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Electrocardiographic and Echocardiographic Abnormalities, and Cirrhotic Cardiomyopathy in Patients Hospitalized at the Campus University Hospital (Lomé)

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Abstract: Cirrhotic cardiomyopathy is a constellation of structural and functional cardiac abnormalities in patients with liver cirrhosis, apart from any underlying cardiac pathology, which potentially worsens the prognosis of these patients. The objectives of this study were to describe electrocardiographic and echocardiographic abnormalities in cirrhotic patients and estimate the prevalence of cirrhotic cardiomyopathy. This is a cross-sectional study with prospective data collection, carried out in hepato-gastroenterology department of the Campus University Teaching hospital of Lomé between July and December 2019, in adult patients hospitalized for liver cirrhosis. Of the 32 patients (men: 62.5%, mean age: 53.3 ± 13.5 years), the main causes of cirrhosis were viral hepatitis (40.6%) and alcoholic cirrhosis (25 %). Cirrhosis was classified Child-Pugh C in 62.5% of patients. The most common electrocardiographic abnormalities were prolonged QTc (59.4%), sinus tachycardia (31,2%), left atrial hypertrophy (25%) and left ventricular hypertrophy (12.5%). The main echocardiographic abnormalities were high cardiac output (68.8%) and left ventricular diastolic dysfunction (56.3%). The diagnostic criteria for cirrhotic cardiomyopathy were met in 37.5% of cases and it was more frequent in Child-Pugh C patients (66.7%) and in cirrhosis of alcoholic etiology (33.4%). Whatever the etiology of cirrhosis, electrocardiographic and echocardiographic abnormalities were common. The prevalence of cirrhotic cardiomyopathy was high, hence the need for systematic cardiological assessment in these patients.

Keywords: Liver Cirrhosis, Electrocardiographic and Echocardiographic Abnormalities, Cirrhotic Cardiomyopathy

1. Introduction

Cirrhosis which is the ultimate outcome of most chronic liver diseases can be complicated by a specific cardiac condition called cirrhotic cardiomyopathy. Described by Lee et al. in 1989, cirrhotic cardiomyopathy corresponds to a constellation of structural and functional cardiac abnormalities found in cirrhotic patients, apart from any underlying cardiac condition [1, 2].

Indeed, cirrhosis is associated with cardiocirculatory abnormalities including increased cardiac output [3], decreased blood pressure and lowered vascular resistance [4, 5] likely to lead to impaired systolic and diastolic function [6–8]. Since, numerous studies have confirmed that the reduction of myocardial contractility under stress is specific to all forms of cirrhosis and that the degree of impairment of cardiac function is similar whatever the etiology of the liver disease [9].

However, the prevalence of cirrhotic cardiomyopathy remains unknown, especially because the disease is usually latent and only manifests when the patient is subjected to stress. It is estimated that half of cirrhotic patients undergoing liver transplantation show signs of cardiac dysfunction, with a postoperative mortality rate from heart failure varying from 7 to 21% [10].

In Western countries since the late 1980s, numerous studies have been conducted on cardiac damage during cirrhosis, particularly from alcoholic origin [11–15]. In sub-Saharan Africa, studies in this field are rare [16]. To date, no study exists on this subject in Togo, hence the interest of the present study whose objectives were to describe the electrocardiographic and echocardiographic abnormalities in patients with cirrhosis, and to estimate the prevalence of the cirrhotic cardiomyopathy.

2. Materials and Methods

2.1. Type and Period of Study

This was a cross-sectional and descriptive study carried out from July to December 2019, on patients hospitalized for liver cirrhosis in the hepato-gastroenterology and cardiology departments of the Lomé Campus University Teaching Hospital.

2.2. Inclusion Criteria

Inclusion has been done consecutively in patients aged 18 years or more, with a diagnosis of liver cirrhosis on the basis of clinical, morphological, biological and endoscopic examinations.

2.3. Non-Inclusion Criteria

Patients with precarious hemodynamic state were excluded from this study.

2.4. Data Collection

Following data were collected prospectively:

1) sociodemographic and clinical data: gender, age, cardiovascular risk factors, etiology and duration of progression of cirrhosis, severity of cirrhosis according to the Child-Pugh classification,
2) paraclinical data: electrocardiography, echocardiography and hemoglobin level.

2.5. Definitions

Patients were considered to have anemia for hemoglobin level < 12 $g.dl^{-1}$. Anemia was considered mild for hemoglobin level between 10 and 11 $g.dl^{-1}$, moderate if the hemoglobin level was between 7 and 9 $g.dl^{-1}$ and severe for hemoglobin level < 7 g/dl. dl [17].

Rest electrocardiogram (ECG) was performed in all patients: left ventricular hypertrophy was defined by Sokolow-Lyon index (SVl + RV5 or V6) >35 mm or Cornell index (RaVL + SV3) ≥ 20 mm in women and ≥ 28 mm in men. Left atrial hypertrophy was considered if duration of P wave ≥120 ms in DII, and/or two-phase P wave with predominantly negativity in V1. Sinus tachycardia was characterized by heart rate of sinus origin >100/minute. The corrected QT interval (QTc, ms) was determined according to the Bazett formula: QTc = measured QT/√RR. The QTc was considered prolonged for a value >440 ms [18, 19].

Echocardiography was performed at rest in all patients. Quantification of cardiac chamber size was done according to the guidelines of the American Society of Echocardiography [20]. Left atrial dilatation was considered for a volume >34 ml/m^2 and the right atrium dilatation for a surface area > 18 cm^2 in apical 4-chamber view. Left ventricle was dilated if its indexed end-diastolic diameter was >31 $mm.m^{-2}$ in men or >32 $mm.m^{-2}$ in women in long-axis parasternal view. Right ventricle was dilated if its basal diameter was > 42 mm and median diameter >35 mm in apical 4-chamber view. Left ventricular mass (LVM, g) was calculated using the cube formula: $LVM = 0.8.[1.04.[(IVS + LVID + PWT)^3 - LVID^3] + 0.6$ where IVS is interventricular septum, LVID is left ventricular internal diameter, and PWT is inferolateral wall thickness. Left ventricular mass index (LVMi, $g.m^{-2}$) was calculated with the following formula: LVMi = LVM/BSA where BSA is body surface area. LVMi was high if > 95 $g.m^{-2}$ in women and > 115 $g.m^{-2}$ in men. Cardiac output ($L.min^{-1}$) was calculated using the following formula: cardiac output = VTI x HR x LVOT area, where VTI is the aortic velocity-time integral, HR the heart rate, and LVOT the left ventricular outflow track. LVOT area was calculated using the formula LVOT area = π x r^2 (r = radius) derived from the measure of the valve annulus diameter; cardiac output was high if > 6 l/min [20]. Left ventricular systolic function was assessed in parasternal long-axis view and was impaired if the left ventricular ejection fraction (LVEF) was <55% [21]. Left ventricular diastolic function was evaluated after performing pulsed Doppler of the mitral flow and tissue Doppler at the mitral annulus and by combining following parameters: E/A, mean E/E', septal and lateral E', maximum velocity of the tricuspid regurgitation flow and left atrial volume; left ventricular diastolic dysfunction was classified into grades I, II and III according to the guidelines of the American Society of Echocardiography [22].

The diagnosis of cirrhotic cardiomyopathy was made based on the criteria established at the World Congress of Gastroenterology in Montreal in 2005 [23]:

1) impaired resting systolic function (LVEF < 55%) or left ventricular diastolic dysfunction with or without the following supporting criteria: prolongation of the QTc interval, left atrial dilatation and increased left ventricular mass,
2) in the absence of clinical signs of heart failure at rest, severe anemia (hemoglobin level <7 g/dl) and cardiovascular history (hypertension, diabetes, known heart disease) which could explain the myocardial dysfunction.

2.6. Data Analysis

Microsoft Excel version 2021 was used to enter data in a case report form. Categorical variables are expressed as counts and percentages and continuous variables as mean ± standard deviation. Data were analyzed using EPI INFO software version 7.2.1.0.

3. Results

3.1. Sociodemographic and Clinical Characteristics of the Study Population

Overall, 32 patients were included with 12 women (37.5%) and 20 men (62.5%), giving a sex ratio of 1.66. The mean age was 53.3 ± 13.5 years (extremes: 29 and 82 years). Cirrhosis was diagnosed for less than 1 year in 19 patients (59.4%). Cirrhosis was of alcoholic etiology in 8 (25%) patients, and due to hepatitis B or C in 13 (40.6%) patients. 20 (62.5%) patients were in Child-Pugh class C. Anemia was found in 25 (78.1%) patients (table 1).

Table 1. *Epidemiological and clinical characteristics of the study population.*

	N (%) or m±SD
Mean age (years)	53.3 ± 13.5
Cardiovascular risk factors	
Diabetes	2 (6.35)
Hypertension	2 (6.35)
Duration of progression of the cirrhosis	
< 1 year	19 (59.4)
1 to 3 year	13 (40.6)
Aetiology of cirrhosis	
Alcoholic cirrhosis	8 (25)
Hepatitis B	7 (21.9)
Hepatitis C	6 (18.8)
Mixed aetiology (alcohol and hepatitis B or C)	6 (18.8)
Unknown	5 (15.5)
Child-Pugh classes	
A	1 (3.1)
B	11 (34.4)
C	20 (62.5)
Anaemia	25 (78.1)
Severe anaemia	3 (9.3)
Moderate anaemia	16 (50)
Mild anaemia	6 (18.7)

n = counts, m = mean, SD = standard deviation

3.2. Electrocardiographic Data

Electrocardiography was normal in 5 (15.6%) patients. The main ECG abnormalities were prolonged QTc (59.4%), sinus tachycardia (32%), left atrial hypertrophy (25%) and low peripheral voltage (25%). Prolonged QTc was more frequent as the cirrhosis severity score was higher; thus 73.7% (14/19) of patients with a prolonged QTc were in Child-Pugh class C and 26.3% (5/19) were in class B (table 2).

Table 2. *ECG abnormalities in cirrhosis patients.*

	Counts	Percentages
Sinus tachycardia	10	31.2
Left atrial hypertrophy	8	25
Left ventricular hypertrophy	4	12.5
Left anterior fascicular block	4	12.5
Negative T wave	4	12.5
Low peripheral voltage	8	25
Prolonged QTc	19	59.4
Prolonged QTc with Child-Pugh class B	5	15.6
Prolonged QTc with Child-Pugh class C	14	43.8

QTc = corrected QT interval

3.3. Echocardiographic Data

The mean cardiac output was 8.2 ± 3.3 l/min with extremes of 3.2 l/min and 15.2 l/min. High cardiac output was found in 22 (68.8%) patients, 15/22 (68.2%) of whom were in Child-Pugh class C. LVMi was high in 9 patients (28.1%). Impaired LVEF was found in 5 patients (15.6%) and left ventricular diastolic dysfunction in 18 patients (56.3%) (table 3).

Table 3. *Echocardiographic abnormalities.*

	Counts	Percentages
High left ventricular mass index	9	28,1
Left atrial dilatation	8	25
Right atrial dilatation	1	3,1
Left ventricular dilatation	5	15,6
Right ventricular dilatation	5	15,6
Impaired left ventricular ejection fraction	5	15,6
Left ventricular diastolic dysfunction	18	56,3
Grade I	17	53,1
Grade II	1	3,1
High cardiac output	22	68,8
Child-Pugh class B	7	21,9
Child-Pugh class C	15	46,9

3.4. Cirrhotic Cardiomyopathy

Overall, 12/32 patients had cirrhotic cardiomyopathy then a prevalence of 37.5%. Mean age of these patients was 55 ± 13.4 years; 8/12 patients (66.7%) with cirrhotic cardiomyopathy were in Child-Pugh class C; 4 patients (33.4%) had alcoholic cirrhosis and 3 (25%) had hepatitis B or C. Cardiovascular investigations showed that 6 patients (50%) with cirrhotic cardiomyopathy had prolonged QTc; left ventricular diastolic dysfunction was found in 9 patients (75%) and 3 patients had impaired LVEF. Eleven patients (91.7%) had high cardiac output (table 4).

Table 4. *Characteristics of patients with cirrhotic cardiomyopathy.*

	Counts (n=12)	Percentages
Female	6	50
Child-Pugh classes		
B	4	33,3
C	8	66,7
Etiology of cirrhosis		
Alcoholic cirrhosis	4	33,3
Hepatitis B and C	2	16,7
Mixed cirrhosis (hepatitis + alcoholic)	2	16,7
Unknown	1	8,3
ECG and echocardiographic abnormalities		
Prolonged QTc	6	50
Left ventricular diastolic dysfunction	9	75

	Counts (n=12)	Percentages
Impaired left ventricular ejection fraction	3	25
High cardiac output	11	91,7

ECG = electrocardiographic

4. Discussion

In this sample of cirrhotic patients, the main electrocardiographic abnormalities were prolonged QTc (59.4%), sinus tachycardia (31.2%), left atrial hypertrophy (25%) and left ventricular hypertrophy (12.5%). Table 5 summarizes the comparison of ECG results between our study and other authors. Indeed, prolonged QTc interval is found in approximately 30 to 60% of cirrhotic patients [9]; it results from abnormal repolarization of the myocardium and is associated with an increased risk of torsade de pointes. Several studies have demonstrated a significant correlation between the severity of liver disease and the duration of the QTc interval [24].

Table 5. *Comparison of the main ECG findings in our study with those of other studies.*

	Our study %	Gueye *et al.* [16] %	Tint *et al.* [25] %	Møller *et al.*[26] %
Tachycardia	32	28.3	38.6	NA
LAH	25	35	NA	38
LVH	12,5	45	NA	58
Prolonged QTc	59,4	40	33	45

LAH: left atrial hypertrophy; LVH: left ventricular hypertrophy; NA: non available

The left ventricle of cirrhotic patients is often moderately hypertrophied in echocardiography [13, 26, 27]. LVMi was high in 28.1% of our patients. Other significant morphological abnormalities in our patients were left atrial dilatation and left ventricular dilatation in 25% and 15.6% respectively. Tint *et al.* reported higher rates with left ventricular dilatation in 70% of cases and left atrial dilatation in 44% [25]. These high rates of left heart chambers dilatation could in part be linked to the chronic anemia frequently encountered during cirrhosis. Left ventricular systolic function, expressed by impaired LVEF, was reduced in 15.6% of our patients. Gueye *et al.* found a lower rate (3.3%) [16]. In fact, both of these results are significantly lower than those of Mandell *et al.* and Wong *et al.* who reported impaired LVEF in 36.7% and 43.6% of cases, respectively [15, 28]. This difference could be explained by the fact that LVEF in cirrhotic patients is normal or supranormal in the basal state. Unlike our study and that of Gueye where it was exclusively a question of resting echocardiography, Mandell and Wong performed stress ultrasounds, thus explaining the relatively high prevalence of impaired LVEF [15, 28]. Stress or dobutamine ultrasound can be useful in detecting an impaired LVEF during cirrhotic cardiomyopathy because left ventricular systolic dysfunction in cirrhotic patients is usually latent [29]. Stress echocardiography reveals chronotropic and inotropic incompetence because the heart is unable to support demand by ensuring adequate cardiac output and mean arterial pressure; the lack of reactivity of the left ventricle is correlated with the severity of the cirrhosis [30]. Thus, a study of the effect of physical exercise in cirrhotic patients, noted an increase in LVEF to 14%, compared to 66% in healthy controls, while the resting values of the 2 groups were similar [15]. Systolic dysfunction can also be demonstrated after an infusion of terlipressin [31], angiotensin II or after liver transplantation [32]. Kanzankov *et al.* reported physiological arguments suggesting that systolic dysfunction can be observed even at rest [33].

There was left ventricular diastolic dysfunction in 56.3% of our patients. The prevalence of left ventricular diastolic dysfunction varies between 45 and 66% and is higher in patients in decompensation stage [34]. Gueye *et al.* found a relatively low rate of 20%, probably because they included more compensated cirrhotic patients [16]. Left ventricular diastolic dysfunction is due to increased rigidity and left ventricular hypertrophy, leading to a decrease in ventricular compliance, thus affecting its filling. The ventricular rigidity is a result of several abnormalities in histopathology, such as cardiac muscular hypertrophy, fibrosis and subendothelial edema. Diastolic function is assessed in echocardiography by measuring the E/A ratio, expressing the blood flow velocity ratio during the early phase of diastole (E wave) and the late phase (A wave). Normally greater than one, this ratio is reduced in cirrhotic patients [35] thus indicating a lack of diastolic relaxation and consequently an alteration of ventricular filling. This ratio is also significantly lower in patients with tension ascites notably in decompensated cirrhosis [36].

High cardiac output was observed in 68.8% of our patients, and it was more frequent in the most severe Child-Pugh classes, B (31.8%) and C (68.2%). Gueye *et al.* reported high cardiac output in 43.3% of patients with hepatitis B cirrhosis [16] and Cohen-Solal *et al.* and Møller *et al.* reported high cardiac output during alcoholic cirrhosis in 48% and 44% of patients, respectively [37, 38]. High cardiac output is common in these patients whatever the etiology of cirrhosis and seems to be a function of its severity.

Application of the criteria of the World Gastroenterology Congress in Montreal allows to estimate the prevalence of cirrhotic cardiomyopathy in our study at 37.5%. The prevalence of cirrhotic cardiomyopathy is still unknown [39], but recently Dash *et al.* observed a prevalence of 39,6% using electrocardiographic and echocardiographic criteria [40]. Enache *et al.* [41] reported an incidence of 23.4% based on rest echocardiographic criteria only including systolic or diastolic dysfunction at rest in the absence of any other clear cause for cardiac impairment. Many diagnostic criteria have been proposed based on association of ECG abnormalities and/or rest and/or stress echocardiographic criteria [42, 43]. Some authors recently suggested to include brain natriuretic peptide plasma levels in the definition of cirrhotic cardiomyopathy [44-46]. The prevalence of cirrhotic cardiomyopathy is high in the present study in which we used

a combination of ECG abnormalities and rest echocardiography; that suggest to proceed systematic cardiovascular screening to improve the prognosis of these patients.

5. Limitations

The sample size is small but it is an initial study in our area. Due to the limits of our technical platform and the financial limitation of patients, it was not possible to study certain parameters such as myocardial biomarkers, stress or dobutamine echocardiography, scintigraphy and cardiac MRI, which allow more refined screening of cirrhotic cardiomyopathy.

6. Conclusion

Whatever the etiology of cirrhosis, electrocardiographic and echocardiographic abnormalities were common. The main electrocardiographic abnormalities were prolonged QTc, sinus tachycardia, left atrial hypertrophy and left ventricular hypertrophy; the main echocardiographic abnormalities were high cardiac output and left ventricular diastolic dysfunction. The prevalence of cirrhotic cardiomyopathy was high. This raises the need for systematic screening of cardiac abnormalities with the aim of adequate management in African cirrhotic patients.

Declarations

Ethical Considerations and Consent

Ethical approval was received from the institutional committee of the Faculty of Health Sciences of the University of Lomé (Togo). We also obtained administrative authorization from the general management of the CHU Campus of Lomé. Oral consent was obtained from each patient; and for those whose consciousness was somewhat impaired by hepatic encephalopathy, consent was obtained from the spouse or close relative. Data confidentiality was guaranteed.

Availability of Data and Material

The datasets generated during the current study are available from the corresponding author on request.

Conflicts of Interest

The authors declare that they have no competing interests.

Authors Contributions

KY designed the study, write the protocol and corrected the manuscript,

TT, LALP managed analysis and discussion and corrected the manuscript,

DAP, TC managed the literature searches,

PS, KWD, BS, BA and DF managed data collection.

References

[1] Lee SS. Cardiac abnormalities in liver cirrhosis. West J Med. 1989; 151 (5): 530.

[2] Netgen. La cardiomyopathie cirrhotique. [Internet]. Rev Med Suisse 2009; 5: 1725-31.

[3] Moller S, Henriksen JH. Cardiovascular complications of cirrhosis. Postgrad Med J 2009; 85: 44-54.

[4] Abelmann WH. Hyperdynamic circulation in cirrhosis: A historical perspective. Hepatology 1994; 20 (5): 1356-8.

[5] Liu H, Gaskari SA, Lee SS. Cardiac and vascular changes in cirrhosis: Pathogenic mechanisms. World J Gastroenterol 2006; 12: 837-42.

[6] Regan TJ, Levinson GE, Oldewurtel HA, Frank MJ, Weisse AB, Moschos CB: Ventricular function in noncardiacs with alcoholic fatty liver: role of ethanol in the production of cardiomyopathy. J Clin Invest 1969, 48: 397-407.

[7] Gould L, Shariff M, Zahir M, Di Lieto M. Cardiac hemodynamics in alcoholic patients with chronic liver disease and a presystolic gallop. J Clin Invest. mai 1969; 48 (5): 860-8.

[8] Limas CJ, Guiha NH, Lekagul O, Cohn JN. Impaired left ventricular function in alcoholic cirrhosis with ascites. Ineffectiveness of ouabain. Circulation. avr 1974; 49 (4): 754-60.

[9] Bayley TJ, Segel N, Bishop JM. The circulatory changes in patients with cirrhosis at rest and during exercise. Clin Sci 1964; 26: 227-35.

[10] Zard EM, Abbate A, Zardi DM, Dobrina A, Margiotta D, Van Tassell BW et al.— Cirrhotic Cardiomyopathy. J Am Coll Cardiol, 2010, 56, 539-49.

[11] Grose RD, Nolan J, Dillon JF, Errington M, Hannan WJ, Bouchier IA, et al. Exercise-induced left ventricular dysfunction in alcoholic and non-alcoholic cirrhosis. J Hepatol. mars 1995; 22 (3): 326-32.

[12] Finucci G, Desideri A, Sacerdoti D, Bolognesi M, Merkel C, Angeli P, et al. Left ventricular diastolic function in liver cirrhosis. Scand J Gastroenterol. mars 1996; 31 (3): 279-84.

[13] Pozzi M, Carugo S, Boari G, Pecci V, de Ceglia S, Maggiolini S, et al. Evidence of functional and structural cardiac abnormalities in cirrhotic patients with and without ascites. Hepatol Baltim Md. nov 1997; 26 (5): 1131-7.

[14] Wong F, Liu P, Lilly L, Bomzon A, Blendis L. Role of cardiac structural and functional abnormalities in the pathogenesis of hyperdynamic circulation and renal sodium retention in cirrhosis. Clin Sci Lond Engl 1979. sept 1999; 97 (3): 259-67.

[15] Wong F, Girgrah N, Graba J, Allidina Y, Liu P, Blendis L. The cardiac response to exercise in cirrhosis. Gut. août 2001; 49 (2): 268-75.

[16] Guèye MN, Louise BM, Malick B, Salamata D, Aïssé TM, Ambdil H, et al. Anomalies électrocardiographiques et échocardiographiques au cours de la cirrhose virale b: à propos de 60 cas au service d'hepato-gastroenterologie de l'Hopital Aristide Le Dantec de Dakar (HALD). Pan Afr Med J 25 juin 2018; 30: 169.

[17] Ponikowski P, Voors AA, Anker SD, Bueno H, Cleland JGF, Coats AJS, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. Eur Heart J.

[18] Luo S, Michler K, Johnston P, Macfarlane PW: A comparison of commonly used QT correction formulae: The effect of heart rate on the QTc of normal ECGs. J Electrocardiol, 2004; 37 (Suppl.): 81–90.

[19] Główczyńska R, Galas M, Ołdakowska-Jedynak U, Peller M, Tomaniak M, Raszeja-Wyszomirska J, et al. Pretransplant QT Interval: The Relationship with Severity and Etiology of Liver Disease and Prognostic Value After Liver Transplantation. In: Annals of transplantation. 2018.

[20] Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for Cardiac Chamber Quantification by Echocardiography in Adults: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc.

[21] Fouad Y, Yehia R. Hepato-cardiac disorders. World J Hepatol. 2014; 6: 41-54.

[22] Nagueh SF, Smiseth OA, Appleton CP, Byrd BF, Dokainish H, Edvardsen T, et al. Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 2016; 29: 277-314. Echocardiogr 2015; 28 (1): 1-39. e14.

[23] Fedorak, Richard & Sherman, Philip. World Congress of Gastroenterology Canadian Bid 2005. Canadian Journal of Gastroenterology. 14. 838-840. 10. 1155/2000/284742.

[24] Genovesi S, Prata Pizzala DM, Pozzi M. QT interval prolongation and decreased heart rate variability in cirrhotic patients: Relevance of hepatic venous pressure gradient and serum calcium. Clinical Science 2009: 116: 851-9.

[25] Tint D, Radoi M, Coman F, Zayarache C, Rosca E. Les atteintes cardiaques dans la cirrhose hépatique-Corré1ations avec l'étiologie. Rev Med Interne. 2002; 23 (Suppl5): 523-713.

[26] Møller S, Henriksen JH. Cirrhotic cardiomyopathy: a pathophysiological review of circulatory dysfunction in liver disease. Heart Br Card Soc. 2002; 87 (1): 9-15.

[27] Torregrosa M, Aguadé S, Dos L, Segura R, Gónzalez A, Evangelista A, et al. Cardiac alterations in cirrhosis: reversibility after liver transplantation. J Hepatol. 2005; 42 (1): 68-74.

[28] Mandell MS, Tsou MY. Cardiovascular dysfunction in patients with endstage liver disease. J Chin Med Assoc 2008; 71: 331–5.

[29] Kim MY, Baik SK, Won CS. Dobutamine stress echocardiography for evaluating cirrhotic cardiomyopathy in liver cirrhosis. Korean J Hepatol. 2010; 16 (4): 376-382.

[30] Baik SK, Fouad TR, Lee SS. Cirrhotic cardiomyopathy. Orphanet J Rare Dis. 2007; 2: 15.

[31] Krag A, Bendtsen F, Mortensen C, Henriksen JH, Moller S. Effects of a single terlipressin administration on cardiac function and perfusion in cirrhosis. Eur J Gastroenterol Hepatol 2010; 22 (9): 1085-92.

[32] Fouad TR, Abdel-Razek WM, Burak KW, Bain VG, Lee SS. Prediction of cardiac complications after liver transplantation. Transplantation 2009; 87 (5): 763-70.

[33] Kazankov K, Holland-Fischer P, Andersen NH et al. Resting myocardial dysfunction in cirrhosis quantified by tissue Doppler imaging. Liver Int 2011; 31 (4): 534-40.

[34] Wong F, Villamil A, Merli M et al. Prevalence of diastolic dysfunction in cirrhosis and its clinical significance. Hepatology 2011; 54 (Suppl.1): Abstract 229.

[35] Friedman SL. Liver fibrosis — from bench to bedside. J Hepatol 2003; 38 (Suppl 1): S38-53.

[36] Alqahtani SA, Fouad TR, Lee SS. Cirrhotic cardiomyopathy. Semin Liver Dis 2008; 28: 59-69.

[37] Møller S, Henriksen JH. Cardiovascular complications of cirrhosis. Gut 2008; 57 (2): 268-78.

[38] Cohen-Solal A, Beauvais F, Seghatol F, Durand F. Problèmes cardiaques du cirrhotique. Hépato-Gastro. 2003; 10 (6): 435-438.

[39] Izzy MJ, Vanwagner LB. Current concepts of Cirrhotic Cardiomyopathy. Clin Liver Dis. 2021; 25 (2): 471–81.

[40] Dash SC, Rajesh B, Behera SK, Sundaray NK, Patil P. Is Cirrhotic Cardiomyopathy Related to Cirrhosis Severity? Rambam Maimonides Med J 2023; 14 (1): e0001.

[41] Enache I, Oswald-Mammosser M, Woehl- Jaegle ML. Cirrhotic cardiomyopathy and hepatopulmonary syndrome: prevalence and prognosis in a series of patients. Respir Med 2013; 107 (7): 1030-6.

[42] Milani A, Zaccaria R, Bombardieri G, Gasbarrini A, Pola P. Cirrhotic cardiomyopathy. Dig Liver Dis 2007; 39: 507e15.

[43] Shahvaran SA, Menyhart O, Csedrik L, Patai AV. Diagnosis and Prevalence of Cirrhotic Cardiomyopathy: A Systematic Review and Meta-analysis. Curr Probl Cardiol 2021; 46: 100821.

[44] Saner FH, Neumann T, Canbay A, et al. High brain-natriuretic peptide level predicts cirrhotic cardiomyopathy in liver transplant patients. Transpl Int 2011; 24: 425e32.

[45] Dourakis SP, Geladari E, Geladari C, Vallianou N. Cirrhotic Cardiomyopathy: The Interplay Between Liver and Cardiac Muscle. How Does the Cardiovascular System React When the Liver is Diseased? Current Cardiology Reviews. 2021; 17, 78-84.

[46] Izzy M, VanWagner LB, Lin G, Altieri M, Findlay JY, Oh JK, Watt KD, Lee SS. The Cirrhotic Cardiomyopathy Consortium. Redefining Cirrhotic Cardiomyopathy for the Modern Era. Hepatology. 2020; 71 (1): 334–45.