



Retinal Involvement in Biopsy-Proven Dense Deposit Disease: A Single-Centre Retrospective Observational Study

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Abstract

Background/Aim: Dense deposit disease (DDD), formerly classified as membranoproliferative glomerulonephritis (MPGN) type II, is a rare renal disorder caused by dysregulation in the activation of the complement system by an alternative pathway associated with retinal abnormalities. However, the frequency and spectrum of ocular involvement remain poorly defined because published evidence is limited to case reports and small case series. This study aimed to determine the frequency of retinal involvement among patients with biopsy-confirmed DDD identified within a consecutive MPGN cohort and to describe their clinical and multimodal retinal imaging findings.

Methods: This retrospective observational study included all consecutive patients who underwent native kidney biopsy at the University Clinical Centre of the Republic of Srpska, Bosnia and Herzegovina, between 1 January 2021 and 31 March 2026. Patients with biopsy-confirmed MPGN underwent comprehensive ophthalmological examination, including multimodal retinal imaging. Clinical, histopathological and ophthalmological findings were analysed descriptively.

Results: Among 110 native kidney biopsies, seven patients had MPGN, including three with biopsy-confirmed DDD. Retinal abnormalities were identified in two patients with DDD, whereas no ocular abnormalities were detected in other MPGN subtypes. One patient presented with acute exudative–ischaemic retinopathy showing partial anatomical and functional recovery, while the other exhibited the typical chronic drusenoid phenotype with drusen-like deposits, retinal pigment epithelium abnormalities and pigment epithelial detachments. Multimodal retinal imaging enabled detailed characterisation of retinal changes and choroidal neovascularisation was not detected.

Conclusions: In this cohort, retinal involvement occurred exclusively in patients with biopsy-confirmed DDD and ranged from acute exudative–ischaemic retinopathy to the characteristic chronic drusenoid phenotype. These findings support comprehensive ophthalmological evaluation with multimodal retinal imaging in patients with DDD. Larger prospective multi-centre studies are needed to better define the prevalence, natural history and clinical significance of ocular involvement.

Key words: Dense deposit disease; Glomerulonephritis, membranoproliferative; Kidney; Tomography, optical coherence; Multimodal imaging.

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Introduction

Dense deposit disease (DDD), formerly designated membranoproliferative glomerulonephritis (MPGN) type II, is an uncommon complement-mediated kidney disorder currently classified within the spectrum of C3 glomerulopathies (C3G).¹⁻³ The estimated prevalence is approximately 2–3 cases per million population and nearly half of affected individuals progress to kidney failure within 10 years of diagnosis.^{4,5} Persistent dysregulation of the alternative complement pathway is now recognised as the principal pathogenic mechanism, resulting in uncontrolled complement activation and progressive tissue injury.⁴ Among C3 glomerulopathies, DDD represents the ultrastructurally defined subtype characterised by highly electron-dense transformation of the glomerular basement membrane.⁶ Although renal disease is the predominant clinical manifestation, complement-mediated damage is not restricted to the kidneys and may also involve other basement membrane-rich tissues, particularly Bruch's membrane within the eye.^{1,6}

The susceptibility of the retina to complement-mediated injury is supported by the close structural and functional relationship between Bruch's membrane and the glomerular basement membrane. Histopathological studies have demonstrated electron-dense deposits within Bruch's membrane, indicating that retinal abnormalities are an integral component of the systemic disease rather than an incidental ocular finding.⁷ Clinical investigations subsequently identified bilateral drusen-like lesions, characteristic fluorescein angiographic abnormalities and increased vitreous fluorescein penetration, further supporting retinal involvement in DDD.^{8,9}

Ocular manifestations associated with DDD encompass a broad spectrum of structural abnormalities, including drusen-like deposits, retinal pigment epithelium alterations, pigmentary changes, pigment epithelial detachments, outer retinal atrophy, subretinal fluid and choroidal neovascularisation.¹⁰⁻¹³ Functional impairment may develop despite relatively preserved visual acuity, suggesting that retinal dysfunction can precede clinically apparent visual loss.¹⁰ Increasing use of multimodal retinal imaging has also demonstrated that ocular involvement in complement-mediated glomerular disease is more heterogeneous than previously appreciated and is

not limited exclusively to the classic drusen-like phenotype.^{12,13}

The introduction of colour fundus photography, fundus autofluorescence, spectral-domain optical coherence tomography, optical coherence tomography angiography and fluorescein angiography has considerably improved the detection and longitudinal assessment of retinal abnormalities in DDD.^{12,13} Nevertheless, evidence regarding ocular involvement remains limited because of the exceptional rarity of the disease. Most available data originate from isolated case reports and small case series, while systematically collected ophthalmological cohorts remain scarce.¹⁴ Recent reports have further highlighted choroidal neovascularisation as a potentially vision-threatening complication requiring long-term ophthalmic surveillance.¹⁵ In addition, histopathological studies have shown that deposits associated with DDD share structural and compositional features with drusen observed in age-related macular degeneration, further supporting a central role of complement dysregulation in their development.¹⁶

Despite growing recognition of retinal involvement in DDD, the frequency and spectrum of ocular abnormalities among consecutively identified patients with biopsy-confirmed disease remain insufficiently characterised.

Therefore, the present study aimed to determine the frequency of retinal involvement in patients with biopsy-confirmed DDD identified within a consecutive native kidney biopsy cohort at a single tertiary referral centre and to characterise the associated clinical findings and multimodal retinal imaging features.

Methods

Study design and patient selection

This retrospective observational study was conducted at the University Clinical Centre of the Republic of Srpska, Bosnia and Herzegovina. Histopathological evaluation of native kidney biopsy specimens was performed in collaboration with the University of Zagreb School of Medicine, Croatia.

The study included all consecutive patients who underwent native kidney biopsy between 1 Jan-

uary 2021 and 31 March 2026. Patients with biopsy-confirmed MPGN were identified through a retrospective review of the renal biopsy database and subsequently underwent comprehensive ophthalmological evaluation to assess for retinal involvement. Medical records, renal histopathology reports and ophthalmological records were retrospectively reviewed.

Histopathological evaluation

Renal biopsy specimens were evaluated using routine light microscopy, immunofluorescence and electron microscopy, which represent the standard diagnostic methods for the evaluation and classification of MPGN. Histopathological assessment was performed according to established diagnostic criteria. The diagnosis of DDD, formerly referred to as MPGN type II, was established by electron microscopy, demonstrating the characteristic intramembranous electron-dense deposits within the glomerular basement membrane.

Ophthalmological examination

All patients with biopsy-confirmed MPGN underwent a comprehensive ophthalmological examination irrespective of the presence of visual symptoms. The examination included assessment of best-corrected visual acuity (BCVA), slit-lamp biomicroscopy, intraocular pressure measurement, dilated fundus examination and multimodal retinal imaging. Multimodal retinal imaging consisted of colour fundus photography and fundus autofluorescence (FAF) using the CLARUS 700 system (Carl Zeiss Meditec AG, Germany), optical coherence tomography (OCT) and optical coherence tomography angiography (OCTA) (CIRRUS 6000 HD-OCT, Carl Zeiss Meditec AG, Germany). Additional ophthalmological investigations, including automated visual field testing (Octopus 900, Haag-Streit AG, Könis, Switzerland), were performed when clinically indicated.

Retinal findings were evaluated for the presence of drusen-like deposits, retinal pigment epithelium abnormalities, pigmentary changes, retinal pigment epithelial detachment, outer retinal atrophy, subretinal fluid and choroidal neovascularisation.

Data collection and statistical analysis

Demographic, clinical, histopathological and ophthalmological data were extracted from the pa-

tients' medical records. Demographic variables included age and sex. Clinical data comprised renal diagnosis, presenting symptoms and ophthalmological findings. Ophthalmological data included BCVA, anterior and posterior segment examination findings and multimodal retinal imaging characteristics. Given the limited number of patients with biopsy-confirmed DDD, the analysis was descriptive.

Results

Study population

During the study period, a total of 110 native kidney biopsies were performed, of which 7 (6.4 %) were diagnosed as MPGN. All patients underwent comprehensive ophthalmological examination (Table 1).

Table 1: Overview of the study population

Variable	n (%)
Native kidney biopsies	110 (100.0)
Patients with biopsy-confirmed MPGN	7 (6.4)
Dense deposit disease (DDD)	3 (42.9)*
MPGN type I	4 (57.1)*
Patients who underwent ophthalmological examination	7 (100.0)
Patients with retinal involvement	2 (28.6)†
Patients without retinal involvement	5 (71.4) †

*Percentage calculated among patients with biopsy-confirmed MPGN (n = 7). † Percentage calculated among patients who underwent ophthalmological examination (n = 7). MPGN: membranoproliferative glomerulonephritis;

Table 2: Baseline characteristics of patients with biopsy-confirmed MPGN

Sex	Age (years)	Histological diagnosis	Ocular involvement
F	27	DDD (formerly MPGN type II)	+
F	35	DDD (formerly MPGN type II)	+
M	50	DDD (formerly MPGN type II)	-
F	28	MPGN type I	-
M	58	MPGN type I	-
M	52	MPGN type I	-
F	55	MPGN type I	-

MPGN: membranoproliferative glomerulonephritis; DDD: dense deposit disease;

The study cohort comprised seven patients (four females [57.1 %] and three males [42.9 %]) with

a mean age of 43.6 ± 13.2 years (Table 2). Histopathological examination identified three patients (42.9 %) with dense deposit disease (DDD) and four patients (57.1 %) with other MPGN subtypes. Retinal involvement was observed in two patients (28.6 %), corresponding to 66.7 % of patients with DDD, whereas no retinal abnormalities were detected in the remaining patient with DDD or in patients with other MPGN subtypes.

Clinical, renal and ophthalmological characteristics of patients with retinal involvement

Renal manifestations of the MPGN were nephrotic syndrome in five patients and rapid-progressive glomerulonephritis in two patients. The clinical characteristics of the two patients with retinal involvement are summarised in Table 3. Although both patients had biopsy-confirmed DDD, they presented with distinct retinal phenotypes. Patient 1 developed an acute retinopathy characterised by bilateral retinal haemorrhages, cotton-wool spots, macular oedema and transient macular star formation during follow-up. In contrast, patient 2 exhibited chronic degenerative retinal changes with bilateral drusen-like deposits, retinal pigment epithelium abnormalities and peripheral retinal rarefaction.

Visual acuity improved during follow-up in both patients. Patient 1 showed regression of retinal lesions following systemic treatment combined with posterior sub-Tenon triamcinolone acetate, whereas patient 2 remained ophthalmologically stable without specific ocular treatment. Both patients progressed to end-stage kidney disease requiring maintenance haemodialysis and were awaiting kidney transplantation.

Multimodal retinal imaging findings

Multimodal retinal imaging findings are summarised in Table 4. OCT demonstrated distinct structural retinal changes in the two patients. Patient 1 demonstrated hyperreflective retinal foci, neurosensory retinal detachment and retinal pigment epithelium irregularities, with marked regression during follow-up (Figure 1). In contrast, patient 2 showed bilateral drusen-like deposits, shallow pigment epithelial detachments and mild retinal pigment epithelium irregularities without intraretinal or subretinal fluid (Figure 2). OCTA demonstrated no evidence of choroidal neovascularisation in either patient. Fundus autofluorescence was unremarkable in patient 1, whereas punctate hyper auto-fluorescent changes temporal to the macula were observed in the right eye of patient 2 (Figure 3).

Table 3: Clinical characteristics of patients with retinal involvement

Variable	Patient 1	Patient 2
Sex	Female	Female
Age (years)	27	35
Year of DDD diagnosis	2025	2021
Renal diagnosis	DDD	DDD
Presenting renal manifestation	Markedly elevated serum creatinine	Proteinuria
Ophthalmic symptoms	Bilateral blurred vision	Bilateral blurred central vision
BCVA OD (initial/final)	0.1 / 0.5	0.1 / 0.7
BCVA OS (initial/final)	0.2 / 0.7	0.9 / 1.0
Anterior segment findings	Unremarkable	Unremarkable
Fundus findings	Acute exudative-ischaemic retinal changes	Chronic drusenoid retinal changes
Systemic treatment	Prednisone, mycophenolate mofetil, haemodialysis	Haemodialysis
Ophthalmic treatment	Posterior sub-Tenon triamcinolone acetate	None
Final ophthalmological outcome	Regression of retinal lesions with improvement in BCVA	Stable retinal findings with improvement in BCVA
Renal outcome	End-stage kidney disease requiring maintenance haemodialysis	End-stage kidney disease requiring maintenance haemodialysis
Kidney transplantation status	Awaiting kidney transplantation	Awaiting kidney transplantation

BCVA: best-corrected visual acuity; OD: oculi dextri (right eye), OS: oculi sinistri (left eye); DDD: dense deposit disease;

Table 4: Retinal imaging findings in patients with dense deposit disease

Imaging finding	Patient 1	Patient 2
Drusen-like deposits	Transient yellow deposits	Multiple medium-sized deposits predominantly temporal to the macula
RPE abnormalities	Present	Present
Hyperreflective retinal foci	Present	Absent
Pigmentary changes	Absent	Present
Pigment epithelial detachment (PED)	Absent	Present
Outer retinal atrophy	Absent	Absent
Subretinal fluid/Neurosensory retinal detachment	Present	Absent
Choroidal neovascularisation	Absent	Absent
FAF abnormalities	No significant fundus autofluorescence abnormalities.	Punctate hyperautofluorescence temporal to the macula (OD)
OCTA abnormalities	No CNV	No CNV; punctate flow-void areas within the sub-RPE/choriocapillaris

RPE: Retinal pigment epithelium; FAF: Fundus autofluorescence; OCTA: optical coherence tomography angiography; CNV: choroidal neovascularisation;

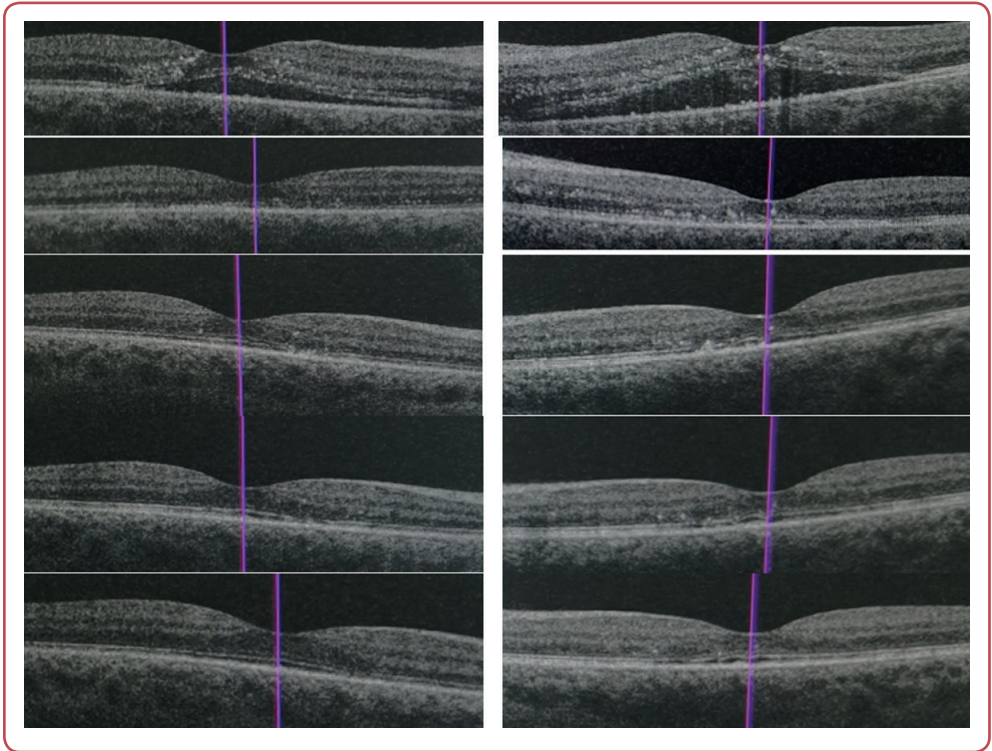


Figure 1: Serial optical coherence tomography (OCT) images of patient 1 obtained at baseline and during follow-up after posterior sub-Tenon's triamcinolone acetate treatment. Images displayed in the left column correspond to the right eye (OD), whereas images displayed in the right column correspond to the left eye (OS)

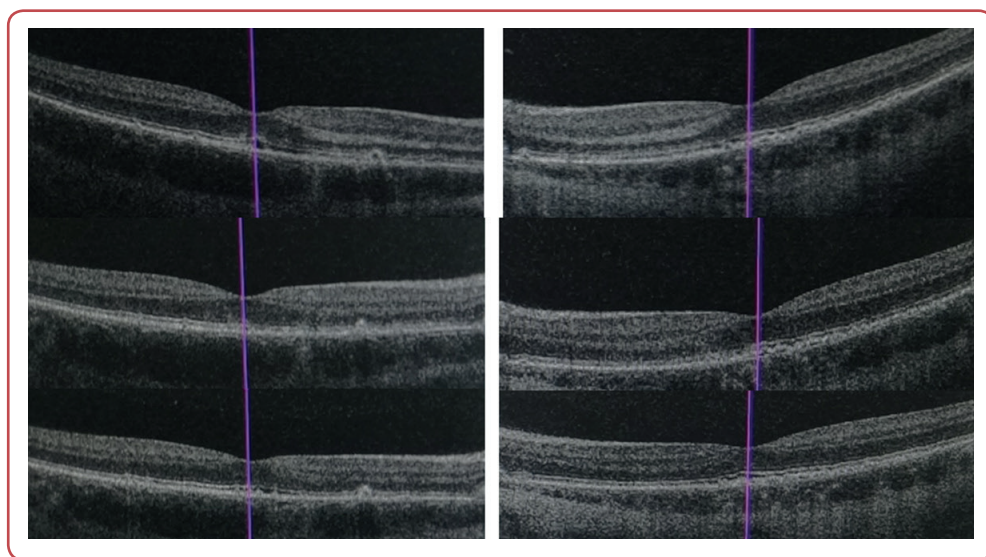


Figure 2: Serial optical coherence tomography (OCT) images of patient 2 during follow-up. Images displayed in the left column correspond to the right eye (OD), whereas images displayed in the right column correspond to the left eye (OS)

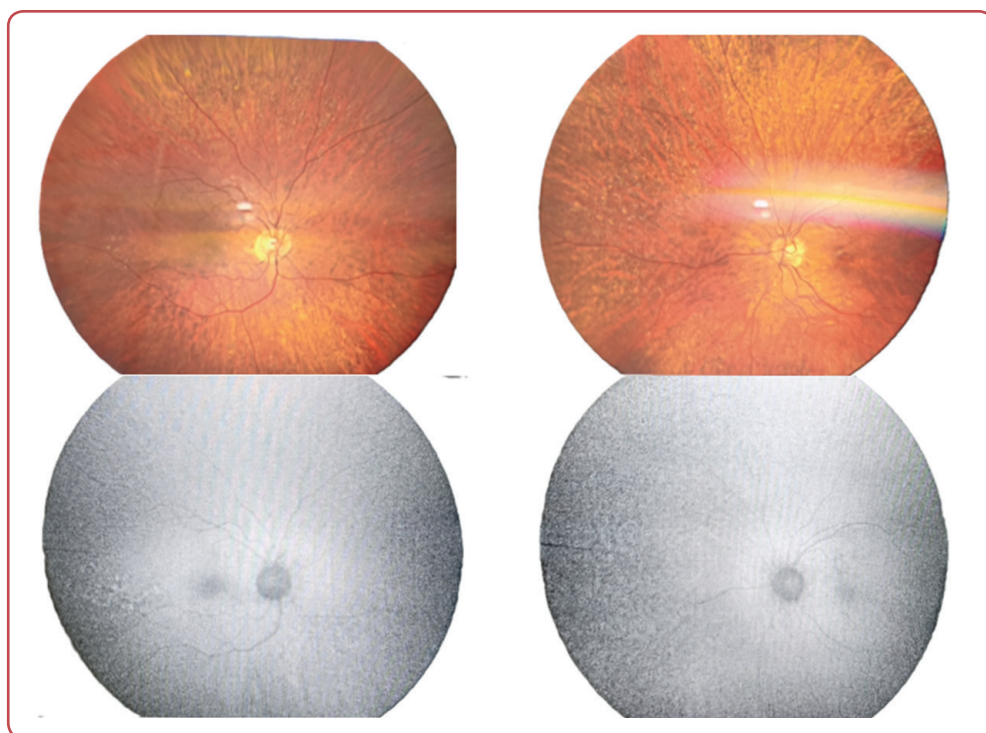


Figure 3: Colour fundus photography and fundus autofluorescence of patient 2 demonstrating chronic drusenoid retinal phenotype associated with dense deposit disease

Discussion

In this retrospective series of patients with biopsy-confirmed membranoproliferative glomerulonephritis, retinal abnormalities were identified exclusively in those with dense deposit disease. Two of the three patients with DDD demonstrated ocular involvement, whereas neither the remaining patient with DDD nor any patient with other MPGN subtypes exhibited retinal abnormalities. Although both affected patients shared the same renal histopathological diagnosis, their ophthalmological presentations differed considerably. One patient developed an acute exudative–ischaemic retinopathy, whereas the other showed the chronic drusenoid retinal phenotype more typically associated with DDD. These findings highlight the marked clinical heterogeneity of retinal involvement in DDD and emphasise the value of multimodal ophthalmic assessment in affected patients.

DDD is a complement-mediated renal disease within the spectrum of C3 glomerulopathies, characterised by persistent dysregulation of the alternative complement pathway and dense intramembranous deposits within the glomerular basement membrane.^{1-6, 17, 18} Because Bruch's membrane shares important structural and biochemical characteristics with the glomerular basement membrane, it has long been considered a second site susceptible to complement-mediated extracellular deposit formation. The characteristic ocular manifestations of DDD were first systematically described by Duvall-Young et al who documented bilateral drusen-like fundus lesions and correlated the clinical findings with fluorescein angiography and histopathological abnormalities of Bruch's membrane.⁷⁻⁹ Subsequently, Mullins et al demonstrated that these deposits contain numerous complement components and other proteins resembling those found in age-related drusen, providing further evidence that chronic complement activation underlies retinal involvement in DDD.¹⁶ Appel et al integrated these ophthalmic and renal observations into the broader concept that the kidney and retina represent anatomically analogous targets of complement-mediated injury in DDD.⁶

Although drusen-like deposits remain the hallmark of ocular involvement in DDD, subsequent reports have expanded the recognised retinal phenotype. Ritter et al described structural

and functional macular abnormalities in affected patients, while Savige et al highlighted the wide spectrum of retinal manifestations associated with complement-mediated renal disease and emphasised the importance of ophthalmic surveillance in these patients.^{10, 11} Nevertheless, available evidence continues to rely predominantly on isolated case reports and small case series, leaving many aspects of the natural history and optimal ophthalmological management insufficiently defined.^{14, 19}

Unlike the classical retinal phenotype, our first patient presented with bilateral retinal haemorrhages, cotton-wool spots, macular oedema and transient macular star formation. Such an acute exudative–ischaemic presentation has been reported only sporadically in patients with DDD and appears to represent an uncommon manifestation of the disease.^{10, 11} The retinal findings observed in this patient were likely multifactorial. In addition to complement-mediated tissue injury, advanced renal failure may contribute to retinal vascular damage through severe hypertension, anaemia, uraemia and disturbances in fluid homeostasis, all of which may promote retinal ischaemia, increased vascular permeability and exudation.²⁰ Consequently, the acute retinal changes in presented patient were probably the result of interactions between systemic disease and the underlying complement dysregulation rather than the direct effect of DDD alone.

Following systemic treatment, haemodialysis and posterior sub-Tenon's triamcinolone acetate, retinal fluid resolved and visual acuity improved substantially. Although it is impossible to determine the relative contribution of each therapeutic intervention, the favourable anatomical and functional outcome suggests that prompt recognition of ocular involvement combined with optimisation of systemic disease may allow at least partial recovery of vision, even in patients presenting with severe retinal manifestations.

The second patient exhibited the chronic retinal phenotype that is most frequently described in DDD. Multiple drusen-like deposits, retinal pigment epithelium abnormalities and pigment epithelial detachments were observed without evidence of choroidal neovascularisation. These findings closely resemble the characteristic

retinal alterations originally described by Duval-Young et al, who correlated drusen-like fundus lesions with abnormalities of Bruch's membrane and later confirmed by Mullins et al, who demonstrated the presence of complement-rich extracellular deposits within these lesions.^{7,8,16}

Multimodal retinal imaging substantially improved the characterisation of retinal involvement in presented patients. Colour fundus photography documented the distribution of drusen-like deposits, spectral-domain OCT identified retinal pigment epithelium irregularities and pigment epithelial detachments, while fundus autofluorescence demonstrated areas of retinal pigment epithelium dysfunction that were less apparent on clinical examination. OCT angiography excluded secondary choroidal neovascularisation in both patients. Similar observations have been reported by Kheir et al, who highlighted the usefulness of multimodal imaging, particularly OCT, fundus autofluorescence and OCT angiography, for evaluating pigment epithelial detachments and retinal pigment epithelium abnormalities in patients with C3 glomerulopathy.²¹ More recently, Huerta Morales et al further illustrated how multimodal imaging enables comprehensive assessment of drusen-like deposits and associated retinal alterations, facilitating recognition of disease extent and potential complications.²²

Neither patient developed choroidal neovascularisation despite advanced retinal abnormalities. Although uncommon, this complication has been increasingly recognised in DDD and may lead to severe central visual loss if left untreated. Inan  et al recently described choroidal neovascularisation as a vision-threatening complication of DDD, emphasising the importance of careful long-term surveillance in patients with extensive retinal pigment epithelium abnormalities.¹⁵ Consequently, the absence of choroidal neovascularisation in presented patients should not preclude continued ophthalmological follow-up.

Retinal abnormalities were detected in two of the three patients with DDD, whereas none of the patients with other forms of MPGN demonstrated ocular involvement. Although the number of patients is too small to permit meaningful comparisons, this observation is consistent with current understanding that retinal disease occurs predominantly in complement-mediated glomerulopathies. In their comparative clinical series, Dalvin et al showed that retinal drusen are con-

siderably more frequent in complement-mediated MPGN than in immune complex-mediated disease.¹² Their subsequent report also challenged the traditional assumption that fundus drusen are confined to MPGN type II, demonstrating that similar retinal changes may occasionally occur across different MPGN subtypes, although they remain most characteristic of DDD.¹³ These observations support the concept that retinal pathology reflects complement dysregulation rather than histological classification alone.

The absence of retinal abnormalities in one patient with biopsy-confirmed DDD also deserves consideration. Previous studies have demonstrated considerable variability in ocular involvement among patients with C3 glomerulopathies, suggesting that retinal disease is influenced not only by the underlying complement abnormality but also by disease duration, cumulative complement-mediated injury and additional genetic or environmental modifiers.^{11, 14, 23, 24} Therefore, the absence of retinal lesions at a single examination should not exclude future ophthalmological surveillance.

The broader literature also suggests that retinal changes in renal disease should not be interpreted solely through the traditional histological classification of MPGN. Harraka et al identified increased retinal drusen in several forms of glomerulonephritis, including diseases with immune deposits and interpreted these findings as indirect evidence of systemic complement activation.²⁵ Their subsequent study in IgA glomerulonephritis reached a similar conclusion, further supporting a relationship between retinal deposit formation and complement-mediated disease mechanisms beyond DDD itself.²⁶ These observations do not reduce the diagnostic relevance of drusen-like lesions in DDD, but they do indicate that such deposits are not entirely disease-specific. In the appropriate clinical setting, their presence should therefore prompt consideration of systemic complement dysregulation rather than be regarded as a pathognomonic marker of one renal histological subtype.

The acute retinal presentation in first patient also requires distinction from the chronic deposit-related phenotype. Retinal haemorrhages, cotton-wool spots, exudation and macular oedema may occur in advanced chronic kidney disease as a consequence of associated microvascular and systemic abnormalities. Deva et al found that

patients with chronic kidney disease stages 3–5 had more frequent and more severe microvascular retinal abnormalities than patients with milder renal impairment and that previously unrecognised vision-threatening lesions were not uncommon in advanced disease.²⁷ This supports the interpretation that renal failure and its systemic consequences may have contributed substantially to the acute exudative–ischaemic picture observed in our patient. It remains possible, however, that the underlying complement disorder increased retinal susceptibility and modified the clinical expression of otherwise nonspecific renal microvascular injury.

The coexistence of acute and chronic retinal phenotypes within the same small DDD cohort is clinically relevant. Drusen-like deposits and Retinal pigment epithelium (RPE) abnormalities may remain asymptomatic for prolonged periods, whereas macular oedema, neurosensory detachment or neovascular complications may result in a more abrupt decline in visual function. The scoping review by McCarney et al, which included 38 studies, confirmed that drusen-like deposits are the most frequently reported retinal manifestation of DDD, while pigment epithelial detachment and macular atrophy are less common. The predominance of case reports in that review also illustrates the limited quality of the available evidence and the lack of standardised data on progression, prognosis and treatment.¹⁴

From a practical perspective, ophthalmological assessment in DDD should not be restricted to visual acuity and routine ophthalmoscopy. Visual acuity may remain relatively preserved despite extensive deposits or peripheral RPE changes and early structural abnormalities may be detected only by retinal imaging. Baseline examination should therefore include dilated fundus evaluation and OCT of the macula. Fundus photography and autofluorescence are useful for documenting the distribution of deposits and RPE disturbance, while OCTA or dye angiography should be considered when neovascularisation is suspected. The exact interval of follow-up cannot be defined from the present evidence and should be individualised according to symptoms, disease duration, retinal phenotype and the presence of outer retinal or RPE abnormalities.

Presented findings also raise the question of whether retinal examination may provide information about systemic complement activity.

The similarity between deposits in the glomerular basement membrane and Bruch's membrane, together with the occurrence of retinal drusen in several complement-associated glomerulopathies, makes the retina a potentially accessible site for observing systemic complement-mediated tissue injury.^{16, 25, 26} At present, however, retinal lesions cannot be regarded as a validated biomarker of renal activity or prognosis. Their relationship with renal function, complement profile, disease duration, treatment response and transplant outcome remains insufficiently defined. Longitudinal studies combining nephrological data with quantified multimodal retinal imaging would be required before such a role could be established.

This study has several limitations. Its retrospective design and the small number of patients with biopsy-confirmed DDD limited statistical analysis and precluded meaningful comparisons between disease subgroups. In addition, the rarity of the disease limits the generalisability of our findings. Nevertheless, the study provides valuable clinicopathological observations from a consecutive biopsy-confirmed cohort of this exceptionally rare complement-mediated disease.

Despite these limitations, the series has several strengths. All renal diagnoses were established by biopsy, all patients with MPGN underwent ophthalmological examination and the affected patients were evaluated using multimodal retinal imaging. The study also illustrates two distinctly different ocular presentations within the same rare renal disease: an acute exudative–ischaemic retinopathy with partial recovery and a chronic drusenoid phenotype with RPE and outer retinal involvement. Presenting these phenotypes together may help clinicians recognise that ocular involvement in DDD is broader than the classical description of asymptomatic fundus drusen.

Conclusion

In conclusion, retinal involvement in this series occurred only in patients with DDD, although its expression varied from an acute, potentially reversible exudative–ischaemic presentation to chronic drusen-like deposits with RPE abnormalities and pigment epithelial detachment. These findings support

ophthalmological evaluation of patients with biopsy-confirmed DDD even in the absence of visual symptoms. Multimodal imaging is particularly useful for defining the retinal phenotype, detecting structural complications and guiding follow-up. Larger prospective studies are needed to clarify the natural history of ocular disease in DDD and to determine whether retinal changes reflect systemic complement activity or have prognostic relevance for renal and visual outcomes.

Ethics

The study was approved by the Ethics Committee of the University Clinical Centre of the Republic of Srpska, Banja Luka, Bosnia and Herzegovina under approval number No: 01-19-576-2/25, dated 23 December 2025.

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Conflicts of interest

The authors declare that there is no conflict of interest.

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Data access

The data that support the findings of this study are available from the corresponding author upon reasonable individual request.

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