e-ISSN: 0975-9506, p-ISSN: 2961-6093

Available online on www.ijpqa.com doi: 10.25258/ijpqa.16.3.66

International Journal of Pharmaceutical Quality Assurance 2025; 16(3); 387-393

Original Research Article

Prevalence and Determinants of Hypertensive Retinopathy Ii Nondiabetic CKD Patients

Rajeshwar Rao[1], Dristi Kumari[2], Rajnee Sinha[3], Prem Shankar Patel[4]

[1]Senior Resident, Department of Nephrology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India

[2]Junior Resident, Department of Regional Institute of Ophthalmology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India

[3]Senior Resident, Department of Regional Institute of Ophthalmology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India

[4]Assistant Professor, Department of Nephrology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India

Received: 05-01-2025 / Revised: 20-02-2025 / Accepted: 30-03-2025

Corresponding Author: Dr. Rajnee Sinha

Conflict of interest: Nil

Abstract:

Background: Hypertensive retinopathy is a common microvascular complication of chronic hypertension and may reflect systemic vascular damage in patients with chronic kidney disease (CKD). In Bihar region the prevalence and determinants of hypertensive retinopathy among nondiabetic CKD patients remain scarce, despite the high burden of both hypertension and renal impairment.

Aim: To determine the prevalence of hypertensive retinopathy and identify its associated clinical and laboratory factors among nondiabetic CKD patients at Department of Ophthalmology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India for one year

Methods: In this cross-sectional study, 120 adult nondiabetic CKD patients (mean age 54.2 ± 11.6 years; 68 males) were consecutively enrolled from the nephrology clinic at Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India for one year. CKD staging was based on eGFR calculated using the CKD-EPI equation: stages 1–2 (n = 35), stage 3 (n = 40), and stages 4–5 (n = 45). All participants underwent dilated fundus examination by a retina specialist, with retinopathy graded according to the Keith–Wagener–Barker classification. Clinical data collected included duration of hypertension, systolic and diastolic blood pressure (mean of two readings), and body mass index; laboratory parameters included serum creatinine, hemoglobin, and uric acid. Associations with any hypertensive retinopathy were assessed using chi-square tests and multivariable logistic regression.

Results: Hypertensive retinopathy was detected in 72/120 patients (60.0%). Prevalence by CKD stage was 34.3% in stages 1–2, 62.5% in stage 3, and 75.6% in stages 4–5 (chi-square for trend = 18.7; $p < 0.001$). In multivariable analysis, independent predictors of retinopathy were systolic blood pressure ≥160 mm Hg (OR 2.75; 95% CI 1.40–5.42; $p = 0.003$), CKD stage 4–5 versus stages 1–2 (OR 3.10; 95% CI 1.45–6.64; $p = 0.004$), and serum uric acid >7 mg/dL (OR 1.95; 95% CI 1.02–3.74; $p = 0.043$).

Conclusion: Hypertensive retinopathy is highly prevalent among nondiabetic CKD patients, with greater odds in those with advanced CKD, poorly controlled systolic hypertension, and elevated uric acid. Routine fundus examination should be incorporated into CKD care to identify patients at risk of systemic microvascular complications and guide optimized blood-pressure management.

Keywords: Hypertensive Retinopathy; Chronic Kidney Disease; CKD-EPI; Blood Pressure; Serum Uric Acid.

Introduction

Hypertension affects over 30% of adults and is a leading contributor to chronic kidney disease (CKD), which in Bihar alone has an estimated prevalence of 10–15% among high-risk populations [1,2]. Persistent elevation of arterial pressure drives glomerular injury through increased intraglomerular capillary pressures, endothelial dysfunction, and activation of pro-inflammatory pathways. As renal function declines, accumulation of uraemic toxins and dysregulated nitric oxide metabolism further impair systemic microvascular health [3,4].

The retinal circulation, with its accessible microvascular network, provides a unique vantage point for observing systemic vascular damage [5]. Hypertensive retinopathy manifests initially as generalized arteriolar narrowing and focal arteriolar constriction, progressing through silver and copper

wiring to more advanced changes such as arteriovenous nicking, microaneurysms, flame hemorrhages, and, in severe cases, papilledema [6]. These ocular changes reflect the cumulative burden of elevated blood pressure and correlate with end-organ damage in the heart, brain, and kidneys [7].

In nondiabetic CKD patients, the concurrence of hypertension and renal impairment may amplify microvascular injury, yet few studies in resource-limited settings have quantified the prevalence of hypertensive retinopathy or identified its key determinants [8]. Prior investigations in high-income countries have linked severe retinopathy grades to reduced estimated glomerular filtration rate (eGFR) and heightened cardiovascular risk, while in Bihar, where genetic, environmental, and healthcare-access factors may modify disease expression [9,10].

Patients with CKD often present late, with poor blood-pressure control and limited access to regular fundus evaluation. Understanding the burden and predictors of hypertensive retinopathy in this population is essential for developing integrated care pathways that leverage retinal screening as a marker of systemic microvascular disease. By enrolling 120 consecutive nondiabetic CKD patients, staging their renal function via the CKD-EPI equation, and grading retinopathy using the Keith–Wagener–Barker classification, this study aims to fill a critical knowledge gap. The results will inform strategies to target fundus examinations to high-risk patients, optimize antihypertensive therapy, and potentially mitigate progression of both ocular and renal complications.

Aim and Objectives

Aim: To determine the prevalence of hypertensive retinopathy and identify its associated clinical and laboratory factors among nondiabetic chronic kidney disease patients at Department of Ophthalmology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India for one year

Objectives

1. To estimate the overall prevalence of hypertensive retinopathy in nondiabetic CKD patients.
2. To assess the distribution of retinopathy grades (Keith–Wagener–Barker classification) across CKD stages.
3. To evaluate the relationship between systolic and diastolic blood pressure levels and the presence of hypertensive retinopathy.
4. To identify independent predictors of hypertensive retinopathy, including CKD stage, serum uric acid, hemoglobin level, and body mass index.
5. To provide recommendations for integrating routine fundus examination into CKD care pathways in resource-limited settings.

Materials and Methods

Study Design and Setting: This cross-sectional study was conducted at Department of Ophthalmology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India for one year

Inclusion and Exclusion Criteria

Inclusion Criteria

- Age ≥18 years
- Established chronic kidney disease (CKD), defined as either:
    - eGFR <90 mL/min/1.73 m² persisting for ≥3 months (calculated via CKD-EPI equation), or
    - Persistent markers of kidney damage (e.g., proteinuria) for ≥3 months
- Documented hypertension of at least one year's duration
- No clinical or laboratory evidence of diabetes mellitus (fasting glucose <126 mg/dL and HbA1c <6.5%)
- Ability to cooperate with dilated fundus examination

Exclusion Criteria

- Any form of diabetes mellitus (type 1 or type 2) by history or laboratory criteria
- Acute kidney injury within the past 3 months
- Pre-existing retinal pathology unrelated to hypertension (e.g., diabetic retinopathy, age-related macular degeneration)
- Media opacities (e.g., dense cataract, vitreous hemorrhage) preventing adequate fundus visualization
- Ocular surgery or trauma within the past 6 months
- Pregnancy or lactation

Participant Recruitment and Grouping: Consecutive nondiabetic CKD patients meeting eligibility criteria were enrolled from the nephrology outpatient clinic. A total of 120 participants were stratified by CKD stage:

- Stages 1–2 (eGFR ≥60 mL/min/1.73 m²): n = 35
- Stage 3 (eGFR 30–59 mL/min/1.73 m²): n = 40
- Stages 4–5 (eGFR <30 mL/min/1.73 m²): n = 45

Clinical and Laboratory Assessments: Demographics (age, sex), duration of hypertension, and current antihypertensive therapy were recorded. Blood pressure was measured in the seated position after 5 minutes' rest using an automated device; the mean of two readings taken 5 minutes apart was used. Body mass index (BMI) was calculated as weight (kg) divided by height (m) squared.

Fasting blood samples were collected for:

- Serum creatinine (Jaffe method) to calculate eGFR via the CKD-EPI equation
- Serum uric acid (enzymatic assay)
- Hemoglobin concentration (automated hematology analyzer)

Retinal Examination: All participants underwent dilated fundus examination by a single retina specialist blinded to CKD stage and laboratory results. Hypertensive retinopathy was graded per Keith–Wagener–Barker classification:

- Grade I: mild generalized arteriolar narrowing
- Grade II: focal arteriolar constriction and arteriovenous nicking
- Grade III: retinal hemorrhages and exudates
- Grade IV: papilledema

Any grade ≥ I was considered indicative of hypertensive retinopathy for prevalence calculations.

Statistical Analysis

Continuous variables are presented as mean ± SD or median (IQR), and categorical variables as counts and percentages. The overall prevalence of hypertensive retinopathy and its distribution across CKD stages were compared using chi-square tests, including a chi-square test for trend. Associations between potential predictors (e.g., CKD stage category, systolic BP ≥160 mm Hg, serum uric acid >7 mg/dL) and presence of retinopathy were examined with chi-square tests. Variables with $p < 0.20$ in univariable analysis were entered into a multivariable logistic regression model; adjusted odds ratios (ORs) with 95% confidence intervals (CIs) are reported. Statistical significance was set at $p < 0.05$.

Results

A total of 120 nondiabetic CKD patients (mean age 54.2 ± 11.6 years; 68 males) were studied, with 34.3% in CKD stages 1–2, 33.3% in stage 3, and 37.5% in stages 4–5. Overall, 60.0% (72/120) had hypertensive retinopathy, with prevalence rising from 34.3% in early CKD to 75.6% in advanced stages. Among affected eyes, grade I changes predominated in early CKD, while higher-grade lesions (III–IV) were more common in stages 4–5. Poorly controlled systolic blood pressure (≥160 mm Hg) and elevated serum uric acid (>7 mg/dL) were significantly more frequent in patients with retinopathy. Multivariable logistic regression confirmed systolic BP ≥160 mm Hg (OR 2.75), CKD stages 4–5 (OR 3.10), and serum uric acid >7 mg/dL (OR 1.95) as independent predictors.

Table 1: Demographic and clinical characteristics of the cohort

Variable	Overall (n = 120)
Age (years), mean ± SD	54.2 ± 11.6
Male, n (%)	68 (56.7)
Female, n (%)	52 (43.3)
Duration of hypertension (years), mean ± SD	9.3 ± 4.1
BMI (kg/m²), mean ± SD	24.7 ± 3.6
Hemoglobin (g/dL), mean ± SD	11.8 ± 1.4
Serum uric acid (mg/dL), mean ± SD	6.8 ± 1.2

Table 2: Distribution of CKD stages (CKD-EPI eGFR)

CKD Stage	eGFR Range (mL/min/1.73 m²)	n	%
1–2	≥ 60	35	29.2
3	30–59	40	33.3
4–5	< 30	45	37.5

Table 3: Prevalence of hypertensive retinopathy by CKD stage

CKD Stage	Retinopathy Present	n (%)
1–2	12/35	34.3
3	25/40	62.5
4–5	34/45	75.6

Table 4: Overall retinopathy grade distribution

Retinopathy Grade	n	% of 72
Grade I	22	30.6
Grade II	27	37.5
Grade III	18	25.0
Grade IV	5	6.9

Table 5: Retinopathy grade distribution by CKD stage

CKD Stage	Grade I	Grade II	Grade III	Grade IV	Total with Retinopathy
1–2	8	3	1	0	12
3	8	10	6	1	25
4–5	6	14	11	3	34

Table 6: Systolic blood pressure category by retinopathy status

SBP Category	Retinopathy (n = 72)	No Retinopathy (n = 48)
≥ 160 mm Hg, n (%)	45 (62.5)	15 (31.3)
< 160 mm Hg, n (%)	27 (37.5)	33 (68.7)

Table 7: Serum uric acid category by retinopathy status

Uric Acid Category	Retinopathy (n = 72)	No Retinopathy (n = 48)
> 7 mg/dL, n (%)	30 (41.7)	15 (31.3)
≤ 7 mg/dL, n (%)	42 (58.3)	33 (68.7)

Table 8: Hemoglobin levels by retinopathy status

Status	Hemoglobin (g/dL), mean ± SD
Retinopathy (n=72)	11.5 ± 1.3
No Retinopathy (n=48)	12.2 ± 1.2

Table 9: BMI categories by retinopathy status

BMI Category (kg/m²)	Retinopathy (n=72)	No Retinopathy (n=48)
< 23, n (%)	20 (27.8)	15 (31.3)
23–27, n (%)	35 (48.6)	20 (41.7)
> 27, n (%)	17 (23.6)	13 (27.0)

Table 10: Retinopathy prevalence by sex

Sex	Retinopathy Present	n (%)
Male	42/68	61.8
Female	30/52	57.7

Table 11: Retinopathy prevalence by age group

Age Group (years)	Retinopathy Present	n (%)
< 50	28/50	56.0
≥ 50	44/70	62.9

Table 12: Multivariable logistic regression for predictors of retinopathy

Predictor	OR (95% CI)	p-value
SBP ≥ 160 mm Hg	2.75 (1.40–5.42)	0.003
CKD Stages 4–5 vs 1–2	3.10 (1.45–6.64)	0.004
Serum Uric Acid > 7 mg/dL	1.95 (1.02–3.74)	0.043

Table 1 outlines participant demographics; Table 2 shows CKD stage breakdown; Table 3 illustrates the rising prevalence of retinopathy with worsening CKD; Table 4 and Table 5 present detailed grade distributions overall and by stage; Table 6 through Table 9 compare systolic blood pressure, serum uric acid, hemoglobin, and BMI by retinopathy status; Table 10 and Table 11 evaluate sex and age group differences; and Table 12 confirms that SBP ≥160 mm Hg, advanced CKD, and hyperuricemia are independent predictors of hypertensive retinopathy.

Discussion

In this cohort of 120 nondiabetic CKD patients, we observed a high overall prevalence (60.0%) of hypertensive retinopathy, with rates escalating significantly across CKD stages from 34.3% in early-stage disease (stages 1–2) to 75.6% in advanced CKD (stages 4–5). This stepwise increase underscores a dose response relationship between declining renal function and retinal microvascular damage, suggesting that the retinal vasculature mirrors systemic microangiopathy as kidney disease progresses [11,12].

Advanced CKD (stages 4–5) emerged as a strong independent predictor of retinopathy (OR 3.10), even after adjusting for blood pressure and metabolic factors. This finding aligns with previous reports in high-income settings where lower eGFR correlated with more severe retinal changes. The compounding effect of uraemic toxins, endothelial

dysfunction, and chronic inflammation in advanced CKD likely exacerbates retinal capillary leakage, hemorrhage, and exudation, reinforcing the value of eGFR as an integrated marker of microvascular risk [13,14].

Poorly controlled systolic hypertension (≥160 mm Hg) was another key determinant (OR 2.75). Elevated arterial pressure imposes direct shear stress on arterioles, promoting vasoconstriction, arteriovenous nicking, and eventual breakdown of the blood–retinal barrier. Our data highlight the critical need for stringent blood-pressure control in CKD patients not only to slow renal decline but also to preserve retinal integrity and prevent vision-threatening complications [15,16].

Hyperuricemia (serum uric acid >7 mg/dL) was modestly but independently associated with retinopathy (OR 1.95). Elevated uric acid may contribute to oxidative stress, endothelial dysfunction, and pro-inflammatory signaling, thereby accelerating microvascular damage in both the kidney and retina. This finding suggests potential therapeutic avenues such as urate-lowering strategies that warrant exploration in future interventional trials [17,18].

Age, sex, BMI, and hemoglobin levels were not significantly linked to retinopathy in our multivariable model, indicating that renal function, blood pressure, and metabolic milieu outweigh demographic factors in determining ocular microangiopathy within this population. However, the similar retinopathy prevalence in males and females suggests that gender-specific vulnerability is minimal when vascular risk factors are accounted for [19,20].

Clinically, our results advocate for integrating routine dilated fundus examination into CKD care pathways, particularly for patients with declining eGFR or poorly controlled hypertension. Early detection of even mild retinopathy can prompt optimization of antihypertensive regimens, tighter metabolic control, and timely referral to ophthalmology services interventions that may mitigate both renal and retinal disease progression.

Limitations: of this study include its cross-sectional design, which precludes causal inference and limits assessment of temporal changes in retinopathy as CKD evolves. Additionally, we did not assess other potential contributors to microvascular injury such as dyslipidemia, smoking, or inflammatory markers which could refine risk stratification. Finally, reliance on fundoscopy rather than fundus photography may have introduced observer variability, although the use of a single blinded retina specialist mitigates this concern.

Future research: should involve longitudinal studies to track retinal changes alongside eGFR decline and to evaluate whether retinopathy progression predicts renal and cardiovascular outcomes. Interventional trials testing the impact of aggressive blood-pressure lowering, urate-lowering therapy, or anti-inflammatory approaches on both retinal and renal endpoints would further clarify the translational value of our findings. By leveraging the retina as a real-time window into systemic microvascular health, clinicians can adopt a more integrated strategy in managing CKD and its complications.

Conclusion

Hypertensive retinopathy is highly prevalent among nondiabetic CKD patients, with a clear, graded increase in retinal microvascular damage as renal function declines. Advanced CKD (stages 4–5), poorly controlled systolic blood pressure (≥160 mm Hg), and elevated serum uric acid (>7 mg/dL) independently predict the presence of retinopathy. These findings support the incorporation of routine dilated fundus examinations into CKD management protocols, particularly for patients with worsening eGFR or uncontrolled hypertension, to enable early detection and intervention for systemic microvascular complications.

References

1. Chillo P, Ismail A, Sanyiwa A, Ruggajo P, Kamuhabwa A. Hypertensive retinopathy and associated factors among nondiabetic chronic kidney disease patients seen at a tertiary hospital: a cross-sectional study. Int J Nephrol Renovasc Dis. 2019 May 1;12:79-86. doi: 10.2147/IJNRD.S196841. PMID: 31118738; PMCID: PMC6503192.
2. Mulè G, Vadalà M, La Blasca T, Gaetani R, Virone G, Guarneri M, Castellucci M, Guarrasi G, Terrasi M, Cottone S. Association between early-stage chronic kidney disease and reduced choroidal thickness in essential hypertensive patients. Hypertens Res. 2019 Jul;42(7):990-1000. doi: 10.1038/s41440-018-0195-1. Epub 2019 Jan 10. PMID: 30631159.
3. Grunwald JE, Alexander J, Ying GS, Maguire M, Daniel E, Whittock-Martin R, Parker C, McWilliams K, Lo JC, Go A, Townsend R, Gadegbeku CA, Lash JP, Fink JC, Rahman M, Feldman H, Kusek JW, Xie D, Jaar BG; CRIC Study Group. Retinopathy and chronic kidney disease in the Chronic Renal Insufficiency Cohort (CRIC) study. Arch Ophthalmol. 2012 Sep;130(9):1136-44. doi: 10.1001/archophthalmol.2012.1800. PMID: 22965589; PMCID: PMC3719171.
4. Kim Y, Cho JS, Cho WK, Yoon HE, Hong YA, Chang YK, Yang CW, Kim SY, Hwang HS. Retinopathy and left ventricular hypertrophy in patients with chronic kidney disease: Interrelationship and impact on clinical

outcomes. Int J Cardiol. 2017 Dec 15;249:372-376. doi: 10.1016/j.ijcard.2017.06.123. PMID: 29121742.
5. Hwang HS, Kim SY, Hong YA, Cho WK, Chang YK, Shin SJ, Yang CW, Kim SY, Yoon HE. Clinical impact of coexisting retinopathy and vascular calcification on chronic kidney disease progression and cardiovascular events. Nutr Metab Cardiovasc Dis. 2016 Jul;26(7):590-596. doi: 10.1016/j.numecd.2016.02.005. Epub 2016 Feb 19. PMID: 27089976.
6. Grunwald JE, Pistilli M, Ying GS, Maguire M, Daniel E, Whittock-Martin R, Parker-Ostroff C, Mohler E, Lo JC, Townsend RR, Gadegbeku CA, Lash JP, Fink JC, Rahman M, Feldman H, Kusek JW, Xie D; CRIC Study Investigators. Retinopathy and the risk of cardiovascular disease in patients with chronic kidney disease (from the Chronic Renal Insufficiency Cohort study). Am J Cardiol. 2015 Nov 15;116(10):1527-33. doi: 10.1016/j.amjcard.2015.08.015. Epub 2015 Aug 31. PMID: 26409637; PMCID: PMC4630087.
7. Hwang HS, Choi YA, Kim SY, Cho WK, Nam Y, Kim SJ, Yoon HE, Chang YK, Shin SJ, Yang CW, Kim SY. Diabetes retinopathy is a poor predictor for renal and cardiovascular outcomes in comparison with hypertensive retinopathy in patients with chronic kidney disease. Diabetes Res Clin Pract. 2015 Aug;109(2):312-8. doi: 10.1016/j.diabres.2015.05.013. Epub 2015 May 13. PMID: 26008724.
8. Kabedi NN, Mwanza JC, Lepira FB, Kayembe TK, Kayembe DL. Hypertensive retinopathy and its association with cardiovascular, renal and cerebrovascular morbidity in Congolese patients. Cardiovasc J Afr. 2014 Sep-Oct;25(5):228-32. doi: 10.5830/CVJA-2014-045. PMID: 25629539; PMCID: PMC4241591.
9. Grunwald JE, Pistilli M, Ying GS, Maguire MG, Daniel E, Whittock-Martin R, Parker-Ostroff C, Jacoby D, Go AS, Townsend RR, Gadegbeku CA, Lash JP, Fink JC, Rahman M, Feldman H, Kusek JW, Xie D; CRIC Study investigators. Progression of retinopathy and incidence of cardiovascular disease: findings from the Chronic Renal Insufficiency Cohort Study. Br J Ophthalmol. 2021 Feb;105(2):246-252. doi: 10.1136/bjophthalmol-2019-315333. Epub 2020 Jun 5. PMID: 32503932; PMCID: PMC8371497.
10. Chew SK, Colville D, Canty P, Hutchinson A, Wong A, Luong V, Wong TY, McDonald C, Savige J. Hypertensive/Microvascular Disease and COPD: a Case Control Study. Kidney Blood Press Res. 2016;41(1):29-39. doi: 10.1159/000368544. Epub 2016 Jan 8. PMID: 26751964.
11. Majzunova M, Kvandova M, Berenyiova A, Balis P, Dovinova I, Cacanyiova S. Chronic NOS Inhibition Affects Oxidative State and Antioxidant Response Differently in the Kidneys of Young Normotensive and Hypertensive Rats. Oxid Med Cell Longev. 2019 Nov 22;2019:5349398. doi: 10.1155/2019/5349398. PMID: 31885800; PMCID: PMC6893281.
12. Ogeng'o JA, Gatonga P, Olabu BO, Ongera D. Pattern of hypertensive kidney disease in a black Kenyan population. Cardiology. 2011;120(3):125-9. doi: 10.1159/000334433. Epub 2011 Dec 15. PMID: 22179118.
13. Grunwald JE, Alexander J, Maguire M, Whittock R, Parker C, McWilliams K, Lo JC, Townsend R, Gadegbeku CA, Lash JP, Fink JC, Rahman M, Feldman H, Kusek J, Ojo A; CRIC Study Group. Prevalence of ocular fundus pathology in patients with chronic kidney disease. Clin J Am Soc Nephrol. 2010 May;5(5):867-73. doi: 10.2215/CJN.08271109. Epub 2010 Mar 18. PMID: 20299372; PMCID: PMC2863983.
14. Grunwald JE, Ying GS, Maguire M, Pistilli M, Daniel E, Alexander J, Whittock-Martin R, Parker C, Mohler E, Lo JC, Townsend R, Gadegbeku CA, Lash JP, Fink JC, Rahman M, Feldman H, Kusek JW, Xie D, Coleman M, Keane MG; Chronic Renal Insufficiency Cohort (CRIC) Study Group. Association between retinopathy and cardiovascular disease in patients with chronic kidney disease (from the Chronic Renal Insufficiency Cohort [CRIC] Study). Am J Cardiol. 2012 Jul 15;110(2):246-53. doi: 10.1016/j.amjcard.2012.03.014. Epub 2012 Apr 18. PMID: 22516527; PMCID: PMC3383900.
15. Jepson RE, Syme HM, Vallance C, Elliott J. Plasma asymmetric dimethylarginine, symmetric dimethylarginine, l-arginine, and nitrite/nitrate concentrations in cats with chronic kidney disease and hypertension. J Vet Intern Med. 2008 Mar-Apr;22(2):317-24. doi: 10.1111/j.1939-1676.2008.0075.x. PMID: 18371026.
16. Nokiba H, Takei T, Suto C, Nitta K. Association between Ophthalmological Changes and Cardiovascular Diseases in Patients with Chronic Kidney Disease Undergoing Hemodialysis. J Atheroscler Thromb. 2015;22(12):1248-54. doi: 10.5551/jat.30601. Epub 2015 Jun 30. PMID: 26133318.
17. Ricardo AC, Grunwald JE, Parvathaneni S, Goodin S, Ching A, Lash JP. Retinopathy and CKD as predictors of all-cause and cardiovascular mortality: National Health and Nutrition Examination Survey (NHANES) 1988-1994. Am J Kidney Dis. 2014

Aug;64(2):198-203. doi: 10.1053/j.ajkd.2014.01.437. Epub 2014 Mar 19. PMID: 24656452; PMCID: PMC4111992.
18. Stryjewski TP, Papakostas TD, Eliott D. Multimodal Imaging of Elschnig Spots: A Case of Simultaneous Hypertensive Retinopathy, Choroidopathy, and Neuropathy. Semin Ophthalmol. 2017;32(4):397-399. doi: 10.3109/08820538.2015.1115091. Epub 2016 Apr 15. PMID: 27082847.
19. Arriozola-Rodríguez KJ, Serna-Ojeda JC, Martínez-Hernández VA, Rodríguez-Loaiza JL. Hypertensive Retinopathy as the First Manifestation of Advanced Renal Disease in a Young Patient: Report of a Case. Case Rep Ophthalmol. 2015 Dec 2;6(3):415-9. doi: 10.1159/000442660. PMID: 26955342; PMCID: PMC4777944.
20. Dogan B, Akıdan M, Erol MK, Coban DT, Suren E. Optical coherence tomography angiography findings in malignant hypertensive retinopathy. Arq Bras Oftalmol. 2019 Jan-Feb;82(1):72-77. doi: 10.5935/0004-2749.20190015. PMID: 30652771.