Review Article - Narrative Review

Taiwan J Ophthalmol 2025;15:212-217

Access this article online

Quick Response Code:

Website: http://journals.lww.com/TJOP

DOI: 10.4103/tjo.TJO-D-25-00032

Comprehensive insights into cytomegalovirus anterior segment infections: A narrative review

Yih-Shiou Hwang[1], Po-Yi Wu[1], Eugene Yu-Chuan Kang[1], Wei-Chi Wu[1], Linda Yi-Hsing Chen[1], Chi-Chun Lai[1], Kyung Seek Choi[2]*

Abstract:

Cytomegalovirus (CMV) anterior uveitis (AU), a significant cause of intraocular inflammation, is increasingly recognized in immunocompetent individuals, often leading to visual morbidity if not promptly addressed. The diagnosis of CMV AU is challenging, owing to its variable clinical manifestations, which can overlap with other forms of AU. CMV AU should be suspected in corticosteroid-recalcitrant inflammatory ocular hypertensive syndrome or corneal endotheliitis with coin-shaped keratic precipitates (KPs). CMV AU differs from herpes simplex virus and varicella-zoster virus AU with milder symptoms, less ciliary injection, smaller KPs, higher intraocular pressure (IOP), and diffuse iris atrophy. Aqueous humor analysis, specifically polymerase chain reaction (PCR), is the gold standard for diagnosis, detecting viral DNA, and quantifying disease severity. While highly effective, PCR can yield false negatives with low viral loads. Clinical judgment remains crucial, alongside PCR results. Early diagnosis and targeted antiviral treatment are key to preserving visual function and preventing complications, such as glaucoma and keratopathy. CMV AU treatment aims to control inflammation, reduce viral activity, and prevent complications. Antiviral therapy is crucial, with topical ganciclovir (GCV) gel often first line. Oral valganciclovir is used for systemic treatment, especially in severe cases. Intravitreal GCV may be used in severe cases, often followed by systemic therapy, but its role remains suspicious. Corticosteroids should only be used with antiviral therapy. Topical corticosteroids manage inflammation and are tapered over time. IOP management is also essential, potentially requiring surgery. Treatment duration varies, and long-term maintenance may be necessary. More research is needed to standardize treatment protocols and further understand the pathogenesis and immunopathogenesis of CMV anterior uveitis.

Keywords:

Anterior uveitis, cytomegalovirus anterior uveitis, ganciclovir, immune mechanisms, intraocular pressure, keratic precipitates, polymerase chain reaction, valganciclovir, viral uveitis

[1]Department of Ophthalmology, Linkou Chang Gung Memorial Hospital, Chang Gung University, Linkou, Taipei, Taiwan, [2]Department of Ophthalmology, Soonchunhyang University Seoul Hospital, Soonchunhyang University College of Medicine, Seoul, Korea

***Address for correspondence:**
Prof. Kyung Seek Choi, Soonchunhyang University Seoul Hospital, Soonchunhyang University College of Medicine, 59 Daesagwan-Ro, Yongsan-Gu, Seoul 04401, Korea.
E-mail: ckseek@naver.com

Submission: 16-02-2025
Accepted: 11-03-2025
Published: 10-06-2025

Introduction

Cytomegalovirus (CMV) is a ubiquitous herpesvirus that can cause a variety of clinical manifestations. In immunocompetent individuals, CMV anterior uveitis (AU) has emerged as a significant ocular condition, often leading to visual impairment if not promptly recognized and treated. However, diagnosing CMV AU remains challenging due to its diverse clinical presentations that can mimic other viral anterior uveitides, such as those caused by herpes simplex virus (HSV) and varicella-zoster virus (VZV).[1,2] This diagnostic difficulty often leads to delays in appropriate treatment, potentially affecting visual prognosis. Given the variability in clinical presentations and the lack of specific clinical indicators, relying solely on clinical findings for diagnosis is problematic. While polymerase chain reaction (PCR) testing of aqueous humor is essential for confirming the diagnosis, this method is not universally

How to cite this article: Hwang YS, Wu PY, Kang EY, Wu WC, Chen LY, Lai CC, *et al*. Comprehensive insights into cytomegalovirus anterior segment infections: A narrative review. Taiwan J Ophthalmol 2025;15:212-7.

accessible. In addition, serological tests have limited diagnostic value because most adults have been exposed to CMV.[1]

Current therapeutic strategies for CMV AU are varied, including topical antivirals such as ganciclovir (GCV), oral antivirals like valganciclovir (VGCV), and intravitreal GCV injections.[3] However, there is currently no standardized treatment protocol for CMV AU, leading to significant variations in treatment efficacy and outcomes. Some studies advocate topical GCV gel as a first-line treatment,[1,3-5] while others promote oral VGCV or combinations of different therapies.[1,2,4,6,7] This variability in treatment options underscores the need for a comprehensive analysis of current practices.[4]

This review paper aims to (a) Provide a comprehensive overview of the clinical manifestations, diagnostic approaches, and treatment strategies for CMV AU. We would like to offer the practical guidance for clinicians to enhance the diagnosis and treatment of CMV AU, ultimately improving patient outcomes. (b) Summarize current research advancements and expert consensus on CMV AU. (c) Highlight the challenges and unmet needs in the diagnosis and management of this condition.

Clinical Manifestations of Cytomegalovirus Anterior Uveitis

CMV AU presents a considerable diagnostic challenge due to its varied clinical manifestations and its similarity to other viral infections. As an infectious condition caused by the CMV, it requires a high index of suspicion for accurate diagnosis, often being mistaken for conditions like HSV or VZV infections.[8] The variable nature of its presentation further complicates diagnosis, and while definitive diagnosis relies on aqueous fluid analysis and PCR to detect CMV DNA, there is a lack of readily available, noninvasive diagnostic tools.

Despite the diagnostic difficulties, CMV AU has several distinguishing clinical features that can aid in its identification. One of the key features is a disproportionately high intraocular pressure (IOP) relative to the degree of inflammation. The presence of coin-shaped keratic precipitates (KPs) on the corneal endothelium is another characteristic finding.[4,9] In contrast to HSV or VZV AU, CMV AU is often associated with mild anterior chamber inflammation, less ciliary injection, and fewer cells and flare. It may also manifest with reduced corneal endothelial cell count and can cause corneal edema.[3] Moreover, CMV AU can resemble conditions such as Posner–Schlossman syndrome or Fuchs uveitis yndrome.[2,6,7] It frequently presents as chronic or recurrent uveitis and is more prevalent in Asia and more common in males.[1,2,4,10] Thus, while diagnosis can be challenging due to varied presentations and similarities to other conditions, there are distinct features, particularly the high IOP and coin-shaped KPs, that can help raise suspicion of CMV as a causative agent.[7,9] Early detection using PCR and prompt treatment combining antiviral and anti-inflammatory therapies are essential for preventing long-term damage. Based on our study, CMV AU should be suspected in cases of non-HSV/VZV corticosteroid-recalcitrant inflammatory ocular hypertensive syndrome (IOHS) or in cases of corneal endotheliitis with specific coin-shaped KPs.[9] The study found that these two clinical profiles had high positive predictive values for CMV anterior segment infection. The first profile, IOHS, refers to AU with elevated IOP that does not respond well to corticosteroid treatment. The second profile is characterized by corneal edema and distinctive coin-shaped KPs. The study suggests that when patients are present with either of these profiles, clinicians should consider the possibility of CMV infection.

Based on Terada's report, the ocular features of CMV AU differed significantly from those of HSV/VZV AU.[3] Although all three were viral infections affecting the anterior segment of the eye, their clinical presentations vary. In terms of symptoms, patients with HSV-AU and VZV-AU often presented with more pronounced eye redness, pain, and blurred vision, while CMV-AU tended to have milder or less noticeable symptoms. Regarding signs, ciliary injection (or ciliary flush) was more common in HSV-AU and VZV-AU, while it was less frequently observed in CMV-AU. Although all three conditions may cause KPs, their size, shape, and distribution differ. HSV-AU and VZV-AU typically exhibit medium-to-large KPs, whereas CMV-AU is characterized by small KPs, particularly coin-shaped KPs. In addition, CMV-AU KPs are less likely to be pigmented, while VZV-AU KPs are more prone to pigmentation. The anterior chamber inflammation also differs: HSV-AU and VZV-AU usually show more prominent anterior chamber cells and flare, while CMV-AU presents milder inflammation. Fibrin is more often observed in the anterior chamber of HSV-AU and VZV-AU compared to CMV-AU. Furthermore, posterior synechiae are more commonly seen in HSV-AU and VZV-AU but less so in CMV-AU. Regarding IOP, CMV-AU is more likely to cause elevated IOP, sometimes requiring glaucoma surgery. In contrast, IOP elevation is less common in HSV-AU and VZV-AU. In terms of the iris, although all three conditions can cause iris atrophy, diffuse iris atrophy is more frequently seen in CMV-AU. Segmental iris atrophy is more typical of VZV-AU. Overall, HSV-AU and VZV-AU share similar clinical features, while CMV-AU differs significantly from both. CMV-AU typically presents with milder inflammation, small KPs, particularly coin-shaped KPs, higher IOP, and diffuse iris atrophy. In contrast, HSV-AU and VZV-AU

are characterized by more pronounced eye redness, pain, ciliary injection, larger KPs, and more significant anterior chamber cells and flare.[3]

Diagnostic Approaches of Cytomegalovirus Anterior Uveitis

Aqueous humor analysis is the most effective method for diagnosing and determining the severity of CMV AU. This analysis involves extracting aqueous humor from the anterior chamber for PCR and Goldmann-Witmer coefficient testing. PCR is a rapid and highly specific assay that can directly identify the virus by detecting even small amounts of DNA. Real-time PCR can quantify viruses and assess disease severity and treatment effects.[1] Multiplex PCR can be used to screen for multiple viruses.[5]

Although PCR testing is highly effective, false negative results can occur if viral DNA levels drop below the detection threshold, and a positive PCR result can be found in the presence of latent viral DNA. Thus, clinical judgment, along with PCR test results, is often considered in the diagnosis.[11] While CMV serology is not usually helpful for diagnosis, as most adults have had prior exposure, some experts, particularly in Europe, may perform serology along with aqueous PCR.[2,4] In summary, diagnosis of CMV AU relies on a combination of clinical findings and laboratory testing, most notably, PCR testing of aqueous fluid, with clinical suspicion driven by key findings including elevated IOP, mild inflammation, and specific patterns of KPs.

It should be notified that cases with an initial negative PCR result but high clinical suspicion were not uncommon. La Distia Nora *et al*. reported that 3 out of 24 eyes had negative PCR results on the first tap but later turned positive on subsequent taps, possibly due to increased viral load as inflammation worsened.[2] Additionally, Pleyer and Chee reported that in cases suspected of CMV infection but with an initial negative PCR, especially during the acute phase with elevated IOP, repeat testing may be necessary due to potential false negatives.[11] Even in patients with unexplained corneal decompensation and graft failure after penetrating keratoplasty, anterior chamber tap for CMV DNA analysis at the time of repeat keratoplasty may be helpful for diagnosis, even if previous PCR results were negative.

Obtaining a positive PCR result from a repeat anterior chamber tap clearly alters clinical management. If the initial negative result caused hesitation in administering antiviral treatment, a subsequent positive result will definitively diagnose viral AU and prompt the clinician to initiate or adjust the antiviral treatment regimen immediately. For example, in patients with a high suspicion of CMV AU, clinicians may have already started antiviral treatment based on clinical suspicion even if the initial PCR was negative. A positive result from a repeat PCR would support the previous clinical judgment and encourage continued antiviral therapy, potentially helping to improve patient prognosis and reduce the risk of complications such as glaucoma. Conversely, if the repeat PCR result remains negative, clinicians will need to re-evaluate the diagnosis, consider other non-viral etiologies, and adjust treatment strategies accordingly. This also highlights the importance of interpreting PCR results in conjunction with clinical presentation, medical history, and other auxiliary examination results. In summary, in cases where the initial anterior chamber tap PCR result is negative but clinical suspicion for viral AU remains high, performing a repeat tap is a prudent course of action. This measure helps to improve diagnostic accuracy and facilitate timely adjustments in clinical management decisions, particularly regarding the initiation and continuation of antiviral treatment, thereby potentially improving the patient's visual outcome.

Treatment Strategies for Cytomegalovirus Anterior Uveitis

Treatment strategies for CMV AU aim to control inflammation, reduce viral activity, and prevent complications such as glaucoma and corneal damage. It has been suggested that there is no single, universally accepted standard treatment protocol for CMV AU, and approaches can vary widely depending on disease severity and chronicity.[1] However, several treatment modalities are commonly employed. Antiviral medications are the cornerstone of therapy for CMV AU. These medications work by inhibiting CMV DNA replication. GCV and its prodrug, VGCV, are the most frequently used antiviral agents. GCV can be administered topically, intravenously, or intravitreally, while VGCV is typically given orally. Topical GCV gel 0.15% is often the first-line treatment, with a majority of experts (70%) choosing this option.[2] For topical treatment, GCV gel 0.15% is typically applied three to four times daily for 1 month. Some sources suggest a higher concentration (0.5%–2%) of topical GCV is needed for endotheliitis, with 6–8 times daily application during the induction phase. A 2% topical GCV solution can be prepared from a lyophilized powder.

Oral VGCV is favored for systemic treatment and is typically used at a dosage of 900 mg twice daily for 2–3 weeks. Some experts, however, would only use systemic antivirals for severe, prolonged, or atypical cases of CMV AU.[4] For chronic CMV AU, some sources recommend an increased antiviral regimen, such as 1%–2% topical GCV six times daily for 2–4 weeks or

oral VGCV 900 mg twice daily for 3 weeks. Maintenance treatment with GCV gel 0.15% twice daily or oral VGCV 450 mg once or twice daily for up to 12 months can be considered for chronic cases or those with frequent recurrences. Monitoring of complete blood counts, renal function, and liver function is recommended for patients on systemic VGCV.

Intravitreal GCV injections have been used, especially in cases of severe disease. A loading dose of intravitreal GCV (2 mg/0.05 mL) is sometimes used, followed by oral VGCV, depending on postinjection inflammation.[7] Some sources suggest that intravitreal injection may achieve a higher concentration at the site of infection. However, intravitreal GCV has shown inconsistent results, and a high rate of recurrence has been observed after intravitreal treatment cessation.[2] Intracameral injections are not favored due to rapid drug elimination and potential corneal toxicity. Combination treatment with topical and systemic antivirals is frequently used, especially in severe cases. The efficacy of early oral VGCV versus topical GCV is being explored.

It is important to note that the first-line treatment strategies for CMV AU vary significantly across the globe, primarily influenced by local clinical practice guidelines and the availability of specific antiviral medications. The TITAN Report 2 provides key insights into these diverse practice patterns by surveying uveitis experts worldwide.[4] According to the findings of this report, there is a general consensus among international uveitis specialists towards topical antiviral treatment as the preferred initial approach for managing CMV AU (85% of experts agree), with GCV gel 0.15% being the most favored topical agent (chosen by 70% of experts). However, opinions diverge regarding the necessity of adding systemic antiviral therapy at the initial stage. Approximately half of the experts (48%) would only consider commencing systemic treatment in cases of severe, prolonged, or atypical CMV AU presentation. When systemic treatment is deemed necessary, oral VGCV is the preferred drug (selected by 78% of experts).

The results of the TITAN Report 2 also indirectly suggest regional differences in practice. While the report does not explicitly detail country-specific first-line treatments, it points out variations in diagnostic and management approaches across different geographical regions such as Asia, Europe, and North America. For instance, a higher percentage of experts in Asia (42%) reported routinely using both topical and systemic GCV on diagnosis compared to Europe (23%) and North America (30%). While other papers provided supplementary information about CMV ocular infections and treatment options, such as studies from Japan focusing on the efficacy of topical GCV gel for CMV endotheliitis[5] and research from Southern Taiwan highlighting the prevalence of CMV in herpetic AU, these documents do not directly compare first-line treatment strategies across different countries. The TITAN Report 2 serves as the most direct evidence of the variations in first-line treatment for CMV AU worldwide, emphasizing the widespread preference for topical GCV gel as initial therapy while also revealing significant differences in the use of systemic antiviral agents at the onset of treatment. These variations are likely driven by local guidelines and the availability of specific medications in different countries and regions.

In addition to antiviral medications, topical corticosteroids are frequently used to manage inflammation.[4] Prednisolone acetate 1% is often the preferred corticosteroid, applied four times daily for 1–2 weeks, then adjusted depending on the clinical response. The corticosteroids are often tapered over time, sometimes over as long as 12 months, in conjunction with antiviral coverage. A critical aspect to understand about CMV AU is that treatment with corticosteroids alone can worsen or prolong the disease course, making it imperative that topical corticosteroids be used only in conjunction with antiviral therapy.[1,2,4]

It is also important to manage IOP, which is a common feature of CMV AU. If elevated IOP is not controlled with medications, surgery may be required.

The duration of treatment is variable. Some experts recommend that antiviral treatment should be continued for at least 5 weeks. In long-term management and recurrence, long-term maintenance treatment for up to 12 months is considered for chronic cases or those with recurrent episodes. Recurrence is common after stopping treatment. Long-term use of topical corticosteroids may be needed, with very slow tapering. Many experts would consider restarting initial dosages of both antiviral and anti-inflammatory medications for recurrences.[4]

Finally, we emphasize the need for more research to establish definitive treatment guidelines, given the variability in current practices.

Pathogenesis of Cytomegalovirus Anterior Uveitis

Several studies suggest that the pathogenesis of CMV iritis involves multiple factors. CMV iritis most commonly occurs in immunocompetent individuals, indicating that immune responses play a critical role in disease development.[12] CMV has the ability to establish latent infections, and inadequate treatment may lead to persistent or recurrent inflammation. Moreover, CMV can modulate the expression of HLA-E through various strategies to evade immune system surveillance,

including the response of natural killer cells. HLA-E is expressed on vascular endothelial cells in the human iris, suggesting the possibility of interactions between CMV and the host immune system via HLA-E.[13] Pattern recognition receptors such as TLR2, TLR3, TLR4, and TLR9 are involved in antiviral responses to HSV, VZV, and CMV infections, and specific T cells play a crucial role in controlling CMV infection.[10] Overall, the pathogenesis of CMV iritis involves complex mechanisms by which the virus evades immune surveillance, as well as the role of host immune responses in disease development.

Current Research Advancements and Expert Consensus on Cytomegalovirus Anterior Uveitis

Other considerations include a global study of uveitis specialists revealing significant variations in preferred management practices, emphasizing the need for standardization. CMV AU is more frequent in Asia, accounting for up to 66% of viral AU.[4] Corneal edema may persist despite the resolution of other clinical signs, possibly due to corneal decompensation. The role of HLA-E in CMV's evasion of immunity is being investigated.[13] Multiple gB genotypes of CMV have been detected in many patients with anterior segment infection, and the gB3 genotype may be linked to poorer IOP control.[14] There is an urgent need for more effective and standardized treatment protocols, especially for chronic and recurrent cases. Further research is needed to optimize the use and duration of corticosteroids, improve long-term strategies, and prevent recurrences. While our understanding of CMV AU is constantly improving, more research is needed to refine diagnostic and treatment guidelines and develop more effective therapies.

Highlight the Challenges and Unmet Needs in the Diagnosis and Management of this Disease

In clinical practice, determining the direction of treatment for patients with CMV AU is often difficult due to inconsistent clinical presentations. Clinicians often face situations where patients have elevated IOP without significant inflammation or significant inflammation without elevated IOP. This challenge primarily arises from the diverse clinical presentations of CMV AU, making it difficult to determine whether the treatment should focus on suppressing immune inflammation or suppressing the virus. The clinical heterogeneity of CMV AU makes it difficult to formulate a consistent treatment approach. Currently, the treatment methods for CMV AU vary, including topical and systemic antiviral drugs, as well as topical corticosteroids. Therefore, extended anti-CMV treatment with or without topical corticosteroids may be used, even the new inflammation is virus-related or not. However, there is no clear consensus on when to use topical or systemic antiviral drugs and when to use corticosteroids to control inflammation.

Clinicians need to comprehensively consider the patient's clinical presentation, laboratory test results (e.g., PCR testing of aqueous humor samples), and the potential risks and benefits of treatment to make the best treatment decision when treating CMV AU. It is necessary to accurately determine whether CMV is the real cause of uveitis and to rule out other possible causes. In addition, it is also necessary to decide how to balance controlling viral replication and regulating immune inflammation, while considering the individual conditions and potential side effects of the patient. Despite advancements in the diagnosis and management of CMV AU, several challenges and unmet needs persist: (a) Limitations in diagnostic methods and the need for early and accurate diagnosis. (b) Uncertainty regarding the optimal use and duration of antiviral medications. (c) The need for standardized treatment protocols to reduce practice variation. (d) Limited understanding of prognostic factors and recurrence mechanisms. (e) Limited understanding of the pathogenesis. Addressing these unmet needs will require multidisciplinary collaborations and large, well-designed studies to explore more effective diagnostic and therapeutic strategies, ultimately leading to improved outcomes for patients with CMV AU.

Data availability statement

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

Financial support and sponsorship

CMRPG3M0321-2, BMRPF29.

Conflicts of interest

Dr. Yih-Shiou Hwang,Wei-Chi Wu, and Chi-Chun Lai, editorial board members at Taiwan Journal of Ophthalmology, had no role in the peer review process of or decision to publish this article. All authors declared no conflicts of interest in writing this paper.

References

1. Zhang J, Kamoi K, Zong Y, Yang M, Ohno-Matsui K. *Cytomegalovirus* anterior uveitis: Clinical manifestations, diagnosis, treatment, and immunological mechanisms. Viruses 2023;15:185.
2. La Distia Nora R, Putera I, Mayasari YD, Hikmahwati W, Pertiwi AM, Ridwan AS, *et al*. Clinical characteristics and treatment outcomes of *Cytomegalovirus* anterior uveitis and endotheliitis: A systematic review and meta-analysis. Surv Ophthalmol 2022;67:1014-30.
3. Terada Y, Kaburaki T, Takase H, Goto H, Nakano S, Inoue Y,

et al. Distinguishing features of anterior uveitis caused by herpes simplex virus, varicella-zoster virus, and cytomegalovirus. Am J Ophthalmol 2021;227:191-200.
4. Thng ZX, Putera I, Testi I, Chan K, Westcott M, Chee SP, *et al*. The infectious uveitis treatment algorithm network (TITAN) report 2-global current practice patterns for the management of *Cytomegalovirus* anterior uveitis. Eye (Lond) 2024;38:68-75.
5. Koizumi N, Miyazaki D, Inoue T, Ohtani F, Kandori-Inoue M, Inatomi T, *et al*. The effect of topical application of 0.15% ganciclovir gel on cytomegalovirus corneal endotheliitis. Br J Ophthalmol 2017;101:114-9.
6. Cheng YC, Kang EY, Hwang YS, Hsiao CH. Treatment of cytomegalovirus anterior segment infection with intravitreal injection of ganciclovir in adjunction with or without oral valganciclovir: A long-term results. Sci Rep 2021;11:3105.
7. Hwang YS, Lin KK, Lee JS, Chang SH, Chen KJ, Lai CC, *et al*. Intravitreal loading injection of ganciclovir with or without adjunctive oral valganciclovir for *Cytomegalovirus* anterior uveitis. Graefes Arch Clin Exp Ophthalmol 2010;248:263-9.
8. Chiang WY, Chen SC, Sheu SJ, Kuo HK. A prospective study of clinical features of anterior uveitis in Taiwan. J Ophthalmol 2023;2023:9647418.
9. Hwang YS, Shen CR, Chang SH, Lai CC, Liu CL, Chen KJ, *et al*. The validity of clinical feature profiles for cytomegaloviral anterior segment infection. Graefes Arch Clin Exp Ophthalmol 2011;249:103-10.
10. Liu Y, Li F, Du L, Pang T, Ma X, Cao Q, *et al*. Association of TLR2 gene polymorphisms with presumed viral-induced anterior uveitis in male Han Chinese. Exp Eye Res 2019;187:107777.
11. Pleyer U, Chee SP. Current aspects on the management of viral uveitis in immunocompetent individuals. Clin Ophthalmol 2015;9:1017-28.
12. Sester M, Sester U, Gärtner BC, Girndt M, Meyerhans A, Köhler H. Dominance of virus-specific CD8 T cells in human primary *Cytomegalovirus* infection. J Am Soc Nephrol 2002;13:2577-84.
13. Pietra G, Romagnani C, Mazzarino P, Falco M, Millo E, Moretta A, *et al*. HLA-E-restricted recognition of *Cytomegalovirus*-derived peptides by human CD8+cytolytic T lymphocytes. Proc Natl Acad Sci U S A 2003;100:10896-901.
14. Huang CY, Cheng YC, Hwang YS, Kang EY, Hsiao CH. *Cytomegalovirus* glycoprotein B genotype in patients with anterior segment infection. Int J Mol Sci 2023;24:6304.