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Research Article

CORRELATION BETWEEN CORRECTED QT INTERVAL AND SEVERITY OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE: A CROSS-SECTIONAL STUDY

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Abstract

Background: Chronic obstructive pulmonary disease (COPD) is a progressive respiratory disorder associated with systemic complications, particularly cardiovascular disease. The corrected QT (QTc) interval, a marker of ventricular repolarization, has been implicated in arrhythmogenesis and sudden cardiac death, and may be prolonged in patients with severe COPD.

Objective: To estimate the QTc interval in patients with COPD, assess pulmonary function and disease severity, and evaluate the correlation between QTc prolongation and COPD severity.

Methods: A cross-sectional study was conducted over 18 months among 166 patients with COPD diagnosed per Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria at a tertiary care hospital. Participants underwent spirometry, electrocardiography (ECG), and two-dimensional echocardiography (2D ECHO). Patients with pre-existing cardiac disease, electrolyte imbalance, or medications affecting the QT interval were excluded. QTc was correlated with GOLD stage and echocardiographic findings using ANOVA and chi-square tests.

Results: Participants were predominantly male (81.9%) with a mean age of 59.81 ± 11.63 years. The overall mean QTc interval was 448.42 ± 28.80 ms (male 454.17 ± 27.13 ms; female 422.33 ± 20.81 ms), and QTc prolongation was present in 59.0% of participants. Mean QTc increased progressively with GOLD stage, from 414.5 ms in mild disease to 480.19 ms in very severe disease ($F = 208.182$, $p < 0.05$), and with worsening 2D ECHO findings ($F = 67.422$, $p < 0.05$). The proportion of

participants with prolonged QTc rose from 1.9% in mild COPD to 100% in very severe COPD ($\chi^2 = 122.005$, $p < 0.05$).

Conclusion: QTc prolongation is common among patients with COPD and correlates strongly with disease severity and echocardiographic evidence of pulmonary hypertension. QTc measurement, a simple and non-invasive ECG-derived parameter, may serve as an adjunct marker for cardiovascular risk stratification in COPD, supporting closer cardiac surveillance in patients with advanced disease.

Keywords: Chronic obstructive pulmonary disease; QTc interval; electrocardiography; cardiovascular disease; pulmonary function; GOLD criteria; arrhythmia; hypoxemia.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common, preventable, and treatable condition characterized by persistent airflow limitation and chronic inflammation resulting from prolonged exposure to noxious particles or gases, most often cigarette smoke.1 Beyond its respiratory manifestations, COPD is increasingly recognized as an independent risk factor for cardiovascular disease (CVD): population cohorts report a 25% increase in major adverse cardiovascular events and up to a 52% higher ten-year cardiovascular risk in COPD patients compared with standard risk estimates, driven by systemic inflammation, oxidative stress, hypoxemia, and shared risk factors such as smoking.2,3

The QT interval, measured from the onset of the QRS complex to the end of the T wave, is a non-invasive electrocardiographic marker of ventricular repolarization. Because QT duration varies with heart rate, it is conventionally expressed after rate correction as the QTc interval. QTc prolongation (>450 ms in men, >460 ms in women) predisposes to early after depolarizations, ventricular arrhythmias, and sudden cardiac death, and has established prognostic value in myocardial infarction, heart failure, and other cardiometabolic conditions.4,5 In COPD, hypoxemia, hypercapnia, systemic inflammation, autonomic dysfunction, and pharmacologic agents such as beta-agonists and anticholinergics may all contribute to QTc prolongation, and prior studies suggest this prolongation becomes more pronounced with advancing disease severity as staged by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria.

Given the need for a simple, accessible, and reproducible marker of cardiovascular risk in COPD beyond existing severity indices, this study was undertaken to estimate the QTc interval in patients with COPD and to examine its correlation with pulmonary function and GOLD-defined disease severity.

OBJECTIVES

- To estimate the QTc interval in patients with COPD.
- To assess pulmonary function testing and disease severity in COPD.
- To study the correlation between QTc interval and COPD severity.

MATERIALS AND METHODS

Study Design and Setting

This was a cross-sectional, observational study conducted over 18 months (May 2023 to October 2024) among patients attending the outpatient and inpatient services of the Department of General Medicine at a tertiary care hospital attached to Bangalore Medical College and Research Institute (BMCRI), Bengaluru, India.

Study Population

Patients aged 40-90 years diagnosed with COPD per GOLD criteria, who provided informed consent, were enrolled. Patients on medications known to affect the QT interval, those with a history of pulmonary disease other than COPD, pre-existing cardiac disease (other than cor pulmonale), or pre-existing electrolyte disturbance, parathyroid disorders, or diabetic autonomic neuropathy were excluded.

Sample Size

Sample size was estimated using the mean QTc interval and standard deviation (437.9 ± 29.5 ms) reported by Sievi et al., with a 95% confidence level ($Z\alpha = 1.96$) and an expected mean difference of 5 ms, yielding a minimum requirement of 135 participants. A total of 166 patients were enrolled, exceeding this requirement.

Data Collection

Following institutional ethics committee approval and informed consent, a detailed history and clinical examination were recorded for each participant. Investigations performed included spirometry, standard 12-lead ECG, chest radiography, and two-dimensional echocardiography (2D ECHO) to exclude cardiac disease other than cor pulmonale; troponin I was used to exclude ischemic heart disease. COPD severity was classified using GOLD criteria based on post-bronchodilator FEV1 (GOLD 1, mild, FEV1 ≥80% predicted; GOLD 2, moderate, 50-79%; GOLD 3, severe, 30-49%; GOLD 4, very severe, <30%). The QTc interval was derived from the ECG and correlated with GOLD stage.

Statistical Analysis

Data were compiled in MS Excel and analyzed using SPSS version 26.0. Quantitative variables were expressed as mean ± standard deviation and qualitative variables as frequency and percentage. One-way ANOVA (or the Kruskal-Wallis test where appropriate) was used to compare QTc interval across three or more groups, and the chi-square test was used to assess association between categorical variables. Pearson's or Spearman's correlation was used as appropriate. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 166 patients diagnosed with COPD were included in the analysis.

Baseline Characteristics

The mean age of participants was 59.81 ± 11.63 years, with the largest proportion (30.1%) in the 61-70-year age group. Males predominated (81.9%). A history of smoking was present in 63.3% of participants, with 7.8% reporting biomass fuel exposure and 7.2% reporting occupational exposure. Cough was reported by 97.6% of participants and breathlessness by 48.2% (Table 1).

Table 1. Sociodemographic and clinical characteristics of study participants (n = 166)

Variable	Category	Frequency (n)	Percentage (%)
Age group (years)	40-50	39	23.5
	51-60	42	25.3
	61-70	50	30.1
	71-80	27	16.3
	81-90	8	4.8
	Mean ± SD	59.81 ± 11.63 years	-
Sex	Male	136	81.9
	Female	30	18.1
Personal / exposure history	Smoking	105	63.3
	Biomass fuel exposure	13	7.8
	Occupational exposure	12	7.2
Cough	Present	162	97.6
	Absent	4	2.4
Breathlessness	Present	80	48.2
	Absent	86	51.8

Laboratory, Spirometric, and GOLD Classification

Mean oxygen saturation was 93.69 ± 2.16%, and mean hemoglobin was 11.90 ± 1.86 g/dL. Serum electrolytes were within normal limits in all participants. Spirometry showed a mean FEV1/FVC ratio of 0.52 ± 0.11 and a mean FEV1 of 54.83 ± 25.39% predicted. Based on GOLD staging, 31.3% of participants had mild, 21.1% moderate, 22.3% severe, and 25.3% very severe COPD (Table 2).

Table 2. Laboratory, spirometric, and GOLD severity classification

Parameter	Category	Value / Frequency	Percentage (%)
Oxygen saturation (%)	Mean ± SD	93.69 ± 2.16	-
Hemoglobin (g/dL)	Mean ± SD	11.90 ± 1.86	-
Serum electrolytes	Normal	166	100.0
FEV1/FVC ratio	Mean ± SD	0.52 ± 0.11	-
FEV1 (% predicted)	Mean ± SD	54.83 ± 25.39	-
GOLD stage	Mild	52	31.3
	Moderate	35	21.1
	Severe	37	22.3
	Very severe	42	25.3

Corrected QTc Interval and Echocardiographic Findings

The overall mean corrected QTc interval was 448.42 ± 28.80 ms, and was longer in men (454.17 ± 27.13 ms) than in women (422.33 ± 20.81 ms). QTc prolongation was observed in 59.0% of participants. On 2D ECHO, 47.6% of participants had normal findings, 4.2% had moderate pulmonary artery hypertension (PAH), 5.4% had severe PAH, and 42.8% had dilated right atrium (RA) and right ventricle (RV) with elevated pulmonary artery systolic pressure (PASP) (Table 3).

Table 3. Corrected QTc interval, QTc prolongation, and 2D echocardiographic findings

Parameter	Category	Value / n	% / SD
Corrected QTc interval (ms)	Male, mean ± SD	454.17	± 27.13
	Female, mean ± SD	422.33	± 20.81
	Overall, mean ± SD	448.42	± 28.80
QTc prolongation	Present	98	59.0%
	Absent	68	41.0%
2D ECHO findings	Normal	79	47.6%
	Moderate PAH	7	4.2%
	Severe PAH	9	5.4%
	Dilated RA/RV, high PASP	71	42.8%

Association Between QTc Interval and Disease Severity

Mean corrected QTc interval increased progressively across GOLD stages: 414.5 ± 15.16 ms in mild, 446.83 ± 11.59 ms in moderate, 461.51 ± 11.60 ms in severe, and 480.19 ± 13.13 ms in very severe COPD, a difference that was statistically significant ($F = 208.182$, $p < 0.05$) (Table 4).

Table 4. Corrected QTc interval by GOLD severity classification

GOLD stage	Mean QTc (ms)	SD	F-value	p-value
Mild	414.5	15.155		
Moderate	446.83	11.585	208.182	<0.05
Severe	461.51	11.597		
Very severe	480.19	13.128		

Similarly, mean QTc increased with worsening 2D ECHO findings, from 426.09 ± 21.42 ms in participants with normal echocardiograms to 470.03 ± 18.28 ms in those with dilated RA/RV and elevated PASP ($F = 67.422$, $p < 0.05$) (Table 5).

Table 5. Corrected QTc interval by 2D echocardiographic findings

2D ECHO finding	Mean QTc (ms)	SD	F-value	p-value
Normal	426.09	21.418		
Moderate PAH	459.29	13.512	67.422	<0.05
Severe PAH	465.44	8.647		
Dilated RA/RV, high PASP	470.03	18.276		

The proportion of participants with prolonged QTc rose markedly with disease severity: 1.9% in mild, 54.3% in moderate, 97.3% in severe, and 100% in very severe COPD, an association that was statistically significant ($\chi^2 = 122.005$, $p < 0.05$) (Table 6).

Table 6. GOLD severity classification and prolonged QTc

GOLD stage	Prolonged QTc: Yes n (%)	Prolonged QTc: No n (%)	χ^2	p-value
Mild	1 (1.9%)	51 (98.1%)		
Moderate	19 (54.3%)	16 (45.7%)	122.005	<0.05
Severe	36 (97.3%)	1 (2.7%)		
Very severe	42 (100%)	0 (0%)		

DISCUSSION

COPD exerts wide-ranging effects on cardiovascular autonomic function that extend beyond the right heart, and even early-stage disease may be regarded as having systemic cardiovascular implications.6,7 Because QTc prolongation predisposes to ventricular arrhythmia and sudden cardiac death, and because it can be derived from a routine ECG without additional cost or invasive testing, it has been proposed as a practical adjunct for cardiovascular risk stratification and severity assessment in COPD.6,8

In this study of 166 patients with COPD, the mean age (59.81 ± 11.63 years) and male predominance (81.9%) were consistent with prior reports; Cherian et al. described a similar mean age of 60.5 years with 80% male participants, and in the study by Jegan and Kumar the largest proportion of patients was similarly concentrated in the 51-70-year range.9,10 Smoking was the predominant exposure (63.3%) in our cohort, in keeping with the established role of smoking in worsening cardiac repolarization in COPD; Jegan and Kumar reported a significantly longer mean QTc in smokers (455 ms) than non-smokers (436.3 ms, $p<0.024$).10

The overall mean corrected QTc interval in our cohort (448.42 ± 28.80 ms) and the prevalence of QTc prolongation (59.0%) were broadly comparable to, though somewhat lower than, the 79% prevalence reported by Cherian et al., in whose study mean QTc rose from 426.8 ms in stage 1 to 465.1 ms in stage 3 disease.9 QTc was also longer in men than women in our cohort (454.17 vs. 422.33 ms), mirroring the gender difference reported by Jegan and Kumar (452.92 ms in men vs. 429.64 ms in women) and by earlier work from Sievi et al. and Nilsson et al., both of whom found COPD patients to have significantly longer QTc intervals than healthy controls.9-12

The central finding of this study - a graded, statistically significant increase in mean QTc interval with worsening GOLD stage (414.5 ms in mild disease to 480.19 ms in very severe disease; $F = 208.182$, $p < 0.05$) and a corresponding rise in the proportion of patients with QTc prolongation (1.9% to 100%; $\chi^2 = 122.005$, $p < 0.05$) - is concordant with the broader literature linking COPD severity to cardiac repolarization abnormalities. Nilsson et al. similarly reported that QTc prolongation prevalence rose with GOLD stage, particularly among men, after adjustment for age and BMI, while Mannino et al., in a large cohort from the Atherosclerosis Risk in Communities study, demonstrated a parallel gradient of increasing mortality with advancing GOLD stage (hazard ratio 5.7 for GOLD 3-4 versus 1.4 for GOLD 1), reinforcing the clinical relevance of disease staging to cardiovascular outcomes.11,13

QTc interval also correlated significantly with echocardiographic severity in our cohort, rising from 426.09 ms in patients with normal 2D ECHO findings to 470.03 ms in those with dilated right heart chambers and elevated pulmonary artery systolic pressure ($F = 67.422$, $p < 0.05$), suggesting that QTc prolongation in COPD may in part reflect the hemodynamic burden of pulmonary hypertension and right ventricular strain, in addition to hypoxemia, systemic inflammation, and autonomic dysfunction described in previous work.6,8,12 Notably, serum electrolytes

were normal in all participants in our cohort, arguing against electrolyte disturbance as a driver of the observed QTc prolongation and instead supporting disease-related mechanisms, consistent with the findings of Zilberman-Itskovich et al., who reported that prolonged QTc in hospitalized COPD patients was not attributable to electrolyte imbalance but was nonetheless associated with significantly higher in-hospital mortality.8

Taken together, these findings support the use of QTc interval measurement as a simple, low-cost, and widely accessible marker to complement GOLD staging in identifying COPD patients at greater cardiovascular risk, particularly those with severe or very severe disease, in whom closer ECG surveillance may be warranted.

LIMITATIONS

This study has several limitations. The cross-sectional design precludes causal inference between COPD severity and QTc prolongation. Potential confounders such as concurrent medication use, comorbidities, and lifestyle factors were not comprehensively assessed. Smoking and occupational exposure histories were self-reported and may be subject to recall bias. Finally, as a single-center study, generalizability to other populations may be limited.

CONCLUSION

Corrected QTc interval was significantly and progressively prolonged with increasing severity of COPD, both by GOLD classification and by echocardiographic findings, in this cohort of 166 patients. QTc prolongation was more prevalent among patients with severe and very severe disease. These findings support QTc interval measurement as a useful, non-invasive, ECG-derived marker for cardiovascular risk stratification in COPD, and highlight the value of routine ECG monitoring - particularly in patients with advanced disease - to enable earlier identification and management of cardiovascular complications and thereby reduce morbidity and mortality.

DECLARATIONS

Ethical approval: This study was approved by the Institutional Ethics Committee, Bangalore Medical College and Research Institute, and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

Conflict of interest: The authors declare no conflict of interest.

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