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Original Article

CIRRHOTIC CARDIOMYOPATHY IN EGYPTIAN PATIENTS

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ABSTRACT

Objective: Liver cirrhosis is a health care problem in Egypt caused by the high prevalence of hepatitis C virus (HCV) infection that affects 15-20 % of the population**.** Cirrhotic cardiomyopathy is the term used to describe a constellation of features indicative of abnormal heart structure and function in patients with cirrhosis.

Aim of this study is to assess the pattern and the extent of cardiac affection in cirrhotic patients and its relation to the presence or absence of ascites.

Medthods: This study was carried out on 70 patients with liver cirrhosis and 30 healthy controls. All persons were subjected to careful history & physical examination, laboratory investigations, abdominal ultrasonography,and echocardiography.

Results: left ventricle end diastolic diameter was significantly increased in cirrhotic patients with ascites (5.40±0.58) and without ascites ((5.31±0.51), compared to the control group (4.52±0.58) ($p<0.05$),. left ventricle end systolic diameter was increased in cirrhotic patients with ascites (3.57±2.2) and without ascites (3.46±3.1), without ascites (3.18±2.5)but the difference was statistically non significant ($p > 0.05$). Left atrium diameter & Right ventricular end diastolic diameter were significantly increased in cirrhotic patients compared to the control group ($p<0.05$ The pulmonary artery pressure was elevated in cirrhotic patients compared to the control group..

Conclusion: In the present study Liver cirrhosis is associated with significant enlarged cardiac chambers and. diastolic dysfunction compared to the control group specially in the presence of ascites.

Keywords: Cirrhosis, Ascites, Echocardiography Cardiomyopathy diastolic dysfunction

INTRODUCTION

Liver cirrhosis is a health care problem in Egypt caused by the high prevalence of HCV infection that affects 15-20 % of the general population [1]**.** Liver cirrhosis is associated with a wide range of cardiovascular abnormalities including hyperdynamic circulation, enlargement or hypertrophy of different cardiac chambers and electrophysiological changes such as QT prolongation [2]**.** In cirrhosis, despite the increased basal cardiac output, cardiac response to physiologic or pharmacologic stimuli is known to be subnormal a phenomenon called cirrhotic cardiomyopathy. The pathogenesis of cirrhotic cardiomyopathy is multifactorial and still incompletely defined; it includes abnormalities in the b-adrenergic signaling pathway, altered cardiomyocyte membrane fluidity, increased myocardial fibrosis, cardiomyocyte hypertrophy and ion channel defects [3]**.** Accumulating evidence suggests that cirrhotic cardiomyopathy plays a major role in the pathogenesis of cardiac dysfunction following liver transplantation or trans-jugular intra-hepatic porto-systemic shunt placement and contributes to the pathogenesis of hepatorenal syndrome [2]**.** Recognition of cirrhotic cardiomyopathy will depend on a high level of awareness and potentially will help better management of patients with cirrhosis [4]**.**

Aim of the work

The aim of this work is to study the pattern and the extent of cardiac affection in cirrhotic patients and its relation to the presence or absence of ascites.

MATERIALS AND METHODS

This study was done in the department of internal medicine, Fayoum teaching hospital; it was carried on 30 patients with liver cirrhosis and without ascites (10 females and 20 males) (Group -1), 40 patients with liver cirrhosis and ascites (12 females and 28 males) (Group - 2), and 30 healthy controls (14 females and 16 males) (Group - 3). Inclusion criteria : Patients diagnosed with liver cirrhosis depending on clinical evidence of stigmata of chronic liver disease (e.g. jaundice, ascites, palmer erythema, etc) and ultrasonographic coarse echo texture and shrunken liver. Exclusion criteria: Patients with cardiovascular diseases e.g. hypertension, ischemic heart disease, valvular heart disease and atrial fibrillartion, patients with severe anemia, renal failure and diabetes mellitus.

Each person included in the study was subjected to Careful history taking and thorough physical examination.

Laboratory investigations

Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), serum alkaline phosphatase, serum albumin, Prothrombin concentration, total and direct bilirubin and hepatitis markers for HBV and HCV

Abdominal ultrasonography

Using GE LOGIQ9 apparatus (GE Healthcare., Milwaukee, WI) with a 2–5 MHz convex array transducer (low frequency probe) and a 5–12 MHz convex array transducer (high frequency probe).recording both static and B-mode imaging in the fasting state. Series of images of both lobes of the liver were obtained for evaluation of the

parenchymal echo texture, focal lesions, volumetric changes, and edge evaluation. High-resolution images of the surface of the right and left lobes of the liver were obtained with high-frequency probes for evaluation of the surface of the liver. Additional images were obtained if required to assess portal tracts as well as and splenic veins and assessment of size of the spleen and the presence of ascites or retroperitoneal masses.

Standard twelve-lead electrocardiogram (12-lead ECG)

For assessment of cardiac rhythm and features suggesting of chamber enlargement or coronary artery disease (CAD).

Echo Doppler study

Echocardiography was performed using and ACUSON CV70 machine equipped with a 2.5/3.25 MHz annular array transducer. Patients underwent a complete echocardiographic examination including 2-D transt-horacic imaging, pulsed wave Doppler, continuous wave Doppler, and color flow mapping. The patients were monitored through a single-lead electrocardiogram. LV internal dimension, left ventricular end-diastolic (LVEDD) and end-systolic (LVESD) interventricular septal thickness (IVS), posterior wall thickness (PWT), right ventricular end-diastolic (RVEDD) and end-systolic (RVESD) and left atrium (LA) end-systolic diameter were obtained; left ventricular EF was calculated by Simpson's biplane method of discs. Pulsed wave Doppler echocardiography was used to evaluate trans-mitral LV filling velocities at the tips of the mitral valve leaflets and trans-tricuspid RV filling velocities at the tips of the tricuspid valve leaflets on the apical four chamber view and the peak early diastolic flow velocities (E), the peak of atrial flow velocities (A) and the ratio of E/A were determined. Color Doppler interrogation of mitral regurgitant jet in 2 orthogonal views to assess the severity of MR (mild MR: mean color flow jet area in the left atrium <4cm2; moderate MR: mean color flow jet area 4 to 8cm2, mean color flow jet area > 8 cm2) and TR jet to assess the severity of TR (mild TR: mean color flow jet area in the right atrium <5cm2; moderate TR: mean color flow jet area 5 to 10 cm2; severe TR: mean color flow jet area > 10 cm2).Pulmonary arterial pressure (PAP) was calculated pulmonary artery systolic pressure using the simplified Bernoulli equation from the velocity of the TR jet and RA pressure [5]."

STATISTICAL ANALYSIS

Collected data were computerized and analyzed using Statistical Package for Social Science (SPSS) version 16. Descriptive statistics were used to describe variables; percent, proportion for qualitative variables. Mean ± SD and range for Quantitative variables. Student's t-test was used to compare the normally distributed continuous variable between the patients with aortic valve stenosis and the healthy control group. Fisher-exact test and Chi-square test were used to compare categorical variables. P values with significance of less than 5% were considered statistically significant. For all statistical tests, a P value less than 0.05 was used to indicate significance.

RESULTS

This study was done in Fayoum university hospital; it was carried on 30 liver cirrhosis patients without ascites (Group -1), 40 liver cirrhosis patients with ascites (Group- 2), and 30 healthy controls (Group- 3).

Group - 1: Included 10 female and 20 male patients with mean age 46.9 ± 7.2 years, 22 patients had HCV infection, 8 patients had HBV, 9 patients had jaundice, 4 patients had hematemesis,3 patients had melena and non of the patients were alcoholic.

Group - 2: Included 12 female and 28 male patients with Included 10 female and 20 male patients with mean age 48.3 ± 4.4 years, 30 patients had HCV infection, 10 patients had HBV, 24 patients had jaundice, 26 patients had hematemesis,10 patients had melena, 35 patients had pitting edema of lower limbs and non of the patients were alcoholic.

Group - 3: Included 14 female and 16 male normal subjects with mean age 49.5 ± 4.5 years and normal parameters of all liver function tests. There was significantly lower serum albumin & PC level in group - 2 patients compared to group -1 (p< 0.001).On the other hand, there was higher total and direct bilirubin level and prolonged PT in patients with group - 2 compared to group - 1 (p< 0.001)

Mean LVEDD was significantly increased in group -1 (5.28 ± 0.51 mm) and in group - 2 (5.41 ± 0.58 mm) compared to group -3 (4.72±0.58 mm) and the difference was statistically significant (p< 0.05). Mean LVESD was 3.46±3.1 mm in group 1; 3.57 ± 2.2 mm in group 2 and 3.28 ± 2.5 mm in the control group and the difference was not statistically significant (p>0.05). Mean LV ejection fraction was 60 ± 4.56 % in group - 1; %, 61 ± 6.61% in group - 2 compared to 60.6 ± 1.9% in the control group and the difference was statistically insignificant (p> 0.05).LA diameter in end-systole was found to be larger in group - 1;4.46 ± 0.4 mm and in group 2, 4.72 ± 0.44 mm vs. 2.98 ±0.31 in the control group) (p< 0.001). RV size was found to be larger in group - 1; 3.03±0.57 mm and in group - 2; 3.66±0.37 mm compared to 1.95±0.13 mm in control group (p< 0.001). Mean PAP was found to be higher in patients of group 1; 25.38±6.94 mmHg and in patients of group 2, 39.29±7.1mmHg vs. 13.2±1.93 mmHg in the control group) (p< 0.001) as shown in table [1].

Mitral valve E/A ratio < 1 was found in 12 patients of group - 1, 30 patients of group - 2 compared to only one of the control group and the difference in E/A ratio was statistically highly significant (p < 0.001) as shown in table [2].

Tricuspid valve E/A ratio < 1 was found in 7 patients of group - 1, 35 patients of group - 2 compared to only two persons of the control group and the difference in E/A ratio was statistically highly significant (p < 0.001) as shown in table [2].

Univariate regression analysis revealed significant correlations between LV diastolic dysfunction and age (r=0.239, p <0.001), presence of ascites (r=0.155, p =0.002), RV enlargement (r=0.123, p =0.037) and pulmonary hypertension (r=0 0.190, p =0.005) as shown in table [3].

Univariate regression analysis revealed significant correlations between RV diastolic dysfunction and presence of ascites (r=0.154, p =0.004), right ventricular enlargement r=0.161, p =0.015) and pulmonary hypertension (r=0 0.211, p =0.001) as shown in table [3].

DISCUSSION

Liver cirrhosis is associated with a wide range of cardiovascular abnormalities including hyperdynamic circulation, cirrhotic cardiomyopathy, and pulmonary vascular abnormalities [6]." Cirrhotic cardiomyopathy is a clinical syndrome in patients with liver cirrhosis characterized by an abnormal and blunted response to physiologic, pathologic, or pharmacologic stress but normal to increased cardiac output and contractility at rest. As many as 50% of cirrhotic patients undergoing liver transplantation show signs of cardiac dysfunction, and 7% to 21% of deaths after orthotopic liver transplantation result from overt heart failure [7]." Strict diagnostic criteria for cirrhotic cardiomyopathy are lacking and the presence of this syndrome should be suspected in patients with worsening hemodynamic [8]."

Our study included 30 liver cirrhosis patients without ascites and 40 liver cirrhosis patients with ascites and fulfilling the criteria of liver cirrhosis diagnosed clinically by the stigmata of chronic liver disease and ultrasonographically by shrunken liver and coarse echo texture. We found that, patients with liver cirrhosis had higher LVEDD when compared to control group (P < 0.05) and there was no significant difference in the LV EF % in patients with liver cirrhosis when compared to control group (P > 0.05). Left ventricular EF has been reported to be normal in some studies [9,10]." increased in others [11,12,13]." and decreased only in one study of patients with cirrhosis and ascites [14].". A relative increase in the EF of patients with cirrhosis could be explained by the hyperdynamic circulation as a result of splanchnic vasodilatation.LV systolic function is usually normal at rest and systolic incompetence is most evident under

stress, whether physical or pharmacological, or when the extent of peripheral arterial vasodilatation demands an increased cardiac output as in the case of bacterial infections. Acute volume overload after insertion of a trans-jugular intra-hepatic porto-systemic shunt or after liver transplantation can also precipitate LV systolic dysfunction [6]." In our study, we found that patients with liver cirrhosis had higher LA diameter and RVEDD when compared to control group (P < 0.001).Our results came in agreement with Papastergiou et al., 2012 [15]. who found echocardiographic evidence of mild to moderate left atrium enlargement was found in 36% of the studied population. Significant correlation between left atrial size and intrapulmonary right to left shunt, characterizing hepatopulmonary syndrome was observed in a previous report [16]." Furthermore Valeriano et al., 2000 [17] " found that right and left atrium and right ventricular diameters were significantly enlarged in cirrhotic patients versus controls. These abnormal structural findings in cirrhotic patients seem to be an adaptation of cardiac hemodynamic to the changes in the peripheral circulation and thus the role of the rennin-angiotensin-aldosterone system (RAAS) and adrenergic hyperactivity has been considered. In addition Wong, 2009 [2] found an increased diameter of the left atrium and some degree of diastolic dysfunction in 39 pre-ascitic and ascitic patients and concluded that ascitic patients had a thicker left ventricular wall and a lower E/A ratio, indicating greater impedance to venous return than pre-ascitic cirrhotic patients.

In our study, we found significant abnormalities in both left and right ventricular diastolic function in patients with liver cirrhosis in the form of reversed E/A ratio with significant decrease in E wave average velocity of both mitral and tricuspid annulus with reversed E/A ratio. LV diastolic dysfunction was detected in 12 patients with liver cirrhosis without ascites and 30 patients with ascites and correlated with age (r=0.239, p <0.001), presence of ascites (r=0.155, p=0.002),right ventricular enlargement (r=0.123, p =0.037) and pulmonary hypertension (r=0 0.190, p =0.005).RV diastolic dysfunction was detected in 7 patients with liver cirrhosis without ascites and 35 patients with ascites and correlated with presence of ascites (r=0.154, p =0.004), right ventricular enlargement r=0.161, p =0.015) and pulmonary hypertension (r=0 0.211, p =0.001).

In our study, diastolic dysfunction appears to be more common in cirrhotic patients with ascites than those without ascites and that some degree of diastolic dysfunction is present in the majority of patients with cirrhosis. Right ventricle diastolic dysfunction may be due to the decrease in cardiac preload, increase in the afterload or right ventricular relaxation or other abnormalities in compliance [18]. Left ventricle Diastolic dysfunction is often linked to cardiac structure abnormalities including a combination of myocardial hypertrophy, fibrosis, and subendothelial edema [19]. The stiff and noncompliant ventricles cannot accommodate the venous return to the heart in early and middle-late diastole. Significant stimulus may not be required to precipitate diastolic dysfunction so that echocardiography may reveal abnormal diastolic function even at rest. Functional component cannot be also excluded, as improvements in diastolic dysfunction have been reported after paracentesis in patients with tense ascites and port systemic shunt insertion [20." Decrease in cardiomyocyte metabolism have been recently proposed in order to explain diastolic dysfunction and its reversibility after liver transplantation [15]."

Our results came in agreement with Salari et al., [21] who reported that diastolic dysfunction was significantly present in all cirrhotic patients but the severity was increased with the increased severity of the chronic liver disease. Møller et al., 2006 [11] showed that A wave velocities and deceleration times are much increased and the E/A-ratio is decreased in cirrhotic patients, especially in those with ascites. Pozzi et al., 1997 [14] found that in cirrhotic patients with tense ascites, the A wave velocity is markedly increased, the E/A ratio is markedly reduced, and the deceleration time is significantly prolonged. Furthermore, removal of the ascitic fluid by rapid total paracentesis was associated with reduction in A wave velocity and increases the E/A ratio to values similar to those of cirrhotic patients without ascites but still abnormal as compared with healthy controls.

Another important finding in our study was the significantly higher pulmonary artery pressure in liver cirrhosis patients (25.38±6.94 mmHg in patients without ascites, 39.29±7.1 mmHg in patients with ascites and 13.2±1.93 mmHg in controls, p<0.001).In fact, pulmonary vascular resistance was tended to decrease in cirrhotic patients. The mechanism of increased PAP is not fully understood, but previous studies suggested the increased levels of vasoactive substances in pulmonary circulation and the probable toxic effect of these substances on endothelial cells [18]." Some authors have suggested that microthrombi can migrate to pulmonary vascular bed along porto-systemic shunts and can cause increase in vascular resistance [22].".

LIMITATIONS OF THE STUDY

The number of the patients was not large enough to have more valuable correlation between the severity of the disease and the prevalence of cardiovascular abnormalities. more sophisticated echocardiographic tools (e.g., tissue Doppler imaging), which could have increased the reliability of our results, were not used. We cannot rule out the decreased cardiac reserve in stress conditions because we evaluated all of our patients in the resting position.

CONCLUSION

Liver cirrhosis is associated with echocardiographic changes in the form of enlarged right cardiac chambers, diastolic dysfunction and pulmonary hypertension and these changes are more evident in cirrhotic patients with ascites than those without ascites. Echocardiography should be part of the screening of patients with chronic liver disease, because patients with systolic and / or diastolic dysfunction and porto-pulmonary hypertension could be at higher morbidity and mortality risk.

“Compliance with Ethical Requirements

For Conflict of Interest statements

Mohamed Mashahit, Hany Younan, Maher El-Amir, A mal Mohamed, Hala Farawela and Alaa Abd El-Hamed declare that they have no conflict of interest.

All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008. Informed consent was obtained from all patients for being included in the study.

This study was approved by Fayoum University ethical committee

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Table 1: Echocardiographic findings in the study groups

Parameter	Group 1	Group 2	Group 3
IVS	0.85 ± 0.11	0.81±0.12	0.82±0.14
	P > 0.05	P > 0.05	
PWT	0.78 ± 0.09	0.79±0.10	0.77±0.12
	P > 0.05	P > 0.05	
LVEDD *(n: 3.7-5.6cm)*	5.31±0.51	5.40±0.58	4.52±0.58
	P < 0.05	P < 0.05	
LVESD *(n: 24-37mm)*	3.46±3.1	3.57±2.2	3.18±2.5
	P > 0.05	P > 0.05	
EF% *(n: 55- 75%)*	60±4.56	61±6.61	60.6±1.9
	P > 0.05	P > 0.05	
LA size *(n: 1.9- 4 cm)*	4.46±0.4	4.72±0.44	2.98±0.31
	P < 0.001	P < 0.001	
RVEDD *(n: 0.7- 5.6cm)*	3.03±0.57	3.66±0.37	1.95±0.13
	P < 0.05	P < 0.001	
PAP *(n: 12-28mmHg)*	25.38±6.94	39.29±7.1	13.2±1.93
	P < 0.05	P < 0.001	

IVS=inter ventricular septum thickness in diastole; PW= posterior wall thickness in diastole, LVEDD= left ventricular end diastolic diameter; LVESD= left ventricular end systolic diameter ;EF%,= ejection fraction; LA= left atrium diameter in end systole; RVEDD= right ventricular end diastolic diameter ;PAP= peak pulmonary artery pressure

Table 2: Prevalence of diastolic dysfunction among the study groups

		Group 1		Group 2		Group3	
E/A ratio		**No**	**%**	**No**	**%**	**No**	**%**
Mitral valve	Normal E/A ratio	18	60%	10	25%	29	96.7
(LVDD)	Reversed E/A ratio	12	40%	30	75%	1	3.3
Tricuspid valve	Normal E/A ratio	23	69 %	5	12.5 %	28	93.3%
(RVDD)	Reversed E/A ratio	7	21%	35	87.5%	2	6.7%

E= mitral valve early diastolic velocity; A=mitral valve late diastolic velocity.

Table 3 : Correlations of LV & RV diastolic dysfunction with different parameters in cirrhotic patients

	LV Diastolic dysfunction		***RV* Diastolic dysfunction**	
Variables	r	P. value	*r*	*P. value*
AgeAge	0.239	0.001	*0.062*	*0.17*
Jaundice	0.115	0.10	*0.078*	*0.11*
Hematemesis	0.035	0.58	*0.011*	*0.86*
Melena	0.064	0.32	*0.033*	*0.57*
Ascites	0.155	0.002	*0.154*	*0.004*
AST	0.081	0.16	*0.022*	*0.73*
ALT	0.043	0.51	*0.038*	*0.42*
Serum albumin	0.007	0.90	*0.009*	*0.89*
PT	0.064	0.32	*0.050*	*0.48*
LVEDD	0.142	0.042	*0.062*	*0.17*
LVESD	0.105	0.081	*0.078*	*0.11*
EF	0.068	0.24	*0.058*	*0.26*
LA Diameter	0.104	0.13	*0.089*	*0.15*
RVEDD	0.123	0.037	*0.161*	*0.015*
PAP	0.190	0.005	*0.211*	*0.001*

AST=Aspartate aminotransferase; ALT= Alanine aminotransferase; PT= prothrombin time; LVEDD= left ventricular end diastolic diameter; LVESD= left ventricular end systolic diameter ;EF%,= ejection fraction; LA= left atrium diameter in end systole; RVEDD= right ventricular end diastolic diameter ;PAP= peak pulmonary artery pressure

Table 4: Mitral & tricuspid valve diastolic inflow velocities in the study groups

		Group 1	**Group 2**	**Group 3**
mitral valve diastolic inflow parameters	E Wave	57.5 ± 1.2 P > 0.05	56.2 ± 1.1 P > 0.05	58.4 ± 2.1
	A Wave	60.3 ± 2.9 P < 0.05	62.8 ± 1.2 P < 0.05	51.8 ± 11.2
	E/A ratio	0.95± 0.6 P < 0.001	0.94 ± 0.8 P < 0.001	1.21 ± 0.42
tricuspid valve diastolic inflow parameters	E Wave	45.25±1.30 P < 0.001	40.11±2.70 P < 0.001	50.31±1.23
	A Wave	47.22±2.12 P = 0.02	50.66±2.27 P = 0.07	43.22±1.62
	E/A ratio	0.94±0.05 P < 0.001	0.83 ± 0.07 P < 0.001	1.25±0.05

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Table 5: Comparison between liver biochemical tests in the study groups

Parameter	**Group 1**	**Group 2**	**Group 3**
AST (n:up to 38mg/dl)	68.44±38.83 P < 0.001	97.08±47.83 P < 0.001	22.9±4.93
ALT (n:up to 40mg/dl)	38±20.08 P < 0.001	59.79±29.79 P < 0.001	21±5.3
ALP (n:40-180U/L)	185.25±93.77 P < 0.001	221.58±89.84 P < 0.001	116.3±20.69
Total protein(n:6.5-8.5g/dl)	6.73±0.49 P < 0.001	5.63±0.65 P < 0.001	7.78±0.64
Albumin (3.5-5gm/dl)	3.29±0.64 P < 0.001	2.13±0.47 P < 0.001	4.02±0.29
Total bilirubin(n: 0.2-1 mg/dl)	1.12±0.90 P < 0.001	3.06±1.29 P < 0.001	0.63±0.2
Direct bilirubin (n:0-0.2mg/dl)	0.31±0.14 P < 0.001	1.26±0.78 P < 0.001	0.06±0.03
PT (n:12sec)	15.34±2.23	29.72±10.47	12.4±0.38

	P < 0.001	**P < 0.001**	
PC (n: 80-100%)	59.21±12.34	45.67±11.56	93.4±5.25
	P < 0.001	**P < 0.001**	

AST=Aspartate aminotransferase; ALT= Alanine aminotransferase; PT= prothrombin time; PC= prothrombin concentration;

1	HCV	Hepatitis C VIRUS
2	QT	QT interval
3	b-adrenergic	Beta – adrenergic
4	AST	Aspartate aminotransferase
5	ALT	Alanine aminotransferase
6	HBV	Hepatitis B VIRUS
7	ECG	electrocardiogram
8	CAD	Coronary artery disease
9	LV	Left ventricle
10	LVEDD	**left ventricular end diastolic diameter**
11	LVESD	**left ventricular end systolic diameter**
12	IVS	**inter ventricular septum thickness in diastole**
13	PWT	**posterior wall thickness in diastole**
14	RVEDD	**right ventricular end diastolic diameter**
15	RVESD	**right ventricular end systolic diameter**
16	LA	**left atrium diameter in end systole**
17	EF	**ejection fraction;**
18	RV	**RIGHT VENTRICLE**
19	E	the peak early diastolic flow velocities
20	A	the peak of late flow velocities
21	E/A	Ratio of E/A
22	MR	Mitral regurgitation
23	TR	Tricuspid regurgitation
24	PAP	**peak pulmonary artery pressure**
25	SD	Standard deviation
26	PC	Prothrombin concentration
26	LVDD	Left ventricular diastolic dysfunction
27	RVDD	right ventricular diastolic dysfunction