The buildup of lipids, cholesterol, and other materials in and on the arterial walls is known as atherosclerosis [1]. Arteries may constrict and get blocked by plaque. Additionally, a blood clot may form if the plaque bursts. It may damage arteries anywhere in the body, atherosclerosis is most frequently linked to the heart [2]. It is possible to cure atherosclerosis. An atherosclerosis prevention diet and lifestyle can assist. Once an artery has constricted or blocked to the point that it is unable to adequately provide blood to organs or tissues, atherosclerotic symptoms usually manifest. One can totally stop blood flow due to a blood clot. Angina or a stroke might result from the clot breaking apart [3]. The primary cause of cardiovascular illness is atherosclerosis, an inflammatory arterial disease linked to cholesterol and other metabolic alterations [4]. Cerebrovascular disease and ischemic heart disease (IHD) are the two main indicators of atherosclerotic cardiovascular disease (ACD). In Asia, ischemic heart disease and stroke rank as the third most prevalent causes of mortality [5]. As per many study articles and current world health organization (WHO) atherosclerotic cardiovascular disease is one of the major causes of mortality worldwide for both men and women. With 3.2 percent yearly increase, the prevalence of this disease is steadily rising in smokers and obese individuals [6]. Atherosclerosis is the result of a complex pathologic process. It is often linked to increased levels of cholesterol, triglycerides, and LDL in cardiovascular patients.
permeability and progressively damages artery walls[6, 7]. By encouraging circulating monocytes to attach to endothelial cells which subsequently produce adhesion molecules and selectin. This substrate triggers an inflammatory response by driving monocyte migration to the sub-endothelial region [8]. After that, monocytes change into foamy macrophages that are high in free fatty acids and cholesterol esters. These macrophages penetrate the artery walls, create a pathological lesion known as intimal thickening, and eventually cause the lipid pool to become necrotic. Atherosclerotic plaque's foamy macrophages are vulnerable to plaque fracture or rupture, which can result in life-threatening thrombosis [9, 10]. The phrase atherosclerotic cardiovascular disease (CVD) refers to a collection of heart and blood vessel conditions [11]. Worldwide, these illnesses are the main contributors to morbidity and early mortality. Coronary heart disease and cerebrovascular disease (stroke) are the most prevalent illnesses[12, 13]]. The second cause of CVD is the oxidation of LDL. Foam cells arise when the oxidation of LDL in the artery wall sets off an inflammatory cascade that initiates the atherogenic pathway. The first discernible atherosclerotic lesion is formed by fatty streaks, which are caused by the buildup of foam cells [14, 15]. The results of several investigations have led to the conclusion that smoking cigarettes is a significant risk factor for the emergence of clinical cardiovascular disease. To investigate the precise functions and interactions between smoking and hyperlipidemia in the development of atherosclerotic cardiovascular disease in people who have been diagnosed[16].

This study was conducted to create more effective preventive and management methods for atherosclerotic cardiovascular disease by providing deeper insights into the roles played by these risk factors in the illness by studying them in a clinical environment.

**METHODS**

A cross-sectional study was conducted in which blood samples were collected from diagnosed cardiac male and female patients. The aims and objectives of this study were to uncover the relationship between hyperlipidemia and smoking with atherosclerotic cardiovascular disease in both male and female genders. Current study was conducted in Medical and Cardiology Departments of Gorki Hospital Lahore, Jinnah Hospital Lahore, and Services Hospital Lahore from June 2023 to February 2024. The ethical approval clearance certificate Ref no.2023/3A was granted by ethical review committee, Faculty of Biological Sciences, Lahore University of Biological and Applied Sciences (UBAS). The age of all participants was in between 35 to 60 years. Body mass index, Smoking habits, Cholesterol, Triglycerides, and LDL and HDL were considered as inclusive biomarkers criteria. Individuals with renal diseases, pregnant women and diabetic patients were exclusive for current study. Total 200 male and female participants with different cardiac complications were selected and divided them into different groups such as Group A and Group B. In Group A 75 males and 25 females with severe chest pain, unexpected numbness or weakness in arms or legs and loss of vision were included while in Group B, 70 male and 30 female individuals with mild chest pain were listed. BMI, Cholesterol, Triglyceride, LDL and HDL levels and other demographics such as age, smoking habits and socioeconomic conditions were measured respectively. The sample size was calculated using a power analysis to assure 90% power to detect a significant difference in cardiovascular risk between smokers and non-smokers at an alpha level of 0.05, resulting in a sample size of 200 individuals.

The Participants were chosen by stratified random sampling to reflect various age groups and genders in the community. For lipid profile tests about 5ml blood sample is taken with a needle inserted into a vein in the arm and stored in blood glass container after centrifugation. BMI values were determined using a well-defined questionnaire, along with other demographic variables including gender, age, smoking, drinking alcohol, and physical activity. Using SPSS version 22.0, bio-statistical processing were applied to the raw data. The correlations between smoking and lipid profile and atherosclerotic cardiovascular disease were investigated using a t-test and the derived means, standard deviations, frequencies, and percentages of participant characteristics. Means and standard deviations (Mean ± SD) were taken into consideration at significant levels (p<0.05). To determine the significance of differences in biomarkers and smoking behaviors, the groups were compared using chi-square tests for categorical variables and t-tests for continuous variables.

**RESULTS**

In Group A (severe disease group) there were 75 males and 25 females with severe chest pain, unexpected numbness or weakness in arms or legs and loss of vision while in Group B (mild disease group) 70 male and 30 female individuals with mild chest pain were listed. The mean age in Group A was (59.09 ± 0.01) and Group B was (59.09 ± 0.01). Whereas the biomarkers such as gender, age, smoking habits, BMI, Cholesterol, Triglyceride, LDL and HDL were compared. The results showed significant difference in Group A and B Cholesterol (279.9 ± 0.04 vs. 239.09 ± 0.04), Triglycerides (187.02 ± 0.01 vs. 127.02 ± 0.01), LDL (153.01 ± 0.02 vs. 123.01 ± 0.02), HDL (49.04 ± 0.01 Vs. 40.01 ± 0.01) and (p-value<0.05) (Table 1).
Table 1: Comparative Analysis of Biomarkers in Severe and Mild Disease

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Category</th>
<th>Group A (Severe) Mean ± SD</th>
<th>Group B (Mild) Mean ± SD</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>Male (%)</td>
<td>75 (75%)</td>
<td>70 (70%)</td>
<td>0.45*</td>
</tr>
<tr>
<td></td>
<td>Female (%)</td>
<td>25 (25%)</td>
<td>30 (30%)</td>
<td>-</td>
</tr>
<tr>
<td>Age</td>
<td>Years</td>
<td>59.09 ± 0.01</td>
<td>59.09 ± 0.01</td>
<td>0.99</td>
</tr>
<tr>
<td>Smoking</td>
<td>Yes (%)</td>
<td>89 (89%)</td>
<td>45 (45%)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>BMI</td>
<td>Kg/m²</td>
<td>34.03 ± 0.01</td>
<td>25.01 ± 0.01</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>mg/dl</td>
<td>273.9 ± 0.04</td>
<td>239.09 ± 0.04</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>mg/dl</td>
<td>187.02 ± 0.01</td>
<td>127.02 ± 0.01</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LDL</td>
<td>mg/dl</td>
<td>153.01 ± 0.01</td>
<td>123.01 ± 0.02</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>HDL</td>
<td>mg/dl</td>
<td>49.04 ± 0.01</td>
<td>40.01 ± 0.01</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

(t-test for continuous variables, chi-square for categorical*)

**DISCUSSION**

Guddeti et al., concluded that even before atherosclerosis develops, the complicated disorder known as hyperlipidemia affects the anatomy and function of the heart. It has long been recognized that serum lipids directly affect heart function in ways unrelated to atherosclerosis [1, 8, 17]. Nevertheless, recent studies by a number of experts have found that serum lipids may build up in the heart, cause oxidative stress and inflammatory cardiac fibrosis, reduce autophagy and microvascular density, and alter the way that cardiomyocytes mitochondria operate. Heart failure, myocardial injury, and electrophysiological abnormalities are more likely as a result of these impacts [10, 18]. The findings of Hitl et al., were described that hypercholesterolemia promotes atherosclerosis and raises the risk of peripheral vascular disease, coronary artery disease, and stroke which has close similarities with the secondary data of different studies [19]. This study has implications for the development of hypercholesterolemia. Raising cholesterol levels causes an increase in the formation of many atherogenic biomolecules, including proinflammatory cytokines interleukin IL-1, IL-2, IL-6, IL-8, and tumor necrosis factor-alpha (TNF-α) [20, 21]. According to the LDL receptor theory, atherosclerosis is caused by high blood levels of LDL cholesterol, and cardiovascular disease can be avoided by reversing or at least delaying atherosclerosis [22]. Heart failure risk has been linked to high non-fasting triglyceride levels and cholesterol. Nonetheless, a low level of serum triglycerides raised the mortality rate of both ischemic and non-ischemic heart failure and was independently linked to a poor prognosis in patients with end-stage heart failure [23, 24]. Present study described that individuals with smoking habits showed high risk of cardiovascular medical complications and similar results were concluded by different studies. Almost every organ in the body is harmed by smoking, including the blood vessels, teeth, mouth, eyes, heart, reproductive organs, bones, bladder, and digestive organs [25]. The inhaled chemicals from smoking damage your heart and blood vessels, raising your risk of atherosclerosis, or the buildup of plaque in the arteries [26]. In this approach, smoking can damage the heart and blood vessels in any amount, even occasionally. Some people are considerably more at risk from smoking, especially those who have diabetes and those who take birth control pills [27]. Current study has significant (p≤0.05) correlation with the previous studies and showed similarities with the secondary data of different studies. The levels of Lipoproteins, Cholesterol and Triglycerides in both male and female participants in Group A vary significantly (p≤0.05) from those in Group B. The individuals how were come in tertiary care units with severe chest pain, unexpected numbness or weakness in arms, legs and loss of vision have a remarkable difference in different lipid profile biomarkers as compared with the individuals how has mild chest pain respectively [28].

**CONCLUSIONS**

Hyperlipidemia and smoking were significant (p≤0.05) risk factors for atherosclerotic cardiovascular disease, as seen by increasing levels of cholesterol, triglycerides and LDL in cardiovascular patients.

**Authors Contribution**

Conceptualization: HUK, HR, KB
Methodology: HR, BI
Formal analysis: HR
Writing-review and editing: BI, HUK, KB, A, SA, AS

All authors have read and agreed to the published version of the manuscript.

**Conflicts of Interest**

The authors declare no conflict of interest.

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**REFERENCES**


