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Case Report On Hepatorenal Syndrome

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ABSTRACT

Hepatorenal syndrome (HRS) is a potentially reversible renal impairment where the kidney function is stopped due to renal vasoconstriction resulting from extreme vasodilation. This case report describes a 64 year old female with Decompensated Chronic Liver Disease. Her clinical history and physical findings shows a decreased renal function secondary to chronic liver disease and was later diagnosed as HRS. HRS can be managed effectively with Terlipressin, Midodrine, Albumin. Such combination therapy at the early stage of diagnosis can prevent further complications.

Keywords: HRS, CLD, Albumin, Terlipressin, Midodrine.

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INTRODUCTION

HRS is a potentially reversible renal impairment where the kidney function is stopped due renal vasoconstrictions resulting from extreme vasodilation. It is a progressive kidney failure usually seen in patients with severe liver damage. HRS is relatively common, with an incidence of 40% seen in patients with cirrhosis and ascites. Prevalence of HRS in patients with cirrhosis and ascites ranges from 13 to 45.8%. [1] High mortality rate is associated with HRS, if left untreated.

HRS is categorized into two types based on the rate of progression of renal dysfunction. Type1 HRS is characterized by abrupt onset, rapidly progressive kidney failure with over production of creatinine and very short survival without treatment; whereas, Type 2 HRS is gradual damage which progress more slowly and longer survival compared to Type 1.[2] The international ascites club (IAC) has provided a set of criteria revised in 2005 to aid in the diagnosis of HRS(Table 1).

Table 1

1. Cirrhosis with ascites.
2. Serum creatinine >133µmol/L (1.5mg/dl)
3. No sustained improvement of serum creatinine after atleast 2 days of diuretic withdrawal and volume expansion with albumin.
4. Absence of shock.
5. No current or recent treatment with nephrotoxic drugs.
6. Absence of parenchymal disease as indicated by proteinuria (>500mg/day), microhematuria, and or abnormal renal ultrasonography.

(Available from: https://www.icascites.org/about/guidelines/, updated 2005 Nov; cited 2014 Nov 16).

Management of HRS is highly challenging since the preferred treatment is liver transplantation which is difficult to accomplish within a short span of time. Therefore patients with HRS are typically managed pharmacologically with albumin, midodrine, and vasopressin analogues with a goal to improve renal function and survival as abridge to transplantation.[3]

Here, we present a case of Type 1 HRS that is successfully treated with midodrine, terlipressin and albumin therapy. This report serves to highlight the role of healthcare practioners in diagnosing HRS and starting its early treatment.

CASE REPORT

A 64 year old Indian women presents a history of T2DM for 2 years and decompensated CLD diagnosed 1 year previously. Since that time she was on Inj. Human Actrapid 16-0-8, Inj. Insulin Lantus 0-0-16, T. Dytor 10 mg OD, T. Cardivas 2.125 mg BD, T. Ursocol 500 mg BD. One year back, she had a surgical history of post ovarian malignancy. She was admitted to gastroenterology

department with complaints of generalized weakness and decreased appetite for 5 days. Her clinical findings were relevant for a blood pressure of 130/90 mmHg, heart rate 86 beats/min. Patient was tired and drowsy. She was presented with ascites and her abdomen was soft and distended on evaluation. Pitting edema was noted in bilateral lower limbs. There were no significant findings from cardiovascular and respiratory examination.

Hematological investigations were performed. Patient was found to be anemic, with Hb count of 9.9 mg/dl at the time of admission. Renal function tests shows markedly elevated serum creatinine level of 2.0 mg/dl. LFT results also shows an elevated AST and ALT levels, ie, 97 U/L and 74 U/L respectively. Patient was hypokalemic with serum potassium level of 2. 9 meq/L. She had an elevated FBS level for all days of admission.

Table 2: laboratory investigations and findings during the time of admission.

Parameters	Patient value	Normal value
Creatinine (mg/dl)	2.0	0.6-1.4
Hb (g%)	9.9	12.5-15.5
K^+ (meq/l)	2.9	3.5- 4.5
FBS (mg%)	283	70-100
AST (U/L)	97	<49
ALT (U/L)	74	<46

On the background of her past medical and medication history and her elevated serum creatinine level patient was suspected to have Type1 HRS. In order to confirm that clinician advised for diuretic withdrawal. With prompt diagnosis, she was started with vasopressin analogue Inj. Terlipressin 1 mg over 2 hours, T. Midodrine 2.5 mg BD and volume expansion with albumin 5% over 1 hour during the entire course of hospital stay. During the therapy, patient shows improvement in serum creatinine level(S.Cr decreased to 1.4 mg/dl). She was hypokalemic and in order to correct her low K^+ level, syrup Keylite has been prescribed. Her elevated FBS was managed by insulin therapy, ie, Inj. Human Actrapid 14-0-8 and Inj. Lantus 0-0-6. Since the patient was suffering from anemia, her low Hb level was corrected with blood transfusion with 1 pint PRBC for 4 hours, two times on the first 2 days.

DISCUSSION

This case report has ruled out that, early diagnosis and further management of HRS, helps to prevent complications to a certain extent. HRS can be diagnosed with the aid of criteria developed by IAC. Pathogenesis of HRS is currently still being explored. Arterial vasodilation of splachnic circulation associated with local release of vaso dilatory substance is considered as a possible explanation. The renal vasoconstriction by activation of renin angiotensin aldosterone, cardiac

dysfunction of cirrhosis, increased circulation of cytokines and vasoactive substances that affect blood circulation.[4]

The mainstay of therapy for patient with HRS remains liver transplantation. It helps in HRS reversal and can improve survival rate among the patients. However liver transplantation may not be suitable for all HRS patients, because it may take long waiting time at most transplant centres. Several pharmacological options are available for the management of HRS. The clinical management of HRS syndrome is currently based on the vasoconstrictors in association with Albumin.[5]

The acute dialysis quality initiative (ADQI) work group recommends the use of vasoconstrictor drugs combined with plasma expansion with albumin, as first line treatment for Type1 HRS. The vasopressin and its analogue like Terlipressin and Ornipressin are the vasoconstrictors that act on splanchnic circulations, are the most effective management of HRS.[6] Midodrine another vasoconstrictor is also effective in HRS management. Midodrine, act on alpha1 adrenergic receptor in vascular smooth muscles, thus increasing effective circulating blood volume and renal perfusion by increasing systemic and splanchnic blood pressure.[7] The use of albumin together with vasoconstrictors has shown to provide good therapeutic outcome and are effective in the management of HRS.

CONCLUSION

In conclusion, we reported a case of Type1 HRS that is successfully treated with Midodrine, Terlipressin, and Albumin. Our study concludes that early diagnosis and proper management of HRS shows an improvement in the survival rates of the patients, when compared to those left untreated. The proper pharmacological management of HRS can reduces the need for liver transplantation to a certain extent.

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