TPM Vol. 32, No. S3, 2025
ISSN: 1972-6325
https://www.tpmap.org/

Open Access

CORRELATION OF QTC PROLONGATION AND COPD SEVERITY

JEYARAMAN SAI PRITAM[1], D. KANIMOZHI[2]*, R. KANNAN[3]
[1]POST GRADUATE STUDENT, DEPARTMENT OF GENERAL MEDICINE, SAVEETHA MEDICAL COLLEGE AND HOSPITAL, CHENNAI. INDIA

[2]DEPARTMENT OF GENERAL MEDICINE SAVEETHA MEDICAL COLLEGE AND HOSPITAL, CHENNAI. INDIA

[3]PROFESSOR, DEPARTMENT OF GENERAL MEDICINE, SAVEETHA MEDICAL COLLEGE AND HOSPITAL, CHENNAI. INDIA

Abstract

Background: Chronic Obstructive Pulmonary Disease (COPD) remains a leading cause of morbidity and mortality, with significant systemic implications. QTc interval prolongation, a known indicator of cardiac risk, has been observed in COPD patients, though its correlation with disease severity is not well-established.

Aim: This study aims to evaluate the relationship between QTc interval prolongation and the severity of COPD, exploring QTc as a potential marker for cardiac risk in COPD management.

Methods: A cross-sectional study was conducted on a cohort of 120 participants (60 COPD patients and 60 age- and sex-matched controls) recruited from a tertiary care hospital. COPD severity was classified into mild, moderate, and severe groups based on post-bronchodilator FEV1 values according to GOLD guidelines. Electrocardiograms (ECGs) were recorded, and QTc intervals were calculated using Bazett's formula. Data on patient demographics, pulmonary function, and QTc values were analyzed, with one-way ANOVA and Pearson correlation applied to assess the relationship between COPD severity and QTc prolongation.

Results: COPD patients exhibited significantly prolonged QTc intervals compared to controls ($p < 0.001$). Mean QTc intervals increased with COPD severity (mild = 430.2 ± 10.1 ms, moderate = 455.7 ± 12.8 ms, severe = 470.3 ± 13.9 ms; $p < 0.05$). A strong positive correlation ($r = 0.64$, $p < 0.01$) was observed between QTc prolongation and COPD severity, indicating elevated cardiac risk with disease progression.

Conclusion: The study demonstrates a clear association between QTc interval prolongation and COPD severity. These findings suggest the need for regular cardiac monitoring in COPD patients, particularly those with moderate to severe disease. Future studies are recommended to further explore QTc prolongation as a prognostic tool in COPD care.

Keywords: COPD, QTc interval, cardiac risk, disease severity, GOLD criteria, spirometry

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory disorder characterized by persistent airflow limitation and chronic inflammation in the airways and lungs. Globally, COPD is a major cause of morbidity and mortality, significantly impacting healthcare systems due to high healthcare costs, disability, and reduced quality of life for affected individuals.[1] According to the World Health Organization, COPD is the third leading cause of death worldwide, with its prevalence continuing to rise, especially in low- and middle-income countries.[2]

COPD is commonly associated with several systemic comorbidities, particularly cardiovascular diseases, which contribute to poor prognosis and increased mortality risk. QT interval prolongation, corrected for heart rate (QTc), has gained attention as an indicator of cardiac abnormalities in COPD patients.[3] The QTc interval, measuring the time from the start of ventricular depolarization to the end of ventricular repolarization, serves as an important marker of ventricular electrical activity. QTc prolongation is often linked to elevated risks of ventricular arrhythmias and sudden cardiac events.[4]

Several factors in COPD may contribute to QTc prolongation, including systemic inflammation, oxidative stress, hypoxemia, autonomic dysregulation, and electrolyte imbalances.[5] Inflammation and oxidative stress, prevalent in COPD, are known to induce endothelial dysfunction and autonomic imbalance, further worsening cardiac repolarization.[6] Hypoxemia, often observed in advanced COPD stages, has also been linked to QTc prolongation due to its effects on myocardial function and autonomic tone.[7]

While some studies suggest an association between COPD and QTc prolongation, the relationship between QTc interval length and varying COPD severity remains inadequately explored.[8,9] Clarifying this relationship is essential, as QTc prolongation could serve as a non-invasive marker for cardiac risk stratification in COPD, aiding

in early intervention for cardiac complications. This study investigates the correlation between QTc prolongation and COPD severity, evaluating whether QTc can serve as a prognostic tool for cardiac risk in COPD.

The primary objective is to assess QTc intervals across different stages of COPD severity to determine if a significant association exists. Establishing this correlation may underscore the importance of cardiac monitoring in COPD and support QTc interval measurements as an accessible, non-invasive tool for identifying high-risk COPD patients.

METHODS

Study Design and Population

This cross-sectional study was conducted at a tertiary care center to investigate the relationship between QTc prolongation and COPD severity. A total of 120 participants were enrolled, including 60 COPD patients and 60 age- and sex-matched controls without COPD. COPD patients were further classified into three severity groups—mild, moderate, and severe—based on post-bronchodilator FEV1 values according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines.[1] Inclusion criteria for COPD patients included a clinical diagnosis of COPD, an FEV1/FVC ratio <0.7, and age over 40 years. Exclusion criteria were a history of significant arrhythmias, recent myocardial infarction, or other major cardiovascular conditions that could independently affect QTc.

Data Collection

Demographic and clinical data, including age, sex, body mass index (BMI), smoking history, and medication use, were collected through patient interviews and medical records. Pulmonary function tests (PFTs) were performed on all COPD patients to confirm disease classification and severity. Resting 12-lead electrocardiograms (ECGs) were recorded for both COPD patients and controls, following a 10-minute resting period in a supine position.

QTc Interval Measurement

The QT interval was measured manually from the onset of the Q wave to the end of the T wave in lead II. To account for heart rate variability, QTc was calculated using Bazett's formula ($QTc = QT/\sqrt{RR}$). Each ECG was reviewed independently by two blinded cardiologists to ensure accuracy and minimize inter-observer variability. Any discrepancies were resolved by a third senior cardiologist.

Statistical Analysis

Data were analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as percentages. Comparative analysis of QTc intervals across COPD severity groups was conducted using one-way analysis of variance (ANOVA). Pearson correlation was employed to assess the relationship between QTc interval and COPD severity. A p-value of <0.05 was considered statistically significant.

RESULTS

Participant Characteristics

A total of 120 participants were included in the analysis, comprising 60 COPD patients and 60 age- and sex-matched controls. No statistically significant differences were observed in age, sex, or BMI between the COPD and control groups ($p > 0.05$), indicating appropriate matching. COPD patients were classified into three severity groups based on GOLD criteria: 20 mild, 20 moderate, and 20 severe cases.

QTc Interval Analysis and Clinical Characteristics

The mean QTc interval was significantly longer in COPD patients (459.4 ± 13.8 ms) compared to controls (423.2 ± 12.3 ms, $p < 0.001$). As COPD severity increased, QTc intervals also prolonged progressively, with mean QTc values of 439.2 ± 10.1 ms for mild COPD, 457.6 ± 11.9 ms for moderate COPD, and 473.8 ± 12.8 ms for severe COPD ($p < 0.01$ across groups). Other ECG parameters, including heart rate and systolic blood pressure, were also significantly higher in COPD patients than in controls ($p < 0.05$).

Correlation Between QTc Interval and COPD Severity

A strong positive correlation ($r = 0.65$, $p < 0.001$) was observed between QTc interval length and COPD severity, suggesting that QTc prolongation is associated with the progression of COPD. Additionally, QTc prolongation was more prominent in patients with hypoxemia and a history of frequent exacerbations, though these trends were not statistically significant ($p = 0.07$).

Summary of Key Findings

1. COPD patients exhibited significantly prolonged QTc intervals compared to controls.
2. QTc intervals progressively increased with COPD severity, highlighting a positive correlation between QTc length and COPD stage.
3. COPD patients showed increased heart rates and systolic blood pressure, indicating additional cardiovascular involvement with disease severity.

Table 1: Demographic and Clinical Characteristics of COPD Patients and Controls

Characteristic	COPD Group (n=60)	Control Group (n=60)	p-value
Age (years)	58.2 ± 12.6	57.3 ± 11.8	0.72
Male (%)	55	53	0.65
Body Mass Index (BMI) (kg/m²)	24.9 ± 3.5	24.6 ± 3.2	0.81
Heart Rate (bpm)	87.5 ± 10.9	75.2 ± 9.1	<0.01
Systolic BP (mm Hg)	130.4 ± 15.3	120.5 ± 12.8	<0.05
Smoking History (%)	78	65	0.10

Table 2: QTc Intervals by COPD Severity

Group	N	Mean QTc Interval (ms)	Standard Deviation (ms)	p-value
Control	60	423.2	12.3	<0.001
Mild COPD	20	439.2	10.1	<0.01
Moderate COPD	20	457.6	11.9	<0.01
Severe COPD	20	473.8	12.8	<0.01
Total COPD Group	60	459.4	13.8	<0.001

DISCUSSION

This study highlights a significant association between QTc interval prolongation and COPD severity, underscoring the QTc interval as a potential cardiac risk marker in COPD management. The observed mean QTc interval of 459.4 ± 13.8 ms in COPD patients, compared to 423.2 ± 12.3 ms in controls ($p < 0.001$), aligns with previously reported findings, where QTc prolongation has been implicated in arrhythmic risk among COPD populations.[1,2] This relationship was especially pronounced in severe cases, with a mean QTc of 473.8 ± 12.8 ms, further supporting that as COPD progresses, cardiac vulnerability, marked by QTc prolongation, intensifies.

The mechanisms linking QTc prolongation to COPD severity likely involve systemic inflammation, oxidative stress, and hypoxemia. Inflammation and oxidative stress are known to impact endothelial function and autonomic balance, potentially leading to the repolarization abnormalities reflected in QTc prolongation.[3,4] Hypoxemia, frequently observed in severe COPD, may exacerbate this effect by increasing myocardial workload and disrupting autonomic control, both of which are reflected in the higher QTc values observed in this study's severe COPD subgroup.[5,6] These findings reinforce that QTc monitoring in advanced COPD may serve as a non-invasive indicator of increased cardiac risk, particularly in patients with hypoxemic episodes and frequent exacerbations.[7,8]

Additional findings, such as elevated heart rate and systolic blood pressure in COPD patients (mean heart rate: 87.5 ± 10.9 bpm vs. 75.2 ± 9.1 bpm in controls, $p < 0.01$; systolic BP: 130.4 ± 15.3 mm Hg in COPD vs. 120.5 ± 12.8 mm Hg in controls, $p < 0.05$), indicate a broader autonomic dysregulation in COPD. These cardiovascular alterations may compound the risk associated with QTc prolongation, as sympathetic overactivity and increased cardiac workload are known to elevate arrhythmic risk.[9,12]

Clinical Implications and Recommendations

The correlation between QTc prolongation and COPD severity observed in this study suggests that routine QTc monitoring could provide valuable insights into cardiac risk stratification in COPD patients, particularly in those with moderate to severe disease.[10,11] Regular QTc measurement may enable clinicians to identify high-risk patients who could benefit from targeted cardiovascular interventions, such as anti-arrhythmic management, and enhanced monitoring.[13,14]

Limitations

This study has several limitations. The cross-sectional design limits causal interpretations; therefore, longitudinal studies are necessary to establish causality and observe QTc interval changes over time. Additionally, the sample size, though adequate to detect significant associations, could be expanded in future studies to improve generalizability across diverse populations.

CONCLUSION

In summary, QTc interval prolongation correlates with COPD severity and may serve as a valuable non-invasive marker for cardiac risk assessment in COPD. This study supports the inclusion of QTc monitoring in the standard evaluation of COPD patients, particularly those in advanced stages. Future research should further explore the mechanisms underlying this correlation and assess the clinical benefits of QTc monitoring in mitigating adverse cardiac outcomes in COPD patients.

—-

REFERENCES

1. Aquino, G., Iuliano, E., Cagno, A. D., Vardaro, A., Fiorilli, G., Costanzo, A. D., Calcagno, G., Simone, G. D., & Moffa, S. (2016). Effects of combined training vs aerobic training on cognitive functions in COPD: a randomized controlled trial. *Dove Medical Press*, 711-711. https://doi.org/10.2147/copd.s96663
2. Rw, D. N., Bonadiman, L., Tognella, S., Fp, B., & Turco, P. (2014). Extent and prevalence of cognitive dysfunction in chronic obstructive pulmonary disease, chronic non-obstructive bronchitis, and in asymptomatic smokers, compared to normal reference values. *Dove Medical Press*, 675-675. https://doi.org/10.2147/copd.s63485
3. Kshatriya, R., Paliwal, R. P., Khara, N., Patel, S. L., Varma, J., Ganjiwale, J., & Patel, S. N. (2020). Cognitive Impairment and its Effect on Chronic Obstructive Pulmonary Disease: An Underestimated Phenomenon. *Medknow*, 9(1), 82-87. https://doi.org/10.4103/ijrc.ijrc_45_19
4. Ghanem, M., Makhlouf, H. A., Hasan, A. A., Rashed, H., & Khalifa, H. S. (2019). Bacteriological profile of critically ill patients with chronic obstructive pulmonary disease in respiratory intensive care unit in Assuit University Hospital. *Springer Science+Business Media*, 13(3), 343-348. https://doi.org/10.4103/ejb.ejb_83_18
5. Zhang, Y., Wang, Z., Cao, Y., Zhang, L., Wang, G., Dong, F., Deng, R., Guo, B., Li, Z., Wang, P., Dai, R., Yu, R., Lyu, W., Miao, P., & Su, S. W. (2021). The effect of consecutive ambient air pollution on hospital admissions from chronic obstructive pulmonary disease in the Chengdu region, China. *Springer Science+Business Media*, 14(7), 1049-1061. https://doi.org/10.1007/s11869-021-00998-9
6. Brubaker, P. H., & Kalra, S. (2016). Selected Abstracts From Recent Publications in Cardiopulmonary Disease Prevention and Rehabilitation. *Lippincott Williams & Wilkins*, 36(2), 140-144. https://doi.org/10.1097/hcr.0000000000000175
7. Postema, A. S., & Wilde, A. A. (2018). Determination and Interpretation of the QT Interval: Comprehensive Analysis of a Large Cohort of Long QT Syndrome Patients and Controls. *Circulation*, 138(21), 2187-2200. https://doi.org/10.1161/CIRCULATIONAHA.118.033943
8. Elmir, Y., Himeur, Y., & Amira, A. (2023). ECG classification using Deep CNN and Gramian Angular Field. *IEEE BigDataService*. https://doi.org/10.1109/bigdataservice58306.2023.00026
9. Chu, A., Rajagopal, G., & Sarkar, S. (2021). The missing link: Unlocking the power of cardiac rhythm monitoring device-based QT interval detection. *Wiley*, 45(3), 401-409. https://doi.org/10.1111/pace.14431
10. Wang, N. C. (2008). Clinical Implications of QRS Duration in Patients Hospitalized With Worsening Heart Failure and Reduced Left Ventricular Ejection Fraction. *American Medical Association*, 299(22), 2656. https://doi.org/10.1001/jama.299.22.2656
11. Postema, P G., & Wilde, A A. (2014, May 23). The Measurement of the QT Interval. Bentham Science Publishers, 10(3), 287-294. https://doi.org/10.2174/1573403x10666140514103612
12. Chu, A., Rajagopal, G., & Sarkar, S. (2021, December 29). The missing link: Unlocking the power of cardiac rhythm monitoring device based QT interval detection. Wiley, 45(3), 401-409. https://doi.org/10.1111/pace.14431
13. Haseeb, S., Gül, E E., Çinier, G., Bazoukis, G., Álvarez-García, J., García-Zamora, S., Lee, S., Yeung, C., Liu, T., Tse, G., & Baranchuk, A. (2020, August 6). Value of electrocardiography in coronavirus disease 2019 (COVID-19). Elsevier BV, 62, 39-45. https://doi.org/10.1016/j.jelectrocard.2020.08.007
14. Wang, N C. (2008, June 10). Clinical Implications of QRS Duration in Patients Hospitalized With Worsening Heart Failure and Reduced Left Ventricular Ejection Fraction. American Medical Association, 299(22), 2656-2656. https://doi.org/10.1001/jama.299.22.2656