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Original Article

Tobacco Exposure and its Possible Electrocardiographic Changes in Female Beedi Rollers in Tirunelveli District

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Abstract

Introduction: Around 5 million female beedi workers in India are rolling beedi for 6–12 h and handling 225–450 g of beedi tobacco/day in a poor household environment. Beedi tobacco dust gets absorbed through the cutaneous and pharyngeal route. Nicotine, the main component, is a potent cardiac myocyte, A-type potassium channel inhibitor. Nicotine, a potent arrhythmogenic agent, causes electrophysiological disturbances such as shortened QTc and prolonged T-wave end (Tp-e) interval, by prolonging ventricular repolarization time. Nicotine accelerates atherogenesis also. Very few Indian studies are available to highlight this aspect.

Aim: This study aims to observe the electrocardiogram (ECG) changes in female beedi rollers in Tirunelveli district.

Materials and Methods: The study population consisted of 35 female beedi rollers, aged between 20 and 50 years, rolling beedi for at least 5 years. Blood pressure and ECGs were performed on all subjects following standard operating procedure. Heart rate, QTc, and Tp-e intervals were measured.

Results: The mean age of beedi rollers is 45.75 ± 7.1 years with mean years of exposure of 19.70 ± 8.05 years rolling 22.8 ± 8.54 beedi bundles/day. There is no correlation found in systolic and diastolic pressure. Mean QTc interval was 432 ± 23.6 ms, mean Tp-e interval is 79.47 ± 3.2 ms; there is no correlation in the duration of beedi rolling with ECG parameters.

Conclusion: Beedi tobacco (nicotine) exposure is associated with risk of accelerated atherogenesis and arrhythmogenesis, but in our study, beedi rollers are not showing changes in ECG.

Key words: Arrhythmias, Atherogenesis, Female beedi roller, Nicotine, Tobacco dust

INTRODUCTION

Beedi, also called as poor man's cigarette, is made of 0.2–0.3 g of tobacco flake wrapped in a tendu leaf (Diospyros melanoxylon) or *Piliostigma racemosum* leaf and secured with colored thread or adhesive at both the ends.[1] It is a thin South Asian cigarette and accounts for over half of the tobacco consumed in India. A beedi is a cheap form of tobacco consumption and it is manufactured traditionally in home-based small-scale industries among the non-affluent groups.[2] Beedi rolling ranks as the top employer in India that employs poor women to hand roll beedis which helps them to earn a meager but crucial sustenance level income. About 4.16 million workers, especially women and children, are employed in this area, but the actual estimate could be over 10 million.[3,4] Tribals from ancient India made tobacco in a pipe made from leaves of trees that gradually developed the practice of wrapping tobacco in a leaf. Beedi smoking has been reported as early as 1711.

Beedis were first introduced in the east coast of India in the early 18th century and sold in bundles of 20–30. According to a report of the Labour Investigation Committee appointed by the Government of India in 1946, merchants from Gujarat introduced beedi manufacture in Madya Pradesh in the 1st decade of the 20th century. According to the Court of Inquiry into Labour Conditions

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in Beedi report published by the Government of Madras in 1947, the oldest beedi-making unit was established as early as 1887. This agrees with the International Labour Organization's estimate that the Indian beedi industry started around 1900.[5]

The beedi industry poses health risks to beedi smokers and the non-smokers who are involved in the beedi manufacturing.[6] Studies state that beedi workers are predisposed to respiratory, dermatological, ophthalmic, neurological, cardiac, and podiatric problems.[7-9] Toxic constituents such as nicotine, nitrosamines, polycyclic aromatic hydrocarbons, formaldehyde, and hydrogen are present in tobacco and these toxins are released into the ambient air during beedi processing and rolling. The inspirable dust in tobacco factory is 150-fold higher than non-factory settings.[10] The nicotine concentration in the tobacco of beedi (21.2 mg/g) is significantly higher than that of commercial filtered (16.3 mg/g) and unfiltered cigarettes (13.5 mg/g) and can be absorbed through the skin, respiratory epithelium, and oral mucous membrane.[11] High levels of tobacco constituents such as cotinine, thioether, promutagen, and direct-acting mutagens have been identified in beedi worker's urine that indicates the increased systemic exposure to tobacco. This resulted in increased chromosomal abnormalities and elevated mutagenic burden among the beedi processors as demonstrated by cytogenetic analysis.[12,13]

The chief complication associated with smoking nicotine is cardiac arrhythmias and cardiac arrest apart from respiratory complications. While in beedi rollers (nonsmokers), nicotine dust is absorbed through the skin and also inhaled and can cause complications similar to a typical smoker in the long run.

Aim

This study aims to observe the electrocardiogram (ECG) changes in female beedi rollers in Tirunelveli district.

MATERIALS AND METHODS

This observational study consisted of 35 female beedi rollers, aged between 20 and 50 years, rolling beedi for at least 5 years. It was ensured that they had never smoked before and they did not have any other pre-existing illness. Beedi rollers who had congenital heart disease, diabetes mellitus, hypertension, and abnormal thyroid tests or who were under other medications such as azithromycin and diphenhydramine were excluded from the study. Proper medical history was obtained and blood pressure (BP) and ECGs were performed on all subjects following standard operating procedure. BP was measured in triplicate after a 10 min seated rest using a mercury sphygmomanometer and appropriate cuff size. Adolescents who had any presenting cardiac disorder in ECHO were also excluded. Heart rate, QTc, and T-wave end (Tp-e) intervals were measured. ECG recorder set at 25 mm/s paper speed and 10 mm/mV voltage was used. All ECGs were recorded when the participants were in the supine position and at

Table 1: Distribution of patients characteristics

Variables	Mean±SD	Minimum	Maximum
Age	45.75±8.42	30	63
Height	148.71±5.25	137	160
Weight	58.59±12.26	35	95
BMI	26.47±5.33	16	44.57
Years	19.71±9.82	3	40
Rolling bundles	22.88±7.85	10	44
Pulse	80.97±11.81	65	115
SBP	127.94±13.88	100	150
DBP	82.06±10.67	60	100
QTc (ms)	434.26±24.53	401	492

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, SD: Standard deviation

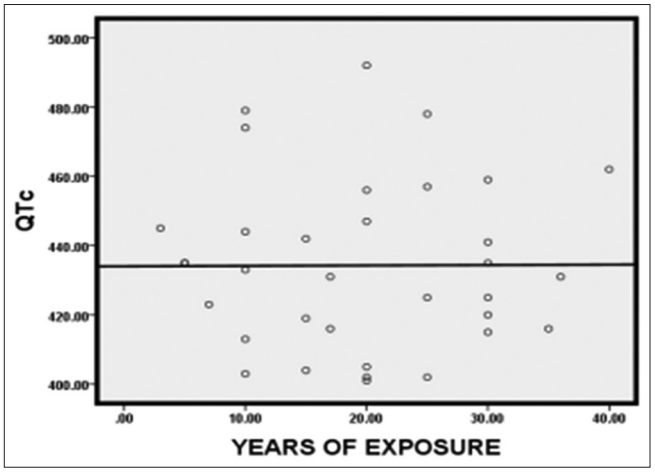

Figure 1: Correlation of QTc and years of exposure

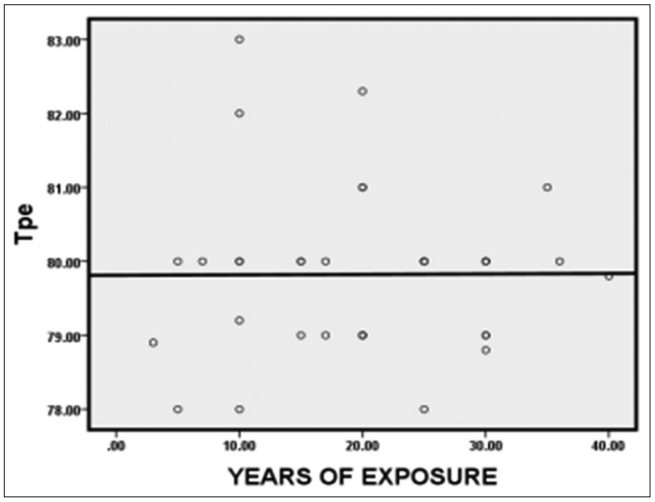

Figure 2: Correlation of T-wave end and years of exposure

rest. Measurements were taken digitally by a cardiologist who was blinded to the actual status of the participants to reduce interobserver variability.

The PR interval was measured from the first visible upward of the P wave to the QRS complex starting point. The QT interval was defined from the beginning of the QRS complex to the end of the T wave, and the Tp-e interval, from the peak of T wave to the point it returns to baseline. This point could also be described as the intersection of the tangent to the slope of the T wave and isoelectric line. At least three P waves, T waves, PR intervals, and QRS-T complexes were evaluated in each derivation and the mean periods of PR, QT, and Tp-e were calculated as milliseconds. The QT intervals were corrected according to Bazett formula, where ($QTc = QT/\sqrt{RR}$). The Tp-e/QT and Tp-e/QTc ratios were calculated based on these measurements. Rhythm, speed, QRS axis, ventricular hypertrophy, and ST-segment and T-wave changes in ECGs were also evaluated. Heart rate was measured in beats/minute. The lipid profile and hematological parameters were also studied.

RESULTS

The mean age of beedi rollers in this study is 45.75 ± 7.1 years with mean years of exposure of 19.70 ± 8.05 years to nicotine dust. They rolled approximately 22.8 ± 8.54 beedi bundles/ day. There is no correlation found in systolic and diastolic pressure and it seemed to be normal. The mean QTc interval was 432 ± 23.6 ms (normal is 400–440 ms), mean Tp-e interval is 79.47 ± 3.2 ms, and this study shows that there is no correlation in the duration of beedi rolling with ECG parameters [Figures 1 and 2]. The heart rates were also within normal limits (68 ± 5 beats/min). Although there may be multiple occupational hazards in female beedi rollers in long-term beedi rolling, this study shows that the cardiac and ECG parameters are seemingly normal when compared to the smokers [Table 1].

A highly significant increase in total cholesterol, low-density lipoprotein (LDL) cholesterol, and triglyceride levels with a concomitant decrease in high-density lipoprotein (HDL) cholesterol level in women beedi rollers was observed when compared with non-beedi rollers. Atherogenic index of plasma was also significantly higher in women beedi rollers when compared with non-beedi roller women. Hematological parameters such as hemoglobin, red blood cells count, white blood cells count, and platelet count were also found to be decreased in women beedi rollers as compared with non-beedi roller women. An altered lipid profile increased atherogenic index and lower hematological parameters in women beedi rollers could be contributory toward the development of cardiovascular disease in them.

DISCUSSION

Beedi making started in India as early as 1887 and it has become so popular in the present scenario that it is the second biggest cottage industry which offers huge potential for employment, next to agriculture. The per capita income is low in backward economics where agriculture is the main occupation of the people.[14] This is why people, especially women easily shift their work to beedi making. India accounts for nearly 85% of the world production of beedies. As beedi leaves and tobacco are the chief raw materials involved in beedi making, substantial revenue is earned by the forest department of various states such as Madhya Pradesh and Andhra Pradesh where these leaves grow. Tobacco is not a native crop. It was brought to India by the Portuguese for their "Hooka" in 1508. Beedies were first manufactured in India in 1902 in Madhya Pradesh.[15] Tendu leaves and the inferior kothra leaves are available for beedi making. The quality of the leaves to certain extent influences the quality of the product. The other vital ingredients in the beedi are the tobacco which gives different taste to the smokers. It is the tobacco that helps the producers to differentiate his product from the products of other manufacturers. Flavors are added with the tobacco to give a different taste to it. There are two types of tobacco available: chewing tobacco and smoking tobacco.

Beedi industry has a history of 95 years in Tirunelveli district. Although agriculture is their chief occupation, it is dependent on rainfall and labor availability is seasonal. The next non-farming alternative to agriculture is beedi rolling and women and children are involved in large amounts in its manufacturing. Even though the female beedi rollers do no smoke or chew tobacco, they are exposed to the toxic substance called nicotine for prolonged periods of time which is absorbed through their skin and mucous membrane into the systemic circulation and also aspirated by nasal routes which can cause serious respiratory illness in them. Many studies have favored the occurrence of occupational health hazards in beedi rollers such as asthma, rhinitis, toxic optic neuropathy, burning eyes, knee and joint problems, and even skin cancer. However, there are very few or no studies that experimented the association of cardiovascular diseases in female beedi rollers. This is the background of this study and it was conducted in the beedi rolling community of Tirunelveli district among 35 female beedi rollers aged between 20 and 50 years.

The acute cardiovascular effects of nicotine, in general, are increased heart rate (up to 10–15 beats/min) and

increased BP (up to 5–10 mmHg).[16-18] Transdermal nicotine absorption causes lesser hemodynamic effects than actual smoking.[19] It increases cardiac output by increasing the heart rate and myocardial contractility. It also causes cutaneous vasoconstriction. It dilates the skeletal muscle vascular beds which can be explained by the increase in cardiac output.[20] Nicotine binds to the cholinergic receptors located in the brain, autonomic ganglia, adrenals, and neuromuscular junction.[21] The chief cyclic vomiting syndrome (CVS) effect is sympathetic neural stimulation which can be due to central nervous system-mediated sympathetic stimulation (by activation of peripheral chemoreceptors) or by intrapulmonary chemoreceptors that, in turn, contribute to the brain-mediated sympathetic arousal. The most sensitive site to low levels of nicotine is the carotid chemoreceptor. Nicotine works by the release of neurotransmitters such as epinephrine, norepinephrine, dopamine, acetylcholine, serotonin, vasopressin, glutamate, and nitric oxide[22] which may also cause effects on the blood vessels.

The mechanisms by which nicotine contributes to acute cardiovascular events are by nature of its hypercoagulable state, increased myocardial work, effects of carbon monoxide in oxygen delivery, coronary vasoconstriction, and catecholamine release. Nicotine exposure or smoking is associated with an increased risk of thrombosis and myocardial infarction.[23] The nicotine in cigarettes and beedi can transiently increase the heart rate and myocardial contractility which, in turn, increases the myocardial workload and blood flow. When the myocardial flow increases, the coronary blood flow must also increase simultaneously to meet the oxygen and nutrient requirements. When the coronary blood flow is limited, it results in ischemia.[24] The carbon monoxide from nicotine smoke increases the risk of ventricular arrhythmias and ventricular dysfunction that results are atherogenesis.[25] The mechanisms by which nicotine promotes atherosclerosis are by their adverse effects on lipids, endothelial toxicity, hemodynamic stress, oxidant injury, neutrophil activation, enhanced thrombosis, and increases fibrinogen and blood viscosity.

Nicotine causes reduction in HDL cholesterol and an increase in LDL cholesterol and also induces endothelial damage that acts as an initiating event in atherosclerosis.[26] All through these changes are quick to occur in beedi smokers, the amount of nicotine absorbed by the beedi rollers is comparatively lesser and it acts as a risk factor for the occurrence of CVS abnormalities. In our study, it was found that there was no correlation between beedi rolling and BP. Both systolic and diastolic BPs seemed normal and no significant changes in heart rates were observed. ECG findings did not show any significant differences. The QTc and Tp-e intervals were within the normal range. Tp-e interval is the distance between the peak and the endpoints of the T wave which is considered as the transmural dispersion index of ventricular repolarization. An alteration in the lipid profile was noticed in the female beedi rollers when compared to normal women. There was an increase in the LDL levels which states that the atherogenic index is significantly high and the risk of arrhythmogenesis is also increased due to cardiovascular toxicity.

Steps should be taken for periodic assessment of systemic illness in these unorganized labor categories to improvise their economic status and reduce the risk of cardiac diseases in them.

CONCLUSION

Beedi tobacco (nicotine) exposure is associated with risk of accelerated atherogenesis and arrhythmogenesis, but in our study, beedi rollers are not showing changes in ECG. Our study concludes that there is no correlation between the duration of beedi rolling and occurrence of cardiovascular events except that they are at a higher risk for these events. The limitations of our study are the small study population and its retrospective nature. More cardiac parameters should be considered in future and elaborated studies should be conducted to give a detailed and clear hypothesis on the long-term effects of nicotine in beedi rollers.

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