

Undiagnosed Stroke and Neurological Manifestations in Chronic Kidney Disease: A Case Series

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ABSTRACT

Chronic kidney disease (CKD) is associated with increased risk of neurological complications, but data describing these conditions and associated neuroimaging findings remain limited, particularly in resource-limited settings. This study aims to describe neurological manifestations and brain imaging abnormalities in hospitalized patients with CKD. We retrospectively analyzed 39 patients with CKD stages II–V who underwent neurological consultation and non-contrast head MSCT (multislice computed tomography) at a tertiary referral hospital in Sukoharjo, Indonesia, during 2025. Data were collected from medical records including demographics, clinical status, neurological complaints, imaging findings, and ICD-10 diagnoses. This case series was reported in accordance with CARE guidelines. The median age was 58 years (IQR 49–67), and 22 patients (56.4%) were male. Most patients had stage V CKD (84.6%), and 29 (74.4%) were on routine hemodialysis. Hypertension was present in 79.5% and diabetes mellitus in 41.0%. Non-contrast head MSCT revealed cerebrovascular pathology in 79.5% of patients, with cerebral infarction in 71.8%. The most common anatomical locations of infarction were the frontal lobe (35.7%), corona radiata (28.6%), and basal ganglia (25.0%). Cerebrovascular disease is highly prevalent among hospitalized CKD patients with neurological symptoms. Cerebral infarction was identified in approximately 71.8% of patients. The high discrepancy between clinical and radiological diagnoses suggests that stroke may be underrecognized in CKD patients, particularly when symptoms are attributed to metabolic encephalopathy. Early neuroimaging should be considered in CKD patients presenting with acute neurological symptoms.

Keywords: CARE, Chronic kidney disease, CT scan, Neurology, Stroke

INTRODUCTION

Chronic kidney disease (CKD) is a major global health challenge, affecting approximately 850 million people. Its impact is most pronounced in low- and middle-income countries. Recent *Global Burden of Disease* (GBD) data indicates that cases of CKD are projected to rise by more than 30% after 2022, and one person dies from CKD every 20 seconds worldwide (Ortiz et al., 2026). The interrelationship between kidney dysfunction and brain disorders is known as the "kidney-brain axis", and it has received significant attention over the past decade (Chagas et al., 2025). Patients with CKD, particularly those with end-stage kidney disease (ESKD), face a substantially elevated risk of neurological complications, including cerebrovascular disease, cognitive impairment, and metabolic encephalopathy (Andrews et al., 2025; Cozza & Boccardi, 2025). Acute brain injury is a known complication of advanced CKD. The underlying mechanisms are complex and may be related to traditional cardiovascular risk factors (i.e., diabetes mellitus and hypertension) (Merlino, Tollitt, et al., 2025; Romadhon, 2020; Wirayudha et al., 2024). It has also been observed that certain factors associated with CKD can lead to brain injury. Neurological injury may be linked to uremia and inflammation (Rivera et al., 2026). Prolonged anemia can also cause brain injury due to a lack of oxygen to the brain (cerebral hypoxia) (Wetmore et al., 2025). The cumulative effect of uremic toxins and CKD potentiates vascular calcification through the actions of indoxyl sulfate and p-cresyl sulfate. Hemodialysis may also cause cerebral hypoperfusion and microemboli (Ogasawara et al., 2025). Brain atrophy (shrinkage of the brain) has also been observed in CKD patients. Patients with CKD are at a higher risk of stroke, and this risk increases as the severity of CKD progresses. The average annual stroke rate is 8.7–10.4 among patients with CKD (stages 4 and 5), while it is 13.5–14.3 among ESKD patients undergoing dialysis (Zhou et al., 2021). A recent meta-analysis of patients undergoing endovascular thrombectomy for acute ischaemic stroke demonstrated that patients with CKD had an increased risk of in-hospital mortality (RR 1.62, 95% CI 1.51–1.77) and unfavorable functional status at 90 days (Ehinder et al., 2025).

Despite these well-documented associations, several knowledge gaps remain. First, the majority of existing studies originate from high-income countries, which differ in demographic profiles and healthcare systems. Second, the correlation between routine clinical diagnoses (based on ICD-10 coding) and detailed neuroradiological imaging findings in real-world practice remains underexplored. Third, the specific anatomical distribution of cerebrovascular lesions in CKD patients warrants further characterization, particularly given that cerebral small vessel disease, characterized by shared microvascular vulnerability of the brain and kidneys, may represent a unifying pathophysiological mechanism (Andrews et al., 2025). This study describes the clinical characteristics and presentations of hospitalized CKD patients requiring neurological consultation, along with the spectrum of non-contrast head MSCT (multislice computed tomography). It further examines discrepancies between clinical and radiological diagnoses and presents a CARE-compliant case series to inform clinical practice in similar settings.

METHODS OF RESEARCH

Study Design and Setting

This study is a retrospective case series designed in accordance with the *CAse REport* (CARE) guidelines (Riley et al., 2017) and was conducted at Ir. Soekarno Regional General Hospital, Sukoharjo,

Indonesia, a tertiary referral center for kidney diseases with an active hemodialysis unit managing hundreds of patients.

Sample

All adult patients (aged ≥ 18 years) with a diagnosis of chronic kidney disease (CKD) who underwent neurological consultation, non-contrast head MSCT (multislice computed tomography) examination, and had complete medical records were consecutively enrolled. Incomplete medical records served as the exclusion criterion. A total of 39 patients met the inclusion criteria and were included in the final analysis.

Data Collection

The following information was abstracted from the medical records: patient demographics and clinical status (age, sex, CKD stage, hemodialysis status, length of hospital stay, and comorbidities such as hypertension, diabetes mellitus, anemia, heart disease, and prior history of stroke), chief complaint and neurological symptoms, head MSCT diagnostic report, discharge diagnosis, ICD-10 codes, and neurological diagnoses.

Imaging Classification

Based on the findings, the MSCT results were classified into the following categories: cerebral infarction (hypodense lesions indicative of ischemic stroke), cerebral atrophy (widened sulci or prominent gyri), cerebral edema, and intracranial hemorrhage (hyperdense lesions indicative of blood clots). The anatomical locations of the lesions were also recorded, including the frontal lobe, parietal lobe, corona radiata, thalamus, basal ganglia, brainstem, cerebellum, and corpus callosum.

Statistical Analysis

Since the objective of this research was to summarize the status of participants, data were synthesized in a tabular format. Continuous data are presented as median and interquartile range (IQR). Categorical data are presented as frequencies and percentages.

Ethical Considerations

The authors adhered to the standards set forth in the World Medical Association's Declaration of Helsinki and obtained ethical approval (number: XX/KEPK/2026) from the Research Ethics Committee of Dr. Moewardi General National Hospital to conduct this study. As the authors conducted a retrospective analysis of anonymized data, the ethical review board waived the requirement for the authors to obtain informed consent from study subjects. To maintain confidentiality, the authors anonymized study subjects' data prior to analysis.

RESULT

Participant Characteristics

During the study period (January 1, 2025, to December 31, 2025), 39 patients with CKD received a neurological consultation and underwent non-contrast head MSCT. Data for these subjects are described below.

Table 1. Demographic and Clinical Characteristics of the Study Population

Characteristic	Value
Total patients, n	39
Sex, n (%)	
Male	22 (56.4)
Female	17 (43.6)
Age (years), median (IQR)	58 (49–67)
Length of hospital stay (days), median (IQR)	8 (5–14)
CKD stage, n (%)	
Stage II	1 (2.6)
Stage III	3 (7.7)
Stage IV	2 (5.1)
Stage V	33 (84.6)
On routine hemodialysis, n (%)	29 (74.4)
Comorbidities, n (%)	
Hypertension	31 (79.5)
Diabetes mellitus	16 (41.0)
Anemia	14 (35.9)
Heart disease (IHD/HHD)	11 (28.2)
Prior history of stroke	8 (20.5)
Number of laboratory results, median (IQR)	6 (2–8)

*IQR: Interquartile range (25th to 75th percentile)

Source: Primary data, 2025

Approximately 56.4% of the participants in this study were men, with a median age of 58 years. The majority (84.6%) had Stage V CKD, and 74.4% were undergoing routine hemodialysis. Of the participants, 79.5% had hypertension, and 41.0% had type 2 diabetes mellitus.

Clinical Presentation

Most (48.7% of the subjects) of the consultations were for altered level of consciousness. Others (35.9% of the subjects) had focal weakness and hemiparesis. Other frequent complaints included dizziness/vertigo (25.6%), nausea/vomiting (23.1%), seizures (15.4%), and headache (12.8%).

Table 2. Clinical Presentation at Neurological Consultation

Primary Complaint	Frequency (n)	Percentage (%)
Decreased level of consciousness	19	48.7
Focal weakness/hemiparesis	14	35.9
Dizziness/vertigo	10	25.6
Nausea/vomiting	9	23.1
Seizures	6	15.4

Headache	5	12.8
Speech disturbance	3	7.7

Source: Primary data, 2025

Note: The sum of the percentages exceeds 100% because patients may have more than one chief complaint.

Neurological Diagnoses Based on ICD-10 Coding

The most common neurological diagnosis at discharge (in 51.3% of patients) was cerebral infarction, unspecified (I63.9). The second most common diagnosis at discharge (in 30.8% of patients) was encephalopathy, unspecified (G93.4).

Table 3. Primary Neurological Diagnoses Based on ICD-10 Codes

ICD-10 Code	Diagnosis Description	Frequency (n)	Percentage (%)
I63.9	Cerebral infarction, unspecified	20	51.3
G93.4	Encephalopathy, unspecified	12	30.8
I69.4	Sequelae of stroke, unspecified	2	5.1
G45.9	Transient ischemic attack, unspecified	2	5.1
G93.6	Cerebral edema	1	2.6
R56.8	Seizure, unspecified	1	2.6
R40.2	Coma, unspecified	1	2.6
Total		39	100

Source: Primary data, 2025

Head MSCT Imaging Findings

Non-contrast head MSCT revealed a range of abnormalities (Table 4). Cerebrovascular pathology was identified in 79.5% of patients. The most frequent finding was cerebral infarction, presenting in both acute and chronic forms, which accounted for 71.8% of cases. Table 5 shows that cerebral infarction was evident in 28 of the 39 patients who underwent head MSCT.

Table 4. Head MSCT Imaging Findings

Imaging Finding	Frequency (n)	Percentage (%)
Any cerebrovascular pathology	31	79.5
Cerebral infarction (acute/chronic)	28	71.8
Cerebral atrophy	17	43.6
Cerebral edema	6	15.4
Intracranial hemorrhage	1	2.6
Normal findings	3	7.7

Source: Primary data, 2025

Note: The sum of the percentages exceeds 100% because patients may have more than one imaging finding.

Table 5. Anatomical Distribution of Cerebral Infarction

Location	Frequency (n)	Percentage of infarct patients (%)
Frontal lobe	10	35.7
Corona radiata	8	28.6
Basal ganglia	7	25.0
Parietal lobe	6	21.4
Cerebellum	5	17.9
Thalamus	4	14.3
Corpus callosum	2	7.1
Brainstem (pons/mesencephalon)	2	7.1

Source: Primary data, 2025

Note: The percentages for anatomical distribution exceed 100% because each patient could have infarcts at multiple sites at once.

Concordance Between Clinical Diagnosis and Imaging Findings

The finding of this case series was a notable discrepancy between clinical diagnosis and imaging findings (Table 6). Although MSCT identified cerebral infarction in 28 patients (71.8%), only 20 patients (51.3%) carried a clinical diagnosis of cerebral infarction. Among the 12 patients clinically diagnosed with encephalopathy (G93.4), imaging revealed evidence of cerebral infarction in 7 patients (58.3%) that had not been apparent in their primary neurological diagnosis.

Table 6. Cross Tabulation of Clinical Diagnosis vs. Imaging Findings of Infarction

Clinical Diagnosis	MSCT Findings	
	Infarct Present	No Infarct
Cerebral Infarction, unspecified (I63.9)	19	1*
Encephalopathy, unspecified (G93.4)	7	5
Other	2	5
Total	28	11

Source: Primary data, 2025.

Footnote: *One patient was given a clinical diagnosis of infarction without confirmatory MSCT findings. This case probably reflects either a transient ischemic attack (TIA) or a clinical diagnosis established on symptomatic grounds without radiological correlation.

DISCUSSION

This case series depicts patients with CKD (chronic kidney disease) who were hospitalized and describes neurological complications. The key points of the study are: (i) about 80% of the patients in the study who underwent neuroimaging had evidence of cerebrovascular disease, and of those patients, about 71.8% had cerebral infarctions, and (ii) about 60% of the patients who were diagnosed with encephalopathy based on clinical judgment had infarcts on neuroimaging. Other neurological issues observed in the patients included in this study were ataxia, myoclonus, and infarcts in the cerebellar hemispheres. The study also revealed the presence of infarcts in the brainstem, brainstem auditory radiations, corona radiata, and frontal lobes.

Prevalence and Patterns of Cerebrovascular Disease

This study found a remarkably high rate of cerebral infarction (71.8%), highlighting the substantial burden of both overt and covert cerebrovascular injury in advanced CKD. Recent evidence indicated that patients with CKD not only experience higher rates of clinical stroke but also carry a greater burden of silent brain infarcts, which are often incidentally detected during neuroimaging (He & Gillespie, 2025). A recent study by the UK Biobank prospectively followed over 43,000 patients aged 40–69 years and found renal dysfunction, defined by abnormal blood tests for blood urea nitrogen (BUN) and estimated glomerular filtration rate (eGFR), was associated with increased volume of white matter lesions and more focal brain atrophy (Nash et al., 2023). There could be several reasons for the high number of patients with kidney impairment in our study. The symptoms in our patients were more severe, which likely prompted the brain MSCT; we observed both recent and old cerebral infarcts, and the majority of patients (84.6%) were in the most severe stage of kidney impairment (CKD Stage V). This risk especially increases in patients who have progressed to kidney failure and require dialysis (He & Gillespie, 2025).

The changes observed in the infarcted areas, particularly in the corona radiata and subcortical regions, closely resemble those seen in CKD-associated cerebrovascular disease (CSVD). CSVD is believed to be linked to the chronic inflammatory changes, endothelial dysfunction, and accumulation of uremic toxins associated with CKD (Capasso et al., 2025). The so-called kidney-brain axis describes CKD and CSVD as associated conditions that occur simultaneously. Recent studies indicate that CKD is a factor that accelerates CSVD, particularly concerning the kidney-brain axis (Ito & Mori, 2025). It is generally understood that CKD leads to cerebral vascular injury (damage to the brain's blood vessels), specifically cerebral microhemorrhages (bleeding from small blood vessels in the brain) (Yang et al., 2025). This study highlights how commonly stroke is misunderstood and/or misdiagnosed in the setting of CKD.

Encephalopathy as a Masking Diagnosis for Stroke

The main finding of this study shows there was a high number of strokes that were missed. The diagnosis of stroke, however, often is not made due to a variety of reasons. Among the 12 patients in this study diagnosed with encephalopathy, 7 (58.3%) exhibited symptoms of stroke. In patients with CKD, particularly those presenting with neurological symptoms, these symptoms are often attributed to metabolic imbalances. The authors of this study believe this occurs because often a diagnosis of stroke is not made; therefore, other causes for the patients' symptoms are explored. Small cerebrovascular accidents in patients with chronic kidney disease (CKD), particularly when asymptomatic, can be detected on various types of scans and are considered a form of end-organ damage (Capasso et al., 2025). The problem with ignoring these small strokes is that often they can be the precursor to larger strokes and other cerebrovascular accidents (Cozza & Boccardi, 2025). A large global collaborative analysis involving over 530,000 CKD patients confirmed that the coexistence of cognitive impairment and CKD is a decisive risk factor for higher mortality and increased adverse outcomes, including cerebrovascular disease and encephalopathy (Merlino, Rainone, et al., 2025).

There are important clinical implications for these undocumented lesions. For example, stroke may be initially misdiagnosed as encephalopathy. Consequently, various methods for stroke prevention and treatment may be delayed. Misidentification can also impact stroke research, particularly when strokes go undetected in administrative data. It is also necessary to review the term "uremic encephalopathy". Uremic toxins may be caused by other factors. Emerging data suggests that brain and other organ injuries caused

by small vessel disease may be the primary causes of symptoms in CKD patients. Multiple mechanisms can contribute to brain injuries, including injury to small blood vessels and stroke, micro-inflammation, and disruptions to neuroprotective pathways and the glymphatic system (Capasso et al., 2025). These injuries can also contribute to strokes and other neurological injuries.

Cerebral Atrophy and CKD

Cerebral atrophy occurred in 43.6% of the patients, which supports the concept of brain aging in CKD. Nash and his colleagues report that low eGFR and high rate of urinary albumin excretion are associated with reduced brain volume and greater brain atrophy. These associations are observed even after accounting for traditional cardiovascular risk (Nash et al., 2023). Frontal and temporal lobe atrophy, seen on brain imaging of CKD patients, is associated with cognitive impairment, particularly with respect to executive function and attention. This type of brain atrophy is dissimilar to that of neurodegenerative disease, including Alzheimer's disease. This indicates that the decline in cognitive function associated with CKD is primarily caused by problems related to blood vessels (Cozza & Boccardi, 2025).

The mechanisms underlying accelerated brain atrophy in CKD are multifactorial and interrelated. Potential contributors include chronic hypertension and diabetes leading to neuronal loss; hemodialysis-related cerebral hypoperfusion; vascular damage resulting in white matter rarefaction; and the direct neurotoxic effects of accumulated uremic toxins. A 2026 review on cerebral neurodegeneration in hemodialysis patients highlighted that brain damage in end-stage CKD is considered a multicomponent process associated with the impact of cerebrovascular, neurodegenerative, and numerous dysmetabolic factors, with special attention given to neurodegeneration mechanisms mediated by the activation of microglia and astrocytes (Khrulev et al., 2026). Longitudinal studies have demonstrated that brain volume loss in dialysis patients progresses more rapidly than in age-matched controls, with estimated brain age being 5-10 years older than chronological age. Interestingly, recent evidence indicates that successful kidney transplantation can normalize cerebral blood flow and improve white matter integrity, suggesting that some of these changes may be reversible with restoration of kidney function (Cozza & Boccardi, 2025).

Hemorrhagic Stroke in CKD

Only one patient (2.6%) had intracranial hemorrhage, a proportion lower than expected given that hemorrhagic stroke accounts for approximately 10-20% of all strokes in Asian populations and CKD is known to increase hemorrhagic risk. A 2025 comprehensive review on intracerebral hemorrhage confirmed that chronic kidney disease is recognized as a non-modifiable risk factor for ICH, with the incidence being higher in Asian populations (18-24%) compared to Western countries (8-15%) (Lee, 2025). However, a recent pooled analysis from the MICON international collaboration involving over 11,000 patients found that while impaired kidney function ($\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$) was associated with a 33% higher risk of recurrent ischemic stroke, it was not significantly associated with intracranial hemorrhage risk (adjusted HR 1.07, 95% CI 0.70-1.60) (Molad et al., 2025). This suggests that the relationship between CKD and hemorrhagic stroke may be more complex than previously thought, possibly modified by factors such as blood pressure control, anticoagulant use, and the presence of cerebral microbleeds.

The single case of intrapontine hemorrhage observed in our series highlights the particular vulnerability of penetrating arteries to hypertensive injury in CKD patients with uncontrolled blood pressure. Experimental studies have demonstrated that CKD promotes cerebral microhemorrhage by

damaging the blood-brain barrier and activating microglia via independent mechanisms that are not related to blood pressure elevation. The gut-derived uremic toxins such as indoxyl sulfate, p-cresyl sulfate, and trimethylamine-N-oxide contribute to the disruption of the endothelial barrier integrity, thus increasing the bleeding tendency of patients with advanced CKD (Yang et al., 2025). The presence of cerebral microbleeds is associated with an increased risk of future intracerebral hemorrhage, particularly in patients undergoing antithrombotic therapy (Molad et al., 2025). These findings highlight the need for strict blood pressure control and careful evaluation of bleeding risk before initiating antithrombotic therapy in this vulnerable population, particularly since CKD patients with cognitive impairment are often prescribed anticoagulants and antiplatelet agents (Merlino, Rainone, et al., 2025).

Implications for Clinical Practice

The results of this study have several important considerations for clinical decision making and patient care. When patients with CKD suddenly exhibit acute neurological symptoms, stroke should be suspected, even if the symptoms point to metabolic encephalopathy. Given the high prevalence of radiologically detected stroke in this population, early neuroimaging should be considered a priority rather than a last resort. The strong association between CKD and posterior reversible encephalopathy syndrome (PRES), which demonstrates a dose-response relationship with CKD stage progression further emphasizes the need for heightened clinical vigilance in this population (He & Gillespie, 2025). The use of detailed diagnostic coding that specifies both the type and location of stroke may contribute to better clinical communication, research quality, and improved healthcare delivery. Nephrology departments should consider incorporating routine neurological assessments or adopting a collaborative approach, particularly for patients who do not exhibit obvious neurological symptoms.

The recognition of CKD as being associated with a specific pattern of cerebrovascular damage, mainly consisting of small vessel disease affecting the frontal subcortical region, should guide the diagnostic and therapeutic approaches. Comprehensive management of vascular risk factors, including hypertension, diabetes mellitus, and dyslipidemia, remains fundamental for reducing the risk of recurrent cerebrovascular events. As impaired renal function has been linked to cerebral microbleeds and recurrent stroke, targeted secondary prevention strategies are particularly important in these patients (Molad et al., 2025). Growing evidence indicates that other interventions, such as renin-angiotensin system inhibitors, anemia management, and even exercise therapy, may help preserve cognitive function in CKD patients (Cozza & Boccardi, 2025). Given the established association between cognitive impairment, higher mortality, and increased risk of adverse outcomes, routine cognitive screening may represent an important component of care for patients with CKD (Merlino, Rainone, et al., 2025). Future research is needed to develop neuroimaging monitoring and evaluate tailored interventions that may reduce the burden of severe cerebrovascular events in this patient population.

CONCLUSIONS

This case series reports on neurologically symptomatic patients with chronic kidney disease (CKD) to understand how frequently cerebrovascular disease occurs in this patient population. Cerebral infarction was identified in 71.8% of the patients who underwent non-contrast head MSCT. In several cases, the patients' encephalopathy was classified as "unspecified," which hindered the classification of the stroke and

its subsequent treatment. Head MSCT showed infarcts in the frontal lobe, the corona radiata, and the basal ganglia. The infarcts led the authors to conclude that cerebral small vessel disease connected the patients' renal disease and brain disease. The authors suggest that when considering potential causes of stroke, acute cerebral infarction should be included in the differential diagnosis for patients with CKD. They recommend that if a patient with CKD is unable to communicate or describe their neurological symptoms, neuroimaging should be performed to check for cerebral infarction. As CKD is a widespread condition, the effects of cerebral infarction that are associated with CKD should be investigated.

Future Directions

This study highlights several avenues for future research. To understand the diverse clinical presentations and progression patterns of cerebrovascular disease (disorders affecting the brain's blood vessels) in patients with chronic kidney disease, there is a need for research that involves long-term monitoring of patient cohorts and utilizes both standard neurological function tests and brain imaging. Further, advanced brain imaging, MRI and its variants, e.g., DWI and SWI, may be useful to distinguish between vascular and nonvascular causes of brain changes. Research that examines the effect of aggressive lowering of blood pressure, the use of antiplatelet agents, and other interventions on the risk of stroke is also needed. Research is also needed to identify the reasons behind the under-recognition of stroke in patients with chronic kidney disease.

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