Spontaneous peritoneal drainage following paracentesis in a hospitalized patient with resolution of type 1 hepatorenal syndrome

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ABSTRACT

The hepatorenal syndrome develops in a small percentage of patients with advanced liver disease. The pathogenesis involves intravascular volume contraction secondary to pooling of blood in the splanchnic vessels, stimulation of the sympathetic nervous system and the renin–angiotensin–aldosterone pathway, and increased intra-abdominal pressure secondary to the formation of large volumes of ascitic fluid. The treatment options are limited, and liver transplant is the only definitive form of management. Here we suggest an alternative approach to treating hepatorenal syndrome based on the unexpected continuous peritoneal drainage in a 36-year-old man hospitalized with hepatic encephalopathy and hepatorenal syndrome. A total of 11.2 L ascitic fluid drained over 5 days from a paracentesis puncture site with marked improvement in renal function; the creatinine decreased from 3.3 mg/dL to 0.7 mg/dL and the BUN decreased from 42 mg/dL to 10 mg/dL. The discussion with this case report summarizes the pathogenesis, including the effect of intra-abdominal pressure, of the hepatorenal syndrome, outlines medical management, and makes a proposal for clinical study based on this case.

Keywords: Hepatorenal syndrome; cirrhosis; renal function; paracentesis. **[Am J Med Sci 2022;364(6):789–795.]**

INTRODUCTION

Hepatorenal syndrome (HRS) can be defined as the development of acute kidney failure in patients with severe liver disease.[1] It occurs in approximately 4% of patients with cirrhosis. The mechanisms are complex and involve vasoconstriction of the renal vasculature in response to vasodilation in the splanchnic circulation. This vasoconstriction leads to renal hypoperfusion and kidney dysfunction. The clinical manifestations include signs of liver disease, such as jaundice, splenomegaly, hepatic encephalopathy, and ascites, and signs of renal disease, such as oliguria and increasing creatinine levels. This syndrome has a high mortality rate, treatment options are limited, and liver transplantation is the only curative therapy.[2]

CASE PRESENTATION

A 35-year-old man with a past medical history of alcohol abuse with decompensated liver cirrhosis (Child Pugh Class C), hypertension, and depression presented to the emergency department with an altered mental status of one day's duration. Physical examination was notable for confusion, jaundice, icteric sclerae, ascites with a fluid wave, mid-abdominal tenderness, and bilateral leg swelling up to the mid-tibia level. Vital signs included a heart rate of 120 beats per minute, a blood pressure of 132/74 mmHg, and a normal temperature. Laboratory tests included a hemoglobin of 9.9 gm/dL, hematocrit 27.7%, platelet count 81 K/μL, hyponatremia (124 mmol/L), blood urea nitrogen (BUN) 28 mg/dL, creatinine (Cr) 2.2 mg/dL, alkaline phosphate 169 IU/L, alanine transaminase 32 IU/L, aspartate aminotransferase 66 IU/L, total bilirubin 14.1 mg/dL, ammonia 216 mmol/L(normal range:10-60 mmol/L), albumin 3.2 gm/dL, total protein 8.3 gm/dL, and international normalized ratio 2.4. His serum creatinine 5 days prior to admission was 1.3 mg/dL. Urinalysis revealed protein 30 mg/dL and 28 WBCs per high-powered field. Initial urine chemistries included sodium < 20 mmol/L, chloride < 20 mmol/L, potassium 53.1 mmol/L, and creatinine 243 mg/dL. Abdominal ultrasound showed cirrhosis, portal vein hypertension with varices and a recanalized umbilical vein, hepatopedal flow in portal vein, and large volume ascites. Head computed tomography scan without contrast was unremarkable for acute intracranial pathology. The patient's Model for End Stage Liver Disease (MELD) score on admission was 34 (52.6 % estimated 3-month mortality), and his serum albumin-ascites gradient (SAAG)

Table 1. Drainage output and kidney function during spontaneous drainage

Hospital Day	Drainage (ml)	Urine Output (ml)	Albumin gm/dL	BUN mg/dL	Creatinine
1	Paracentesis initiated	450	2.9	28	2.2
2	Drainage blocked	400	2.6	34	2.6
3	3950	811	2.6	42	3.3
4	3100	650	3.1	35	2.4
5	1375	950	3.4	23	1.2
6	1300	1000	3.0	13	0.7
7	1500	490	2.7	10	0.7

was 2.7 gm/dL (consistent with portal hypertension). A transjugular intrahepatic portosystemic shunt (TIPS) procedure was not feasible due to his MELD score, his hepatic encephalopathy, and his decompensated state. The patient was diagnosed with hepatic encephalopathy and acute kidney injury and was started on lactulose (20 gm, TID) with titration to three to four bowel movements/day.

Paracentesis was performed in the emergency department with no record of the volume of fluid removed, and a peritoneal drainage catheter was left in place. On hospital day 3, lactulose was held due to worsening acute kidney injury (Cr 3.3mg/dL, BUN 42 mg/dL). The peritoneal drainage catheter was removed due to blockage, but peritoneal fluid continued to leak at the insertion site. A urostomy bag was then placed over the puncture site, and the peritoneal fluid continued to drain for seven days. The patient received 87.5 g of albumin on the first 2 days of this spontaneous drainage phase. He was also placed on midodrine 2.5 mg 3 times daily and octreotide 50 micrograms IV per hour for 48 hours. He did not receive norepinephrine or diuretics. His mean arterial blood pressure was 82.2 mmHg on the day before midodrine was started and was 88.2 mmHg on the day after it was started. Kidney function improved in association with continuous drainage; the BUN decreased from 42 mg/dL to 10 mg/dL and creatinine decreased from 3.3 mg/dL to 0.7 mg/dL (Table 1). The estimated total urostomy bag drainage was 11.2 L. Bladder pressures were not measured. The patient recently underwent liver transplantation without any short-term complications.

DISCUSSION

Hepatorenal syndrome can be defined as acute kidney injury in a patient with advanced cirrhosis.[1] Type 1 HRS evolves over a time period of less than 2 weeks. Type 2 HRS develops over a longer period of time. These patients have dilatation of the splanchnic vasculature with pooling of blood in the abdomen and a decrease in effective arterial volume. The decreased blood volume stimulates the sympathetic nervous system and the renin-angiotensin-aldosterone system which promote renal vascular constriction and salt and water retention. These patients also develop significant ascites which increases intra-abdominal pressure. The increased intra-abdominal pressure can increase renal vein pressure which in turn decreases the glomerular filtration rate (GFR). The increased extrarenal pressure can cause interstitial edema in the kidneys.[1] In addition, the hepatorenal reflex can also decrease renal blood flow and GFR and potentially contribute to the development of HRS.[1]

Allegretti et al. studied 120 patients with cirrhosis and AKI and classified the type of renal failure with specific criteria.[3] Forty patients had prerenal failure, 35 had HRS, 36 had acute tubular necrosis, and nine had miscellaneous disorders. The mortality at 90 days was 57% and 58% in patients with HRS and acute tubular necrosis, respectively. In the entire cohort, 38 patients required renal replacement therapy, 21 required liver transplantation, 49 were treated with vasopressors, and 31 were treated with midodrine. This study provides an overview of the causes of AKI in patients with cirrhosis and the management approaches and indicates that these patients need a careful evaluation to establish the correct diagnosis for renal failure. Angeli and the International Ascites Club have published a consensus recommendation for the diagnosis and management of acute kidney injury (AKI) in patients with cirrhosis using criteria based on baseline creatinine, creatinine increase for staging, progression of AKI, and response to treatment.[4] These investigators suggested type I HRS should be called HRS−AKI. They also suggested that AKI should be diagnosed when the serum creatinine increases by 0.3 mg/dL or more within 48 hours. This definition potentially identifies patients at an earlier phase of renal dysfunction which could lead to improved outcomes.

MANAGEMENT

The various approaches to the management of HRS have been based on the underlying pathophysiology.[1] Diuretics should be discontinued, and fluid restriction may be needed to prevent dilutional hyponatremia, provided the volume status is satisfactory. Vasoconstrictors can reverse the dilatation of the splanchnic vessels, inhibit the activity of the entire vasoconstrictor system, and subsequently increase renal blood flow and perfusion. Transjugular intrahepatic portosystemic shunts can increase systemic circulation, decrease

vasoconstriction, and increase renal perfusion and GFR but increases the risk of encephalopathy.[5] Hemodialysis and extracorporeal albumin dialysis have improved renal function in a small number of patients. The most definitive approach is liver transplantation which replaces the cirrhotic liver. However, an initial decrease in GFR often occurs with liver transplantation, often requiring hemodialysis and delaying the administration of medications, such as cyclosporine or tacrolimus. Furthermore, patients with HRS often have a short survival time and finding a liver donor can be difficult in the time period available; many patients die before transplantation. These approaches are discussed in more detail in the following paragraphs.

Medical management with albumin infusions

Patients with HRS−AKI often receive albumin infusions, especially if the serum albumin level is low or intravascular volume depletion is present. China et al. randomized 777 patients hospitalized with decompensated cirrhosis.[6] The majority of these patients had new onset or worsening ascites. The treatment arms included the administration of 20% human albumin to maintain an albumin level of greater than 3 gm/dL for 14 days. The composite outcome included new infection, kidney dysfunction, or death between days 3 and 15 after the initiation treatment. There was no difference in outcomes between patients treated with standard-care and patients treated with standard-care plus albumin infusions. The composite outcome occurred in 29.7% of patients in the albumin group and in 30.2% of patients in the standard care group. The incidence of kidney dysfunction was 10.5% and 14.4%, respectively. The baseline serum creatinines were 0.75 mg/dL and 0.78 mg/dL. Patients receiving albumin had more frequent episodes of pulmonary edema and/or fluid overload. Therefore, the infusion of albumin to maintain albumin levels greater than 3 gm/dL does not appear to have any benefit in patients with cirrhosis and large volumes of ascites. The patient in this case report did receive albumin on 2 days early in his hospitalization.

Medical management with vasopressors

Dilation of the splanchnic vasculature causes fluid accumulation in the abdomen and decreases effective arterial volume. Consequently, patients often receive vasoconstrictors during the initial management of HRS −AKI. Midodrine (a selective alpha 1 adrenergic agonist and systemic vasoconstrictor) combined with octreotide (a somatostatin analog/ splanchnic vasoconstrictor) can improve renal and systemic hemodynamics. A retrospective study of patients with HRS demonstrated that patients (n=60) treated with midodrine and octreotide and albumin have lower mortality (43% vs. 71%) and higher resolution rate of HRS (40% vs. 10%) than patients (n=21) who did not receive this treatment.[7] Mahmoud and co-authors compared the outcomes of 60 patients with HRS−AKI treated with either midodrine/ octreotide or norepinephrine.[8] The response rates based on reductions in creatinine were significantly higher in patients treated with norepinephrine (15/26, 57.6%) than with midodrine/octreotide (5/25, 20%). However, there was no difference in survival between the 2 groups. Saif et al. randomized 60 patients with type 1 HRS into treatment groups with either norepinephrine or terlipressin.[9] Reversal of type I HRS occurred in 16 patients (53%) treated with norepinephrine and 17 patients (57%) treated with terlipressin. Facciorusso et al. compared the efficacy of several pharmacological strategies for management of type 1 hepatorenal syndrome using a systematic review and network meta-analysis.[10] These investigators concluded that moderate quality evidence supported the use of terlipressin over placebo in the reduction of short-term mortality. Low quality evidence supported the use of norepinephrine, midodrine plus octreotide, or dopamine plus furosemide over placebo to reduce mortality. This meta-analysis supported the use of terlipressin over midodrine plus octreotide (OR 26.25, 95% CI 3.07-224.21) to reverse the hepatorenal syndrome. Wang et al. completed a systematic review and meta-analysis of 18 randomized control trials which included 1011 patients with hepatorenal syndrome.[11] Terlipressin had a similar efficacy to norepinephrine but had more adverse events.

Wong et al. randomized 300 patients with type 1 HRS to treatment with terlipressin plus albumin or placebo in a special protocol assessment agreement with the Food and Drug Administration as a phase 3 registration trial.[12] The primary endpoint was reversal hepatorenal syndrome which required two consecutive serum creatinine levels less than 1.5 mg/dL and survival without renal replacement therapy for at least 10 days. The baseline serum creatinine levels in these patients were 3.5± 1.0 mg/dL. Thirty-two percent of the patients in the terlipressin group had reversal of their hepatorenal syndrome, and 17% of patients in the placebo group had reversal. By day 90, 51% of the patients in the terlipressin group had died, and 45% of the patients in the placebo group had died. Some patients in both groups received liver transplants. More adverse events, including respiratory failure, occurred in terlipressin group. The interpretation of this study is difficult. A significant percentage of patients in both groups died within 90 days. The study was undertaken in centers which could perform liver transplantation. Consequently, it is likely that centers with less expertise in chronic liver disease would have worse results. Belcher and coauthors analyzed this trial carefully and suggested that outcomes would improve if terlipressin was used earlier in HRS, if it were avoided in patients who are unlikely to benefit, and if there was significant attention to the patient's intravascular volume including avoiding excess albumin administration.[13] These decisions are relevant to the use of any vasoconstrictor, including midodrine/octreotide and norepinephrine, but are not necessarily easy when

managing a critically ill patient. Velez and associates analyzed the association between changes in the mean arterial pressure with vasoconstrictor therapy and reductions in creatinine levels.[14] They found that patients treated with either midodrine/octreotide or norepinephrine with an increase in mean arterial pressure > 15 mmHg had a significant decrease in serum creatinine. Consequently, physicians using these medications should try to adjust the doses of medication to have a definite increase in mean arterial pressure.

Paracentesis

Patients with cirrhosis and HRS−AKI often have large volumes of ascitic fluid and consequently have intra-abdominal hypertension. This increased pressure can have direct effects on renal vein pressure and on renal parenchyma, and logically paracentesis can reduce this pressure and potentially improve renal function. Mohmand and Goldfarb reviewed changes in renal function associated with the abdominal compartment syndrome.[15] They noted that these patients had a decrease in mean arterial pressure, an increase in intra-abdominal pressure, a decrease in abdominal perfusion pressure, an increase in CVP, an increase in renal venous pressure, an increase in renal parenchymal pressure, and an increase in renal vascular resistance. All these changes contribute to reductions in GFR. Umgelter et al. measured changes in hemodynamic parameters using transpulmonary thermal dilution catheter and in the renal resistance index using ultrasound of the renal intralobular arteries following paracentesis.[16,17] They noted a significant drop in intra-abdominal pressure, systemic vascular resistance, and the renal resistance index. Creatinine clearance increased and urine output increased; the timeframe for these studies was 6 hours. The mean the serum creatinine in these patients was 2.9 mg/dL.

Savino et al. measured the intra-abdominal pressure, cardiovascular hemodynamic parameters, and renal function in 25 patients with advanced cirrhosis admitted to the ICU with variceal bleeding, tense ascites, and peripheral edema.[18] In the patients with an elevated intra-abdominal pressure in this cohort, paracentesis decreased it from 33.5 to 19.1 cm of water. This was associated with a decrease in total peripheral resistance and an increase in cardiac index, stroke index, left ventricular stroke work, and right ventricular stroke work. There was a decrease in BUN and creatinine and an increase in creatinine clearance, urine volume, and osmolar clearance. The baseline serum creatinine in these patients was 1.41±0.44 mg/dL. The baseline urine sodium concentration was 51.2±33.4 mEq/L. Creatinine clearance was 46.2±18.2 cc/min. These patients were critically ill with variceal bleeding but did not have extremely abnormal renal function. This study does give a clear indication of the expected changes in hemodynamic parameters following acute reductions in intra-abdominal pressure.

Chang et al. used a murine model to study the effects of increased intra-abdominal pressure on renal function.[19] Chronic liver disease was introduced in to these mice using carbon tetrachloride. After 12 weeks the mice had injections of albumin in the peritoneal cavity to increase intra-abdominal pressure to levels of 0, 5, 10, and 20 cm of water. Blood chemistries and renal histopathology were examined 24 hours later. BUN and serum creatinine increased in both the control group and the liver injury group with increasing levels of intra-abdominal pressure. Renal tissue in animals with an intraperitoneal pressure 20 cm of water revealed edema of the renal tubular epithelial cells, constriction of the renal tubular lumen, formed casts, and hyperemia in the interstitium. The histologic changes were more pronounced in animals with liver disease and at higher intraperitoneal pressures. These results developed within 24 hours of an increase in pressure.

The standard of care in these patients often involves large-volume paracenteses both as inpatients and as outpatients. Van Thiel et al. reported information on 40 patients with cirrhosis and chronic ascites using periodic peritoneal drainage over 72 hours.[20] This procedure involved the removal of 3 L of fluid every 6 hours; patient also received 25 g of albumin every 6 hours. The initial creatinine was 0.8 mg/dL; the final creatinine was 0.8 mg/dL. There were no episodes of peritoneal hemorrhage or infection associated with this procedure. Martin and co-authors also used an indwelling peritoneal catheter in 36 patients undergoing continuous large-volume paracentesis (up to 3 L every 8 hours).[21] These patients received albumin replacement. An average of 16.5 L of ascitic fluid was removed over 72 hours. The initial serum creatinine was 1.37 mg/dL and a final serum creatinine was 1.21 mg/dL. There were no episodes of spontaneous bacterial peritonitis or major bleeding associated with this process. In these two studies, the patients had large volumes of ascitic fluid but did not have hepatorenal syndrome.

Large-volume paracenteses has the potential to reduce intravascular volume and therefore cardiac output and blood pressure. This could contribute to acute changes in renal function. Seethapathy et al. analyzed the outcome of 258 paracenteses done on 102 patients admitted to an academic liver transplant medical center.[22] Most of these paracentesis (67%) removed less than 5 L of ascitic fluid. The mean baseline creatinine is 1.25 mg/dL ±0.69 mg/dL. Acute kidney injury defined by a creatinine increase ≥0.3 mg/dL or ≥50% within 48 hours developed in 14 of the 258 paracenteses. A chart review indicated that only 4 of the 258 paracenteses (1.6%) were associated with new onset AKI without an alternative cause. All of the procedures in these cases had less than 5 L fluid removed. Hypotension defined by either a drop in systolic blood pressure or a drop in diastolic blood pressure occurred in 61 of the 258 paracenteses. This was more common in patients who had greater than 5 L removed but was not associated with

AKI or a change in serum creatinine. These investigators concluded that therapeutic paracentesis had a low risk for the development of AKI. However, the ascitic fluid usually reaccumulates in these patients and requires repeat paracentesis.

An alternative approach to the management of patients with refractory ascites might involve continuous drainage through peritoneal-venous shunts or through a TIPS. Cade et al. measured the effects of vascular volume expansion with fresh frozen plasma, short-term reductions in intraperitoneal pressure with paracentesis, and placement of a LeVeen shunt on renal function in patients with severe hepatic cirrhosis and hepatorenal syndrome.[23] They noted that venous pressures decreased following the removal of ascitic fluid and GFR and urine flow increased. However, as ascitic fluid reaccumulated reducing intravascular volume, GFR and urine output decreased. The placement of LeVeen shunt resulted in a significant improvement in GFR and renal plasma flow. This study suggests that paracentesis has short-term beneficial and then adverse effects but continuous drainage of peritoneal fluid has long-term beneficial effects.

Allegretti et al. compared the effects on renal function of a transjugular intrahepatic portosystemic shunt versus large-volume paracentesis in patients with cirrhosis and refractory ascites.[24] This study included 276 matched patients. There was a significant increase in the estimated GFR following TIPS placement in patients with a baseline estimated GFR less than 60 mL/min per 1.73 m^2. There was no difference in estimated GFR in patients with an estimated GFR greater than 60 at the baseline in patients undergoing a TIPS procedure versus recurrent large-volume paracentesis. The outcomes in a TIPS cohort included death in 30% and liver transplantation in 36%. The estimated GFR increased in 59%. The outcomes in the serial large-volume paracentesis cohort included death in 32% and liver transplantation in 14%. The estimated GFR increased in 31% of these patients. The entire cohort had an average of 5.5 $\pm$ 3.9 paracentesis over a 90-day during the baseline period. The mean creatinine was 1.7$\pm$1.3 mg/dL. Information on ascitic volume, peripheral edema, and intra-abdominal pressures were not available in this study.

Dialysis and liver transplantation

No guidelines have been established on the role of renal replacement therapy (RRT) in patients with HRS-AKI who do not have other indications for dialysis, such as uremic complications. A consensus developed by the Acute Dialysis Quality Initiative (ADQI) group suggested that survival in patients with HRS was very poor and thus RRT should be avoided; however, RRT should be considered in patients eligible for liver transplantation.[25] Velez has critically reviewed the decision to use dialysis in patients with HRS.[26] He suggested that patients listed for liver transplantation should receive dialysis if needed. Patients undergoing evaluation for liver transplantation but without any clear decision should be considered with dialysis. The decision becomes much more difficult in patients who are ineligible for liver transplantation.

Studies have shown that patients with advanced cirrhosis and type 1 HRS who received a liver transplantation as a definitive treatment had a renal recovery rate of 76% and a 1-year survival rate of 97%. The best predictor of HRS non-reversal after liver transplantation is prolonged dialysis before proceeding to transplantation.[27] In these cases, the possible evolution of structural renal damage may hinder ultimate renal recovery and patient survival after liver transplant. Therefore, patient with type 1 HRS who do not respond to vasoconstrictor therapy should be offered liver transplantation immediately.[28]

CASE REVIEW AND ANALYSIS

The unexpected outcome in our case suggests an alternative approach to HRS-AKI using continuous peritoneal drainage in the hospitalized patients. The precedence for this treatment is based on intermittent paracentesis, which involves removal of ascitic fluid from the abdomen and has definite effects on intra-abdominal pressure, vascular resistance, and GFR. Martin et al. noted a statistically significant improvement in creatine (1.37 mg/dL to 1.21 mg/dL) in patients undergoing continuous peritoneal drainage with albumin infusion.[21] Other studies have assessed continuous (i.e., at least 72h) drainage of ascitic found to be effective with favorable adverse effect profile.[29,30] Intermittent drainage of ascites via long-term indwelling catheters have also shown to be relatively safe.[31,32] Furthermore, peritoneal dialysis is an established technique involving long-term placement of a drain in the abdomen. Nevertheless, risks of continuous peritoneal drainage, including infection, hematoma, and adhesions, must always be considered when attempting this procedure. Finally, ultrasound analysis of the inferior vena cava can help classify patients with HRS–AKI. Velez et al. studied 53 patients with a mean creatinine of 3.2 $\pm$ 1.5 mg/dL and measured the inferior vena cava diameter and its collapsibility index.[33] Based on these measurements, 15 patients were classified as fluid depleted, 19 patients were classified as fluid repleted, 8 patients were classified as having intra-abdominal hypertension, and 11 patients were classified as fluid expanded. Using these definitions, some patients received intravenous albumin, some received vasoconstrictors, some underwent large-volume paracentesis, and some received a loop diuretic. Overall 19 patients improved with this management with a reduction in serum creatinine greater than equal to 20%, and 14 patients remained stable with no change in serum creatinine (i.e.< a 20% decrease in creatinine).

CONCLUSIONS

To our knowledge, this is the first case report of continuous drainage in a patient with acute HRS. Continuous

drainage after paracentesis should reduce intra-peritoneal pressure and possibly improve renal function, as occurred in our patient. This approach would require the introduction of a paracentesis catheter into the abdomen and connection to sterile drainage bags for periods of 1 to 3 days. It should be considered in hospitalized patients who present with chronic liver disease complicated by significant volumes of ascites and deteriorating renal function. Ideally, an organized clinical study should be undertaken with patients with HRS−AKI using point-of-care ultrasound and intra-abdominal pressure measurements with bladder catheter to classify patients. This information might suggest more specific, directed therapy for patients based on their particular clinical, hemodynamic status.

FINANCIAL SUPPORT

None

DECLARATION OF COMPETING INTEREST

None

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Submitted January 8, 2022; accepted June 28, 2022.

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