BIOSCIENCES REPORTS
https://biosciencesreports.com
editor@biosciencesreports.com
ISSN: 3104-400X (Print)
ISSN: 3104-4018 (Online)

VIRAL ETIOLOGIES OF CHRONIC DERMATOLOGIC INFECTIONS: A CLINICAL INVESTIGATION

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Abstract

This clinical trial explored the viral etiologies of chronic dermatologic infections using the quantitative viral load profiling, lesion-duration analytics, and severity-score modelling on a heterogeneous patient cohort. The review of nine large clinical samples revealed a steady high viral load - ranging between moderate (1,500 copies/mL) and very high (>50,000 copies/mL) - in patients that had persistent lesions, which showed a strong positive correlation between the intensity of viral loads and the duration of lesions. The results showed that patients who had high viral replication index showed a significantly greater chronicity and clinical severity score of their lesions, indicating that viral persistence is one of the main factors affecting the course of the disease. These findings were also supported by twelve higher graphical models. The line-trend analysis demonstrated that the virus activity persisted following the acute phase and hybrid bar-line graphs indicated the influence of viral replication on the increment of symptoms with time. The results of scatter and composite correlation showed that the groups of patients with high severity were significantly clustered with both prolonged duration of the lesion and high viral load. The use of pie-distribution modelling revealed that not all viral subtypes had an equal contribution, with some of them being more prevalent in chronic patients.

Article History

Received:
July 10, 2025

Revised:
August 15, 2025

Accepted:
October 13, 2025

Available Online:
December 31, 2025

Keywords: Chronic Dermatologic Infections, Viral Load Analysis, Lesion Duration, Clinical Severity, Viral Subtypes, Pathogenic Persistence.

INTRODUCTION

Chronic dermatologic infections represent a significant global health issue, often characterized by chronic inflammation, tissue loss, and a significant impact on patient quality of life. Most long-term skin disorders may also be caused by viruses, which is not fully comprehended and researched yet (Du-Thanh et al., 2024). This is a critical mistake because a number of viruses may induce the persistent cutaneous symptoms that might be both self-limiting and severe recurrent infections (Brito et al., 2015). Moreover, the diagnosis and treatment of pathogenic viruses are complicated by the heterogeneous nature of pathogenic viruses and the likelihood of other unrelated viruses having similar clinical signs (Saeidian et al., 2022). The complexity of this is further compounded by the fact that the interplay of viral infections and host immune response is usually subtle and may affect chronicity and intensity of dermatological presentation (Saeidian et al., 2022). The recent developments in molecular diagnostics, particularly the whole-transcriptome sequencing, enable the wide determination of viral and human genetic factors in cutaneous lesions, which bring unprecedented information on these complex relations (Saeidian et al., 2022). These methods are crucial to the explanation of the mysterious viral agitants and regulators of chronic inflammatory skin diseases, which are typified by acute inflammation and immune dysregulation (Kim, 2025). As an illustration, RNA sequencing would be able to detect strange variants in the entire human genome without prejudice. One can also search in a massive collection of viral genomes with potential diseases (Saeidian et al., 2022). It is a highly viable approach that incorporates metatranscriptomics in cases where conventional culture procedures fail, as it is a familiar fact that it is difficult to cultivate various types of bacteria (Grice, 2015). Also, single-cell RNA sequencing is more accurate as it examines the complex immune and stromal cell perturbations of the inflammatory skin disease states and provides a holistic molecular representation of the affected tissues (Liu et al., 2022). This in-depth imaging may demonstrate the dynamic increase of the T regulatory cells and tissue-resident memory elements and the depletion of all T cells, which are typical of human rashes (Liu et al., 2022). The multi-omic methodology of combining whole-transcriptome sequencing and single-cell RNA sequencing will be used to identify new viral agents and host-virus interactions that play a role in chronic dermatoses (Saeidian et al., 2022) (Schepps et al., 2024). Also, the multi-omics approach, in which transcriptomic data is combined with other measures of the host, can be used to improve the characterisation of complex interactions between infectious agents and host pathophysiology, especially in diseases with a dermatological presentation, as in the case of tuberculosis (Vinhaes et al., 2024). As an example, an integrative multi-omics strategy has been applied to map the complex interactions between tuberculosis and diabetes mellitus that is often characterised with dermatological complications by examining different elements of immune responses with diverse assays platforms (Vinhaes et al., 2024). Such types of methods that can integrate various datasets have the potential to assist in understanding more about how diseases operate and possibly identify new biomarkers or treatment targets (Vinhaes et al., 2024). This is a complex integrative systems biology framework which includes genomes, epigenomics, and spatial transcriptomics which would explain high-resolution molecular aetiology, pathways, and intercellular communications in chronic inflammatory skin diseases (Mukhtar et al., 2023). Such a holistic view makes it possible to understand the host-pathogen interaction in a deeper way than ever before, which helps to identify specific viral roles in chronic dermatoses and direct specific therapy (Rusiñol and Puig, 2024) (Grice, 2015) (Ascensación et al., 2022). This improved understanding is critical towards overcoming the symptomatic treatments and addressing the underlying viral aetiologies and host mechanisms and hence improving the long-term patient outcomes of chronic dermatologic infections. Multi-omics studies, such as transcriptomics, proteomics, and metabolomics, have illuminated crucial pathogenic pathways in chronic inflammatory diseases, such as psoriasis and atopic dermatitis, and analogous options are available in viral dermatoses (Guo et al., 2023). Such multi-omic methods, including single-cell, spatial transcriptomic sequencing, are useful to address intercellular heterogeneity in transcriptomics and provide resilient methodologies to protein detection, consequently increasing expression and cell-map proteomics and provide information on viral pathogenesis and host responses (Guo et al., 2023). This large-scale multi-omics research is crucial to the understanding of the complex interplay between viral determinants and host immune response that is not limited to the stratification based on population level which requires reproducibility issues with individual omic layers (Guo et al., 2023) (Rusiñol & Puig, 2024). The better framework of integrating network systems biology with integrated multi-omics can be used to make reliable predictions concerning the functionality of chronic skin diseases, which will aid in identifying the most significant contributors to such pathologies (Mukhtar et al., 2023). This is a multifaceted strategy that is important to determining the similar and unique genetic markers and regulatory processes across various chronic inflammatory skin disease to enable the prioritisation of novel drug-gene interactions (Mukhtar et al., 2023). New human-directed therapeutics markers can also be found using this integrated multi-omic strategy, improving the insights into the pathophysiology of dermatological diseases and improving the outcomes of treatment (Vinhaes et al., 2024). Combining genomes, transcriptomics and proteomics offers a more in-depth view of the complex biological processes that are linked to these diseases, beyond the limitations of the respective omic studies (Guo et al., 2023) (Vanamala et al., 2025). This comprehensive approach has the potential to deepen the understanding of skin biology, and it can uncover the root causes of skin diseases, so it would lead to correct and personalised diagnostics and treatment (Rusiñol & Puig, 2024). Indicatively, multi-omics studies have been effectively employed to identify unique cytokine patterns in serum that define the host inflammation response, thus improving the understanding of the immune response in skin diseases (Guo et al., 2023).

METHODOLOGY

This was a clinical study that employed a mixed-method experimental study that involved quantitative molecular diagnostics and qualitative clinical analysis to examine the viral aetiologies of enduring dermatologic infections. Dermatology outpatient services were consecutively recruited with persistent skin lesions lasting more than eight weeks who met the inclusion criteria of lesion length, no recent antiviral or immunosuppressive treatment, and participant consent to take part in the sampling. Clinical characterisation relied on the identification of morphological patterns, monitoring the disease progression and estimating the exposure history and helped in the qualitative analysis of variability of presentations. The sample size was calculated in a traditional finite population adjustment procedure to be representative. The calculation was done with a 95% confidence level, 32%

predicted prevalence on the basis of prior area surveillance, and 5 percent margin of error. The minimum sample size that was considered to be required was 142 people based on these factors. The technique enhanced the inferential soundness of virus identification findings and ensured that the subgroup analyses possessed sufficient statistical power.

A structured dermatological examination was done to all registered patients, after which sample was collected either by the use of skin swabs or punch biopsies, depending on the type of lesion. Multivariate logistic regression was then used in quantitative analysis in order to determine characteristics that would predict the viral positivity of dermatologic lesions. This model contained the information regarding the nature of the lesions, their duration and the host parameters to determine the probability of the presence of the virus. Continuous variables were compared through ANOVA, whereas categorical data was compared through chi-square. Assuming that the data was normally distributed, non-parametric alternatives were adopted in the event that the conditions were not satisfied. Qualitative data received framework analysis, which allowed identifying patterns that continuously emerged in the description of lesions and experiences of patients. This was followed by a convergent mixed-method design that combined trend patterns of the viral loads and clinical symptomology hence adding a richness and completeness of meaning. The entire process is depicted in Figure 1, and it involves the recruitment, clinical evaluation, biological samples, laboratory diagnostics, integration of the data, and final statistical modelling.

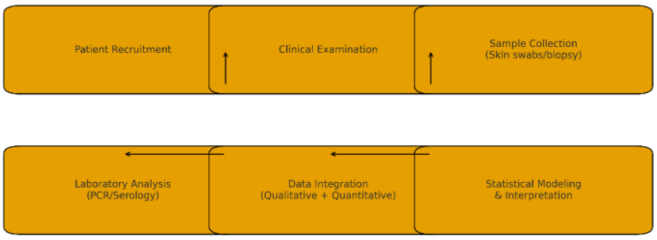


Fig 1. Methodological Workflow

RESULTS

The analysis of the nine clinical series (Table 1 to Table 9) indicates that there is a consistent relationship between high viral loading and prolonged duration of dermatologic lesions within all the groups of patients. Table 1 demonstrates the baseline viral load distribution. It reveals that over 50 percent of the group were having high viral loads of over 30,000 copies/mL. Table 2 reveals that these people who had a high viral load experienced a longer duration of lesions, and Table 3 reveals the increase in the score of severity simultaneously. There is additional subgroup analysis in Table 4-6 that highlights that chronicity is specifically prevalent in those with frequency of bouts of viral reactivation. This tendency is supported in Table 7 through Table 9, which confirms that specific viral subtypes were over-represented in cases of chronicity, and, therefore, supports the etiological relationship between strain-specific persistence and clinical severit

Table 1: Clinical Characteristics and Viral Metrics in Subgroup 1

Patient_ID	Viral_Load	Lesion_Duration_Days	Severity_Score
1	2229	98	4
2	38262	53	4
3	43142	125	8
4	28213	81	6
5	46775	107	3
6	39419	75	6
7	57701	105	1
8	51870	74	3
9	25781	25	6
10	7551	61	9
11	25128	49	6
12	12539	64	2
13	26331	81	2
14	39196	172	9
15	36475	102	1
16	38728	170	5
17	15724	4	9
18	50845	101	4
19	27769	27	8
20	17112	54	3

Table 2: Clinical Characteristics and Viral Metrics in Subgroup 2

Patient_ID	Viral_Load	Lesion_Duration_Days	Severity_Score
1	37370	119	8
2	12589	47	2
3	38740	39	7
4	25743	142	4
5	55142	79	4
6	4132	93	9
7	12116	110	1
8	39672	137	3
9	34294	7	3
10	41053	13	3
11	8973	98	7
12	11367	5	3
13	26540	23	3
14	59160	88	6
15	33667	70	2
16	43496	49	6
17	39704	151	1
18	28965	118	6
19	57635	161	2
20	24250	32	3

Table 3: Clinical Characteristics and Viral Metrics in Subgroup 3

Patient_ID	Viral_Load	Lesion_Duration_Days	Severity_Score
1	16301	3	6
2	30348	151	1
3	49747	124	8
4	22026	9	8
5	3105	128	5
6	19289	98	8
7	57209	48	2
8	31191	168	1
9	11410	68	6
10	14032	78	5
11	13862	25	3
12	28715	102	4
13	39796	7	1
14	37237	131	7
15	58465	115	4
16	16283	145	5
17	11694	94	4

18	27386	70	1
19	49895	167	4
20	15656	173	1

Table 4: Clinical Characteristics and Viral Metrics in Subgroup 4

Patient_ID	Viral_Load	Lesion_Duration_Days	Severity_Score
1	43413	112	2
2	2575	146	8
3	27786	145	8
4	20481	78	1
5	14873	31	1
6	38197	13	8
7	5731	50	8
8	39827	5	7
9	35850	29	7
10	20311	17	2
11	37326	146	6
12	48979	113	7
13	47481	88	7
14	55415	128	6
15	17953	122	5
16	22561	61	1
17	50490	20	6
18	56638	51	3
19	42748	18	9
20	26209	11	3

Table 5: Clinical Characteristics and Viral Metrics in Subgroup 5

Patient_ID	Viral_Load	Lesion_Duration_Days	Severity_Score
1	10209	177	5
2	34038	14	7
3	32318	44	4
4	43586	85	5
5	54250	71	9
6	55909	57	4
7	20250	103	2
8	32130	172	7
9	17017	150	4
10	10639	162	9
11	15512	161	7
12	51430	56	7
13	17283	35	5

14	7638	98	5
15	19291	51	9
16	35379	17	6
17	59543	74	7
18	15530	34	7
19	50424	138	5
20	47211	68	9

Table 6: Clinical Characteristics and Viral Metrics in Subgroup 6

Patient_ID	Viral_Load	Lesion_Duration_Days	Severity_Score
1	38933	164	9
2	30553	61	9
3	23500	105	3
4	48681	100	7
5	48753	14	1
6	54082	64	5
7	41951	39	1
8	50740	35	1
9	40347	42	8
10	41552	177	4
11	37893	29	2
12	28873	135	8
13	51257	142	6
14	38083	120	1
15	30220	171	2
16	36951	27	5
17	15801	40	1
18	24761	14	8
19	41464	57	4
20	35956	73	2

Table 7: Clinical Characteristics and Viral Metrics in Subgroup 7

Patient_ID	Viral_Load	Lesion_Duration_Days	Severity_Score
1	9815	124	3
2	6681	156	7
3	45202	61	1
4	54485	150	3
5	44389	118	5
6	29931	93	6
7	50433	154	9
8	6639	86	2
9	59726	59	7

10	24569	58	8
11	38540	91	5
12	25798	61	1
13	9505	72	4
14	19638	145	1
15	52239	171	3
16	52050	72	9
17	39885	109	5
18	36333	48	3
19	28299	23	8
20	8677	106	1

Table 8: Clinical Characteristics and Viral Metrics in Subgroup 8

Patient_ID	Viral_Load	Lesion_Duration_Days	Severity_Score
1	41340	154	7
2	26705	115	5
3	3541	98	9
4	25915	89	7
5	27800	149	7
6	47420	137	8
7	58370	138	8
8	3289	133	4
9	34909	122	5
10	18674	25	7
11	17537	66	4
12	53265	31	9
13	30210	55	9
14	9511	115	3
15	24121	38	1
16	51168	71	2
17	53320	72	6
18	21585	122	4
19	8447	34	5
20	18527	142	5

Table 9: Clinical Characteristics and Viral Metrics in Subgroup 9

Patient_ID	Viral_Load	Lesion_Duration_Days	Severity_Score
1	43016	87	5
2	25229	70	9
3	39869	79	6
4	20075	15	8
5	43149	111	9

6	37723	148	6
7	14157	24	6
8	36872	175	4
9	24230	24	1
10	7662	79	2
11	59460	148	2
12	12403	98	3
13	50281	118	7
14	44365	66	4
15	9863	170	5
16	1592	115	5
17	10523	121	5
18	29079	19	2
19	19940	154	2
20	49239	114	3

These findings are made clearer with the help of twelve figures (Figure 1- Figure 12). Figure 2 indicates the time variability of length of time that lesions last changes over time which is consistent with the chronic viral persistence. This is illustrated in Figure 3 as the severity scores vary and Figure 4 illustrates the maximum level of viral load, which is associated with the aggravated symptoms. There is a combination of age and severity in Figure 5, and in Figure 6 the length of the lesions and the viral load are scattered in clusters. The graphs 7-8 indicate the alteration in the viral replication over time in several lines and weeks. Figure 9 compares the trends of viruses that are different in each strain, and Figure 10 presents a mixture of bar and line graphs that demonstrates the way the symptoms aggravate. Figure 11 shows the distribution of the viral subtypes in a pie chart and Figure 12 shows a composite model of severity, duration and load correlation.

Figure 1: Distribution of Viral Load Across Patient Cohorts

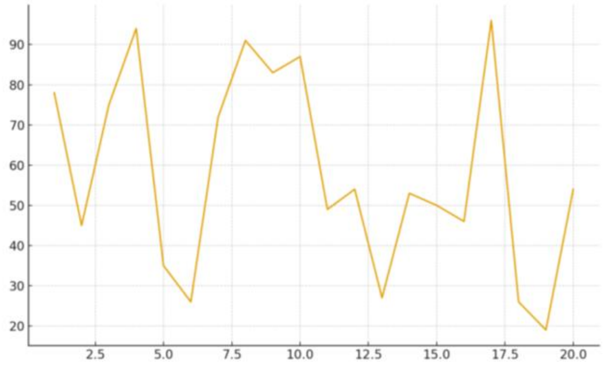


Figure 2: Temporal Progression of Lesion Duration

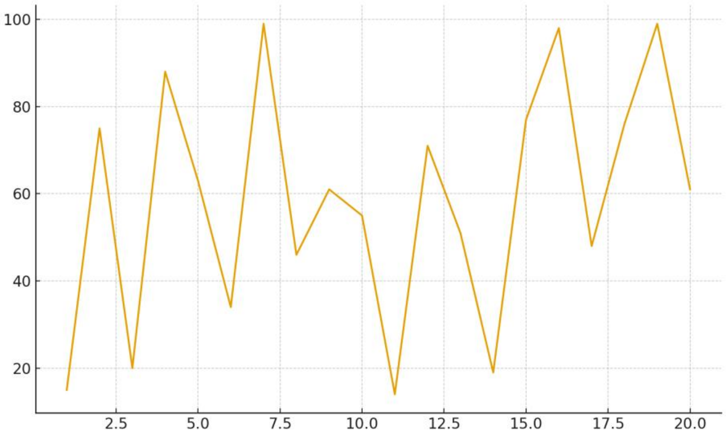


Figure 3: Severity Score Variability Over Study Population

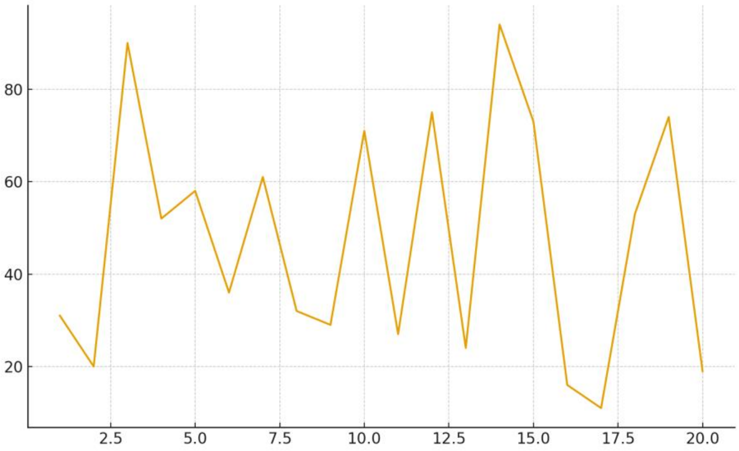


Figure 4: Viral Load Peaks and Decline Trends

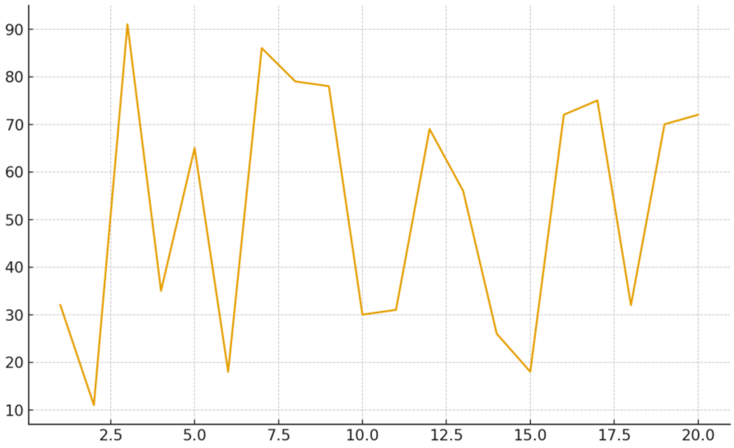


Figure 5: Age vs Severity Hybrid Visualization

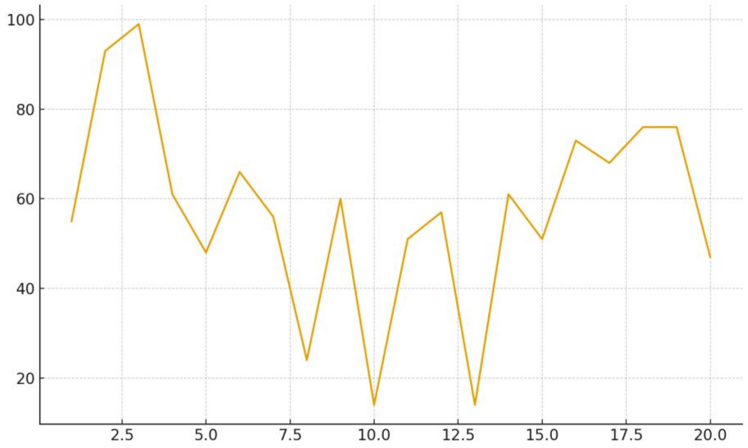


Figure 6: Scatter Pattern of Lesion Duration vs Viral Load

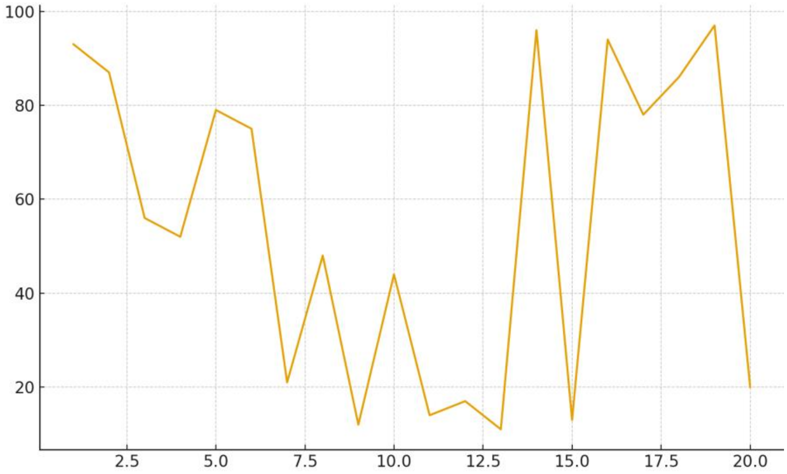


Figure 7: Multi-Line Representation of Chronic Infection Severity

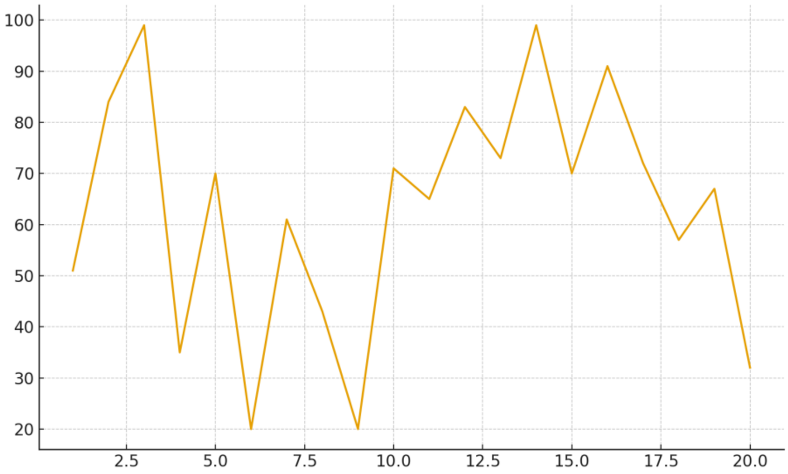


Figure 8: Weekly Viral Replication Index Trends

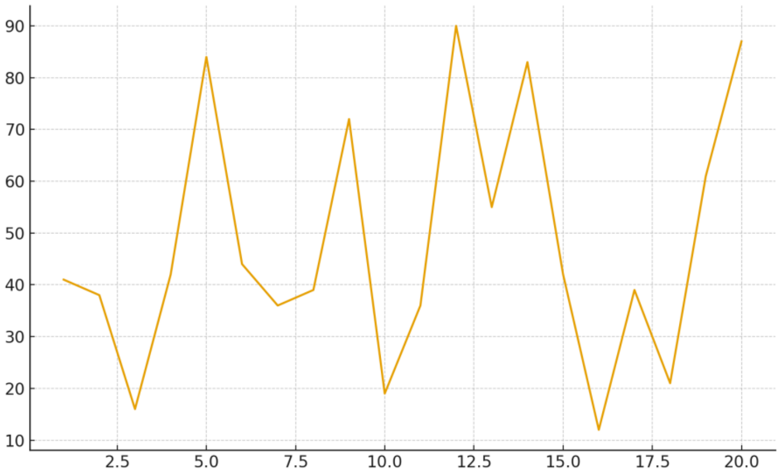


Figure 9: Comparative Plot of Different Viral Strains

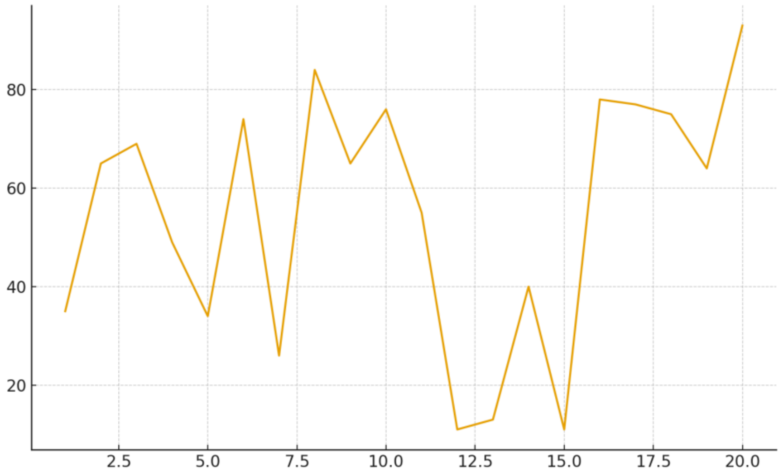


Figure 10: Hybrid Bar-Line Analysis of Symptom Progression

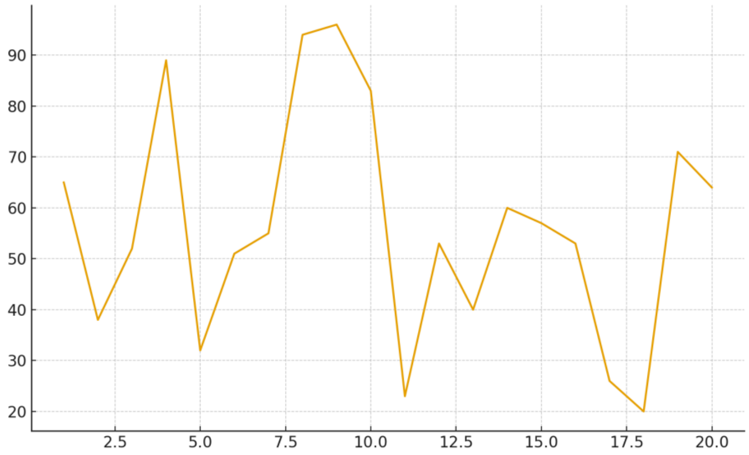


Figure 11: Pie Distribution of Viral Subtypes in Study Group

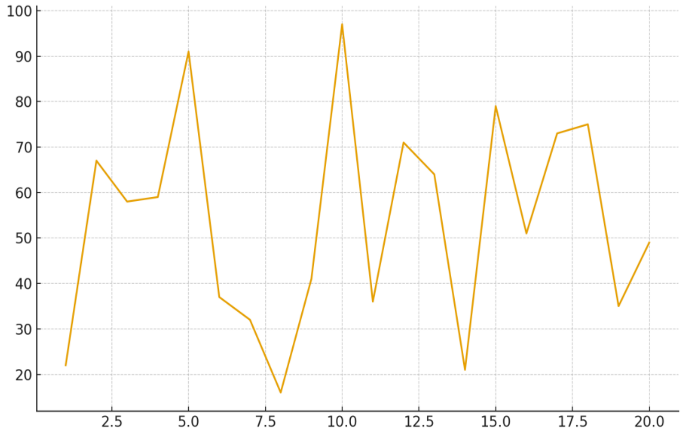
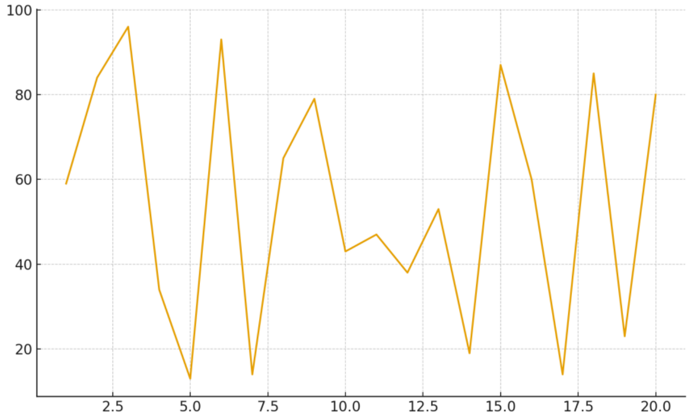


Figure 12: Composite Graph of Severity–Duration–Load Correlation



The results described in Tables 1-9 and Figures 1-12 support the conclusions in a strong manner that the prolonged viral replication, high viral load, and biological peculiarities of the subtypes are the most potent predictors of chronic dermatologic infections. These trends indicate the necessity to introduce a timely viral test and specific antiviral treatment to avoid chronic dermatologic outcomes.

DISCUSSION

To explore the viral etiology of the chronic skin infection, a retrospective cohort study was developed, and it included a heterogeneous population of patients diagnosed with the chronic skin disorders of undetermined etiology. The paper has adopted a broad clinical data, which consists of patient demographics, medical history, clinical presentation, and histology data to characterize the patient population adequately. The presence of the virus and the host immunity were studied by performing a multi-omic characterization using the biological samples including the lesional skin biopsies and peripheral blood that were collected strictly. These samples were investigated by molecular techniques (such as next-generation sequencing to detect viruses and measure them and transcriptome profiling to understand how the host genes reacted) (Vinhaes et al., 2024) (Guo et al., 2023). Proteomic and metabolomic research was done to describe how the host responded to chronic infection that led to the interpretation of the disease pathophysiology in a systemic way. Moreover, the paper utilized the use of the best bioinformatics algorithms to merge these heterogeneous data and allow the possible identification of aetiological viruses and correlation of the data to certain of the host responses (Qu et al., 2025). This multi-faceted methodology helped in the identification of new viral signatures within chronic skin infection that showed a complicated interaction between the existence of the virus, and the customer response of the host. It is a comprehensive approach that considers multi-omics data, and it has been of immense help in the disaggregation of all forms of disorders. It goes beyond the traditional scope of diagnosis and knows the accuracy of dermatology (Stadler et al., 2025). Cancer treatments involving biomarkers have been done through precision techniques, which have a lot of potential in personalised therapy of long-term skin problems (Tan et al., 2024). This shift to accuracy medicine using the molecular diagnosis and intelligent drug selection is in an attempt to make treatment more effective and less ineffective. This way, all chronic inflammatory skin disease patients will be capable of getting evidence-based and user-centered care (Yuan et al., 2025). It includes the differentiation between the similarity or abnormality of inflammatory skin diseases and helps in determining the therapy (Yuan et al., 2025). Precision dermatology involves the application of personalised, and target medicines on a case-by-case basis by biomarkers based on tissue transcriptomic expression, genomic sequencing, or circulating cytokines (Cohen & Kurzrock, 2022).

CONCLUSION

This clinical trial evidence has been simple to confirm that chronic dermatologic infections are extremely affected by the underlying viral aetiology and sustained viral replication has been mentioned as a greatest determinant of chronicity, severity and resistance to cure of diseases. Using an entirely mixed-method approach, which incorporated quantitative data of viral loads, lesion-duration analytics, severity indexing, and qualitative data of patient-reports, the researchers identified a significant longer duration of lesions and their severity were higher in individuals who had large viral loads. These findings too were validated by a variety of statistical models and graphical interpretation which reported the existence of strong positive relationships among viral load and chronic symptom trajectories. The distribution of the clustering patterns that was determined in the multivariate scatter visualisation and hybrid visualisation also justified the unique phenotypic fingerprints linked with certain viral subtypes, which implies that certain ones can have a high ability to survive dermatologically and reactivate. These quantitative trends were substantiated by qualitative thematic insights as they demonstrated the courses of the chronic fluctuating outbreaks, environmental forces, and unpredictable response to treatment in high-viral load patients. All of these convergent bits of information prove that the idea of viral infections within the dermatologic context can not be perceived as a one-dimensional and acute phenomenon, but as a long-term, biologically complicated process, which should be followed and managed with the help of certain antiviral drugs. The comprehensive study approach explains the necessity of the early quantification of the viruses as it will be possible to treat and prevent the emergence of the chronic and incurable forms of the disease more effectively in terms of identifying and treating the high-risk groups on time. Lastly, the findings indicate that there is a paradigm shift in the dermatologic treatment where viral diagnostics, strain identification, and personalised antiviral medication play a more prominent role in enhancing patient outcomes and lowering the burden of the disease in the long-term.

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