ORIGINAL RESEARCH

Assessment of Dyslipidaemia and Echocardiographic Markers of Myocardial Contractility in Smokers with Ischaemic Heart Disease

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ABSTRACT

Background: Cardiovascular diseases (CVDs) remain a leading cause of morbidity and mortality worldwide; with ischaemic heart disease (IHD) being one of the most prevalent conditions affecting millions of individuals annually.This study aims to evaluate the impact of dyslipidaemia on echocardiographic markers of myocardial contractility in smokers with ischaemic heart disease (IHD). The study investigates the relationship between lipid abnormalities and myocardial dysfunction in this high-risk population.

Material and Methods: A total of 100 patients diagnosed with IHD, all of whom were smokers with a history of smoking for at least five years, were included in this study. Baseline demographic data, including age, sex, body mass index (BMI), duration of smoking, and blood pressure, were recorded. Laboratory investigations included fasting lipid profile assessments of total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides. Echocardiographic evaluations were performed to assess left ventricular ejection fraction (LVEF), fractional shortening (FS), and regional wall motion abnormalities (RWMA), as well as myocardial velocities using tissue Doppler imaging (TDI). Statistical analysis was conducted using SPSS, with a p-value of <0.05 considered statistically significant.

Results: The mean age of the participants was 56.4 ± 10.2 years, with 85% being male. The mean BMI was 27.6 ± 3.4 kg/m², and the mean duration of smoking was 18.3 ± 7.5 years. The lipid profile analysis revealed dyslipidaemia, with elevated total cholesterol (210.5 ± 35.2 mg/dL), LDL-C (135.7 ± 28.4 mg/dL), and triglycerides (185.6 ± 45.3 mg/dL), while HDL-C was reduced (38.5 ± 6.2 mg/dL). Echocardiographic findings showed left ventricular dysfunction, with a mean LVEF of 48.3 ± 8.7% and evidence of diastolic dysfunction in 45% of patients. RWMA was present in 65% of patients, indicating a high prevalence of ischemia-induced myocardial dysfunction. A significant association was observed between lipid abnormalities and impaired myocardial contractility, with higher LDL-C and triglyceride levels in patients with reduced LVEF.

Conclusion: The study highlights the adverse effects of smoking-induced dyslipidaemia on myocardial contractility in patients with IHD. The findings underscore the strong correlation between abnormal lipid profiles and left ventricular dysfunction, emphasizing the need for targeted interventions, including smoking cessation and lipid-lowering therapies, to mitigate cardiovascular risks.

Keywords: Ischaemic heart disease, Smoking, Dyslipidaemia, Myocardial contractility, Echocardiography.

INTRODUCTION

Cardiovascular diseases (CVDs) remain a leading cause of morbidity and mortality worldwide, with ischaemic heart disease (IHD) being one of the most prevalent conditions affecting millions of individuals annually.

Among the numerous risk factors contributing to the development and progression of IHD, smoking stands out as a significant modifiable factor that profoundly impacts cardiovascular health. The deleterious effects of smoking on the cardiovascular system are well documented, with evidence linking it to endothelial dysfunction, atherosclerosis, oxidative stress, and increased inflammatory responses. However, beyond these broad pathological mechanisms, smoking also influences lipid metabolism and myocardial contractility, factors that play crucial roles in the clinical outcomes of patients with IHD.[1]Dyslipidaemia, characterized by abnormal lipid levels in the blood, is a critical risk factor for atherosclerosis and coronary artery disease (CAD). Smoking is known to alter lipid metabolism, leading to an atherogenic lipid profile, which includes elevated levels of low-density lipoprotein cholesterol (LDL-C), decreased high-density lipoprotein cholesterol (HDL-C), and increased triglycerides (TG). These alterations contribute to the progression of atherosclerosis, exacerbating coronary artery narrowing and predisposing individuals to acute coronary syndromes (ACS). The assessment of lipid abnormalities in smokers with IHD is essential, as dyslipidaemia not only serves as an indicator of cardiovascular risk but also represents a modifiable factor that can be targeted for therapeutic interventions.[2]In addition to dyslipidaemia, myocardial contractility—the ability of the heart muscle to contract and generate force—plays a pivotal role in determining cardiac function and overall cardiovascular health. Echocardiographic evaluation of myocardial contractility provides valuable insights into the structural and functional changes occurring in the heart, particularly in individuals with established IHD. Parameters such as left ventricular ejection fraction (LVEF), global longitudinal strain (GLS), and tissue Doppler imaging (TDI) indices help assess myocardial performance, detect early signs of myocardial dysfunction, and guide clinical management strategies. In smokers with IHD, smoking-induced alterations in myocardial contractility may further compromise cardiac function, increasing the risk of heart failure and adverse cardiovascular events.[3]

Despite the well-established relationship between smoking and CVD, the combined evaluation of dyslipidaemia and echocardiographic markers of myocardial contractility in smokers with IHD remains an area that requires further exploration. While dyslipidaemia is widely recognized as a major contributor to atherosclerosis, its interaction with smoking-related myocardial dysfunction warrants deeper investigation. The synergistic impact of these factors could potentially exacerbate cardiovascular impairment, influencing disease progression and treatment outcomes in affected individuals.[4]

Understanding the interplay between lipid abnormalities and myocardial contractility in smokers with IHD has important clinical implications. By identifying specific patterns of dyslipidaemia and myocardial dysfunction in this population, clinicians can tailor therapeutic strategies aimed at optimizing lipid levels and preserving myocardial function. Pharmacological interventions such as statins, lipid-lowering agents, and renin-angiotensin system inhibitors, along with lifestyle modifications including smoking cessation and dietary changes, may offer significant benefits in mitigating the cardiovascular risks associated with smoking. Moreover, advancements in echocardiographic imaging techniques provide opportunities for early detection and monitoring of myocardial dysfunction, enabling timely intervention to prevent disease progression.[5,6]

AIM& OBJECTIVES

The current study aims to evaluate the impact of dyslipidaemia on echocardiographic markers of myocardial contractility in smokers with ischaemic heart disease (IHD) and evaluate the relationship between lipid abnormalities and myocardial dysfunction in this high-risk population.

MATERIALS & METHODS

Study Design: The current study was a The Observational Cross-Sectional study.

Study place*:* This study was conducted at Department of General Medicine, Major S.D. Singh Medical College & Hospital, Farrukhabad, Uttar Pradesh, Indiain collaboration with Department of Physiology, Major S.D. Singh Medical College & Hospital, Farrukhabad, Uttar Pradesh, IndiaandDepartment of Biochemistry, Santosh Medical College & Hospital, Ghaziabad, NCR Delhi, India.

Study period: The study was carried out from January2018 to October 2019.

Ethical consideration: The study was ethical approval was obtained from the institutional review board.

Study Population: The Study included 100 patients (85 males and 15 females) diagnosed with IHD, all of whom had a history of smoking.

Informed written consent was secured from all patients before their inclusion in the study.

Inclusion Criteria:

- Patients who give written informed consent.
- Patients of both genders diagnosed with IHD.
- History of smoking.
- Available for follow-up.

Exclusion Criteria:

- Patients do not give written, informed consent.
- Patients with renal disease, hepatic disease, or any other neurological disorders.
- Individuals with diabetes mellitus, hypertension, or other systemic illnesses that could influence lipid metabolism or cardiac function.
- Patients on lipid-lowering medications or those with a history of alcohol abuse.
- Not available for follow-up.

Data Collection:

- **Lipid Profile Assessment:** Serum Total Cholesterol (TC), Triglycerides (TG), High-Density Lipoprotein (HDL), and Low-Density Lipoprotein (LDL) levels were measured using an auto-analyzer. Very Low-Density Lipoprotein (VLDL) was calculated using Friedewald's equation. Venous blood samples were collected after an overnight fast of 12 hours and analyzed using an automated biochemical analyzer.
- **Echocardiographic Evaluation:** Two-dimensional M-mode and Doppler echocardiography were performed to assess left ventricular ejection fraction and other cardiac parameters.

Baseline demographic data such as age, sex, body mass index (BMI), and duration of smoking were recorded. Parameters evaluated included left ventricular ejection fraction (LVEF), fractional shortening (FS), and regional wall motion abnormalities (RWMA). Tissue Doppler imaging (TDI) was also utilized to measure myocardial velocities, including peak systolic (S'), early diastolic (E'), and late diastolic (A') velocities, to assess myocardial contractility. All echocardiographic measurements were taken following the recommendations of the American Society of Echocardiography.

STATISTICAL ANALYSIS

- Data were analyzed using appropriate statistical methods to compare lipid profiles and echocardiographic parameters.
- Statistical analysis was performed using SPSS software. Continuous variables were expressed as mean ± standard deviation, while categorical variables were represented as frequencies and percentages. Comparisons between groups were conducted using the Student's t-test for normally distributed variables and the Mann-Whitney U test for non-normally distributed variables. A p-value of <0.05 was considered statistically significant.

RESULTS

The results of the study provide a comprehensive analysis of the clinical, biochemical, and echocardiographic parameters in smokers with ischaemic heart disease (IHD).

Table 1: Baseline Characteristics of Study Population

Characteristic	Mean ± SD / Percentage
Age (years)	56.4 ± 10.2
Male (%)	85%
Female	15%
BMI (kg/m²)	27.6 ± 3.4
Duration of Smoking (years)	18.3 ± 7.5
Systolic Blood Pressure (mmHg)	140.5 ± 15.2
Diastolic Blood Pressure (mmHg)	85.2 ± 10.8
Heart Rate (beats per minute)	76.3 ± 8.5
Fasting Blood Glucose (mg/dL)	110.7 ± 18.3

The study included 100 patients diagnosed with IHD, all of whom were smokers. The mean age of the participants was 56.4 ± 10.2 years, with the majority (85%) being male. The mean body mass index (BMI) was 27.6 ± 3.4 kg/m², indicating that most participants were overweight. The duration of smoking was recorded at an average of 18.3 ± 7.5 years, highlighting the long-term exposure to tobacco and its potential contribution to cardiovascular risk.

The mean systolic and diastolic blood pressures were 140.5 ± 15.2 mmHg and 85.2 ± 10.8 mmHg, respectively, suggesting a high

prevalence of hypertension among the participants. Additionally, the mean heart rate was 76.3 ± 8.5 beats per minute, which is within the normal range but may indicate increased sympathetic activity. The fasting blood glucose levels averaged 110.7 ± 18.3 mg/dL, indicating that a significant proportion of the patients had impaired fasting glucose or diabetes mellitus, which is a known risk factor for cardiovascular disease[Table 1, Figure I].

Figure I: Gender wise distribution of the patients

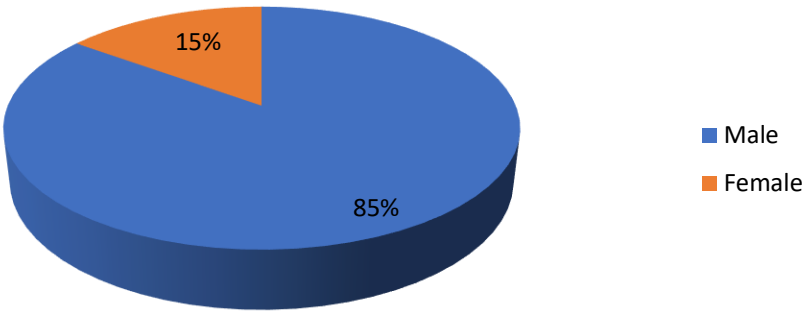

Table 2: Lipid Profile of the Study Population

Parameter	Mean ± SD
Total Cholesterol (mg/dL)	210.5 ± 35.2
LDL-C (mg/dL)	135.7 ± 28.4
HDL-C (mg/dL)	38.5 ± 6.2
Triglycerides (mg/dL)	185.6 ± 45.3
Non-HDL Cholesterol (mg/dL)	172.0 ± 32.5
Total Cholesterol/HDL Ratio	5.6 ± 1.4
LDL/HDL Ratio	3.5 ± 1.1

Table 2 shows that the lipid profile analysis revealed dyslipidaemia among the participants. The mean total cholesterol level was 210.5 ± 35.2 mg/dL, which is elevated and suggests a higher risk of atherosclerosis. The mean low-density lipoprotein cholesterol (LDL-C) level was 135.7 ± 28.4 mg/dL, which is considered borderline high and is strongly associated with cardiovascular events.

The mean high-density lipoprotein cholesterol (HDL-C) level was 38.5 ± 6.2 mg/dL, which is lower than the recommended level, indicating reduced cardioprotective effects. Triglyceride levels were also elevated, with a mean of 185.6 ± 45.3 mg/dL, further contributing to an atherogenic lipid profile. The mean non-HDL cholesterol level, an important marker of cardiovascular risk, was found to be 172.0 ± 32.5 mg/dL. The total cholesterol to HDL ratio and LDL to HDL ratio were 5.6 ± 1.4 and 3.5 ± 1.1, respectively, both of which suggest an increased risk of cardiovascular disease.

Table 3: Echocardiographic Parameters of Myocardial Contractility

Parameter	Mean ± SD
Left Ventricular Ejection Fraction (LVEF) (%)	48.3 ± 8.7
Fractional Shortening (%)	25.4 ± 4.5
Peak Systolic Velocity (S') (cm/s)	7.8 ± 1.3
Early Diastolic Velocity (E') (cm/s)	9.5 ± 1.6
Late Diastolic Velocity (A') (cm/s)	8.2 ± 1.4
Left Ventricular End-Diastolic Diameter (mm)	52.4 ± 5.6
Left Ventricular End-Systolic Diameter (mm)	38.2 ± 4.9
E/A Ratio	0.85 ± 0.14

Table 3 shows that Echocardiographic assessment showed evidence of impaired myocardial contractility in many patients. The mean left ventricular ejection fraction (LVEF) was 48.3 ± 8.7%, which is lower than the normal reference range, indicating left ventricular systolic dysfunction in a significant proportion of patients. The mean fractional shortening was 25.4 ± 4.5%, further supporting the presence of compromised myocardial contractility.

Tissue Doppler imaging (TDI) parameters were also assessed. The mean peak systolic velocity (S') was 7.8 ± 1.3 cm/s, indicating impaired myocardial contraction. The early diastolic velocity (E') and late diastolic velocity (A') were 9.5 ± 1.6 cm/s and 8.2 ± 1.4 cm/s, respectively, suggesting alterations in diastolic function. The mean left ventricular end-diastolic diameter (LVEDD) was 52.4 ± 5.6 mm, and the left ventricular end-systolic diameter (LVESD) was 38.2 ± 4.9 mm, both of which suggest ventricular remodeling and possible dilatation due to chronic ischemic injury. The mean E/A ratio were 0.85 ± 0.14, which is indicative of diastolic dysfunction.

Table 4: Comparison of Lipid Profile between Normal and Impaired Myocardial Contractility Groups

Parameter	Normal Contractility (Mean ± SD)	Impaired Contractility (Mean ± SD)	p-value
Total Cholesterol (mg/dL)	195.2 ± 30.4	225.7 ± 40.3	0.01
LDL-C (mg/dL)	120.5 ± 25.8	150.8 ± 30.2	0.02
HDL-C (mg/dL)	42.3 ± 5.8	34.6 ± 4.9	0.03
Triglycerides (mg/dL)	160.4 ± 38.7	210.7 ± 50.6	0.005
Non-HDL Cholesterol (mg/dL)	153.5 ± 29.2	190.6 ± 35.1	0.008
LDL/HDL Ratio	2.8 ± 0.9	4.2 ± 1.2	0.001

Table 4 shows the comparative analysis of lipid parameters between patients with normal and impaired myocardial contractility showed significant differences. Patients with impaired contractility had significantly higher total cholesterol levels (225.7 ± 40.3 mg/dL vs. 195.2 ± 30.4 mg/dL, p = 0.01) and LDL-C levels (150.8 ± 30.2 mg/dL vs. 120.5 ± 25.8 mg/dL, p = 0.02).

HDL-C levels were notably lower in the impaired contractility group (34.6 ± 4.9 mg/dL vs. 42.3 ± 5.8 mg/dL, p = 0.03), indicating a reduced protective effect against atherosclerosis. Triglyceride levels were significantly elevated in the impaired contractility group (210.7 ± 50.6 mg/dL vs. 160.4 ± 38.7 mg/dL, p = 0.005).

Non-HDL cholesterol, which encompasses all atherogenic lipoproteins, was significantly higher in patients with impaired contractility (190.6 ± 35.1 mg/dL vs. 153.5 ± 29.2 mg/dL, p = 0.008). The LDL/HDL ratio, a strong predictor of cardiovascular risk, was also significantly higher in the impaired contractility group (4.2 ± 1.2 vs. 2.8 ± 0.9, p = 0.001). These findings suggest that dyslipidaemia plays a crucial role in the worsening of myocardial contractility in smokers with IHD.

Table 5: Prevalence of Regional Wall Motion Abnormalities (RWMA) and Myocardial Dysfunction

Parameter	Number of Patients (%)
RWMA Present	65 (65%)
RWMA Absent	35 (35%)
Global Hypokinesia Present	28 (28%)
Global Hypokinesia Absent	72 (72%)
Diastolic Dysfunction Present	45 (45%)
Diastolic Dysfunction Absent	55 (55%)

Table 5 shows the regional wall motion abnormalities (RWMA) were present in 65% of the study population, indicating a high prevalence of ischemia-induced myocardial dysfunction. In contrast, 35% of the patients had no detectable RWMA.

Global hypokinesia, which reflects generalized impairment of myocardial contraction, was present in 28% of the patients. This finding

suggests that a subset of patients had diffuse myocardial involvement rather than localized ischemia. Diastolic dysfunction was identified in 45% of the study population, further indicating that nearly half of the patients had impaired ventricular relaxation, which is a common feature in smokers and individuals with ischemic heart disease.

DISCUSSION

The mean age of 56.4 ± 10.2 years and predominance of male participants (85%) in our study are consistent with previous research indicating a higher prevalence of IHD among middle-aged men. The average body mass index (BMI) of 27.6 ± 3.4 kg/m² suggests that many participants were overweight, a known risk factor for cardiovascular disease. The mean duration of smoking was 18.3 ± 7.5 years, underscoring prolonged exposure to tobacco-related cardiovascular risks.Elevated mean systolic (140.5 ± 15.2 mmHg) and diastolic (85.2 ± 10.8 mmHg) blood pressures indicate a high prevalence of hypertension, a common comorbidity in smokers with IHD. This finding aligns with studies suggesting that smoking exacerbates hypertension, thereby increasing cardiovascular risk (Craig et al., 1989).[7] The mean fasting blood glucose level of 110.7 ± 18.3 mg/dL points to impaired glucose metabolism, which, in conjunction with smoking, heightens the risk of developing type 2 diabetes mellitus (Eliasson, 2003).[8]Our study revealed dyslipidaemia characterized by elevated total cholesterol (210.5 ± 35.2 mg/dL) and low-density lipoprotein cholesterol (LDL-C) levels (135.7 ± 28.4 mg/dL), coupled with reduced high-density lipoprotein cholesterol (HDL-C) levels (38.5 ± 6.2 mg/dL). These findings are consistent with earlier research indicating that smokers exhibit higher LDL-C and lower HDL-C levels compared to non-smokers (Craig et al., 1989). Elevated triglyceride levels (185.6 ± 45.3 mg/dL) further contribute to an atherogenic lipid profile, corroborating the association between smoking and adverse lipid alterations.[7]The mean left ventricular ejection fraction (LVEF) of 48.3 ± 8.7% observed in our study indicates systolic dysfunction among participants. This finding aligns with previous studies demonstrating that smoking contributes to left ventricular dysfunction, potentially through mechanisms involving increased oxidative stress and myocardial fibrosis (He et al., 1999). Tissue Doppler imaging parameters, including reduced peak systolic velocity (S') and early diastolic velocity (E'), suggest impaired myocardial relaxation and contraction, which have been associated with chronic smoking.[9]Our comparative analysis revealed that patients with impaired myocardial contractility had significantly higher total cholesterol and LDL-C levels, along with lower HDL-C levels, compared to those with normal contractility. These findings are in line with studies indicating that dyslipidaemia exacerbates myocardial dysfunction, particularly in the context of smoking-induced oxidative stress and endothelial dysfunction (Gepner et al., 2011).[10]The high prevalence of RWMA (65%) and diastolic dysfunction (45%) in our study population underscores the detrimental impact of chronic smoking on myocardial structure and function. These findings are consistent with earlier research demonstrating that smoking is associated with regional myocardial dysfunction and impaired ventricular relaxation, contributing to the progression of heart failure (Heeringa et al., 2008).[11]

LIMITATIONS OF THE STUDY

- Small Sample Size
- Short Follow-Up Duration

CONCLUSION

This study highlights the significant impact of smoking on dyslipidaemia and myocardial contractility in patients with ischaemic heart disease. The findings reveal a high prevalence of abnormal lipid profiles, including elevated LDL-C and triglycerides with reduced HDL-C, which contribute to impaired myocardial function. Echocardiographic parameters demonstrated notable left ventricular dysfunction, including reduced LVEF and diastolic abnormalities. The strong association between smoking, dyslipidaemia, and myocardial dysfunction underscores the need for aggressive risk factor modification, including smoking cessation and lipid-lowering therapy.

REFERENCES

1. Rigotti NA, Clair C. Managing tobacco use: the neglected cardiovascular disease risk factor. *Eur Heart J*. 2013;34(42):3259-67.
2. Mons U, Muezzinler A, Gellert C, et al. Impact of smoking and smoking cessation on cardiovascular events and mortality among older adults: meta-analysis of individual participant data from prospective cohort studies of the chances consortium. *BMJ*. 2015;350:h1551.
3. Piano MR, Benowitz NL, Fitzgerald GA, et al. Impact of smokeless tobacco products on

cardiovascular disease: implications for policy, prevention, and treatment. *Circulation*. 2010;122(15):1520-44.
4. Teo KK, Ounpuu S, Hawken S, et al. Tobacco use and risk of myocardial infarction in 52 countries in the inter heart study: a case-control study. *Lancet*. 2006;368(9536):647-58.
5. Critchley JA, Capewell S. Mortality risk reduction associated with smoking cessation in patients with coronary heart disease: a systematic review. *JAMA*. 2003;290(1):86-97.
6. Wilson K, Gibson N, Willan A, Cook D. Effect of smoking cessation on mortality after myocardial infarction: meta-analysis of cohort studies. *Arch Intern Med*. 2000;160(7):939-44.
7. Craig WY, Palomaki GE, Haddow JE. Cigarette smoking and serum lipid and lipoprotein concentrations: an analysis of published data. *BMJ*. 1989;298(6676):784-8.
8. Eliasson B. Cigarette smoking and diabetes. *ProgCardiovasc Dis*. 2003;45(5):405-13.
9. He J, Vupputuri S, Allen K, Prerost MR, Hughes J. Passive smoking and the risk of coronary heart disease: a meta-analysis of epidemiologic studies. *N Engl J Med*. 1999;340(12):920-6.
10. Gepner AD, Piper ME, Johnson HM, Fiore MC, Baker TB, Stein JH. Effects of smoking and smoking cessation on lipids and lipoproteins: outcomes from a randomized clinical trial. *Am Heart J*. 2011;161(1):145-51.
11. Heeringa J, Kors JA, Hofman A, van Rooij FJ, Witteman JC. Cigarette smoking and risk of atrial fibrillation: the Rotterdam Study. *Am Heart J*. 2008;156(6):1163-9.