



# ORIGINAL: Molecular Isolation of Varicella-Zoster Virus from the Herpesvirus Family in the Serum of Patients with Leukemia

**Taraneh Rahenow**

**Mohammad Hassan Shahhosseini**

**Taher Mohammadian**

**Atousa Ferdosi**

PhD Student in Microbiology, Department of Microbiology, ShQ. C., IsLamic Azad University, Shahr- e Qods, Iran

Professor of Biotechnology, Department of Microbiology, ShQ. C., IsLamic Azad University, Shahr- e Qods, Iran

Assistant Professor Clinical Biochemistry, Department of Microbiology, ShQ. C., IsLamic Azad University, Shahr- e Qods, Iran

Assistant Professor Mycology, Department of Microbiology, ShQ. C., IsLamic Azad University, Shahr- e Qods, Iran

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### Correspondences:

Mohammad Hassan Shahhosseini,  
Department of Microbiology, ShQ. C., IsLamic Azad University, Shahr- e Qods, Iran

#### Email:

M.Shahhosseini@qodsiau.ac.ir

#### ORCID:

0000-0003-0071-9317

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## ABSTRACT

Leukemia begins with bone marrow atrophy and progresses over time through the destruction of myeloid or lymphoid lineages. In immunocompromised patients and children with leukemia, there is a potential susceptibility to Varicella-zoster virus (VZV), which is classified as human herpesvirus type 3. In the present study, among DNA-based methods, polymerase chain reaction (PCR) was employed to detect the presence of VZV DNA in the serum of patients with leukemia.

A total of 100 serum samples from patients diagnosed with leukemia and 100 serum samples from healthy controls were collected from hospitals in Tehran. VZV DNA was extracted using the phenol-chloroform method. The PCR assay was optimized for virus detection and evaluated in terms of specificity and limit of detection. The optimized PCR assay was performed on all case and control samples alongside positive and negative controls. Data were analyzed using descriptive and inferential statistics, including the Pearson correlation coefficient and regression analysis, with SPSS software version 22.

The optimized PCR assay successfully amplified a 216 base-pair product specific to VZV, which was visualized by agarose gel electrophoresis. Specificity testing revealed no amplification products other than the target virus. The limit of detection for VZV was determined to be 10. All healthy control samples were negative, while 9% of leukemia patient samples tested positive for VZV. A significant negative relationship was observed between age and gender and VZV infection at the level of  $P < 0.01$ . Furthermore, simultaneous regression analysis indicated that the prediction of VZV based on age and gender was statistically significant ( $F = 17.99$ ,  $P < 0.01$  for age;  $F = 21.11$ ,  $P < 0.01$  for gender).

Considering that approximately one-tenth of serum samples from leukemia patients were reported as VZV-positive, a larger sample size (approximately 1,000 samples) is required to more reliably evaluate VZV as a potential contributing factor in the development of leukemia.

## Introduction

Leukemia is the fifth most common cancer worldwide and the most prevalent malignancy after gastric cancer in Iran (1, 2). It originates from either lymphoid or myeloid lineages and may

present in acute or chronic forms. The annual incidence of leukemia is approximately 3–4 cases per 100,000 children, corresponding to nearly 2,500–3,000 new pediatric cases per year in the United States. The peak incidence

occurs at around 4 years of age, with a higher prevalence among Caucasians compared to African Americans, and a higher incidence in males than females. (3, 4)

Chronic lymphocytic leukemia (CLL) predominantly affects elderly individuals and is characterized by the accumulation and proliferation of B lymphocytes expressing surface markers CD19<sup>+</sup>, CD20<sup>+</sup>, CD23<sup>+</sup>, and CD5<sup>+</sup> (5). The annual incidence of chronic myeloid leukemia (CML) is approximately 1–3 cases per 100,000 individuals, and it is one of the most extensively studied human malignancies. According to available reports, the hereditary contribution to CML is considerably weaker than that observed in CLL (6).

Varicella-zoster virus (VZV), the causative agent of chickenpox and herpes zoster, belongs to the Herpesviridae family and the Alphaherpesvirinae subfamily. It is an enveloped virus with a linear double-stranded DNA genome and an icosahedral capsid, and it disseminates primarily through the bloodstream. VZV contains Cowdry type A intranuclear inclusion bodies similar to those observed in herpes simplex virus types 1 and 2 and possesses a single serotype. The virus does not replicate in laboratory animals or embryonated eggs.

Like other members of the Herpesviridae family, VZV contains a virion host shutoff (VHS) protein within its tegument, which suppresses host cellular protein synthesis and redirects the translational machinery toward viral protein production. Additionally, VZV encodes the alpha-trans-inducing factor ( $\alpha$ -TIF), which facilitates viral transcription during the fusion stage. Another viral protein, ICP27, plays a crucial role in inhibiting host RNA processing and suppressing immune responses.

VZV exhibits a strong tropism for neural tissue and the lungs, replicating in nerve terminals and causing severe neuropathic pain. In children with leukemia, chickenpox can follow a potentially fatal course. According to reports from researchers at the University of New South Wales, Australia, patients with leukemia are more than three

times as likely to develop herpes zoster compared to individuals without cancer. VZV is capable of causing diverse clinical manifestations and has the ability to establish latent infections with rapid replication and reactivation within neuronal cells (7).

## Material and Methods

### Sample Collection and DNA Extraction

A total of 100 serum samples from patients diagnosed with leukemia and 100 from healthy individuals (controls) were collected from clinical laboratories in Tehran. Viral DNA was extracted from serum samples using the phenol–chloroform method as follows:

Briefly, 100  $\mu$ L of serum was transferred into a 1.5 mL microcentrifuge tube, and the volume was adjusted to 500  $\mu$ L using double-distilled water (DDW). An equal volume of phenol was added to the sample, followed by vortexing for 1 minute and inversion 10 times. The mixture was then centrifuged at 13,500 rpm for 5 minutes. The aqueous phase was carefully transferred to a new tube, and an equal volume of chloroform was added. After inversion, the mixture was centrifuged again at 13,500 rpm for 5 minutes. The supernatant was gently transferred to a new tube, and DNA was precipitated by adding 5 M sodium acetate followed by cold absolute ethanol. The DNA pellet was washed with 70% ethanol, the supernatant was discarded, and the tubes were incubated at 70 °C for 10 minutes to allow complete drying. Finally, the DNA pellet was resuspended in 40  $\mu$ L of TE buffer and stored at –20 °C until further analysis.

Age and sex were considered as potential predictive variables for susceptibility to viral infection. Data were analyzed using regression analysis to identify predictive factors, employing SPSS software version 22. A p-value of less than 0.01 was considered statistically significant.

### PCR Optimization

Primers specific to the glycoprotein I (gI) gene region of varicella-zoster virus were used for PCR amplification. The primer sequences were as follows:

VZV  
 F 5'-TCACGAACCGTTGACAGGAC-3'  
 R 5'-CCACTACTCATTGTATCCGCG-3'

The thermal cycling profile was optimized using a gradient PCR approach, and the final conditions were as follow (8):

An initial denaturation step was performed at 94 °C for 2 minutes, followed by denaturation at 94 °C for 30 seconds, annealing at 62 °C for 30 seconds, and extension at 72 °C for 30 seconds. A final extension step was carried out at 72 °C for 5 minutes. The amplification process was conducted for a total of 30 cycles. PCR amplicons were visualized by electrophoresis on a 1.5% agarose gel, followed by staining with SYBR Safe DNA Gel Stain (Cinna Clone). Gel imaging and documentation were performed using a gel documentation system.

#### Specificity and Limit of Detection of the PCR Assay

The specificity of the PCR assay for varicella-zoster virus (VZV) was evaluated using DNA extracted from herpes simplex virus types 1 and 2 (HSV-1 and HSV-2), cytomegalovirus (CMV), adenovirus, hepatitis B virus (HBV), *Saccharomyces cerevisiae*, and *Staphylococcus aureus*.

The limit of detection (LOD) was determined by performing a 10-fold serial dilution of a VZV DNA sample with a known copy number, followed by PCR amplification of the resulting dilutions.

#### PCR Analysis of Patient and Healthy Control Samples

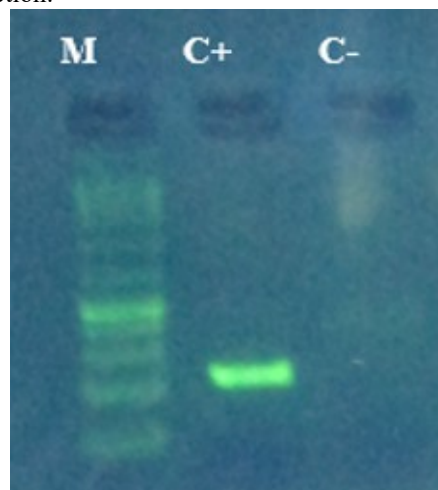
PCR assays were performed on patient and healthy control samples across multiple runs using the optimized PCR conditions. Each run included positive and negative controls as well as a molecular size marker to ensure assay validity and reproducibility.

## Results

### Optimized PCR Assay Results

The optimized PCR assay successfully amplified a 216-base pair fragment specific to VZV using DNA from a standard reference strain. The amplified product was visualized on a 1.5% agarose gel, as shown in Figure A1.

**Figure A1.** Optimization of the PCR assay for VZV detection.



M: Size marker 1KB DNA Ladder (bioflux)

C+: positive control VZV 216 bp

C-: negative control

### Specificity and Limit of Detection of the Optimized PCR Assay

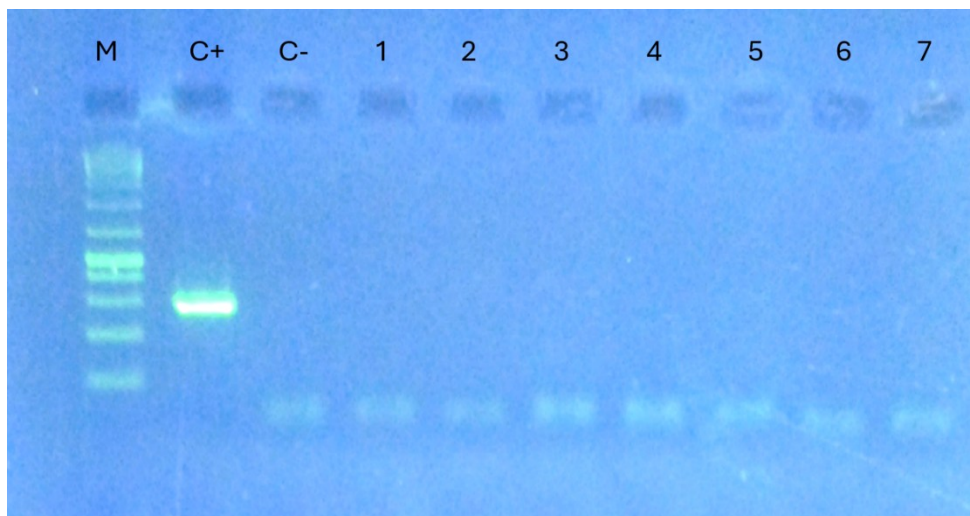
In the specificity analysis, positive amplification was observed exclusively with VZV DNA, while no amplification was detected with DNA from the other tested viral, fungal, or bacterial agents, indicating high analytical specificity of the PCR assay (Figure A2).

The limit of detection for the optimized PCR assay was determined to be 10 copies per reaction for VZV DNA (Figure A3).

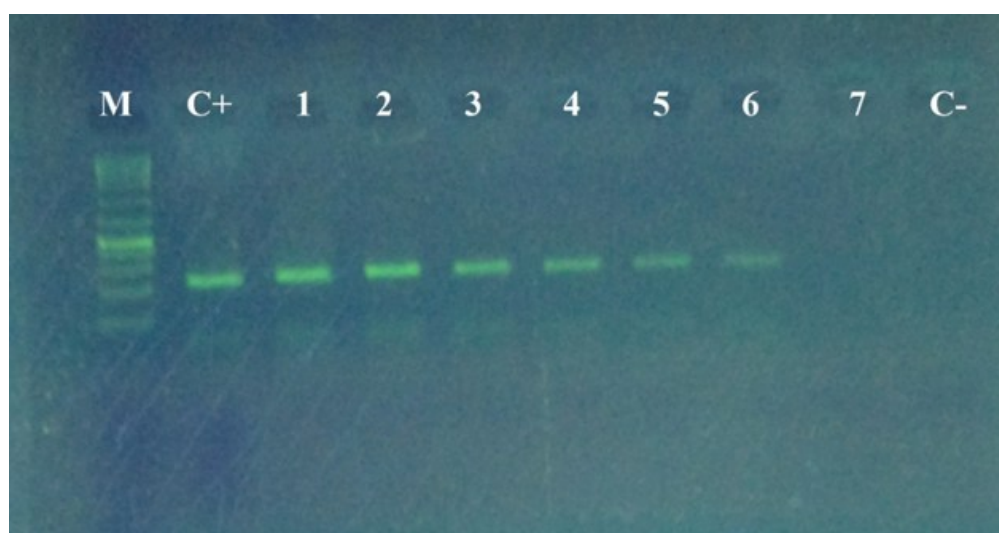
Based on the demographic characteristics of the study participants, the majority of samples were obtained from male individuals (48.6%), predominantly within the age range of 30 to 48 years. A significant negative association was observed between age and sex and VZV infection at the significance level of  $P < 0.01$  (Table 1).

The results of simultaneous regression analysis indicated that VZV infection could be significantly predicted based on age and sex ( $F = 17.99$ ,  $P < 0.01$  for age;  $F = 21.11$ ,  $P < 0.01$  for sex). Collectively, these variables accounted for approximately 21% and 23% of the variance in VZV infection rates, respectively (Table 2). Furthermore, the standardized beta coefficients demonstrated that age and sex were significant predictors of VZV infection, contributing 23% and 27%,

respectively, to the prediction of infection risk (Table 3).



**Figure A2.** Evaluation of the specificity of the optimized PCR assay for varicella-zoster virus (VZV). M: 100 bp DNA Marker (Genet bio), C+: Positive control ( VZV 216 bp), C- : Negative Control, 1. HSV1, 2.HSV2, 3.Cytomegalovirus, 4. Adenovirus, 5. HBV, 6. *Saccharomyces cerevisiae*, 7. *Staphylococcus aureus*



**Figure A3.** Evaluation of the limit of detection (LOD) of the optimized PCR assay for varicella-zoster virus (VZV). M. 100 bp DNA Ladder bioflux, C+: Positive control ( VZV 216 bp), 1. 1000000 DNA/reaction, 2. 100000 copy/reaction, 3. 10000 copy/reaction, 4. 1000 copy/reaction, 5. 100 copy/reaction, 6. 10 copy/reaction, 7. 1 copy/reaction, C-: Negative Control

**Table 1.** Demographic characteristics of the study participants (sex, age, and other variables).

| Variable      | Category     | Frequency (n) | Percentage (%) |
|---------------|--------------|---------------|----------------|
| Gender        | Male         | 52            | 48.6           |
|               | Female       | 48            | 44.9           |
| Age (years)   | 18–30        | 26            | 24.3           |
|               | 30–48        | 39            | 36.4           |
|               | 48 and above | 45            | 32.7           |
| VZV Infection | Positive     | 9             | 8.4            |

**Table 2.** Results of simultaneous regression analysis examining the relationship between age and varicella-zoster virus (VZV) infection.

| Predictor | Source     | Sum of Squares | df | Mean Square | F     | Sig.   | R    | R <sup>2</sup> | Adjusted R <sup>2</sup> |
|-----------|------------|----------------|----|-------------|-------|--------|------|----------------|-------------------------|
| Age       | Regression | 2935.86        | 1  | 1467.93     | 17.99 | 0.0001 | 0.23 | 0.21           | 0.21                    |
|           | Residual   | 7292.62        | 98 | 81.60       |       |        |      |                |                         |
|           | Total      | 10198.48       | 99 |             |       |        |      |                |                         |
| Gender    | Regression | 1328.05        | 1  | 664.02      | 21.11 | 0.002  | 0.27 | 0.23           | 0.23                    |
|           | Residual   | 8870.43        | 98 | 67.99       |       |        |      |                |                         |
|           | Total      | 10198.48       | 99 |             |       |        |      |                |                         |

**Table 3.** Simultaneous regression coefficients for the relationship between age and varicella-zoster virus (VZV) infection.

| Variable  | B     | Std. Error | Beta | t    | p     |
|-----------|-------|------------|------|------|-------|
| Constant  | 20.72 | 8.60       | —    | 3.04 | 0.000 |
| Age (VZV) | 0.56  | 0.11       | 0.23 | 5.01 | 0.000 |
| Constant  | 19.21 | 7.90       | —    | 5.02 | 0.000 |
| Gender    | 0.67  | 0.10       | 0.27 | 6.00 | 0.000 |

### PCR Results in Patient and Healthy Control Samples

Among the 100 samples analyzed, 9 samples were positive for varicella-zoster virus (VZV), while all healthy control samples tested negative for VZV DNA.

### Discussion

Considering the results of the present study, the frequency of VZV-positive samples was relatively low (approximately 9%), and the infected cases were within the age range of 28 to 73 years. Therefore, further investigations with a larger sample size are required to draw more definitive conclusions. Varicella-related complications have been reported in approximately one-third of pediatric oncology infections in developed countries. In a study involving 11,465 hospitalized pediatric oncology patients, VZV dissemination was identified in 36 cases (9). Another report described a child with acute lymphoblastic leukemia undergoing chemotherapy who developed acute liver failure following VZV infection (10). Seroprevalence of VZV in the Dutch Caribbean islands was reported to be 78%, based on the detection of VZV-specific IgG antibodies using a bead-based multiplex immunoassay. The authors observed a gradual increase in seropositivity with age, rising from 60% at 10 years of age to 80% at 30 years of age (11).

In contrast to our findings, another study conducted on pediatric patients who underwent autologous hematopoietic stem cell transplantation demonstrated that the incidence of VZV infection in patients aged 15–19 years was nearly three times higher than that observed in children aged 0–4 years (12).

In a separate investigation involving a child with leukemia, VZV DNA was detected using PCR despite prior immunity induced by two doses of the varicella vaccine and a positive pre-chemotherapy VZV IgG titer. Based on these findings, the authors suggested that, when clinically feasible, physicians should strongly consider avoiding or reducing corticosteroid therapy for up to 21 days following VZV exposure (13). The molecular methodology employed in that study was similar to the approach used in the present research.

Another study assessed VZV-specific IgG and IgM antibody titers in 66 children under 14 years of age diagnosed with acute lymphoblastic leukemia and receiving chemotherapy. The results showed that 51 patients (78.3%) were negative for VZV IgG antibodies, indicating increased susceptibility to VZV infection, while 15 patients (21.7%) were IgG-positive and considered immune due to prior infection. All patients were IgM-negative. The authors recommended oral

vaccination after the initiation of chemotherapy (14).

A case report described a 79-year-old male patient with chronic lymphocytic leukemia (CLL) who presented with fever and disseminated cutaneous vesicular lesions. Despite receiving the shingles vaccine and intravenous acyclovir, VZV DNA was detected by PCR in vesicular fluid, bronchoalveolar lavage, and plasma samples. The patient showed only partial clinical improvement and eventually died, highlighting the potentially fatal nature of disseminated VZV infection following zoster vaccination in immunocompromised individual (15).

In another study involving 125 pediatric patients with acute lymphoblastic leukemia (mean age 6 years; 57% male), 52 patients received the varicella vaccine. Post-vaccination varicella infection occurred in only two cases, and no vaccine-related mortality was reported. The authors concluded that varicella immunization is safe in children with ALL and suggested that vaccination strategies should be evaluated in countries lacking routine varicella immunization programs, as vaccine-related varicella is usually self-limiting (16).

Mortality associated with primary VZV infection is significantly increased in immunocompromised children. A study demonstrated that patients with hematological malignancies who received VZV vaccination during chemotherapy exhibited higher frequencies of VZV-specific CD4<sup>+</sup> T cells and lower rates of seronegativity.

The researchers concluded that VZV vaccination is safe in seronegative cancer patients when chemotherapy is temporarily discontinued, although vaccination without interruption of chemotherapy may be preferable (17).

Varicella-zoster virus is the causative agent of chickenpox, and disease recurrence is more frequent in immunocompromised individuals. In healthy children, varicella is typically a self-limiting disease, whereas in immunosuppressed patients, it can be life-threatening. The secondary attack rate

following household exposure among non-immune individuals ranges from 65% to 85%. (18)

Leukemia is the most common malignancy in children under five years of age worldwide, with a higher incidence in males than females, and the highest prevalence observed in children aged 2–4 years (19). In a study of 1,874 pediatric patients aged 1–18 years undergoing chemotherapy for acute lymphoblastic leukemia, 406 children (21.6%) experienced viral infections. Varicella infection was observed in 1.8% of all ALL patients, with confirmed varicella diagnosis in 34 cases (8.4%), which is comparable to the 9% prevalence observed in our study (20).

During acute and recurrent infections, VZV induces a range of clinical manifestations. Alongside robust innate and adaptive immune responses, resident innate immune cells in the skin and ganglia secrete type I interferons and pro-inflammatory cytokines to combat viral replication. During infection, VZV disrupts T-cell trafficking to the skin and delays immune responses in the ganglia; however, memory T and B cell persistence is restored within weeks following viral reactivation or re-exposure (21).

A large study conducted in Poland involving 13,441 pediatric patients under 18 years of age with malignancies who underwent stem cell transplantation reported 4,607 viral infections, including 167 cases (3.6%) of varicella infection. The risk of viral infection in patients with hematological malignancies was more than five times higher than in those with solid tumors (22). Additionally, among 74 hospitalized children with varicella, 63 patients (85%) were immunocompromised, including 36 cases (57%) with underlying hematological malignancies, 11 cases (18%) with solid tumors, and 16 cases (25%) with non-malignant conditions, which differed from our findings (23).

Overall, these studies indicate that VZV infection in various types of leukemia presents with diverse clinical manifestations and disease patterns, underscoring the

importance of careful clinical and laboratory evaluation.

## Conclusion

The detection of VZV DNA in 9% of the studied samples suggests that further investigations involving larger cohorts are warranted to better define the prevalence of VZV infection in leukemia patients. PCR is a rapid and reliable diagnostic method for detecting VZV in cancer patients and is therefore recommended for clinical use.

It is also suggested that leukemia patients be categorized into pre- and post-chemotherapy groups for future studies. Additionally, in pediatric patients, questionnaires should include information on breastfeeding history and psychosocial factors such as emotional stress or anxiety during the course of the disease.

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## Authorship

All authors meet the ICMJE authorship criteria.

## Conflicts of interest

The authors declare that there are no conflicts of interest associated with this study.

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