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Evaluation of the Analytical Value of ECG-Derived Tpeak-Tend Interval in Hypertensive Patients in the Babylon Governorate

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ABSTRACT

Background: *Hypertension remains one of the most prevalent global health challenges and is widely recognised as a major predisposing factor for a spectrum of cardiovascular diseases.* ***Aim:*** *To evaluate the presence of ventricular repolarisation abnormalities and conduction system abnormalities in a group of patients with systemic arterial hypertension.* ***Methods:*** *The analysis was performed using standard 12-lead electrocardiogram (ECG) recordings at paper speed of 25 mm/s and amplitude of 10 mm/mV. Heart rate and QRS duration were derived from automated ECG reports. QT, RR and Tpe intervals were assessed with semi-automated digital software. Only patients who had sinus rhythm were included.* ***Results:*** *Compared to the healthy controls, patients had statistically significant prolongations of QTd, QTc and Tpe intervals ($p < 0.05$). The higher prevalence of abnormal values was seen for the Tpe and corrected Tpe intervals (94% and 100%) than it was in the QTc interval (52%). In the hypertensive group, comparison analyses showed a strongly positive correlation between QT/QTc intervals and Tpe, TpecBa, and TpecFd ($p < 0.001$).* ***Conclusion:*** *The corrected Tpeak-Tend interval with Bazett's or Fridericia's formulae may be a useful marker for evaluating the variability of ventricular repolarisation in hypertensive patients. The significant Tpe and QTc interval prolongation in the results implies that these patients have an increased risk of repolarisation dispersion leading to cardiac arrhythmias.*

Keywords: Tpeak-Tend interval, QTc, Hypertension, Ventricular Repolarisation, Electrocardiography.

Article Information

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INTRODUCTION

Hypertension is a common global public health issue because of its high prevalence and being the leading risk factor for many cardiovascular diseases [1, 2]. Hypertension rarely has symptoms when it develops but untreated can lead directly to life-threatening entities such as kidney disease, stroke, peripheral vascular disease, and hypertensive heart disease. Most importantly, prolonged time to ventricular repolarisation is a strong independent predictor of CVD morbidity and mortality in patients with resistant hypertension [3]. The conventional 12-lead electrocardiogram (ECG) continues to be a key non-invasive diagnostic tool in the clinical setting, yielding numerous information essential for assessing and treating cardiovascular diseases. In recent years, the T peak-T end (Tp-e) a novel electrocardiographic sign that shows higher transmural dispersion of ventricular repolarization is the interval of the T wave, which has gained attention. [4–8].

In resource limited environments, access to precise and easily obtainable clinical and laboratory predictors of high cardiovascular risk hypertensive patients is essential. Such screening has been classically facilitated using the QT interval, which reflects total

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ventricular depolarisation and repolarisation time. Evaluating the risk of QTc interval prolongation allows for focused strategies to prevent morbidity and mortality. Nevertheless, the search for ECG-derived parameters at higher sensitivity lingers on. As a result, many studies focused on the relationship between ventricular repolarisation markers and arrhythmogenesis within various populations [9–14]. Yet despite these advances it is difficult to define which patients are at highest risk of life-threatening arrhythmias in the coming months. Current studies suggest a close link between the Tpe duration, and notably the Tpe/QTc ratio [15].

Systemic arterial hypertension (SAH) is associated with comorbidities that are indeed predictive of ventricular arrhythmias and sudden cardiac death [16–18], Thus, the purpose of our study was to investigate the alterations in Tpe interval in SAH patients in order to assess the incidence of ventricular repolarisation disturbances and cardiac conduction disturbances. The QT interval was rate corrected with QTc and the Tpe interval was also corrected with Bazett's and Fridericia's formulas.

METHODS

The present case-control study was carried out at Merjan Medical City, Al-Hilla, Babylon Governorate from September 2023 to June 2024.

Exclusion criteria:

Patients with diabetes, relevant arrhythmias, or taking drugs known to impact the ventricular repolarisation and T-wave parameters (cardioprotective agents) were excluded from this study [69]. 50 patients with hypertension and 50 healthy age-matched-control subjects (sex, weight, height) entered the study.

Ethical Approval

The study protocol was granted by the Publication Ethics Committee at the College of Medicine, University of Babylon / Iraq.

Electrocardiographic Measurements

Using a standard recording velocity of 25 mm/s and a calibration of 10 mm/mV, standard 12-lead electrocardiograms were recorded. These were the heart rate and the length of the QRS, both of which were determined from the electrical cardiogram reports. Calculations of QT, RR, and Tpe intervals were performed with the use of digital semi-automated software..

The stress test analysis was conducted only in the subjects with sinus rhythm. We measured the Tpe interval from the peak to the end of the T wave (in milliseconds) mainly in lead V5. If V5 was suboptimal, the leads V4 or V6 were utilized [19, 20]. Lead with T-wave amplitude < 1·5 mm were excluded from the analysis. The QTc interval was derived from the initial onset of the QRS complex to the end of T wave. The T-wave offset was defined as the point at which an upslope of the descending limb of the T wave intersects the TP baseline, regardless of any intervening U wave.If a low-amplitude terminal signal overlapped with the T wave, a tangent line was drawn from the steepest slope of the descending limb to the TP segment to the right, and the intersection with the TP segment was considered as the end of the T wave [21].

QT interval was adjusted for heart rate (QTc) using the Bazett's formula: $QT/\sqrt{RR}$ [22]. In case of Tpe interval, the Bazett´s formula [22] were used for correction of results [23]. The previous RR interval in seconds from the lead used for Tpe measurement. To simplify, these corrected values of Tpe (Tpec) were represented in milliseconds according to the convention used for QTc despite variances in the mathematical equivalent.

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RESULTS

Demographic and electrocardiographic variables of the study population, consisting of 50 hypertensive patients and their corresponding 50 age- and sex-matched healthy ones. The two groups were matched for age (46.42 ± 9.89 vs 45.84 ± 8.01 years; p =0.54) and BMI (33.13 ± 5.74 vs 31.98 ± 5 ·72 kg/m²; p =0·66), thus confirming successful matching with respect to these variables, which otherwise may have confounded the measured electrocardiographic parameters (Table S1). Much like the systolic and diastolic blood pressure, the RR interval between end-diastole to food present non significantly in the hypertensive versus control groups (0.81 ± 0.48 vs. 0.79 ± 0.11 seconds; p = 0.79), suggesting a similar baseline heart rate between cohorts as well (**Table I**).

On the other hand, for all ventricular repolarisation parameters: there was prolongation in the hypertensive group compared with controls and this finding was statistically significant. QT interval and rate-corrected QTc interval were longer in the hypertensive group than those with normotension (366.33 ± 41.97 vs. 338.78 ± 31.73 ms; p < 0.001) for QT; (427.72 ±74.02 vs 383.07±32 /15ms, p <0.001) for QTc respectively In the hypertensive group, there was an obvious prolongation of Tpeak-Tend (Tpe) interval (94.29 ± 16.33 vs. 67.14 ± 15.00 ms; p < 0.001).

In addition, the corrected Tpe intervals from Bazett's formula (TpecBa: 110.41 ± 26.38 vs. 75.65 ± 15.41 ms; p < 0.001) and Fridericia's formula (TpecFd: 98.58 ± 17.57 vs. 72.08 ± 15.07 ms; p <0·01) were all markedly raised in the patient cohort. These results collectively suggest that hypertensive patients have extensive changes in ventricular repolarisation, as indicated by the homogeneous prolongation of various time indices originating from the ECG and considered to be representative of the transmural dispersion of repolarisation, which is, in turn, an arrhythmoggenic substrate favoring the development of ventricular arrhythmias.

Table 1: Demographic and ECG variables in hypertensive and control subjects.

Parameter	Group	N	Mean	Std. Deviation	P-value
Age (years)	HT	50	46.42	9.89	0.54
	Control	50	45.84	8.01	
BMI (kg/m²)	HT	50	33.13	5.74	0.66
	Control	50	31.98	5.72	
RR (s)	HT	50	0.81	0.48	0.79
	Control	50	0.79	0.11	
QT (ms)	HT	50	366.33	41.97	<0.001
	Control	50	338.78	31.73	
QTc (ms)	HT	50	427.72	74.02	<0.001
	Control	50	383.07	32.15	
Tpe (ms)	HT	50	94.29	16.33	<0.001
	Control	50	67.14	15.00	
TpecBa (ms)	HT	50	110.41	26.38	<0.001
	Control	50	75.65	15.41	
TpecFd (ms)	HT	50	98.58	17.57	<0.001
	Control	50	72.08	15.07	

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The analysis revealed that 52% (n=26) of the hypertensive patients exhibited a prolonged QTc interval. In contrast, a prolonged Tpe interval was observed in 94% (n=47) of the patients, and abnormal TpecBa and TpecFd values were present in 100% of the hypertensive group. Although the uncorrected QT intervals were statistically longer in patients than in controls, all values remained within the normal physiological range (below 450 ms). The study showed 52% (no.=26) patients with prolonged QTc, while prolonged Tpe was 94% (no.=47) , TpecBa and TpecFd were 100%. QT intervals despite they were statistically longer than control , but all values were with in normal range(bellow 450ms). **Figure 1.**

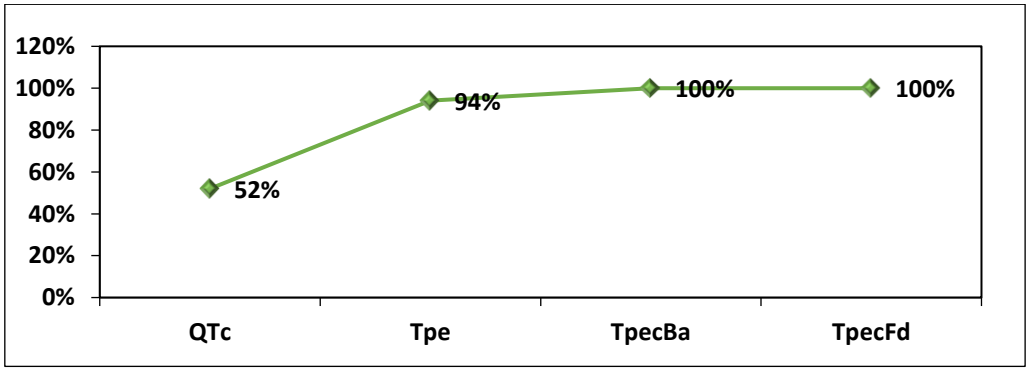

Figure 1: Curve showing the percentage of prolonged QTc, Tpe, TpecBa, and TpecFd in hypertensive patients.

Correlation analysis showed a very significant positive correlation between the QT/QTc intervals and Tpe, TpecBa, and TpecFd intervals ($p < 0.001$), indicating that the repolarization parameters are interrelated **(Figures 2, 3, 4).**

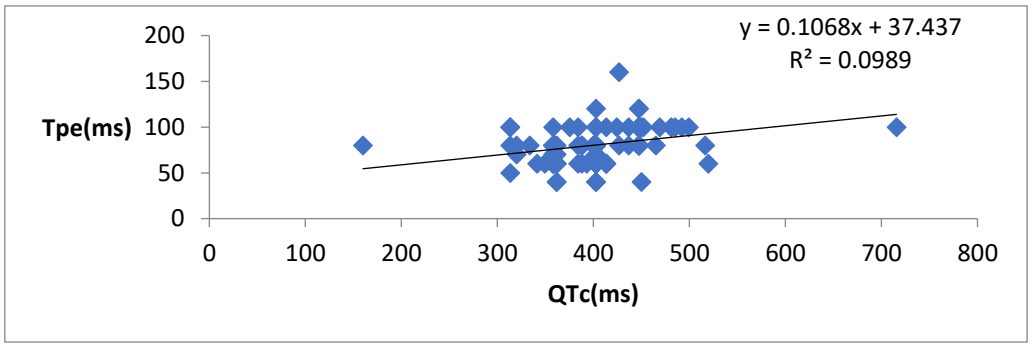

Figure 2: Correlation studies of QTc and Tpe of ECG in hypertensive patients.

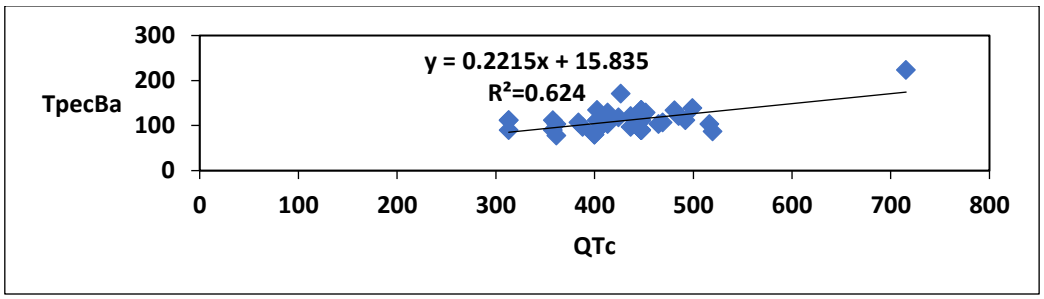

Figure 3: Correlation studies of QTc and TpecBa of ECG in hypertensive patients.

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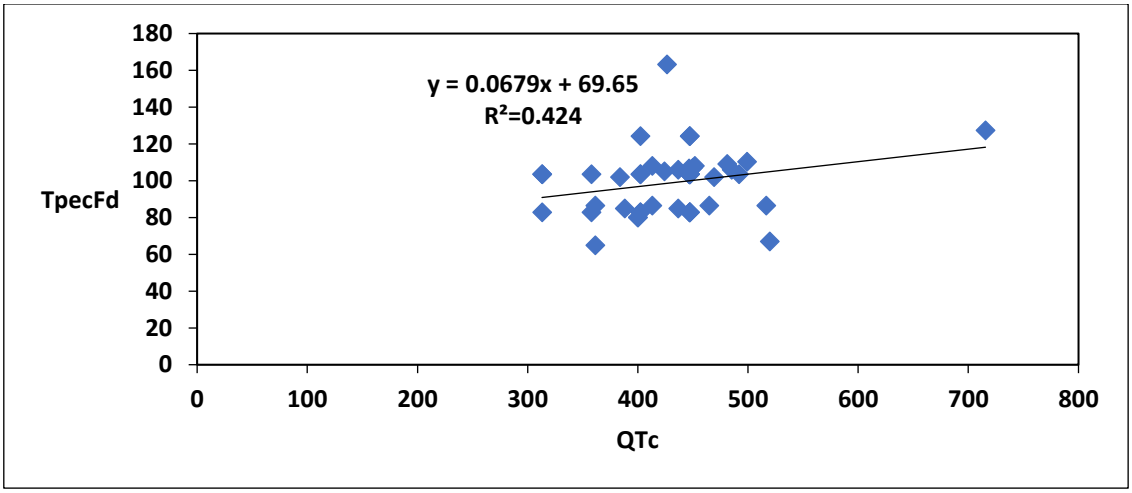

Figure 4: Correlation studies of QTc and TpecFd of ECG in hypertensive patients.

The AUC for the QT interval was 0.704 (sensitivity: 44%, specificity: 91% at a cut-off of 380 ms). The AUC for the QTc interval was higher at 0.776 (sensitivity: 75%, specificity: 85% at a cut-off of 402 ms). **Table 2.**

Table 2 ROC: for QT intervals, QTc, Tpe, TpecB, and TpecF.

Test Result Variable(s)		Area	Std. Error[a]	Asymptotic Sig.[b]	95% CL	
					Lower Bound	Upper Bound
Dimension 0	QT(ms)	.704	.054	.000	.599	.809
	QTC	.776	.051	.000	.676	.876
	T pe(ms)	.886	.033	.000	.821	.951
	TpeB	.887	.034	.000	.820	.954
	TpeF	.870	.036	.000	.800	.940

Notably, the Tpe interval demonstrated a superior AUC of 0.886 (sensitivity: 95%, specificity: 63% at a cut-off of 75 ms). The corrected Tpe using Bazett's formula (TpecBa) yielded an AUC of 0.887 (sensitivity: 97%, specificity: 57% at a cut-off of 77 ms), while Fridericia's correction (TpecFd) resulted in an AUC of 0.870 (sensitivity: 93%, specificity: 63% at a cut-off of 77 ms). **Figure 5.**

AUC of QTc was 0.77 with sensitivity and specificity of cutoff value 402 were 75% and 85%, respectively. **Figure 6.**

The ROC analysis showed AUC 0.88, which is very close to corrected Tpe by Bezalt formula (TpeB), but the sensitivity and specificity of Tpe were 95 % and 63% for a cut off value 75ms, while TpeB the cutoff value of 77 with sensitivity 97% and specificity 57%. AUC of TpeF was 0.87, and sensitivity and specificity for a cutoff value of 77 were 93% and 63%, respectively. **Figures 7,8,9**

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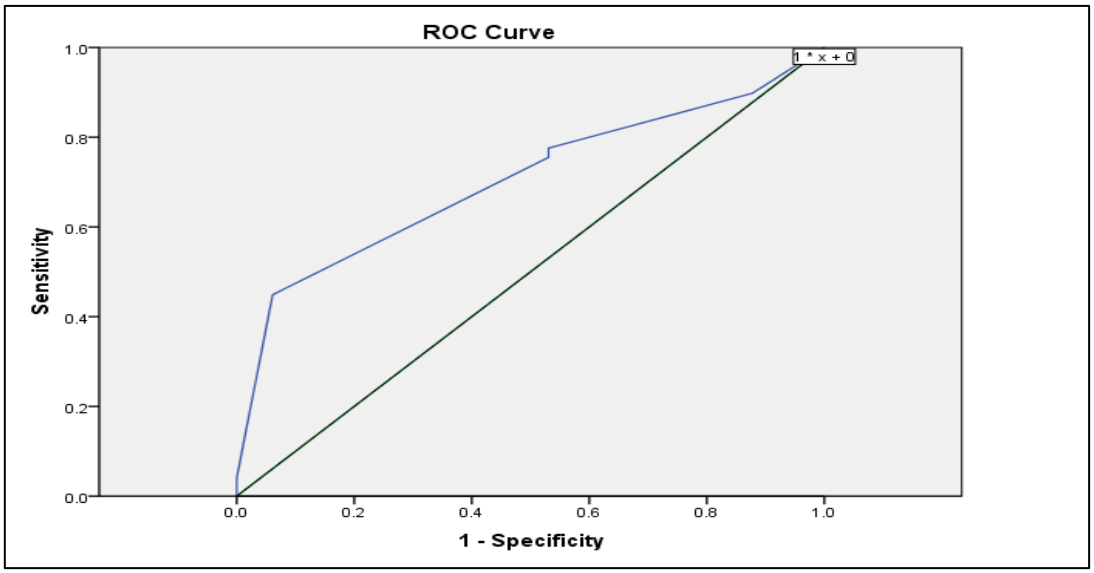

Figure 5: Receiver operating characteristic for QT interval from ECG of hypertensive patients.

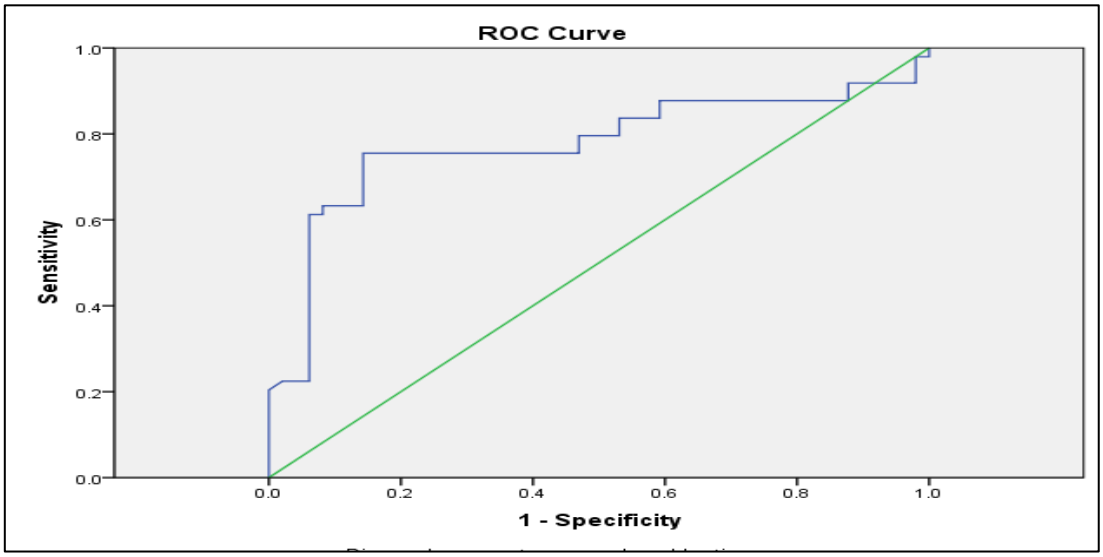

Figure 6: Receiver operating characteristic for QTc interval from ECG of hypertensive patients.

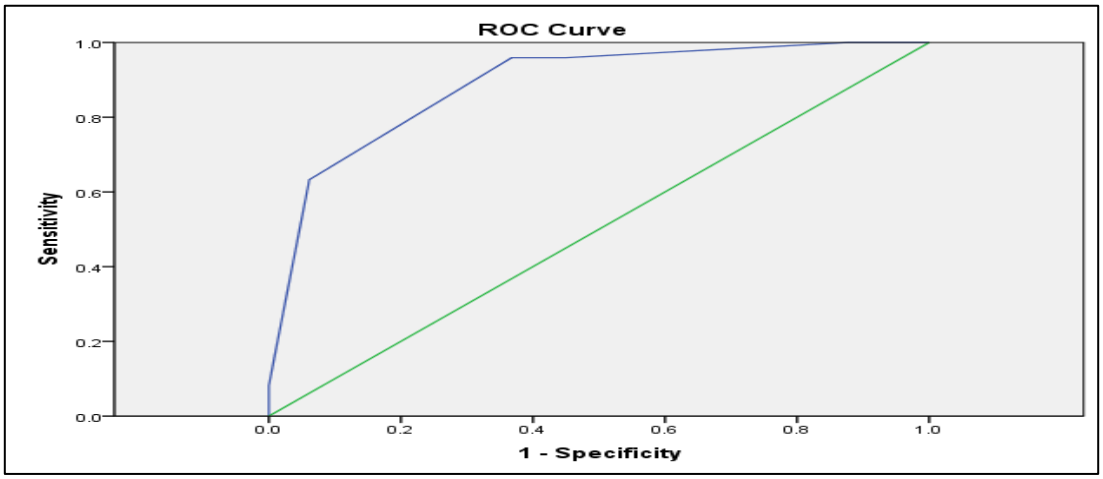

Figure 7: Receiver operating characteristic for Tpeak-end from ECG of hypertensive patients.

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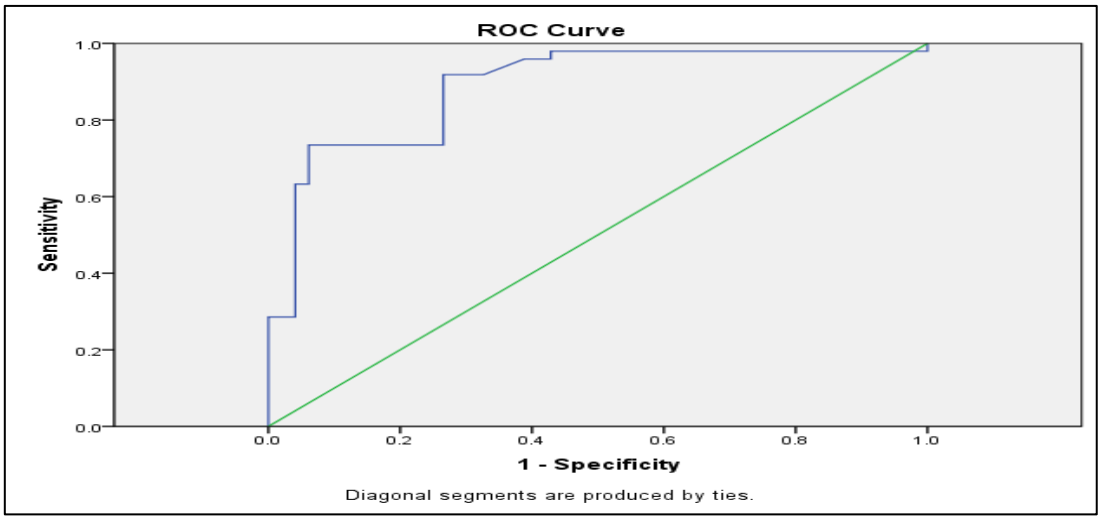

Figure 8: ROC for TpecDd interval from ECG of hypertensive patients

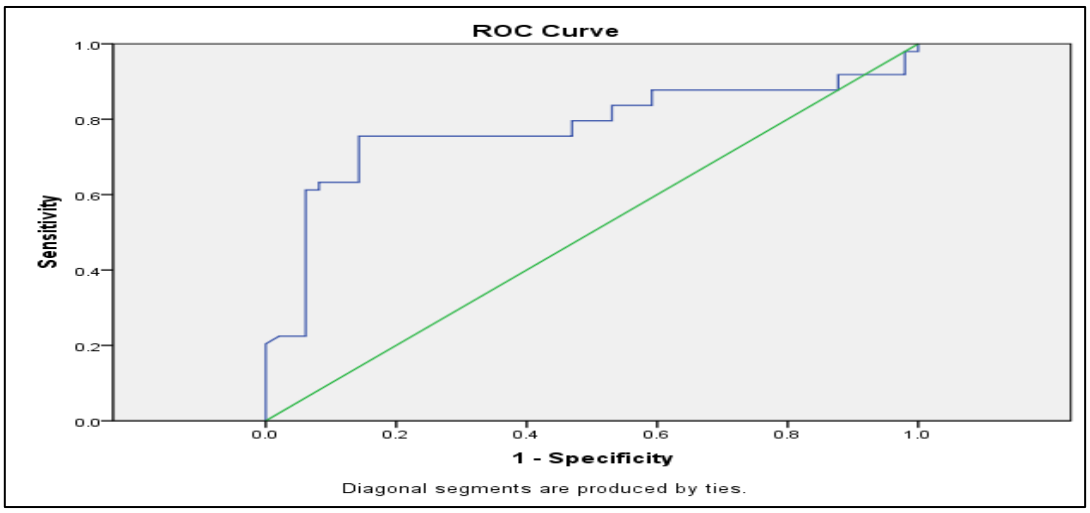

Figure 9: ROC for TpecDd from ECG of hypertensive patients.

DISCUSSION

Hypertension is well known to cause abnormal ventricular repolarisation which is one of the main mechanisms in arrhythmogenesis and increased cardiovascular risk . QT interval is a surrogate for the duration of electrical depolarisation and repolarisation of the cardiac ventricle, that is to say the cardiac action potential duration in the ventricle. Lengthening of the QT interval is a powerful determinant of cardiovascular, and sudden cardiac, death due to electrical instability [24]. As already noted, there "are several studies in the past, that proved the significance of the QT-prolonging effect of high blood pressure [24].

In this study, all repolarisation parameters (QT, QTc, Tpe, TpecBa and TpecFd) were also found prolonged with high significance, with hypertensive cohort closely controlled to the age and BMI of controls. In addition, the incidence of prolonged QTc interval was in fact very low (52%) compared with near-complete prolongation of Tpe and corrected Tpe intervals (94–100%). The presence of such abnormal Tpe parameters in such a high percentage underline their possible usefulness as a better marker for early repolarisation abnormalities compared to the classical QTc interval. Moreover, the tight correlation of corrected Tpe (TpecBa) with QTc underlines its clinical relevance.

The interval Tpeak-Tend has been established as a reproducible electrocardiographic index of transmural dispersion of repolarisation [25] The physiological basis for this prolongation is related to the voltage gradients that exist

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across the "M region" (mid-myocardium) of the left ventricular wall during phases 2 and 3 [16]. The peak of the T wave is completion of epicardial repolarisation and the end of the T wave is repolarisation of M cells. Thus, the Tpe interval is an adjunct descriptor to the transmural dispersion of repolarisation, a more refined measure than QT alone [26]. An abnormal dispersion (prolonged Tpe) suggests an unstable myocardial substrate with heterogeneous refractoriness, which is critically predisposing to re-entrant ventricular arrhythmias.26

ROC analysis demonstrated that QTc was superior to the uncorrected QT interval (AUC, 0.776 vs. 0.704) with higher sensitivity (75% vs. 44%). The Tpe also and its error-corrected variants (TpecBa and TpecFd) performed best in terms of diagnostic accuracy (all with AUC ~0.88). The high sensitivities of Tpe and corrected Tpe intervals (>93%) demonstrate their use for screening for abnormal myocardial repolarisation. However, these parameters were less specific (57-63%). A similar inverse relationship between sensitivity and specificity is well documented by Shenthar et al. (2015) study which showed high sensitivity but low specificity of the Tpe interval in ST-elevation myocardial infarction [28]. Such a low specificity is a deliberate trade-off when a threshold is set to maximise the number of true positives identified.

Study Limitations

The present study can be improved in the following aspects. First, a small sample size was used, which might impair the generalization of the findings to the larger population of hypertensive individuals. Secondly, the cross-sectional nature of this study does not allow any conclusions about causality of hypertension and the electrocardiographic changes observed. Thirdly, the exclusion of patients with diabetes mellitus, pre-existing arrhythmias and those on medications known to affect ventricular repolarisation, whilst essential for consistency in order to minimise confounding also reduces generalisability of these findings to an out-patient population where such co-morbidities frequently coexist. Moreover, the study was limited to a single center (Merjan Medical City, Babylon Governorate) which could introduce selection bias. Another limitation was the lack of echocardiographic data to assess left ventricular mass and structural changes. Left ventricular hypertrophy is a classical factor leading to repolarisation abnormalities occurring in patients with essential hypertension. Finally, there was no longitudinal follow-up data to determine if the observed increases in Tpe intervals were related to real-life events or negative cardiovascular outcomes.

CONCLUSION

The Tpeak-Tend interval, especially when corrected through Bazett's or Fridericia's formulae, is an extremely sensitive measure of ventricular repolarisation variability in hypertensive patients. The marked Tpe interval prolongation with QTc interval prolongation indicates that these patients have a vulnerable myocardial substrate and are at risk of arrhythmogenesis due to repolarisation dispersion.

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Conflict of Interest: None.

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Author Contributions

Ekhlas Hatem AL-Asadi contributed to the study conception, design, and supervision. Hawraa Hamid Hussein was responsible for data collection, ECG measurements, and statistical analysis. Ahlam Kadhim Abbood contributed to data interpretation and manuscript preparation.

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