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SERUM LEVELS OF INTERLEUKIN-1β ALONG WITH ITS GENETIC POLYMORPHISM IN PATIENTS OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Abstract

Background: Chronic Obstructive Pulmonary Disease (COPD) is a progressive inflammatory lung disorder characterized by persistent airflow limitation and systemic inflammation. Interleukin-1β (IL-1β), a key pro-inflammatory cytokine, has been implicated in COPD pathogenesis; however, data on its serum levels and genetic polymorphisms in the Indian population remain limited. **Material & Methods:** A case–control study was conducted involving 160 participants, comprising 80 COPD patients and 80 healthy controls aged ≥40 years. Serum IL-1β levels were measured using sandwich ELISA. Genotyping of IL-1β polymorphism was performed using ARMS-PCR following DNA extraction. **Results:** Levels of serum IL-1β were significantly higher in the COPD group at 2.28 (1.7–2.685) pg/dl compared to 1.52 (1.05–2.17) pg/dl in controls ($p < 0.0001$). The CC genotype was significantly more frequent among COPD patients (32.5%) than controls (15%), whereas the TT genotype was more prevalent in controls (43.75% vs 17.5%; $p=0.0006$). The C allele was significantly associated with increased COPD risk (57.5% vs 35.63%; $p<0.0001$), while the T allele demonstrated a protective association. **Conclusion:** Elevated serum IL-1β levels support the role of persistent systemic inflammation in COPD. The IL-1β C allele appears to confer increased susceptibility to COPD, whereas the T allele may be protective. These findings emphasize the combined contribution of

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inflammatory and genetic factors in COPD pathogenesis and highlight IL-1β as a potential biomarker for disease risk assessment.

Keywords: COPD, IL-1β, Genotype, Alleles.

INTRODUCTION

COPD is a diverse lung disease defined by chronic respiratory symptoms (cough, sputum production, dyspnea) brought on by abnormalities of the alveoli (emphysema), airways (bronchitis, bronchiolitis), or both that lead to a persistent, frequently worsening blockage of airflow [1]. Globally, the prevalence of COPD in individuals over 40 is 9–10%; in India, it ranges from 6.5 to 7.68% [2]. According to the worldwide burden of illnesses, COPD caused 212.3 million cases, 3.32 million deaths, and 74.4 million disability-adjusted life years (DALYs) in 2019 [3]. Cigarette smoking is linked to COPD as the main cause. Chronic inflammation causes airway constriction and decreased lung recoil, which can progress from being asymptomatic to respiratory failure. Alpha-1 antitrypsin deficiency (AATD), exposure to the environment and at work place, and second-hand smoke are possible additional causes. AAT (Alpha-1 antitrypsin) is a protein made in the liver, deficiency of AAT in the liver, is the cause of AATD. When COPD patients show signs of liver damage, AATD should be suspected, lower lobes are mostly affected in AATD [3].

Chronic bronchitis (CB) and emphysema are the two main causes of COPD [4]. A productive cough lasting longer than three months and returning more than twice a year is the classic indicator of CB [5]. Lung emphysema a condition in which, the alveolar walls are irreversibly destroyed, which causes the distal airspaces to expand [6]. A record of FEV1/FVC ratio (after bronchodilator) less than 0.70 was categorized as an obstructive pattern and confirmed COPD in accordance with GOLD criteria [2].

The pathogenesis of COPD depends on IL-1, a critical proinflammatory cytokine mostly generated by monocytes and macrophages [7]. The airway epithelium and macrophages, which strongly express IL-1β in the lungs of COPD patients, are thought to be the cause of these higher levels of IL-1β [8].

This study aims to fill this gap by evaluating the pattern, association, and risk posed by IL1β polymorphisms at the chromosome 2q14 locus, exploring their potential role in modulating COPD in an Indian population. The research represents a step toward understanding the molecular basis of COPD by examining polymorphisms in candidate genes.

Aim and Objectives

Aim was to study the serum levels of Interleukin-1β along with its Genetic Polymorphism in patients of COPD.

Objectives were to estimate serum level of Interleukin-1β in patients of COPD and control subjects and to investigate the genotype of Interleukin-1β in patients of COPD and control subjects.

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MATERIAL AND METHODS

This case-control research study was conducted in the Department of Biochemistry and Department of Respiratory Medicine, Sharda Hospital, SMSR, Sharda University. The participants for the study were recruited following the acquisition of approval from the Institutional Ethical Committee.

Written informed consent was obtained from the participants. Demographic information was gathered in the study's case record form. The participants chosen for this study consisted of adult patients with COPD, aged 40 years and above, who were either newly diagnosed with the condition or currently receiving treatment and monitoring at the Department of Respiratory Medicine at Sharda Hospital. The study collected details of blood investigations, and genetic studies, respectively.

A case control study was done on human volunteers, consisting of two groups: a group of patients with COPD (n=80) and a control group of healthy subjects (n=80). **Inclusion criteria** Age more than or equal to 40 years. All the cases of COPD diagnosed according to GOLD guidelines for COPD and patient willing to be a part of this study. **Exclusion criteria** Age less than 40 years, non-cooperative and unwilling patients to participate in the study and patient suffering from asthma, history of coronary heart disease, malignancy, systemic infection.

Blood samples were collected in plain and EDTA vials for the biochemical measurement of interleukin 1 beta and DNA isolation respectively. For the estimation of Interleukin- 1 Beta level and polymorphism, serum samples were stored at -20 degrees C. In all the samples, DNA extraction was done from the peripheral blood samples by commercial available Qaigen kits. Interleukin 1beta Primers. Pre-Designed SNP genotyping assays were used to detect the polymorphisms, master mix was added to run for Polymerase Chain Reaction (PCR) for Interleukin 1 beta levels. Samples were analyzed in PCR through electrophoresis and with the help of Gel Doc system, and thus the desired band was achieved. With the help of ELISA kit, serum IL-1β levels were measured.

RESULTS

This study was conducted on 160 participants in which 80 were cases having COPD and 80 were controls.

In COPD cases, age was 64.24 ± 9.74 years in males and 61.7 ± 7.24 years in females. While in controls age was 53.53 ± 9.83 years in males and 55.96 ± 8.62 years in females.

Table 1:- Comparison of IL-1β between COPD and control

IL-1β (pg/dl)	COPD(n=80)	Control(n=80)	Total	P value
Mean ± SD	2.28 ± 0.76	1.76 ± 0.98	2.02 ± 0.91	<.0001§
Median(25th-75th percentile)	2.28 (1.7-2.685)	1.52 (1.05-2.17)	1.89 (1.418-2.512)	
Range	0.84-4.29	0.44-4.78	0.44-4.78	

§ Mann Whitney test

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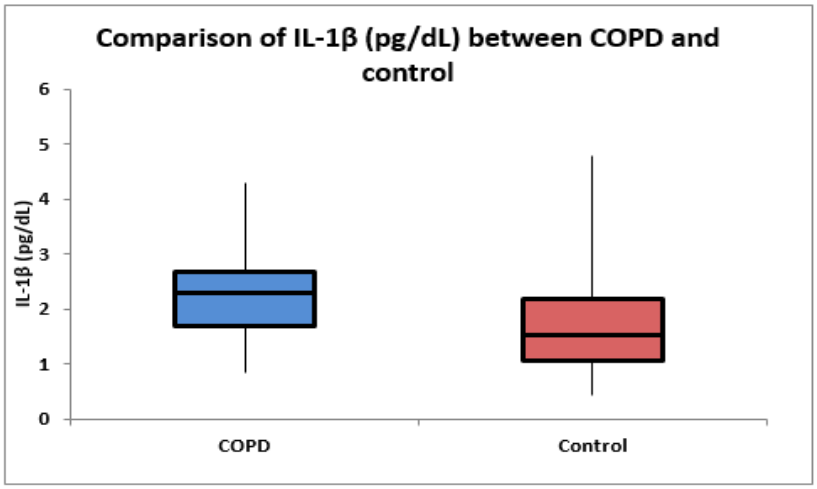

Figure 1: Comparison of IL-1β between COPD and control (non-parametric variable, Box-whisker plot)

Median (25th–75th percentile) IL-1β levels were significantly higher in the COPD group at 2.28 (1.7–2.685) pg/dl compared to 1.52 (1.05–2.17) pg/dl in controls ($p < 0.0001$) (Table 1, Figure 1)

Table 2: Comparison of genotype between COPD and control

Genotype	COPD(n=80)	Control(n=80)	Total	P value	Odds ratio (95% CI)
CC	26 (32.50%)	12 (15%)	38 (23.75%)	0.0006†	1
CT	40 (50%)	33 (41.25%)	73 (45.63%)		0.565 (0.248 to 1.288)
TT	14 (17.50%)	35 (43.75%)	49 (30.63%)		0.188 (0.075 to 0.472)
Total	80 (100%)	80 (100%)	160 (100%)		-

† Chi square test

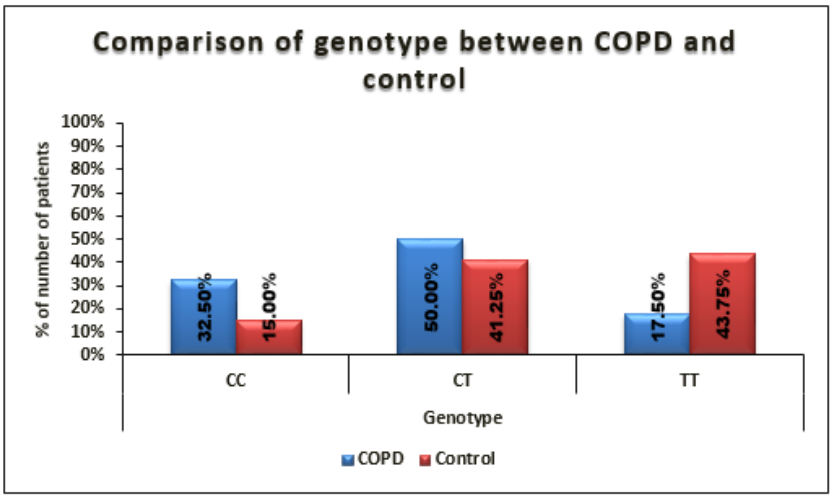

Figure 2: Comparison of genotype between COPD and control

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Genotype distribution differed significantly between COPD and control groups (p value = 0.0006). The CC genotype was more frequent in the COPD group (32.50% vs. 15%), while the TT genotype was more frequent in controls (43.75% vs. 17.50%). Compared to the CC reference genotype, the CT genotype showed an odds ratio of 0.565 (95% CI: 0.248–1.288), while the TT genotype demonstrated a significantly lower odds ratio of 0.188 (95% CI: 0.075–0.472) in COPD compared to controls (Table 2, Figure 2).

Table 3: Comparison of alleles between COPD and control

Alleles	COPD(n=160)	Control(n=160)	Total	P value	Odds ratio (95% CI)
C	92 (57.50%)	57 (35.63%)	149 46.56%)	<.0001†	1
T	68 (42.50%)	103 (64.38%)	171 (53.44%)		0.411 (0.262 to 0.645)
Total	160 (100%)	160 (100%)	320 (100%)		-

† Chi square test

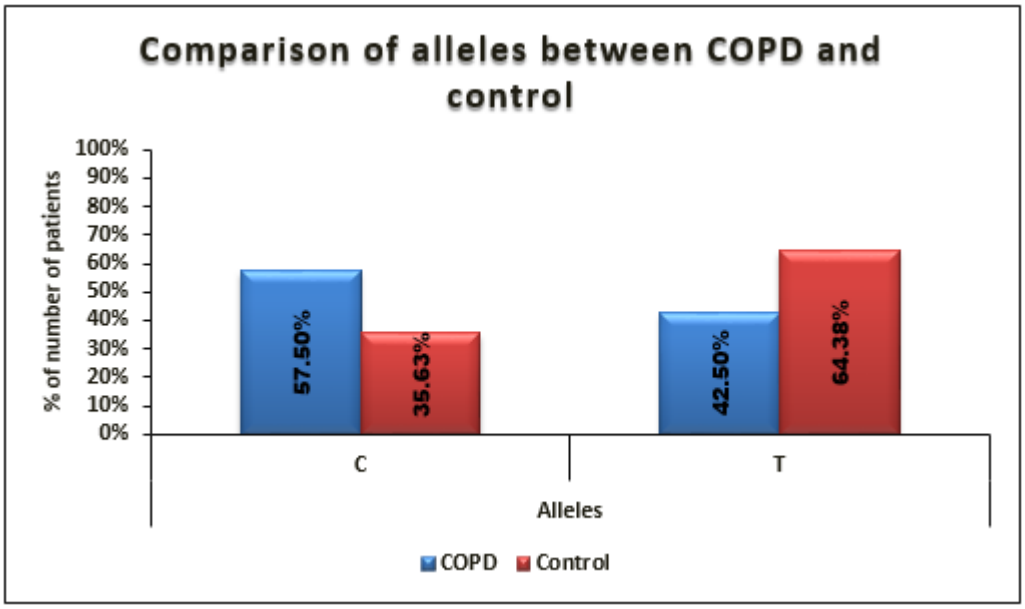

Figure 3: Comparison of alleles between COPD and control

Allele distribution differed significantly between COPD and control groups (p value < 0.0001). The C allele was more frequent in COPD (57.50% vs. 35.63%), whereas the T allele was more frequent in controls (64.38% vs. 42.50%). Compared to the C allele, the T allele showed a significantly lower odds ratio of 0.411 (95% CI: 0.262–0.645) for COPD compared to controls (Table 3, Figure 3).

Table 4: Hardy-Weinberg equilibrium in cases

Genotype	Observed frequency	Expected frequency	p value
CC	26 (32.50%)	26.5	0.83†
CT	40 (50%)	39.1	
TT	14 (17.50%)	14.45	

† Chi square test

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This p value is non-significant, the null hypothesis is not rejected, and so the population is in Hardy-Weinberg equilibrium (Table 4).

Table 5: Hardy-Weinberg equilibrium in controls

Genotype	Observed frequency	Expected frequency	p value
CC	12 (15%)	10.14	0.36†
CT	33 (41.25%)	36.7	
TT	35 (43.75%)	33.14	

† Chi square test

This p value is non-significant, the null hypothesis is not rejected, and so the population is in Hardy-Weinberg equilibrium (Table 5).

DISCUSSION

COPD, a major cause of mortality and morbidity, is becoming an even more serious health issue. Environmental and genetic factors interact to develop COPD, a complicated genetic illness [3]. An inflammatory response, structural remodelling that thickens the airway wall and alveolar destruction that results in emphysema are the main anomalies in COPD patients.

Nuclear factor (NF)-kB, a transcription factor that boosts the expression of multiple inflammatory genes, is activated by pro-inflammatory cytokines, including as IL-1, TNF-alpha and IL-6, which are higher in COPD and appear to worsen inflammation [9]. The primary environmental risk factor for COPD development is cigarette smoking. The other environmental risk factors (diet, lower socioeconomic level, etc.) may interact with smoking to raise the risk of COPD, although they are probably far less significant than cigarette smoking. However, only 10–20% of smokers go on to develop clinically severe COPD, suggesting that the pathophysiology is influenced by genetic factors [3].

The polymorphism of the IL-1 gene cluster is linked to a number of inflammatory illness [10].

This study was conducted on 160 participants in which 80 were cases having COPD and 80 were controls. The serum IL-1β levels were 2.28 pg/dl (1.7–2.685 pg/dl). Which were significantly higher in COPD patients than 1.52 pg/dl (1.05–2.17 pg/dl) in controls. IL-1β involved in neutrophil recruitment, airway remodelling, oxidative stress, and epithelial damage—all hallmark processes in COPD. The elevated levels observed in this study support the concept of persistent systemic inflammation in COPD, these findings agree with previous studies reporting increased IL-1β in COPD patients, linking it to disease severity, exacerbation frequency, and airflow limitation. Although IL-1β levels varied within both groups, the clear elevation in COPD indicates its role as an inflammatory biomarker. Studies also showed that levels of serum IL-17 and IL-1β were significantly higher in patients with COPD than those in the healthy control group [11]. According to a study done in southern India, patients with COPD had higher levels of pro-inflammatory cytokines such IL-1β and TNF-α [9].

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The genotype distribution shows CC genotype was more frequent in COPD (32.5% vs 15%), while TT was more frequent in controls (17.5% vs 43.75%) (p= 0.0006). While CT genotype frequencies were similar (50% vs 41.25%). The C allele was more common in COPD (57.5% vs 35.63%), while the T allele predominated in controls (64.38% vs 42.5%) (p<0.0001). In respective of CC, the TT genotype was protective. T allele also associated with lower COPD risk, this suggests the C allele may be a risk allele for COPD.

A Korean study conducted in the year 2008 concluded that out of the four polymorphisms of the IL1β gene (1464G / C, 3737C / T, 511C / T, and 31T / C), the 511C / T and 31T / C polymorphisms, contribute to the risk of COPD [12]. Polymorphic variations of the TNF α, LTA, and IL1β genes are risk markers for the development of COPD, according to a study done on the Russian population that demonstrates the molecular underpinnings of the disease's development and phenotypic variability [13].

In our study both cases and controls were in HWE (Hardy-Weinberg equilibrium**)**, indicating the population is genetically stable, and the genotype distributions are not biased by sampling error. There was no significant association observed between IL-1β genotype and age or gender in either group. IL-1β levels were also not significantly different across genotypes in COPD or controls. This implies that while the C allele increases susceptibility to COPD, it does not significantly affect the systemic IL-1β concentration, which may be influenced more by disease status or environmental exposures rather than genotype alone**.**

CONCLUSION

Chronic Obstructive Pulmonary Disease (COPD) represents a significant global health challenge, notably affecting millions of individuals worldwide. The interplay between genetic factors, environmental exposures, and lifestyle choices, particularly smoking, underlines the complexity of this heterogeneous condition.

The disease is marked by persistent airflow obstruction, significantly contributing to substantial mortality rates. The study highlights the significant role of the pro-inflammatory cytokine IL-1β in the context of COPD. The elevated IL-1β serum levels in COPD patients reinforce the concept of persistent systemic inflammation associated with the disease, aligning with findings from previous research.

The genotype analysis indicates that the C allele may serve as a risk allele for the development of COPD, Despite the association of the C allele with increased susceptibility to COPD, it appears that IL-1β concentrations are more influenced by disease status and environmental factors than by genotype alone.

This underscores the multifactorial nature of COPD, where both genetic predispositions and environmental exposures converge to influence disease severity and outcomes. To understand the underlying mechanisms and investigate potential treatment targets within the inflammatory pathways involved in COPD, this area merits further exploration.

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