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ACUTE LOSS OF VISION FOLLOWING DENGUE FEVER: AN UNUSUAL CASE OF MACULAR NEURORETINOPATHY. A SYSTEMATIC REVIEW

Misbah Naeem[1], Adila Anwar[2]

[1]Postgraduate Trainee, KRL Hospital, Islamabad, Pakistan.

[2]Senior Registrar, Federal Government Polyclinic Hospital, Islamabad, Pakistan.

Corresponding author: Misbah Naeem

ABSTRACT

Purpose: *Dengue fever (DF), caused by the dengue virus (DENV), is a rapidly expanding global health threat. While systemic features dominate its presentation, ocular manifestations are increasingly recognized, occurring in up to 10% of hospitalized patients. Among these, Acute Macular Neuroretinopathy (AMN) represents a rare, sight-threatening presentation characterized by acute-onset central or paracentral scotomas and distinct outer retinal changes. This review synthesizes the current literature on DENV-associated AMN, focusing on its immune-mediated microvascular pathophysiology, characteristic multimodal imaging findings, and therapeutic strategies.*

Methods*: A comprehensive literature search was conducted across major electronic databases (PubMed, Google Scholar, and Scopus) using keywords including "Dengue", "Dengue Maculopathy", "Acute Macular Neuroretinopathy", "Spectral-Domain Optical Coherence Tomography (SD-OCT)", and "Deep Capillary Plexus". Peer-reviewed case reports, retrospective cohort studies, and systematic reviews were analyzed.*

Results*: DENV-associated AMN primarily affects young to middle-aged adults, typically presenting 7 to 10 days after the onset of fever, closely mirroring the nadir of thrombocytopenia and the rise of the secondary immune response. The pathogenesis is driven by a combination of viral-induced endothelial activation (via non-structural protein 1, NS1), immune-complex deposition, and subsequent microvascular ischemia of the deep capillary plexus (DCP). Spectral-domain optical coherence tomography (SD-OCT) classically reveals hyperreflectivity at the level of the outer plexiform layer (OPL) and outer nuclear layer (ONL), followed by long-term disruption of the ellipsoid zone (EZ) and interdigitation zone (IZ). Optical coherence tomography angiography (OCTA) confirms a corresponding flow deficit in the DCP. While conservative management is suitable for mild cases, severe or progressive visual loss frequently warrants systemic, local, or intravitreal corticosteroid therapy.*

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Conclusions*: DENV-associated AMN is an underdiagnosed, ischemic outer-retinopathy driven by post-viral hyperinflammation and microvascular impairment. Clinicians must maintain a high index of suspicion in dengue-endemic regions or in returning travelers presenting with acute visual symptoms. Early multimodal imaging is essential for diagnosis and prognosis.*

INTRODUCTION

Dengue fever (DF) is a major vector-borne viral illness caused by the dengue virus (DENV, serotypes 1–4), an enveloped, single-stranded RNA virus belonging to the *Flaviviridae* family. Transmitted primarily by the day-biting *Aedes aegypti* and *Aedes albopictus* mosquitoes, dengue represents an escalating global public health challenge. Driven by urbanization, climate change, and global travel, the geographic footprint of the virus has expanded dramatically. Millions of infections occur annually across tropical and subtropical regions of Southeast Asia, the Western Pacific, the Americas, and Africa.[1-3]

The clinical spectrum of DENV infection is remarkably diverse, ranging from an asymptomatic state or mild, self-limiting febrile illness (classic dengue fever) to life-threatening complications such as dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS), characterized by severe plasma leakage, thrombocytopenia, and multi-organ dysfunction.[4]

Historically, ocular manifestations of dengue were considered rare. However, with the integration of advanced ophthalmic imaging in clinical settings, the reported incidence of ocular involvement among hospitalized dengue patients ranges from 7.9% to 40.3% in endemic zones. Ocular complications can affect both the anterior and posterior segments. Common presentations include subconjunctival hemorrhages, anterior uveitis, vitreous haze, retinal vasculitis, optic neuritis, and various phenotypes of dengue maculopathy—such as diffuse macular edema, cystoid macular edema, and foveolitis.[5-7]

Acute Macular Neuroretinopathy (AMN) has emerged as a distinct, rare, and visually debilitating manifestation of dengue maculopathy. Originally described in 1975 by Bos and Deutman in association with oral contraceptive use, AMN is characterized clinically by the acute onset of paracentral or central scotomas and reddish-brown, wedge-shaped macular lesions. Over the past decade, a growing body of literature has linked AMN to DENV infections, particularly during the convalescent or post-febrile phase of the disease.[8-10]

This review provides a comprehensive synthesis of DENV-associated AMN. We explore the complex interplay of viral and immunological mechanisms driving its pathogenesis, delineate the clinical and multimodal imaging features necessary for its diagnosis, and evaluate current therapeutic strategies to optimize visual outcomes.

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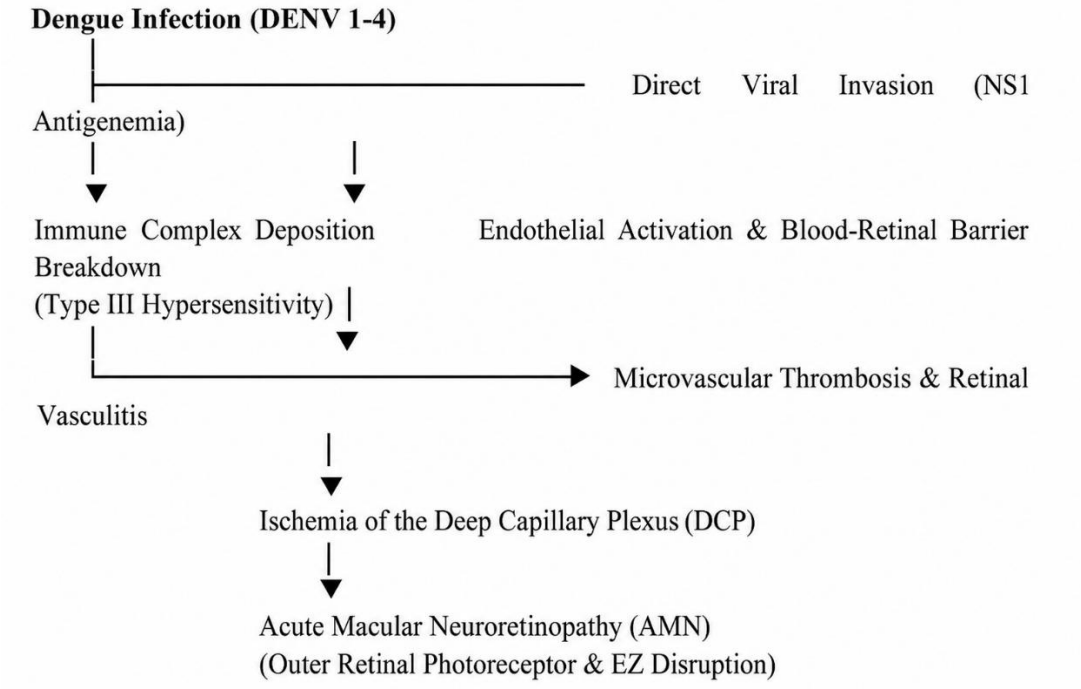
PATHOPHYSIOLOGY

The precise pathogenesis of DENV-associated ocular disease remains a subject of active investigation, but current evidence points toward a multi-factorial process involving direct viral cytopathic damage, immune-mediated vascular inflammation, and coagulopathy. In the context of AMN, the primary insult is localized to the microvasculature of the outer retina—specifically the deep capillary plexus (DCP).



ROLE OF THE VIRAL NS1 PROTEIN AND ENDOTHELIAL DYSFUNCTION

The DENV non-structural protein 1 (NS1) is a glycoprotein secreted in high concentrations into the circulation during the acute phase of infection. NS1 acts as a powerful driver of endothelial activation and vascular permeability. It triggers the p38 mitogen-activated protein kinase (p38-MAPK) pathway, leading to the upregulation and shedding of tight-junction proteins, matrix metalloproteinases (such as MMP-2), and adhesion molecules (such as ICAM-1) on the vascular endothelium. This process directly compromises the integrity of the inner and outer blood-retinal barriers (BRB), allowing systemic inflammatory cytokines, immunoglobulins, and complement proteins to infiltrate the retinal parenchyma.

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IMMUNE-MEDIATED VASCULITIS AND THROMBOCYTOPENIA

The temporal presentation of AMN—which typically occurs 7 to 10 days after the onset of fever, coinciding with the clearance of viremia and the rise of IgG/IgM antibodies—strongly supports an immune-mediated mechanism rather than direct viral replication. This delay aligns with a Type III hypersensitivity reaction. Circulating antigen-antibody complexes (DENV-IgG/IgM) deposit along the endothelial walls of the retinal microvasculature, activating the classical complement pathway.

The resulting inflammatory response leads to:

1. Local perivasculitis and capillaritis: Restricting microvascular blood flow.

2. Thrombocytopenia and coagulopathy: Transiently predisposing the patient to microthrombi formation within the high-resistance capillary networks.

VULNERABILITY OF THE DEEP CAPILLARY PLEXUS (DCP)

The retinal vasculature is organized into three distinct layers: the superficial capillary plexus (SCP), the intermediate capillary plexus (ICP), and the deep capillary plexus (DCP). The DCP, situated at the outer border of the inner nuclear layer (INL) adjacent to the outer plexiform layer (OPL), consists of thin, highly anastomotic capillary channels. Unlike the SCP, which is directly connected to larger feeding arterioles, the DCP has a lower perfusion pressure and higher resistance, making it uniquely vulnerable to:

Hypotension: Associated with dengue-related plasma leakage or shock.

Thrombotic or inflammatory occlusion: Secondary to immune complex-induced endothelial swelling.

Ischemia of the DCP deprives the watershed zone of the outer retina—comprising the OPL, outer nuclear layer (ONL), and the highly metabolically active photoreceptor inner/outer segment junctions—of necessary oxygen and nutrients. This acute ischemic insult manifests structurally and functionally as AMN.

CLINICAL PRESENTATION

DENV-associated AMN primarily affects young to middle-aged adults, showing no significant gender predisposition, though individual series have highlighted varied cohorts.

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SYMPTOMATOLOGY

Patients classically present with a sudden, painless decrease in visual acuity or, more characteristically, the acute onset of persistent central or paracentral scotomas in one or both eyes. Symptoms typically emerge during the convalescent phase of the systemic illness, approximately 7 to 14 days after the initial onset of febrile symptoms (such as high-grade fever, severe headache, retro-orbital pain, myalgia, and arthralgia).

Other reported visual disturbances include:

Metamorphopsia (distorted vision)

Micropsia (objects appearing smaller)

Decreased color sensitivity

Mild photophobia

CLINICAL FINDINGS

Standard anterior segment slit-lamp examination is typically unremarkable, showing no signs of active inflammation (such as anterior chamber cells or flare), although mild vitritis or vitreous haze may occasionally be noted in cases with concurrent posterior uveitis.

On funduscopic evaluation, classic AMN presents with:

One or more well-circumscribed, faint, grayish-white or reddish-brown wedge-shaped lesions in the parafoveal region, with the apices of the wedges pointing directly toward the foveola.

Accompanying features of dengue maculopathy, which may co-exist, such as flame-shaped retinal hemorrhages, soft exudates (cotton-wool spots), and mild macular edema.

Notably, because these outer retinal lesions can be highly subtle and faint on standard white-light ophthalmoscopy, fundus examination alone may miss the diagnosis, making advanced multimodal imaging critical.

MULTIMODAL OCULAR IMAGING

The integration of advanced ophthalmic imaging modalities has revolutionized the detection, characterization, and longitudinal tracking of DENV-associated AMN.

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NEAR-INFRARED (NIR) REFLECTANCE IMAGING

Near-infrared reflectance is highly sensitive for identifying AMN. On NIR, the lesions appear as distinct, hyporeflective, wedge-shaped or teardrop-shaped areas in the parafoveal region. NIR provides a clearer contrast of the lesions against the normal retinal background than conventional color fundus photography, making it an excellent screening tool.

SPECTRAL-DOMAIN OPTICAL COHERENCE TOMOGRAPHY (SD-OCT)

SD-OCT is the gold standard for diagnosing AMN, providing high-resolution cross-sectional visualization of the individual retinal layers. The structural changes observed on SD-OCT evolve through distinct stages:

As demonstrated in Figure 1, optical coherence tomography is crucial for identifying the precise depth of involvement:

Acute Phase: Characterized by localized hyperreflectivity at the level of the outer plexiform layer (OPL) and outer nuclear layer (ONL). This hyperreflectivity reflects acute intracellular edema and mitochondrial swelling of the photoreceptor axons (Henle's fiber layer) and cell bodies, secondary to hypoxia.

Subacute to Chronic Phase: Over the following weeks, the hyperreflectivity subsides, giving way to thinning of the outer nuclear layer and structural disruption of the ellipsoid zone (EZ) (historically referred to as the photoreceptor inner segment/outer segment junction) and the interdigitation zone (IZ). This disruption corresponds to the permanent loss of photoreceptor outer segments, which correlates closely with the persistence of central scotomas.

OPTICAL COHERENCE TOMOGRAPHY ANGIOGRAPHY (OCTA)

OCTA is a non-invasive dye-free imaging technique that allows for depth-resolved visualization of the retinal microvasculature. In eyes with DENV-associated AMN:

SCP: Typically remains intact or shows minimal, focal capillary dropouts.

DCP: Consistently demonstrates severe, localized flow deficits and capillary non-perfusion corresponding directly to the areas of outer retinal disruption seen on SD-OCT and the hyporeflective lesions on NIR.

Foveal Avascular Zone (FAZ): Frequently appears enlarged or irregular due to the loss of surrounding deep capillaries.

FUNDUS FLUORESCEIN ANGIOGRAPHY (FFA) AND INDOCYANINE GREEN ANGIOGRAPHY (ICGA)

Unlike OCTA, traditional FFA is often normal in isolated AMN because the large superficial vessels imaged by FFA mask the deep capillary plexus. However, in DENV-associated cases, FFA is useful to rule out or characterize co-existing pathologies, showing late optic disc staining, retinal vasculitis (with perivascular dye leakage), or areas of macular capillary non-perfusion in cases of severe macular infarction. ICGA may occasionally reveal hypofluorescent spots in the early and late phases, suggesting secondary hypoperfusion of the choriocapillaris.

DIFFERENTIAL DIAGNOSIS

When evaluating a patient with acute visual loss or scotoma following a febrile illness in a tropical environment, several clinical entities must be differentiated from DENV-associated AMN:

MANAGEMENT AND CLINICAL COURSE

Currently, there are no standardized, universally accepted guidelines for the management of DENV-associated AMN. Treatment strategies are largely individualized based on the severity of visual impairment, the degree of structural macular damage, and the presence of co-existing systemic or ocular complications.

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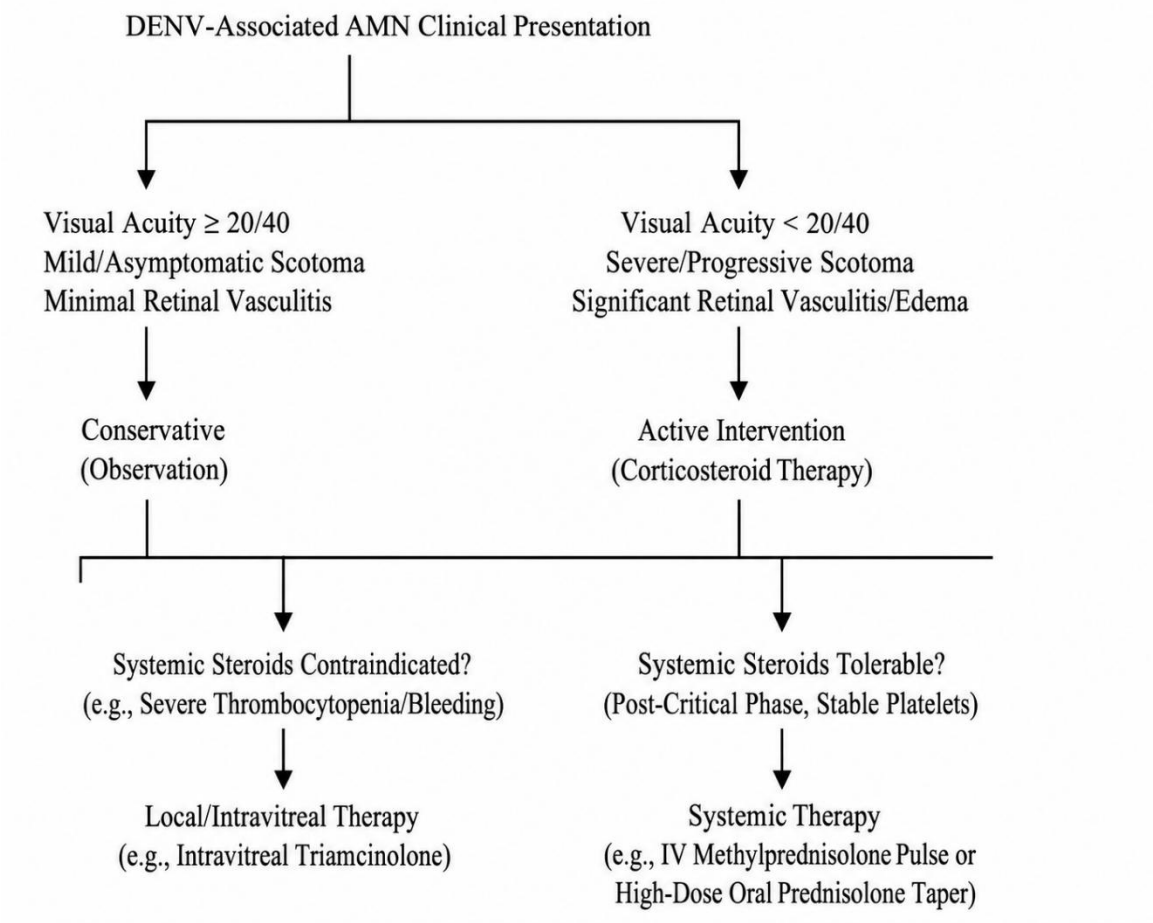

CONSERVATIVE MANAGEMENT (OBSERVATION)

A significant proportion of patients with mild DENV-associated AMN and preserved baseline visual acuity (20/40) undergo spontaneous resolution without medical intervention. Active surveillance with serial SD-OCT and visual acuity monitoring is highly appropriate for these patients. Visual symptoms often improve gradually over weeks to months, although mild paracentral scotomas may persist long-term due to residual EZ disruption.

CORTICOSTEROID THERAPY

In cases presenting with severe visual loss (< 20/40), rapidly progressive scotomas, or significant co-existing inflammatory changes—such as retinal vasculitis, optic neuritis, or diffuse macular edema—anti-inflammatory therapy is indicated to limit structural outer retinal damage and prevent permanent photoreceptor loss.

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SYSTEMIC CORTICOSTEROIDS

Intravenous Methylprednisolone: For rapidly deteriorating or bilateral, severe sight-threatening cases, a pulse dose of intravenous methylprednisolone for 3 consecutive days) is often administered, followed by an oral prednisolone taper.

Oral Prednisolone: Alternatively, initiating oral prednisolone at typically starting at 40 mg/day with a gradual, structured taper over 4 to 8 weeks is widely utilized.

Systemic Safety Warning: Systemic corticosteroids must be introduced with extreme caution during the acute or critical phases of dengue fever. Initiating high-dose steroids in a patient with severe thrombocytopenia, active plasma leakage, or hemorrhagic manifestations can exacerbate bleeding diathesis and suppress vital immune clearance of the virus. Close coordination with the treating physician or infectious disease specialist is mandatory. Platelet counts must be stabilized before systemic steroid initiation.

LOCAL AND INTRAVITREAL CORTICOSTEROIDS

To bypass the systemic risks of high-dose corticosteroids, local ocular delivery has emerged as a highly effective, targeted alternative.

Intravitreal Triamcinolone Acetonide (IVTA): Injection of 4 mg/0.1 Ml of triamcinolone acetonide can deliver therapeutic levels of anti-inflammatory medication directly to the posterior segment. Studies have shown rapid resolution of macular edema and restoration of foveal architecture following IVTA, with minimal systemic side effects.

Intravitreal Dexamethasone Implant: Provides sustained drug release over several months and is particularly useful in chronic or refractory cases.

VISUAL PROGNOSIS

The visual prognosis for patients with DENV-associated AMN is variable. While many patients achieve a significant recovery of their best-corrected visual acuity (BCVA) with or without treatment, complete functional recovery of the visual field is less common. Persistent, localized paracentral scotomas frequently remain as permanent sequelae. This functional deficit correlates structurally with the irreversible loss of the ellipsoid zone (EZ) and thinning of the outer nuclear layer (ONL) on follow-up SD-OCT.

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DISCUSSION

The clinical entity of DENV-associated AMN represents a fascinating and critical intersection between virology, immunology, and microvascular ophthalmology. As the global incidence of dengue fever continues to rise year-over-year, recognizing and understanding this specific ocular complication is of paramount importance for ophthalmologists, infectious disease specialists, and primary care physicians alike.[11-13]

Historically, AMN was considered an idiopathic, highly rare condition predominantly affecting healthy young females on oral contraceptives. However, the expanding literature on post-viral AMN—triggered not only by DENV, but also by influenza, COVID-19, and other systemic infections—has reshaped our understanding of its etiology. It is now widely accepted that AMN is a clinical phenotype of deep retinal capillary ischemia, and any systemic state that induces microvascular inflammation, hypercoagulability, or severe endothelial dysfunction can serve as a trigger.[14]

In the context of dengue, the timing of onset of AMN is a crucial clinical clue. Occurring typically in the post-febrile, convalescent phase (days 7–14), the presentation aligns perfectly with the window of maximal immune complex deposition and immune-mediated vasculitis. During this phase, direct viral replication has largely subsided, and the patient's symptoms are driven by the host's robust immunological response. This pathophysiology explains why patients frequently present with ocular complaints after their systemic symptoms (fever, myalgia) have begun to resolve.[15-17]

The primary diagnostic challenge of DENV-associated AMN is its subtle presentation on standard fundus ophthalmoscopy. Grayish-white parafoveal lesions can easily be missed during routine examinations, leading to misdiagnoses of "malingering" or functional visual loss. The absolute necessity of utilizing multimodal imaging—specifically NIR and SD-OCT—cannot be overstated. NIR provides an immediate, highly-contrasted blueprint of the ischemic regions, while SD-OCT localizes the damage to the outer retinal layers (OPL, ONL, EZ, and IZ) with micron-level precision. Furthermore, the introduction of OCTA has provided the final, definitive proof of the underlying ischemic mechanism, demonstrating a direct, co-localized flow deficit within the deep capillary plexus (DCP).[17-20]

The management of DENV-associated AMN remains a clinical tightrope. The decision of whether to observe or treat must balance the potential for spontaneous visual recovery against the risk of permanent photoreceptor loss from prolonged ischemia. If treatment is deemed necessary, corticosteroids are the mainstay of therapy due to their potent anti-inflammatory and immunomodulatory properties. However, systemic administration during the acute or early recovery phase of dengue is fraught with risk, particularly concerning the worsening of thrombocytopenia or bleeding.

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To mitigate these systemic risks, the use of intravitreal corticosteroid therapy (such as triamcinolone acetonide or dexamethasone implants) represents a major therapeutic advancement. By delivering high concentrations of anti-inflammatory agents directly to the vitreous cavity and macula, clinicians can achieve rapid control of local inflammation and macular edema without compromising systemic safety or platelet recovery.

Ultimately, the visual prognosis in DENV-associated AMN is dictated by the structural integrity of the outer retinal layers. Disruption of the ellipsoid and interdigitation zones on SD-OCT serves as a reliable surrogate marker for permanent photoreceptor damage and long-term scotomas. This highlights the critical importance of early diagnosis and, where appropriate, timely intervention to suppress microvascular inflammation before irreversible outer retinal atrophy occurs.

CONCLUSIONS AND FUTURE PERSPECTIVES

DENV-associated Acute Macular Neuroretinopathy (AMN) is a distinct, ischemic outer-retinopathy driven by post-viral hyperinflammation, immune-complex mediated vasculitis, and deep capillary plexus hypoperfusion. As the global burden of dengue continues to expand, it is vital for clinicians to recognize this clinical entity.

A thorough ophthalmic examination, complemented by near-infrared imaging, spectral-domain OCT, and OCT angiography, is mandatory for any patient presenting with acute visual disturbances following a dengue infection.

Future research should focus on:

1. Prospective clinical trials to establish standardized management protocols, particularly evaluating the efficacy and safety of early local (intravitreal) corticosteroid therapy versus conservative observation.

2. Identifying specific genetic or viral biomarkers (e.g., DENV serotypes such as DENV-1) that may predispose certain individuals to severe ocular complications.

3. Determining the long-term functional impact of persistent outer retinal scotomas on patient quality of life.

REFERENCES

1. *Li M, Zhang X, Ji Y, Ye B, Wen F. Acute macular neuroretinopathy in dengue fever: short-term prospectively followed up case series. JAMA Ophthalmol. 2015;133(11):1329–1333. doi:10.1001/jamaophthalmol.2015.2687.*

2. *Aggarwal K, Agarwal A, Katoch D, Sharma M, Gupta V. Optical coherence tomography angiography features of acute macular neuroretinopathy in dengue fever. Indian J Ophthalmol. 2017;65(11):1235–1238. doi:10.4103/ijo.IJO_969_18.*

3. *Bacsal KE, Chee SP, Cheng CL, Flores JV. Dengue-associated maculopathy. Arch Ophthalmol. 2007;125(4):501–510. doi:10.1001/archopht.125.4.501.*

4. *Teoh SC, Chee CK, Laude A, et al. Optical coherence tomography patterns as predictors of visual outcome in dengue-related maculopathy. Retina. 2010;30(3):390–398. doi:10.1097/IAE.0b013e3181bd2fc6.*

5. *Tavassoli S, Carreño E, Teoh SC, et al. Optical coherence tomography angiography findings in dengue-related maculopathy: A Case report. Ophthalmic Surg Lasers Imaging Retina. 2016;47(11):1057–1060. doi:10.3928/23258160-20161031-12.*

6. *Fang PP, Pfau M, Holz FG, Finger RP. Persistent visual loss in dengue fever due to outer retinal damage. Clin Exp Ophthalmol. 2017;45(7):747–749. doi:10.1111/ceo.12960.*

7. *Agarwal A, Aggarwal K, Dogra M, et al. Dengue-induced inflammatory, ischemic foveolitis and outer maculopathy: a swept-source imaging evaluation. Ophthalmol Retina. 2019;3(2):170–177. doi:10.1016/j.oret.2018.09.008.*

8. *Su DH, Bacsal K, Chee SP, et al. Prevalence of dengue maculopathy in patients hospitalized for dengue fever. Ophthalmology. 2007;114(9):1743–1747. doi:10.1016/j.ophtha.2007.03.054.*

9. *Rajurkar K, Thakar M. Optical coherence tomography angiography features of dengue retinopathy manifesting as acute macular neuroretinopathy, branch vein vasculitis and neurosensory detachment. Oman J Ophthalmol. 2021;14(3):179–182. doi:10.4103/ojo.ojo_210_21.*

10. *Ooi KG, Inglis H, Paramanathan N, Downie JA, Hennessy MP. Dengue fever-associated maculopathy and panuveitis in Australia. Case Rep Ophthalmol Med. 2016;2016:5704695. doi:10.1155/2016/5704695.*

11. *Bos PJ, Deutman AF. Acute macular neuroretinopathy. Am J Ophthalmol. 1975;80(4):573–584. doi:10.1016/0002-9394(75)90387-6.*

12. *Bhavsar KV, Lin S, Rahimy E, et al. Acute macular neuroretinopathy: a comprehensive review of the literature. Surv Ophthalmol. 2016;61(5):538–565. doi:10.1016/j.survophthal.2016.03.003.*

13. *Munk MR, Jampol LM, Cunha Souza E, de Andrade GC, Esmaili DD, Sarraf D, Fawzi AA. New associations of classic acute macular neuroretinopathy. Br J Ophthalmol. 2016;100(3):389–394. doi:10.1136/bjophthalmol-2015-306845.*

14. *Fawzi AA, Pappuru RR, Sarraf D, et al. Acute macular neuroretinopathy: long-term insights revealed by multimodal imaging. Retina. 2012;32(8):1500–1513. doi:10.1097/iae.0b013e318263d0c3.*

15. *Lee SY, Cheng JL, Gehrs KM, et al. Choroidal features of acute macular neuroretinopathy via optical coherence tomography angiography and correlation with serial multimodal imaging. JAMA Ophthalmol. 2017;135(11):1177–1183. doi:10.1001/jamaophthalmol.2017.3790.*

16. *Thanos A, Faia LJ, Yonekawa Y, Randhawa S. Optical coherence tomographic angiography in acute macular neuroretinopathy. JAMA Ophthalmol. 2016;134(11):1310–1314. doi:10.1001/jamaophthalmol.2016.3513.*

17. *Lim WK, Mathur R, Koh A, Yeoh R, Chee SP. Ocular manifestations of dengue fever. Ophthalmology. 2004;111(11):2057–2064. doi:10.1016/j.ophtha.2004.03.038.*

18. *Yip VC-H, Sanjay S, Koh YT. Ophthalmic complications of dengue fever: a systematic review. Ophthalmol Ther. 2012;1(1):2. doi:10.1007/s40123-012-0002-z.*

19. *Ng AW, Teoh SC. Dengue eye disease. Surv Ophthalmol. 2015;60(2):106–114. doi:10.1016/j.survophthal.2014.07.003.*

20. Gupta A, Srinivasan R, Setia S, Soundravally R, Pandian DG. Uveitis following dengue fever. Eye (Lond). 2009;23(4):873–876. doi:10.1038/eye.2008.124.