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Changes of Portal Flow in Patients with an Acute Exacerbation of Heart Failure and Liver Congestion

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Abstract

Background: *Acute heart failure may be either new heart failure or worsening of pre-existing chronic heart failure. Liver congestion causes transmission of flow waveforms portal vein.*

Aim: *to describe changes of portal vein flow in patients with an acute exacerbation of heart failure.*

Patients and methods: *Thirty patients with exacerbation of chronic heart failure presenting at Heart failure unit, National Heart Institute. Doppler ultrasound and measurement of portal vein pulsatile index (PI) were done for all patients.*

Results: *Based on PI, the following 4groups were defined: Group 1 (PI < 0.5): 4 patients (13.3%), Group 2 (PI 0.5 – 0.99): 11 patients (36.7%), Group 3 (PI =1): 9 patients (30%) and Group 4 (PI >1): 6 patients (20%). Mean total serum bilirubin was 1.2 ± 0.4 in group 1, 1.7 ± 0.6 in group 2, 2.1 ± 0.3 in group 3, and 2.8 ± 1 in group 4. This represents a statistically significant linear correlation between PI and total serum bilirubin (p value= 0.01). The mean right atrial pressure (RAP) was 8.5 ± 4 mmHg in group 1, 13.5 ± 5 mmHg in group 2, 20 ± 3.5 mmHg in group 3, and 22 ± 4 mmHg in group 4. This represents a statistically significant linear correlation between PI and RAP (p value= 0.01).*

In conclusion: *portal vein flow has significant changes in patients with acute exacerbation of heart failure which are proportional to serum level of total bilirubin, RAP, severity of tricuspid regurge and severity of heart failure*

Introduction

Heart failure (HF) is a complex syndrome with multiple organ manifestations. Extracardial organ damage (kidney, liver, lung, and skeletal muscles) present typically in the advanced stages of chronic heart failure and have nonspecific morphological as well as functional manifestations. Liver disorders in chronic heart failure result from either congestion and/or hypo perfusion [(1).]

Liver damage in heart failure is manifested by hepatomegaly and subicterus, especially in cases where right-heart failure is present. In patients with acute HF and a low cardiac index (CI), in which the predominant

mechanism of liver damage is hypoperfusion, liver transaminases rise significantly as a manifestation of ischemic hepatitis. [2].

Increased serum levels of liver amino transferases and total bilirubin in patients with chronic heart failure [3] or pulmonary arterial hypertension indicate a worse prognosis [4].

The portal vein is interposed between the capillary network of splanchnic circulation and hepatic sinusoids. There are some reports demonstrating the unique changes of portal vein flow in HF with right ventricular dysfunction [5].

Liver congestion causes structural changes permitting the transmission of flow waveforms during right ventricular filling and ventricular contraction to the portal vein. A simple measurement of portal flow could be a good tool for detection and quantification of systemic congestion [6].

This study aimed to describe the changes of the portal vein flow in patients with an acute exacerbation of HF in relation to central hemodynamics and biochemical indicators of liver lesion.

Patients and methods

This single center, hospital-based study was conducted on 30 patients with exacerbation of chronic heart failure presenting at Heart failure unit, National Heart Institute (NHI) for evaluation and treatment of HF between July 2012 and December 2013. The diagnosis of HF was based on a combination of physical examination, laboratory tests, and imaging.

Exclusion criteria: Patients with primary liver disease associated with heart failure including any patient who had history of chronic liver disease, biliary disease, or other serious liver illness.

A written informed consent was obtained from all participating patients prior to inclusion in the study.

All included patients were subjected to the following:

Liver tests: included serum bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), and serum albumin.

Prothrombin time, activity and international normalized ratio (INR)

Viral markers: HBsAg and HCV antibody

Abdominal ultrasonography

Echocardiography

Doppler examination of portal vein

Protocol of Doppler examination:-

- Fasting 8-12 hours before examination.
- Approach: intercostal approach in supine position.
- Portal vein sample was taken from its main ramification. If it's not possible samples was taken from main right branch.
- Sample volume adjusted to cover maximum amount of flow.
- Samples taken during short interruption of breathing in a slight inspirum.
- We calculated pulsatile index (PI) as the ratio of difference of maximal and minimal velocity to maximal flow velocity.

(Vmax- Vmin/ Vmax).

Based on PI, the flow will be categorized to one of four groups: - (***5 and 7).***

- If the PI was less than 0.5 the PV flow was classified as continuous or sub continuous (Group 1).
- We considered the flow pulsatile when the PI was 0.5 - 0.99 (Group 2).
- If the flow over the cardiac cycle decreased to zero, we considered it as intermittent, PI = 1 (Group 3).
- In cases when flow reversed in systole (PI > 1), it was considered as alternating (Group 4).
- Central haemodynamics were evaluated by using central venous catheter to measure right atrial pressure (RAP)**.**
- All patients received diuretics, 95% received angiotensin converting enzymes inhibitors (ACEI), 90% received spironolactone, 92% received digoxine (no B-blocker was administrated).

Results

The total number of the study population was 30 patients with mean age of 56 ± 9 years and 24 of them were males (80%).

Thirteen patients (43.3%) had heart failure due to ischemic heart disease, while 17 patients (56.7%) were due to dilated cardiomyopathy.

The mean blood pressure (systolic/diastolic) (mmHg) was 100/65 (± 7/4).

The mean left ventricular ejection fraction (LVEF) (%) was 23 ± 8.

Seventeen patients (56.7%) had tricuspid regurge (TR) III/IV.

Based on PI, the flowing four groups were defined:

Group 1 (PI < 0.5): the PV flow is classified as continuous or sub continuous, 4 patients (13.3%) were categorized into this group

Group 2 (PI 0.5 – 0.99): the PV flow is classified as pulsatile, 11 patients (36.7%) were categorized into this group

Group 3 (PI =1): the PV flow over the cardiac cycle decreases to zero, 9 patients (30%) were categorized into this group

Group 4 (PI > 1): the PV flow is classified as alternating, 6 patients (20%) were categorized into this group.

The mean total serum bilirubin (mg/dec) was 1.2 ± 0.4 in group 1, 1.7 ± 0.6 in group 2, 2.1 ± 0.3 in group 3, and 2.8 ± 1 in group 4. This represents a statistically significant linear correlation between PI and total serum bilirubin (p value= 0.01).

The mean ALT (U/L) was 49 ± 8 in group 1, 51 ± 7 in group 2, 48 ± 10 in group 3, and 52 ± 8 in group 4. This represents a statistically non-significant correlation between PI and ALT (p value= 0.283).

The mean AST (U/L) was 48 ± 5 in group 1, 49 ± 5 in group 2, 46 ± 7 in group 3, and 48 ± 6 in group 4. This represents a statistically non-significant correlation between PI and AST (p value= 0.308).

The mean ALP (U/L) was 20 ± 5 in group 1, 22 ± 9 in group 2, 23 ± 7 in group 3, and 21 ± 4in group 4. This represents a statistically non-significant correlation between PI and ALP (p value= 0.327).

The mean serum albumin (mg/dL) was 3.2 ± 0.2 in group 1; 3.1 ± 0.2 in group 2; 2.9 ± 0.3 in group 3; and 3 ± 0.1in group 4. This represents a statistically non-significant correlation between PI and serum albumin (p value= 0.327).

The mean INR was 1.4 ± 0.2 in groups 1, 1.4 ± 0.3 in group 2, 1.3 ± 0.4in group 3, and 1.6 ± 0.2 in group 4. This represents a statistically non-significant correlation between PI and serum albumin (p value= 0.066).

The mean RAP was 8.5 ± 4 mmHg in group 1, 13.5 ± 5 mmHg in group 20, 20 ± 3.5 mmHg in group 3, and 22 ± 4 mmHg in group 4. This represents a statistically significant linear correlation between PI and RAP (p value= 0.01).

Out of the whole study population, 14 patients had TR. Four patients were classified to have mild to moderate TR (TR I, II), while 10 patients were classified to have moderate to severe TR (TR III, IV).

Among patients with TR (I, II), 2 had continuous flow, one patient had pulsatile flow, and one patient had intermittent flow. While in patients with TR (III, IV), one patient had pulsatile flow, 6 had intermittent flow, and 3 had alternating flow as shown in table (1).

All the studied patients were classified for severity by NYHA classification as NYHA **I** in 8 patients, NYHA **II** in 7 patients, NYHA **III** in 8 patients and NYHA **IV** in 7 patients.

Among patients with NYHA (I), 3 had continuous flow, 3 had pulsatile flow, and 1 had intermittent flow 1alternating. While in patients with NYHA (II), 2 continuous flow, 3 had pulsatile flow, 1 had intermittent flow, and 1 had alternating flow. In patients with NYHA (III), 1 continuous flow, 2 had pulsatile flow, 3 had intermittent flow, and 2 had alternating flow. In patients with NYHA (IV), 1 continuous flow, 1 had pulsatile flow, 2 had intermittent flow, and 3 had alternating flow as shown in table (2).

Table (1): Correlation between PI and TR

Number of patients with TR			
TR (III,IV)	**TR (I,II)**		
0	2	***Group 1(continous flow)***	
1	1	***Group 2 (pulsatile flow)***	
6	1	***Group 3 (intermittent flow)***	
3	0	***Group 4 (alternating flow)***	
3.836		X^2	**Chi-Square**
0.044		p-value	

Table (2): Correlation between PI and stage of heart failure

p value	*Total no in each group*	*Group 4 alternating flow*	*Group 3 intermittent flow*	*Group 2 pulsatile flow*	*Group 1 continous flow)*	**Pulsatility index**
	8	1	1	3	3	**Stage** I HF
	7	1	1	3	2	**Stage** II HF
	8	2	3	2	1	**Stage** III HF
	7	3	2	1	1	**Stage** IV HF
0.021	30					

Discussion

Acute and chronic heart failure may lead to acute ischemic hepatitis or chronic congestive hepatopathy [(8)].The deleterious effects of HF on the liver are a consequence of two mechanisms: (1) hepatic congestion secondary to volume and pressure overload; and (2) reduced cardiac output and compromised liver perfusion causing hypoxic injury [(9)].

Changes in hepatic function that are proven by laboratory tests are significant in predicting the survival of patients with severe heart failure. Therefore, the evaluation of cardiac and hepatic function is very important in patients with severe HF and hepatic injury. Their treatment options should be revised in order to ensure stable hemodynamics, as well as optimal liver function, and so in this way their survival and prognosis could be improved [(10)].

In our study, we found that in pure cardiac liver disease, the PV flow becomes pulsatile. This comes in agreement with ***Duerinckx et al.,*** who studied the portal vein waveforms in heart failure patients and compared portal vein spectral patterns to right atrial pressures and concluded that portal vein pulsatility is a sign of elevated systemic venous pressure [(11)]. Also ***Gaasch,* et al., 2004** showed that in primary liver disease (cirrhosis, steatosis, hepatitis). The flow through the PV is flat. This difference in flow pattern could be very useful in the diagnosis of primary liver disease in CHF patients [(12)].

In the current, study there was significant correlation between PI and total bilirubin. These findings were comparable to the findings of **Sun et al., 2012** who analyzed the relationship between serum total bilirubin in relation to exacerbation of congestive heart failure (CHF) and subsequent long-term mortality in patients with CHF [(13)]. They found that increased total bilirubin is coincident with cardiac decompensation and it also predicts a worse long-term death of CHF, presumably through the potential liability to both decompensated RV function and lower cardiac output syndrome which occurs simultaneously when HF deteriorates. This also comes in agreement with **Chintanaboina , et al.2013** who concluded that in patients with acute decompensated HF, elevated serum TB on admission with or without low EF (< 35%) predicts a worse prognosis and early future readmission secondary to HF (14). Finally, **Allen et al., 2009** found that elevated total bilirubin is one of the strongest predictor for poor prognosis of CHF [(15)].

Although the serum AST, ALT and ALP in this study were elevated, there was no significant correlation between PI and elevated level of neither serum aminotransferases nor ALP.

Hypoalbuminemia in CHF is thought to result mainly from malnutrition, inflammation and cachexia. Other causal factors include hemodilution, liver dysfunction, protein-losing enteropathy, increased transcapillary escape rate, and nephrotic syndrome. Low albumin may be because of decreased albumin synthesis from hepatic congestion and right heart failure [16].

Serum albumin level was declined in our patients but there was no significant correlation between PI and decreased level of serum albumin. The decreased level of albumin was shown also by **Tamara, et al. 2008** who studied the effect of serum albumin level on survival in patients with advanced HF. The study concluded that hypoalbuminemia is common in HF and is independently associated with increased risk of death in HF [17]. Also, **Ambrosy, et al. 2012** studied the prevalence and importance of liver function test (LFT) abnormalities in HF patients and have concluded that baseline and in-hospital changes in serum albumin and total bilirubin provide additional prognostic value [18].

The elevation of INR in HF patients can be explained by fact that in end-stage HF there is often congestive hepatopathy. The underlying pathophysiology is related first to the poor end-organ perfusion leading to ischemic parenchymal changes with hepatocellular necrosis especially in cases of acute decompensation. Second, passive hepatic venous congestion which develops in the setting of right heart dysfunction with increased RAP [19].

INR was mildly elevated in our patients but there was no significant correlation between PI and elevated level of INR. The mild elevation of INR comes in agreement with **Giallourakis et al, 2002** who concluded that HF patients have mild elevation in INR which is usually multifactorial but most probable cause is liver dysfunction in GHF [20].

We found that there was significant correlation between PI and RAP. The correlation between PI and RAP can be explained by that in CHF there is reduced EF of the heart so there is increased afterload in the right ventricle. The increased afterload will decrease evacuation of right atrium during diastole leading to elevation of RAP. As there are no valves between right atrium and inferior vena cava this will lead to transmission of the elevated pressure to inferior vena cava and hepatic veins. The liver in passive "backward" congestion status can develop hepatomegaly and synchronous pulsation. The high pressure of the hepatic veins will be transmitted through the liver to the portal vein leading to increase in its pressure which will appear in the form of changes of flow by Doppler. [21].

Our finding is coincident with the finding of **Rengo et al. 1998** who studied portal vein flow in patients with CHF and control subjects. The portal flow was recorded by color Doppler sonography**.** The study found that hepatic congestion and TR were associated with a higher portal vein PI, thus indicating that portal vein pulsatility ratio (PR) reflects the level of impairment of the right heart [22]. Also, **Jui-Ting Hu, et al. 2003** who studied the peak-to-peak PI of portal blood flow in order to predict the right-sided congestion in CHF patients. They concluded that the measurement of Portal vein flow changes identify patients with CHF [7]. This finding adds more evidence to our result. This also comes in agreement with **Shih et al. 2006** who studied the use of PI for evaluation of right heart function. He found that that the occurrence of congestive liver is common in patients with CHF. This was accompanied by dilatation of inferior vena cava and hepatic veins during abdominal sonography, and PI was coincident with CI. So, he concluded that the measurement of both CI and PI is helpful for the indirect non-invasive evaluation of right heart function [23].

In this study there was significant correlation between PI and TR.

The relation between portal vein flow and TR is well established. One of the interesting studies in this point is the work of **Sakai et al., 1983,** he studied TR by analyzing the blood flow pattern in the portal vein using a combined system of a pulsed Doppler technique and two-dimensional echocardiography Inferior vena cava dimension (IVCD), hepatic vein dimension (HVD) and the blood flow pattern in the portal vein were compared with the severity (negative, mild, moderate and severe) of TR assessed by right ventriculography and with right atrial and ventricular pressures. The following conclusions were derived from the study: IVCD and HVD in a group of TR severe were significantly larger than those of the other groups. By portal flow patterns, it was possible to differentiate the TR of severe and moderate groups from the TR of mild group. The Doppler shifts were well correlated with RAP and right ventricular end-diastolic pressure [(24)].

The relation between TR and PI can be clarified by the anatomical position of the tricuspid valve which lies between right atrium and right ventricle. Normally, during systole blood passes from systemic and portal veins through superior and inferior vena cava to be evacuated in right atrium while tricuspid valve is closed. During diastole the tricuspid valve opens and blood passes from right atrium to right ventricle. In case of TR during systole the tricuspid valve is opened so blood passes from right ventricle to right atrium so decrease evacuation of vena cava leading to venous congestion in both systemic and portal circulations. As the severity of tricuspid valve regurgitation increases more blood will be pumped backwards from right ventricle to right atrium to cause inferior vena congestion which will be transmitted to portal vein leading to its pulsatility wave changes [(25)].

In 1990 **Abu-Yousef , et al,** made an important study about Pulsatile portal vein flow as a sign of TR on duplex Doppler sonography. He analyzed the pulsatile portal venous waveform in which minimum velocity dropped to or below zero on Doppler and investigated its possible association with TR. At the end of the study he showed that detection of a pulsatile portal venous waveform on Doppler in patients with liver dysfunction should raise the possibility of TR [(26)]. This finding is also comparable to the work of **Loperfido, et al. 1993** who studied Portal and hepatic vein flow-velocity profiles by pulsed Doppler in patients with TR and normal subjects to determine if portal vein flow analysis is useful in the evaluation of TR. They concluded that there were changes in portal vein flow associated with TR especially alternating flow. This finding can be explained by the fact that TR is associated with high right ventricular filling pressures leading to congestion of liver and causes secondary changes in portal vein flow [(27)].

As regarding the severity of HF which was detected by using NYHA classification, we found that there was significant correlation between it and PI. The correlation between PI and NYHA stages can be explained by the fact that with increasing severity of heart failure there is increase in volume overload. This volume will lead to increase in systemic and portal congestion which will be reflected in the form of changes in portal vein flow [(28)].

The earliest evidence of that finding came from **Hosoki et al. 1990** who studied patients with CHF were examined with duplex sonographic scanning of the portal vein. Increasing pulsatility of the Doppler signals was demonstrated in 11 patients with severe CHF. Two patients with severe CHF showed decreasing pulsatility of portal Doppler signals in response to therapeutic procedures the time-velocity waveform shape of portal flow is, to a large degree, influenced by the mechanical events in the right side of the heart in severe CHF which is compatible with severity of HF [(29)]. This finding was also shown by **Catalano, et al. 1998** who examined the relationships between measurements of liver and spleen dimensions and blood flow in portal and hepatic veins, assessed non- invasively by Doppler sonography, and compared them with echo-cardiographic data. He found that patients with more severe left ventricular failure (NYHA class III-IV) showed more dilatation of the left ventricle and atrium, reduced systolic function, and reduced portal vein mean velocity compared with patients with milder heart failure (NYHA class I-II); in addition, the hepatic

vein diameter was increased and portal vein PR was reduced (1).This also comes in agreement with **Eriksen et al. 2006** who studied Hepatic and renal haemodynamic changes in CHF. They ultrasonographically measured portal PR, maximal velocity in the hepatic vein during atrial contraction and in systole, the vena cava inferior diameter and collapsibility index, and renal resistive and pulsatility indexes**.** So they concluded that CHF causes caval hypertension and thus increased pressure within the portal and also renal vein [(30)].

In conclusion

Portal vein flow shows significant changes in in patients with an acute exacerbation of HF. The changes in portal vein flow are proportional to serum level of total bilirubin, RAP, severity of TR and severity of HF.

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