



## Acute Retinal Necrosis Complicated by Serous Retinal Detachment Associated with Varicella-Zoster Virus Infection in an Immunocompetent Young Adult

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### Article History

Received: 04.01.2024  
Accepted: 09.02.2024  
Published: 24.03.2024

**Abstract:** Acute retinal necrosis syndrome (ARN syndrome) is a rare, rapidly progressive viral retinitis with a grim functional prognosis. We present a case of a 43-year-old immunocompetent woman who experienced unilateral acute retinal necrosis, with Varicella-Zoster Virus (VZV) infection as the etiology. A unilateral vesicular facial rash accompanied by rapidly declining visual acuity over 7 days prompted consultation. Intravenous antiviral treatment and panretinal photocoagulation laser (PPR) were initiated, followed by antiviral prophylaxis. The patient developed ophthalmic zona lesions, peripheral retinal ischemia, and unilateral macular serous retinal detachment (SRD). Treatment included valacyclovir, corticotherapy and PPR laser. Knowledge of this condition, diagnosed clinically, is crucial due to its rapid progression and severe complications. Early intervention appears to be a significant prognostic factor, emphasizing its therapeutic urgency.

**Keywords:** Acute Retinal Necrosis, Serous Retinal Detachment, Varicella-Zoster Virus, Immunocompetent Young Adult.

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## INTRODUCTION

Acute retinal necrosis syndrome (ARN syndrome) is a severe, rapidly progressing viral retinitis [1]. The implicated viruses belong to the Herpesviridae family, including Varicella-Zoster Virus (VZV), Herpes Simplex Virus (HSV), and occasionally Cytomegalovirus (CMV). We report a case of a 43-year-old immunocompetent woman with unilateral acute retinal necrosis attributed to VZV infection, complicated by serous retinal detachment, and treated with antivirals, corticotherapy and PPR laser.

## CASE PRESENTATION

A 43-year-old woman with no significant medical history presented to ophthalmic emergency services with pain and declining vision in the right eye over 7 days, along with progressively worsening facial periocular lesions indicative of ophthalmic zona. Corrected visual acuity was counting fingers at 2 meters in the right eye and 10/10 in the left eye. The examination of the right eye revealed conjunctival hyperemia, superficial punctate keratitis (KPS), minimal Tyndall effect, minimal vitreous Tyndall effect, significant occlusive vasculitis, serous retinal detachment, exudates in the posterior pole and temporal quadrant, and avascular vessels (Figure 1). The left eye examination was normal (Figure 2).

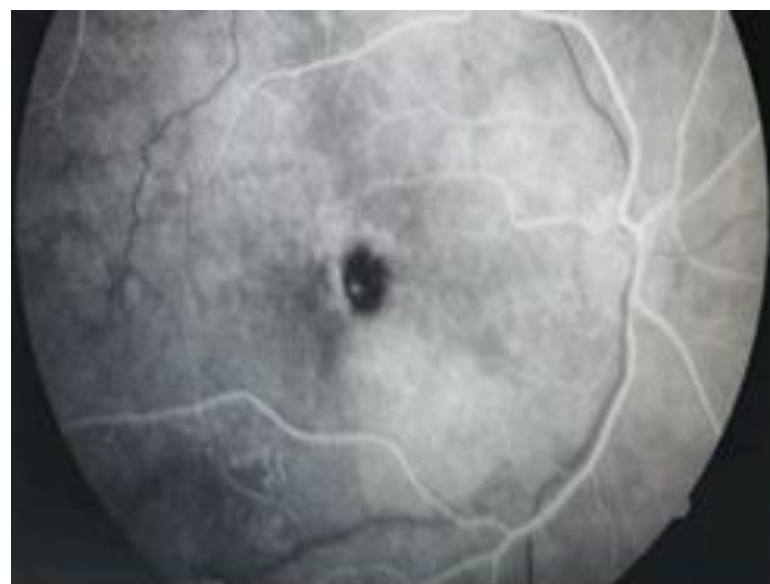
**Citation:** D. Batungwanayo, A. Najah, S. Nidal, B. Salaheddin, K. Majid, S. Belghmaidi, I. Hajji, A. Moutaouakil (2024). Acute Retinal Necrosis Complicated by Serous Retinal Detachment Associated with Varicella-Zoster Virus Infection in an Immunocompetent Young Adult. *Glob Acad J Med Sci*; Vol-6, Iss-2 pp- 53-57.



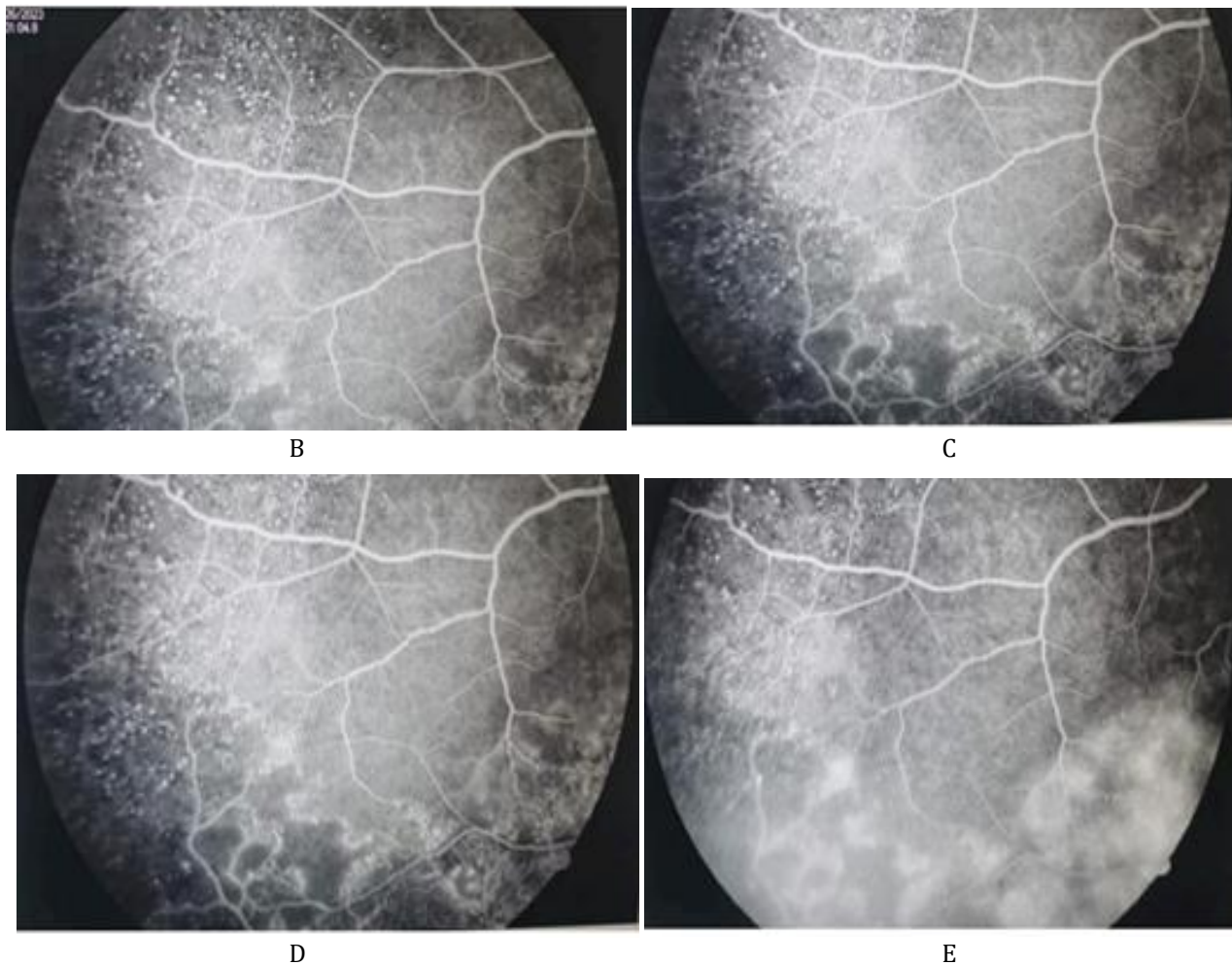
**Figure 1: Significant occlusive vasculitis, serous retinal detachment exudates in the posterior pole and temporal quadrant, and avascular vessels**



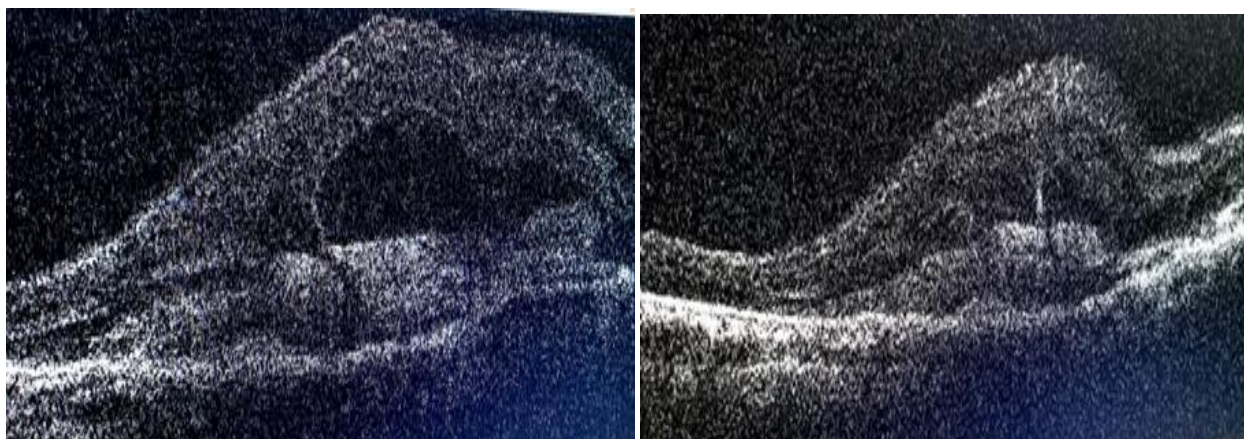
**Figure 2: Normal left eye**



A



**Figure 3: Fluorescein retinal angiography of the right eye demonstrated delayed arterial filling (A), marked absence of peripheral retinal perfusion (B, C) and necrosis retinal area (D, E)**

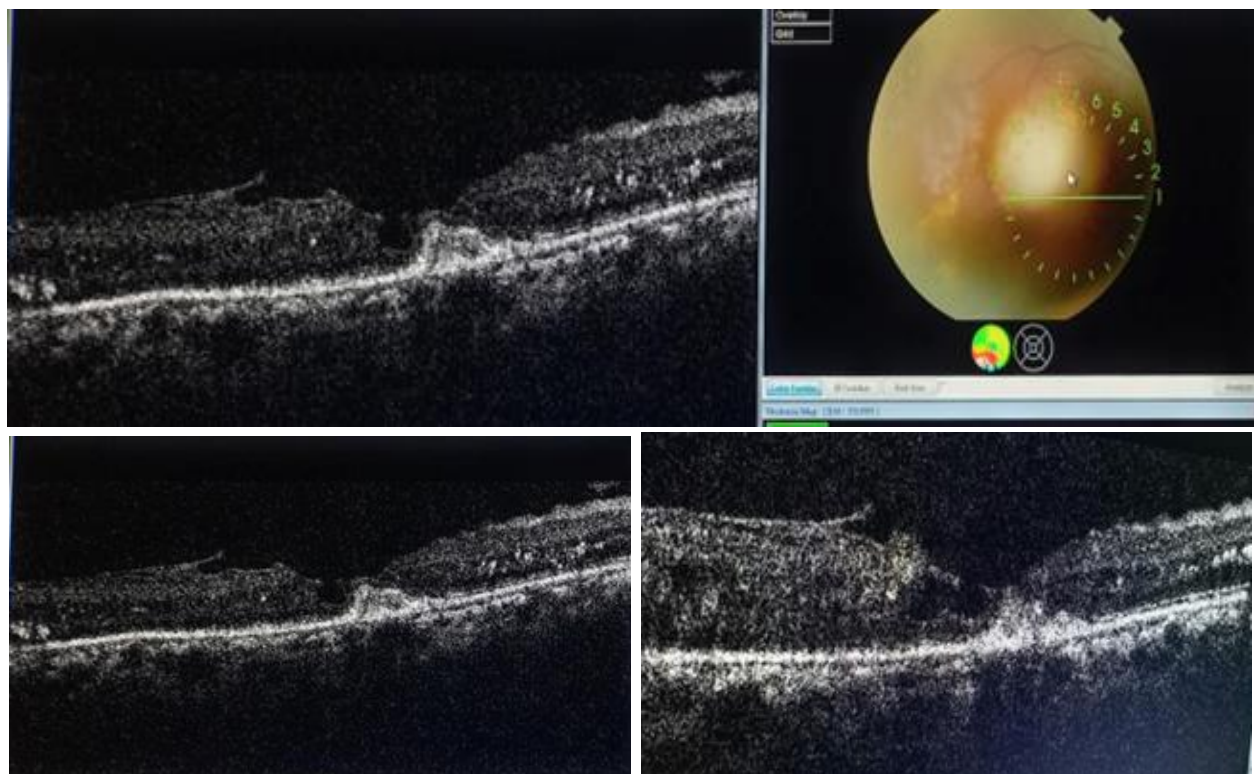


**Figure 4: Macular optical coherence tomography (OCT) revealed DSR in the right eye**

Given the unilateral involvement and unknown immune status, treatment involved valacyclovir, corticotherapy and PPR laser. Serosurveys were negative except for VZV, which tested positive. Three corticosteroid boluses were

administered, followed by oral tapering. The patient showed favorable progress, with healing of necrotic areas observed. At 4 months, there was a noticeable reduction in retinal thickness and SRD regression (Figure 5).





**Figure 5: Reduction in retinal thickness and SRD regression**

## DISCUSSION

Clinical examination serves as the gold standard for diagnosing acute retinal necrosis syndrome. Reduced visual acuity, conjunctival hyperemia, and myodesopsias were the presenting complaints in our patient. Examination of the anterior segment revealed conjunctival hyperemia and minimal Tyndall effect. The posterior segment exhibited minimal vitreous Tyndall effect, complicated by peripheral retinal necrosis with centripetal extension, exudates in the posterior pole extending to the temporal quadrant, avascular vessels, and serous retinal detachment. Similar symptoms were noted by Essakhi *et al.*, [2], although N. Cassoux and P. Gastaud [3] observed this symptomatology with cystoid macular edema.

Acute retinal necrosis syndrome is associated with severe, rapidly progressing complications requiring urgent intervention. In Morel *et al.*'s series [4], 4 patients experienced total retinal detachment, one became monophthalmic due to uveitis, and another had bilateral involvement. Our patient presented with serous retinal detachment, possibly due to her prompt consultation (7 days).

Upon admission, our patient's visual acuity (VA) was severely reduced to counting fingers at 2 meters. Similar findings of reduced VA were reported by other authors [3, 4]. VA is a crucial parameter in evaluating the prognosis of acute retinal necrosis syndrome. Areas of retinal necrosis were

documented through fluorescein retinal angiography. Macular optical coherence tomography (OCT) provides necessary insight into retinal and macular structure, guiding treatment decisions and patient monitoring. In our case, serology revealed VZV as the causative agent. Lau *et al.*, [5] found VZV coinfection in three EBV-positive patients, suggesting a potential pathogenic role for VZV. VZV is frequently identified and appears more aggressive (with necrosis extension) than other viruses, contributing to acyclovir resistance [1, 5, 6]. The at-risk population includes individuals with a history of viral infections [2, 6, 8], such as chickenpox (70.6%), shingles (29.2%), ophthalmic shingles (20.7%), pseudogripal syndrome due to HSV (25%), and HSV encephalitis (15.4%). However, our patient had no specific antecedents.

Recommended treatment involves intravenous acyclovir at a dose of 10 mg/kg/8 hours for 3 weeks until lesion healing. A switch to oral valacyclovir (Zelitrex®) at 3 g/day with a tapering dose is initiated. The duration is not standardized but is suggested to be at least 4 months, the period of maximum recurrence risk [6]. In cases of acyclovir resistance, foscarnet (Foscavir®) can be used. Intravitreal injection of 2 mg ganciclovir (Cymévan®) is also an option for monophthalmic or immunocompromised patients.

Our patient's treatment included decreasing doses of valacyclovir (Zelitrex®), corticosteroid

bolus therapy in three doses of methylprednisolone with oral continuation and PPR laser on the necrotic retinal area to prevent complications like neovascularization and retinal detachment. Lau *et al*, report a detachment rate after laser treatment of 35.3% versus 80% without laser [5]. Our patient's progression showed improved VA and a remarkable reduction in SRD. Early diagnosis and treatment are pivotal factors in this favorable outcome.

## CONCLUSION

The functional prognosis of acute retinal necrosis syndrome remains guarded. However, early management through the administration of antivirals and corticosteroids, coupled with PPR laser therapy, appears to mitigate necrosis and improve the prognosis.

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