Research Article

Prevalence & Risk Factors of Right-sided Pleural Effusion in Patients with Liver Cirrhosis and Ascites

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Abstract

Pleural Effusion is an important complication in patients with liver cirrhosis and ascites. Aim: to evaluate the prevalence and risk factors of right-sided pleural Effusion in patients with liver cirrhosis and ascites. Methods: Consecutive patients with liver cirrhosis and ascites admitted to our inpatient department from January to December $\gamma \cdot \gamma \gamma$ were included in the study. Clinical history and examination, abdominal ultrasonography, ascitic fluid aspiration, chest x ray and echocardiography were done for every patient. **Results:** Y (12.1%) patients out of 127 (had right sided pleural effusion. Patients with right-sided pleural effusion were considered group A, compared to the rest of the patients (group B). Jaundice and abdominal tenderness were significantly more prevalent in group A than in group B (p = $\cdot \cdot \cdot \cdot , \cdot \cdot \cdot , \cdot \cdot \cdot$ respectively). The mean Hb level was significantly lower in group A $(p=\cdot,\cdot)$. The total leucocytic count and the ascitic fluid cell number were significantly higher in group A ($p=\dots$). Patients in group A had significantly higher MELD score $(\Upsilon^{q}, \Upsilon^{\pm} \circ, \Upsilon)$ compared to patients in group B $(\Upsilon^{\bullet}, \Upsilon^{\pm}, \Lambda)$ (p=•.•). Pulmonary systolic pressure was significantly elevated in patients with right- sided pleural effusion ($p=\dots$). Multivariate analysis revealed that abdominal tenderness, abnormal bilirubin level and pulmonary hypertension were independent factors associated with pleural effusion in patients with liver cirrhosis and ascites. Conclusion: Pleural Effusion is a common finding in our patients with liver cirrhosis and ascites. Abdominal tenderness, abnormal bilirubin level and pulmonary hypertension may predict patients at risk for pleural effusion

Key words: liver cirrhosis-pleural effusion-ascites

Introduction

Right- sided pleural effusion is transudative pleural effusion in the absence of underlying cardiac or pulmonary disease in patients with portal hypertension^{(1).}

Its prevalence has been estimated to be o-10.7% in patients with liver cirrhosis^(1,7). The presence of portal hypertension is the key factor in the development of ascites and right- sided pleural effusion in cirrhosis. Important mechanism leading to the passage of ascetic fluid from the peritoneal cavity into the pleural cavity is the presence of diaphragmatic defects. These defects were corroborated by passage of ⁹^mTc-human showing albumin from the abdominal into the pleural cavity, even in the absence of underlying ascites^(°). Most diaphragmatic

defects, also referred to as pleuroperitoneal communications or blebs, are $<^{1}$ cm in size and are predominantly located on the right hemi diaphragm⁽ⁱ⁾. In addition, negative intrathoracic pressure is believed to contribute to the one-way directional flow of ascetic fluid from the abdominal cavity. Ascites is detectable in over $\wedge \cdot \stackrel{?}{\times}$ of those with right- sided pleural effusion, but is not required for diagnosis^(°, °). The aim of this study was to determine the prevalence and risk factors of right- sided pleural in patients with cirrhotic ascites.

Patients and Methods

The setting for this study was the gastroenterology and hepatology inpatient department, Minia University Hospital, Minia, Egypt. The study was approved by the institutional review board of the Minia Faculty of Medicine, Minia University, Egypt. Written, informed consent was obtained from every participant. A crosssectional study design was initiated in the inpatient setting. Consecutive patients with liver cirrhosis and ascites admitted to our department were enrolled from January to December **Y**. Y. Full clinical history and examination, abdominal ultrasonography, chest x ray and echocardiography were performed for every patient. Ascitic fluid analysis and blood examination was also performed for all patients. Patients with pleural effusion due to other causes than cirrhosis: infectious diseases, heart and kidney disease or tuberculosis were excluded from the study. Patients with hematemesis, encephalopathy, deteriorated renal function, coagulopathy, and patients with secondary bacterial peritonitis, peritoneal tuberculosis, and peritoneal carcinomatosis were also excluded.

Data were collected for all patients and analysis included classification of patients to two groups according to the presence or absence of pleural effusion.

Statistical analyses were performed using Stats Direct version $7.7.\circ$ statistical software (Stats Direct Ltd., Sale, Cheshire, UK) and SPSS for Windows version $11.\circ$ (SPSS, Inc., Chicago, IL, USA). All data are presented as means \pm SD. Chi square, Fisher's tests, Student's t test and multivariate analysis were used when appropriate. Significance was accepted at $P = *..\circ\circ$.

Results

Among $1\xi\gamma$ consecutive patients with Liver cirrhosis and ascites prospectively evaluated, γ patients (1ξ . \forall ?) had right sided pleural effusion one of them had bilateral pleural effusion. The \uparrow patients with right-sided pleural effusion were $\uparrow \uparrow$ male and \circ female, with a mean age $\circ \uparrow$. $\uparrow \pm \lor$. ϵ years. We considered these patients as group A and the rest of the patients ($\uparrow \uparrow \uparrow$ patients) with liver cirrhosis and ascites were taken as control (group B).

The demographic, clinical, laboratory and radiologic data are shown in table). The history of jaundice, and abdominal tenderness were significantly more prevalent in •.• ° respectively). The mean Hb level was significantly lower in group A $(p=\cdot,\cdot)$. The total leucocytic count and the ascetic fluid cell number were significantly higher in group A $(p=\cdot,\cdot,\cdot)$. The MELD score (ranges from Λ - $\tau\Lambda$), with significantly higher values in group A $(\Upsilon^{9}, \Upsilon^{\pm} \circ. \Upsilon)$ indicating more severe disease than in group B $(\uparrow \cdot, \uparrow \pm \lor, \land)$ (p=·.·). The score was calculated using the original formula without including the cause of liver disease. Also Child classification was used to classify patients in the two groups according to the severity of the disease and results are shown in Table¹. the Echocardiography performed for all studied patients in group A and group B, and revealed a significantly more prevalent abnormalities in patients with right- sided pleural effusion (table γ). Pulmonary systolic pressure was significantly elevated in patients with right- sided pleural $(p=\cdot,\cdot,\cdot)$.

Multivariate analysis revealed that abdominal tenderness, abnormal bilirubin level and pulmonary hypertension were independent factors associated with HH in patients with liver cirrhosis and ascites (table \mathcal{T})

	Group A	Group B	P value
	(^ү patients)	(<i>\Y\</i> patients)	
Age (Mean ±SD)	07.7±4.2	٦٠ _. ٥±٦.٩	•_٦
Gender			
Male	۱۶ (V٤.۲%)	۸۶ (۲ ۱ .۱٪)	۰.۰
Female	٥ (٢٥.٨٪)	۳۰ (۲۸.۹)	
Smoking			
Smokers	۱۶ (۷٤.۲٪)	٦٩(٥٧٪)	•.1
Non smokers	٥ (٢٥.٨٪)	٥٢ (٤٣٪)	
Residence			
Rural	۱۷(۸۱٪)	۱۰۹ (۹۰٪)	•.•
Urban	٤ (١٩٪)	14 (1・%)	
Abdominal tenderness	10 (V1.£%)	۳٥ (۲۹٪)	• . • ٣
Jaundice	۲۰ (۹۰.۲٪)	۸۶ (۲ ۱. ٤٪)	• • • •
encephalopathy	٤ (١٩٪)	٤ • (٣٣٪)	• • • •
Bleeding	11(07.5%)	۲۳ (۲۷٪)	•.•
splenomegaly	۲۱(٥٢.٤٪)	٦٣(٥٢٪)	•.•
ALT	۷۱.۹±۱۰.۷	۶.۷±۷.۹	•.٢
Bilirubin	٤.٧±٠.٤	۳.۳±۰.۳	•.• *
Albumin	۳.۲±۰.۳	۲.۳±۰.۳	•.1
INR	۱.۸±۰.۳	۱.۸±۰.٤	•.٧
Hb	^.Y±•.^	۹.۸±۰.۹	•.• 1
Platelet count	×٤.١±٢٠.٣	۷٤.۸±۱۳.۲	•.1
WBCs $(1 \cdot 7)$	۸.۹±۳.۲	۳.۱±۱.۰۳	•.••1
Creatinine	۱.۱±۰.۳	۰.۸±۰.۳	•.٢
Alkaline phosphatise	۳.۳.٬±۱۸.٤	22.7±0.52	•.1
MELD score	۷۹.۳± ۵.۷	۲۰.۱± ۷.۸	•.• 1
Child-pugh	Child A · (· ½)	Child A1 · (^.٣%)	•.••1
classification	Child Bo (۲۳.۸%)	Child B^、(いいど)	
	Child C17 (77.7%)	Child C でい(ての. 7%)	
Ascitic fluid cells	7795.0±70X7.7	۲۳٤. <u>۸±۱٤۷.</u> ۱	1
Ascitic fluid albumin	۱.۱±۰.۲	11±7	•.٢

Table (1): Demographic characteristics, clinical and laboratory data of the patients included in the study

Table $({}^{\gamma})$: The echocardiographic findings in patients with and without hydrothorax.

Variables	With hydrothorax	Without hydrothorax	
	No= ۲ ۱	No= 171	
Normal (%of patients)	·.·(·.·٪)	٨٧(٧٢٪)	
TR(%of patients)	۷(۳۳٪)	۲۸(۳۳٪)	• • * *
TR, MR(%of patients)	٤(١٩٪)	٦(%)	
Pericardial effusion (% of patients)			
	٤(١٩٪)	١٢(٩.٩٪)	
IHD(%of patients)	۱(٤.٨٪)	۰ <u>.</u> •(۰.•٪)	A .11.
PASP (Mean ±SD)	۳۸.۱±۲.۹	40.N±2.7	•.••)**

Variables	dds ratio	१०% СІ	
Bilirubin	09.9	0.1_2927.1	. • • 2*
Jaundice	•_17	•- 1.989	.^
Abdominal tenderness	•_£	1.9-27.2	.•• \ *
Creatinin	•_^	•.1- *1*.*	.ź
Pulmonary hypertension	•_^	۱.۱-٦.٩	.• *
Alkalin phosphates	• • • ٣	11-10	.)
WBCs	• • • • •	1_1	.)
Platelets		1-1	.)
Hb	• 14	•.•٢-١.٤	.)
Ascitic fluid cells	•_17	• • • • 9 - ٣ 1	۲.

Table ($^{\gamma}$): Multivariate analysis of factors associated with development of pleural effusion among patients with liver cirrhosis.

Discussion

Right-sided pleural effusion is the presence of transudative pleural effusion in the absence of cardiac or pulmonary disease, in patients with portal hypertension. Passage of ascitic fluid from the peritoneal cavity into pleural cavity through diaphragmatic defects is the mechanism of rightsided pleural effusion(r). The prevalence of right-sided pleural effusion in our study was 15.1% which goes in accordance with other studies, where the prevalence ranged from $1.\circ$ to $1\circ.\%^{(1,7)}$. Malnutrition in cirrhosis is thought to contribute to thinning of the diaphragmatic muscle and to the development of these defects. In addition, negative intrathoracic pressure is believed to contribute to the one-way directional flow of ascitic fluid from the abdominal cavity $^{(1)}$.

Our results showed that all cases of pleural effusion were right sided except for one case of bilateral pleural effusion. In other studies, pleural effusion was right sided in $\vee \cdot \%$ of cases, left sided in $\vee \wedge \%$ and

bilateral in 1%?⁽¹⁾. In the review by Krok K., et al., $(\checkmark,)$, it was reported that effusion may affect one or both of the hemithoraces but largely affects the right hemithorax^(Y). Lazaridis KN, et al., (1999), found that The hydrothorax is usually right sided ($\land\circ$?), with isolated left sided presentation in 1%? and bilateral hydrothorax seen in only %?^(A) Absence of left sided cases in our study may be attributed to the small number of patients included.

Right- sided pleural effusion in the absence of ascites (as detected by clinical examination and attempted paracentesis) has been reported previously. $(^{(T, \hat{\eta})})$

Worsening of liver functions presented with infections and encephalopathy was more common in patients with right- sided pleural effusion^(``). Diagnosis of rightsided pleural effusion is usually suspected in patients with advanced cirrhosis or chronic liver disease. In our study the results of MELD score were $Y \P. T \pm \circ. Y$ in group A and $Y \cdot . Y \pm Y. A$ in group B. Present study is comparable with study done by Kuiper et al., $({}^{\tau} \cdot \cdot {}^{\tau})$, they found that rightsided pleural effusion was seen in patients with advanced stage of cirrhosis $({}^{(\cdot)})$. Worsening of liver function was shown to be associated with not only with rightsided pleural effusion but also with spontaneous bacterial pleuritis $({}^{(1)},{}^{(t)})$

In our study, pulmonary hypertension is significantly more common in patients with right- sided pleural effusion. Pulmonary hypertension may occur in the setting of portal hypertension, with or without advanced liver disease and called portopulmonary hypertension⁽¹⁷⁾ with an estimated prevalence of \circ .^{\circ} to \land . \circ ?^{$(15, 1\circ)$}.

The pathogenesis of portopulmonary hypertension (POPH) is triggered by vascular injury reflected by the development of plexiformarteriopathy, concentric intimal fibrosis, and proliferation and muscularization of the pulmonary arterioles⁽¹¹⁾. Initial symptoms of POPH are subtle patients and may remain asymptomatic at the time of diagnosis despite advanced disease^{$(1 \vee)$}. Whether it has direct relation to right- sided pleural effusion or it is a reflection of severe portal hypertension, pulmonary hypertension is a common finding in patients with rightsided pleural effusion. Puncho Gurung, et al., $(7 \cdot 1)$ found a high prevalence of diastolic dysfunction and left atrial enlargement in this group of patients with right- sided pleural effusion^(1^).

The presence of abdominal tenderness with elevated ascetic fluid cell count (spontaneous bacterial peritonitis) and jaundice was more significant in patient with right-sided pleural effusion in our study. Worsening of liver function was shown to be associated with not only right-sided pleural effusion but also with spontaneous bacterial pleuritis (SBP) $^{(15,7)}$.

In summary, right- sided pleural effusion is a common finding in our patients with liver cirrhosis and ascites. Jaundice, SBP and pulmonary hypertension are independent factors associated with right- sided pleural effusion in patients with cirrhotic ascites.

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