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Hepatorenal Syndrome with Metabolic Sequelae: A Case Report

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ABSTRACT

When intrinsic kidney damage does not occur, functional renal impairment is the hallmark of Hepatorenal Syndrome (HRS), a serious consequence of advanced liver illness. Two kinds can be identified: Type 1, which is typified by abrupt renal failure, and Type 2, which is characterized by long-term, persistent renal impairment. The etiology of HRS is mostly related to severe renal vasoconstriction, which is impacted by peripheral vasodilation, cardiac dysfunction, sympathetic nervous system activation, and vasoactive mediators.

A 64-year-old woman with a history of pituitary microadenoma, hepatic encephalopathy, secondary adrenal insufficiency, and chronic liver disease manifested as widespread weariness, discomfort, and insomnia. Hyperpigmentation, bilateral NVBS (non-specific findings), and a history of repeated hospital hospitalizations were discovered during the physical examination. Liver parenchymal alterations, portal hypertension, and related disorders such as splenic hemangioma and cholelithiasis were all seen on ultrasonography. Tests in the lab revealed high levels of bilirubin and liver enzymes.

A multidisciplinary approach to care is crucial since this case emphasizes the complexity of HRS in the context of advanced liver disease

INTRODUCTION

An altered kidney function known as hepatorenal syndrome (HRS) is a condition that affects people with severe liver disease. The kidneys are not structurally harmed in people with hepatorenal syndrome, nor is there a known cause of their kidney impairment. As a result, hepatorenal syndrome could be described as a "functional" type of kidney damage.

Two HRS subtypes have been identified: Type 1 HRS is characterized by a 50% decline in creatinine clearance to a level of less than 20 milliliters per minute in less than two weeks, or by doubling initial serum creatinine to a level greater than 2.5 mg/dl. Serum creatinine greater than 1.5 mg/dl indicates type 2 HRS, a mild and persistent renal failure. While type 2 HRS develops gradually and is the primary underlying cause of refractory ascites, type 1 HRS usually has a precipitating event identified.[2]

Severe renal vasoconstriction, which begins early and gets worse as the liver disease worsens, is a hallmark of HRS. Although the underlying mechanisms causing HRS are poorly understood, they may involve components operating on the renal circulation that are both lowered and elevated in vasoconstrictor activity. The pathophysiology of HRS has been linked to four interrelated mechanisms. Each of these

mechanisms may have a different effect on renal vasoconstriction and the onset of HRS in different patients. Some of those paths are:

1. renal vasoconstriction that follows peripheral arterial vasodilation with hyperdynamic circulation; 2. stimulating the sympathetic nervous system (SNS) in the kidneys;3. renal hypoperfusion and circulatory disturbances caused by cardiac failure; 4. various vasoactive mediators and cytokines affect renal circulation and other vascular beds.[3]

In patients with severe liver disease, hepatorenal syndrome develops when the kidneys fail to function properly. The body produces less urine, which causes nitrogen-containing waste materials to accumulate in the circulation (azotemia). Orthostatic hypotension, diuretic usage, gastrointestinal bleeding, infection, and paracentesis are risk factors. A few symptoms are ascites, decreased urine production, mental disorientation, jerky movements of the muscles, dark urine, nausea, vomiting, weight gain, and jaundice.[4]

Key requirements for the diagnosis of HRS include advanced liver failure with portal hypertension; elevated levels of the organic acid creatine; lack of other kidney-toxic drug use, shock, or bacterial infection; no improvement in renal function with diuretic withdrawal and increased plasma with albumin (a liver-produced protein that is low in liver disease patients); low levels of protein in the urine without evidence of parenchymal renal disease or urinary disease (uropathy).[1]

A liver transplant corrects both the liver disease and the corresponding reduced renal function, making it the sole curative therapy for patients with hepatorenal syndrome. People with a history of hepatorenal syndrome may not entirely restore their kidney function even after a successful liver transplant. Only a tiny portion could develop irreversible damage that requires dialysis.[1]

CASE REPORT

A 64-year-old female patient complained of generalized tiredness, unexplained discomfort, and altered sleep disturbance and was admitted to the department of general medicine. Upon physical examination, the patient was hyperpigmented throughout the face and hands, bilateral NVBS was visible in the chest, and the patient was conscious and oriented. She had a history of many admissions for hepatic encephalopathy, pituitary microadenoma, decompensated CLD, and 2◦ adrenal insufficiency, for which she has been receiving steroid replacement treatment since 2014.

On evaluation, ultrasonography revealed characteristics of long-term liver parenchymal disease, including portal hypertension, cholelithiasis, regenerative nodule, mild gall bladder wall oedema, reactive splenic cyst, small hyperechoic lesion in spleen-hemangioma, and mildly enlarged uterus.

The lab tests indicated that the levels of ALP and total and direct bilirubin were higher than normal. After consulting a gastroenterologist, it was recommended to keep up the anti-hepatic coma treatments.

Cefoperazone and sulbactam IV antibiotics, Rifaxamine, Proton pump inhibitors, tabs, carvedilol, antihypertensives, anti-hepatic coma measures, in. hydrocortisone, OHA, insulin (HbA1c-9.8%), tabs of thyroxine (TSH-0.2, FT4-0.7, FT3-1.64), and other supportive measures were administered to the patient. By the end of the course, the patient's symptoms were improved.

After being discharged, she requires more follow-up in the endocrinology department.

CONCLUSION

The complicated pathophysiology of hepatorenal syndrome, a serious consequence of liver illness, involves renal and systemic circulation disruptions. The definitive treatment for reversing both liver and renal failure is liver transplantation, thus accurate diagnosis and care are essential. Enhanced care and ong-

oing monitoring are crucial for maximizing results and resolving concerns arising after therapy.

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