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Continuous Furosemide Infusion in the Management of Ascites

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Abstract

Background—The current therapy for patients hospitalized with ascites requires titration of oral diuretics and often needs several days. A faster method for predicting the response to a given dose of diuretic may allow this process to be completed more rapidly.

Aim—Describe the short-term safety and efficacy of a diuretic infusion to predict net sodium excretion in patients with cirrhosis, ascites and edema using a fractional excretion of sodium (FENa) of $\geq 1\%$ as the target.

Methods—We conducted a retrospective case series of patients admitted for management of ascites who received intravenous furosemide by continuous infusion in ascites management. Patients were stratified depending on whether they had edema, received an intravenous bolus of furosemide or a large-volume paracentesis. The primary outcome was the proportion of patients achieving a FENa of $\geq 1\%$ during the infusion. Secondary outcomes included development of electrolyte abnormalities or acute kidney injury (AKI) during or immediately following the infusion and natriuresis on titrated oral furosemide.

Results—47 patients meeting criteria were identified from 721 patients seen in consultation. 10 of the patients had edema and received neither bolus intravenous diuretic therapy nor therapeutic paracentesis; all ten achieved a FENa $\geq 1\%$. One patient had transient hypokalemia. Of 37 patients who either had no edema or received additional treatment options, all but six patients achieved a FENa of $\geq 1\%$. Transient complications in the 31/37 patients with natriuresis included hyponatremia ($n = 1$), hypokalemia ($n = 5$) and AKI ($n = 3$). 24 hour urine sodium averaged > 4 g/d on the titrated oral furosemide regimen in 19 patients completing the collection.

Conclusion—Use of a short continuous furosemide infusion can achieve a FENa $\geq 1\%$ in patients with cirrhotic ascites and may be safe and efficacious for diuresis, meriting further study.

Introduction

Ascites is the most common complication from cirrhosis of the liver, developing in ~ 50 % of patients observed over a 10 year period [1]. The occurrence of ascites usually portends a poor prognosis and heralds the onset of other complications of portal hypertension, such as variceal hemorrhage and hepatic encephalopathy [2]. Thus, uncontrolled ascites is often the reason for hospital admission in patients with decompensated cirrhosis [3], [4].

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Standard therapy for ascites includes a combination of dietary sodium restriction, oral spironolactone and furosemide, with large volume paracentesis (LVP) if needed [5]. It can take several days to titrate the dose of oral diuretics needed to produce the desired effect, if measured by sequential daily weight loss.

Ideally, there would be a rapid method to determine the dose of diuretics needed to produce effective diuresis on hospital discharge. This requires: a) a method to predict effective diuresis, and b) an efficient approach to titrating diuretic dose. Use of a continuous intravenous infusion of furosemide, titrated to achieve a fractional excretion of sodium (FENa) $\geq 1\%$, may meet these requirements. Fractional excretion of sodium can be calculated using urine and plasma sodium and creatinine ($\text{FENa} = 100 \times [(\text{urine sodium} \times \text{plasma creatinine}) / (\text{plasma sodium} \times \text{urine creatinine})]\%$, see Supplemental Table 1), which are easily measured and widely available. The daily excretion of sodium can then be calculated, by substituting in the equation: $24 \text{ hour urine sodium} = \text{plasma sodium} \times \text{glomerular filtration for 24 hours} \times \text{FENa}$ (see Supplemental Table 1). If the FENa averages 1% throughout the day (24 hours), it predicts effective diuresis because the plasma sodium multiplied by glomerular filtration times 1% equals about 4 grams of sodium excreted per day (see Supplemental Table 1). Thus calculating the FENa may be a method to predict effective diuresis. If insufficient sodium excretion is predicted, then the diuretic dose needs to be increased until efficacy is expected. Continuous intravenous infusion of a loop diuretic such as furosemide, which is generally widely available, may be an efficient approach to titrating diuretic dose to achieve a FENa $\geq 1\%$ because the onset of diuretic action is rapid and a stable response is maintained throughout the infusion [6]. Moreover, once the effective IV dose is determined, oral diuretic requirements can be estimated [6], [7].

Based on these principles, in 2008, one of the authors (JC) commenced a new clinical practice of calculating the FENa during diuretic therapy. The recommended approach was that of using an infusion of furosemide, after measuring baseline FENa, and increasing the rate of furosemide delivery until the FENa was $\geq 1\%$. While continuous infusions of furosemide are both safe and effective for acute management of volume overload in heart failure [6], [7], no previous studies or case reports have described the use of a continuous infusion of intravenous furosemide in patients with portal hypertensive ascites. Herein we report a retrospective case series of cirrhotic patients admitted with ascites who received an infusion of furosemide. Our findings suggest that this method may be both effective and safe, and merits further study.

Methods

We conducted a retrospective study of patients with cirrhosis and ascites receiving a continuous infusion of furosemide. Patients were identified through review of records from all patients seen by the inpatient hepatology consultation service at our hospital from November 2008 to January 2010. The names and medical record numbers of patients were obtained from shared lists in the electronic medical record prospectively collected by the Division of Digestive and Liver Diseases. A comprehensive electronic medical record (Epic®), maintained since October 2005, was the primary data source for this study.

Patient inclusion criteria

Data were collected for patients with cirrhosis and portal hypertensive ascites who received a continuous intravenous infusion of furosemide for at least two hours during their hospital stay and had urine chemistries measured at least two hours after commencement of the infusion (Supplemental Figure 1). Cirrhosis was documented by standard clinical, histologic and/or radiologic criteria. This included nodularity of the liver surface seen on imaging, elevated total bilirubin, low serum albumin and/or prolonged prothrombin time. Ascites was

demonstrated by diagnostic paracentesis or imaging. Portal hypertensive ascites was characterized by ascites fluid with a serum albumin-ascites gradient (SAAG) of ≥ 1.1 . Edema was defined by documentation of pitting peripheral edema in the medical record.

Patient exclusion and stratification criteria

Patients were excluded if they had chronic kidney disease, stage 5 or a prior transhepatic portal-systemic shunt for ascites. Patients were stratified depending on presence or absence of edema and whether they received an intravenous bolus dose of furosemide or an LVP during their hospital stay in addition to continuous infusion intravenous furosemide. Specifically, group 1 is defined as those patients with cirrhosis, ascites, and edema who received continuous infusion furosemide without prior inpatient treatment for fluid overload; while group 2 is defined as patients with cirrhosis and ascites, both with and without peripheral edema, who received bolus intravenous furosemide or LVP or both for management before the continuous intravenous infusion of furosemide was commenced. The distinction was made because, in group 1, the presence of edema and the absence of bolus furosemide and of therapeutic paracentesis permits assessment of the effects of the infusion without these co-interventions. In contrast, the data collected for group 2 patients reflects the more commonly encountered hospitalized patients.

Oral diuretic regimen

All patients were prescribed spironolactone, 100 – 200 mg/d but no other oral diuretic. Following titration to achieve a FENa $\geq 1\%$, oral furosemide was prescribed. The total daily oral dose was calculated by multiplying the hourly infusion rate by 24, then divided by 3 and scheduled for administration at 6 a.m., 12 noon and 6 p.m. 24 hour urine for sodium, potassium and creatinine on the oral regimen was requested (Supplemental Figure 2).

Measurements

Demographic information, etiology of cirrhosis, presence or absence of peripheral edema and baseline laboratory data at presentation were recorded. The furosemide infusion doses, results of urine chemistries obtained before the initiation of the infusion and at varying times thereafter and results of contemporaneous routine blood tests were analyzed. Model of End-stage Liver Disease (MELD) scores were calculated from laboratory values at the time of admission. FENa was calculated using the following equation:

$$FENa = 100 \times \frac{\text{urinary sodium} \times \text{plasma creatinine}}{\text{plasma sodium} \times \text{urinary creatinine}} \%$$

Outcomes and analyses

Our primary outcome was the proportion of patients achieving a FENa of $\geq 1\%$ during the furosemide infusion. This primary outcome was chosen because, as mentioned previously, all patients with normal renal function who are maintained on a four-gram sodium diet should have a net excretion of sodium (natriuresis) and water at a FENa of $\geq 1\%$. Secondary outcomes included proportion of patients with each of the following complications during or within 18 hours of discontinuation of the infusion: 1) new hypokalemia (serum K < 3.0 mmol/L), 2) new hyponatremia (serum Na < 130 mmol/L), 3) significant hypernatremia (Δ serum Na ≥ 10 mmol/L in a 24-hour period), and 4) acute kidney injury (AKI, increase in serum creatinine ≥ 0.3 mg/dL = ≥ 26.5 μ mol/L or $\geq 50\%$ from pre-infusion). The other secondary outcome was 24 hour urine sodium excretion on the titrated oral dose of furosemide with continuation of spironolactone. The study was approved by the UT Southwestern and Parkland Health and Hospital System Institutional Review Boards.

Results

From November 2008 to January 2010, 721 patients were seen in consultation by the inpatient hepatology service at Parkland Memorial Hospital. Inclusion and exclusion criteria were met by 47 patients. Of these, ten patients with edema received neither bolus furosemide nor LVP for inpatient management before the intravenous infusion of furosemide (Supplemental Figure 1, Group 1). The remaining 37 patients received bolus furosemide intravenously or LVP or both for management or did not have edema (Supplemental Figure 1, Group 2). Demographic characteristics and admission data of the patient population in each group are listed in Table 1. Combining groups 1 and 2, our total population was 66% male with a mean age of 51 years and a mean MELD score of 16. Caucasians made up the largest group of the patients at 40% followed by those of Hispanic and African American ethnicity or race at 32% and 28% respectively. All but three patients had cirrhosis from alcohol consumption, chronic hepatitis C or a combination of the two. All patients were prescribed spironolactone (100 – 200 mg/d). No patients received another oral diuretic during data collection either before or after furosemide infusion.

Achievement of FENa $\geq 1\%$

With the furosemide infusion, all ten patients in group 1 achieved the primary outcome of attaining a FENa of $\geq 1\%$ (Table 2). Six patients achieved the goal FENa at an infusion rate of 5 mg/hr while the other four patients required a rate of 10 mg/hr (Figures 1 and 2). The average time on the infusion was 27 hours (range 5 – 102 hours) and patients lost an average of 3.5 kg (median 3.4 kg, range = gained 0.8 to lost 9.5 kg, $n = 9$) during the infusion. The total furosemide dose received during the entire infusion period averaged 210 mg (median 99 mg, range 26 – 884 mg). After initiation of the titrated oral furosemide regimen, a 24 hour urine collection was completed successfully in 6 patients, average sodium excretion was 183 mmol/d = 4.2 g/d (median 183, range 111 – 253 mmol/d).

In group 2, most patients (31/37, 84%) also achieved a FENa of $\geq 1\%$; 6 patients did not (Table 2, Figure 1). The successful furosemide dose ranged from 2.5 mg/hr to 15 mg/hr (Figures 1 and 2). Of the 6 patients without edema, all but one (83%) achieved a FENa of $\geq 1\%$ (Supplemental Table 2). This subset included both patients with diuresis on the lowest furosemide infusion dose, 2.5 mg/hr.

Three of the 6 patients who failed to achieve a FENa $> 1\%$, had either FENa results just below our threshold ($n = 2$) or evidence of diuresis during the infusion ($n = 2$). One patient (FENa 0.8%) lost 1.9 kg during the 23.8 hr infusion. The second patient (FENa 0.9%) had a 7 hour infusion and was discharged the next day. The third patient (FENa 0.3%) had net natriuresis during the 38.1 hr infusion documented by weight loss (3.8 kg) and a 24 hour urine sodium (187 mmol/d excreted = 4.3 g/d) but without a corresponding creatinine value for FENa calculation. A second 24 hour urine sodium on oral furosemide confirmed natriuresis (183 mmol/d = 4.2 g/d, FENa = 1.2%). The spot urine during the infusion (FENa = 0.3%) was apparently collected 12 hours after the infusion commenced; the result does not match the other data and is unexplained.

The mean infusion length was 36 hours (median 22 hours, range 3 – 229 hours) during which time patients lost an average of 4.7 kg (median 3.5 kg, range = gained 1.0 to lost 23.1 kg, $n = 29$). The total furosemide dose received during the entire infusion period averaged 296 mg (median 131 mg, range 17 – 3,284 mg). After initiation of the titrated oral furosemide regimen, a 24 hour urine collection was successfully completed in 13 patients, average sodium excretion 288 mmol/L = 6.6 g/d (median 211, range 64 – 784 mmol/d). Eight patients excreted at least 4 g sodium/d.

In this group, 22 patients also received bolus furosemide, average dose 149 mg (median 80 mg, range 20 – 1,380 mg). Therapeutic paracentesis was undertaken in 22 patients, with an average of 5 liters being removed (median 5, range 1 – 14 liters).

Safety of continuous infusion furosemide

Electrolytes and renal parameters were monitored before, during and after the infusion. As shown in Table 3, the mean and median sodium levels were below the normal range at our institution whereas the mean and median potassium levels were in the lower normal range. Mean and median creatinine levels increased minimally during and after the infusion (between admission and post infusion, mean increase = 0.07 mg/dL = 6.2 μ mol/L and median increase = 0.03 mg/dL = 2.7 μ mol/L).

Electrolyte abnormalities during or immediately following the furosemide infusion occurred in 8 patients, 1 patient in group 1 (hypokalemia) and 7 patients in group 2, including 2 of the 6 without a natriuretic response. One patient developed hyponatremia (129 mmol/L), while hypokalemia (2.6 – 2.8 mmol/L) was recorded in a total of 6 patients and hyperkalemia in 1 patient (Supplemental Table 3). Both the hyponatremia and hypokalemia resolved rapidly. The patient with hyperkalemia also had AKI (see below).

In 6 patients, all in group 2, an increased creatinine was observed (Supplemental Table 4). Whereas electrolyte abnormalities resolved rapidly in all but one patient, 2 of the 6 patients with AKI expired in the hospital with complications of cirrhosis and 1 patient was discharged with hospice for metastatic hepatocellular carcinoma. Three patients with an increased creatinine failed to achieve a FENa > 1% and had findings consistent with pre-existing AKI (Supplemental Table 4). Thus, the creatinine level at the beginning of the infusion was higher than on admission, continued to rise with each time period and no weight loss was documented. Overall, 15 patients are known to have died in follow-up, including both patients with AKI and underlying chronic kidney disease.

Discussion

We found that a continuous infusion of furosemide achieved a FENa of $\geq 1\%$ in ten patients with ascites, cirrhosis and edema who did not receive an LVP or intravenous bolus dose of furosemide. In a more heterogeneous group, receiving other therapies for fluid loss or lacking edema for rapid resorption, a FENa of $\geq 1\%$ was achieved in most patients (31/37, 84%). Diuresis accompanied natriuresis and was documented by both weight loss (median 3.5 Kg) and 24 hour urinary sodium excretion (median ~ 4 g/d). Therefore, our data suggest that continuous infusion of furosemide with dose titrated to achieve a FENa > 1% is feasible for hospitalized patients with cirrhosis and ascites, and may be effective for achieving diuresis in patients with cirrhotic ascites.

The safety of the infusion technique was also evaluated. Electrolyte abnormalities were transient. However, AKI was documented in 6 patients (16%). While we believe that there was no definite direct causal relationship between the infusion and the rising creatinine, the findings reinforce the potential complications encountered in this group of patients with advanced cirrhosis, portal hypertension and ascites.

Current treatment guidelines for patients with portal hypertensive ascites are based on studies completed more than 20 years ago, and generally suggest use of oral diuretics for hospitalized patients with ascites, titrated over several days [8], [9], [10]. Possible reasons why titration requires several days include erroneous assumptions of renal function and dietary sodium intake. The time lost in titration likely leads to increased hospital costs, and, as important, a longer time to relief of patient symptoms. Our approach of using continuous

infusion furosemide to achieve a FENa of $\geq 1\%$ may be much more time efficient for achieving diuresis and predicting oral diuretic requirements because the response is titrated for the individual. We propose study of a new algorithm for ascites management that includes measuring the FENa and adjusting diuretic regimens based on urinary sodium excretion, measured renal function and anticipated sodium intake (Supplemental Figure 2).

Of note, there are a number of important limitations to our report. Our study design did not allow comparison of the patients in our series to a control group of patients treated with the current standard of care. This limited our ability to examine other important outcomes such as relative benefits of the continuous infusion approach versus standard of care diuretic management on weight loss, length of hospital stay, and safety. In addition, since the ultimate decisions on management were made by the primary inpatient service, the hepatology consultation service had minimal control of the duration of the furosemide infusions. Despite these restrictions, this case series shows the potential for using short-term continuous infusions of diuretics in patients with cirrhosis and ascites, titrating to a FENa $> 1\%$.

We conclude that short-term infusions of furosemide can be used to achieve a FENa of $\geq 1\%$ in patients with cirrhosis, edema and ascites. Future studies, including randomized controlled trials, should compare effectiveness of continuous furosemide infusion with standard diuretic titration and management of fluid overload examining weight loss, length of hospital stay, and safety as primary outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1. Achievement of peak FENa by each individual is determined by infusion rate
 Maximum FENa obtained for each subject is shown with the associated furosemide infusion dose on the X-axis. Group 1 subjects had edema and received neither bolus furosemide nor therapeutic paracentesis as management for ascites before commencement of the furosemide infusion. Group 2 subjects either had no edema or received other management for ascites before commencement of the furosemide infusion (bolus furosemide or therapeutic paracentesis or both). White symbols = group 1; black symbols = group 2; gray symbols = Group 2 subjects who failed to achieve a FENa of at least 1%. Most subjects achieved a FENa $\geq 1\%$ with 5 mg/hr infusion rate.

Figure 2. Response of FENa to dose escalation with FENa before and after increase in furosemide infusion rate

Group 1 subjects had edema and received neither bolus furosemide nor therapeutic paracentesis as management for ascites before commencement of the furosemide infusion. Group 2 subjects either had no edema or received other management for ascites before commencement of the furosemide infusion (bolus furosemide or therapeutic paracentesis or both). Symbols for group 2 subjects include black triangles, black and gray reverse triangles and white, black and gray circles to permit differentiation of individuals. Most subjects requiring dose escalation achieved a FENa $\geq 1\%$ with 10 mg/hr infusion rate or less.

Table 1

Patient Demographics and Admission Data

Characteristic	Group 1 n = 10	Group 2 n = 37
<i>Mean (% or median, range)</i>		
Age (years)	52 (55, 36 – 59)	50 (50, 35 – 67)
Gender (male)	7 (70%)	24 (51%)
<i>Ethnicity or race:</i>		
African-American	3 (30%)	10 (27%)
Hispanic	3 (30%)	12 (32%)
White	4 (40%)	15 (41%)
<i>Etiology of cirrhosis:</i>		
Alcohol	5 (50%)	13 (35%)
Alcohol and HCV	3 (30%)	17 (46%)
HCV	1 (10%)	5 (14%)
Cryptogenic	1 (10%)	2 (5%)
Peripheral edema	10 (100%)	31 (84%)
Plasma Na (mmol/L)	133 (134, 124 – 141)	132 (132, 116 – 139)
Plasma K (mmol/L)	4.0 (3.9, 3.7 – 4.8)	3.8 (3.9, 1.8 – 5.4)
Plasma creatinine (mg/dL)	0.9 (0.7, 0.3 – 1.9)	1.1 (0.9, 0.5 – 3.8)
Plasma creatinine (μ mol/L)	80 (62, 27 – 168)	97 (80, 44 – 336)
MELD *	14 (13, 6 – 24)	17 (16, 7 – 32)
Weight (kg) **	85 (77, 50 – 166)	89 (86, 45 – 161)

* Missing one patient in each group (1 patient on warfarin, 1 without INR on admission)

** Missing 3 patients without weights on admission, 1 without other Rx, 2 with other Rx

Table 2

Natriuresis with Furosemide Infusion

Successful or final infusion rate	Number with effective natriuresis (%)	Mean FENa of responders	Median FENa of responders (range)
Group 1, n = 10			
5 mg/hr (n = 6)	6 (100%)	4.5%	3.4% (2.0% – 8.4%)
10 mg/hr (n = 4)	4 (100%)	7.9%	7.1% (4.9% – 12.4%)
Group 2, n = 37			
2.5 mg/hr (n = 2)	2 (100%)	1.5%	(1.4%, 1.7%)
5 mg/hr (n = 25)	19* (76%)	3.2%	2.6% (1.4% – 8.4%)
10 mg/hr (n = 7)	7 (100%)	2.9%	2.6% (1.0% – 5.6%)
15 mg/hr (n = 3)	3 (100%)	4.0%	2.4% (2.0% – 7.6%)

* Non-responders (n = 6), average FENa = 0.4%, median = 0.3%, range = 0.2% – 0.9%. Only 1 patient received a higher infusion rate (10 mg/hr), no FENa results are available for that rate.

Table 3

Plasma Chemistries Associated with Furosemide Infusion

Component	Admission	Start infusion	End infusion	Post infusion
Mean $\pm$ SD, n (median, range)				
Sodium, mmol/L	131 $\pm$ 6, 47 (133, 106 – 141)	133 $\pm$ 6, 47 (134, 109 – 145)	133 $\pm$ 4, 44 (134, 120 – 145)	133 $\pm$ 4, 42 (133, 120 – 144)
Potassium, mmol/L	3.9 $\pm$ 0.7, 47 (3.9, 1.8 – 5.4)	3.9 $\pm$ 0.6, 47 (3.9, 2.5 – 5.2)	3.7 $\pm$ 0.5, 43 (3.6, 2.5 – 5.1)	3.9 $\pm$ 0.7, 42 (3.8, 2.6 – 6.3)
Creatinine, mg/dL	1.06 $\pm$ 0.61, 47 (0.85, 0.32 – 3.78)	1.06 $\pm$ 0.59, 47 (0.83, 0.31 – 3.78)	1.08 $\pm$ 0.47, 43 (0.93, 0.45 – 2.23)	1.13 $\pm$ 0.58, 42 (0.88, 0.46 – 2.40)
<i>Creatinine, μmol/L</i>	<i>94 $\pm$ 54, 47 (75, 28 – 334)</i>	<i>94 $\pm$ 52, 47 (73, 27 – 334)</i>	<i>95 $\pm$ 42, 43 (82, 40 – 197)</i>	<i>100 $\pm$ 51, 42 (78, 41 – 212)</i>