The impact of large-volume paracentesis on renal haemodynamics in cirrhotic patients with ascites

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Background and aim
Ascites in liver cirrhosis is associated with a poor prognosis and impairment of the quality of life and may be complicated by hepatorenal syndrome. Renal functions and haemodynamic changes after large-volume paracentesis (LVP) in cirrhotic patients with tense ascites were evaluated.

Patients and methods
A total of 50 cirrhotic patients with tense ascites were divided into two groups: group I 25 patients without renal impairment and group II 25 patients with renal impairment (type II hepatorenal syndrome).

Results
In groups I and II, the serum creatinine decreased significantly 24 h after LVP ($P < 0.05$ and 0.01, respectively). The glomerular filtration rate and the urine output increased significantly 24 h after LVP ($P < 0.05$, $P < 0.01$ and $P < 0.01$, $P < 0.05$, respectively, in groups I and II). The renal artery resistive index (RI) was significantly higher in group II compared with group I ($P < 0.01$). LVP caused a significant increase in the cardiac output, the stroke volume and the cardiac index ($P < 0.01$) and a significant decrease in the RI in both groups ($P < 0.01$). There was significant correlation between serum and ascetic fluid electrolyte levels in all patients.

Conclusion
LVP causes a significant reduction of heart rate and mean arterial pressure, serum creatinine, blood urea nitrogen and RI with a significant glomerular filtration rate increase, but had no effect on the plasma renin activity.

Keywords:
ascites, cirrhosis, hepatorenal syndrome, large-volume paracentesis, renal resistive index

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Introduction

Hepatorenal syndrome (HRS) is a clinical condition that occurs in patients with chronic liver disease, advanced hepatic failure and portal hypertension due to impaired renal function and marked abnormalities in the arterial circulation and the activity of endogenous vasoactive systems. There is marked renal vasoconstriction that results in a low glomerular filtration rate (GFR), whereas in the extrarenal circulation, there is a predominance of arterial vasodilatation, which results in the reduction of the total systemic vascular resistance and arterial hypotension [1].

HRS is characterized by a combination of liver failure, circulatory abnormalities and renal failure (RF) [2]. Type I HRS is characterized by rapidly progressive RF with a doubling of serum creatinine to a level greater than 2.5 mg/dl [3], with a median survival of 2 weeks, and type II HRS is characterized by a slowly progressive increase in the serum creatinine level to greater than 1.5 mg/dl and urine sodium less than 10 mEq/dl with a median survival of 4–6 months [4].

About 18% of the cirrhotic patients with ascites develop HRS after 1 year and 39% after 5 years, and up to 10% of the hospitalized patients with liver failure can also develop HRS [5].

Renal Doppler indices have been used to analyse renal haemodynamics for a long time, and the renal resistive index (RI) correlates with the renal function in a variety of kidney disorders [6] and increases along the clinical stages of cirrhotic renal dysfunction [7]. High values of RI predict the occurrence of HRS and have also been shown to correlate with the intra-abdominal pressure (IAP) [8].

Intra-abdominal hypertension affects kidney function and is an important factor contributing to acute RF in critical care patients [9]. Intra-abdominal hypertension

may reduce the renal perfusion pressure and contribute to RF in cirrhotic patients with ascites [10].

Abnormalities of circulatory function in patients with HRS include a high cardiac output (CO), a low arterial blood pressure and decreased total systemic vascular resistance. Although it was traditionally considered that the increased vascular resistance in HRS occurred only in the renal circulation, vascular resistance is also increased in upper and lower limbs as well as in the cerebral circulation [11].

Large-volume paracentesis (LVP) is an optimum choice for the management of tense ascites. The main findings of studies comparing LVP with diuretics in patients with tense ascites are summarized as follows:

(a) LVP combined with an infusion of albumin is more effective than diuretics and shortens the duration of hospital stay significantly.
(b) LVP plus albumin is safer than diuretics as the frequency of hyponatraemia, renal impairment and hepatic encephalopathy is lower in patients treated with LVP in the majority of the studies.
(c) LVP is a safe procedure, and the risk of local complications such as haemorrhage or bowel perforation is extremely low [12].

Patients and methods

This prospective open-label un-controlled study was conducted at the National Liver Institute, Menoufiya University. A total of 50 cirrhotic patients with tense ascites were enrolled in this study after obtaining their informed consent. Patients were divided according to their renal function into two groups:

(1) Group I: 25 patients with tense ascites without renal impairment.
(2) Group II: 25 patients with tense ascites and renal impairment (type II HRS).

Exclusion criteria

(1) Sepsis as spontaneous bacterial peritonitis or any other infection.
(2) Hepatocellular carcinoma, cardiac, pulmonary or intrinsic RF.
(3) Acute gastrointestinal bleeding 1 week before the study.
(4) Use of diuretics, β-blockers, plasma expanders and paracentesis within 1 month.
(5) International normalized ratio greater than 1.5 or platelet count less than $100 \times 10^3/mm^3$.

Laboratory investigations

(1) Complete blood count, biochemical liver tests: serum bilirubin (total and direct), serum albumin, prothrombin time and concentration, serum aspartate transaminase, serum alanine transaminase, alkaline phosphatase, gamma glutamyl transferase.
(2) Renal functions [blood urea, serum creatinine and blood urea nitrogen (BUN)], plasma renin activity (PRA) by the radioimmunoassay and the estimated GFR (eGFR) using the Mayo Quadratic formula [13] before and 24 h after LVP.
(3) Serum and ascetic fluid sodium (Na^+), potassium (K^+) and chloride (Cl^-) and the total ascetic fluid white blood cell count for the exclusion of spontaneous bacterial peritonitis.
(4) A high-resolution machine (Philips ATL, HDI 5000 with SonoCT manufacturer, Philips Medical Systems, Nederland B.V., Amsterdam, The Netherlands) with different transducers was used to perform the following examinations:
    (1) Abdominal ultrasound
    Ultrasound-guided LVP was performed on all cases. About 5–8 l, with mean of 6.4 ± 1.04 l, were withdrawn. Human albumin was infused simultaneously at a dose of 1 U (100 ml) for every 3 l of ascites drained.
    (2) Renal Doppler was performed to assess renal haemodynamics, especially the renal artery RI.
    (3) Echocardiography was performed to assess the stroke volume (SV), the CO and the cardiac index (CI).

Statistical procedures

Descriptive statistics were presented as the mean ± SD for normally distributed data and median, range for non-normally distributed data. The Student *t*-test was used for normally distributed quantitative variables to measure the mean and SD. The Mann–Whitney test was used for quantitative variables that were not normally distributed. Pearson's correlation test was used to study the correlation between two normally distributed quantitative variables. The paired *t*-test was used to detect the mean and SD of normally distributed prevalue and postvalue of the same variable of the same group of patients. The Wilcoxon test was used to detect the mean and SD of non-normally distributed prevalue and postvalue of the same variable of the same group of patients. Repeated measures of analysis of variance test were performed to differentiate changes in different follow-up results of normally distributed studied variables, and the Friedman test was performed to differentiate changes in different follow-up results of the different studied variables. *P*-value less than 0.05 was considered significant for all variables.

Results

The age in group I ranged between 38 and 63 years, with a mean ± SD of 52.7 ± 7.3 years: there were 8 (32%) female and 17 (68%) male participants. In group II, the age ranged between 40 and 66 years, with a mean ± SD of 54.9 ± 8.3 years: there were 11 (44%) female and 14 (56%) male participants with no significant difference.

The body weight was 74.9 ± 12.7 vs. 74.1 ± 14.7 kg, the height 168.4 ± 7.9 vs. 167.4 ± 9.04 cm and the body surface area 1.85 ± 0.19 vs. 1.83 ± 0.21 m^2 in groups I and II, respectively, with no significant difference.

Regarding baseline laboratory measurements of all patients, haemoglobin and albumin were significantly lower in group II compared with group I ($P < 0.05$): haemoglobin was 10.3 ± 1.45 against 9.5 ± 1.06 g/dl, whereas albumin was 2.35 ± 0.4 against 2.11 ± 0.43 g/l in groups I and II, respectively. There was no statistically significant difference regarding red blood cells, white blood cells and platelet counts, prothrombin time, prothrombin activity, total and direct bilirubin, aspartate transaminase, alanine transaminase, gamma glutamyl transferase and alkaline phosphatase.

There was a statistically significant difference in the level of serum and ascitic fluid electrolytes (Na^+, K^+ and Cl^-) between both groups. The serum Na^+ was 125.2 ± 7.58 against 126.4 ± 6.9 mEq/l, the ascitic fluid Na^+ was 126.8 ± 6.78 against 128.2 ± 6.1 mEq/l, the serum K^+ was 4.44 ± 0.52 against 4.67 ± 0.86 mEq/l, the ascitic fluid K^+ was 4.31 ± 0.54 against 4.46 ± 0.85 mEq/l, the serum Cl^- was 99.4 ± 4.96 against 101.6 ± 3.62 mEq/l and the ascitic fluid Cl^- was 105.12 ± 4.81 against 106.8 ± 4.38 mEq/l in group I and group II, respectively. Urinary Na^+ was significantly lower in group II (4.79 ± 1.7 mEq/l) compared with group I (6.85 ± 3.72 mEq/l) ($P < 0.05$).

On comparing renal function tests [urea, creatinine, BUN, eGFR, urine output (UO) and PRA] in both groups before and 24 h after LVP, there was a highly significant increase in urea, creatinine, BUN and PRA levels in group II compared with group I ($P < 0.01$): before LVP, urea was 70.4 ± 46.2 against 140.9 ± 37.01 mg/dl, creatinine 0.89 ± 0.3 against 2.2 ± 0.59 mg/dl, BUN 32.9 ± 21.6 against 65.4 ± 17.6 mg/dl and PRA 4.68 ± 3.11 against 11.5 ± 3.25 ng/ml/h in groups I and II, respectively; and 24 h after LVP, urea was 68.4 ± 49.9 against 134.4 ± 42.7 mg/dl, creatinine 0.81 ± 0.32 against 1.92 ± 0.51 mg/dl, BUN 31.9 ± 23.2 against 62.4 ± 20.2 mg/dl and PRA 4.68 ± 3.14 against 11.8 ± 3.7 ng/ml/h in groups I and group II, respectively. Overall, eGFR estimates were less in group II than in group I ($P < 0.01$). Before LVP, eGFR was 104.8 ± 50.6 against 33.12 ± 10.1 ml/min, and 24 h after LVP, it was 120.6 ± 56.7 against 40.4 ± 16.5 ml/min in groups I and II, respectively ($P < 0.01$).

Serum creatinine decreased significantly 24 h after LVP in groups I and II ($P < 0.05$ and 0.01, respectively). However, the eGFR and the UO increased significantly 24 h after LVP ($P < 0.05$ and 0.01, respectively, in group I; $P < 0.01$ and 0.05, respectively, in group II) as shown in Table 1.

The renal RI ratio was significantly higher in group II compared with group I, whether before, 1 h after

Table 1 Renal function changes in all patients (before and 24 h after LVP)

Studied variables	Mean ± SD (Range)		*t*-test	*P*-value
	Group I (*n* = 25)	Group II (*n* = 25)		
Urea (mg/dl)				
Before LVP	70.4 ± 46.2 (13–207)	140.9 ± 37.01 (75–208)	4.78[a]	<0.01**
24 h after LVP	68.4 ± 49.9 (17–215)	134.4 ± 42.7 (67–221)	4.27[a]	<0.01**
Creatinine (mg/dl)				
Before LVP	0.89 ± 0.3 (0.42–1.4)	2.2 ± 0.59 (1.52–3.7)	6.06[a]	<0.01**
24 h after LVP	0.81 ± 0.32 (0.3–1.6)	1.92 ± 0.51 (1–3.12)	5.75[a]	<0.01**
BUN (mg/dl)				
Before LVP	32.9 ± 21.6 (6–97)	65.4 ± 17.6 (35–99)	4.69[a]	<0.01**
24 h after LVP	31.9 ± 23.2 (8–100)	62.4 ± 20.2 (31–103)	4.24[a]	<0.01**
eGFR (ml/min)				
Before LVP	104.8 ± 50.6 (56–168)	33.12 ± 10.1 (17–52)	4.24[a]	<0.01**
24 h after paracentesis	120.6 ± 56.7 (48–169)	40.4 ± 16.5 (21–83)	4.69[a]	<0.01**
Urine output (ml/day)				
Before LVP	808 ± 259.3 (300–1500)	856 ± 183.3 (600–1400)	0.76	<0.05
24 h after LVP	991.2 ± 298.9 (400–1600)	1032 ± 342.1 (600–1600)	0.45	<0.05
PRA (ng/ml/h)				
Before LVP	4.68 ± 3.11 (1.2–14)	11.5 ± 3.25 (6.2–22)	5.29[a]	<0.01**
24 h after LVP	4.68 ± 3.14 (1.2–14)	11.8 ± 3.7 (6–22)	5.34[a]	<0.01**

BUN, blood urea nitrogen; eGFR, estimated glomerular filtration rate; LVP, large-volume paracentesis; PRA, plasma renin activity;
[a]The Mann–Whitney test; **Highly significant.

or 24 h after LVP ($P < 0.01$), although there was a significant decrease in RI in both groups ($P < 0.01$). The right RI was 0.704 ± 0.0357 against 0.814 ± 0.0279 before LVP, 0.681 ± 0.0411 against 0.789 ± 0.0299 1 h after LVP and 0.6812 ± 0.0435 against 0.788 ± 0.0309 24 h after LVP in groups I and II, respectively. The left RI was 0.703 ± 0.0355 against 0.814 ± 0.0272 before LVP, 0.678 ± 0.0431 against 0.788 ± 0.0289 1 h after LVP and 0.679 ± 0.046 against 0.786 ± 0.028 24 h after LVP in groups I and II, respectively.

There was a significant increase in CO, SV and CI ($P < 0.01$) in group I: CO was 7486.8 ± 1265.1, 7647.04 ± 1325.9 and 7552.2 ± 1311.7 ml/min, SV was 91.4 ± 16.3, 98.8 ± 17.9 and 95.2 ± 17.3 ml/beat and CI was 4056.6 ± 570.3, 4171.9 ± 609.3 and 4090.2 ± 576.8 ml/min/m^2. In group II, CO was 7576.5 ± 1012.3, 7886.9 ± 969.4 and 7644.6 ± 999.6 ml/min, SV was 92.04 ± 13.98, 98.4 ± 13.7 and 95.5 ± 14.23 ml/beat and CI was 4139.04 ± 522.2, 4245.4 ± 685.8 and 4175.12 ± 511.5 ml/min/m^2 before, 1 and 24 h after LVP, respectively (Table 2).

The heart rate (HR) (beats/min) showed a statistically significant reduction in both groups before, 1 h after and 24 h after LVP, respectively, showing values of 91.1 ± 9.8, 85.2 ± 10.3 and 88.0 ± 11.9 in group I and values of 87.9 ± 11.8, 82.7 ± 13.9 and 84.5 ± 12.8 in group II ($P < 0.05$).

The mean arterial pressure (MAP) (mmHg) showed a significant decrease when compared in both groups before LVP, 1 h after and 24 h after LVP, respectively, showing values of 86.3 ± 11.8, 81.7 ± 10.9 and 84.5 ± 12.1 in group I and values of 83.2 ± 12.2, 79.4 ± 11.6 and 80.7 ± 10.6 in group II ($P < 0.05$).

Discussion

In this study, all patients showed a significant decrease in HR 1 and 24 h after LVP compared with the baseline HR. This is in agreement with a study conducted by Appenrodt *et al.* [14] who found a significant decrease in the HR 1 and 24 h after paracentesis compared with the baseline HR (80, 76 and 72 beats/min) in cirrhotic patients with ascites. Similarly, Umgelter *et al.* [15] reported that there was a reduction in the HR from 101 beats/min (85–116) before paracentesis to 91 beats/min (68–106) after paracentesis, and Savino *et al.* [16] found a reduction in the HR from 104.46 ± 18.36 beats/min before paracentesis to 100.4 ± 16 beats/min after paracentesis ($P < 0.001$).

In the current study, there was a significant reduction in the MAP after LVP. Similar to this finding, García-Compean *et al.* [17] observed a reduction in the MAP from 89 ± 11 mmHg before paracentesis to 84 ± 11 mmHg 24 h after paracentesis with albumin substitution ($P < 0.05$). Also, Appenrodt *et al.* [14] found a significant decrease in the MAP 1 and 24 h after paracentesis compared with that before paracentesis [77 mmHg (63–83), 73 mmHg (67–78) and 72 mmHg (65–77), respectively]. Finally, Umgelter *et al.* [15] showed that the MAP reduced

Table 2 Haemodynamic parameters before, 1 h after and 24 h after LVP

Studied variables	Mean ± SD (Range)		*t*-test	*P*-value
	Group I (n = 25)	Group II (n = 25)		
CO (ml/min)				
Before paracentesis	7486.8 ± 1265.1 (5605–10 370)	7576.5 ± 1012.3 (5160–9095)	0.28	<0.01
1 h after paracentesis	7647.04 ± 1325.9 (5840–10 266)	7886.9 ± 969.4 (5644–9516)	0.73	<0.01
24 h after paracentesis	7552.2 ± 1311.7 (5600–10 320)	7644.6 ± 999.6 (5229–9125)	0.28	<0.01
SV (ml/beat)				
Before paracentesis	91.4 ± 16.3 (59–122)	92.04 ± 13.98 (60–126)	0.14	<0.01
1 h after paracentesis	98.8 ± 17.9 (59–146)	98.4 ± 13.7 (68–122)	0.09	<0.01
24 h after paracentesis	95.2 ± 17.3 (60–136)	95.5 ± 14.2 (63–125)	0.06	<0.01
CI (ml/min/m^2)				
Before paracentesis	4056.6 ± 570.3 (3051–5185)	4139.04 ± 522.2 (3080–4947)	0.53	<0.01
1 h after paracentesis	4171.9 ± 609.3 (3073–5110)	4245.4 ± 685.8 (2884–5265)	0.4	<0.01
24 h after paracentesis	4090.2 ± 576.8 (5160–2947)	4175.12 ± 511.5 (3197–5100)	0.55	<0.01
Right RI (ratio)				
Before paracentesis	0.704 ± 0.0357 (0.61–0.77)	0.814 ± 0.0279 (0.77–0.86)	12.2	<0.01
1 h after paracentesis	0.681 ± 0.0411 (0.58–0.75)	0.789 ± 0.0299 (0.72–0.83)	10.7	<0.01
24 h after paracentesis	0.6812 ± 0.0435 (0.57–0.78)	0.788 ± 0.0309 (0.71–0.83)	9.9	<0.01
Left RI (ratio)				
Before paracentesis	0.703 ± 0.0355 (0.61–0.77)	0.814 ± 0.0272 (0.76–0.85)	12.42	<0.01
1 h after paracentesis	0.678 ± 0.0431 (0.56–0.76)	0.788 ± 0.0289 (0.72–0.82)	10.6	<0.01
24 h after paracentesis	0.679 ± 0.046 (0.55–0.78)	0.786 ± 0.028 (0.72–0.83)	9.86	<0.01

CI, cardiac index; CO, cardiac output; LVP, large-volume paracentesis; RI, resistive index; SV, stroke volume.

from 81 mmHg (74–100) before paracentesis with albumin substitution to 77 mmHg (68–93) 24 h after paracentesis, and Lai *et al.* [18] reported a significant decrease in the MAP during the first 2 h of paracentesis. This decrease in MAP is probably due to a decreased intravascular volume as a result of rapid reformation of ascites. Furthermore, Phillip *et al.* [19] showed a decrease in the MAP and the systemic vascular resistance immediately, 2 h after and 6 h after paracentesis.

Regarding other haemodynamic changes in this study, the mean value of CI increased in both groups after LVP, but not to a significant level. Also, Umgelter *et al.* [20] reported an increase in CI from 4.12 l/min/m^2 before paracentesis to 4.55 l/min/m^2 after paracentesis with albumin substitution, and Savino *et al.* [16] reported an increase in CI from 3.90 ± 1.21 l/min/m^2 before paracentesis to 4.42 ± 1.21 l/min/m^2 after paracentesis, (P < 0.001) in cirrhotic patients with tense ascites. Increasing CI after paracentesis has been attributed to an improved venous return and right ventricular filling, as impingement of the elevated diaphragm on the right heart is reduced by the decreased IAP as a consequence of LVP [21]; also, as the MAP decreased, it was believed that a decrease in the afterload caused by a decrease in the systemic vascular resistance due to decreased IAP after paracentesis was the reason for the enhanced CI [20].

Singh *et al.* [22] observed that there was no significant increase in the PRA after LVP with albumin substitution (45.90 ± 8.59 ng/ml/h) compared with that before LVP (43.18 ± 10.73 ng/ml/h) (P = 0.273). Similarly, Appenrodt *et al.* [14] reported no significant difference in plasma renin before and after paracentesis (385 and 402 μU/ml, respectively). Lai *et al.* [18] revealed similar results. All previous results are comparable to the current study. However, comparing the PRA between both groups, a significant increase in PRA was found in the HRS group (P < 0.01). These findings were similar to Ruiz Del Arbol *et al.* [23], who found an increase in the PRA from 9.9 ± 5.2 ng/ml/h in cirrhotic patients without HRS to 17.5 ± 11.4 ng/ml/h in cirrhotic patients complicated by HRS. As in HRS, there is a primary peripheral arterial vasodilatation and mesenteric blood pooling, resulting in a poorly effective arterial blood volume and compensatory stimulation of endogenous vasopressor systems [15]; also, in HRS, a mild increase in portal pressures leads to the up-regulation of nitric oxide synthase [24].

The present study showed that LVP with albumin substitution improved the RI significantly in cirrhotic patients with tense ascites in both groups. Any decrease in RI may be the consequence of diminished renal vascular resistance caused by reduced IAP and retroperitoneal pressure after LVP [6]. Also, the finding of decreasing RI despite decreasing HR may be interpreted as indicating a reduced renal vascular resistance [20]. However, on comparing both groups, the RI was significantly higher in the HRS group compared with the ascitic group before or after paracentesis.

An increased UO and an enhanced haemodynamic status after paracentesis were initially observed in cirrhotic patients with an elevated IAP. Paracentesis influenced the haemodynamic status favourably as expressed by an increased CO and improved the renal function in patients with cirrhosis and tense ascites [21,25]. Umgelter *et al.* [20] reported an increase in the UO from 12 ml/h before paracentesis to 16 ml/h after paracentesis with albumin substitution in cirrhotic patients with tense ascites and HRS. In addition, García-Compean *et al.* [17] reported that there was a significant increase in the UO from 612 ± 593 ml/day before paracentesis to 904 ± 502 ml/day after paracentesis (P < 0.05) in cirrhotic patients with tense ascites. Maslovitz *et al.* [26] detected a significant increase in the daily UO from 925 ± 248 ml/day before paracentesis to 1523 ± 526 ml/day after paracentesis (P < 0.001), and Savino *et al.* [16] found an increase in the UO from 46.74 ± 26.46 cm^3/h before paracentesis to 54.95 ± 24.52 cm^3/h after paracentesis (P < 0.01).

García-Compean *et al.* [17] reported that there was no significant difference in serum creatinine before and 24 h after LVP (0.8 ± 0.3 and 0.8 ± 0.4 mg/dl, respectively). Also, Maslovitz *et al.* [26] reported no significant difference in serum creatinine before and after paracentesis (0.84 ± 0.17 and 0.8 ± 0.12 mg/dl, respectively). In contrast, results of the present study showed a significant reduction in serum creatinine 24 h after LVP. Similar findings were observed by Savino *et al.* [16], who showed a significant decrease in serum creatinine from 1.37 ± 0.49 mg/dl before paracentesis to 1.32 ± 0.58 mg/dl after paracentesis (P < 0.001). The decrease in serum creatinine attributed to the increased CO due to increased cardiac compliance after paracentesis and the decreased IAP improved renal perfusion by lowering venous and retroperitoneal pressures [15] as the impairment of renal function caused by direct renal compression due to increased IAP. These events might be the reason for the improvement in renal perfusion and in serum creatinine as a consequence [16].

Umgelter *et al.* [20] reported an increase in GFR from 5 ml/h before paracentesis to 9 ml/h after paracentesis with albumin substitution in cirrhotic patients with tense ascites and HRS. Also, in another study, Umgelter

et al. [15] discovered a significant elevation in GFR from 23 ml/min before paracentesis to 34 ml/min 24 h after paracentesis in cirrhotic patients with tense ascites and HRS, which is consistent with the finding of an improved renal function and GFR after LVP and albumin substitution in the current study [20].

Nguyen-Khac *et al.* [27] reported a strong correlation of electrolytes in the serum of cirrhotic patients with that in the ascitic fluid as the ascitic Na^+ was 133.1 ± 6.6, blood Na^+ was 131.8 ± 6.3 mmol/l ($P < 0.0001$), ascitic K^+ 4.1 ± 0.8, blood K^+ 4.3 ± 0.9 mmol/l ($P < 0.0001$) and ascitic Cl^- was 107.2 ± 7.6, and blood Cl^- was 101 ± 7 mmol/l ($P < 0.0001$). Finally, in the present study, there was a positive correlation between serum and ascitic electrolytes (Na^+, K^+ and Cl^-) in both groups.

Conclusion

PRA is significantly higher in patients with HRS and LVP, with albumin substitution leading to a significant reduction in HR and MAP, serum creatinine, BUN and RI and a significant increase in GFR, but there was no significant effect on the PRA. Finally, there was a significant correlation between the level of serum and ascitic fluid electrolytes.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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