activity of other proteins (e.g., GGTP, 5'-nucleotidase, alkaline phosphatase isoenzymes) confirms the presence of hepatobiliary disease. Aminotransferases: Alanine aminotransferase (ALT), a cytosolic enzyme found primarily in hepatocytes, and Aspartate aminotransferase (AST), an isozyme found in hepatocytes and cells from several other tissues—are usually detected in serum at low concentrations. Conditions that result in hepatocellular injury (e.g., viral hepatitis, toxic liver injury, ischemic hepatitis) increase plasma membrane permeability and release of aminotransferases into the plasma. A predominant increase in serum aminotransferase levels (relative to alkaline phosphatase) indicates that jaundice is caused by hepatocellular injury. Leucocytosis could be a sign of cholestasis-related inflammatory diseases or biliary blockage. Anaemia raises the possibility that a haemolytic is the source of jaundice, particularly in cases when isolated hyperbilirubinemia is shown in liver biochemical testing without any other abnormalities (1).

Thrombocytopenia is a typical finding in cirrhosis and appears to result from a decreased synthesis of thrombopoietin, a regulator of platelet production, or increased splenic sequestration associated with portal hypertension. Prothrombin time reflects the activity of coagulation factors I, II, V, VII, and X. Prothrombin time is prolonged (typically reported as an increase in international normalized ratio (INR)) with reduced hepatic production of these proteins; however, this is not specific to conditions associated with hepatocellular injury. Prolongation of prothrombin time can also be seen in intrahepatic cholestasis or prolonged biliary obstruction due to impaired absorption of vitamin K, a fat-soluble cofactor required for the synthesis of factors II, VII, IX, and X (1).

Abdominal ultrasound is the initial imaging test performed in jaundiced patients with suspected hepatobiliary disease. Ultrasound examination can also show cholelithiasis (although bile duct stones may not be well visualized) and intrahepatic lesions larger than one cm in diameter. Abdominal CT with intravenous contrast is an alternative non-invasive means of evaluating hepatobiliary disease. An additional non-invasive technique for assessing hepatobiliary illness is abdominal CT with intravenous contrast. Abdominal CT is operator-independent, produces technically more precise images in obese patients, and can detect space-occupying intrahepatic lesions as small as 5 mm. Magnetic Resonance Cholangiopancreatography (MRCP) allows visualization of bile ducts. MRCP is superior to conventional ultrasound or CT for detecting biliary obstruction and plays an important role as a diagnostic test. A standard Magnetic resonance imaging (MRI) can be performed during the same examination time if there is suspicion of a hepatobiliary mass or an allergy to contrast. Endoscopic Retrograde Cholangiopancreatography, or Endoscopic retrograde cholangiopancreatography (ERCP), allows direct visualization of the bile ducts. ERCP is more invasive than ultrasound, CT, and MRCP and is comparable in cost to MRCP. ERCP is highly accurate in diagnosing biliary obstruction. If a focal cause (e.g., choledocholithiasis, biliary stricture) is identified, manoeuvres to relieve the obstruction (e.g., sphincterotomy, stone extraction, stricture dilation, stent placement) can be performed in the same session.

Patients with jaundice accompanied by biochemical evidence of hepatocellular dysfunction or cholestasis in which imaging studies do not show biliary obstruction should be evaluated for underlying liver disease. Depending on the suspected abnormality, screening laboratory studies include viral serology; serum iron, transferrin, and ferritin levels (for hemochromatosis); ceruloplasmin (for Wilson's disease); AMA (for PBC); ANA, smooth muscle antibodies, and serum immunoglobulins (for autoimmune hepatitis); and tissue transglutaminase antibodies (for celiac disease). A liver biopsy may be performed if a diagnosis is not established based on serological tests. Liver biopsy provides accurate information about lobular architecture and the degree and pattern of liver inflammation and fibrosis and is very helpful in patients with persistent jaundice and undiagnosed jaundice. Liver biopsy enables the diagnosis of fatty liver disease (alcohol-related or not), hemochromatosis, Wilson's disease, autoimmune hepatitis, primary biliary cirrhosis

(PBC), granulomatous hepatitis, and neoplasms with the use of specific histological stains and, if necessary, with quantification of iron or copper content. Liver histology may be, as expected, in acute biliary obstruction. A liver biopsy has a low rate of complications, mostly from bleeding and perforation, and a 1 % hospitalization rate; the death rate is approximately 0.01% (1) (Figure 3).

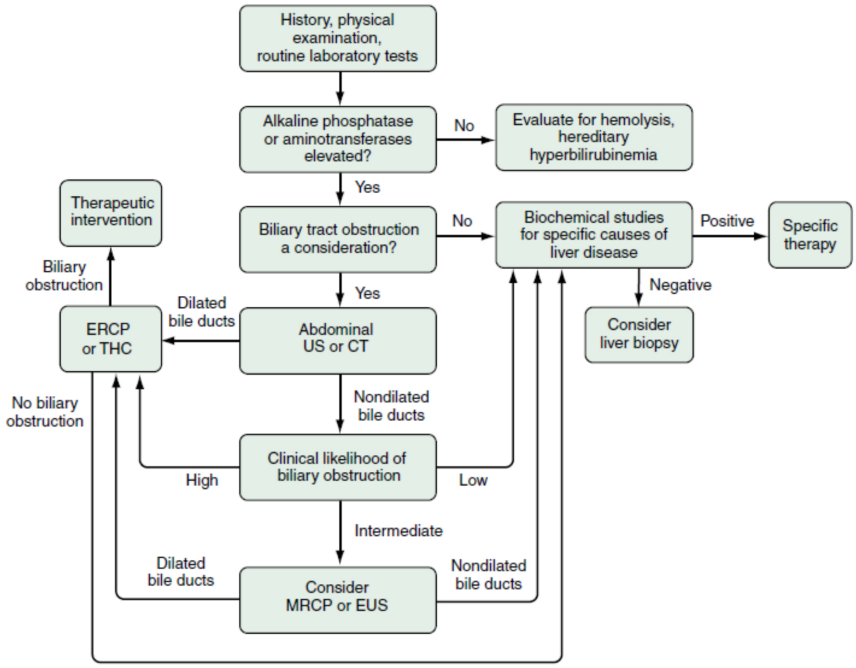

Figure 3. Algorithm for evaluation and management of jaundice and hyperbilirubinemia. THC, Transhepatic cholangiography (1).

NAFLD is a group of liver disorders associated with metabolic dysfunction. It is defined by the presence of steatosis in more than 5 % of hepatocytes, linked to metabolic risk factors (particularly obesity and type 2 diabetes), and the absence of excessive alcohol consumption (≥30 g per day for men and ≥20 g per day for women) or other chronic liver diseases (7). T2DM accelerates the progression of NAFLD to cirrhosis and increases the risk of liver-related and all-cause mortality by two to three times (2). Among patients with NASH-related cirrhosis, T2DM is associated with a four-fold increased risk of hepatoma incidence (8).

Diabetes was found to be the strongest independent predictor of the development of hepatoma in a recent study involving 18 million persons with NAFLD in Europe. NAFLD also negatively impacts T2DM, both in its occurrence and clinical deterioration. According to a recent meta-analysis comprising over 500 000 middle-aged people from Asia, the US, and Europe (30 % with imaging- or biopsy-confirmed NAFLD), NAFLD was linked to a two-fold increased risk of acquiring T2DM (3). Most NAFLD cases occur in middle-aged people in their fourth or fifth decades of life, while it is now increasingly seen in younger children and teenagers. NAFLD is generally more common in men than women, but its prevalence is higher in older women, particularly postmenopausal women, suggesting a relationship between sex hormones and menopause. NAFLD (and especially NASH) is often associated with diabetes mellitus, with

prevalence rates of 60 % to 76 % for NAFLD and 22 % for NASH. The Dallas Heart Study suggested ethnicity as a risk factor, with Hispanics showing the highest NAFLD prevalence (45 %), compared to 33 % in Caucasians and 24 % in African Americans (1).

NAFLD results from free fatty acid accumulation in the liver. Fatty acid metabolism is influenced by regulators like catecholamines, glucagon, growth hormone, and insulin (1). Insulin resistance in type 2 diabetes can cause liver and extrahepatic tissue disorders, contributing to NAFLD pathogenesis (9). Insulin resistance triggers insulin signaling disorders in adipose tissue and the liver, leading to adipose tissue dysfunction and a shift towards pro-inflammatory adipokine production (10,11). Glucotoxicity and lipotoxicity, along with mitochondrial dysfunction, oxidative and endoplasmic reticulum stress, and altered gut microbiome, collectively promote liver inflammation and hepatic sinusoidal endothelial cell capillarization, causing hepatic stellate cell activation and liver fibrosis development (12,13).

Asymptomatic individuals comprise 48–100% of NAFLD cases. Symptoms can include non-specific chronic problems such as lethargy and malaise, and abdominal discomfort in the upper right quadrant. Hepatomegaly may be the only physical abnormality detected. NAFLD is often discovered incidentally during other examinations. Some patients present with cirrhosis complications such as splenomegaly, telangiectasia, ascites, variceal bleeding, or even hepatoma (1,14).

In metabolic fatty liver disease, mild to moderate elevations (1.5 to 4 times) in serum AST, ALT, or both are widely followed. However, levels rarely exceed 10 times the upper limit of normal. In contrast to alcohol-related fatty liver disease, when AST levels are often at least twice as high as ALT levels, serum ALT levels are generally greater than AST levels. Alkaline phosphatase and GGTP levels may be elevated, but serum bilirubin, prothrombin time, and serum albumin levels are generally normal, except in patients with NAFLD-related cirrhosis. Low-titer ANA (<1:320) is found in about 25% of individuals with NAFLD, but it does not affect the patient's clinical course or prognosis. Laboratory tests for other chronic liver diseases are negative. NAFLD can coexist with HCV, although hepatitis C virus (HCV) infection (especially HCV genotype 3) itself can cause hepatic steatosis. Serum and hepatic iron levels may be elevated in patients with NAFLD. Specifically, serum ferritin levels may be elevated in 20 % to 50 % of patients with NAFLD and may be a marker of more advanced disease. Hepatic ultrasound may reveal a "bright" liver with increased echogenicity, consistent with hepatic steatosis. Additionally, fatty liver can be identified using MRI (fat shows up strongly on T1-weighted imaging) or abdominal CT (fatty liver has a lower density than the spleen). Ultrasound is good at detecting steatosis, particularly if the liver fat level is more than 20%. CT and MRI, on the other hand, are excellent at detecting steatosis, with an area under the receiver operating characteristic (AUROC) of 0.90 and greater. Conventional cross-sectional imaging cannot only show evidence of portal hypertension in advanced disease but also evaluate liver masses. These modalities cannot identify the severity or validate the existence of NASH (1).

Diagnosing NAFLD is generally straightforward when imaging reveals hepatic steatosis, and alternative chronic liver conditions are excluded. However, despite the relative ease of diagnosis, a liver biopsy remains essential for identifying NASH cases. In practice, the majority of NAFLD patients do not undergo this procedure. Liver biopsy is an invasive technique that, while rare, can lead to serious complications such as bleeding and, in extreme cases, death. Differentiating between NASH and Isolated Fatty Liver (IFL) is of paramount importance, as NASH patients have a higher risk of progressing to cirrhosis (1).

While liver biopsy remains the gold standard for diagnosing NAFL and NASH, it is not universally recommended due to its invasive nature, high cost, and associated risks. Liver biopsy should be considered in all patients with persistently elevated aminotransferase levels whose diagnosis remains uncertain. Biopsy also plays a crucial role in excluding other liver conditions, particularly when serum ferritin is elevated, or autoantibodies are present. Furthermore, it offers prognostic insights into NAFLD cases, as the identification of NASH and/or fibrosis can indicate the likelihood of cirrhosis development and liver-related mortality.

The American Association of Liver Disease practice guidelines for NAFLD recommend considering liver biopsy in patients with NAFLD who are at high risk of NASH and fibrosis. These guidelines suggest that the presence of metabolic syndrome and NAFLD fibrosis score be used to identify high-risk patients. In addition to proving a definitive diagnosis, liver biopsy is useful for and determining the stage of liver disease and assessing the likelihood of disease progression (15).

The cornerstone of NAFLD treatment continues to be lifestyle changes, with a particular emphasis on weight reduction. Research has demonstrated a correlation between the extent of weight loss and improvements in liver histology, indicating that a 7 %-10 % or greater weight reduction is necessary to resolve NASH and ameliorate liver fibrosis. For T2DM patients with NAFLD, especially those presenting with NASH and fibrosis, certain diabetes medications are given priority. However, it's worth noting that no drugs have yet received approval from US and European food and drug agencies. Nonetheless, randomized controlled trials have shown that some diabetes medications offer benefits beyond their primary role of HbA1c reduction. The 2022 American Diabetes Association guidelines specifically recommend pioglitazone and glucagon-like peptide-1 (GLP-1) receptor agonists for this purpose. NAFLD is also closely related to the occurrence of cardiovascular disease and chronic kidney disease, so the administration of drugs with protective functions for the heart and kidneys is recommended (3).

A 57-year-old female patient presented with complaints of jaundice accompanied by abdominal distension. The patient reported feeling weak and had a history of diabetes mellitus for the past 2 years, during which she rarely sought medical attention and infrequently used insulin medication. Physical examination revealed ascites but no hepatomegaly. Laboratory tests showed elevated bilirubin levels, decreased albumin levels, increased alkaline phosphatase, and metabolic disorders, including diabetes mellitus and dyslipidemia. In addition to positive anti-smooth muscle antibody (ASMA) and negative anti-mitochondrial antibody (AMA) tests, they also showed increased Gamma GT. Imaging studies revealed liver fibrosis on fibroscan examination. CT and MRI of the abdomen showed hepatomegaly without biliary system obstruction. Nevertheless, there was no measurement of the patient's immunoglobulins. Even after these examinations, a conclusive diagnosis was still unattainable. Consequently, a liver biopsy was carried out, and the results showed that liver cirrhosis of the F4 stage was present.

CONCLUSION

This case highlights the complex interplay between Type 2 Diabetes Mellitus (T2DM) and Non-Alcoholic Fatty Liver Disease (NAFLD), culminating in liver cirrhosis. The patient's symptoms of jaundice and ascites, coupled with a history of inadequately managed diabetes, emphasize the critical need for careful monitoring and treatment of metabolic conditions to avert serious liver complications. The diagnostic process highlights difficulties in distinguishing between various liver ailments, especially when several risk factors coexist. Although initial tests and imaging provided useful information, the liver biopsy was essential in confirming the diagnosis of cirrhosis. This case reaffirms the importance of liver biopsy as the definitive method for diagnosing and staging NAFLD, particularly when non-invasive tests are inconclusive.

The progression to cirrhosis in this patient with T2DM aligns with the current understanding of the bidirectional relationship between diabetes and NAFLD. It emphasizes the need for early intervention and comprehensive management strategies in patients with metabolic risk factors. This case demonstrates the need for a collaborative, multi-specialty approach in treating patients with coexisting metabolic and liver disorders. It advocates for greater awareness among medical professionals regarding the liver-related complications of T2DM and the necessity of regular liver function tests in diabetic patients. Furthermore, it reinforces the need for lifestyle modifications and targeted therapies to address both diabetes and liver health, potentially slowing disease progression and improving overall outcomes.

Ethics and Consent

The study participants provided their informed consent upon admission, allowing the presentation of their case details for publication and educational purposes. The publication of this information did not require institutional approval.

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Disclosure Statement

The authors stated no conflicts of interest in this work.

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