

VARICELLA ZOSTER VIRUS ESOPHAGITIS IN AN IMMUNOCOMPETENT PATIENT

Tayure Agayeva

*Infectious disease physician, Shabran District Central Hospital
e-mail: T.agayeva21@gmail.com*

Received 20.01.2025

Accepted 16.05.2025

Abstract: *Varicella Zoster Virus (VZV) usually presents with severe clinical manifestations in immunocompromised individuals, but it has been rarely described to cause esophagitis in immunocompetent individuals. We present a case of VZV-associated esophagitis in a 37-year-old male patient with immunocompetent status. The patient presented with dysphagia and retrosternal pain. Endoscopy revealed multiple mucosal erosions in the esophagus, and biopsy and PCR results were consistent with VZV. Antiviral treatment (acyclovir) was initiated, and the patient recovered completely. This case highlights the possibility of VZV esophagitis in immunocompetent individuals and highlights the importance of early diagnosis.*

Keywords: *Varicella-zoster virus, Esophagitis, Immunocompetent patient, Clinical case, Endoscopy, Antiviral treatment*

Introduction

Varicella-zoster virus (VZV) esophagitis is extremely rare in immunocompetent individuals, with only a few cases reported in the literature. We present the case of a 37-year-old previously healthy male who was admitted with fever, nausea, epigastric pain, dysphagia, and retrosternal discomfort. Endoscopy revealed multiple mucosal erosions and ulcerative lesions in the esophagus. Histopathological examination of biopsy samples showed intranuclear inclusion bodies, and immunohistochemistry was positive for VZV but negative for HSV-1 and HSV-2. Serum VZV IgM levels were elevated, and PCR confirmed VZV infection. On the third day of hospitalization, vesiculopapular eruptions appeared on the trunk, further supporting the diagnosis. The patient was treated with intravenous acyclovir and achieved full clinical recovery.

This case emphasizes that VZV esophagitis, although rare, should be considered in the differential diagnosis of infectious esophagitis even in immunocompetent patients. Early endoscopic evaluation and laboratory confirmation are crucial for appropriate management and favorable outcomes.

Experimental part

A 37-year-old male patient has himself ill for approximately 5–6 days. The illness began with fever, chills, nausea, and acute pain in the epigastric region. Gastroscopy revealed an infectious lesion in the esophagus (suspected HSV), and the patient was admitted to our clinic for inpatient treatment. In his anamnesis, the fact of contact with an infectious patient is denied. He does not remember any infectious diseases or vaccination history in childhood.

Objective examination: Body temperature – 37.2 °C. Skin integuments are of normal color. The tongue is moist, covered with crust. Vesiculopustular rash accompanied by mucositis is observed on the oral mucosa. Heart sounds are muffled, pulse rate is 98 beats/min, arterial pressure is 140/90 mm Hg. Hard breathing is heard over the lungs, SpO₂ – 98%. The abdomen was soft on palpation, painful in the epigastric region. Urination and defecation were normal.

On the 3rd day of hospitalization, a papulo-vesicular rash appeared on the patient's body (Fig. 1). The rash was grouped, typical of the Varicella Zoster virus. The appearance of these skin lesions confirmed the connection of the infection causing esophagitis with the Varicella Zoster virus.



Fig. 1. Papulovesicular rash observed in the abdominal region during Varicella zoster virus infection.

Laboratory tests: Leukopenia ($3.42 \times 10^9/L$), lymphocytosis were observed in the complete blood count. CRP 28 mg/L, ESR – 30 mm/h were recorded. Liver and kidney function tests were within the normal range. HIV, syphilis test were negative

Esophagogastroduodenoscopy examination (Fig. 2.) showed local 5-6mm ulcerated bumps on the esophageal mucosa, and a diagnosis of infectious damage to the esophagus (HSV?), erythematous gastropathy, and cardiac failure was made.

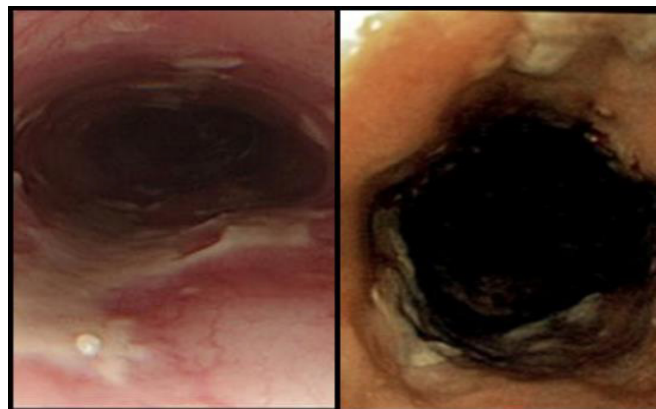


Fig. 2. Esophagogastroduodenoscopy examination

The opening in the esophagus was open and freely passable. On the mucous membrane, there were ulcerated bumps, the largest of which was 5-6 mm in size (**herpes simplex?**). The Z line was passed at 42 cm from the anterior incisors. The gastric fundus was normal. The mucous membrane in the corpus and antrum was diffusely hyperemic and edematous. The bulb and postbulbar duodenum were normal.

Biopsies were taken from the center and edges of the bumps for pathological examination. Immunohistochemistry was positive for Varicella Zoster virus and negative for Herpes simplex virus 1 and Herpes simplex virus 2. Serum varicella IgM levels were high, and PCR analysis detected Varicella Zoster virus.

The patient was prescribed intravenous acyclovir (10 mg/kg, 3 times a day), a proton pump inhibitor, and symptomatic therapy (analgesics, fluid support). From the 5th day of treatment, the patient's complaints decreased, fever and epigastric pain subsided. After a 10-day course of antiviral treatment, the patient was discharged home for observation by a field physician with a repeat gastroscopy scheduled 1 month later.

Results and Discussion

Varicella Zoster virus (VZV) causes chickenpox during the initial infection, then remains latent in nerve nodes and can be reactivated by various factors, manifesting as herpes zoster. VZV is usually observed in people with weakened immune systems with a severe clinical course and rare localizations. Esophagitis is considered one of the rarest complications of this virus.

Damage to the gastrointestinal tract by Varicella-zoster virus (VZV) is rare and has been observed in only a few cases, particularly in immunocompetent patients. According to the literature, VZV esophagitis has been described more often in patients with immunodeficiency (hematological diseases, organ transplantation, immunosuppressive therapy). However, in recent years, isolated cases have been reported in individuals with normal immune systems [1–6].

In our 37-year-old immunocompetent patient, the disease began with esophagitis and was later accompanied by the development of a typical papulo-vesicular rash. Esophagogastroduodenoscopy showed ulcerated bumps on the esophageal mucosa, and histological examination revealed classic intranuclear inclusion bodies, and immunohistochemical analysis showed positive results for VZV and negative results for HSV-1 and HSV-2. These results confirmed the etiological role of VZV.

In contrast to cases reported in the literature, our patient developed skin rashes several days after symptoms of esophagitis. As described by Akiyama et al. (2024), esophagitis may precede skin lesions in some patients [7]. This makes early clinical diagnosis difficult and necessitates differentiation of VZV from other herpes viruses.

On the other hand, as reported by Yu et al. (2025), the presence of skin lesions in addition to gastrointestinal symptoms facilitates the diagnosis [6]. In addition, Timotijevic et al. (2025) emphasized that not only the esophagus but also the stomach and intestines can be affected during widespread VZV infection [8]. These cases indicate that VZV is not limited to the skin and nervous system, but also has the potential to affect internal organs.

According to treatment protocols, intravenous acyclovir remains the drug of choice even in immunocompetent patients, with a treatment duration of usually 7–10 days. In the clinical case presented by Shallal A et al. (2019), the treatment duration was 15 days of IV acyclovir, followed by oral valacyclovir for 24 days [1]. In our patient, 10 days of IV acyclovir treatment was effective.

Conclusion

Varicella Zoster virus (VZV) causes chickenpox in the primary infection, then remains latent in the nerve ganglia and can be reactivated under the influence of various factors and manifests as herpes zoster.

Varicella-zoster virus (VZV) damage to the gastrointestinal tract is rare and has been observed in very few cases, especially in immunocompetent patients. According to literature data, VZV esophagitis has been described more often in patients with immunodeficiency. However, in recent years, isolated cases have been reported in individuals with normal immune systems [1–6].

We present a 37-year-old immunocompetent patient with a clinical presentation of esophagitis, which was later accompanied by the development of a typical papulo-vesicular rash. Early antiviral therapy helps both to reduce clinical symptoms and to prevent complications.

References

1. Shallal A., Valdez H., McKinney W.P. Varicella Zoster Virus Esophagitis in an Immunocompetent Patient. *Cureus*, 2019, V. 11(3), e4283. DOI:10.7759/cureus.4283
2. Pierte de Paula J.M., Franco Hidalgo S., Eiros Bouza J.M., de la Calle Valverde F. Esofagitis por el virus varicela zóster en un paciente inmunocompetente. *Medicina de Familia – SEMERGEN*, 2012, V. 38(3), p. 191–193. DOI: [10.1016/j.semerg.2011.09.010](https://doi.org/10.1016/j.semerg.2011.09.010)

3. Shallal A., Patel P., Reaume M., Tariq T., Gard D. Varicella-Zoster Virus Esophagitis in an Immunocompetent Patient. *ACG Case Rep. J.*, 2019, V. 6(4), e00044. DOI: 10.14309/crj.0000000000000044.
4. Lee S.K., et al. Varicella zoster virus esophagitis in an immunocompetent adult. *Endoscopy*. 1998, V. 30(Suppl 1), S59.
5. Niller H.H., Wolf H., Minarovits J. Regulation and dysregulation of Epstein-Barr virus latency: implications for the development of autoimmune diseases. *Autoimmunity*, 2008, V. 41(4), p. 298–328.
6. Yu Z., Ding Y., Sun F. Upper Gastrointestinal and Cutaneous Varicella in an Adult Male. *Clin Case Rep.* 2025, V. 13(3), e70296. DOI:10.1002/ccr3.70296.
7. Akiyama S., Hara S., Inokuma T. Varicella-Zoster Virus Esophagitis Prior to the Onset of Skin Lesions. *Intern Med.* 2024, V. 63(20), p. 2865–2866. DOI:10.2169/internalmedicine.3235-23.
8. Timotijevic A., Kodela P., Glusac V., Bokonjic S., Joksimovic B., Gomes J.V., Ladin D., Domic I. Disseminated Varicella-Zoster Virus Infection with Internal Organ Involvement. *Viruses*. 2025, V. 17(8), 1135. <https://doi.org/10.3390/v17081135>