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A Case of Non - Alcoholic Fatty Liver Disease

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CASE STUDY:

Patient Information:

- Name: Mr . Dhanush
- Age: 45 years
- Sex: Male
- Occupation: Office worker
- Height: 175 cm
- Weight: 95 kg
- BMI: 31 kg/m (Obese)

Medical History:

- Type 2 Diabetes Mellitus (diagnosed 5 years ago)
- Hypertension (diagnosed 3 years ago)
- Dyslipidemia
- Sedentary lifestyle

Family History:

- Father had coronary artery disease
- Mother had type 2 diabetes

Presentinoft Complaints:

Mr. Dhanush presents with fatigue and occasional discomfort in the right upper quadrant of his abdomen. He denies any alcohol consumption and has not noticed jaundice or signiflcant weight loss. He has a history of poorly controlled type 2 diabetes and has gained 10 kg in the last 2 years due to a sedentary lifestyle and an unbalanced diet.

Physical Examination:

Vitals:

- Blood Pressure: 145/90 mmHg
- Heart Rate: 82 bpm
- BMI: 31 kg/m

Abdominal Examination:

- Mild tenderness in the right upper quadrant
- No hepatomegaly or splenomegaly
- No signs of ascites or peripheral edema

Other Findings:

No signs of jaundice, palmar erythema, or spider angiomas.

Initial Investioftations:

1. **Blood Tests:**

- Liver Function Tests (LFTs):
- ALT: 82 U/L (Normal: 7-56 U/L)
- AST: 62 U/L (Normal: 10-40 U/L)
- GGT: 70 U/L (Normal: 9-48 U/L)
- ALP: 105 U/L (Normal: 44-147 U/L)
- Bilirubin: 0.9 mg/dL (Normal: 0.1-1.2 mg/dL)
- Albumin: 4.3 g/dL (Normal: 3.5-5.0 g/ dL)
- Fasting Blood Glucose: 155 mg/dL (elevated)
- HbA1c: 8.2% (poorly controlled diabetes)

Lipid Profile :

- Total Cholesterol: 230 mg/dL (Normal: <200 mg/dL)
- LDL Cholesterol: 150 mg/dL (Normal: <100 mg/dL)
- HDL Cholesterol: 35 mg/dL (Normal:>40 mg/dL)
- Triglycerides: 250 mg/dL (Normal: <150 mg/dL)

2. **Imaging:**

Abdominal Ultrasound: Shows increased liver echogenicity consistent with hepatic steatosis. No signs of cirrhosis or hepatomegaly.

Diaoftnosis:

- Non-Alcoholic Fatty Liver Disease (NAFLD) based on:
- Evidence of hepatic steatosis on ultrasound
- Exclusion of signiflcant alcohol intake (less than 30 g/day)
- Elevated liver enzymes, particularly ALT and AST, without other identiflable causes of liver disease (e.g., viral hepatitis, autoimmune liver disease)

Risk Factors Identified:

- Obesity (BMI of 31)
- Poorly controlled Type 2 Diabetes Mellitus
- Dyslipidemia (elevated LDL and triglycerides, low HDL)

- Sedentary lifestyle
- Hypertension

MANAGEMENT PLAN:

1. **Lifestyle Modiflcations:**

- Diet: Introduce a calorie-restricted, Mediterranean-style diet rich in fruits, vegetables, whole grains, and healthy fats (e.g., olive oil, nuts).
- Avoid saturated fats, trans fats, and fructose-containing drinks (e.g., sugary beverages).
- Physical Activity: Recommend at least 150 minutes of moderate-intensity aerobic exercise per week (e.g., walking, cycling).
- Weight Loss: Target a gradual weight loss of 7-10% over the next 6-12 months.

2. **Medical Management:**

- Metformin: Adjust dosage for better glycemic control in diabetes (target HbA1c < 7%).

Lipid-Lowering Therapy:

- Statin (Atorvastatin): 20 mg daily for dyslipidemia (aim to reduce LDL < 100 mg/dL).

Antihypertensive Medication:

- Continue current antihypertensive therapy (ACE inhibitor) and monitor blood pressure.
- Vitamin E: 800 IU/day (considered for non-diabetic patients with biopsy-proven NASH, but Mr. Dhanush' diabetes and cardiovascular risk may limit its use due to safety concerns).

3. **Monitoring:**

- Reassess liver function tests in 3 months to track enzyme improvement.
- Annual monitoring with non-invasive flbrosis assessments (e.g., transient elastography/FibroScan) to evaluate progression of flbrosis.
- Regular follow-ups to monitor cardiovascular risk factors and reinforce lifestyle changes.

4. **Referral:**

- Refer to a dietitian for a personalized meal plan.
- Consider referral to a hepatologist if there is no improvement in liver function or if progression to cirrhosis is suspected.

Follow-Up (6 Months Later):

Weight: 89 kg (6 kg weight loss, BMI 29) Liver Function Tests:

ALT: 52 U/L

AST: 40 U/L

HbA1c: 7.4%

Lipid Profile:

Total Cholesterol: 190 mg/dL LDL Cholesterol: 110 mg/dL HDL Cholesterol: 42 mg/dL

Triglycerides: 170 mg/

DISCUSSION

This case highlights the close association between NAFLD, metabolic syndrome, and cardiovascular risk. Early intervention through lifestyle modiflcation and management of comorbidities like diabetes, hypertension, and dyslipidemia is crucial for preventing the progression of NAFLD to more severe stages like NASH and cirrhosis.

Both environmental and genetic factors contribute to the development of non-alcoholic fatty liver disease (NAFLD) and its progression. First-degree relatives of patients with NAFLD are at higher risk than the general population. Histone amino-terminal ends maintain the chromatin structure and gene expression that is cAMP-responsive element-binding protein H (CREBH) or sirtuin (SIRT1). Genetic studies have shown that activation of SIRT is thought to play a role in the development of NAFLD. The trigger of the progression of NAFLD to cancer is via abnormal DNA methylation.

The development of NAFLD is multifactorial. The "two-hit" hypothesis has been widely accepted to explain its pathogenesis:

1. First Hit: Hepatic steatosis occurs primarily due to insulin resistance, leading to the accumulation of triglycerides in the liver. This initial phase often remains asymptomatic which leads to the accumulation of fat droplets that are triglycerides in the cytoplasm of hepatocytes, leading to the development of steatosis. Insulin resistance causes excess delivery of free fatty acid and triglycerides to the liver and decreased excretion leading to accumulation. Also, excess carbohydrates are a stimulus for de novo fatty acid synthesis in the liver.
2. Second Hit: Oxidative stress, lipid peroxidation, mitochondrial dysfunction, and inflammatory cytokines (such as TNF-α and IL-6) lead to hepatocellular injury, inflammation, and flbrosis, which characterizes the progression from simple steatosis to NASH.The second hit causing hepatocellular injury and the development of NASH is multifactorial.Excessive fatty acids in the liver make the liver more vulnerable to injury. Peroxisomal fatty acid oxidation, reactive oxygen species (ROS) production from the mitochondrial respiratory chain, cytochrome P450 metabolism of fatty acids, and hepatic metabolism of gut-derived alcohol are hypothesized to cause the injury. Obesity also contributes to the second hit as adipose tissue releases inflammatory mediators such as leptin, tumor necrosis factor (TNF)- alpha, and interleukin (IL)-6, causing hepatocyte damage. The hepatocytes undergo ballooning, cytoskeletal aggregation, apoptosis, and necrosis.

Insulin resistance is also a part of the second hit. The sinusoidal collagen deposition caused by the activation of hepatic stellate cells and the portal flbrosis caused by the ductular proliferation leads to the development and progression of NASH. These changes have correlated with insulin resistance, which is now believed to cause the progression of steatosis to NASH and progressive flbrosis .

CONCLUSION

NAFLD is a rapidly growing cause of chronic liver disease, mirroring the rising incidence of obesity and the metabolic syndrome. Management requires a multidisciplinary approach with clear risk stratiflcation. The therapeutic options for advanced disease stages will signiflcantly improve in the next decade.

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