

CASE REPORT

Herpetic Pharyngotonsillitis in an Immunocompetent Adult Male

Chalisara Subongkot¹, Panuwat Wongwattana², Nattarat Trinusonth³, Alena Santeerapharp⁴, Pannipa Wiriyaamornchai⁵

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ABSTRACT

Background: Herpetic pharyngotonsillitis is a rare clinical entity, especially in immunocompetent adults. While herpes simplex virus (HSV) type I is commonly associated with oropharyngeal infections, HSV type II is infrequently reported as a causative agent.

Case presentation: A 30-year-old immunocompetent male presenting with acute sore throat and whitish membranous pharyngotonsillitis without initial vesicular lesions. Initial antibiotic treatment failed to improve symptoms. Further investigation revealed HSV type II via polymerase chain reaction (PCR) analysis. The patient was successfully treated with oral acyclovir, with complete resolution of symptoms.

Discussion: Herpes simplex virus pharyngotonsillitis mimics bacterial tonsillitis and may result in misdiagnosis and inappropriate antibiotic use. Molecular diagnostics such as PCR are essential for accurate diagnosis in atypical cases. Although HSV type II is commonly associated with genital infections, it can also present with pharyngeal manifestations due to orogenital transmission.

Conclusion: Clinicians should consider HSV, including HSV type II, in the differential diagnosis of pharyngotonsillitis, particularly in cases unresponsive to antibiotics. Early antiviral treatment can accelerate recovery and reduce transmission.

Keywords: Antiviral therapy, Case report, Herpetic pharyngotonsillitis, Herpes simplex virus type II, Sore throat.

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INTRODUCTION

Generally, patients presenting with acute sore throat are often caused by viral infections, accounting for up to 42%. Among the viruses. The most frequently identified virus is adenovirus. Other viruses found less frequently include Epstein-Barr virus (EBV), parainfluenza, influenza A, herpes simplex virus (HSV), and respiratory syncytial virus.¹ The other most common group of bacterial pathogens includes group A beta-hemolytic *streptococcus* (GABHS), *Streptococcus dysgalactiae* subsp. *equisimilis* (hemolytic group C and G streptococci), *Fusobacterium necrophorum*, *Borrelia vincentii*, *Arcanobacterium haemolyticum*, *Neisseria gonorrhoeae*, *Mycoplasma pneumoniae*, and *Chlamydophila pneumoniae*.^{2,3}

In patients with viral tonsillitis, viral infections can be found from EBV (72%), human herpes virus 7 and 6B (54 and 16%, respectively), enterovirus (18%), parvovirus (7%), and other viruses (<4%).⁴ Herpetic tonsillitis is therefore considered a rare disease.

In adults, primary herpes infection usually causes acute pharyngotonsillitis, while reactivation typically leads to herpes labialis. Most cases are clinical diagnoses, and only in some cases need to be confirmed with additional tests.⁵

CASE PRESENTATION

A 30-year-old man presented with a 2-day history of acute sore throat and mild odynophagia, without cough and fever. The patient was diagnosed with acute pharyngitis and was subsequently discharged with a prescription for oral amoxicillin 500 mg, two capsules twice a day. Three days later, he revisited the otolaryngology department with persistent symptoms. On examination, the patient had whitish membranous pharyngotonsillitis with tonsillar gland enlargement 2+, uvular and soft palatal mild edema. However, no evidence of vesicular lesions or mucosal ulcerations was observed upon clinical examination. Bilateral cervical nodes were palpable in subcentimeters.

¹⁻⁵Department of Otolaryngology, Faculty of Medicine, Srinakharinwirot University, Nakhon Nayok, Thailand

Corresponding Author: Panuwat Wongwattana, Department of Otolaryngology, Faculty of Medicine, Srinakharinwirot University, Nakhon Nayok, Thailand, Phone: +66-37-395085, e-mail: panuwatw@g.swu.ac.th

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The patient was postulated to be afflicted with a partially ameliorated manifestation of streptococcal tonsillitis, given the clinical presentation and incomplete resolution of symptoms. The patient was hospitalized for intravenous fluid resuscitation due to dehydration and intravenous amoxicillin/clavulanic acid 1.2 grams q 8 hours. Forty-eight hours after admission, the clinical symptoms were stable. Physical examination showed a decrease of whitish membranous pharyngotonsillitis, multiple shallow ulcers on an erythematous base at the bilateral tonsils and posterior pharyngeal wall (Fig. 1). The indirect laryngoscopic examination shows multiple shallow ulcers on an erythematous base at the posterior pharyngeal wall, extending to the hypopharynx.

For the investigation, the complete blood count showed white blood cell count 10,600, neutrophil 80%, lymphocyte 12%, monocyte 6.2%. Serological analysis revealed immunological markers indicative of a prior EBV infection (VCA-IgG positive, EBNA-IgG positive, and VCA-IgM negative). The preliminary polymerase



Fig. 1: Oropharyngeal examination revealed a whitish membranous appearance of the pharyngeal mucosa with erythematous and injected tonsils. Bilateral tonsillar enlargement (grade 2+) was noted, accompanied by multiple shallow ulcers with erythematous bases on both tonsillar surfaces and the posterior pharyngeal wall.

chain reaction (PCR) assay performed on a nasopharyngeal swab specimen yielded a negative result for SARS-CoV-2. Chest radiography was normal.

Polymerase chain reaction analysis of a throat swab specimen identified the presence of HSV type II, but was negative for HSV type I, Varicella zoster virus, EBV, and cytomegalovirus. The throat swab for bacterial culture was normal flora, and the throat swab KOH preparation was reported as negative for fungus.

A definitive diagnosis of HSV-induced pharyngotonsillitis was established based on clinical presentation and corroborative molecular diagnostic findings. The patient was administered oral acyclovir (400 mg five times/day) for 5 days as part of the treatment regimen. On 1 week follow-up, the patient's clinical condition had improved, and on examination, a decrease in size of multiple shallow ulcers on an erythematous base at bilateral tonsil and posterior pharyngeal wall was observed. At the two-week follow-up appointment, the patient exhibited complete clinical resolution of pharyngotonsillitis, with no residual signs or symptoms noted on examination.

DISCUSSION

Pharyngotonsillitis patients typically present with nonspecific upper respiratory symptoms, including nasal congestion, low-grade fever, hoarseness, and malaise. For bacterial infections, patients often have a high-grade fever, chills, painful swallowing, and difficulty swallowing, but usually lack the general symptoms typical of viral infections.⁶

In practice, distinguishing between viral and bacterial causes of acute bacterial pharyngitis is based on history-taking and physical examination. To confirm GABHS infection, rapid antigen testing (streptococcal carbohydrate), or other methods like ELISA and throat swab cultures, may be used. The Centers for Disease Control and Prevention (CDC), American Academy of Family Physicians (AAFP), and the American College of Physicians (ACP) recommend using the Centor scoring system to differentiate between causes. This system uses the patient's history, symptoms, and signs to aid in treatment decisions. The criteria include: (1) fever ($>38^{\circ}\text{C}$), (2) swollen cervical

lymph nodes, (3) tonsillar exudates (pus on the tonsils), and (4) absence of cough. If the 0–1 criteria are met, symptomatic treatment is recommended. If 2–3 criteria are met, a rapid antigen test should be performed, and if positive, antimicrobial therapy is given. If all 4 criteria are met, antimicrobial therapy should be considered.⁷ If the criteria are not met, a viral infection should be suspected.

Acute pharyngitis is among the most infectious conditions encountered by primary care physicians. Viral etiology is the most prevalent cause of disease.⁶

Acute pharyngotonsillitis caused by HSV type II was first reported in 1978.⁸ The clinical presentation of acute HSV infection is influenced by multiple factors, including the patient's age, the route of viral transmission, and the integrity of the host's immune system. Gingivostomatitis represents the most prevalent clinical manifestation of oropharyngeal HSV infection in the pediatric population. In addition to gingival inflammation and ulceration of the oral mucosa, patients may also present with systemic symptoms such as fever, malaise, and lymphadenopathy. Up to one-third of cases of gingivostomatitis may also be accompanied by membranous pharyngotonsillitis.⁹

In most cases, viral pharyngotonsillitis is a short-term illness, so treatment will be focused on symptomatic treatment. However, in some cases, especially in patients with poor immune status, the infection can develop into severe forms and may become life-threatening.

Herpes simplex virus is one of the most prevalent human pathogens, with direct contact being the main route of transmission. Herpes simplex virus type I is primarily associated with orofacial lesions, while HSV type II is associated with genital herpes. However, there has been a rise in the prevalence of HSV type I genital herpes in recent years. Even though primary infections with HSV type I most commonly occur during infancy and childhood, those with HSV type II primarily affect adolescents and adults, as the virus is primarily acquired through sexual activity.¹⁰

Most herpes pharyngotonsillitis cases are caused by HSV type I. However, HSV type II can present with similar clinical features.¹¹ Herpes simplex virus type II typically causes anogenital lesions, as it is primarily transmitted through sexual contact.¹² Nevertheless, the sites of HSV infection now often overlap due to orogenital transmission.¹³

Herpetic pharyngotonsillitis is usually a self-limited disease, often presenting with adenoid or tonsillar hypertrophy and chronic adenoiditis or tonsillitis. Previous literature has shown that only a few cases have undergone histological examination to confirm the pathogen.^{8,14–17}

The study by Annunziato and Gershon¹⁸ and the study by McMillan et al.¹⁹ reported the clinical manifestations of pharyngitis due to HSV type I infection: Pharyngeal edema (71%), tonsillar exudate (40%), exudative and ulcerative lesions in the throat (34%), other symptoms include fever, fatigue, and swollen cervical lymph nodes.

McMillan et al.¹⁹ study reported that acute pharyngitis caused by HSV accounted for 5.7% of cases. There has been a reported decline in HSV type I and HSV type II infections among young people in Europe, North America, and Asia. However, primary HSV infection is more common in young adults than in children due to sexual activity.^{20–23} Therefore, in young adult patients with acute infectious pharyngitis, it may be necessary to inquire about sexual history to aid in differential diagnosis.²⁴

During the diagnostic evaluation, the use of clinical scoring systems can assist healthcare providers in more accurately

identifying cases with a higher likelihood of bacterial etiology, thereby guiding the selection of patients who would benefit from rapid antigen detection testing. In the absence of such diagnostic strategies, there is a tendency toward overdiagnosis of streptococcal pharyngitis, frequently leading to the unwarranted administration of antibiotics, predominantly broad-spectrum agents—which contributes to antimicrobial overuse and resistance.

The treatment for herpetic pharyngotonsillitis depends on whether the infection is primary or recurrent, alongside the severity of clinical manifestations and the immunological competence of the host. Antiviral therapy aims to shorten the duration of symptoms and accelerate the healing process to prevent disease transmission.²⁵

According to the review, the recommended treatment for acute pharyngitis caused by HSV includes: (1) Acyclovir: 400 mg taken orally three times daily or 200 mg taken orally five times daily for 7–10 days, (2) Famciclovir: 250 mg taken orally three times daily or 500 mg taken orally twice daily for 7–10 days, and (3) Valacyclovir: 1 gram taken orally twice daily for 7–10 days.^{26–28}

CONCLUSION

Herpetic pharyngotonsillitis is an uncommon disease and typically self-limiting. The treatment depends on the immune status and severity of the disease. An antiviral drug may shorten the duration of symptoms and help to improve the healing process. Early recognition and appropriate antiviral therapy can reduce complications and healthcare burden.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The Human Ethics Committee of Srinakharinwirot University approved the study (SWUEC/X/M-014/2566). Informed consent was obtained from the participant before including him in the trial.

ORCID

Chalisara Subongkot  <https://orcid.org/0009-0005-0547-6002>

Panuwat Wongwattana  <https://orcid.org/0000-0003-1348-0739>

Alena Santeerapharp  <https://orcid.org/0000-0001-5592-0849>

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