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Original Research Paper

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PERI-OPERATIVE MANAGEMENT OF LIVER CIRRHOSIS WITH HEPATO-RENAL SYNDROME: CASE SERIES

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ABSTRACT

INTRODUCTION: Liver Transplant is a highly challenging and demanding procedure for the anaesthesiologist. The surgery is usually lengthy and complicated with critical patient condition. HEPATORENAL SYNDROME is a serious complication of Liver Cirrhosis with critically poor prognosis.Pathophysiological hallmark is Renal Vasoconstriction because of complex changes
In Splanchnic vasoconstriction and general circulation.The definite treatment is Liver Transplant.

METHODS: 5 cases of Chronic Liver Cirrhosis with Hepatorenal Syndrome were studied retrospectively.
Three patients had NASH -related Cirrhosis, two had Alcoholic Liver disease.Patients
were optimised preoperatively either by vasoconstrictor therapy or Renal replacement therapy.All were induced using the same Anaesthetic agents and invasive monitoring of BP, CVP and PAP was employed for all.Intraoperative intravascular volume was maintained based on hourly input-output data.Coagulation control was guided by intra-operative TEG.

RESULTS: Three patients had intra-op fluid intake more than output, renal function was regained earliest in the one with min input-output difference.Two patients had intra-op output more than input, renal function was regained almost immediately. patients who received intra-op continuous renal replacement therapy; regained renal function last .

CONCLUSION:
Most cases of renal dysfunction in cirrhosis are functional.Vasocontrictor therapy, renal replacement therapy improve short-term outcome and buy time for
Liver transplant.A thorough assessment of cardiovascular,renal,hematological,fluid and electrolyte status of the patient and a good knowledge of the pathophysiology of HRS and its anaesthetic implications are mandatory for successful management.

KEYWORDS : Liver Transplant, HRS

INTRODUCTION:

Liver Transplant is a highly challenging and demanding procedure for the anaesthesiologist. The surgery is usually lengthy and complicated with critical patient condition. HEPATORENAL SYNDROME is a serious complication of Liver Cirrhosis with critically poor prognosis. It is defined as volume unresponsive, refractory pre-renal azotemia in patients with chronic liver disease, characterised by systemic and splanchnic vasodilatation but profound renal vasoconstriction, without parenchymal kidney injury[1]. There are two types of HRS. Type-1 is a rapidly progressive acute renal failure which is usually temporally related with a precipitating factor like infection(SBP) or large volume paracentesis. Type-2 HRS is a slowly progressive renal failure. It reflects the cardiac dysfunction that is unavoidable in a cirrhotic patient[2]. Treatment strategies include improvement of renal function by volume replacement, splanchnic vasoconstrictors or reversal of portal hypertension by TIPPS or Liver Transplant. The definite treatment is Liver transplant. Maintaining intravascular volume, coagulation control and venous outflow to hepatic allograft impose major anaesthetic challenge. A thorough assessment of cardiovascular, renal, haematological, fluid and electrolyte status of the patient and a good knowledge of the pathophysiology of HRS and its anaesthetic implications are mandatory for successful management.

We studied 5 cases of cirrhosis complicated by HRS retrospectively with the objective of

1) Assess return of renal function post Liver Transplant and correlate with pre-op and intra-op management.
2) Characterise the current peri-operative management and to suggest evidence-based guidelines for the anaesthetic management.

METHODS:

A retrospective analysis of 5 patients with Chronic Liver Cirrhosis complicated by HRS who underwent Liver Transplant was done. 3 patients had NASH cirrhosis , 2 patients had Alcoholic cirrhosis.

Patients were optimised preoperatively either by vasoconstrictor therapy or Renal replacement therapy. All were induced using the same protocol. Intraoperatively intravascular volume was maintained based on hourly input-output data, using crystalloids and albumin. Blood loss was replaced with whole blood. Coagulation control was guided by intra-operative TEG (thromo boelastography).

RESULTS:

TABLE 1: Patient Data

	Diagnosis	MELD	Baseline Creatinine	Intra-op fluid balance	Treatment modalities	POD 10 S.Creat
A	NASH	23	2.8	I<O	CRRT+Alb + Terli.	1.47
B	NASH	19	1.6	I=O	Alb+ NA	0.82
C	NASH+DM	22	2.4	I>O	Alb+ NA	0.86
D	ALD	18	1.78	I>O	Alb+ NA	0.42
E	ALD	23	1.88	I<O	Alb+ Terli.	1.40

- Patients (B, C, D) in whom positive fluid balance was maintained ; implying adequate intravascular volume, gained renal function earlier as shown by serum Creatinine values on 10th post-operative day (POD-10).
- Patients (A, D) who had negative fluid balance regained function late with adequate vasoconstrictor therapy continued into the post op period. Instead of the usual Nor -adrenaline used in other cases Inj. Terlipressin was used in these cases. These patients had the highest MELD score amongst the 5 cases.
- The patient (A) who required pre-op and intra-op CRRT (continuous renal replacement therapy) regained function the last. A negative balance was maintained on purpose to avoid

overloading the system. Post op vasoconstrictor therapy was continued.

*I = Input
O = output
CRRT = continuous renal replacement therapy
Alb = Albumin
NA = Nor - adrenaline
Terli.= Terlipressin
NASH = non - alcoholic steatohepatitis
ALD = Alcoholic Liver Disease
DM = Diabetes Mellitus

MELD = Model for end stage liver disease score

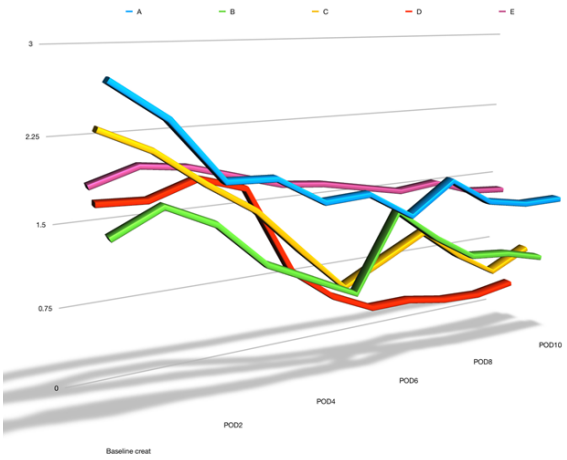

FIGURE 1 : This graph shows the serum creatinine trend over 10 days post Liver transplant.

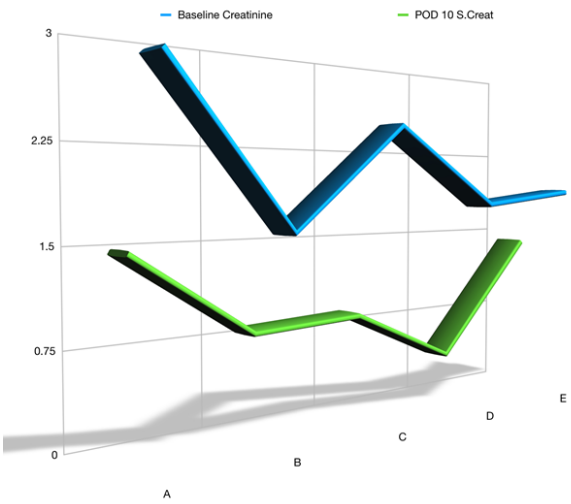

FIGURE 2: This figure shows a comparison of baseline creatinine before transplant with post liver transplant 10 th day serum creatinine values.

DISCUSSION:

HRS is a major decompensation in advanced chronic liver disease .HRS has a very poor prognosis (increased short term mortality) with high recurrence and resource utilisation rates.

Frerichs and Flint (1861) reported Oliguria in the absence of renal hisotological changes in patients with advanced cirrhosis and ascites. Term "HRS" was used in 1939 to describe the occurrence of renal failure after biliary surgery in patients showing a pathological pattern of ATN or TIN. Koppel et al. in 1960 also showed that kidneys from patients who succumbed from HRS functioned normally when transplanted into patients with chronic uraemia.

AKI (acute kidney injury) is seen in 15-20% cases of cirrhosis and 40-50% cases of cirrhosis with ascites. HRS is seen in < 25% cases of AKI in cirrhosis[3]. The diagnosis of HRS is made by the following criteria -Cirrhosis with ascites, S. Creatinine > 133umol/lt (1.5mg/dl), no improvement in serum creatinine after at least 2 days of diuretic withdrawal and volume expansion with albumin (the recommended dose of albumin is 1gm/kg/day up to a maximum of 100gms/day), absence of Shock, no current or recent treatment with nephrotoxic drugs, absence of renal parenchymal disease, proteinuria > 500mg/day, microhematuria (>50 RBCs/hpf) and/or abnormal USG [4]. HRS can be precipitated by bacterial infections, large volume paracentesis, GI bleeding, acute alcoholic hepatitis etc. Cirrhotic cardiomyopathy and worsening hyper dynamic circulation lead to further increase in renal vasoconstriction.

Actuarial probability to survive in cirrhotic patients with HRS- Mean Survival without treatment ➢HRS1 : 10% at 90 days ➢HRS2 : 6 months, with treatment ➢3 months survival ➢HRS1 20% ➢HRS2 40%. Treatment strategies include improvement of renal perfusion by volume expansion, splanchnic vasoconstriction; Reversal of portal hypertension by TIPSS or Liver transplantation. The definite treatment however is Liver transplant. Albumin at 1gm/kg/day (100gms max) followed by 25 to 50 gms per day until therapy continued is used for improving renal perfusion. Barcelona group showed patients receiving albumin prevented development of RF (10% vs 33%, P 0.002) and reduced short term mortality at 3 months in recently developed HRS(22% vs 41%, P = 0.03) [5]. Vasoconstrictor therapy includes the use of Terlipressin or Nor adrenaline. Midodrine + Octreotride (Selective alpha1 Adr agonist, Long acting Somatostatin analogue) are newer strategies for improving renal perfusion in HRS complicated cirrhotic patients. Patients with SIRS have registered good response to Terlipressin vs placebo (42.9% vs 6.7%) [6].

CONCLUSION:

Three patients had intra-op fluid intake more than output, renal function was regained earliest in the one with min input-output difference.Two patients had intra-op output more than input, renal function was regained almost immediately. patients who received intra-op continuous renal replacement therapy; regained renal function last. Vasoconstrictors [terlipressin + albumin] are effective in less than 50% of HRS patients. Liver transplantation is the only effective treatment. Most cases of renal dysfunction in cirrhosis are functional.Vasocontrictor therapy, renal replacement therapy improve short-term outcome and buy time for Liver Transplant. Thorough assessment of cardiovascular,renal,hematological,fluid and electrolyte status of the patient and a good knowledge of the pathophysiology of HRS and its anaesthetic implications are mandatory for successful management.

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