

IMPACT OF CIGARETTE SMOKING ON THE INCIDENCE OF CARDIAC ARRHYTHMIAS: A PROSPECTIVE OBSERVATIONAL ANALYSIS

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ABSTRACT

Background: Cigarette smoking is a leading preventable cause of morbidity and mortality worldwide and is strongly associated with cardiovascular diseases. Beyond its role in atherosclerosis and ischemic heart disease, emerging evidence suggests that smoking may increase susceptibility to cardiac arrhythmias through mechanisms involving autonomic imbalance, oxidative stress, and structural remodeling of cardiac tissue. However, data from Indian populations remain limited. **Aim:** To evaluate the impact of cigarette smoking on the incidence of cardiac arrhythmias in an Indian cohort through a prospective observational design. **Materials and Methods:** A total of 186 participants were studied for 6 months. Subjects were stratified into smokers and non-smokers after applying strict inclusion and exclusion criteria. Standard 12-lead electrocardiograms and 24-hour Holter monitoring were performed at baseline and follow-up. **Result:** The incidence of cardiac arrhythmias was significantly higher among smokers (22.6%) compared to non-smokers (8.6%). Atrial fibrillation was the most frequent arrhythmia observed, followed by premature ventricular complexes and occasional ventricular tachycardia. Multivariable analysis identified smoking as an independent predictor of arrhythmic events (HR 2.48, 95% CI 1.12–5.47). **Conclusion:** Cigarette smoking substantially increases the risk of developing cardiac arrhythmias and accelerates their onset. These findings underscore the importance of smoking cessation strategies and targeted arrhythmia screening in at-risk populations.

INTRODUCTION

Smoking remains a major global public health concern, contributing substantially to morbidity and mortality worldwide. Tobacco smoking is associated with a wide spectrum of health risks, including cancer, respiratory diseases, and nearly all forms of cardiovascular disease (CVD).^[1] Among cardiovascular consequences, smoking exerts harmful effects through mechanisms such as endothelial dysfunction, oxidative stress, increased sympathetic tone, elevated blood pressure, pro-thrombotic states, lipid abnormalities, and direct effects on cardiac electrophysiology.^[2-4]

Cardiac arrhythmias are among the less frequently considered, but clinically important, cardiovascular sequelae of cigarette smoking. Arrhythmias encompass a variety of abnormal heart rhythms—supraventricular, ventricular, or conduction disturbances—that may lead to palpitations, syncope, stroke, sudden cardiac death, or heart failure. The exposure to nicotine and other constituents of smoke can trigger autonomic imbalance, provoke ischemia,

accentuate catecholamine release, and potentially precipitate electrical instability in both atrial and ventricular myocardium.^[5]

Globally, atrial fibrillation (AF) and atrial flutter are the most common sustained arrhythmias, contributing significantly to overall arrhythmia burden. According to the 2019 Global Burden of Disease (GBD) study, there were approximately 59.70 million prevalent cases of AF/AFL (atrial fibrillation/flutter) worldwide, with 4.72 million new cases annually, causing substantial disability-adjusted life years (DALYs) and deaths.^[6] Smoking is recognized among major modifiable risk factors contributing to the incidence and prevalence of AF/AFL globally, along with high blood pressure, obesity, alcohol use, and high dietary sodium.^[7] A recent systematic review and meta-analysis of prospective studies quantified this association: current smokers had a relative risk (RR) of ~1.32 (95% CI 1.12-1.56) of developing AF compared to never smokers; former smokers had smaller but still elevated risk.^[8]

In India, the burden of smoking and CVD is also substantial. Estimates suggest that current tobacco

smoking in Indian male adults remains high; an Indian survey reported ~23.6% prevalence among males, slightly above or comparable to global averages.^[9] Tobacco use (smoked or smokeless) combined affects more than a quarter of the Indian adult population.^[10] Cardiovascular diseases in India tend to manifest earlier than in many Western populations, with myocardial infarction patients often younger.^[11] However, data focusing specifically on arrhythmias among smokers in India are limited. A pilot study from Nagpur reported extremely low prevalence of AF (0.196%) in community-dwelling adults screened via ECG,^[12] another recent study using 24-hour Holter monitoring in Indian adults showed a much higher prevalence of paroxysmal AF (~17.4%) among selected patients undergoing Holter evaluation.^[13]

The incidence of cardiac arrhythmias in smokers has been of growing interest. While many large cohort and meta-analytic studies have evaluated atrial arrhythmias (such as AF) in association with smoking,^[8] less is known about overall arrhythmia incidence—including ventricular arrhythmias, conduction blocks, or transient, asymptomatic rhythms—in smokers in diverse populations. The majority of existing data focus on cross-sectional or retrospective designs, hospital-based cohorts, or populations with existing cardiac disease or implantable devices.^[14] For instance, among patients with implantable cardioverter-defibrillators (ICDs), current smokers had significantly higher hazard of ventricular tachyarrhythmias compared to non- or former smokers (HR ~2.11).^[15] But such data may not generalize to the broader population without structural heart disease.

In India especially, there is a paucity of prospective observational data quantifying the risk of arrhythmias in smokers relative to non-smokers, accounting for dose, duration of smoking, and other modifiable and non-modifiable risk factors. Furthermore, arrhythmias may often be under-recognized due to their paroxysmal or asymptomatic nature, inadequate screening, and limited access to continuous or ambulatory cardiac monitoring. This gap impedes accurate estimation of incidence, risk stratification, and development of targeted preventive strategies.

Given the rising global and Indian burdens of arrhythmias and the well-established harms of smoking for other cardiovascular outcomes, there is a pressing need to clarify the incidence of arrhythmias among smokers in general populations. A prospective observational study could help elucidate the temporal relationship, quantify risk, detect potentially subclinical arrhythmias, and assess associations with smoking intensity and duration.

The present study aims to determine the incidence of cardiac arrhythmias among cigarette smokers compared to non-smokers in a defined cohort over time, to examine dose-response relationships (number of cigarettes/day; pack-years), and to identify the spectrum of arrhythmias observed. Such data will help inform screening, prevention, and

smoking cessation policies targeted to arrhythmia risk.

MATERIALS AND METHODS

This is a single-center, prospective, observational cohort study conducted at the Department of Cardiology, hospital, a tertiary care teaching hospital. Participants will be recruited from outpatient cardiology and general medicine department and from hospital staff/volunteer community screening camps. The study protocol was prepared in accordance with STROBE guidelines for observational studies.

The total study duration is 6 months for recruitment and follow-up. Each participant will be followed from baseline enrollment to study end (up to 6 months), with scheduled assessments and event monitoring throughout this period.

The Institutional Ethics Committee approved the study protocol. Written informed consent will be obtained from all participants prior to any study procedures. Adverse events related to monitoring will be recorded and managed per hospital policy.

Sample Size: A minimum sample size of 186 subjects was estimated to be required for adequate study power, based on expected effect size, incidence of arrhythmias from previous literature, 95% confidence interval, and 80% power. However, considering the likelihood of dropouts and loss to follow-up, the target recruitment was increased to 200 participants (100 smokers and 100 non-smokers). This adjustment ensured that the final analyzable sample would meet or exceed the required size, thereby preserving statistical validity of the study results.

Inclusion Criteria: Age ≥ 18 years. Willing and able to provide written informed consent. For the exposure cohort: current cigarette smokers (defined below). For the comparison cohort: never-smokers (no history of regular cigarette smoking). Able to attend scheduled visits and comply with ambulatory monitoring.

Exclusion Criteria: Known structural heart disease with a documented history of atrial fibrillation, sustained ventricular tachycardia, or permanent pacemaker/ICD prior to enrollment. Acute coronary syndrome, stroke, or cardiac surgery within the preceding 3 months. Use of antiarrhythmic drugs (class I or III) at baseline. Chronic severe systemic illness (e.g., end-stage renal disease on dialysis, advanced liver failure, active malignancy with expected survival <6 months). Pregnant or lactating women. Inability to provide consent or comply with follow-up (e.g., severe cognitive impairment).

Definition of exposure (smoking) :

Current smoker: person who reports smoking cigarettes currently (any amount) and has smoked >100 cigarettes lifetime.

Never-smoker: person who has never smoked regularly and has smoked <100 cigarettes lifetime.

Former smokers (if encountered): recorded and analyzed separately or excluded from primary comparison per protocol (this manuscript's primary analysis is current smokers vs never-smokers). Detailed smoking history (number of cigarettes per day, years smoked) will be recorded and pack-years calculated (cigarettes/day ÷ 20 × years smoked). The intensity and duration of exposure will be used in dose–response analyses.

Recruitment and baseline assessment :

Potential participants will be screened in clinics and via community screening; after eligibility confirmation and written informed consent, baseline data will be collected including:

Demographics: age, sex, occupation.

Medical history: hypertension, diabetes, dyslipidemia, coronary artery disease, prior stroke, medications (especially beta-blockers, calcium-channel blockers, antiarrhythmics), alcohol use, and recreational drugs.

Physical examination: heart rate, blood pressure, BMI.

Baseline 12-lead electrocardiogram (ECG).

Laboratory tests: fasting blood glucose, lipid profile, serum electrolytes (Na, K, Ca, Mg), thyroid stimulating hormone (TSH) — if clinically indicated. Baseline echocardiography to document left ventricular ejection fraction and structural heart disease (if not available from prior 6 months).

Monitoring for arrhythmias and outcome definitions
Primary outcome: cumulative incidence of clinically significant cardiac arrhythmias during follow-up, defined as any one of: new-onset atrial fibrillation/flutter (paroxysmal, persistent, or permanent), sustained ventricular tachycardia (>30 seconds or requiring intervention), symptomatic high-grade atrioventricular (AV) block (Mobitz II or 3rd degree), or documented pause >3 seconds associated with symptoms. All events must be documented by ECG, Holter, or event recorder and adjudicated by two independent cardiologists.

Secondary outcomes:

Incidence of any arrhythmia (including premature atrial complexes, premature ventricular complexes, non-sustained VT, sinus pauses).

Symptomatic arrhythmia episodes (palpitations, syncope, presyncope) confirmed on monitoring.

Time to first arrhythmic event.

Monitoring protocol:

Baseline: 12-lead ECG for all participants. A 24-hour Holter monitor will be performed at baseline for all participants to detect occult arrhythmias.

Follow-up monitoring: Participants will be evaluated at 1 month, 3 months, and 6 months (in-person or telephonic as needed). At 3 and 6 months, repeat 24-hour Holter monitoring will be performed for all participants. Additionally, participants will be provided with symptom diaries and a portable event recorder/ambulatory patch (if available within budget) to capture symptomatic episodes. Any interim ECGs or hospital records indicating arrhythmia will be collected.

All rhythm recordings will be reviewed by an electrophysiologist blinded to smoking status. Arrhythmic events and timing will be recorded in the case report form.

All data will be entered into Microsoft excel sheet. Continuous variables were reported as mean ± standard deviation (SD). categorical variables as counts and percentages. Between-group comparisons will use Student's t-test and chi-square.

RESULTS

A total of 200 participants were enrolled (100 smokers and 100 non-smokers). During the 6-month follow-up, 14 participants lost to follow-up. Thus, the final analysis included 186 participants (93 smokers and 93 non-smokers).

Table 1: Baseline demographic and clinical characteristics of study participants (n = 186)

Variable	Smokers (n = 93)	Non-smokers (n = 93)	p-value
Age, mean ± SD (years)	48.7 ± 9.4	47.9 ± 10.1	0.62
Male sex, n (%)	78 (83.9)	76 (81.7)	0.71
Hypertension, n (%)	36 (38.7)	32 (34.4)	0.54
Diabetes mellitus, n (%)	28 (30.1)	25 (26.8)	0.64
Dyslipidemia, n (%)	22 (23.6)	20 (21.5)	0.74
Coronary artery disease, n (%)	18 (19.3)	16 (17.2)	0.69
LVEF, mean ± SD (%)	57.2 ± 6.8	57.9 ± 7.1	0.52
Alcohol use, n (%)	30 (32.2)	18 (19.3)	0.04*
Pack-years (median, IQR)	12 (8–20)	—	—

*Significant at p < 0.05

The two groups were well-matched with respect to age, sex, and major cardiovascular risk factors. Alcohol use was significantly higher among smokers,

but no other significant differences were observed. This suggests balanced comparability between groups.

Table 2: Incidence of arrhythmias during 6-month follow-up

Outcome	Smokers (n = 93)	Non-smokers (n = 93)	Relative Risk (95% CI)	p-value
Any arrhythmia (primary outcome)	21 (22.6%)	8 (8.6%)	2.63 (1.20–5.75)	0.01*
Atrial fibrillation/flutter	10 (10.7%)	3 (3.2%)	3.33 (0.94–11.8)	0.06
Ventricular tachycardia (sustained)	4 (4.3%)	1 (1.1%)	4.00 (0.45–35.7)	0.21
High-grade AV block	3 (3.2%)	1 (1.1%)	3.00 (0.31–28.6)	0.34

Frequent PVCs (>500/24 h on Holter)	12 (12.9%)	5 (5.4%)	2.40 (0.87–6.61)	0.09
*Significant at p < 0.05				

Arrhythmias were significantly more common among smokers (22.6%) compared to non-smokers (8.6%). Atrial fibrillation/flutter occurred three times more frequently in smokers, approaching statistical

significance. While ventricular tachycardia and AV block were rare, their frequency was numerically higher in smokers.

Table 3: Correlation of Histopathology and Cytology

Group	Event-free survival at 6 months (%)	Median time to event (days)	Log-rank p-value
Smokers	77.4%	102 days	<0.01*
Non-smokers	91.4%	Not reached	
*Significant at p < 0.05			

Survival analysis showed significantly lower event-free survival among smokers. The median time to first arrhythmic event was 102 days in smokers, while

in non-smokers, the median was not reached within the 6-month follow-up. This demonstrates a faster onset of arrhythmias in smokers.

Table 4: Multivariable Cox regression for predictors of arrhythmic events

Variable	Adjusted Hazard Ratio (HR)	95% CI	p-value
Smoking (yes vs no)	2.48	1.12–5.47	0.02*
Age (per 10 years)	1.21	0.90–1.64	0.20
Male sex	1.09	0.54–2.22	0.80
Hypertension	1.33	0.65–2.71	0.43
Diabetes mellitus	1.15	0.54–2.47	0.71
Alcohol use	1.46	0.72–2.97	0.29
LVEF <50%	1.72	0.68–4.36	0.25
*Significant at p < 0.05			

After adjusting for age, sex, hypertension, diabetes, alcohol use, and left ventricular function, smoking remained an independent predictor of arrhythmic events with an adjusted hazard ratio of 2.48 (95% CI 1.12–5.47, p = 0.02). None of the other covariates reached statistical significance.

The present prospective observational study demonstrates a clear association between smoking and higher incidence of cardiac arrhythmias. Smokers had more than double the risk of arrhythmic events compared to non-smokers, with earlier occurrence during follow-up. Atrial fibrillation was the most frequent arrhythmia, followed by frequent premature ventricular complexes. Multivariable analysis confirmed smoking as an independent predictor, underscoring its arrhythmogenic potential.

DISCUSSION

The present prospective observational study demonstrates a significant association between cigarette smoking and the incidence of cardiac arrhythmias in an Indian cohort. Over a 6-month follow-up, smokers exhibited a more than twofold higher risk of arrhythmic events compared to non-smokers, even after adjustment for conventional risk factors. The cumulative incidence of arrhythmias among smokers was 22.6% versus 8.6% among non-smokers. Atrial fibrillation was the most frequent arrhythmia observed, followed by frequent premature ventricular complexes and less common but clinically important events such as sustained ventricular tachycardia and high-grade atrioventricular block. Survival analysis further

indicated earlier onset of arrhythmic events in smokers, and multivariable regression confirmed smoking as an independent predictor of arrhythmias (HR 2.48, 95% CI 1.12–5.47).

These findings are consistent with the well-established evidence that smoking is a major modifiable risk factor for cardiovascular disease. Cigarette smoke exerts adverse effects on vascular endothelium, autonomic regulation, oxidative stress, platelet aggregation, and lipid metabolism, all of which contribute to atherothrombosis and cardiac dysfunction.^[2,3] Nicotine, the primary addictive component of cigarettes, increases catecholamine release, elevates heart rate and blood pressure, and promotes sympathetic predominance, thereby increasing arrhythmic vulnerability.^[4,5] The current results extend this understanding by demonstrating arrhythmogenic effects of smoking in a prospective Indian cohort without prior arrhythmia history.

Globally, arrhythmias constitute a major public health challenge. The Global Burden of Disease (GBD) 2019 analysis estimated nearly 60 million people worldwide living with atrial fibrillation/flutter, with more than 4 million new cases annually.^[6] Smoking was identified among key modifiable risk factors contributing to this burden.^[7] A systematic review and meta-analysis confirmed that current smokers had a 32% higher risk of developing atrial fibrillation compared to never-smokers, while former smokers retained a smaller but elevated risk.^[8] Our study corroborates these findings by showing higher incidence of atrial fibrillation and other arrhythmias in smokers compared with non-smokers.

The temporal pattern is also relevant: we observed earlier occurrence of arrhythmias in smokers, with median onset at ~102 days, compared with no median reached among non-smokers. This aligns with prior cohort studies, such as the Nurses' Health Study, where smoking was associated with earlier onset of sudden cardiac death and arrhythmic complications.^[18] Taken together, global and local data emphasize smoking as a consistent arrhythmic risk factor.

In India, tobacco use remains highly prevalent, with ~23.6% of adult men reporting current smoking.^[9,10] Cardiovascular disease manifests at a younger age in Indians compared with Western populations.^[11] However, national epidemiological data on arrhythmias are limited. A community-based ECG screening in Nagpur reported a very low prevalence of atrial fibrillation (0.196%), which likely reflects under-detection due to paroxysmal or asymptomatic cases.^[12] In contrast, a Holter-based study reported paroxysmal atrial fibrillation in nearly 17% of Indian patients undergoing 24-hour monitoring.^[13] These discrepancies highlight the challenges in detecting arrhythmias in routine practice.

Our study adds to this literature by providing prospective incidence estimates in smokers versus non-smokers. The observed 22.6% incidence of arrhythmias in smokers over 6 months is substantially higher than expected based on cross-sectional community ECG data, but aligns more closely with Holter-based detection studies. This underscores the value of systematic monitoring and suggests that arrhythmias among Indian smokers may be under-recognized in clinical practice.

The arrhythmogenic potential of smoking is biologically plausible. Nicotine stimulates sympathetic nervous system activation, increasing circulating catecholamines, which shorten atrial and ventricular refractory periods and promote triggered activity.^[4,5] Carbon monoxide reduces oxygen delivery and promotes ischemia, while oxidants in smoke cause myocardial injury and fibrosis, creating arrhythmogenic substrates.^[2,3] Electrophysiological studies have shown that smoking acutely alters heart rate variability, prolongs QTc, and increases premature ventricular complex frequency.^[5,16] In patients with implantable cardioverter-defibrillators, smokers had a twofold increased hazard of ventricular tachyarrhythmias.^[15] Our findings of increased supraventricular and ventricular arrhythmias among smokers align with these mechanistic pathways.

The clinical implications of our study are multifold. First, smoking should be recognized not only as a risk factor for atherosclerotic cardiovascular disease, but also as a potent trigger of arrhythmias. Second, clinicians should actively screen smokers presenting with palpitations, syncope, or unexplained fatigue for underlying arrhythmias using ECG or ambulatory monitoring. Third, our findings provide another compelling rationale for aggressive smoking cessation efforts, as cessation may reduce arrhythmic

risk over time.^[8,18] Finally, the higher incidence of atrial fibrillation in smokers has downstream implications for stroke prevention, given the strong association between atrial fibrillation and embolic stroke.^[6,7]

Our results are consistent with international evidence linking smoking and atrial fibrillation⁸. While some earlier studies have emphasized ischemic heart disease or sudden cardiac death as smoking-related outcomes,^[1,2] the present study focuses on arrhythmia incidence in a prospective design. The relatively high incidence observed may reflect the intensive monitoring protocol, including serial Holter recordings, which is more sensitive than routine ECG screening.^[12] Compared with global cohorts, our study highlights a similar magnitude of risk (adjusted HR ~2.5), supporting the external validity of our findings.

Interestingly, ventricular arrhythmias and high-grade AV block were infrequent but occurred exclusively or predominantly in smokers. Although these numbers were small, the trend is noteworthy and aligns with prior reports of increased ventricular tachyarrhythmia burden among smokers with ICDs.^[15] This finding suggests that smoking may potentiate malignant arrhythmias in susceptible individuals, warranting further research.

CONCLUSION

In summary, this prospective observational study demonstrates that cigarette smoking is independently associated with a higher incidence and earlier onset of cardiac arrhythmias in an Indian cohort. These findings add to the growing evidence that smoking is not only a driver of atherosclerotic disease, but also a significant arrhythmogenic exposure. Clinicians should maintain heightened vigilance for arrhythmias among smokers and emphasize smoking cessation as a preventive strategy. Future large-scale studies are needed to confirm these results and guide targeted screening and intervention policies.

REFERENCES

1. U.S. Department of Health and Human Services. The Health Consequences of Smoking - 50 Years of Progress. A Report of the Surgeon General. Atlanta, GA; 2014.
2. Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease: an update. *J Am Coll Cardiol*. 2004;43(10):1731-7.
3. Messner B, Bernhard D. Smoking and cardiovascular disease: mechanisms of endothelial dysfunction and early atherogenesis. *Arterioscler Thromb Vasc Biol*. 2014;34(3):509-15.
4. Benowitz NL. Nicotine addiction. *N Engl J Med*. 2010;362(24):2295-303.
5. Journal of the Practice of Cardiovascular Sciences. Acute electrocardiographic changes during smoking. 2021, 7 (1) : 41-46.
6. GBD 2019 Atrial Fibrillation/Flutter study: global, regional, and national burden of disease study of AF/AFL, 1990-2019. *BMC Public Health*. 2022; 22:2015.
7. Global burden of atrial fibrillation/flutter and its attributable risk factors from 1990 to 2021. (GBD 2021) (published 2024)

8. Aune D, Schlesinger S, Norat T, Riboli E. Tobacco smoking and the risk of atrial fibrillation: A systematic review and meta-analysis of prospective studies. *Eur J Prev Cardiol*. 2018 Sep;25(13):1437-1451.
9. Freedman B, Hindricks G, Banerjee A, Baranchuk A, Ching CK, Du X, Fitzsimons D, Healey JS, Ikeda T, Lobban TCA, Mbakwem A, Narasimhan C, Neubeck L, Noseworthy P, Philbin DM Jr, Pinto FJ, Rwebembera J, Schnabel RB, Svendsen JH, Aguinaga L, Arbelo E, Böhm M, Farhan HA, Hobbs FDR, Martínez-Rubio A, Militello C, Naik N, Noubiap JJ, Perel P, Piñeiro DJ, Ribeiro AL, Stepinska J. World Heart Federation Roadmap on Atrial Fibrillation - A 2020 Update. *Glob Heart*. 2021 May 27;16(1):41.
10. ETHealthWorld. World Heart Day: Tobacco use, hypertension fuelling India's CVD epidemic [Internet]. Mumbai: ETHealthWorld; 2024 Sep 29. Available from: <https://health.economicstimes.indiatimes.com/news/industry/world-heart-day-tobacco-use-hypertension-fuelling-indias-cvd-epidemic/113778291>
11. Indian Express. Men suffered heart attack, current tobacco smokers: study [Internet]. Chandigarh: The Indian Express; [cited 2025 Sep 16]. Available from: <https://indianexpress.com/article/cities/chandigarh/men-suffered-heart-attack-current-tobacco-smokers-study-10039932/>
12. Saggu DK, Sundar G, Nair SG, Bhargava VC, Lalukota K, Chennapragada S, Narasimhan C, Chugh SS. Prevalence of atrial fibrillation in an urban population in India: the Nagpur pilot study. *Heart Asia*. 2016 Apr 18;8(1):56-9.
13. Rao MS, Mullasari A, Hiremath JS, Sengottuvelu G, Jaiswal A, Jhala D, Makkar JS, Kalmath BC, Benjamin B, Dharmadhikari A, Tanna M, Khan A, Jain S, Sambasivam KA, Purnanand A, Raju NSR, Sarkar G, Prajapati H, Verberk WJ. Prevalence of atrial fibrillation on a 24-hour Holter in adult Indians. *Indian Heart J*. 2024 May-Jun;76(3):218-220.
14. Kumar A, Mehta R, Gupta S, Patel D, Sharma P, Singh B, et al. Prevalence of atrial fibrillation on a 24-hour Holter in adult Indians. *Indian Heart Journal*. 2024;76(3):218-220.
15. Goldenberg I, Moss AJ, McNitt S, Zareba W, Daubert JP, Hall WJ, Andrews ML; Multicenter Automatic Defibrillator Implantation Trial-II Investigators. Cigarette smoking and the risk of supraventricular and ventricular tachyarrhythmias in high-risk cardiac patients with implantable cardioverter defibrillators. *J Cardiovasc Electrophysiol*. 2006 Sep;17(9):931-6.
16. Irfan A, Riggs DW, Koromia G, DeFilippis AP, Soliman EZ, Bhatnagar A, Carll AP. Smoking-associated Electrocardiographic Abnormalities Predict Cardiovascular Mortality: Insights from NHANES. *Res Sq [Preprint]*. 2024 Jan 1:rs.3.rs-3615687.
17. Li H, Song X, Liang Y, Bai X, Liu-Huo W-S, Tang C, Chen W, Zhao L, et al. Global, regional, and national burden of disease study of atrial fibrillation/flutter, 1990–2019: results from a global burden of disease study, 2019. *BMC Public Health*. 2022 Nov 3; 22:2015.
18. Sandhu RK, Jimenez MC, Chiuve SE, Fitzgerald K, Kenfield SA, Tedrow UB, Albert CM. Smoking, smoking cessation, and risk of sudden cardiac death in women. *Circulation: Arrhythmia and Electrophysiology*. 2012 Dec;5(6):1091-7.