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Case Report

Liver Metastasis: An Unsung Etiology of Hepatorenal Syndrome

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ABSTRACT

Hepatorenal syndrome is a reversible acute kidney injury affecting patients with acute liver failure, cirrhosis or acute on chronic liver failure. The tsunami of metastatic liver disease and their various presentations has always intrigued the clinicians. Liver metastasis attributing to hepatorenal syndrome in otherwise normal liver parenchyma is a less considered and often an ignored etiology. Secondly less aggressive treatment approach of such patients has also shielded its presence. We present a case of pancreatic malignancy with liver metastasis who had Icterus, ascites, coagulopathy and altered sensorium. In due course of stay in the hospital patient's urine output reduced and serum creatinine got elevated. Diagnosis of hepatorenal syndrome was made and treatment was initiated. Outcome of such patients on a large scale could provide a better picture for their prognosis and at the same time empower efforts of clinicians.

KEYWORDS

Hepatorenal syndrome, Liver metastasis, Acute kidney injury, Metastatic cancer, Parenchymal liver diseases

INTRODUCTION

Hepatorenal syndrome is a reversible acute kidney injury occuring in patients with acute liver failure, cirrhosis or acute on chronic liver failure. The complex of renal vasoconstriction, causing reduction in glomerular filtration rate, splanchnic vasodilatation causing decreased systemic vascular resistance, cardiocirculatory dysfunction secondary to portal hypertension along with inflammation paves the path of it's pathogenesis [1].

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The increasing burden of metastatic liver disease and their various presentations has always intrigued the clinicians. There has been a systematic review and metanalysis on pseudocirrhosis and portal hypertension in patients with metastatic liver disease [2]. But there are a very few descriptions of malignancy and metastasis associated hepatorenal syndrome in otherwise normal liver parenchyma making it a rather less considered and often ignored etiology.

PRESENTATION OF CASE

A 78 years old male patient presented with chief complaints of on and off pain since 2 weeks, reduced appetite, abdominal distension and fatigue. He was non-diabetic (HBA1C – 4.6), non-hypertensive, a non-smoker and non-alcoholic. On clinical examination patient was icteric, on abdominal examination shifting dullness in the abdomen was present proving significant ascites. Respiratory, cardio vascular and central nervous systemic examination were unremarkable.

Patient was admitted in general ward where he received medications and supportive care. On computed tomography; a pancreatic head mass and multiple low-density lesions in the liver consistent with pancreatic head cancer with liver metastases. Positron emission tomography-computed tomography (PET-CT) showed a pancreatic mass with high 18F-fluorodeoxyglucose (FDG) uptake, suggesting metastasis, liver metastasis. An endoscopic ultrasound guided biopsy was suggestive of poorly differentiated adenocarcimona. The patient was not stable for liver biopsy thus was deferred. His carcino embryonic antigen and CA-19.9 were both elevated being >10000 and >25000 respectively. In the light of above reports and clinical presentation diagnosis of pancreatic cancer with liver metastasis was kept.

The patient had deranged liver function tests with raised sgpt, sgot, ggt being 113,632 and 517 respectively. Total bilirubin, direct bilirubin and indirect bilirubin were 12.66,6.60 and 6.06 respectively. Total protein, albumin, globulin and albumin globulin were 7.83,3,34,4.48,0.75 respectively. Patient's prothrombin time was 38.3 and international normalized ration were 38.3,3.5 respectively. Patient didn't have any bleeding manifestation in the form of epistaxis, malena, echymosis or petechiae.

Patient on due course of stay at hospital developed reduced urine output from 900 ml/day ON DAY 1 to 300ml/day on day 3. This reduction in urine output progressed from oliguria to anuria on day 6. Meanwhile serum creatinine which was 0.7mg/dl also increased to 1.2 on day 2 of admission to 2.1 on day 3 and it progressed further. By the time patient was anuric on day 6 it was 4.7. During this time frame patient developed shortness of breath and altered mental status for which he was shifted to medical intensive care unit. During this period he received albumin infusion for 3 days. Creatinine didn't improved on diuretic withdrawal as well.

Urine routine, microscopic and culture were unremarkable. Ultrasound done was suggestive of 11.5 x 4.8 cm right kidney with preserved cortico medullary differentiation. Left kidney being 9.7 x 5.1 cm with preserved cortico medullary differentiation. Urinary bladder was normal. Prostrate was normal in size, shape and parenchymal echopattern. It weighed 17 gm. No renal parenchymal abnormality was seen.

In MICU he received nursing care, Non-invasive ventilatory support, intravenous medications including antibiotics. Nephrotoxic drugs were being avoided right from the very beginning of decrease in urine output and raised serum creatinine levels. All antibiotics were being administered in renal dosage. Gastro-medicine and nephrology teams concluded on the point that it was a case on hepatorenal syndrome and hence terlipressin was initiated at a low dosage 1mg/24 hour as infusion.

The urine output improved and patient became oliguric from anuric but after some time he again went into anuric phase. Attendants were counselled regarding the need for dialysis but they refused for an aggressive approach; hence the patient was not offered haemodialysis sessions.
We lost this patient the following day.

DISCUSSION

As per International ascitic club the diagnostic criteria of hepatorenal syndrome includes cirrhosis, acute liver failure or acute on chronic liver disease, increase in serumcreatinine >/= 0.3mg/dl within 48 hours or >/=50% from baseline value and/or urinary output </=0.5ml/kg/body weight >/=6 hours, with no full/partial response after 2days or diuretic withdrawal and volume expansion with albumin, in absence of shock, no current/recent treatment of nephrotoxic drugs, absence of renal parenchymal disease, evident by hematuria, proteinuria or an obstructive uropathy [1].

There are a few case reports describing hepatorenal syndrome in patient secondary to metastatic liver disease.
Rashidi A et al. [3] in his article -Hepatorenal syndrome in metastatic Cancers; described 3 patients having hepatorenal syndrome associated with metastatic breast cancer and cholangiocarcinoma [3]. Rosansky S J et al. [4] has also described a patient who developed the hepatorenal syndrome associated with an angiosarcoma of the gallbladder that metastasized to the liver; in the article. The Hepatorenal Syndrome Associated with Metastatic Angiosarcoma of the Gallbladder [4].

Recently a systematic review and metanalysis done by Villani R et al. [2] named Pseudocirrhosis and portal hypertension in patients with metastatic cancers: a systematic review and meta-analysis has described an entity known as pseudocirrhosis and its manifestations. They defined pseudocirrhosis as a clinical and radiological entity mimicking liver cirrhosis in patients without a history of chronic liver disease. 80% of the patients in this metanalysis had breast cancer. Most patients had at least one clinical sign of portal hypertension and ascites was

the most common one. They concluded that pseudocirrhosis may have a negative impact on survival and clinical management of patients because of the potential development of portal hypertension and its complications [2]. Interestingly portal hypertension is also a contributor to pathogenesis of hepatorenal syndrome.

CONCLUSION

With plenty of metastatic liver diseases being diagnosed and with advent of newer therapies; there has been a paradigm shift in their clinical presentation. Our patient is a classical example of hepatorenal syndrome secondary to chronic liver disease due to metastasis. Icterus, ascites, coagulopathy, altered sensorium and finally hepatorenal syndrome; the patient had all manifestations of chronic liver insult. Early diagnosis of hepatorenal syndrome becomes a priority in such case. It can slow the progression of acute kidney injury and help in early initiation of renal replacement therapy. However aggressive management of such cases becomes a tough decision for the relatives and a new chapter of prognosis for such patients. Hence while dealing oncology cases gastro-medicine and nephrology teams should be keen enough to start recognizing liver metastasis as an etiology of hepatorenal syndrome.

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