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Sequential Hemodynamic Changes for Large Volume Paracentesis in Post-hepatitic Cirrhotic Patients with Massive Ascites

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ABSTRACT

Large volume paracentesis (4.8 to 15.5 liters) was performed in 42 patients with post-hepatitic cirrhosis and massive ascites, not only to derive parameters capable of predicting the development of severe clinical hypotension after large volume paracentesis, but also to determine the optimal time to introduce preventive volume expanders. Systemic hemodynamics were sequentially measured for 72 hours in thirty-two patients. Severe clinical hypotension occurred in 13 (31.0%) patients 4-62 hours from the start of paracentesis. Univariate analysis, with the Mantel-Cox test used to compare Kaplan-Meier curves, and the subsequent multivariate analysis by stepwise Cox regression procedure were utilized to identify two variables, withdrawn ascitic fluid greater than 7.5 liters ($p = 0.0121$) and the absence of peripheral edema ($p = 0.0148$), reaching statistical significance to predict the occurrence of severe clinical hypotension. Compared to the baseline value, the cardiac output of patients not developing severe clinical hypotension increased (6.26 ± 0.66 vs. 6.65 ± 0.69 liter/min, $p < 0.01$) one hour from the start of paracentesis and right atrial pressure decreased (11.2 ± 2.4 vs. 8.7 ± 2.3 mmHg, $p < 0.05$). The cardiac output returned to the baseline value at the 9th hour. Based on the results presented herein, we can conclude that severe clinical hypotension occurs in a high percentage of patients with post-hepatitic cirrhosis and massive ascites within 72 hours from the start of large volume paracentesis. At potential risk of this occurring are those patients without peripheral edema and withdrawn ascitic fluid greater than 7.5 liters. Volume expanders should be introduced before 4th hour from the start of large volume paracentesis.

Key Words: cirrhosis; ascitic fluid; hemodynamics; paracentesis.

I. Introduction

Large volume paracentesis (LVP) has recently served as a highly promising clinical approach to effectively control intractable ascites and for the first-line management of massive ascites (Gines *et al.*, 1987; Kao *et al.*, 1985; Quentero *et al.*, 1985; Salerno *et al.*, 1987; Smart and Triger, 1990; Tito *et al.*, 1990). However, the necessity of preventive volume expanders in a LVP procedure and the optimal time to introduce them still remain controversial (Antillon *et al.*, 1990; Gines *et al.*, 1988; Pinto *et al.*, 1988; Simon *et al.*, 1987).

Most investigations involving the necessity of preventive volume expanders in a LVP procedure were conducted without taking hemodynamic measurements or with hemodynamic data of only the period during the staged withdrawal of ascites (Gines *et al.*, 1987; Gines *et al.*, 1988; Pinto *et al.*, 1988; Simon *et al.*, 1987; Salerno *et al.*, 1987). The only study concerning sequential hemodynamic changes after LVP examined patients mainly with alcoholic cirrhosis; in addition, severe clinical hypotension occurred in two out of the twenty-three

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patients (Panos *et al.*, 1990). Our previous investigation of LVP also revealed that severe clinical hypotension can occur in some non-alcoholic cirrhotic patients (Wang *et al.*, 1994). To our knowledge, sequential hemodynamic data of LVP in a large series of patients with post-hepatitic cirrhosis have never been reported. Therefore, this study not only delineates sequential hemodynamic changes during the 72-hour period after LVP in patients with post-hepatitic cirrhosis, but also derives parameters to accurately predict how severe clinical hypotension develops.

II. Materials and Methods

Patients in this study were admitted to our hospital for treating massive ascites. The criteria required for inclusion included the following: (a) post-hepatitic cirrhosis with the diagnosis based on histopathologic examination or compatible laboratory data and sonographic findings (Hess *et al.*, 1989; Leio *et al.*, 1989); (b) being hemodynamically stable without evidence of congestive heart failure, chronic obstructive pulmonary disease, gastrointestinal bleeding, encephalopathy, acute pancreatitis, bloody ascites, peritoneal carcinomatosis or spontaneous bacterial peritonitis; (c) serum bilirubin < 10 mg/dL; (d) prothrombin time (INR) < 1.6; (e) peripheral platelet count > 40000/mm^3; and (f) serum creatinine < 3.5 mg/d L. Our Hospital Ethics Committee approved the study protocol, and the patients gave written consent.

After admission, diuretic treatment was withdrawn and patients were administered a daily 2 gm sodium diet with free fluid intake for 3 days before paracentesis. Paracentesis was performed using a standard peritoneal dialysis kit with a flexible sheath introducer in the morning (Day 0). In sterile condition and after local anesthesia, the needle was inserted in the midline, 2 cm below umbilicus. Once the needle entered the peritoneal cavity, the inner part was removed and the ascitic fluid was mobilized by static pressure. The physician remained at the patient's bedside during the entire procedure to ensure that no bleeding occurred at time of paracentesis by the color of ascitic fluid withdrawn. Ultrasonography was performed to confirm that the ascitic fluid was completely removed. No diuretics were administered and a daily 2 gm sodium diet was given with free fluid intake until day 5.

In the first ten patients (Group I), hemodynamic measurements were taken only before and after paracentesis for the first ten patients with hepatic vein and right atrium catheterization method as in our previous reports (Wang *et al.*, 1994). After paracentesis for the patients in Group I was completed, blood pressure was measured every 0.5-4 hours with an external sphygmomanometer for 5 days. The remaining patients (Group II) were continuously monitored in the intensive care unit for 5 days with systemic hemodynamics sequentially measured for 72 hours. A Swan-Ganz catheter was introduced for right atrium catheterization via the internal jugular vein. After 30 minutes of equilibration, pulmonary artery mean (PAMP), pulmonary capillary wedged pressure (PCWP), right atrial pressure (RAP), mean arterial pressure (MAP), heart rate (HR) and the cardiac output (CO) were measured at baseline and 1, 2, 3, 4, 6, 9, 12, 18, 24, 48 and 72 hrs from the start of paracentesis. The cardiac output was measured by thermodilution, using 10 ml of 0-4 °C 5% dextrose water injected through the Swan-Ganz catheter into the pulmonary artery (Forrester *et al.*, 1972). The zero reference point was set at the midpoint between the anterior sternal surface and the dorsal surface of the patient. Pressure and CO were measured in duplicate. Systemic vascular resistance (SVR) was calculated using the formula: SVR=79.9 (MAP-RAP)/CO, where SVR is in dyn.sec.cm^{-5}, MAP and RAP are expressed in millimeters of mercury and CO in liters per minute.

Severe clinical hypotension was defined when systolic pressure fell below 80 mmHg and at least 15 mmHg lower than baseline pressure within 5 days. When it occurred, albumin (10 gm for per liter of ascitic fluid withdrawn) and other colloid were infused to maintain blood pressure. To find predictors on how severe clinical hypotension develops, an analysis was made of the 24 variables—including age, sex, time to complete paracentesis, the amount of withdrawn ascitic fluid, baseline body weight, the ratio of withdrawn ascitic fluid to baseline body weight, absence of lower leg edema, baseline MAP, baseline CO, hemoglobin, blood sugar, prothrombin time, liver function tests (serum levels of total protein, albumin, bilirubin, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase and cholesterol), renal function tests (blood urea nitrogen and serum creatinine) and levels of serum electrolyte (potassium and sodium) and the score of Pugh's modification of Child's classification (Pugh *et al.*, 1973).

Statistical Analysis: Data were presented as mean ±SEM. Univariate analysis of severe clinical hypotension was performed for each of the twenty-four variables by the Kaplan-Meier method; the curves were also compared by the Mantel-Cox test (Wang *et al.*, 1991). For the calculation, all patients were grouped according to the presence or absence of each qualitative variable or the value higher or lower than the median value as a cut-off level for each quantitative variable. Variables reaching statistical significance in the univariate analysis were introduced into a multivariate analysis using a stepwise Cox regression procedure.

Those calculations were made using the BMDP (1L and 2L) program (Dixon, 1985). The paired Student's *t* test or Wilcoxon signed rank test was used for the other statistical analyses when deemed appropriate. A *p* value less than 0.05 is considered significant.

III. Results

Forty-two patients (thirty-seven men and five women, aged 43-76 years with a mean of 61.2 ± 1.4 years), ten in group I and thirty-two in group II, were included in this study. The etiology of cirrhosis was hepatitis B in thirty-six patients, hepatitis C in six patients. Table 1 lists their clinical and biochemical characteristics. The amount of withdrawn ascitic fluid was 4.8-15.5 liters, with a mean of 8.2 ± 0.5 liters in all patients. The total time taken to complete paracentesis was 128 ± 7 minutes in group I and 106 ± 6 minutes in group II.

Severe clinical hypotension occurred in thirteen (31.7%) of forty-two patients, two in group I and 11 in group II, during the period from 4 hours 15 minutes to 62 hours from the start of paracentesis (Fig. 1). No severe clinical hypotension or other life-threatening complications occurred in patients of both groups before paracentesis was completed. In the univariate analysis of twenty-four variables with data obtained from forty-two patients of both groups, three variables, withdrawn ascitic fluid greater than 7.5 liters ($p = 0.0121$), the absence of peripheral edema ($p = 0.0148$) and the ratio of withdrawn ascitic fluid to baseline body weight greater than 12% ($p = 0.0226$) reached statistical significance to accurately predict the occurrence of severe hypotension. Only two variables, i.e. withdrawn ascitic fluid greater than 7.5 liters and absence of peripheral edema, reached statistical significance in the subsequent multivariate analysis by the stepwise Cox regression procedure (Fig. 2).

The hemodynamic data of patients of Group I were not included in the calculation because hemodynamic measurements were taken immediately before and after paracentesis in patients of Group I, and the approach to measure hemodynamics was different from that used in patients of Group II. In addition, because albumin and other colloid were infused to maintain blood pressure when severe clinical hypotension occurred, the subsequent hemodynamics were changed in eleven patients. Two patients were dropped out due to either esophageal variceal bleeding or refusal of further investigation. Therefore, 72-hr hemodynamic changes were completely observed in only nineteen of the patients in Group II (Fig. 3). Compared to the baseline value, CO significantly increased 1 hr later (6.26 ± 0.66 vs. 6.65 ± 0.69 liter/min, $p < 0.01$), reached peak value at the second hr, remained so for the next 2 hours, and then dropped to the baseline value or a lower level from 9 to 72 hours. RAP (11.2 ± 2.4 vs. 8.7 ± 2.3 mmHg, $p < 0.05$), PCWP, MAP and SVR significantly decreased 1 hr later and progressively dropped to a nadir at the 9th hr. Next, SVR gradually returned to baseline value at 12th hr; however, RAP, PCWP and MAP remained at low values until 72 hr later. In ten patients of Group I, compared to the baseline value before paracentesis, CO was significantly increased after paracentesis; MAP and SVR significantly decreased.

Table 1. Clinical and Laboratory Features of the Patients Studied

	Group I (n=10)	Group II (n=32)
Sex (M/F)	7/3	30/2
Age (yrs)	62.1 ± 2.9	59.8 ± 1.6
Peripheral edema	6	22
Baseline systolic pressure (mmHg)	121 ± 4	117 ± 4
Withdrawn ascitic fluid (liters)	8.1 ± 0.9	8.7 ± 0.6
Albumin (gm/dL)	2.7 ± 0.2	2.4 ± 0.2
Bilirubin (mg/dL)	2.8 ± 0.6	2.5 ± 0.5
Aspartate aminotransferase (IU/L)	122 ± 34	87 ± 32
Alkaline phosphatase (U/L)	143 ± 11	129 ± 23
Cholesterol (mg/dL)	138 ± 6	107 ± 15
Prothrombin time (INR)	1.42 ± 0.09	1.46 ± 0.07
Glucose (mg/dL)	118 ± 15	113 ± 12
Hemoglobin (gm/dL)	10.7 ± 0.5	10.1 ± 0.4
Creatinine (mg/dL)	1.3 ± 0.2	1.2 ± 0.2
Blood urea nitrogen (mg/dL)	32 ± 3	24 ± 4
Serum sodium (meq/L)	135 ± 2	137 ± 2
Pugh's classification	10.5 ± 0.4	10.7 ± 0.4

Group I, patients with hemodynamics measured only before and after paracentesis; Group II, patients with sequential hemodynamic measurements for 72 hours

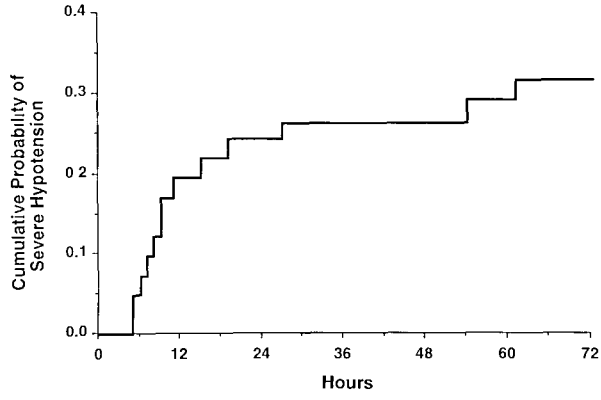

Fig. 1. Cumulative probability of severe clinical hypotension within 72 hours from the start of large volume paracentesis in 42 post-hepatitic cirrhotic patients with massive ascites.

IV. Discussion

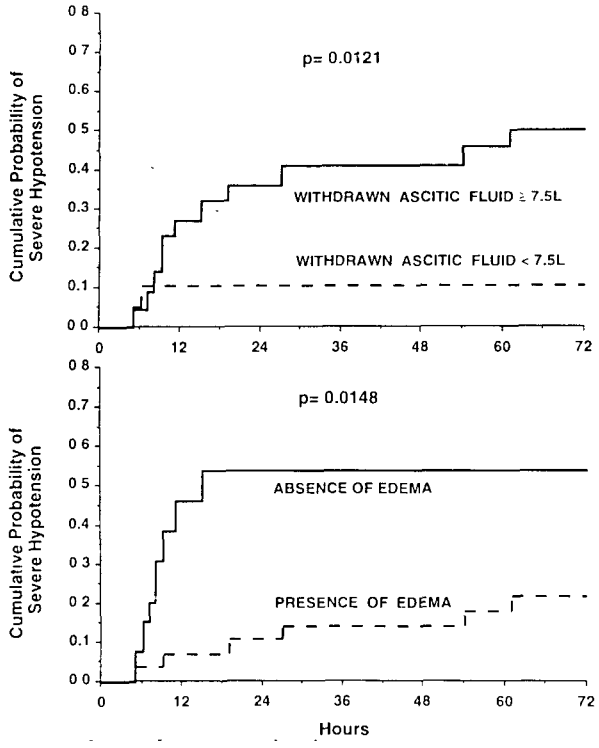

Fig. 2. Cumulative probability of severe clinical hypotension within 72 hours from the start of large volume paracentesis in 42 post-hepatitic cirrhotic patients with massive ascites, classified according to the only two variables — the amount of withdrawn ascitic fluid and the absence of peripheral edema — reaching predictive value in the multivariate analysis.

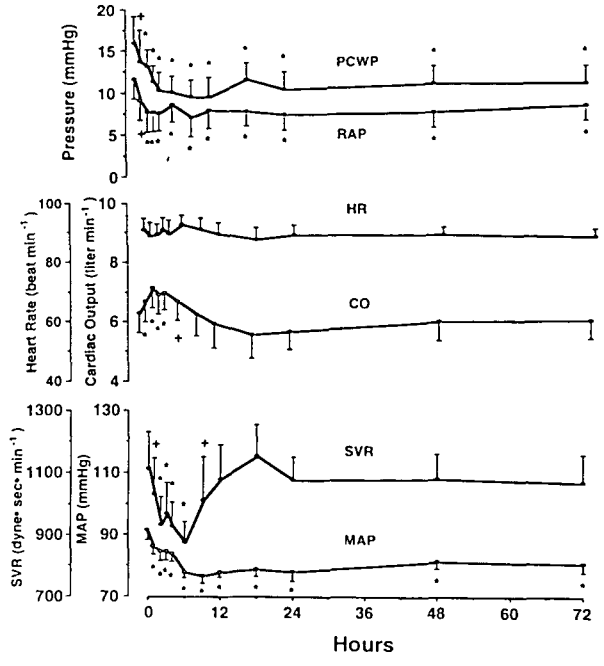

Fig. 3. Sequential changes in pulmonary capillary wedged pressure (PCWP), right atrial pressure (RAP), heart rate (HR), cardiac output (CO), systemic vascular resistance (SVR) and mean arterial pressure (MAP) within 72 hours from the start of large volume paracentesis in nineteen post-hepatitic cirrhotic patients with massive ascites without development of severe clinical hypotension All values are mean ±SEM. Results are compared with the baseline value. $^{*}p < 0.01$, $^{+}p < 0.05$.

This preliminary study is part of a series of studies involving hemodynamic changes of the period lasting 72 hours from the start of large volume paracentesis in patients with post-hepatitic cirrhosis. Most investigations involving paracentesis lacked hemodynamic measurements or involved a procedure with less ascitic fluid removed each time (Antillon *et al.*, 1990; Kao *et al.*, 1985; Pinto *et al.*, 1988). A few studies concerning hemodynamic changes after LVP, including our previous one, either evaluated the period during the staged withdrawal of ascitic fluid (Wang *et al.*, 1994), with limited-volume paracentesis (Guazzi *et al.*, 1975; Kowalski *et al.*, 1954) or assessed the changes only within 24 or 48 hours in patients with mainly alcoholic cirrhosis (Panos *et al.*, 1990; Simon *et al.*, 1987). This study indicates that severe clinical hypotension occurs in more than 30% of the patients, i.e. significantly higher than the 8.7% reported in patients with mainly alcoholic cirrhosis (Panos *et al.*, 1990). The reasons for the discrepancy in the two studies remain unknown. Ethnic differences, different etiologies of cirrhosis or different definitions of systemic clinical hypotension may partially account for such a discrepancy. Herein, severe clinical hypotension was strictly defined. However, comparing the two series is impossible since Panos *et al.* (1990) did not clearly define severe clinical hypotension in their study. Nevertheless, results obtained from both series confirm the importance of preventive volume expanders for LVP in patients with alcoholic or post-hepatitic cirrhosis.

The sequential hemodynamic measurements taken herein after LVP in patients without severe clinical hypotension reveal a gradual decrease in cardiac output to near or even lower levels than the baseline value at the 9th hour. Our previous investigation (Wang *et al.*, 1994) indicated LVP may deteriorate renal function, even in patients without severe clinical hypotension if volume expanders were not infused. These findings not only imply the insufficiency of effective blood volume possibly owing to relative hypovolemia caused by progressive reaccumulation of ascites, but also confirm the necessity of preventive volume expanders after LVP in patients with or without severe clinical hypotension.

The multivariate analysis in this study reveals that both variables, withdrawn ascitic fluid greater than 7.5 liters and the ratio of withdrawn ascitic fluid to baseline body weight greater than 12%, reach statistical significance to predict the occurrence of severe clinical hypotension after LVP. Above findings can be accounted for by the greater ascitic fluid withdrawal or by a higher ratio of withdrawn ascitic fluid to the body weight in Chinese patients with a smaller body size than Westerners, resulting in a distinct and accelerated decrease of intravascular volume to compensate for.

Previous investigations concerning LVP without preventive volume expanders revealed no difference in hemodynamics between those with and without peripheral edema (Panos *et al.*, 1990; Simon *et al.*, 1987). Nevertheless, Pockros and Reynolds (1986) demonstrated the importance of peripheral edema in protecting from plasma volume contraction while treating with a large dose of oral diuretics. This study shows that patients without peripheral edema are at risk of developing severe clinical hypotension after LVP. Our results confer that peripheral edema is a prerequisite for protecting from plasma volume contraction when treating massive cirrhotic ascites.

No severe hypotension or other life-threatening complications occurred during the withdrawal of ascitic fluid in our series. Immediately after the start of paracentesis, CO increased and RAP decreased. These findings not only correspond to previous reports (Panos *et al.*, 1990; Salerno *et al.*, 1990; Simon *et al.*, 1987), but also confirm that cardiac function is transiently improved after LVP. Furthermore, the prominent driving forces affecting hemodynamics during the initial stage are the relief of right atrial compression and increased venous return caused by the relief of intraabdominal and intrathoracic pressures. After the initial stage, unfortunately, cardiac function returns to the baseline value and relative hypovolemia caused by progressive reaccumulation of ascites may cause severe clinical hypotension in some patients, particularly in those with a greater ascitic fluid withdrawal or without the protection of peripheral edema.

All episodes of severe clinical hypotension in our series occur after the 4th hr from the start of paracentesis. The sequential hemodynamic changes reveal that CO reaches the peak level at the 2nd hr and gradually falls to the baseline value at the 9th hr. Based on above results, we can conclude that preventive volume expanders should be administered before 4th hour from the start of large volume paracentesis in all patients with or without severe clinical hypotension.

In summary, severe clinical hypotension occurs in a high percentage of patients with post-hepatitic cirrhosis and massive ascites within 72 hours from the start of LVP. Patients without peripheral edema and withdrawn ascitic fluid greater than 7.5 liters are potentially at risk. Moreover, infusion of preventive volume expanders is prerequisite for LVP.

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肝炎後肝硬化合併巨量腹水病患施行大量腹水放液術之連續血流動力變化

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爲尋找大量腹水放液術後併發嚴重低血壓的臨床指標，以及決定靜脈注射預防性血容擴張劑的恰當時間，我們於42位肝炎後肝硬化合併巨量腹水病患施行大量腹水放液術(4.8~15.5公升)。其中32位病患施行72小時連續性系統血流動力學檢查。結果顯示，十三位病患(31.0%)於腹水放液後4~62小時間發生嚴重低血壓。我們以Mantel-Cox檢驗比較Kaplan-Meier曲線作單變項分析，並進一步以Stepwise Cox Regression步驟做多變項分析，獲得預測發生嚴重低血壓的兩個變項—腹水放液超過7.5公升(P = 0.0121)及無末稍水腫(P = 0.0148)—具有統計學上的意義。與基礎值比較，未發生嚴重低血壓病患放液 1 小時後的心輸出量增高 (6.26 ± 0.66 vs. 6.65 ± 0.09 L/min，$p < 0.01$)，而右心房壓降低(11.2 ± 2.4 vs. 8.7 ± 2.3 mmHg，$p < 0.05$)。心輸出量於第 9 小時回復至基礎值。結論：肝炎後肝硬化合併巨量腹水病患施行大量腹水放液術之 72 小時內產生嚴重低血壓之比率甚高，而無末稍水腫病患或腹水放液超過 7.5 公升病患之危險性更高。大量腹水放液術後 4 小時內應施行靜脈注射預防性血容擴張劑。