

Suspected immune-complex glomerulonephritis in FIV-infected cats: a clinical case series

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Abstract Feline immunodeficiency virus (FIV) is a retroviral infection that causes progressive immunosuppression in cats and predisposes them to systemic complications, including renal disorders such as immune-complex glomerulonephritis (ICGN). ICGN may develop due to immune-complex deposition within the glomeruli. Early detection of renal involvement in FIV-positive cats is essential for clinical management and prognosis. This report presents two suspected cases of ICGN in FIV-infected domestic cats. Both were tested for retroviral infections using a commercial rapid test kit for FIV/FelV. Clinical examination, serial complete blood count, and serum biochemical analysis were performed, including blood urea nitrogen (BUN), creatinine, and symmetric dimethylarginine (SDMA) to assess renal function. Both cats tested positive for FIV and negative for FelV. The first cat showed normal BUN (33.8 to 29.9 mg/dL) but elevated creatinine (2.47 to 2.6 mg/dL) and increased SDMA (12.5 to 20.6 µg/dL), consistent with early renal dysfunction. The second cat showed progressive and severe deterioration with elevated BUN from 89.91 to 149.84 mg/dL, creatinine from 6.05 to 9.66 mg/dL, and SDMA from 29.9 to 43.3 µg/dL, indicating moderate renal impairment. Both cats also exhibited progressive anemia and leukocytosis, reflecting chronic inflammation and immunosuppression associated with FIV infection. The combination of retroviral infection, hematological changes, and renal biomarker elevation strongly suggests immune complex-mediated renal injury. This case series emphasizes the importance of integrating retroviral testing, hematology, and renal biomarkers, particularly SDMA, for early identification of kidney involvement in FIV-infected cats. Further histopathological confirmation is required to establish the ICGN diagnosis.

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1 Introduction

Feline immunodeficiency virus (FIV) was first reported in 1986 in a cat colony in California [9]. FIV is a retrovirus of the genus *Lentivirus* closely related to human immunodeficiency virus (HIV). However, humans are not susceptible to this virus [9,5]. Five genetically distinct subtypes (A–E) have been identified, showing geographic clustering that is relevant for diagnostic PCR assay design [5,8]. In Indonesia, there are no national prevalence data, but individual case reports of FIV infection have been documented [6].

Globally, the prevalence of FIV infection remains around 4.7% and has been relatively constant since the 1980s. Older, intact male outdoor cats show higher risk due to fighting behaviour and bite transmission [5,8]. Following infection, cats may experience an acute phase with transient fever and lymphadenopathy, a prolonged asymptomatic stage, and finally a terminal phase marked by progressive immunodeficiency and secondary infections [14].

FIV infection typically progresses through three clinical phases. The acute phase is characterized by transient fever, malaise, and generalized lymphadenopathy lasting several weeks to months. The asymptomatic phase may persist for years with minimal viral replication, followed by a terminal phase marked by progressive immunodeficiency, declining CD4+ T lymphocyte counts, and opportunistic infections [5, 4]. Terminal FIV infection represents a stage of progressive immune depletion, followed by viral evasion from immune surveillance and the emergence of clinical manifestations indicate severe immunodeficiency. Weight loss, resembling HIV-associated “wasting syndrome,” is commonly observed [4].

Cats with FIV or FeLV infection were 11-times more likely to be diagnosed with ICGN than non-ICGN [12]. From all types of retroviral as a potential cause of CKD, only FIV plays an important role in the pathophysiology of kidney inflammation [15]. Histopathological studies in naturally and experimentally infected cats have demonstrated mesangial widening, segmental glomerulosclerosis, thickened Bowman’s capsule, and nephrosclerosis [7]. The WSAVA-Renal Standardization Study Group (RSSG) classifies feline glomerular disease into immune-complex glomerulonephritis (ICGN) and non-ICGN categories [12]. Although detailed morphological criteria are limited, this framework facilitates clinical interpretation of feline renal pathology.

Histopathological change of the kidney with mesangioproliferative glomerulonephritis (MPGN) was described as a unique feature in FIV-infected cats [10, 12]. Mesangial widening and segmental sclerosis associated with immune-complex deposition are common findings, emphasizing the role of chronic antigenemia and immune dysregulation in the development of glomerular injury. Cats infected with FIV or FeLV are significantly more likely to develop ICGN than uninfected cats, highlight the clinical importance of early detection and differentiation between ICGN and non-ICGN renal disease [12].

Renal involvement is increasingly recognized as a consequence of chronic immune activation in FIV-infected cats. According to the WSAVA-RSSG classification, feline glomerular diseases are broadly divided into immune-complex glomerulonephritis (ICGN) and non-ICGN [10, 12]. While specific morphological criteria have not been defined for cats, reports indicate that membranous and mesangioproliferative patterns predominate [7, 10]. These renal changes are attributed to immune-complex deposition, complement activation, and subsequent glomerular inflammation.

2 Case Presentation

2.1 Case 1 – Olanje

A domestic cat named Olanje presented on 30 May 2025 with anorexia, limping of the left hind limb, and recurrent flu-like symptoms. The cat lived outdoors and frequently engaged in fights with feral cats. Physical examination revealed edema extending from the left inguinal region to the lower limb, with pain and contusion. No nasal discharge was observed, only mild stertor. Radiographic findings were unremarkable.

A blood test on 6 June 2025 revealed anemia and mild leukocytosis. The cat was hospitalized and treated with amoxicillin and metronidazole. After clinical improvement, Olanje was discharged on 12 June 2025. However, a follow-up blood test on 20 June 2025 showed worsening anemia despite improved appetite and demeanor. The blood smear revealed neutrophils with toxic change, activated lymphocytes, and occasional giant thrombocytes, suggesting possible FIV or FeLV infection.

An FIV/FeLV rapid test performed on 27 June 2025 using PetX™ FIV Ab and FeLV Ag combo test kit yielded FIV-positive and FeLV-negative results. The anemia gradually improved by 18 August 2025, but azotemia with elevated SDMA developed. Ultrasonography indicated indistinct corticomedullary definition, cortical hyperechogenicity, and irregular renal surface—findings consistent with chronic kidney disease (CKD). Comparison over ten days revealed kidney size reduction and decreased inflammation of the bladder and right renal pelvis. These features collectively suggested chronic renal pathology consistent with ICGN and CKD.

Treatment was continued with Renacor™, telmisartan, vitamin B12 with iron supplementation, methylprednisolone, and renal support diet (Royal Canin Renal). Despite transient improvement, the cat escaped home between 9–15 September and subsequently deteriorated, dying approximately three weeks later.

Table 1. Summary of hematology results (June–September 2025)

Date	Hb (g/dL)	RBC ($\times 10^6/\mu\text{L}$)	Hct (%)	WBC ($/\mu\text{L}$)	Platelet ($\times 10^5/\mu\text{L}$)
Jun-06	8.3	5.22	24.1	48,7	0.89
Jun-20	7.6	4.89	23.0	19,7	1.16
Jul-06	9.0	5.49	27.5	13,4	1.10
Aug 18–24	9.6–10.4	5.99–6.56	28.8–31.8	15,800–20,900	0.80–1.66
Sep-04	7.9	4.88	23.7	29,7	2.04

Table 2. Summary of biochemistry results (June–September 2025)

Date	BUN (mg/dL)	Cr (mg/dL)	SDMA ($\mu\text{g/dL}$)	Interpretation
Jul-19	89.91	6.05	29.9	Azotemia
Aug-25	113.22	7.50	32.7	Progressive CKD
Sep-04	149.84	9.66	43.3	IRIS Stage 4

2.2 Case 2 – Moza

Moza, an intact adult male feral cat, was FIV-positive and FeLV-negative based on a test performed on 3 December 2024 using PetX™ FIV Ab and FeLV Ag combo test kit. The cat had chronic rhinitis and marked gingivostomatitis. On 13 June 2025, the condition worsened, showing hypersalivation, entropion, low body condition score (3/9), and mild dehydration despite continued food intake.

Laboratory evaluation on 30 June 2025 showed hypochromic anemia, neutrophilia, and thrombocytopenia, indicating chronic inflammation. A repeat test on 9 July 2025 demonstrated pancytopenia with severe anemia and leukopenia, consistent with advanced bone marrow suppression [4, 1]. Biochemical results revealed SDMA elevation (12.5 to >20.6 µg/dL) with marginal creatinine increase (2.47–2.60 mg/dL), indicating early glomerular dysfunction. Progressive weight loss (from 3.45 kg in December 2024 to 2.6 kg in July 2025) and hypothermia (36.9°C declining to 33.3°C) were noted before death. Treatment included methylprednisolone, Hematodin™, Fluimucil™, amoxicillin, and Ringer’s lactate with dextrose 40% infusion.

Table 3. Summary of hematology results (June and July 2025)

Date	Hb (g/dL)	RBC (×10 ⁶ /µL)	Hct (%)	WBC (/µL)	Platelet (×10 ⁵ /µL)
Jun-30	7.7	5.96	24.0	17,6	1.08
Jul-09	5.3	4.18	17.1	3,9	1.70

Table 4. Summary of biochemistry results (June and July 2025)

Date	BUN (mg/dL)	Cr (mg/dL)	SDMA (µg/dL)	Interpretation
Jun-30	33.8	2.47	12.5	Non-Azotemic, borderline Creatinine
Jul-09	29.9	2.60	20.6	Progressive CKD

3 Discussion

Feline immunodeficiency virus (FIV) infection can result in a wide range of systemic manifestations, including renal lesions secondary to chronic immune activation. In this report, two unrelated FIV-positive, FeLV-negative cats developed progressive hematologic and renal abnormalities consistent with immune-complex glomerulonephritis (ICGN) and chronic kidney disease (CKD). These findings align with previous studies demonstrating the central role of circulating immune complexes (CIC) and glomerular immune deposits in FIV-associated renal pathology [10, 12, 11, 15].

In Olanje, early anemia progressed despite initial clinical improvement. Subsequent azotemia, elevated SDMA, and ultrasonographic changes—such as cortical hyperechogenicity and corticomedullary loss—were consistent with chronic immune-mediated nephropathy. Although histopathologic confirmation was not performed due to the owner’s request, previous reports have described comparable lesions in FIV-infected cats, including mesangioproliferative and membranoproliferative glomerulonephritis with IgG, IgM, and C3 immune deposits [10,12]. These findings from the literature provide theoretical

support for immune-complex-mediated mechanisms underlying the renal changes observed in this case. Similarly, Rossi et al. [12] documented immune complexes along the glomerular basement membrane associated with thickening, confirming the structural consequences of chronic immune injury. These published data strengthen the interpretation that chronic antigenemia in FIV infection can facilitate circulating immune-complex formation, glomerular deposition, and progressive renal damage, these mechanisms likely applicable to the clinical pattern observed in Olanje.

Experimental infection studies further support these mechanisms. Although renal biopsy and urinalysis were not performed in the present cases, findings from Mesquita et al. [7] and others describe membranous and membranoproliferative glomerular lesions, glomerulosclerosis, and amyloidosis consistent with immune-complex mediated disease. Baxter et al. [3] reported that proteinuria often develops before azotemia in FIV-infected cats, reflecting early glomerular involvement that may remain undetected in the absence of urinalysis. Together, these prior investigations provide a theoretical diagnostic framework suggesting that the renal abnormalities in both cats likely represent immune-complex glomerulonephritis (ICGN) within the chronic course of lentiviral infection.

Moza's case represented a terminal stage of lentiviral immune-complex nephropathy, marked by pancytopenia, severe weight loss, and progressive renal failure. The elevation in SDMA prior to creatinine changes suggests early CIC-related glomerular impairment [10, 12, 15]. From a diagnostic and therapeutic perspective, these cases highlight the importance of monitoring FIV-positive cats for CIC-mediated renal disease. The ABCD guidelines [5] and recent clinician updates [14, 8] emphasize lifelong monitoring, including proteinuria, SDMA, and creatinine screening, to detect early immune-mediated renal injury. The feral lifestyle and intact-male status of Moza, consistent with high-risk profiles described by Kurnianto [6], likely contributed to sustained antigenic stimulation and immune-complex formation.

Comparatively, Olanje exhibited a compensated phase of chronic immune-complex glomerulonephritis, while Moza progressed to terminal renal failure. Together, these cases illustrate the spectrum of FIV-associated renal pathology, from chronic CIC formation to irreversible glomerular scarring. The potential evolution toward secondary amyloidosis reported by Asproni et al. [2] and Mesquita et al. [7] further mirrors HIV-associated nephropathy in humans, reinforcing FIV's translational relevance as a natural lentiviral nephropathy model.

Therapeutically, recognizing ICGN as distinct from non-ICGN renal disease is crucial because treatment strategies differ substantially. Non-ICGN or degenerative CKD management focuses on renal diets, blood pressure control, and nephron preservation. In contrast, ICGN requires additional modulation of immune activity using corticosteroids or other immunosuppressive agents to limit immune-complex formation and deposition [11, 13]. FIV-positive cats with CIC-mediated glomerulonephritis may also benefit from anti-inflammatory adjuncts and close monitoring for proteinuria recurrence. The differing therapeutic goals urge the need to identify the underlying pathogenesis to optimize outcomes.

4. Conclusion

These two cases illustrate progressive renal involvement in FIV-positive cats, emphasizing the role of circulating immune complexes in the pathogenesis of immune-complex glomerulonephritis. Distinguishing ICGN from non-ICGN renal disorders is essential, as the management and therapy differ. Understanding these clinical outcomes in feline medicine underscores the value of FIV infection research not only for the advancement of veterinary medicine but also as a model of HIV/AIDS research.

5. References

1. Nahid A., et al. Feline immunodeficiency virus: current insights into pathogenesis, clinical impact, and advances in treatment and vaccine development *Front. Vet. Sci.* (2025). <https://doi.org/10.3389/fvets.2025.1665999>
2. Asproni P., Abramo F., Millanta F., Lorenzi D., Poli A. Amyloidosis in association with spontaneous feline immunodeficiency virus infection. *J. Feline Med. Surg.* **15**(4):300–306 (2012). <https://doi.org/10.1177/1098612X12467997>
3. Baxter K.J., Levy J.K., Edinboro C.H., Vaden S.L., Tompkins M.B. Renal disease in cats infected with feline immunodeficiency virus. *J. Vet. Intern. Med.* **26**:238–243 (2012). <https://doi.org/10.1111/j.1939-1676.2011.00871.x>
4. Bęczkowski P.M., Beatty J.A. Feline immunodeficiency virus: current knowledge and future directions. *Adv. Small Anim. Care.* (2022). <https://doi.org/10.1016/j.yasa.2022.05.007>
5. Hosie M.J., Addie D.D., Belák S., et al. ABCD guidelines on prevention and management: Feline immunodeficiency. *J. Feline Med. Surg.* **11**:575–584 (2009). <https://doi.org/10.1016/j.jfms.2009.05.006>
6. Kurnianto A. Laporan kasus: Feline immunodeficiency virus pada kucing Moi di Surabaya. *J. Kajian Veteriner.* **11**(2):103–113 (2023). <https://doi.org/10.35508/jkv.v11i2.12803>
7. Mesquita L.P., Grandi F., Rocha N.S., Dagli M.L.Z. Aspectos histopatológicos das lesões renais em gatos experimentalmente infectados pelo FIV. *Pesq. Vet. Bras.* **34**:865–872 (2014). <https://doi.org/10.1590/S0100-736X2014000900011>
8. Nehring M., et al. Study of feline immunodeficiency virus prevalence and expert opinions on standards of care. *J. Feline Med. Surg.* (2024). <https://doi.org/10.1177/1098612X241245046>
9. Pedersen N.C., Ho E.W., Brown M.L., Yamamoto J.K. Isolation of a T-lymphotropic virus from domestic cats with an immunodeficiency-like syndrome. *Science.* **235**:790–793 (1987). <https://doi.org/10.1126/science.3643650>
10. Poli A., Tozon N., Guidi G., Pistello M. Renal alterations in feline immunodeficiency virus (FIV)-infected cats: a natural model of lentivirus-induced renal disease. *Viruses.* **4**(9):1372–1389 (2012). <https://doi.org/10.3390/v4091372>
11. Rayhel L.H., Quimby J.M., Cianciolo R.E., et al. Clinicopathologic and pathologic characteristics of feline proteinuric kidney disease. *J. Feline Med. Surg.* **22**(12):1219–1229 (2020). <https://doi.org/10.1177/1098612X20921056>
12. Rossi F., Aresu L., Martini V., et al. Immune-complex glomerulonephritis in cats: a retrospective study based on clinico-pathological data, histopathology and ultrastructural features. *BMC Vet. Res.* **15**:2046 (2019). <https://doi.org/10.1186/s12917-019-2046-y>
13. Sugar N., Chen H., Segev G. Clinical findings, prognostic factors, and outcome of protein-losing nephropathy in cats: a retrospective study. *J. Vet. Intern. Med.* **38**:3111–3118 (2024). <https://doi.org/10.1111/jvim.17240>
14. Westman M.E., Malik R., Norris J.M. Diagnosing FIV and FeLV infection: an update for clinicians. *Aust. Vet. J.* **97**(9):371–371 (2019). <https://doi.org/10.1111/avj.12781>
15. Hartmann, K., Pennisi, M. G. & Dorsch. Infectious Agents in Feline Chronic Kidney Disease: What is the Evidence? *Advances in Small Animal Care.* 1:189–206. <https://doi.org/10.1016/j.yasa.2020.07.013>