# The Role of Ascitic Paracentesis in Liver Cirrhosis in Improving the Function and Structure of the Heart

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**ABSTRACT**

*Heart abnormalities in cirrhotic patients have been known for five decades, with atria and ventricular dilatation. Pozzi et al reported that in cirrhotic patients with or without ascites, the diastolic function was lower than control. Ascitic paracentesis improved diastolic function. The diameter of both atria was larger in cirrhotic patients with or without ascites. The diastolic diameter of left ventricle did not differ significantly in cirrhotic patients with or without ascites compared to control, but there was an increase after paracentesis. Ejection fraction was lower in cirrhotic and increased after ascitic parancentesis although the increase was not significant. The aim of the Study: To compare the function and structure of the heart before and after ascitic paracentesis in cirrhotic patients.*

***Methods**: This study took place from February 2000 to April 2001 in dr. Pringadi Hospital/H.Adam Malik Hospital. There were 18 samples (12 men and 6 women), 15 of which were cirrhotic patients with tense ascites and 3 cirrhotic patients with refractory ascites. The mean age was 51,8 + 8,28 years, the youngest being 29 years and the oldest 65 years. The mean ascitic fluid removed by paracentesis was 7,20 liters with a range of 5 to 9 liters. Immediately following paracentesis, Dextran 40 % was administered at a dose of 8g/ 1L ascitic fluid aspirated.*

***Results**: The diameter of the four heart chambers decreased after paracentesis, but the decrease was not statistically significant. There was increase in E/A ratio after ascitic paracentesis, from 0,93 + 0,370 to 1,06 + 0,383 (significant, $p<0,05$), meaning that there was an improvement in diastolic function after ascitic paracentesis. There was also an increase in ejection fraction from 68,99 + 13,26 % to 72,10 + 11,10 %, but this was not significant ($p>0,05$).*

***Conclusion**: After paracentesis, there was a significant improvement in diastolic function while the diameter of the four heart chambers decreased and the ejection fraction increased insignificantly.*

***Keywords**: Ascitic paracentesis - liver cirrhosis - heart function and structure.*

## INTRODUCTION

Ascites is a symptom indicating the severity of liver cirrhosis. The prognosis of cirrhosis following the appearance of ascites ranges from 1-2 years. The prevalence of ascites in liver cirrhosis ranges from 10-89%, with annual incidence rates ranging from 3-10%.[1] Heart disorders in liver cirrhosis patients have been known since over 50 years, but the more specific possibilities have been detected just recently.[2] The disorders found are marked with electrophysiological and mechanical disruptions, mainly as a response to stress loads.[3] Cirrhotic patients undergoing maximum treadmill test have only a 97% increase in cardiac output, compared to control subjects who have an up to 300% increase in cardiac outputs under the same load.[4] This situation is accompanied with an increase in thickness and rigidity

of ventricle walls that leads to disruptions of diastolic function with a variety of fractions.[3,5] Atrial and ventricle diameters are greater in liver cirrhosis patients with ascites.[6,7,8] The diameter of the left atrium, which is greater than in control subjects, demonstrates the presence of fluid retention, and disorder in left ventricle contraction. The increase in systolic and diastolic ventricle diameter in liver cirrhosis is interpreted as a secondary impact to the increase in circulating blood volume. Pozzi et al reported that the ejection fraction of the left ventricle is lower in liver cirrhosis patients with ascites compared to normal people, and will increase following paracentesis even though the increase was not significant.[9] His study in liver cirrhosis patients with ascites also demonstrate increased ventricle diameter 24 hours following total paracentesis, whereas the atrial diameter was not changed compared to pre-paracentesis.[6,8]

In liver cirrhosis with or without ascites, failure in diastolic function is found, marked by decreased E/A ratio compared to normal subjects. There is improved A wave and E/A ratio following paracentesis. Ejection fraction is an evaluation of the left ventricle systolic function. It can be measured by measuring the end diastolic volume and end systolic volume. The E wave rapid filling phase of the left ventricle is followed by an opening of the distal valve. The A wave left ventricle filling is followed by normal atrial contraction E/A ratio of >1.[10]

Based on the information above, the author studied the relationship between ascites in liver cirrhosis and the changes of heart function and structure.

Diastolic function consists of 4 phases:
1. Iso-volume relaxation time
2. Rapid filling phase (E wave)
3. Diastalsis (equal pressure of left ventricle and right ventricle)
4. Atrial contraction phase (A wave)

## THE AIM OF STUDY

To compare the function and structure of the heart prior to and following ascites paracentesis in liver cirrhosis.

## MATERIALS AND METHODS

This study is experimental, conducted from February 2000 to April 2001 at Dr.Pirngadi Hospital/ Adam Malik Hospital, Medan.

**Case group**: Patients with liver cirrhosis with ascites who were not responsive to high-dose diuretics therapy (400 mg spinorolakton and 40 mg Furosemide). The patients were diagnosed based on Suharyono Subandiri criterias and USG.

**Exclusion**: heart diseases (valve disorder, hypertension, congenital heart disease, cardiomyopathi, cardiac arrhythmia), severe lung disease, systemic disease, bleeding in less than 3 months, anemia of Hb<7 gr/dl, liver cancer, hepatic encephalopathy, creatinine levels of more than 1.5 mg%, alcohol consumption in less than 3 months, those undergoing treatment for hypertension, bilirubine levels of more than 10 mg%, platelet counts of less than 40.000/mm$^3$.

Patients underwent routine laboratory check up, liver function test, SPE and renal function test. Then echocardiography was taken as a preliminary data. Ascites liquid paracentesis was conducted to the minimum with abocathe number 16 under ultrasound guidance. After parasentesis was performed, the patient received infusion with Dextran 40% 8 gr/l ascites fluid. Repeat echocardiography was performed 24 hours following paracentesis.

The parameters measured:
- Left and right atrial diameters
- Left and right ventricle diameters
- Left ventricle ejection fraction
- E/A ratio

Echocardiography was performed with Logic 500 made by General Electric USA, using the American Society of echocardiography criteria.[11]

## RESULTS

Out of 18 patients with liver cirrhosis with ascites (12 males and 6 females), 15 patients had tense ascites and 3 patients were not responsive to high dose diuretics therapy (400 mg Spironolactone and 40 mg Furosemide). The mean age was 51.8 + 8.28 years. The youngest was 29 years old and the oldest was 65 years old.

**Table 1 A Comparison of Blood Pressures Prior to and Following Paracentesis**

| Variable | n | X (mmHg) | SD | p |
|---|---|---|---|---|
| S-BP-1 | 18 | 119.17 | 8.09 | 0.079 |
| S-BP-2 | 18 | 115.28 | 7.57 | |
| D-BP-1 | 18 | 74.5 | 5.11 | 0.049 |
| D-BP-2 | 18 | 72.50 | 4.93 | |

Note
S-BP : Systolic blood pressure
D-BP : Diastolic blood pressure
1 : Prior to paracentesis
2 : Following paracentesis

From the table above, it can be seen that following paracentesis, there was a decrease in both systolic and diastolic blood pressures. Yet, the decrease was not statistically significant (p>0.05).

**Table 2. A Comparison of the Diameter of the Four Heart Chambers Prior to and Following Paracentesis**

| Variable | n | X (mmHg) | SD | p |
|---|---|---|---|---|
| D-RA-1 | 18 | 26.87 | 5.73 | 0.591 |
| D-RA-2 | 18 | 26.31 | 6.67 | |
| D-LA-1 | 18 | 32.78 | 3.72 | 0.155 |
| D-LA-2 | 18 | 31.40 | 3.90 | |
| D-RV-1 | 18 | 33.57 | 6.67 | 0.637 |
| D-RV-2 | 18 | 33.20 | 6.81 | |
| D-LV-1 | 18 | 49.81 | 7.38 | 0.154 |
| D-LV-2 | 18 | 48.37 | 6.84 | |

Note:
D-RA : Diameter of the right atrium
D-LA : Diameter of the left atrium
D-RV : Diameter of the right ventricle
D-RV : Diameter of the left ventricle

From the table above, it can be seen that in all four chambers of the heart, there was a decrease in diameter following paracentesis. Yet, the decrease was not statistically signicant (p>0.05).

**Table 3. A Comparison of the A/A Ratio Prior to and Following Paracentesis**

| Variable | n | X (mmHg) | SD | p |
|---|---|---|---|---|
| E/A-1 | 18 | 0.93 | 0.370 | 0.002 |
| E/A-2 | 18 | 1.06 | 0.383 | |

From the table above, it was revealed that there was an increase in the E/A ratio from 0.93 + 0.370 to 1.06 + 0.383. This increasing was statistically significant (p<0.05). In other words, there was an improvement in diastolic function following ascites paracentesis.

**Tabel 4. A Comparison of E-wave Prior to and Following Paracentesis**

| Variable | n | X (mmHg) | SD | Df | t-test | p |
|---|---|---|---|---|---|---|
| E-WAVE-1 | 18 | 0.626 | 0.185 | 17 | 0.48 | 0.640 |
| E-WAVE-2 | 18 | 0.636 | 0.191 | | | |

Note:
E-wave on echocardiography

From table above, it is demonstrated that there was no difference in the velocity of the E wave, prior to and following paracentesis (p>0.05).

**Table 5. A Comparison of A-wave Prior to and Following Paracentesis**

| Variable | n | X (mmHg) | SD | p |
|---|---|---|---|---|
| A-WAVE-1 | 18 | 0.690 | 0.157 | 0.001 |
| A-WAVE-2 | 18 | 0.621 | 0.122 | |

Note:
A-WAVE: A-wave on echocardiography

From table above, it is shown that there was a significant decrease in A wave following paracentesis from 0.690 + 0.157 m/s to 0.621 + 0.122 m/s (p>0.05).

**Table 6. A Comparison of Ejection Fraction Left Ventricle Prior to and Following Paracentesis**

| Variable | n | X (mmHg) | SD | p |
|---|---|---|---|---|
| EF-1 | 18 | 68.99 | 13.26 | 0.214 |
| EF-2 | 18 | 72.10 | 11.10 | |

From table above, it is shown that there was an increase in ejection fraction from (68.99 + 13.26)% prior to paracentesis to (72.10 + 11.10)%. However, the increase was not statistically significant (p>0.05).

## DISCUSSION

In portal hypertension, peripheral vasodilatation and sodium retention are hemodynamic disorders preceding plasma volume expansion leading to a hyperdynamic situation in liver cirrhosis. The pathogenesis of this hyperdynamic situation in liver cirrhosis is the increase in vasodilator substances that are normally metabolized by the liver.[12] These substances are: intestinal polypeptide vasoactive, prostacyclin, endotoxin, glucagons, and endothelial relaxation factor, nitric oxide and alpha TNF.[13,14] Another mechanism is the failure of the liver to produce vasoconstrictor substances. There is also proof that the several vasodilators produced and some substances that are inactivated or secreted by the lungs are inable to pass through the lungs due to arterial venous shunting.

The study by Kowalski et al in the year 1950 demonstrated a hyperdynamic circulation in liver cirrhosis manifested in increased cardiac output and decreased systemic vascular resistance and the

presence of peripheral vasodilatation.[9,11] Several studies also show that in portal hypertension with or without primary liver disease, there is limited heart function. The limitations include minimum preload reservation and failure of heart contraction. Elements correlating with vasodilatation cause disorders of the two. The chronic increase in cardiac output and humoral factors such as alpha TNF could disrupt cardiac function.[14] In liver cirrhosis, hepatocellular failure is obviously found. This failure may result in the accumulation of toxins, such as endotoxins, which are normally cleaned or inactivated by the liver. Such toxic substances have a direct effect on the heart. In the liver cirrhosis, there is also an increase in norepinephrine, which is known to be able to cause the myocardial damage and desensitized beta adrenergic receptors.[14]

Ventricular failure in liver cirrhosis often appears due to medical intervention such as liver transplantation, portosistemic shunt operation and Transjugular intrahepatic portosystemic operation. These procedures induce physical stress to the heart, and installing a shunt will lead to high venous return. In a study, 7.3% of deaths following heart transplantation was caused by congestive heart failure. Several heart failures have been reported following the insertion of Transjugular intrahepatic portosistemik stent shunts, as quoted from. [9]

Atrial and ventricular diameters are greater in liver cirrhosis patients with ascites. The diameter of the left atrium, which is greater than in control subjects, show fluid retention and abnormal contraction of the left ventricle.[11] The increase in systolic and diastolic diameters of the ventricles in liver cirrhosis are interpreted as a secondary impact to the increase in circulating blood volume.[9] Pozzi et al reported from a study on liver cirrhosis patients with ascites that there was an increase in ventricle diameter 24 hours following total paracentesis, whereas the atrial diameter was not changed as compared to pre-paracentesis.[17] On the other hand, Arbol et al reported that on the first day of paracentesis cardiac index tended to increase, but it returned to the figure prior to paracentesis following the sixth day.[15]

In this study, both atrial and ventricle diameters decreased following paracentesis even though the decrease was not statistically significant ($p>0.05$). The results we obtained differed from the study by Pozzy et al. It may be due to differences in the method of study. Pozzy used albumin to replace plasma fluid, while our study used Dextran-40. The use of Dextran-40 as plasma replacement produced nearly 40-50% circulation failure, marked by an over 50% increase of plasma renin activity compared to the level prior to paracentesis, while the use of albumin as plasma replacement produced only 15% circulation failure.[15] In this study, plasma renin activity was not examined.

The study by Pozzi et al reported a lower left ventricle ejection fraction in liver cirrhosis patients with ascites compared to non-ascites cirrhotic patients and healthy subjects, even though still within the normal range, and a slight increase following paracentesis.[9]

In this study there was an increase in the ejection fraction following paracentesis from 68.99 + 13.26% to 72.10 + 11.10%, but it was not statistically significant ($p>0.05$).

Doppler echocardiography was able to monitor ventricular filling. The filling was divided into the velocity of the peak wave filling during early ventricle diastolis (E wave) and the velocity of wave filling during atrial systolis (A wave). The E/A wave velocity ratio is usually used as an index for diastolic function. Under normal circumstances, the E wave is greater than the A wave, while under abnormal conditions, it demonstrates abnormal diastolic function where the role of the atrium is more dominant than the early ventricle dilatation component due diastolic rigidity of ventricle.[6]

In liver cirrhosis, there is an increase in the thickness and rigidity of the ventricle wall, which leads to the disruption in diastolic function.[3] In a study, diastolic function disorders were generally found in liver cirrhosis with ascites, and there was also an increase in preload caused by increased intravascular circulation volume and decreased afterload caused by peripheral vasodilatation, where both factors could prevent abnormal diastolic function. On the other hand, the physical effect of ascites liquid liquid pushes the diaphragm into the thoracic cavity, thus disturbing the function of the ventricles.[3,6]

This study revealed a significant increase in E/A ratio from 0.93 + 0.370 to 1.06 + 0.383 ($p<0.05$). From the two components of wave that determine the diastolic function in this study, there was an increased A wave and E wave, even though the increase in E wave was not statistically significant (table IV-V). In this study, the difference in the diameter of the heart chambers was not statistically significant prior to and following paracentesis, thus, it may not be the dominant factor leading to diastolic dysfunction.[3,6]

In conclusion the diameters of all four chambers of the heart decreased following paracentesis but the decrease was not statistically significant, the ejection

fraction increased following paracentesis but the increase was not statistically significant, there was a significant improvement in diastolic function following ascites paracentesis which is marked by the increase in E/A ratio, there was a significant decrease in cardiac pulse rate following ascites paracentesis.

Further study involving more samples and longer follow up is required. Neurohumoral measurements are needed to determine its role in changing the hemodynamic status in this study.

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