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MCP-1, IL-6, TMPRSS2, and CRP Gene Expression in Peripheral Blood of COVID-19 Patients with a History of COPD and Comparison with Healthy Individuals

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ABSTRACT

Introduction: Chronic obstructive pulmonary disease (COPD) is associated with persistent airway inflammation and an increased risk of adverse outcomes following coronavirus disease 2019 (COVID-19). While the lower airways have been the primary focus of most studies, the upper respiratory tract plays a critical role in SARS-CoV-2 entry and early symptom development. Inflammatory mediators, including interleukin-6 (IL-6), monocyte chemoattractant protein-1 (MCP-1), and C-reactive protein (CRP), as well as the viral entry–related protease TMPRSS2, are involved in both COPD-associated inflammation and COVID-19 pathogenesis. This study investigated the expression of these markers in COVID-19 patients with underlying COPD.

Materials and Methods: In this case–control study, peripheral blood samples were collected from COVID-19 patients with a documented history of COPD and from age- and sex-matched healthy controls. Total RNA was extracted, reverse transcribed into cDNA, and relative mRNA expression levels of IL-6, MCP-1, CRP, and TMPRSS2 were quantified using real-time polymerase chain reaction. Statistical analyses were performed using parametric tests, with statistical significance defined as $p < 0.05$.

Results: Compared with healthy controls, COVID-19 patients with COPD demonstrated significantly higher mRNA expression levels of IL-6, MCP-1, and CRP ($p < 0.05$). TMPRSS2 expression was also elevated in the patient group, indicating a potential contribution to enhanced viral entry. Overall, the findings revealed a distinct inflammatory expression profile in patients with COPD during SARS-CoV-2 infection.

Conclusion: The increased expression of IL-6, MCP-1, CRP, and TMPRSS2 in COVID-19 patients with underlying COPD suggests a combined effect of chronic inflammation and viral entry mechanisms. These markers may be valuable as accessible blood-based indicators of disease severity, although further studies are needed.

Keywords: COVID-19; COPD; Gene expression; IL-6; MCP-1; TMPRSS2; C-reactive protein.

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Introduction

Chronic obstructive pulmonary disease (COPD) is a significant global health burden characterized by persistent airflow limitation, chronic airway inflammation, and progressive structural remodeling of the lungs [1]. Although COPD has traditionally been regarded as a disease primarily affecting the lower airways, increasing evidence indicates that the upper respiratory tract—including the nasal cavity, pharynx, and nasopharynx—also plays a significant role in disease pathogenesis [2]. Patients with COPD frequently experience upper airway symptoms, including nasal congestion, excessive mucus production, chronic cough, and pharyngeal irritation, reflecting mucosal barrier dysfunction and dysregulated local immune responses [3].

Chronic airway inflammation in COPD is mediated by a complex network of pro-inflammatory cytokines and chemokines, among which interleukin-6 (IL-6) and monocyte chemoattractant protein-1 (MCP-1/CCL2) are particularly prominent [4-6]. IL-6 contributes to both local immune activation and systemic inflammation by inducing hepatic synthesis of C-reactive protein (CRP), thereby linking airway pathology to systemic inflammatory burden [6]. MCP-1 plays a key role in recruiting monocytes and macrophages to inflamed mucosal tissues, promoting mucus hypersecretion, tissue edema, and airway remodeling [4]. Elevated circulating levels of IL-6, MCP-1, and CRP have been associated with increased disease severity, frequent exacerbations, and adverse clinical outcomes in COPD patients [7].

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the etiologic agent of coronavirus disease 2019 (COVID-19), primarily enters the host through the upper airway epithelium via angiotensin-converting enzyme 2 (ACE2) and transmembrane serine protease 2 (TMPRSS2)-mediated mechanisms [8]. TMPRSS2 facilitates viral entry by priming the spike protein, enabling efficient epithelial infection and replication [9]. Notably, TMPRSS2 expression is upregulated in the airway epithelium of patients with COPD, potentially creating a more permissive environment for SARS-CoV-2 infection in the context of impaired antiviral interferon responses [10]. Clinical observations indicate that COVID-19 patients with underlying COPD experience more severe upper respiratory manifestations, including pronounced nasal obstruction, excessive secretions, persistent cough, and early onset dyspnea, suggesting a synergistic interaction between chronic airway inflammation and viral infection [11-13]. In this setting, IL-6, MCP-1, and CRP—already elevated in COPD—may contribute to exaggerated inflammatory responses during COVID-19, correlating with increased viral burden, acute respiratory distress syndrome (ARDS) progression, and mortality [13]. This inflammatory convergence is likely to exacerbate mucosal injury and immune dysregulation in the upper airways. Despite extensive investigation of lower airway pathology in COPD and COVID-19, the molecular mechanisms underlying upper airway involvement in patients with COPD infected with SARS-CoV-2 remain insufficiently explored [2]. In particular, the coordinated expression of inflammatory mediators (IL-6, MCP-1, and CRP) alongside viral entry–related genes such as TMPRSS2, and their relationship to upper airway symptomatology, represents a critical knowledge gap. Therefore, examining the expression pattern of these genes in COVID-19 patients with a history of COPD can provide valuable information. Therefore, the aim of this study is to examine the expression of the aforementioned genes in the peripheral blood of COVID-19 patients with a history of COPD and compare it with healthy individuals.

Materials and Methods

Study Design and Participants

This case–control study investigated the expression of interleukin-6 (IL-6), monocyte chemoattractant protein-1 (MCP-1), transmembrane serine protease 2 (TMPRSS2), and C-reactive protein (CRP) genes in peripheral blood samples from COVID-19 patients with a confirmed history of chronic obstructive pulmonary disease (COPD) compared with healthy controls. Ethical approval was obtained from the institutional Ethics Committee (Approval Code: IR.SBMU.NRITLD.REC.1404.102). Written informed consent was obtained from all participants before enrollment, A total of 60 participants were enrolled: 30 COVID-19 patients with documented COPD (case group) and 30 age- and sex-matched healthy individuals without a history of pulmonary or infectious diseases (control group). Patients were recruited from the Department of Oral and Maxillofacial Pathology, School of Dentistry, Tehran University of Medical Sciences.

Inclusion criteria

1. age between 40 and 75 years with confirmed COPD (GOLD stages II–III) and active SARS-CoV-2 infection.

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2. presence of upper respiratory symptoms, including nasal congestion, excessive secretions, or cough, with a visual analog scale score ≥ 4.

3. stable COPD without exacerbation during the previous four weeks.

Exclusion criteria

1. Active malignancy, autoimmune disease, or immunosuppressive conditions

2. Use of corticosteroids, immunomodulatory agents, or antibiotics within four weeks before enrollment.

3. Hospitalization for COVID-19 pneumonia or oxygen saturation < 92%.

4. Smoking cessation within the previous six months or use of electronic cigarettes

Sample Collection and RNA Extraction

Peripheral venous blood (2 mL) was collected from each participant into EDTA-containing tubes. Immediately after collection, samples were processed for total RNA extraction according to the manufacturer's instructions [14-16].

RNA Extraction and Quantification

Total RNA, including small RNAs, was extracted from plasma using RNA Blood Mini Kit (Qiagen, Cat. No. 52304) according to the manufacturer's protocol. All procedures were conducted under RNase-free conditions using certified RNase-free microtubes and filter tips. RNA purity and concentration were assessed using a NanoDrop spectrophotometer (NanoDrop Technologies), and only samples with an A260/A280 ratio between 1.8 and 2.1 were used for downstream analysis [14].

cDNA Synthesis and Real-Time RT-PCR

cDNA was synthesized from 1 µg RNA, using the Viva 2-Step RT-PCR Kit (Cat. No. RTPL12). The 18S rRNA gene was used as the reference control, according to the manufacturer's instructions. Synthesized cDNA was stored at –20 °C until Real-time RT-PCR analysis.

Quantitative Real-Time PCR

Real-time RT-PCR was performed using CinnaGreen qPCR Mix 2X (Cat. No. MM2041). The genes of interest and their respective primers are presented in [Table 1]. Each 20 µL reaction contained 4 µL master mix, 1 µL of forward and 1 µL of reverseprimers, 2 µL cDNA, and nuclease-free water to reach the final volume [17,18]. Thermal cycling conditions were as follows:

- Initial activation: 95 °C for 5 minutes.
- 40 cycles of:
- Denaturation: 94 °C for 15 seconds.
- Primer Annealing: 56 °C for 60 seconds.
- Extension: 72 °C for 25 seconds.
- Final extension at 72°C for 5 minutes.

Relative gene expression was calculated using the 2^{-} ΔΔCt method to compare patients with severe symptoms to those with mild symptoms.

Statistical Analysis

Threshold cycle (Ct) values obtained from Real-time RT-PCR were analyzed using SPSS software (version 20, IBM Corp., USA). Relative gene expression levels were calculated using the ΔCt method and normalized to the reference gene. Data normality was assessed using the Shapiro–Wilk test, and homogeneity of variance was evaluated with Levene's test. Between-group differences in gene expression were analyzed using independent t-tests. Correlations between gene expression and clinical variables, including age, sex, and smoking status, were assessed using Spearman's rank correlation coefficient. Statistical significance was defined as $p < 0.05$.

Results

Participant Characteristics

A total of 60 participants were enrolled, including 30 COVID-19 patients with a confirmed history of COPD (case group) and 30 healthy controls. The cohorts were comparable in baseline demographics, with mean ages of 45.8 ± 10.8 years for cases and 48.2 ± 12.5 years for controls ($p > 0.05$). Gender distribution did not differ significantly between groups ($p > 0.05$), confirming balanced comparability.

Gene Expression Profiles

Real-time RT-qPCR analysis demonstrated a normal distribution of ΔCt values for all target genes (Shapiro-Wilk test: all $p > 0.05$). In the COPD-COVID-19 cohort, MCP-1 was detectable in 28/30 participants (93.3%), IL-6 in 26/30 (86.7%), TMPRSS2 in 25/30 (83.3%), and CRP in 27/30 (90.0%). In contrast, healthy controls exhibited substantially lower positivity: MCP-1 in 8/30 (26.7%), IL-6 in 7/30 (23.3%), TMPRSS2 in 10/30 (33.3%), and CRP in 14/30 (46.7%).

Between-group comparisons revealed highly significant differences for all markers (all $p < 0.001$, independent t-test; Figures 1-4).

Fold Change Analysis

Relative expression calculated by the $2^{-\Delta\Delta Ct}$ method revealed that MCP-1 expression increased 2.3-fold, IL-6 by 2.1-fold, TMPRSS2 by 2.5-fold, and CRP by 2.6-fold compared to controls (all $p < 0.001$). Boxplot distributions demonstrated clear separation between cohorts with minimal overlap, and Levene's test indicated consistent variance across groups (all $p > 0.10$), validating parametric test assumptions [Figures 5-8].

Clinical Correlations

Gene expression levels of IL-6, MCP-1, CRP, and TMPRSS2 were higher in COVID-19 patients with COPD compared to healthy controls. Upregulation of IL-6 was associated with increased nasal congestion and mucus production. Elevated MCP-1 expression corresponded with more persistent chronic cough and pharyngeal inflammation. Higher CRP levels were observed in patients with more severe upper respiratory symptoms, and TMPRSS2 expression was associated with early onset and the intensity of COVID-19 symptoms. These findings demonstrate a clear molecular–clinical linkage in COVID-19 patients with COPD.

Table 1. Primer sequences and amplicon sizes.

Parameters	*MCP-1*	*IL-6*	*TMPRSS2*	*CRP*	*18s rRNA*
Forward Primer (5′–3′)	*AGAATCACCAG-CAGCAAGTGTCC*	*GGTACATCCTC-GACGGCATCT*	*AAGTCCTCAG-GAGCACTGTGCA*	*TTTTCTCGTATG-CCACCAAG*	*GTAACCCGTT-GAACCCCATT*
Length of primer	*23*	*21*	*22*	*20*	*20*
Reverse Primer (5′–3′)	*TCCTGAAC-CCACTTCT-GCTTGG*	*GTG-CCTCTTTGCT-GCTTTCAC*	*CAGAACCTC-CAAAGCAAGA-CAGC*	*TTTCCAAT-GTCTCCCACCAG*	*CCATCCAATCGG-TAGTAGCG*
Length of primer	*22*	*21*	*23*	*20*	*20*
Annealing Temperature (°C)	*62*	*59*	*61*	*57*	*53.5°C*

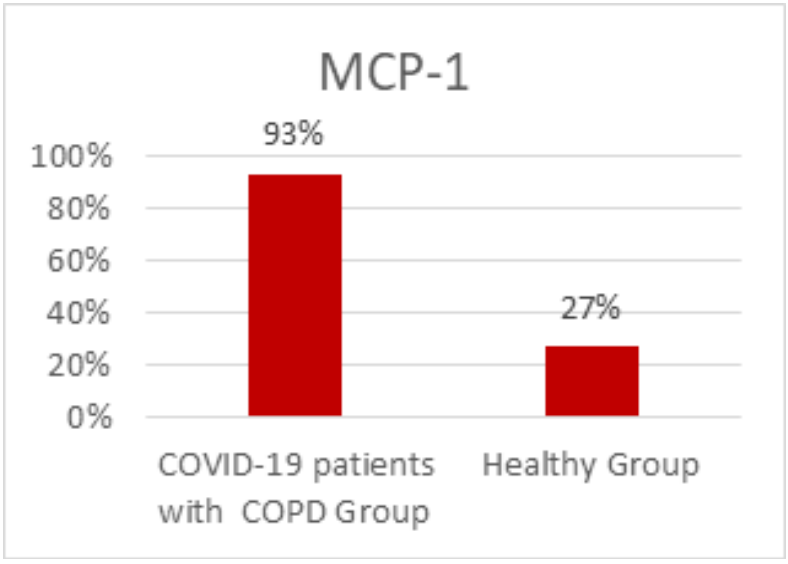

Figure 1. MCP-1 gene expression levels in COPD–COVID-19 patients versus healthy controls.

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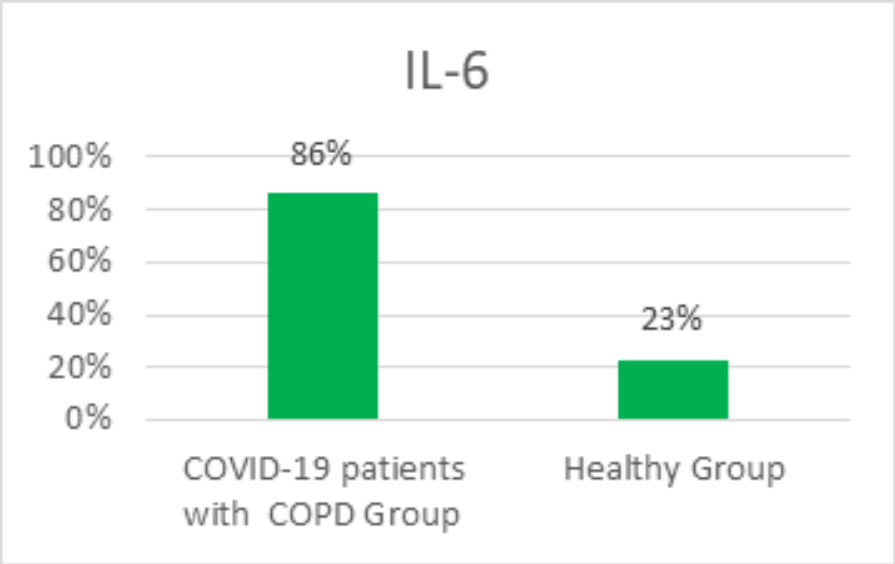

Figure 2. IL-6 gene expression levels in COPD–COVID-19 patients versus healthy controls.

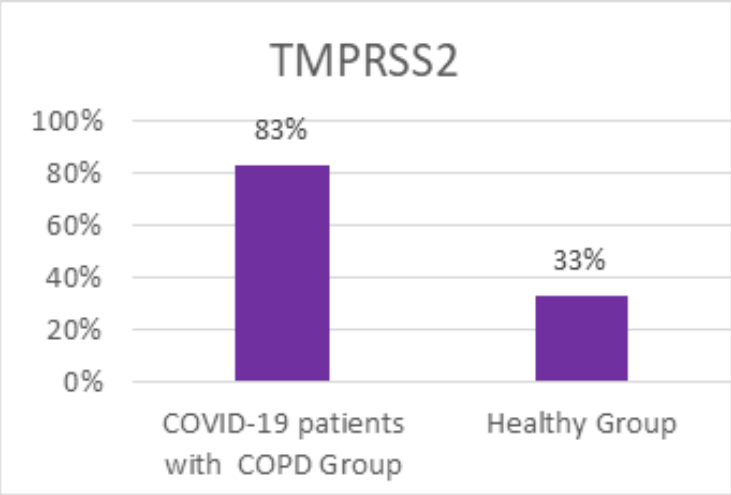

Figure 3. TMPRSS2 gene expression levels in COPD–COVID-19 patients versus healthy controls.

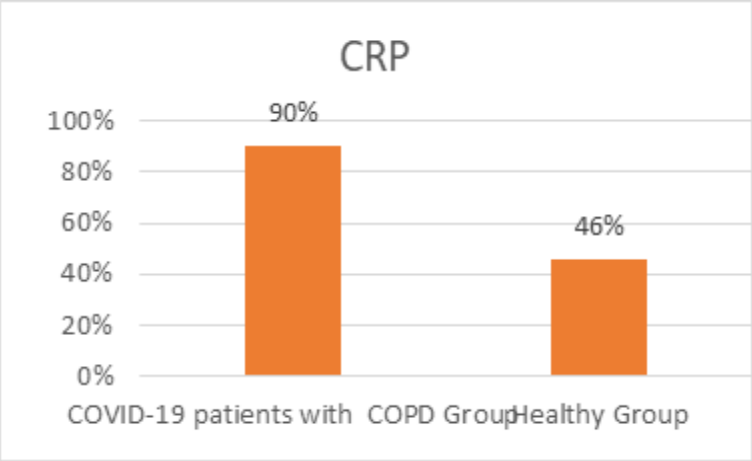

Figure 4. CRP gene expression levels in COPD–COVID-19 patients versus healthy controls.

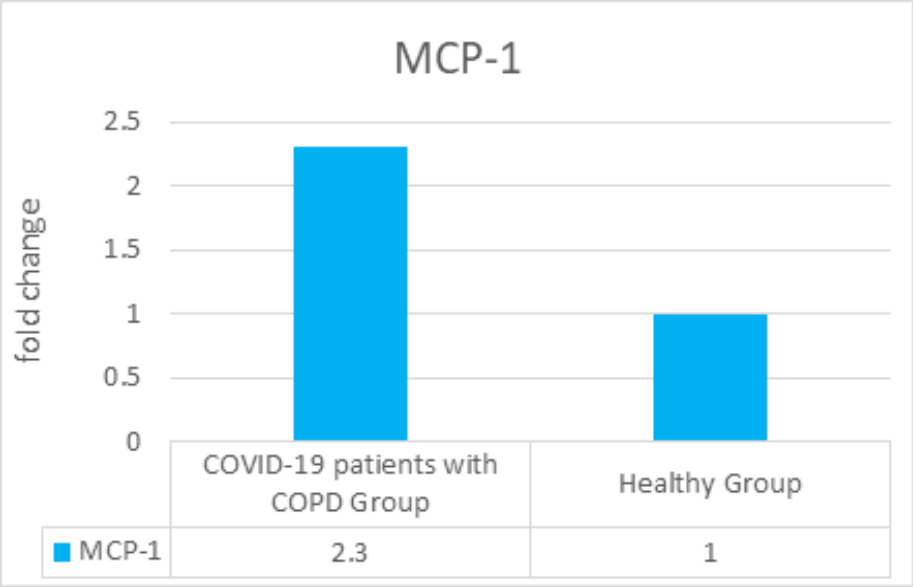

Figure 5. Differences in MCP-1 gene expression between the COPD–COVID-19 group and the healthy control group.

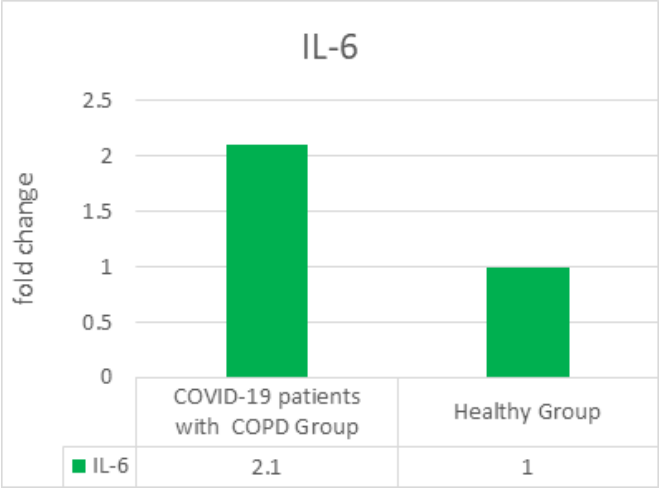

Figure 6. Differences in IL-6 gene expression between the COPD–COVID-19 group and the healthy control group.

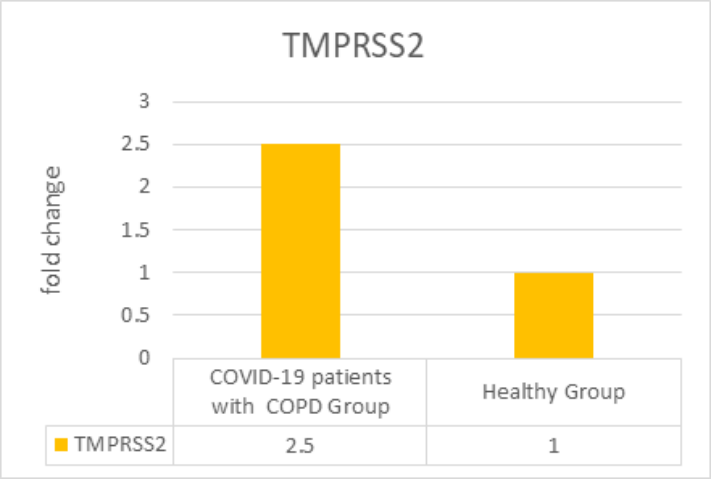

Figure 7. Differences in TMPRSS2 gene expression between the COPD–COVID-19 group and the healthy control group.

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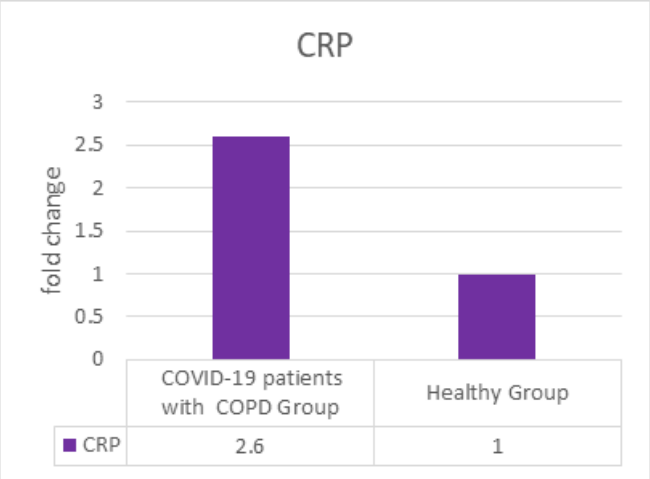

Figure 8. Differences in CRP gene expression between the COPD–COVID-19 group and the healthy control group.

Discussion

This study demonstrated an evident upregulation of MCP-1, IL-6, CRP, and TMPRSS2 gene expression in patients with COVID-19 and underlying COPD compared with healthy controls. These findings directly support the study hypothesis and indicate that pre-existing chronic inflammation in COPD amplifies both inflammatory and viral entry–related molecular pathways during SARS-CoV-2 infection, particularly affecting the upper respiratory tract. The elevated expression of IL-6, MCP-1, and CRP reflects pronounced activation of inflammatory cascades in this comorbid population. IL-6 is a central mediator of mucosal inflammation and has been repeatedly linked to nasal congestion, increased secretions, and persistent cough in COPD patients with COVID-19 [19]. Its increased expression in the present study underscores its role in bridging local airway inflammation with systemic immune activation.

MCP-1, a key chemokine responsible for monocyte recruitment, plays a critical role in sustaining inflammatory cell infiltration within the respiratory mucosa [20]. The observed increase in MCP-1 expression suggests enhanced immune cell migration to the upper airway, contributing to mucosal edema and worsening of upper respiratory symptoms. This finding is consistent with previous studies reporting a direct association between MCP-1 levels and nasal and pharyngeal symptom severity in COPD patients [21]. In parallel, increased CRP expression indicates a heightened systemic inflammatory state. CRP is known to promote vascular permeability and amplify innate immune responses, thereby exacerbating mucosal inflammation in the upper airways [22]. The combined elevation of IL-6 and CRP supports the presence of a sustained inflammatory loop that may intensify clinical manifestations in patients with COPD and COVID-19. Notably, the upregulation of TMPRSS2 suggests increased susceptibility of respiratory epithelial cells to SARS-CoV-2 entry. TMPRSS2 facilitates viral spike protein priming and is overexpressed in the nasal epithelium of patients with COPD [10]. Its increased expression in this study may partly explain the enhanced viral invasion and more severe early upper airway symptoms observed in this population. Chronic inflammation in COPD is associated with impaired epithelial barrier integrity, reduced mucociliary clearance, and dysregulated immune responses [23]. These alterations create a permissive environment for viral infection and exaggerated inflammatory signaling. The concurrent elevation of inflammatory mediators and TMPRSS2 observed here suggests a synergistic mechanism through which chronic airway inflammation and viral entry pathways interact to worsen upper respiratory involvement in COVID-19 [24,25]. Several limitations should be considered. The relatively small sample size may limit generalizability, and gene expression was assessed at the mRNA level in peripheral blood, which may not fully reflect localized airway events. Future studies incorporating protein-level validation and tissue-based analyses are warranted.

Conclusion

In summary, the present findings indicate that upper respiratory symptoms in COVID-19 patients with underlying COPD arise from the combined effects of chronic inflammation and enhanced viral entry mechanisms. Therefore, examining the expression of IL-6, MCP-1, TMPRSS2, and CRP genes in the peripheral blood of patients with COVID-19 with a history of COPD may be of effective help in continuing the treat-

ment and recovery of patients.

Conflict of Interest

There is no conflict of interest to declare.

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