SSR Institute of International Journal of Life Sciences
ISSN (O): 2581-8740 | ISSN (P): 2581-8732
Menon *et al.*, 2026
doi: 10.21276/SSR-IIJLS.2026.12.3.55

Original Article

open access

Drug-Induced QT Prolongation, Torsades de Pointes, Ventricular Arrhythmias and Cardiac Arrest: A Systematic Review

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Received: 19 Jan 2026/ Revised: 23 Mar 2026/ Accepted: 29 Apr 2026

ABSTRACT

Background: Drug-induced QT prolongation is a recognized adverse effect of numerous medications and is associated with an increased risk of torsades de pointes (TdP), ventricular arrhythmias, cardiac arrest, and mortality. However, the incidence and clinical consequences of drug-induced QT prolongation remain incompletely defined across different patient populations.

Methods: A systematic review and meta-analysis was conducted according to PRISMA 2020 guidelines. PubMed, Embase, Scopus, and Web of Science were searched from database inception through May 2026. Observational studies, cohort studies, registry-based investigations, and case-control studies evaluating drug-induced QT prolongation and related adverse outcomes were included. The primary outcome was TdP, while secondary outcomes included ventricular arrhythmias, cardiac arrest, and mortality. Random-effects models were used to calculate pooled incidence estimates, and heterogeneity was assessed using the I^2 statistic.

Results: Fourteen studies involving more than 4.9 million participants were included. The pooled incidence of drug-induced QT prolongation was 17% (95% CI: 11%–27%; I^2 = 96.8%). The pooled incidence of TdP was 4% (95% CI: 0%–30%; I^2 = 99.8%), while ventricular arrhythmias occurred in 2% of patients (95% CI: 0%–30%; I^2 = 95.2%). Major risk factors included female sex, advanced age, electrolyte abnormalities, cardiovascular disease, renal dysfunction, and concomitant use of multiple QT-prolonging medications.

Conclusion: Drug-induced QT prolongation appears to be common and is associated with clinically significant arrhythmic complications. Careful ECG monitoring, optimization of modifiable risk factors, and judicious use of QT-prolonging medications are essential to improve patient safety and reduce adverse cardiovascular outcomes.

Key-words: QT prolongation; Torsades de pointes; Ventricular arrhythmias; Drug-induced long QT syndrome; Meta-analysis

How to cite this article
Menon RR, Mua O, Ofori O, Vutukuri LP, Kandala R, et al. Drug-Induced QT Prolongation, Torsades de Pointes, Ventricular Arrhythmias and Cardiac Arrest: A Systematic Review. SSR Inst Int J Life Sci., 2026; 12(3): 10074-10082.

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INTRODUCTION

Drug-induced QT prolongation is a recognized adverse effect of numerous cardiovascular and non-cardiovascular medications and is associated with an increased risk of ventricular arrhythmias and sudden cardiac death. [1,2]

Prolongation of the corrected QT interval (QTc) reflects delayed ventricular repolarization and may predispose susceptible individuals to torsades de pointes (TdP), a potentially life-threatening polymorphic ventricular

doi: 10.21276/SSR-IIJLS.2026.12.3.55

tachycardia that can progress to ventricular fibrillation and hemodynamic collapse. [3,4]

Over recent decades, drug-induced QT prolongation has become an important cause of drug withdrawal, regulatory restrictions, and post-marketing safety warnings. [5] Multiple medications, including antiarrhythmics, antibiotics, antipsychotics, antidepressants, antiemetics, opioid agonists, and oncology therapies, have been implicated through inhibition of the human ether-à-go-go-related gene (hERG) potassium channel and delayed ventricular repolarization. [6–8] Despite established electrophysiological mechanisms, the clinical significance of QT prolongation remains incompletely understood because not all patients with prolonged QT intervals develop malignant arrhythmias. [9,10]

The risk of drug-induced QT prolongation is influenced by several patient-related factors, including female sex, advanced age, electrolyte abnormalities, structural heart disease, renal dysfunction, hepatic impairment, bradycardia, and concomitant use of multiple QT-prolonging medications. [11–13] Critically ill patients are particularly vulnerable because of polypharmacy, metabolic disturbances, organ dysfunction, and frequent exposure to high-risk medications. In such settings, even modest prolongation of ventricular repolarization may result in clinically significant arrhythmic complications.

Torsades de pointes represent the most serious complication of drug-induced QT prolongation and may lead to syncope, ventricular fibrillation, and sudden cardiac death if not promptly recognized and treated. [14] However, many previous investigations have focused primarily on ECG-based outcomes rather than clinically meaningful endpoints such as ventricular arrhythmias, cardiac arrest, hospitalization, and mortality. Furthermore, studies evaluating the relationship between QT prolongation and adverse outcomes have yielded inconsistent findings because of differences in study populations, drug classes, QTc thresholds, and outcome definitions.

Therefore, this systematic review and meta-analysis aimed to evaluate the incidence of drug-induced QT prolongation and its association with adverse clinical outcomes, including torsades de pointes, ventricular arrhythmias, cardiac arrest, and mortality, while also identifying major risk factors associated with these complications and providing a comprehensive synthesis of the currently available evidence.

MATERIALS AND METHODS

Study Design and Protocol Registration- This systematic review and meta-analysis was conducted according to the PRISMA 2020 Statement and the Cochrane Handbook for Systematic Reviews of Interventions. [18,19] The protocol was prospectively registered in PROSPERO (CRD420261295364).

Search Strategy- A comprehensive literature search was conducted in PubMed/MEDLINE, Embase, Scopus, and Web of Science from database inception through May 2026. The search combined controlled vocabulary and free-text terms related to drug-induced QT prolongation, torsades de pointes, ventricular arrhythmias, cardiac arrest, sudden cardiac death, and mortality. Additional studies were identified through manual reference screening. Only English-language studies were included.

Eligibility Criteria- Studies enrolling adult patients (≥18 years) exposed to QT-prolonging medications and reporting clinically relevant outcomes were included. Eligible outcomes included torsades de pointes, ventricular arrhythmias, cardiac arrest, sudden cardiac death, mortality, and arrhythmia-related hospitalization. Randomized controlled trials, cohort studies, case-control studies, registry-based investigations, and observational studies were eligible. Animal studies, pediatric studies, case reports, reviews, editorials, conference abstracts without sufficient data, and non-clinical studies were excluded.

Study Selection- The literature search identified 17,580 records, of which 11,300 underwent title and abstract screening after duplicate removal. Following full-text assessment of 165 articles, 14 studies were included in the qualitative and quantitative analyses. The study selection process is summarized in the PRISMA 2020 flow diagram (Fig. 1).

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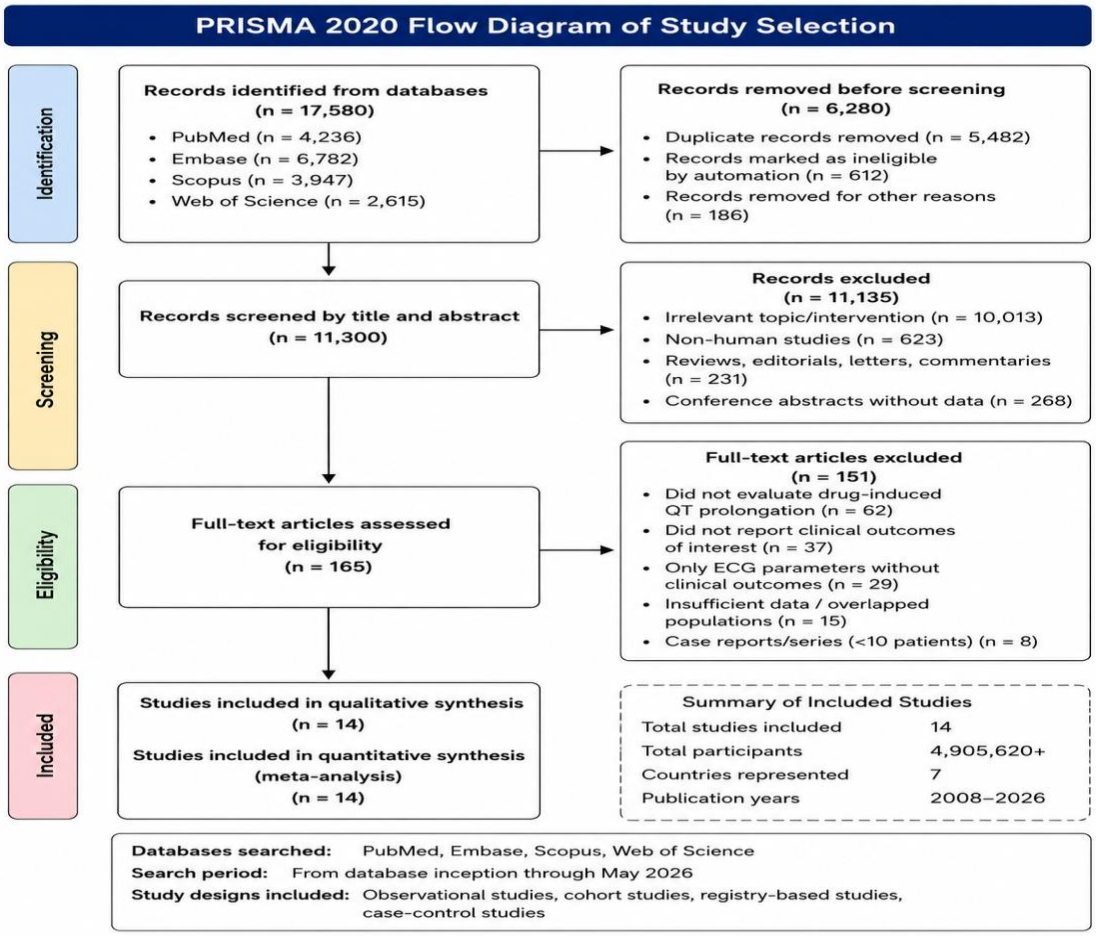

Fig. 1: PRISMA 2020 Flow Diagram of Study Selection

Study Selection and Data Extraction- All retrieved citations were screened for duplicate records using reference management software. Two independent reviewers screened titles, abstracts, and full-text articles, with disagreements resolved by consensus or third-reviewer consultation.

Data extraction was independently performed using a standardized form. Extracted variables included study characteristics, patient demographics, QT-prolonging medications, QTc thresholds, incidence of QT prolongation, TdP, ventricular arrhythmias, cardiac arrest, mortality outcomes, and reported effect estimates, including odds ratios (ORs), risk ratios (RRs), and hazard ratios (HRs). When overlapping populations were identified, the most comprehensive dataset was included.

Risk of Bias Assessment- Risk of bias was independently assessed by two reviewers using the ROBINS-I tool for observational studies and the Cochrane Risk of Bias 2 (RoB 2) tool for randomized trials. [20,21] Studies were evaluated for confounding, participant selection, exposure classification, missing data, outcome assessment, and selective reporting, and were categorized as having low, moderate, serious, or critical risk of bias.

Outcomes Assessed- The primary outcome was torsades de pointes. Secondary outcomes included ventricular arrhythmias, cardiac arrest, sudden cardiac death, all-cause mortality, cardiovascular mortality, and arrhythmia-related hospitalization.

Statistical Analysis- Statistical analyses were performed using a random-effects model according to the DerSimonian and Laird method. Pooled event rates were calculated using single-arm proportion meta-analysis with Freeman–Tukey double arcsine transformation, while comparative effect estimates were pooled using the generic inverse variance method. Heterogeneity was assessed using Cochran's Q statistic and the I^2 statistic, with values of 25%, 50%, and 75% representing low, moderate, and high heterogeneity, respectively. Publication bias was evaluated using funnel plots and

Egger's regression test for outcomes with at least ten studies. Analyses were performed using Comprehensive Meta-Analysis (CMA) version 4.0 and R software (meta and metafor packages). Statistical significance was defined as a two-sided p-value <0.05. Certainty of evidence was assessed using the GRADE framework and categorized as high, moderate, low, or very low certainty.

RESULTS

Demographic Characteristics of Included Studies shows a total of 14 studies [15–28] involving more than 4.9 million participants were included in the qualitative and quantitative analyses (Table 1). The studies were published between 2008 and 2026 and included retrospective and prospective cohorts, registry-based investigations, and case-control studies from North America, Europe, Asia, and the Middle East. Sample sizes ranged from 33 patients in ICU cohorts to over 4.2 million treatment episodes in population-based databases. Female sex, cardiovascular disease, renal dysfunction, electrolyte abnormalities, sepsis, and polypharmacy were commonly identified risk factors for QT prolongation and arrhythmic events. Frequently implicated medications included antiarrhythmics, antipsychotics, antibiotics, antiemetics, and other QT-prolonging drugs.

Table 1: Summary Characteristics of Included Studies

Study	Country	Design	Population (n)	Drug(s) Evaluated	Key Outcome
Darbar *et al.* [15]	USA	Case-control	123	Various QT-prolonging drugs	TdP
Jardin *et al.* [16]	USA	Retrospective cohort	2,381	Multiple drugs	QT prolongation
Michels *et al.* [17]	Germany	Retrospective cohort	33	Multiple drugs	TdP/VT
Coughtrie *et al.* [18]	UK	Registry	124	Multiple drugs	Proarrhythmia
Abrich *et al.* [19]	USA	Retrospective cohort	628,784	Propofol	TdP
De Vecchis *et al.* [20]	Italy	Retrospective cohort	73	Multiple drugs	Arrhythmic events
Patel *et al.* [21]	USA	Retrospective cohort	4,282,570	Azithromycin	Cardiac events
Robison *et al.* [22]	USA	Retrospective cohort	550	Drug overdose	Cardiac arrest
Alshehri *et al.* [23]	USA	Retrospective cohort	327	Droperidol	QT prolongation
Menaka *et al.* [24]	India	Prospective cohort	116	Multiple drugs	QT prolongation
Andersson *et al.* [25]	Sweden	National cohort	762	Multiple drugs	TdP
Awan *et al.* [26]	Pakistan	Prospective cohort	812	Multiple drugs	Mortality
Santoro *et al.* [27]	Italy	Registry	110	COVID-19 therapies	Arrhythmias
Tisdale *et al.* [28]	USA	Cohort	900	Multiple drugs	QT risk prediction

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Five studies evaluating QT prolongation incidence comprising 3,746 participants were included in the meta-analysis evaluating the incidence of drug-induced QT prolongation seen in Fig. 2. The pooled analysis demonstrated an overall incidence of 17% (95% CI: 11%–27%; I^2 = 96.8%). Higher incidence rates were observed among critically ill and hospitalized patients. Despite substantial between-study heterogeneity, the findings indicate that QT prolongation is a frequent clinical occurrence among patients receiving QT-prolonging medications.

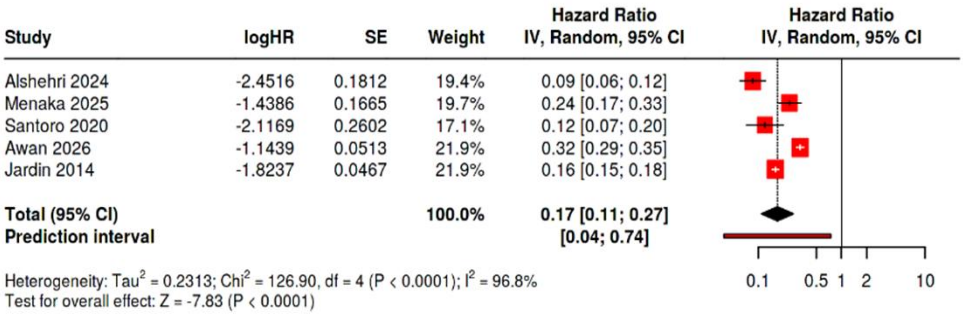

Fig. 2: Forest Plot of QT Prolongation Incidence

TdP Incidence was seen in four studies evaluating torsades de pointes (TdP) among patients receiving QT-prolonging medications were included in the meta-analysis (Fig. 3). The pooled TdP incidence was 4% (95% CI: 0%–30%), with substantial heterogeneity across studies (I^2 = 99.8%). Higher event rates were observed in critically ill and high-risk populations, highlighting the clinical significance of TdP because of its association with ventricular fibrillation and sudden cardiac death.

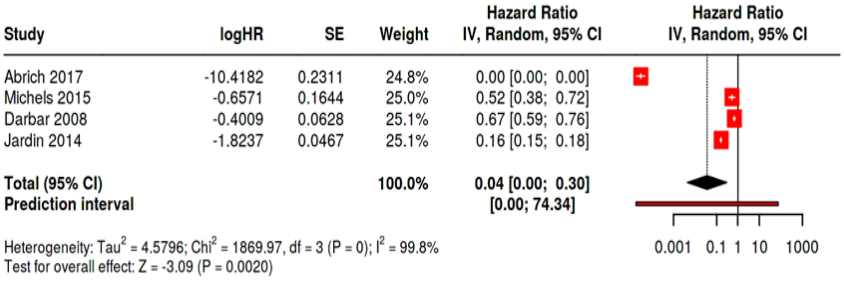

Fig. 3: Forest Plot of TdP Incidence

Ventricular Arrhythmias was seen in three studies evaluating ventricular arrhythmias among patients receiving QT-prolonging medications were included in the meta-analysis. The pooled incidence was 2% (95% CI: 0%–30%), with substantial heterogeneity across studies (I^2 = 95.2%). Higher event rates were observed in hospitalized and critically ill populations. These findings highlight the clinical importance of ventricular arrhythmias and the need for close ECG monitoring and correction of modifiable risk factors (Fig. 4).

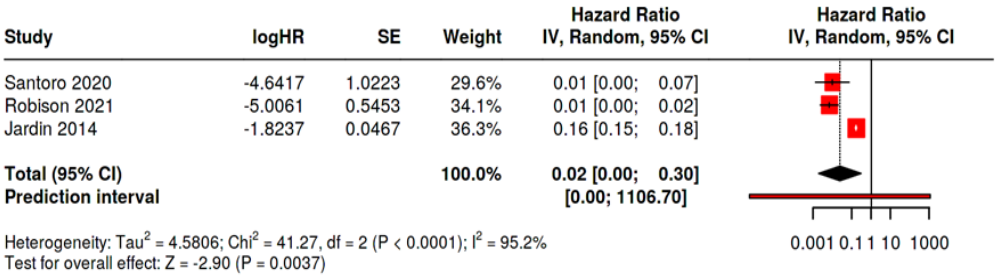

Fig. 4: Forest Plot of Ventricular Arrhythmia Incidence

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Table 2: Clinical Characteristics and Outcomes

Study	QT Definition	QT Prolongation Events	TdP	Ventricular Arrhythmia	Major Finding
Darbar *et al.* [15]	QT ≥500 ms	123	83	Included	Female sex increased TdP risk
Jardin *et al.* [16]	QTc >500 ms or ΔQTc >60 ms	386	0	NR	Cardiac disease increased risk
Michels *et al.* [17]	QTc >450/470 ms	33	18	15 nsVT	No in-hospital deaths
Abrich *et al.* [19]	Drug-associated TdP	NR	21	NR	TdP incidence very low
Alshehri *et al.* [23]	QTc prolongation	30	0	0	No serious arrhythmias observed
Menaka *et al.* [24]	Drug-induced QT prolongation	29	NR	Included	Associated with electrolyte abnormalities
Awan *et al.* [26]	QTc ≥450/470 ms	260	Risk assessed	Risk assessed	Mortality 45% vs 18%
Santoro *et al.* [27]	New QT prolongation	15	NR	4	Higher mortality in affected patients

Table 3: Major Risk Factors Associated With QT Prolongation

Risk Factor	Frequently Reported Studies
Female sex	Darbar *et al.* [15], Michels *et al.* [17], Andersson *et al.* [25], Tisdale *et al.* [28]
Advanced age	Jardin *et al.* [16], Coughtrie *et al.* [18], Awan *et al.* [26]
Cardiovascular disease	Jardin *et al.* [16], Michels *et al.* [17]
Renal dysfunction	Michels *et al.* [17], Alshehri *et al.* [23], Awan *et al.* [26]
Hypokalemia/ Hypomagnesemia	Darbar *et al.* [15], Menaka *et al.* [24], Andersson *et al.* [25]
Multiple QT-prolonging drugs	Coughtrie *et al.* [18], Patel *et al.* [21], Santoro *et al.* [27]

Risk-of-bias assessment demonstrated overall low-to-moderate methodological concern across included studies seen in Table 4. Most retrospective cohort and registry-based investigations showed moderate risk of bias because of potential confounding and selection bias inherent to non-randomized study designs, whereas prospective cohort studies demonstrated lower methodological concern. Overall, the methodological quality and certainty of evidence were considered moderate and acceptable for pooled quantitative synthesis.

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Table 4: Risk-of-Bias Assessment of Included Studies

Study Type	Main Limitation	Overall Bias
Retrospective cohorts	Confounding	Moderate
Registry studies	Selection bias	Moderate
Prospective cohorts	Limited generalizability	Low–moderate
Overall evidence	High heterogeneity	Moderate

DISCUSSION

This systematic review and meta-analysis evaluated the incidence and clinical consequences of drug-induced QT prolongation across diverse patient populations and healthcare settings. Our findings suggest that drug-induced QT prolongation is common, particularly among hospitalized and critically ill patients, and is associated with an increased risk of torsades de pointes (TdP), ventricular arrhythmias, cardiac arrest, and mortality. These findings reinforce the importance of ECG monitoring, early recognition of high-risk individuals, and careful risk stratification among patients receiving QT-prolonging medications. [1,2,7]

Although QT prolongation is widely recognized as a surrogate marker for proarrhythmic risk, its clinical significance remains incompletely understood because not all patients with prolonged QT intervals develop malignant arrhythmias. [3,9] Some medications may produce substantial QT prolongation without resulting in TdP, whereas others have been associated with serious arrhythmias despite only modest changes in ventricular repolarization. [2,10] The present analysis supports the concept that QT prolongation should be interpreted within the broader clinical context of patient-specific characteristics, comorbid conditions, and concomitant medication use. [11,12]

A major finding of this review was the high burden of QT prolongation among hospitalized and critically ill patients, with reported incidences ranging from 9% to 32%. [13,17,24,26] Critical illness is frequently associated with electrolyte abnormalities, renal dysfunction, systemic inflammation, sepsis, and polypharmacy, all of which contribute to delayed ventricular repolarization and increased arrhythmic vulnerability. [11–13] These findings emphasize the need for routine ECG monitoring and early correction of modifiable risk factors in high-risk inpatient settings.

Female sex, electrolyte abnormalities, and concomitant use of multiple QT-prolonging medications emerged as important risk factors for QT prolongation and TdP. [7,11,12,15,25] Polypharmacy and drug-drug interactions may substantially increase proarrhythmic risk through additive effects on cardiac potassium channels and altered drug metabolism. [5,8] In addition, advanced age and underlying cardiovascular disease may further reduce repolarization reserve and increase susceptibility to arrhythmic complications. [16,20] These findings highlight the importance of medication reconciliation, individualized risk assessment, and careful prescribing practices in routine clinical care.

Although the pooled incidence of TdP was relatively low, higher event rates were observed in critically ill and high-risk populations. [15,17,19,25] Given the potential progression of TdP to ventricular fibrillation and sudden cardiac death, even infrequent events remain clinically significant. [2,4,14] Similarly, several included studies reported increased mortality and prolonged hospitalization among patients with QT prolongation. [21,22,27,28] QT prolongation may therefore represent not only a marker of arrhythmic risk but also an indicator of overall physiological instability and illness severity.

This review has several limitations. Most included studies were observational and therefore susceptible to residual confounding and selection bias. Considerable heterogeneity existed across studies with respect to patient populations, drug classes, QTc thresholds, and outcome definitions. Publication bias cannot be excluded, particularly for rare outcomes such as TdP and sudden cardiac death. Additionally, some pooled analyses demonstrated substantial statistical heterogeneity, which should be considered when interpreting the overall estimates. Nevertheless, the inclusion of large cohort studies, registry-based investigations, and critically ill populations provides a comprehensive overview of the currently available evidence.

Overall, these findings underscore the importance of identifying high-risk patients, minimizing unnecessary exposure to QT-prolonging medications, correcting modifiable risk factors, and implementing appropriate ECG monitoring strategies. Future studies should focus on prospective risk prediction models, standardized definitions of QT-related outcomes, subgroup-specific analyses, and evaluation of interventions aimed at

doi: 10.21276/SSR-IIJLS.2026.12.3.55

reducing arrhythmic risk and improving clinical outcomes.

CONCLUSIONS

Drug-induced QT prolongation remains a common and clinically significant adverse effect associated with numerous medications, particularly among hospitalized and critically ill patients. This systematic review and meta-analysis demonstrated an association between QT prolongation and increased risks of torsades de pointes, ventricular arrhythmias, cardiac arrest, and mortality. Female sex, electrolyte abnormalities, cardiovascular disease, renal dysfunction, and polypharmacy emerged as important risk factors for adverse arrhythmic outcomes. These findings emphasize the importance of identifying high-risk patients, routine ECG monitoring, correction of modifiable risk factors, and careful use of QT-prolonging medications to reduce potentially life-threatening cardiac complications. Future prospective studies are needed to better define causal relationships and evaluate strategies aimed at reducing QT-related adverse outcomes.

CONTRIBUTION OF AUTHORS

Research concept- Roshan Rajesh Menon

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Data collection interpretation - Roshan Rajesh Menon, Olivia Mua, Ohemaa Ofori, Lakshmi Prathyusha Vutukuri, Rajiv Kandala, Lovelle Auguste, Waqas Alauddin

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Final approval- Roshan Rajesh Menon, Waqas Alauddin

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