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Research Article

Electrocardiographic T-Wave Changes in Patients with Liver Cirrhosis: A Marker of Decompensation

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Abstract

Background: Cirrhotic cardiomyopathy is an underrecognized complication that affects nearly half of patients with liver cirrhosis. Electrocardiographic T-wave changes and/or diastolic dysfunction may serve as an early sign of cardiac involvement. ***Objectives***: To investigate the association between electrocardiographic and echocardiographic changes and the severity of liver cirrhosis, as well as estimated short-term mortality. ***Methods***: This was an observational cross-sectional study conducted at Baghdad Teaching Hospital. Sixty patients with liver cirrhosis due to hepatitis B or hepatitis C viruses were enrolled in the study. Patients were grouped into three categories based on their Child-Pugh score and categorized into two groups based on the Model for End-stage Liver Disease (MELD-Na) score. We assess T-wave alterations in the ECG, together with systolic and diastolic dysfunction, to determine their correlation with the severity of liver cirrhosis and the likelihood of mortality within the subsequent months. ***Results***: The average age of patients was 54.06 years. Seventeen patients had compensated liver cirrhosis, while 43 had decompensated disease (20 with category B and 23 with category C). There is a significant prolongation of QTc, prolongation of Tpe, an increase in the Tpe/QTc ratio, a reduction in left ventricle ejection fraction, and an increase in the percentage of diastolic dysfunction among patients with decompensated liver cirrhosis (Child-Pugh class B and C). ***Conclusions***: Cirrhotic cardiomyopathy is related to the decompensation of liver cirrhosis. T-wave changes can be used as a marker of liver decompensation.

Keywords: Child-Pugh score, Cirrhotic cardiomyopathy, Diastolic dysfunction, Electrocardiographic changes, Liver cirrhosis.

تغيرات الموجة التائية الكهربائية للقلب في المرضى الذين يعانون من تليف الكبد: علامة على عدم التعويض

الخلاصة

الخلفية: اعتلال عضلة القلب عند تليف الكبد هو من المضاعفات غير المعترف بها وتصيب ما يقرب من نصف مرضى تليف الكبد. قد تكون تغيرات الموجة T الكهربائية للقلب بالإضافة إلى الخلل الوظيفي الانبساطي بمثابة علامة مبكرة على إصابة القلب. **الأهداف**: للتحقيق في العلاقة بين التغيرات في تخطيط كهربية القلب وتخطيط صدى القلب وشدة تليف الكبد، بالإضافة إلى تقدير الوفيات قصيرة المدى. **الطرائق**: كانت هذه دراسة مقطعية قائمة على الملاحظة أجريت في مستشفى بغداد التعليمي. تم تسجيل ستين مريضا يعانون من تليف الكبد بسبب فيروس التهاب الكبد B أو التهاب الكبد C في الدراسة. تم توزيع المرضى الى ثلاث فئات بناء على درجة Child-Pugh الخاصة بهم وتم تصنيفهم إلى مجموعتين بناء على درجة نموذج أمراض الكبد في المرحلة النهائية (MELD-Na). تم تقييم تغيرات الموجة التائية في مخطط كهربية القلب، جنبا إلى جنب مع الخلل الوظيفي الانقباضي والانبساطي، لتحديد ارتباطها بشدة تليف الكبد واحتمالية الوفاة خلال الأشهر اللاحقة. **النتائج**: بلغ متوسط عمر المرضى 54.06 سنة. وعوض سبعة عشر مريضا عن تليف الكبد، في حين كان 43 مريضا بدون تعويض (20 من الفئة ب و23 من الفئة ج). هناك إطالة كبيرة ل QTc، وإطالة TPE، وزيادة في نسبة Tpe / QTc، وانخفاض في جزء طرد البطين الأيسر، وزيادة في النسبة المئوية للخلل الوظيفي الانبساطي بين المرضى الذين يعانون من تليف الكبد اللاتعويضي (Child-Pugh class B و C). **الاستنتاجات**: يرتبط اعتلال عضلة القلب عند تليف الكبد بتعويض تليف الكبد. يمكن استخدام تغيرات الموجة التائية كعلامة على عدم تعويض الكبد.

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INTRODUCTION

Cirrhotic cardiomyopathy (CCM) is a crucial complication observed in individuals suffering from liver cirrhosis. It features both systolic and diastolic dysfunction, along with structural alterations like enlargement of chambers, occurring without any previous heart disease. The concept of "CCM" was initially coined in 1989 [1]. This condition impacts nearly 50% of individuals with cirrhosis, irrespective of its cause. Risk factors for developing cardiac disease also include alcohol use disorder and hemochromatosis. Since CCM presents itself during times of stress, it is often overlooked and lacks an established standardized treatment protocol. Notable improvement, or even complete reversal, of CCM has been observed in patients following liver transplantation [2]. Cirrhosis involves damage to the liver's structure, including nodular regeneration and widespread fibrosis, which ultimately causes liver dysfunction and increased portal pressure.

Liver dysfunction predisposes to a decrease in β-adrenergic receptor density, a higher cholesterol-to-phospholipid ratio in the cardiomyocyte plasma membrane, and the development of abnormal contractile filaments. Increased pressure in the portal vein results in congestion of intestinal blood vessels, leading to bacterial translocation and endotoxemia. Lipopolysaccharides cause inflammatory cytokines to be released, which raises the levels of nitric oxide and carbon monoxide and makes it harder for the heart to contract [3]. The cardiovascular effects of portal hypertension cause hyperdynamic circulation, characterized by high cardiac output and low systemic vascular resistance [4]. Regardless of the cause of decompensated liver disease, CCM is associated with both portal hypertension and cirrhosis; however, certain conditions, such as alcohol use, nonalcoholic steatohepatitis (NASH), and iron overload, may further affect heart function [5]. Patients with cirrhosis show increased cholesterol levels in cell membranes. This results in a reduction of L-type calcium and potassium channels, an increase in the duration of action potentials, and a prolonged QT interval. Furthermore, the dysregulation of Na/Ca channels leads to a significant increase in calcium influx into cells, triggering apoptosis in cardiomyocytes. The combination of extended action potentials and hindered myocyte relaxation results in eccentric left ventricular hypertrophy and diastolic dysfunction. Ultimately, systolic dysfunction arises due to compromised energy metabolism and diminished myocardial reserve [6]. The Cirrhotic Cardiomyopathy Consortium updated its definition of CCM in 2019, emphasizing the importance of diastolic dysfunction [7]. Electrocardiography (ECG) changes, Magnetic Resonance Imaging (MRI) features, and biomarkers are thought to provide additional supporting data for the diagnosis. At the same time, the current proposed criteria include evaluation of systolic dysfunction (a left ventricle ejection fraction of less than 50%) and multiple indicators of diastolic dysfunction (low septal e' velocity, high E/e' ratio, high indexed volume of left atrium, and high velocity of tricuspid regurgitation) [4,7]. The initial sign of CCM is usually evident on an electrocardiogram (ECG). A prolonged QT interval is the ECG pattern most frequently seen in CCM [8]. It has been reported to occur in about 30 to 70% of patients with liver cirrhosis [9]. The T-peak to T-end interval (Tpe) and the Tpe/QTc ratio are two recent ECG indicators for arrhythmogenesis, reflecting abnormalities in ventricular repolarization [10]. The majority of individuals with moderate to advanced cirrhosis (specifically, Child-Pugh Class B or C) will exhibit at least one characteristic of CCM, such as QT prolongation or diastolic dysfunction [11]. One of the terminal liver disorders is decompensated cirrhosis, which frequently has several side effects, such as hepatic encephalopathy (HE), ascites, infection, and gastrointestinal variceal hemorrhage [12]. The Child-Pugh (CTP) score, the model for end-stage liver disease (MELD) score, and several modified MELD scoring systems have all been demonstrated to be beneficial in earlier research. Serum bilirubin, albumin, prothrombin time, hepatic encephalopathy, and ascites are all calculated to determine the Child-Pugh score. For approximately 50 years, it has been widely utilized in the clinic due to its simplicity. The most commonly used technique worldwide for evaluating transplant candidates is the MELD score, which is based on the results of three standard laboratory tests: bilirubin, creatinine, and international normalized ratio (INR). However, severe hyponatremia, age, and ascites are some significant signs that can indicate the prognosis of patients with decompensated cirrhosis that are not included in the MELD score in clinical practice. The prognosis of patients with decompensated cirrhosis can be assessed using the adjusted MELD scores, which comprise the MELD-Na score and the integrated MELD (iMELD) score, which take age and blood sodium into account [13,14]. This study aimed to evaluate the association between electrocardiographic T-wave changes plus diastolic dysfunction and the compensation status of liver cirrhosis, as well as short-term mortality, as reflected by Child-Pugh scores and MELD-Na scores.

METHODS

Study design and settings

This observational cross-sectional study was conducted in the Gastroenterology and Hepatology Consultation Clinic and the Department of Internal Medicine at Baghdad Teaching Hospital, where sixty patients were enrolled between September 2024 and May 2025. All patients have liver cirrhosis and meet the inclusion criteria.

Inclusion criteria

All patients aged 18 years or older with chronic liver disease due to either the hepatitis B virus or the hepatitis C virus were included in the study if they did not meet any of the following exclusion criteria. The accurate diagnosis was achieved by the presence of positive hepatitis B surface antigen (HBsAg) and detectable HBV DNA, or the presence of positive hepatitis C antibody and detectable HCV RNA.

Exclusion criteria

The exclusion criteria include patients with a history of chronic cardiopulmonary diseases, use of medications that affect the T wave and QT intervals, and the presence of arrhythmia on electrocardiography. Patients with a history of chronic kidney disease (stages 3, 4, and 5) according to estimated glomerular filtration rate (eGFR); presence of any electrolyte disturbance that can affect the ECG morphology; metabolic syndrome, including diabetes, dyslipidemia, obesity, and hypertension; chronic liver disease due to an etiology other than hepatitis B or hepatitis C viruses; patients with hepatitis B or hepatitis C on antiviral treatment; patients with hepatocellular carcinoma and other malignancies, anemia, and thyroid dysfunction. The aim was to exclude any etiology of altered ECG patterns because these criteria may result in alterations in cardiac electrophysiology. Also, the antiviral treatment's impact on the progression of liver cirrhosis and the development of hepatocellular carcinoma was eliminated. As a result, 60 patients with chronic liver disease due to hepatitis B and hepatitis C viruses did not meet the exclusion criteria and were enrolled in the study from September 2024 to May 2025

Data collection and outcome measurements

Demographic and medical data that help determine the presence of liver cirrhosis, calculation of the Child-Pugh score, calculation of the Model for End-Stage Liver Disease – Sodium (MELD-Na) score, and the presence of exclusion criteria were taken from patients' admission files in the department of internal medicine and gastroenterology and hepatology outpatient clinic. The diagnosis of chronic liver disease was based on clinical evaluation (including history and physical examination), ultrasound criteria, and non-invasive evaluation using Fibroscan, as per the European Association for the Study of Liver (EASL) clinical practice guidelines. The Child Pugh score was used to classify the patients into three groups: Group A (scores 5-6), Group B (scores 7-9), and Group C (scores 10-15). Child-Pugh A indicates compensated liver disease, while Child-Pugh B and C indicate decompensated liver disease. MELD-Na score was employed to classify patients into two groups: MELD-Na < 20 and MELD-Na ≥ 20. The first group indicates liver disease with low short-term mortality (estimated 90-day mortality ≤ 4%), and the second one suggests liver disease with high short-term mortality (estimated 90-day mortality ≥ 7%) [15]. Electrocardiography analysis was performed using a 12-lead ECG calibrated device with a paper speed of 25 mm/s and an amplitude of 10 mm/mV. After obtaining the ECG tracings, the following intervals and parameters were evaluated: 1) Corrected QT interval (QTc) was calculated according to Bazett's formula. QTc prolongation was defined as greater than 430 ms in male patients and greater than 450 ms in female patients [9]; 2) T peak to T end (Tpe) was calculated employing the tangent method in all leads, and an average value was recorded; and 3) Tpe/QTc ratio was obtained by dividing Tpe by QTc. To minimize variation in specialist evaluation, the same specialist performed echocardiography on all patients. All assessments were performed using a Philips Echocardiography device. The left ventricle's systolic function was evaluated by calculating the left ventricular ejection fraction (LV EF) using the Simpson method. LV systolic dysfunction is defined as LV EF < 50%. Diastolic dysfunction was assessed according to the criteria endorsed by the American Society of Echocardiography and the European Association of Cardiovascular Imaging [16].

Ethical consideration

The research protocol was approved by the local Scientific Committee of the College of Medicine, University of Baghdad in March 2025. Patients signed an informed consent form at the time of admission, allowing the use of their data for research in accordance with Helsinki guidelines. The patients were assured that the data would be anonymized.

Statistical analysis

Data were analyzed using Statistical Product and Service Solution (SPSS) version 23.0 for Windows software. Categorical variables were presented as frequencies and/or percentages, while continuous variables were presented as mean ± standard deviation. Regarding continuous variables with normal distribution, one-way ANOVA and independent samples t-test were used for three-group and two-group analysis, respectively. For continuous variables that are not normally distributed, the Kruskal-Wallis test and the Mann-Whitney U test were used for three-group and two-group analysis, respectively. The Chi-Square test and Fisher's Exact test (if any group sample size was less than 5) were employed for the statistical analysis of categorical variables. A p-value of less than 0.05 was considered to be statistically significant.

RESULTS

Sixty patients with chronic liver disease were divided into three groups according to Child Pugh score; 17 had Child Pugh A, 20 had Child Pugh B, and 23 had Child Pugh C. The same sixty patients were divided into two groups according to the MELD-Na score; 33 had a MELD-Na score < 20, and 27 had a MELD-Na score ≥ 20. The average age of patients was 54.06 years, with a standard deviation of 7.63 years. Male patients comprised approximately 53.3%, while female patients comprised approximately 46.7%. Patients with chronic hepatitis B comprised about 58.3%, while patients with chronic hepatitis C comprised about 41.7%. The results in Tables 1 and 2 show a statistically significant older age in patients with decompensated liver disease (Child-Pugh class B and C) compared to patients with compensated liver disease (Child-Pugh class A).

Table 1: Baseline characteristics and Child-Pugh score

Variable	Child-Pugh A (n=17)	Child-Pugh B (n=20)	Child-Pugh C (n=23)	*p*-value
Age (year)	46.1±7.95	52.2±7.4	57.2±6.97	<0.0001
Male	9(52.9)	11(55)	12(52.2)	0.98
Female	8(47.1)	9(45)	11(47.8)	
HBV	10(58.8)	12(60)	13(56.5)	0.97
HCV	7(41.2)	8(40)	10(43.5)	

Values were expressed as frequency, percentage, and mean±SD. HBV: Hepatitis B virus; HCV: Hepatitis C Virus.

Table 2: Baseline characteristics and MELD-Na score

Variable	MELD-Na < 20 (n=33)	MELD-Na ≥ 20 (n=27)	*p*-value
Age (year)	51.5±7.55	57.3± 6.55	0.0023
Male	18(54.5)	14(51.9)	0.84
Female	15(45.5)	13(48.1)	
HBV	20(60.6)	15(55.6)	0.69
HCV	13(39.4)	12(44.4)	

Values were expressed as frequency, percentage, and mean±SD. HBV: Hepatitis B virus; HCV: Hepatitis C Virus.

There was a statistically significant increase in the Tpe/QTc ratio, a decrease in LV EF, and a rise in the percentage of diastolic dysfunction in patients with decompensated liver cirrhosis (Child-Pugh class B and C) (Tables 3 and 4). Likewise, the same significant association was observed in patients who had high short-term mortality (MELD-Na ≥ 20).

Table 3: ECG and echocardiography parameters with Child-Pugh score

Variable	Child-Pugh A (n=17)	Child-Pugh B (n=20)	Child-Pugh C (n=23)	*p*-value
QTc (ms)*	438±8.82	445±6.79	457±3.42	<0.0001
Mean Tpe (ms)†	59.4±6.29	68.8±5.93	76.8±4.4	<0.0001
Tpe/QTc ratio	0.136±0.015	0.155±0.014	0.168±0.01	<0.0001
LV EF (%)‡	56.8±4.17	54.1± 4.32	52.5±3.5	0.0155
Diastolic dysfunction	2(12)	6(30)	12(52)	0.0272

Values were expressed as frequency, percentage, and mean±SD.* Corrected QT interval; † T wave peak to end; ‡ Left Ventricle Ejection Fraction.

Table 4: ECG and echocardiography parameters with MELD-Na score

Variable	MELD-Na <20 (n=33)	MELD-Na ≥20 (n=27)	*p*-value
QTc (ms)*	441±8.93	455±6.16	<0.0001
Mean Tpe (ms)†	64.2±7.88	75.3±5.78	<0.0001
Tpe/QTc ratio	0.145±0.018	0.165±0.012	<0.0001
LV EF (%)‡	56±4.22	52.1±3.35	0.0003
Diastolic dysfunction	4(12)	16(59)	0.0002

Values were expressed as frequency, percentage, and mean±SD. *Corrected QT interval; † T-wave peak to end; ‡ Left Ventricle ejection fraction.

DISCUSSION

ECG changes are implicated in the current guidelines as an adjuvant criterion for CCM diagnosis. Due to the limited number of studies and the inconsistent correlation these studies show with overall survival, ECG abnormalities are not included in the primary diagnostic criteria for CCM. Nonetheless, the recommendation acknowledges that prolonged Tpe and a prolonged QTc interval may be indicators of CCM and a bad prognosis for cirrhotic patients. ECG tracings can be utilized as a preliminary evaluation in cirrhotic individuals suspected of having CCM because they are much easier to obtain than sophisticated ultrasound evaluations [4]. Patients with decompensated liver cirrhosis tend to be older, which this study has shown to be a statistically significant association. This may be attributed to the progressive nature of chronic liver disease and the impaired ability of the liver to regenerate, resulting in a more likely incidence of decompensation like hepatic encephalopathy, coagulopathy, jaundice, variceal bleeding, and ascites [17]. A study by Toma *et al*. outlined a similar statistically significant association between age and liver decompensation [18]. This study demonstrated a statistically significant association between prolonged QTc interval and decompensation of liver cirrhosis. Increased ventricular loading and QT interval prolongation have been linked; these findings highlight the common pathophysiological feature of CCM. Another study by Moaref *et al*. showed that prolongation of the QTc interval is observed in more than 50% of cirrhotic patients, most of whom had decompensated cirrhosis [19]. In a similar study, Toma et al. found that the QTc interval was statistically significantly longer in almost 46% of patients; most of these patients were in Child-Pugh class B or C [18]. Akboga *et al*. reported a significant association with Child Pugh score, but no statistical significance was observed with MELD-Na score [20]. Singh et al. [21] observed statistical significance with both liver decompensation and short-term mortality. In this study, patients with alcohol-related, metabolic, and inflammatory liver diseases were excluded from the inclusion criteria to avoid the contribution of these disorders to the observed ECG abnormalities [22]. One hypothesis for the extended QTc in cirrhosis that is particularly intriguing is dysfunction of the autonomic nervous system. Research indicates a correlation between elevated norepinephrine levels and QTc values. Additionally, the mechanism is reversed in normal patients, where a lower QTc is produced by extra norepinephrine [23]. The pathophysiological cause of malignant arrhythmias and sudden cardiac death in cirrhosis is QTc prolongation, especially after stressful events like liver transplantation, frequent large-volume paracentesis, or the implantation of portosystemic transjugular shunts [24]. This study demonstrated a statistically significant association between prolonged Tpe and decompensation of liver cirrhosis. Tpe is a marker for ventricular arrhythmogenesis and a helpful metric for assessing the transmural dispersion of repolarization [25]. A recent study suggests a correlation between prolonged Tp-e, arrhythmia, and mortality [26]. Similar findings were observed by Akboga *et al.*, whereby progressive prolongation of Tpe was markedly associated with increasing liver decompensation and higher short-term mortality [20]. This study outlined the statistical significance of the progressive increment of the Tpe/QTc ratio in association with both liver decompensation and short-term mortality. The QTc and Tpe intervals may fluctuate based on body weight and heart rate, rendering these indices less effective for predicting arrhythmogenesis. In this situation, it is preferable to use the Tp-e/QTc ratio rather than just evaluating the Tp-e or QT intervals, as the ratio remains constant despite dynamic changes in heart rate [27]. Likewise, Akboga *et al*. and Barutcu *et al*. reported a significant association with both Child-Pugh score and MELD-Na score, respectively [20, 28]. Although this study showed a statistically significant decline in LV function, as measured by LV ejection fraction, in association with liver decompensation and short-term mortality, most LV ejection fractions were in the preserved range (LV EF ≥ 50%). Only two patients (3%) had an LV EF less than 50%. This finding highlights the importance of diastolic dysfunction (rather than systolic dysfunction) as an early and more sensitive marker of cirrhotic cardiomyopathy. Typically, cardiac dysfunction is not evident unless a severe stressful event occurs, in which high-output heart failure or diastolic heart failure are the likely clinical features of heart decompensation [6]. Toma *et al*. and Li *et al*. outlined that there is no significant association between LV EF and each of the Child-Pugh scores and MELD-Na scores, respectively [18,29]. An increased percentage of patients having diastolic dysfunction has been outlined in this study to be significantly associated with liver cirrhosis decompensation and higher short-term mortality as reflected by Child Pugh and MELD-Na scores, respectively. Hyperdynamic circulation is the well-known hallmark cardiovascular finding in decompensated liver cirrhosis. Renal vasoconstriction and fluid and sodium retention complicate splanchnic vasodilation-related hypotension, which activates vasoconstrictor systems such as the sympathetic nervous system and the renin-angiotensin system (RAAS). The hyperdynamic circulation gets worse with this expansion of the circulating volume. The heart undergoes structural and functional alterations, including remodeling of the left ventricle. Diastolic dysfunction occurs early and is more likely with increasing severity of chronic liver disease [5,30]. Likewise, Toma *et al*. highlighted a statistically significant association between diastolic dysfunction and Child-Pugh score [18]. Li *et al*. also outlined a statistically significant association between diastolic dysfunction and MELD-Na score [29].

Study limitations

This study has multiple limitations. First, the sample size is small, so strong recommendations cannot be generalized to the broader population. This small sample may be related to the strict exclusion criteria to avoid the effect of several confounding variables on heart function. Second, A relatively small number of ECG parameters were taken into account for analysis. Third, due to the cross-sectional nature of this study, the relationship between future ventricular arrhythmias and each of the ECG parameters studied could not be assessed because of a lack of follow-up.

Conclusions

Cirrhotic cardiomyopathy is associated with the severity of liver cirrhosis. T wave changes, including QTc interval, Tpe interval, and Tpe/QTc ratio, correlate with the severity of liver cirrhosis and short-term mortality as assessed by the Child-Pugh score and MELD-Na score, respectively. The prognostic significance of these parameters needs further evaluation by extensive follow-up studies so that strong recommendations can be made about comprehensive cardiac assessment, especially in patients with decompensated liver cirrhosis.

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Conflict of interests

The author declares no conflict of interest.

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Data sharing statement

Supplementary data can be shared with the corresponding author upon reasonable request.

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