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# Syphilitic Necrotizing Retinitis Mimicking Cytomegalovirus Retinitis: A Case Report

Maria Garcia [1], Daniela Busquets [1], Armando Oliver [1]

1. Department of Ophthalmology, School of Medicine, Medical Sciences Campus, University of Puerto Rico, San Juan, PRI

**Corresponding author:** Maria Garcia, maria.garcia49@upr.edu

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## Abstract

Retinitis is an inflammation of the neurosensory retina. Herein, we describe a case of a 40-year-old man diagnosed with syphilitic necrotizing retinitis that was initially suspected to be cytomegalovirus (CMV) retinitis. The patient presented with a one-month history of blurred vision, floaters, and a dark silhouette in the left eye. Ophthalmic examination revealed anterior chamber and vitreous inflammation, along with a large, well-demarcated area of ground-glass yellow-white necrotizing retinitis with numerous creamy-yellow superficial retinal precipitates clustered along its inferior border - an appearance that initially raised concern for CMV retinitis. A comprehensive infectious work-up revealed positive results for rapid plasma reagin and fluorescent treponemal antibody absorption testing and a negative HIV test status, supporting the diagnosis of syphilitic necrotizing retinitis. The patient was treated with intravenous penicillin G and oral prednisone, with marked resolution of the retinitis and disappearance of the precipitates following the 14-day course. This case underscores the importance of including syphilis in the differential diagnosis of necrotizing retinitis, including in HIV-negative patients, and highlights superficial retinal precipitates overlying ground-glass retinitis as a potential diagnostic clue.

**Categories:** Ophthalmology
**Keywords:** case report, cytomegalovirus, hiv, infectious retinitis, syphilis, syphilitic retinitis

## Introduction

Retinitis is an inflammation of the neurosensory retina that can lead to irreversible vision loss if left untreated [1]. Cytomegalovirus (CMV) is a leading cause of infectious retinitis in immunocompromised patients, particularly those with advanced HIV infection with very low CD4 counts. CMV retinitis may present as areas of full-thickness retinal necrosis, with or without intraretinal hemorrhage, and may be accompanied by tiny, granular, dot-like “satellites” at the interface between infected and unaffected retina [2]. Three clinical patterns have been described: a fulminant pattern, with hemorrhagic necrosis on white-yellow cloudy retinal lesions that may be centered around the vasculature; a granular pattern, found more often in the retinal periphery, with little to no necrosis or hemorrhage; and a perivascular pattern resembling “frosted branch” angiitis [3]. The confluent retinal whitening with hemorrhages has given rise to the classic descriptors “pizza pie” and “cottage cheese and ketchup” retinopathy [3,4]. Conversely, syphilis, often referred to as “the great imitator,” can mimic nearly every form of intraocular inflammation and remains in the differential diagnosis for all anatomical subtypes of uveitis because it generally lacks pathognomonic clinical features [5].

Syphilis has re-emerged as a systemic infection [6], and increases in suspected ocular syphilis cases have been reported [7]. In a surveillance report from eight United States jurisdictions, suspected ocular syphilis accounted for 0.60% of reported syphilis cases, and half of the affected patients were HIV-positive [7]. Although syphilis and CMV retinitis may appear similar, syphilis produces a broad spectrum of posterior segment manifestations, including acute syphilitic posterior placoid chorioretinitis, punctate inner retinitis, and focal or necrotizing retinitis, and the focal necrotizing phenotype, which is rare, may bear considerable similarity to CMV retinitis [6,8]. Recognizing the distinguishing elements of each entity is essential because they require different antimicrobial treatment: CMV retinitis is managed with antiviral therapy, such as oral valganciclovir or intravenous (IV) ganciclovir, whereas ocular syphilis is treated as neurosyphilis with IV penicillin G [2,9]. This distinction is particularly relevant in HIV-negative patients, in whom viral or autoimmune etiologies may be presumed, subsequently resulting in delayed or omitted syphilis serologic testing and, consequently, delayed appropriate treatment and possible irreversible vision loss. This report describes syphilitic necrotizing retinitis mimicking CMV retinitis in an HIV-negative patient, documents, with multimodal imaging, the superficial retinal precipitates overlying the necrotizing lesion as a potential diagnostic clue, and emphasizes the importance of prompt syphilis serologic testing and HIV testing in all patients presenting with necrotizing retinitis.

## Case Presentation

A 40-year-old Hispanic man with a history of myopia and fatty liver disease presented with a one-month history of blurred vision, floaters, and a dark silhouette in the left eye. He was taking no immunosuppressive

or corticosteroid medications and had no history of malignancy, organ transplantation, or cytotoxic chemotherapy. His social history included alcohol, tobacco, and cannabis use, as well as multiple female sexual partners and inconsistent use of protection. The review of systems was negative for headache, fever, chills, arthralgia, and skin rash.

On ophthalmological examination, the patient's best-corrected visual acuity (BCVA) was 20/25 in the right eye and 20/100 in the left eye. Intraocular pressure measured 12 mmHg in the right eye and 10 mmHg in the left eye. The pupils were round and reactive to light, bilaterally, with no afferent pupillary defect. A slit-lamp examination (SLE) of the left eye showed 1+ conjunctival injection, 1+ anterior chamber cells, and 2+ vitreous cells. The SLE of the right eye was essentially unremarkable. A fundus examination of the right eye showed clear media with no lesions in the macula or the peripheral retina. In contrast, that of the left eye revealed a large, well-demarcated confluent area of ground-glass yellow-white necrotizing retinitis involving the superotemporal posterior pole and extending peripherally toward the ora serrata, with an active border surrounding a central area of relative inactivity, suggesting different stages of lesion progression. Numerous discrete, round, creamy-yellow superficial retinal precipitates were clustered along the inferior border of the lesion; no intraretinal hemorrhages were present (Figure *1A*).

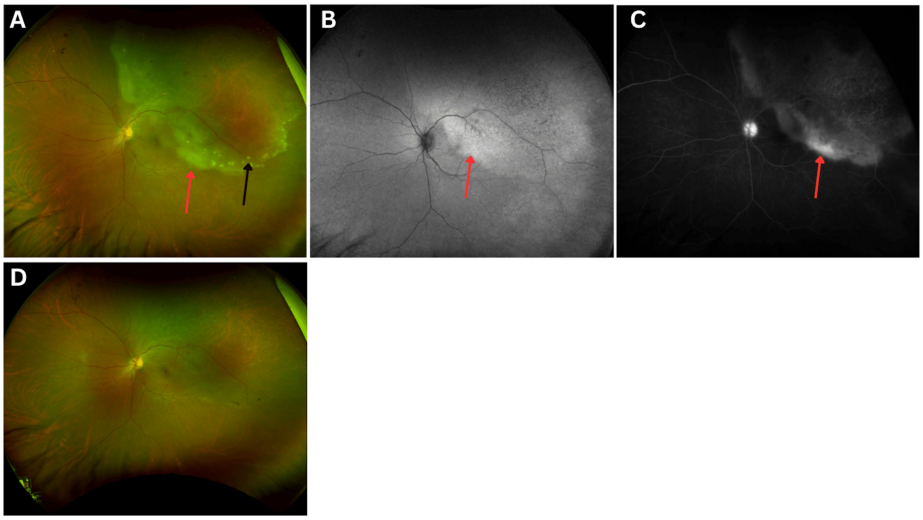



**FIGURE 1: Multimodal retinal imaging of the left eye**

(A) Ultra-widefield color fundus photograph at presentation demonstrating a large, well-demarcated area of ground-glass yellow-white retinitis involving the superotemporal posterior pole and extending peripherally toward the ora serrata. The lesion exhibits an active border (red arrow) with a central area of relative inactivity, suggesting different stages of lesion progression. Numerous discrete, round, creamy-yellow superficial retinal precipitates (black arrow) are clustered in a curvilinear distribution along the inferior margin of the lesion. No intraretinal hemorrhages are present. (B) Fundus autofluorescence at presentation demonstrating hyperautofluorescence along the active border of the lesion (red arrow) with granular hypoautofluorescence centrally, indicating extensive outer retinal and retinal pigment epithelium (RPE) involvement. (C) Late-phase fluorescein angiography at presentation demonstrating hyperfluorescence and staining along the active border of the retinitis (red arrow), with a transition to heterogeneous stippled fluorescence, centrally, and mild optic disc leakage. (D) Ultra-widefield color fundus photograph at the 14-day follow-up, after a 14-day course of intravenous penicillin, demonstrating marked resolution of the retinitis, with disappearance of the superficial retinal precipitates and residual granular RPE changes in the previously involved retina.

Fundus autofluorescence (FAF) of the right eye was within normal limits. However, FAF of the left eye demonstrated hyperautofluorescence along the active border of the lesion, with central granular hypoautofluorescence (Figure *1B*). Fluorescein angiography (FA) of the right eye revealed normal findings; however, the left eye showed hyperfluorescence and staining along the active border of the retinitis, with a transition to heterogeneous stippled fluorescence centrally and mild optic disc leakage (Figure *1C*). Spectral-domain optical coherence tomography (OCT) of the left eye demonstrated disruption and attenuation of the ellipsoid zone and outer retinal bands, with granular irregularity of the retinal pigment epithelium (RPE) temporal to the fovea and hyperreflective vitreous opacities consistent with vitreous cells. The foveal contour was preserved without intraretinal or subretinal fluid (Figure *2*).

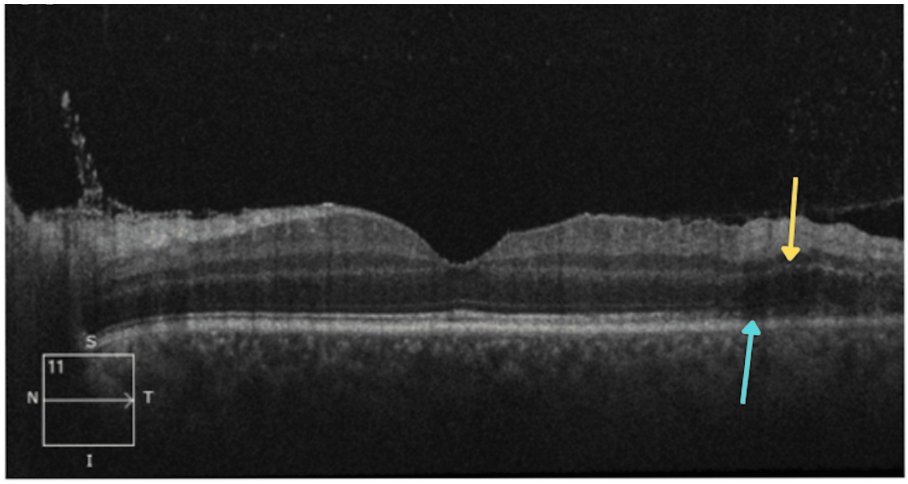



**FIGURE 2: Spectral-domain optical coherence tomography of the left eye**

Spectral-domain optical coherence tomography demonstrating disruption and attenuation of the ellipsoid zone (blue arrow) and outer retinal bands (yellow arrow), with granular retinal pigment epithelium irregularity temporal to the fovea, corresponding to the area of retinitis, and hyperreflective vitreous opacities consistent with vitreous cells. The foveal contour is preserved without intraretinal or subretinal fluid.

The findings of multimodal imaging led us to consider several causes of necrotizing retinitis - acute retinal necrosis (ARN) caused by herpes simplex virus (HSV) or varicella-zoster virus (VZV), progressive outer retinal necrosis, ocular syphilis, toxoplasmic retinochoroiditis, endogenous endophthalmitis, and vitreoretinal lymphoma - with CMV retinitis as the leading one. However, the degree of anterior chamber and vitreous inflammation was atypical for CMV retinitis in an apparently immunocompetent host, and a comprehensive work-up was therefore obtained before any antimicrobial therapy was initiated. Serologic testing revealed a reactive rapid plasma reagin test at a titer of 1:32 and a positive confirmatory fluorescent treponemal antibody absorption test. An HIV test was nonreactive. Toxoplasma gondii IgG and IgM serologies were nonreactive; hepatitis A, B, and C serologies showed no evidence of active infection; and rheumatoid factor was within normal limits. A lumbar puncture was performed, and the cerebrospinal fluid (CSF) Venereal Disease Research Laboratory (VDRL) test was nonreactive. An anterior chamber paracentesis was performed, and aqueous humor polymerase chain reaction (PCR) testing for HSV was negative; PCR testing for CMV and VZV was not performed due to sample size limitations. The positive syphilis serology, together with the clinical findings, led to a diagnosis of necrotizing syphilitic retinitis and consequently to the decision not to initiate antiviral therapy. Once the diagnosis of syphilis had been made, IV penicillin G (24 million units/day for 14 days), the recommended regimen for ocular syphilis [10], was initiated two days after presentation, and oral prednisone (1 mg/kg/day) was added the following day and tapered according to a standard schedule over a total of two weeks. The timeline of presentation, investigations, treatment, and outcomes is summarized in Table *1*.

| Time point | Events and findings |
|---|---|
| Approximately 1 month before presentation | Onset of blurred vision, floaters, and a dark silhouette in the left eye. |
| Presentation (day 0) | BCVA 20/100 in the left eye; anterior chamber and vitreous inflammation; necrotizing retinitis with superficial retinal precipitates; multimodal imaging; CMV retinitis considered the leading diagnosis; infectious work-up obtained. |
| Day 0 to day 2 (before treatment) | Reactive RPR (1:32) and positive FTA-ABS; nonreactive HIV test; aqueous humor HSV PCR negative; CSF VDRL nonreactive. |
| Day 2 | IV penicillin G 24 million units/day started (14-day course). |
| Day 3 | Oral prednisone 1 mg/kg/day started, tapered according to a standard schedule over a total of 2 weeks. |
| 1-week follow-up | BCVA 20/30 in the left eye; 2+ vitreous cells; no disease progression. |
| Completion of the 14-day course | BCVA 20/20 in the left eye; marked resolution of vitreous cells and retinitis; disappearance of superficial retinal precipitates (Figure 1D). |

**TABLE 1: Timeline of presentation, investigations, treatment, and outcomes**

BCVA: best-corrected visual acuity; CMV: cytomegalovirus; CSF: cerebrospinal fluid; FTA-ABS: fluorescent treponemal antibody absorption; HSV: herpes simplex virus; IV: intravenous; PCR: polymerase chain reaction; RPR: rapid plasma reagin; VDRL: Venereal Disease Research Laboratory

At the one-week follow-up, BCVA improved to 20/30 in the left eye, and the SLE of the left eye showed 2+ vitreous cells but was otherwise unremarkable. The fundus examination of the left eye showed no evidence of disease progression. Upon completion of the 14-day course of penicillin, the patient's BCVA had improved to 20/20 in the left eye, with marked resolution of the vitreous cells and retinitis (Figure *1D*). Treatment was tolerated without adverse effects.

## Discussion

Syphilis remains one of the greatest diagnostic challenges in ophthalmology because of its ability to mimic a broad spectrum of infectious and inflammatory ocular diseases [11]. Its posterior segment manifestations range from acute syphilitic posterior placoid chorioretinitis to punctate inner retinitis and, rarely, to necrotizing retinitis; within this spectrum, the focal necrotizing phenotype may closely resemble viral necrotizing retinitis, particularly CMV retinitis [11,12]. This resemblance may contribute to diagnostic delay, which is associated with poorer visual outcomes [13], underscoring the importance of maintaining a high index of suspicion and performing a comprehensive infectious evaluation in patients presenting with necrotizing retinitis.

In our patient, CMV retinitis was clinically plausible because of the confluent, geographic area of yellow-white retinal whitening consistent with necrotizing retinitis extending toward the periphery. Also, the patient had a border surrounding a central area of relative inactivity, suggesting different stages of lesion progression. Classic CMV retinitis, however, is typically characterized by diffuse, indistinct borders with hemorrhages at the advancing edge, granular satellite lesions, and adjacent areas at different stages of progression within a minimally inflamed eye [2,14]. In contrast, our patient's lesion was well demarcated, lacked intraretinal hemorrhage, had a translucent ground-glass quality, and was studded with discrete, creamy-yellow superficial retinal precipitates along its inferior margin - a finding that has been described in syphilitic retinitis and that, in retrospect, provided an important diagnostic clue [15]. Multimodal imaging reinforced the predominantly outer retinal-RPE involvement of the disease: FAF demonstrated a hyperautofluorescent active border with central granular hypoautofluorescence, and FA showed staining of the active border with central transition to heterogeneous stippled fluorescence and optic disc leakage, a pattern well described in posterior syphilitic retinitis [16-18]. In addition, OCT further supported this predominantly outer retinal-RPE involvement, demonstrating ellipsoid zone and outer retinal disruption with granular RPE irregularity in the involved retina, without preretinal deposits along the scanned section [17,19].

Nevertheless, several findings should prompt early reconsideration of classic CMV retinitis in a similar scenario. Cytomegalovirus retinitis characteristically develops in profoundly immunosuppressed hosts, in whom the limited immune response typically results in minimal anterior chamber and vitreous inflammation [2]. The presence of anterior chamber cells and 2+ vitreous cells in an HIV-negative patient without known immunosuppression, together with optic disc leakage on FA, should therefore raise suspicion for an alternative etiology such as syphilis, in which moderate-to-severe vitritis and optic nerve involvement are common [12,20]. Importantly, no single clinical or imaging finding is diagnostic; rather, it is the

constellation of prominent intraocular inflammation, preserved immune status, and a necrotizing phenotype that should broaden the differential diagnosis. A negative HIV test does not by itself establish immunocompetence, as CMV retinitis has been reported in patients with other forms of immune dysfunction [14,21]; immune status is therefore best appraised clinically rather than inferred from HIV serology alone. In our patient, an HIV test was nonreactive, and there was no history of immunosuppressive or corticosteroid therapy, malignancy, organ transplantation, or cytotoxic chemotherapy, nor of antecedent opportunistic infection or unexplained constitutional illness, together rendering clinically significant cell-mediated immunodeficiency unlikely.

Beyond CMV and syphilis, the differential diagnosis of necrotizing retinitis includes ARN caused by HSV or VZV, progressive outer retinal necrosis, toxoplasmic retinochoroiditis, endogenous endophthalmitis, and masquerade syndromes such as vitreoretinal lymphoma. The key features distinguishing syphilitic necrotizing retinitis, CMV retinitis, and ARN are summarized in Table *2*. In our patient, the confluent posterior distribution of the lesion, without the peripheral onset, rapid circumferential spread, or occlusive arteriolitis characteristic of ARN, argued against a herpetic etiology, a conclusion further supported by the negative aqueous humor HSV PCR, although VZV was not assayed [22]. The absence of a pigmented chorioretinal scar with an adjacent focus of retinochoroiditis, together with nonreactive Toxoplasma gondii serology, made toxoplasmosis less likely [5]. Endogenous endophthalmitis was also less likely in the absence of fever or an identifiable extraocular source of infection [5,15], and vitreoretinal lymphoma was not supported by the necrotizing rather than subretinal infiltrative phenotype or by the rapid and complete response to antimicrobial therapy [23]. Reactive nontreponemal and treponemal serology, in conjunction with the clinical findings, supported the diagnosis of syphilis.

| | Syphilitic necrotizing retinitis | CMV retinitis | Acute retinal necrosis |
|---|---|---|---|
| Clinical presentation | Variable clinical presentation that may mimic other forms of necrotizing retinitis. | Patients may develop decreased vision, floaters, flashes, and scotomas. | Patients may develop ocular or periocular pain, redness, photophobia, floaters, decreased vision, and visual field constriction. |
| Progression | Slowly progressive. | Granular variant progresses slowly, whereas hemorrhagic variant progresses rapidly. | Rapid onset with active retinitis typically lasting 4–6 weeks. |
| Location and distribution | Lesions often begin in the posterior pole; may involve the macula, midperiphery, or peripheral retina. | Usually begins in the peripheral retina and progresses toward the posterior pole, often following retinal vessels; may also begin posteriorly. | Usually begins in the peripheral retina, with the posterior pole initially spared. |
| Retinal appearance | Mottled areas of necrosis, often with an exudative membrane obscuring the underlying retina. | Yellow-white lesions with granular, hemorrhagic, or perivascular patterns. Hemorrhagic lesions may exhibit extensive necrosis. | Multifocal, well-demarcated areas of peripheral retinal whitening and relatively homogeneous necrosis that subsequently become confluent. |
| Complications and outcomes | Lesions may heal without disrupting the underlying retinal pigment epithelium. | Retinal detachment may occur, particularly with extensive retinal involvement. | Retinal detachment may occur. |
| Sources | [24,25] | [3,26] | [27] |

**TABLE 2: Key features of syphilitic necrotizing retinitis, CMV retinitis, and acute retinal necrosis**

CMV: cytomegalovirus

This report has several limitations. First, intraocular fluid testing was limited in scope. Although aqueous humor PCR for HSV was negative, CMV and VZV PCR were not performed, and CMV retinitis was therefore considered unlikely on clinical, serologic, and therapeutic grounds rather than excluded by direct virologic testing. Three converging findings argued against CMV retinitis: the patient's HIV-negative status with no identifiable cause of cell-mediated immunosuppression, which is typically present in CMV retinitis [2,14]; the marked anterior chamber and vitreous inflammatory response, which is atypical of CMV retinitis [2,14]; and the regression of the retinitis with restoration of 20/20 acuity after 14 days of penicillin treatment administered without any antiviral agent. This therapeutic response is consistent with syphilitic necrotizing retinitis, although it does not constitute virologic proof. Coinfection cannot be formally excluded, although the absence of immunosuppression makes CMV coinfection less likely. Second, the findings derive from a single patient with limited follow-up, so the diagnostic value of superficial retinal precipitates cannot be

generalized without comparative data. Comprehensive ocular-fluid PCR testing, including testing for CMV and VZV, remains an important component of the diagnostic evaluation of atypical necrotizing retinitis and may be particularly useful when the diagnosis remains uncertain or the clinical response to therapy is inadequate [21,22].

Management followed the recommended treatment of ocular syphilis with IV penicillin G [9,10]. Current guidelines state that, in patients with isolated ocular symptoms, confirmed ocular abnormalities on examination, and reactive syphilis serology, a CSF examination is unnecessary before treatment [9]. Nonetheless, CSF abnormalities have been reported in approximately 60% to 70% of patients with ocular syphilis, reflecting the frequent coexistence of central nervous system involvement [5,28]. In our patient, a lumbar puncture was performed in accordance with institutional practice, and it yielded a nonreactive CSF VDRL test. Because the CSF VDRL test is highly specific but lacks sensitivity, a nonreactive result does not exclude neurosyphilis and does not alter management, as ocular syphilis is treated with a neurosyphilis regimen irrespective of CSF findings [9,10,29]. Systemic corticosteroids were initiated one day after penicillin to control the intraocular inflammatory response and to mitigate a potential Jarisch-Herxheimer reaction, an acute febrile reaction that can occur within the first 24 hours after the initiation of any syphilis therapy [9] and that may transiently worsen intraocular inflammation [30]. The improvement in visual acuity from 20/100 to 20/20, together with the resolution of the ground-glass retinitis and disappearance of the superficial retinal precipitates after two weeks of penicillin treatment, was consistent with the diagnosis, reinforcing the importance of prompt serologic testing. Syphilis and HIV serologies should be included in the initial evaluation of all patients presenting with necrotizing retinitis, regardless of the leading clinical diagnosis.

## Conclusions

This case demonstrates that syphilitic necrotizing retinitis - a rare manifestation of ocular syphilis - can closely mimic CMV retinitis, presenting a significant diagnostic challenge. In our patient, creamy-yellow superficial retinal precipitates overlying ground-glass retinitis, combined with well-demarcated borders, an absence of intraretinal hemorrhage, and prominent intraocular inflammation in a patient without identifiable immunosuppression, were suggestive clues that should prompt consideration of syphilis. However, these observations derive from a single case and are not diagnostic in isolation. Prompt serologic testing for both syphilis and HIV is essential to enable timely treatment and help preserve vision.

## Additional Information

### Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

**Concept and design:** Maria Garcia, Daniela Busquets, Armando Oliver

**Drafting of the manuscript:** Maria Garcia, Daniela Busquets

**Critical review of the manuscript for important intellectual content:** Maria Garcia, Armando Oliver

### Disclosures

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