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Research Article

The Impact of *CXCL12* rs1801157 Genetic Variation on Viral Skin Infections

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A B S T R A C T

Introduction: Viral skin diseases constitute a significant portion of skin disorders worldwide, encompassing a wide spectrum of illnesses exhibiting diverse clinical manifestations and varying degrees of severity. A body's immunity plays a vital role in the elimination of viral infections. The chemokines are a type of cytokine that are considered essential for the regulation of the migration of immune cells during inflammation. Among these chemokines is CXCL12 that shows pro-inflammatory characteristics and cytotoxic activity. There are many genetic variations in the gene encoding this chemokine, including rs1801157, positioned in the 3' untranslated region, resulting in the conversion of guanine to adenine.

Methods: Sixty-one patients with viral skin diseases and 31 healthy individuals were enrolled in this investigation. Patients were categorised into two groups: those infected with human papillomavirus (HPV) and those with molluscum contagiosum virus (MCV). HPV and MCV infections were clinically diagnosed by dermatologists. The genotype of the *CXCL12* rs1801157 polymorphism was determined using restriction-length polymorphism analysis on peripheral blood samples.

Results: Patients infected with HPV and MCV ranged in age from 4 to 50 years and from 2 to 64 years, respectively. The male-to-female ratio among infected patients was 1.4:1. A statistically significant association was found between the A allele of the *CXCL12* rs1801157 polymorphism and susceptibility to HPV infection, with carriers of the A allele showing an increased risk of infection (odds ratio = 2.52; 95% confidence interval: 1.18–5.38; p = 0.02). No statistically significant association was observed between this polymorphism and MCV infection.

Conclusion: This investigation proposes that *CXCL12* rs1801157 A allele may serve as a possible biogenetic marker of susceptibility to HPV infection and provides insight into the interaction between virus and host in skin infections.

Keywords: CXCL12, Gene Polymorphism, Skin Infections, RFLP, Viral Warts

Introduction

Viral skin diseases constitute a significant portion of skin disorders worldwide, encompassing a wide spectrum of illnesses exhibiting diverse clinical manifestations and varying degrees of severity.[1] Viral rashes are a prominent clinical manifestation characterised by the appearance of a rash caused by various viral agents, notably rubella virus, measles virus, and roseola virus. These infections are usually self-limiting and are diagnosed based on the morphological characteristics, appearance, distribution, and configuration of the skin lesions. Numerous viruses exhibit a direct impact on the dermal and mucosal surfaces. Herpesviruses and zoonotic pathogens, such as those causing infectious ecthyma, Ebola, monkeypox, and smallpox, are known to produce distinctive mucosal skin manifestations, whereas various other viruses are implicated in skin infections, including human papillomavirus, molluscum contagiosum virus, and those causing Gianotti-Crosti syndrome and hand-foot-mouth disease.[2]

Human papillomaviruses (HPVs) infect keratinocytes in the basal layer of epithelia[3,4] and stem cells[5,6]. Keratinocytes, as the initial target for HPV, are central to the initial stages of HPV infection and also subsequently cooperate in inducing an effective adaptive immune response. Keratinocytes are immune sentinels that are part of the body's innate immunological defence mechanism.[7]

Molluscum contagiosum is a viral skin infection caused by the Molluscum contagiosum virus (MCV), a virus with a double-stranded DNA belonging to the Poxvirus family. Although it infects both adults and children, it is most prevalent among children, producing dome-shaped pearl-like skin lesions that may appear singly or in clusters.[8–11]

Moreover, the immune system is instrumental in the eradication of viral infections. Chemokines are recognised as essential factors that regulate the progression of viral infections.[12] They are responsible for directional migration of keratinocytes, particularly in white blood cells in an inflammation. Long-term inflammation may contribute to carcinogenesis by creating an optimal milieu for tumour growth and progression.[13] Many chemokines are involved in the process of inflammation, including CXCL12, which has a pro-inflammatory feature and acts as a chemoattractant to immune cells like lymphocytes.[14]

The *CXCL12* gene is found on the long arm of chromosome 10 and was principally recognised as pre-B cell growth-stimulating factor after being cloned from a stromal cell line generated from bone marrow.[15] A variety of polymorphisms have been identified within the *CXCL12* gene, including *CXCL12* gene rs1801157 in the 3' untranslated region (UTR), which was first reported by Cheryl Winkler in 1998 and is categorised by a guanine-to-adenine replacement.[16] This investigation aimed to look at the role of *CXCL12* rs1801157 polymorphism in viral skin infections in Babylon province because of the lack of related data.

Methodology

Collection of Samples and Ethical Approval

This investigation was approved by the Dermatology Consultation at Marjan Medical City. All patients were informed of the study's purpose and provided written informed consent. Between 2018 and 2019, 62 patients (31 infected with HPV and 31 infected with MCV) were enrolled for the study, along with 31 healthy individuals (negative for both HPV and MCV), enrolled as the control group. Patients and healthy individuals ranged in age from 2 to 64 years and were of both sexes. Approximately 2.5 mL of peripheral blood was collected in tubes containing Ethylene Diamine Tetra Acetic acid (EDTA) as an anticoagulant and stored at -20°C until genomic DNA extraction. Demographic data (age and sex) were collected using a standardised questionnaire.

Detection of Skin Viral Infections

HPV and MCV infections were detected clinically by a dermatology specialist at Medical Marjan City, based on distinctive clinical characteristics.

Extraction of Genomic DNA

Favrogen Kit (Korea) was used to extract genomic DNA from peripheral white blood cells.

Detection of *CXCL*12 rs1801157 Genetic Polymorphism by Polymerase Chain Reaction

After extracting genomic DNA from peripheral blood samples, the *CXCL12* rs1801157 gene polymorphism was detected using polymerase chain reaction (PCR). Primers were used to amplify a portion of the 3'UTR region of the *CXCL12* gene, and these primers were constructed using the nucleotide sequence located in the GenBank under the number L36033.

The PCR procedure included:

- Initial denaturation at 95°C for 3 minutes
- A total of 37 cycles comprising denaturation at 94°C for 20 seconds
- Annealing at 64°C for 45 seconds
- Extension at 74°C for 30 seconds
- A final extension lasting 5 minutes

The PCR mixture consisted of 0.8 µL of each of the primers (forward and reverse), approximately 5 µL of extracted DNA, 12.5 µL of hot start green master mix (Promega, USA), and 5.9 µL of nuclease-free water.

The amplification of the *CXCL12* product yields a 293-bp fragment. PCR-RFLP was used to perform enzymatic

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restriction using the PCR product in the presence of the restriction enzyme MspI (Promega, USA). In the presence of guanine, this enzyme cuts the amplified DNA fragment into two 100- and 193-base-pair fragments, while in the presence of adenine, the 293-base-pair fragment remains intact. Electrophoresis was performed on a 2.5% agarose gel (Promega, USA), stained with RedSafe™ dye (Intron Biotechnology, Seoul, South Korea), under a voltage of 50 volts for 3 hours.

Statistical Analysis

The effect of this mutation on the infected group compared to the control group was assessed using the chi-square (X^2) test (with $p \leq 0.05$ regarded as statistically significant). Odds ratio with 95% CI was calculated to examine the possible correlations of *CXCL12* with the risk of HPV and MCV infections.[17,18]

Results

According to the clinical diagnosis, the 62 individuals included in this investigation were separated into two groups: HPV-infected patients and MCV-infected patients. They varied in age from 4 to 50 years, and from 2 to 64 years, respectively. Most of the HPV infections occurred in the age range of 16–30 years (48.4%), while the majority of MCV infections were in the age range of 1–15 years (41.94%), as shown in Table 1.

In this study, the distribution of two infected groups according sex were 18 males from 31(58.06%) and 13 females from 31(41.94%). The patient's genome was analysed to identify the *CXCL12* genotype. The PCR results showed that the polymorphism of *CXCL12* revealed two alleles, A and G, and three genotypes (AA, GA and GG), as shown in Figure 1. The A allele resulted in an undigested PCR product of 293 bp (homozygote), and the allele contributed to a digested PCR product of two 139 and 100 bp fragments, while the GA genotype resulted in three 239, 139, and 100 bp (heterozygote).

All patients had a substantial correlation between allele A and HPV infections, demonstrating that the polymorphism is linked with HPV infection independently. Allele A carriers had a higher incidence of HPV infection (OR = 2.52; 95% CI: 1.18–5.38, p = 0.02) in this study, while there was no association between allele A and MCV infections, as shown in Tables 2 and 3.

Table (3) displays the logistic regression analysis for CXCL2 Gene polymorphism among human papillomavirus (HPV)-infected patients and uninfected Controls. Compared with GG reference genotype, carriers of GA genotype displayed 2.36-fold increasing odds of human papillomavirus; however, p value (0.1288) reflects non-significant association. On the other hand, AA genotype had a stronger impact, showed a 6.61-fold increasing odds of human papillomavirus with significant association as reflected by p value at 0.0362. The negative value of constant term reflects lower baseline odds of papillomavirus among GG carriers.

Table (5) displays the logistic regression analysis for CXCL2 Gene polymorphism among Molluscum Contagiosum Virus (MCV)-infected patients and uninfected controls. Relative to GG reference genotype, carriers of GA genotype demonstrated about two-fold (OR=1.63) increased odds of MCV, but the p value (0.357) reflects non-significant association; also, AA carriers had about two-fold higher odds (OR=1.96) but the p value (0.493) reflects non-significant association.

Table 1.Distribution of Patients According to Age

Age Groups (Years)	Infected with HPV		Infected with MCV	
	n	%	n	%
1–15	10	32.30	13	41.94
16–30	15	48.40	9	29.03
31–45	5	16.10	5	16.13
More than 46	1	3.20	4	12.90
Total	31	100.00	31	100.00

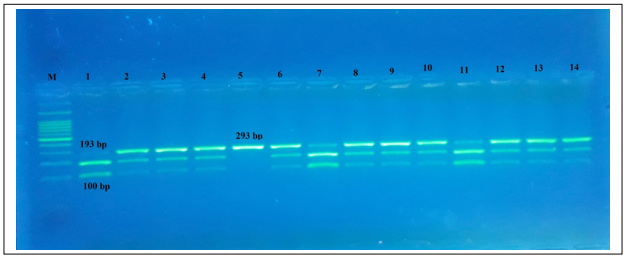

Figure 1.Agarose Gel Electrophoresis (Promega, USA) with a Concentration of 2.5% Stained with Red Safe 2TM (Intron Biotechnology, Seoul, South Korea) under 50 V for 3 h: Size Marker; Lane 1: GG Homozygote; 2,3,4,6,7,8,9,10,11,12,13,14: GA Heterozygote; 5: AA Homozygote

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Table 2.Allele and Genotype Frequencies of CXCL2 Gene Polymorphism among Human Papillomavirus (HPV)-Infected Patients and Uninfected Controls

Model	Control	HPV-infected	OR	95% CI	p Value
Codominant model					
GG[a]	17	9			
GA	12	15	2.36	(0.77–7.15)	0.1288
AA	2	7	6.61	(1.12–38.6)	0.0362*
Dominant model					
GG[a]	17	9			
GA + AA	14	22	2.96	(1.03–8.47)	0.04*
Recessive model					
GG + GA	29	24			
AA	2	7	4.22	(0.80–22.28)	0.07
Alleles					
G	46	33			
A	16	29	2.52	(1.18–5.38)	0.02*

Analysis employed a chi-square (X2) test (p≤ 0.05 regarded statistically significant); OR: Odds Ratio; CI: Confidence Interval. SPSS Inc., Chicago, Illinois, USA. *Significant p values are indicated in bold; a: Dominant.

Table 3.Logistic Regression Analysis for CXCL2 Gene Polymorphism among Human Papillomavirus (HPV)-Infected Patients and Uninfected Controls

Genotypes	B coefficient	SE	Wald χ^2	OR	95% CI	p Value
GG[a]	--	--	--	1.000		
GA	0.85	0.25	1.92	2.36	0.779 - 7.154	0.1288 [NS]
AA	1.88	0.86	4.94	6.61	1.129-38.69	0.0362*
Constant	-0.63	--	--	--	--	--

SE: Standard error; χ^2: chi-square; OR: Odds Ratio; CI: Confidence Interval; NS: Non-significant; *: significant outcomes

Table 4.Allele and Genotype Frequencies of CXCL2 Gene Polymorphism among Molluscum Contagiosum Virus (MCV)-Infected Patients and Uninfected Controls

Model	Control	MCV-infected	OR	95% CI	p Value
Codominant model					
GG[a]	17	13			
GA	12	15	1.63	(0.57–4.66)	0.3579
AA	2	3	1.96	(0.284-13.506)	0.4937
Dominant model					
GG	17	13			
GA + AA	14	18	1.6813	0.615-4.591	0.310
Recessive model					
GG + GA	29	28			
AA	2	3	1.55	(0.24–10.01)	0.6430
Alleles					
G	46	41			
A	16	21	1.47	(0.67–3.19)	0.43

Analysis employed a chi-square (X2) test (p ≤ 0.05 regarded significant); OR: Odds Ratio; CI: Confidence Interval SPSS Inc., Chicago, Illinois, USA. *Significant p values are indicated in bold; a: Dominant

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Table 5.Logistic Regression Analysis for CXCL2 Gene Polymorphism among Molluscum Contagiosum Virus (MCV)-Infected Patients and Uninfected Controls

Genotypes	B coefficient	SE	Wald χ^2	OR	95% CI	p Value
GG[a]	--	--	--	1.000		
GA	0.48	0.51	0.98	1.63	(0.57–4.66)	0.357 NS
AA	0.67	0.95	0.49	1.96	(0.284-13.506)	0.493 NS
Constant	- 0.28	--	--	--	--	--

SE: Standard error; χ^2: chi-square; OR: Odds Ratio; CI: Confidence Interval; NS: Non-significant

Discussion

Most of the HPV infections occurred in the age range of 16–30 years (84.4%). This result was consistent with the findings reported by Faik et al.[19] The result showed that most of the infected people were in the working-age group. They were exposed to numerous work accidents, which could have damaged the skin and compromised its defence, facilitating viral entry. HPV infections were common in school children who were under the age of 15 years (approximately 32%), probably due to direct contact with each other. These results are supported by the study conducted by van Haalen *et al.*[20]

The majority of MCV infections were in the age range of 1–15 years (41.5%). This result is similar to a study done by Reynolds et al., Jallab & Al-Shamarti, and Shisler.[21–23] The cause of this result may be the direct contact among children while playing and in the childcare centres.

The MCV infection in childhood was higher than in adolescence and youth, probably due to lower immunity against infections. Most of these infections occurred during shaving and by using the towels of other infected people.[24]

In this study, MCV-infected and HPV-infected are 18male:13 females; the male to female ratio is 1.4:1. This low ratio between males and females was similar to that observed in a study of the detection of MCV in the German population done by Sherwani et al.[25] The results show that males were more susceptible to HPV and MCV infections than females.

In addition to environmental and lifestyle influences, genetic factors have been reported to play a role in the persistence of HPV.[26] Genetic variations among individuals within this complex system may have a role in viral survival. Identifying susceptibility alleles is still a potential research topic since a grouping of genetic variations may govern risk factors.[27] In the current study, *CXCL12* variation rs1801157 was investigated in the HPV and MCV patients. We demonstrated that there is a positive association of allele A in the studied gene with HPV infection, while no such association was identified with MCV infection. *CXCL12* serves as a major chemokine involved in the trafficking of immune cells and local immune surveillance within epithelial tissue; therefore, genetic variations that control its expression or signalling pathways may significantly impact the interactions between host and virus. Infection with HPV is nonlytic and non-viraemic, limited solely to epithelial surfaces, relying on tightly regulated cell-mediated immunity and chemokine-driven recruitment of immune effector cells for effective viral clearance. Within this context, alterations in *CXCL12*-mediated immune cell trafficking attributable to the presence of the A allele may potentially facilitate HPV evasion of immunological surveillance and consequent persistence, leading to inadequate viral clearance and improved susceptibility to persistent infection.

Conversely, MCV usually prompts a more substantial innate and inflammatory response and is often resolved naturally in immunocompetent individuals, indicating that *CXCL12*-dependent immune responses may not be critical for the clearance of viral infection. The absence of a durable relationship between *CXCL12* polymorphism and MCV infection suggests that the effect is virus-specific rather than a general effect on cutaneous antiviral immunity. This variable association is biologically reasonable and highlights the heterogeneity of immune pathways that are operated by various viruses associated with skin infections. Collectively, these results underscore the significance of the *CXCL12* A allele as a genetic determinant in the host that selectively influences susceptibility to HPV infection, thereby emphasising the role of chemokine-mediated immune regulation in defining virus-specific outcomes.

The variation was not shown to be a risk factor for cervical cancer development in certain investigations.[28–30] However, according to a study by Okuyama et al., only a few studies have investigated whether the variation is linked to HPV risk.[31]

Extensively, *CXCL12* has been thought of as a basic proinflammatory chemokine because it attracts white blood cells to inflammatory sites and aids in their activation.[14,32] Both the decrease of cellular immunity, prompted by T helper 1 cell response, and the creation of

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the immunosuppressor Treg profile, appear to be required for neoplastic development.[33] Jaafar et al. found a significant rise in *CXCL12* expression as neoplastic lesions in epithelium advanced from pre-aggressive to aggressive malignancy, as evaluated by Immunohistochemistry (IHC) and Enzyme Linked Immunosorbent Assay(ELISA).[34] Additionally, they observed that there was no expression of *CXCL12* in ordinary glandular and squamous tissues, which agrees with Zanotta et al.'s findings, who observed that *CXCL12* levels were found to be low or absent in healthy tissue.[35] Although the effect of the rs1801157 variation in *CXCL12* on its expression or plasmatic levels in HPV infections is yet to be determined, it has been studied extensively in other illnesses and malignancies, with mixed findings. Individuals carrying Allele A have much lower levels of *CXCL12* mRNA in their circulating blood specimens compared to those with the GG genotype, according to De Oliveira et al.[36] Surprisingly, in prostate cancer patients, Hirata et al. discovered that expression of *CXCL12* was higher in carriers with the A allele compared with G allele patients.[37] In colorectal cancers, *CXCL12* immunohistochemistry revealed negative or feeble immunostaining in ordinary mucosa and dramatically increased immunostaining in tissues of cancer, particularly in well-distinguished cancers; a high percentage of patients with robust *CXCL12* immunostaining in the cytomembrane and membrane had GA or AA genotypes. On the contrary, the GG genotype was found in 88.6% of individuals with negative immunoreactivity.[38] The plasma level of *CXCL12* was not associated with the A allele or GA genotype/ AA genotype in another investigation involving colorectal cancer patients.[39] Numerous regulatory motifs are found in the 3'UTR of genes, which are targets of post-transcriptional organisation via interactions with RNA-binding proteins, long uncoding RNAs and microRNAs; all of them alter stability, localisation and turnover of mRNA. The Single Nucleotide Polymorphisms in these motifs may inhibit the binding of miRNA, resulting in the stability of mRNA transcripts and enhanced protein production.[40] Despite the contradictory nature of research on the impact of the *CXCL12* rs1801157 variation on chemokine levels,[41–44] indication suggests that miRNAs have a role in the synthesis and regulation of proteins. *In-silico* investigation found that rs1801157 is positioned in a seven-base homologous region (the fourth base is multimorphic), which is a probable goal of miR-941. As a result, the existence of the SNP may result in the deletion of the mi941 binding site. However, dual-luciferase assays were utilised in stem cells of healthy donors to explore the link between miR-941 and the 3'UTR. The co-expression of miR-941 had no effect on 3'UTR expression.[45] The current state of information concerning the interaction of the 3'UTR and miRNAs is inadequate, and more research is needed. The aberrant and unequivocal *CXCL12* gene expression in keratinocytes of HPV-productive mucosal or cutaneous abrasions provided evidence for the participation of *CXCL12* in the HPV life cycle.[46]

CXCL12 expression in keratinocytes may be stimulated by the expression of the HPV genome, thereby producing an autoreactive signalling pathway necessary for keratinocyte proliferation and migration.[47] The presence of six Sp1 binding sites in the 5'_untranslated and 5'_flanking regions of the *CXCL12* proximal promoter recommends that the Sp1 transcription factor is the primary positive controller of *CXCL12* production, providing a plausible description for this mechanism.[48] The E6 and E7 oncoproteins are expressed after infection of epithelial cells by HPV, and they may link definitively to the protein 1 transcription factor (Sp1). E6-Sp1 and E7-Sp1 complexes have the ability to move into the nucleus, where they are likely to induce *CXCL12* gene expression.[49]

Conclusions

This investigation explains that the *CXCL12* rs1801157 polymorphism, specifically allele A, is significantly linked with increased exposure to infection with HPV in the Iraqi population. No association was detected between this polymorphism and infection with MCV. These results propose that *CXCL12* rs1801157 may serve as a possible biogenetic marker of susceptibility to HPV infection and provide insights into the interaction between virus and host in skin infections. Further investigations with larger cohorts and more comprehensive analyses are necessary to explain the molecular pathways behind this association.

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