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The Association of Interferon Lambda 4 (IFNL4) rs12979860 Polymorphism with COVID-19 Outcome

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ABSTRACT

Background & Objective: Coronavirus Disease 2019 (COVID-19) has a different fatality rate and clinical effects and exhibits a wide range of clinical symptoms, from asymptomatic to critically ill infections that require intensive care units. IFNL4 has shown antiviral activity against RNA viruses. IFNL4 is classified as a type-III interferon the newest addition to the interferon lambda protein family. The receptor of INFL4 (IFNLR1) is primarily restricted to tissues of epithelial origin. Therefore, interferon lambda proteins may have evolved specifically to protect the epithelium. Genetic polymorphism is one of the factors impacting COVID-19 outcomes. Variants of the IFNL4 gene have been linked to the clinical response to some viral infections. Many studies have found a correlation between the T Allele of IFNL4 (rs12979860) SNP and COVID-19 outcomes. Our goal was to investigate the correlation between such a polymorphism and the severity of COVID-19 infection.

Materials & Methods: A total of 150 positive COVID-19 patients (75 severe, 75 mild/moderate) were enrolled in the present study. The polymorphism was genotyped using TaqMan real-time PCR (polymerase chain reaction).

Results: The present study found the TT genotype of the rs12979860 variants was significantly prevalent in severe patients compared to mild and moderate infection patients, suggesting that this genotype could be a risk factor for COVID-19.

Conclusion: Between the two study groups, there is a statistically significant association between COVID-19 outcome and the T Allele of the IFNL4 (rs12979860) SNP.

Keywords: IFNL4, rs12979860, COVID-19, SARS-CoV-2

1. Introduction

Coronavirus Disease 2019 (COVID-19), which is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), was found to have a significant fatality rate (1-3). The symptoms and progression of SARS-CoV-2 infection vary greatly among patients, and this variation cannot be attributed to age or pre-existing conditions alone (4). Research increasingly shows that major shifts in cytokine levels in the blood are a key driver of the disease process in severe COVID-19 (5). The cytokine storm is a key factor in triggering the pathological chain of events—including plasma leakage, vascular permeability, and coagulopathy—that leads to life-threatening respiratory symptoms in COVID-19 (6). The infection progresses when SARS-CoV-2 moves into the lung's air sacs (alveoli) and attaches to type 2 pneumocytes, initiating viral replication. Following replication, the self-destruction of these lung cells causes them to emit inflammatory mediators, which in turn recruit immune cells like macrophages and neutrophils (7).

The stimulated macrophages further secrete cytokines such as IL-1, IL-6 and Tumor Necrotic Factor α (TNF-α). The released cytokines trigger a “cytokine storm”, which stimulates the release of vascular endothelial growth factor (VEGF), monocyte chemoattractant protein-1 (MCP-1), IL-8, and additional IL-6, as well as reduced E-

cadherin expression on endothelial cells causing vasodilation and increased capillary permeability (8). The resulting plasma leakage causes interstitial and alveolar edema. The alveolar fluid decreases surfactant levels, leading to increased surface tension and subsequent alveolar collapse. Radiologically, this edema and collapse manifest as multiple peripheral ground-glass opacities in both lungs, a frequent observation in patients (9). The wide variation in COVID-19 symptom severity suggests that an individual's genetic makeup significantly influences their susceptibility to the virus and the seriousness of their illness (10, 11). Investigations into the role of host genetics in COVID-19 progression have pinpointed numerous susceptibility genes, albeit with differing levels of supporting evidence (12-15). Interferon lambda 4 (IFNL4) gene encodes the IFNL4 protein, which contributes to the innate immune response against viral infection. This protein is categorized as type-III interferons because its signal is through IFNLR1 and IL10R2 (16, 17). Contrary to the widespread expression of IFN-α receptors, IFNLR1 is primarily present in epithelial tissues (18, 19). A human IFNL4 single nucleotide polymorphism (SNP) rs12979860 was associated with good response to treatment of chronic hepatitis C (20, 21) and self-limited HCV infection (22, 23). Also, IFNL4 (rs12979860) has been linked to how SARS-COV-2 infection progresses, with the T allele that have an influence on clinical progression to severity (24, 25). Our objective was to determine whether the IFNL4 (rs12979860) was associated with the severity of COVID-19 infection.

2. Materials and Methods

Blood samples were collected from 150 COVID-19 patients (75 severe, 75 mild/moderate) who were hospitalized at the internal Teaching Hospital in Sulimaniyah city/ Iraq from October 2022 to March 2023 due to respiratory symptoms and were positive for SARS-CoV-2 (PCR-test). All subjects, were non-vaccinated previously, and there were 83 males (mean age 40 years) and 67 females (mean age 37 years). Each subject signed an informed consent for the genetic study approved by the Ethical Research Committee of the College of Medicine /AL-Nahrain University. DNA was extracted from the blood samples of each subject by using conventional kits addbio genomic DNA extraction kit according to the manufacturer's instructions (addbio, South Korea). IFNL4 (rs12979860) C/T SNP was determined in all the patients by real time PCR genotyping with a TaqMan probe (Fisher Scientific, assay id. C___7820464_10) with Probe sequence of IFNL4 gene rs12979860:

TGAACCAGGGAGCTCCCCGAAGGCG[C/T] GAACCAGGGTTGAATTGCACTCCGC. The PCR amplification reaction was done according to the manufacturer's instructions using the Add Star Taq master mix PCR kit (addbio, Korea). The PCR reaction was done by mixing 5 µL of Green Master Mix and 0.5 µL of each primer at 10 pmol/L concentration, 0.5 µL of each probe, 0.5 µL of MgCl2 and 2 µL of DNA template. Using nuclease-free water, the capacity was increased to 10 µL. The thermocycler (Bio-Rad, USA) was set up for an initial denaturation phase of 10 min at 95 °C, followed by 40 cycles of denaturation at 94 °C for 30 s, annealing at 50 °C for 30 s, extension at 72 °C for 40 s, and a final extension phase of 7 min at 72 °C. Detection genotyping kits based on the use of 2 fluorophores. These are based on two probes stained with two different fluorophore dyes, VIC and FAM. The probe stained with FAM indicates the presence of the Mutant Allele (C and T) of IFITM3 rs12252 IFNL4 rs12979860 respectively. The amplification signal for each dye will be considered whenever the detector curve crosses its threshold value. Therefore, samples will be considered to have the wild allele whenever they display a VIC positive signal. By contrast, samples will be considered having a Mutant allele whenever they display a FAM positive signal. Furthermore, samples will be considered Heterozygous of wild and mutant alleles whenever they display a VIC and FAM positive signal.

2.1 Statistical analysis

Data were analyzed with SPSS v25.0. Group comparisons of allele and genotype frequencies were performed using linear logistic regression. The relationship between COVID-19 severity and the IFNL4 gene (rs12979860) was determined with multiple logistic regression (linear generalized model). Statistical significance was set at $p \leq 0.05$.

3. Result

Patients with mild-moderate infection showed a lower mean age (45.58±15.96 years) than those with severe infection (57.73±10.85 years) with highly significant differences ($p < 0.001$), females were more frequent in both group (54.67% vs. 52.00%), the difference was not significant ($p < 0.56$) Table 1.

The wild homozygous genotype (CC) was more frequent among mild/moderate group than severe group (73.33% vs. 52.33%) with a significant difference. In contrast, the heterozygous genotype (TC) and mutant homozygous genotype (TT) were more common among Sever group (29.33% and 18.33%, respectively) than mild/moderate group (18.6% and 8.00%, respectively) with significant differences (OR= 0.45, 95%CI= 0.19-1.06, and OR= 0.30, 95%CI= 0.09-0.94, p= 0.021, respectively).

Analysis of allele distribution (Figure1) revealed a higher frequency of T allele among severe group than mild/moderate group (33.33% versus 17.33%) with a highly significant difference (OR= 0.27, 95%CI= 0.10-0.64, p= 0.002) Table 2.

Table 1. Demographic characteristics of the study population.

Factor	Mild-moderate	Severe	P value (sig≤0.05)
Age, years			
Mean ±SD	45.58±15.96	57.73±10.85	**<0.001**
Gender			
Male	35(45.33%)	48(48.00%)	**0.56**
Female	45(54.67%)	52(52.00%)	

Table 2. Variations of IFNL4 rs12979860 in the COVID-19 patients.

rs12979860	Mild-moderate	Severe	P value (sig≤0.05)	OR (95%CI)
Genotypes				
CC	55(73.33%)	39(52.00%)		**1.0**
CT	14(18.66%)	22(29.33%)	**0.021***	**0.45(0.19-1.06)**
TT	6(8.00%)	14(18.66%)		**0.30(0.09-0.94)**
Alleles				
C	124(82.66%)	100(66.66%)	**0.002***	**1.0**
T	26(17.33%)	50(33.33%)		**0.27(0.10-0.64)**

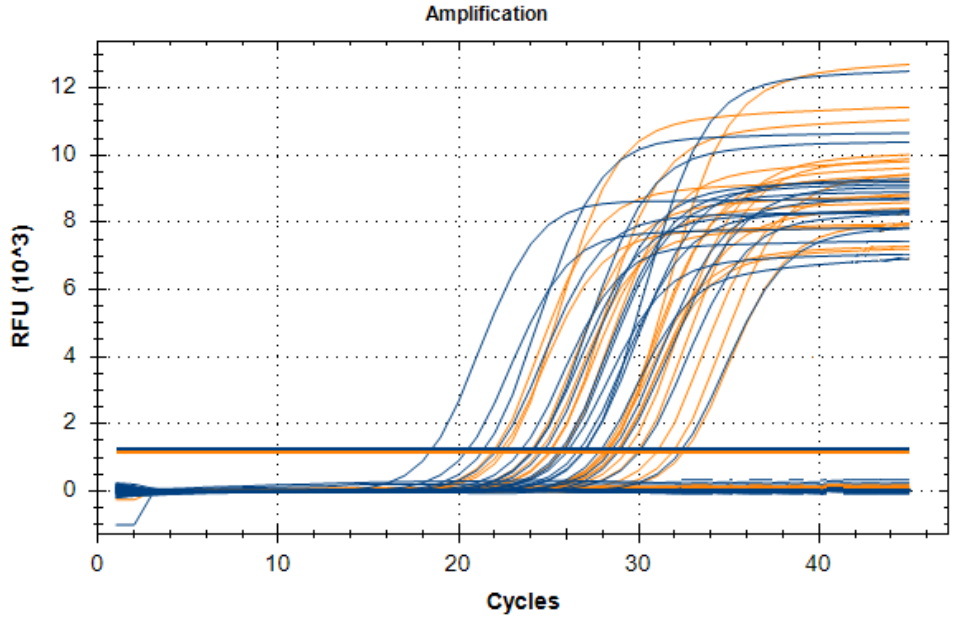

Figure 1. Genotyping of Alleles using TaqMan Real time PCR the blue line represent T Allele and orange line represent C Allele (**Prepared by Authors, 2025**).

4. Discussion

According to our results, TT genotype is significantly expressed in COVID-19 sever patients versus mild/moderate patients. Also, T allele is higher risk of COVID-19 severity. Many associations have been found between interferon lambda 4 rs12979860 polymorphism and various clinical outcomes such as viral clearance of HCV and other RNA viruses (26-28). The association between the polymorphism in the IFN-lambda gene (rs12979860) and COVID-19 has been reported in many papers (24, 25, 29-36). In this sense, a study done in Spain by Saponi-Cortes et al (24), the T allele of rs12979860 was more prevalent in COVID-19 patients than in healthy controls, indicating its potential role as a risk factor for infection (24). Furthermore, another study that included 750 SARS-CoV-2 positive patients (375 survivors and 375 nonsurvivors) disclosed that the results of that study proved that the severity of COVID-19 infection was associated with unfavorable genotypes of IFNL4 rs1297860 (TT) (25). In another study done by Zahid et al (37) included 117 subjects found that T allele more frequent in sever patients compared to mild patients with no statistical difference (37). Agwa et al (38) remarked that rs12979860 can predispose to COVID-19 and they mentioned that the highest frequency of T allele of rs12979860 was more frequent in COVID-19 patients with regard to the general population (36.16% vs. 26.40%) so they conclude that the rs12979860 polymorphism of IFNL4 can predispose to COVID-19 (38). According to the results in this study as well as the other results it's revealed that the C allele of rs12252 and T allele of rs12979860 are related to the poor outcome of the disease, which may be due to producing nonfunctional protein. However, it's essential to note that the association between IFNL4 rs12979860 T polymorphism and COVID-19 outcomes is still under study, and results may vary between different populations and studies.

5. Conclusion

We conclude that TT variation as well as T allele are associated with the severity of COVID-19 infection and the severity of disease was positively associated with advancing age among patients' groups. IFNL4, a type III interferon, is critical for antiviral defense in epithelial tissues. The rs12979860 polymorphism influences viral clearance and disease outcomes, with the T allele associated with increased risk of severe COVID-19, while the C allele may offer protective effects.

6. Declarations

6.1 Acknowledgments

None.

6.2 Ethical Considerations

This study was carried out under the ethical approval of the institutional board review (IRB) in the College of Medicine Al-Nahrain University, under the number 20211063.

6.3 Authors' Contributions

Conceptualization and Design: Sara F. Ahmed and Muhammed A. H. Aldabagh. Data Acquisition: Sara F. Ahmed and Aryan F. Khorshid. Analysis and Interpretation: Sara F. Ahmed. Methodology: Sara F. Ahmed and Muhammed A. H. Aldabagh. Writing: Sara F. Ahmed. Supervision: Sara F. Ahmed and Muhammed A. H. Aldabagh. Resources: Aryan F. Khorshid. Data Curation: Sara F. Ahmed. All authors reviewed, edited, and approved the final version of the manuscript.

6.4 Conflict of Interest

The authors declare no conflict of interest.

6.5 Fund or Financial Support

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

6.6 Using Artificial Intelligence Tools (AI Tools)

The authors were not utilized AI Tools.

7. Publisher's Note

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