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Trajectories of Serum Creatinine and Associated Factors in an Outpatient Chronic Kidney Disease Cohort in Burkina Faso: A Latent Class Analysis

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Abstract

Background: Chronic kidney disease (CKD) progression varies considerably between individuals. Identifying these trajectories in sub-Saharan Africa could significantly improve patient management. **Objective**: To identify CKD progression trajectories and analyze associated factors in a cohort of patients in Burkina Faso. **Patients and Methods:** We conducted a retrospective study including 123 patients aged ≥ 18 years, followed at a nephrology outpatient clinic between January 1, 2014, and December 31, 2017. Renal function was modeled using serial serum creatinine measurements. We used a latent class model to identify serum creatinine trajectories. The optimal model was selected based on the lowest Akaike Information Criterion (AIC) and clinical interpretability. Multivariable logistic regression analysis was used to identify factors associated with each trajectory. **Results:** The mean age was 55.5 ± 15.9 years, with a sex ratio of 1.9. Most patients (65.9%) had low socioeconomic status. Three distinct trajectories were identified: rapid progression (Class 1, 15.45%), stable (Class 2, 31.7%), and moderate progression (Class 3, 52.85%). Anemia was a strong independent predictor across all classes. Urban residents had a lower probability of belonging to the moderate progression class compared to the stable Class. Chronic NSAID use specifically increased the odds of moderate progression. The relationship with age was non-linear, with advanced age being linked to a stable trajectory rather than rapid decline. **Conclusion:** CKD progression in this setting is characterized by heterogeneous trajectories, shaped by clinical and socioeconomic factors. These findings are the first step towards developing context-specific predictive tools that will enable the personalized, risk-stratified management of CKD in West Africa.

Keywords

Chronic Kidney Disease, Progression, Serum Creatinine Trajectories, Latent Class Analysis, Burkina Faso, Sub-Saharan Africa

1. Introduction

Chronic kidney disease (CKD) is a major public health concern, affecting around 13.4% of people worldwide [1] [2]. The progression of CKD is of particular concern in resource-limited countries. This is linked to the concurrent rise in the prevalence of hypertension, diabetes and obesity, an aging population, and the high prevalence of communicable diseases [1] [3] [4]. In West Africa, the prevalence of CKD is estimated at 16.5%, which ranks among the highest rates worldwide [3]. This may be partly explained by genetic factors, particularly the high prevalence of APOL1 gene variants that predispose individuals to CKD [1].

CKD progresses through five stages of increasing severity. At end-stage renal disease (ESRD), renal replacement therapy becomes necessary for patient survival [2]. However, the rate of progression varies significantly between individuals. Consequently, patients with comparable CKD stages may follow distinct evolutionary trajectories: some maintain stable renal function for prolonged periods, while others experience accelerated decline [5]. Rapid CKD progression is associated with factors such as advanced age, male sex, the presence of proteinuria, and a low baseline estimated glomerular filtration rate (eGFR) [6]-[8]. The progression of CKD is accompanied by an increased prevalence of complications, leading to higher healthcare costs [2]. CKD is responsible for approximately 2.3 million deaths worldwide and is projected to become the fifth leading cause of death by 2040 [1]. Access to renal replacement therapy remains limited in Africa, where only 9% - 16% of eligible patients receive it [9]. Early interventions are crucial in reducing the burden of this disease before it reaches the terminal stage, through targeted prevention and management strategies [4].

Serum creatinine is the most commonly used biomarker for routine assessment of renal function. It is also a predictive factor for CKD progression [10]. Studying serum creatinine trajectories over time provides valuable insight for monitoring CKD progression, with results comparable to those obtained by eGFR trajectory analysis [11].

However, serum creatinine remains an imperfect GFR marker, influenced by physiological factors (muscle mass, dietary protein intake) and analytical factors [12]. Two main assay methods exist: the Jaffe method and the enzymatic method.

Regardless of technique, calibration must be standardized, ideally by reference to isotope dilution mass spectrometry (IDMS), the gold standard [12]. The Jaffe method, although less expensive, is subject to interferences (glucose, bilirubin, uric acid, medications) and tends to overestimate serum creatinine, leading to eGFR underestimation compared to the enzymatic method [12] [13].

In our study context, serum creatinine assays were performed using the Jaffe method, without IDMS standardization, and in different laboratories. Given this potential bias, we chose to study serum creatinine trajectories directly.

An increase in serum creatinine over time is a key indicator of CKD progression. Identifying subgroups of patients with similar evolutionary profiles is critical for personalizing care and improving prognosis. Latent class analysis (LCA) provides a useful methodological framework for this purpose. This statistical approach enables the identification of homogeneous subgroups that are not directly observable, termed "latent classes". This approach models diversity in individual trajectories by inferring each individual's class membership based on their observed evolutionary profile [5] [14].

To our knowledge, no previous study has characterized CKD progression trajectories by analyzing serum creatinine in Burkina Faso. We therefore conducted the present study, which aimed to identify serum creatinine trajectories and analyze associated factors among patients attending a nephrology outpatient clinic.

2. Patients and Methods

2.1. Study Design and Population

This retrospective cohort study was conducted in the Nephrology and Hemodialysis Department of the Yalgado Ouedraogo University Hospital, Ouagadougou, from January 1, 2014, to December 31, 2017. All outpatients aged 18 years or older who were treated for CKD during this period and underwent regular follow-ups at least every six months for at least 12 months were included, provided that they had at least three serum creatinine measurements available (at initiation, after six months and after 12 months). Patients who were followed up until the end of the study (36 months) had at least seven creatinine measurements (at baseline and at six, 12, 18, 24, 30, and 36 months). Patients with incomplete or unusable medical records, those already undergoing hemodialysis at the time of recruitment, and those who had undergone a kidney transplant were excluded. Data were extracted from medical records using a standardized collection form.

The collected variables included sociodemographic parameters (age, sex, geographical origin, socioeconomic status and education level), clinical parameters (etiology of nephropathy, non-steroidal anti-inflammatory drug [NSAID] use, herbal medicine use, blood pressure, weight, height and body mass index [BMI]) and laboratory parameters (blood urea nitrogen, serum creatinine, serum uric acid, sodium, potassium, calcium, phosphate, fasting blood glucose, complete blood count and 24-hour proteinuria). Data on lifestyle habits (alcohol and tobacco consumption) were available for only a few patients and were not analyzed.

2.2. Definitions

Socioeconomic status was estimated based on occupation and categorized as follows:

- High for senior public and private sector executives, business owners, and military officers;
- Middle for civil servants, private sector employees with secondary education, small business owners, and non-commissioned officers; and
- Low for manual labourers, farmers, and unemployed persons.

NSAID use was defined as the regular use of NSAIDs for at least 90 days. Herbal medicine use was defined as the use of herbal decoctions or powders for disease prevention or treatment, regardless of duration or frequency, as documented in medical records.

CKD was defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) initiative [15]. Serum creatinine was measured using the Jaffe method. The estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease (MDRD) study equation: eGFR = 186.6 × (serum creatinine/88.4) − 1.154 × age − 0.203 × (0.742 if female) − (1.21 if black), where serum creatinine is in μmol/L and age is in years [16]. CKD staging followed the KDIGO classification [15]: Stage 1 (eGFR ≥ 90 mL/min/1.73m^2), Stage 2 (eGFR 60 to 89 mL/min/1.73m^2), Stage 3a (eGFR 45 to 59 mL/min/1.73m^2), Stage 3b (eGFR 30 to 44 mL/min/1.73m^2), Stage 4 (eGFR 15 to 29 mL/min/1.73m^2), and Stage 5 (eGFR < 15 mL/min/1.73m^2).

In the absence of a renal biopsy, the etiology of CKD was inferred based on clinical and paraclinical arguments:

- Hypertensive nephropathy: long-standing hypertension (≥5 years); low-output proteinuria (less than 1 g per 24 hours); absence of hematuria; symmetrical kidneys on ultrasound; and hypertensive retinopathy.
- Chronic glomerulonephritis: proteinuria of more than 1.5 g/24 hours, hematuria (as indicated by a dipstick test or the presence of more than five red blood cells per high-power field), symmetrical kidneys that appear normal on ultrasound, and exclusion of secondary causes such as diabetes, hepatitis B or C, HIV or lupus.
- Chronic tubulointerstitial nephropathy was defined by the presence of persistent leukocyturia, with low-grade proteinuria of less than 1 g per 24 hours and kidneys with irregular contours on ultrasound.
- Diabetic nephropathy is diagnosed in patients with type 1 diabetes for ≥10 years or type 2 diabetes for ≥5 years, persistent albuminuria and diabetic retinopathy, and no hematuria.
- HIV-associated nephropathy (HIVAN): positive HIV serology, nephrotic-range proteinuria (>3 g/24hours), enlarged hyperechoic kidneys on ultrasound and no other secondary glomerular cause (e.g. hepatitis B/C, diabetes). This can be diagnosed without a renal biopsy.
- Autosomal dominant polycystic kidney disease (ADPKD) was diagnosed using Pei's criteria [17].

BMI was calculated as weight (kg) divided by height squared (m^2). Categories were defined as underweight (BMI < 18.5 kg/m^2), normal weight (BMI 18.5 to 24.9 kg/m^2), overweight (BMI 25 to 29.9 kg/m^2), and obesity (BMI ≥ 30 kg/m^2) [18].

Hypertension was defined as systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg, or the use of antihypertensive medication [19]. Diabetes mellitus was defined as a fasting blood glucose level ≥ 1.26 g/L on two separate occasions or a random plasma glucose ≥ 2 g/L (11.1 mmol/L) in the presence of suggestive signs and/or the use of antidiabetic medication [20]. Significant proteinuria was defined as 24-hour urinary protein ≥0.5 g/24h in the absence of a urinary tract infection. Anemia was defined as a hemoglobin level < 12 g/dL in women and <13 g/dL in men [21].

2.3. Statistical Analysis

Analyses were performed using R (version 3.5.2) with the lcmm package. Continuous variables were summarized as the mean ± standard deviation (sd) and categorical variables as counts and frequencies.

A latent class model was used to study potential trajectories of serum creatinine and their predictors. The time scale was defined in months (range: 0 - 36 months). Due to its non-Gaussian distribution, serum creatinine trajectories were modelled using splines with three knots located at the 33rd, 66th and 90th percentiles of the measurement time distribution, corresponding approximately to 12, 24 and 30 months. Random effects were specified for the intercept and linear time slope with an unstructured covariance matrix.

Initial univariate analysis identified factors with p-value < 0.15, which were included in a multivariate model. A backward stepwise selection procedure (retention threshold: $p < 0.05$) was then employed to determine the final model. BMI was included in the model for physiological reasons, regardless of statistical significance, due to its correlation with muscle mass. The optimal number of trajectory classes was determined by comparing models with two to five classes, based on the Akaike information criterion (AIC), with the lowest AIC value being prioritized, as well as clinical interpretability and a minimum class proportion of 10%. Class characteristics were described using posterior membership probabilities.

3. Results

Between January 1, 2014, and December 31, 2017, 123 patients were included at their first consultation for chronic kidney disease (CKD). All 123 patients were seen at the 6- and 12-month follow-up visits. At 18 and 24 months, 74 and 72 patients were seen, respectively. At 30 and 36 months, 57 patients (46.3%) remained in follow-up (at least biannual check-ups), while 66 patients (53.7%) were lost to follow-up.

3.1. Baseline Characteristics of the Cohort

The mean age was 55.5 ± 15.9 years (range: 18 - 86), with a male predominance of

65% (sex ratio 1.86). Most patients had a low socioeconomic status (65.9%), and 39.8% had no formal education. Hypertensive nephropathy was the leading cause of CKD (52%). The mean eGFR was 30.4 ± 18 mL/min/1.73m^2, and 93.5% of patients (n = 115) had CKD stages 3 to 5. Table 1 compares the baseline characteristics of patients who were followed up for 36 months (n = 57) with those who were lost to follow-up (n = 66). Patients lost to follow-up had a significantly higher mean baseline serum creatinine level (447.0 vs 279.4 µmol/L; p = 0.004) and were more frequently at CKD stage 5 (73.3% vs 26.7%; p = 0.027). No other significant differences were observed between the two groups.

Table 1. Characteristics of the study population according to the follow-up (n = 123).

Parameters	All patients (n = 123)	Follow-up		p-value
		Completed 36 months (at least 7 visits) (n = 57)	Lost (between 3 and 6 visits) (n = 66)	
Age	55.5 ± 15.9	55.1 ± 18.2	55.8 ± 13.8	0.826
Age categories (years)				
≥60	56 (45.5)	28 (49.1)	28 (42.4)	0.457
<60	67 (54.5)	29 (50.9)	38 (57.6)	
Sex				
Male	80 (65)	36 (63.2)	44 (66.7)	0.684
Female	43 (35)	21 (36.8)	22 (33.3)	
Education level				
No formal education	49 (39.8)	17 (29.8)	32 (48.5)	0.101
Primary school	27 (22)	13 (22.8)	14 (21.2)	
Secondary school	39 (31.7)	21 (36.8)	18 (27.3)	
University	8 (6.5)	6 (10.5)	2 (3)	
Income level				
Low	81 (65.9)	36 (63.2)	45 (68.2)	0.503
Middle	41 (33.3)	20 (35.1)	21 (31.8)	
High	1 (0.8)	1 (1.8)	0 (0)	
Geographic origin				
Urban	68 (55.3)	31 (54.4)	37 (56.1)	0.944
Semi-urban	37 (30.1)	17 (29.8)	20 (30.3)	
Rural	18 (14.6)	9 (15.8)	9 (13.6)	
Comorbidities				
Hypertension	95 (77.2)	44 (77.2)	51 (77.3)	0.992
NSAIDs use	41 (33.3)	15 (26.3)	26 (39.4)	0.125
Herbal medicine use	35 (28.5)	19 (33.3)	16 (24.2)	0.265

Continued

Diabetes mellitus	26 (21.1)	16 (28.1)	10 (15.2)	0.08
Gout	15 (12.2)	6 (10.5)	9 (13.6)	0.599
Urogenital schistosomiasis	4 (3.3)	3 (5.3)	1 (1.5)	0.256
Primary disease				
Hypertensive nephropathy	64 (52)	30 (52.6)	34 (51.5)	0.9016
Chronic glomerulonephritis	24 (19.5)	9 (15.8)	15 (22.7)	0.3329
Diabetic nephropathy	22 (17.9)	12 (21.1)	10 (15.2)	0.3944
Chronic tubulointerstitial nephropathy	9 (7.4)	4 (7)	5 (7.6)	>0.9999
HIV-associated nephropathy	3 (2.4)	1 (1.8)	2 (3)	>0.9999
Polycystic kidney disease	1 (0.8)	1 (1.8)	0 (0)	>0.9999
Serum creatinine (µmol/L)	369.3 ± 333.7	279.4 ± 204.1	447 ± 399.8	0.004
eGFR (mL/min/1.73m²)	30.4 ± 18	32.7 ± 15.2	28.4 ± 20	0.188
CKD stage at inclusion				
1	0	0	0	
2	8 (6.5)	2 (3.5)	6 (9.1)	0.027
3a	16 (13)	10 (17.5)	6 (9.1)	
3b	36 (29.3)	18 (31.6)	18 (27.3)	
4	33 (26.8)	19 (33.3)	14 (21.2)	
5	30 (24.4)	8 (14)	22 (33.3)	
24-hour proteinuria (g/24h)	1.2 ± 1.5	1.2 ± 1.8	1.1 ± 1.2	0.607

CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate; HIV: human immunodeficiency virus; NSAIDs: non-steroidal anti-inflammatory drugs. Values are presented as mean ± standard deviation (sd) or number (percentage).

3.2. Evolution of Serum Creatinine during Follow-Up

Figure 1 shows the evolution of serum creatinine levels for each individual over

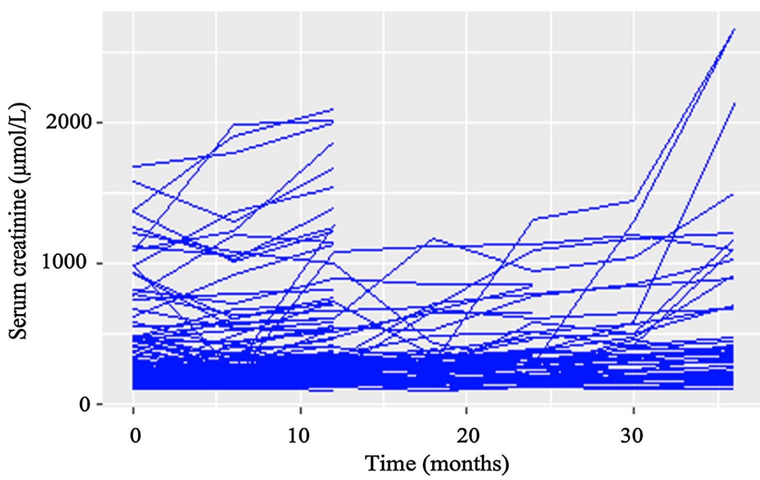

Figure 1. Individual serum creatinine trajectories over 36 months of follow-up.

36 months. The mean serum creatinine level increased from 369.3 ± 333.7 µmol/L at baseline to 524.7 ± 569.6 µmol/L after 36 months (Supplementary Table S1).

3.3. Factors Associated with Creatinine Evolution in Univariate Analysis

Five factors were significantly associated with the creatinine trajectory ($p < 0.05$): advanced age (coefficient = −0.029, p = 0.022); urban origin (coefficient = −1.859, p < 0.001); history of urogenital schistosomiasis (coefficient = −3.600, p < 0.001); NSAIDs use (coefficient = 1.007, p = 0.009); and the presence of anemia (coefficient = 3.131, p < 0.001) (Table 2).

Table 2. Factors significantly associated with serum creatinine evolution (univariate analysis).

Variable	Categories/units	Adjusted coefficient	Wald	p-value	p-value (Type 3 test)
Age	Years	−0.029	2.290	0.022	
Geographic origin	Semi-urban *vs* rural	0.151	0.262	0.793	<0.001
	Urban *vs* rural	−1.859	−3.469	<0.001	
NSAID use	Yes *vs* no	1.007	2.605	0.009	
Urogenital schistosomiasis	Yes *vs* no	−3.600	−3.559	<0.001	
Anemia	Yes *vs* no	3.131	5.980	<0.001	

NSAIDs: non-steroidal anti-inflammatory drugs.

3.4. Identification of Serum Creatinine Trajectories

We hypothesized the existence of two, three, four or five distinct classes. Figure 2 shows the evolution of serum creatinine according to these different hypotheses. The three-class model was selected as the most relevant for predicting patient creatinine trajectories based on its clinical interpretability and having the lowest Akaike Information Criterion (AIC = 7549.97) (Table 3). The mean posterior probabilities were >0.80 for all three classes, and the entropy was 0.76, indicating good class separation. In this model, we observed the following: class 1 (rapid progression of CKD), characterized by a rapid, linear increase in serum creatinine; class 2 (stable CKD), characterized by stable serum creatinine; and class 3 (moderate progression of CKD), characterized by a moderate, non-linear increase in serum creatinine (Figure 2(b)).

Table 3 presents the assessment parameters of the different models based on the class hypothesis.

3.5. Descriptive Analysis of the Final Model According to Different Trajectories

Nineteen patients (15.45%) were identified as Class 1. Classes 2 and 3 comprised respectively 39 (31.7%) and 65 (52.85%) patients. Patients in class 1 (rapid progression) were significantly older (73 ± 34.6 years) and had a higher prevalence of diabetes (31.6%) (Table 4).

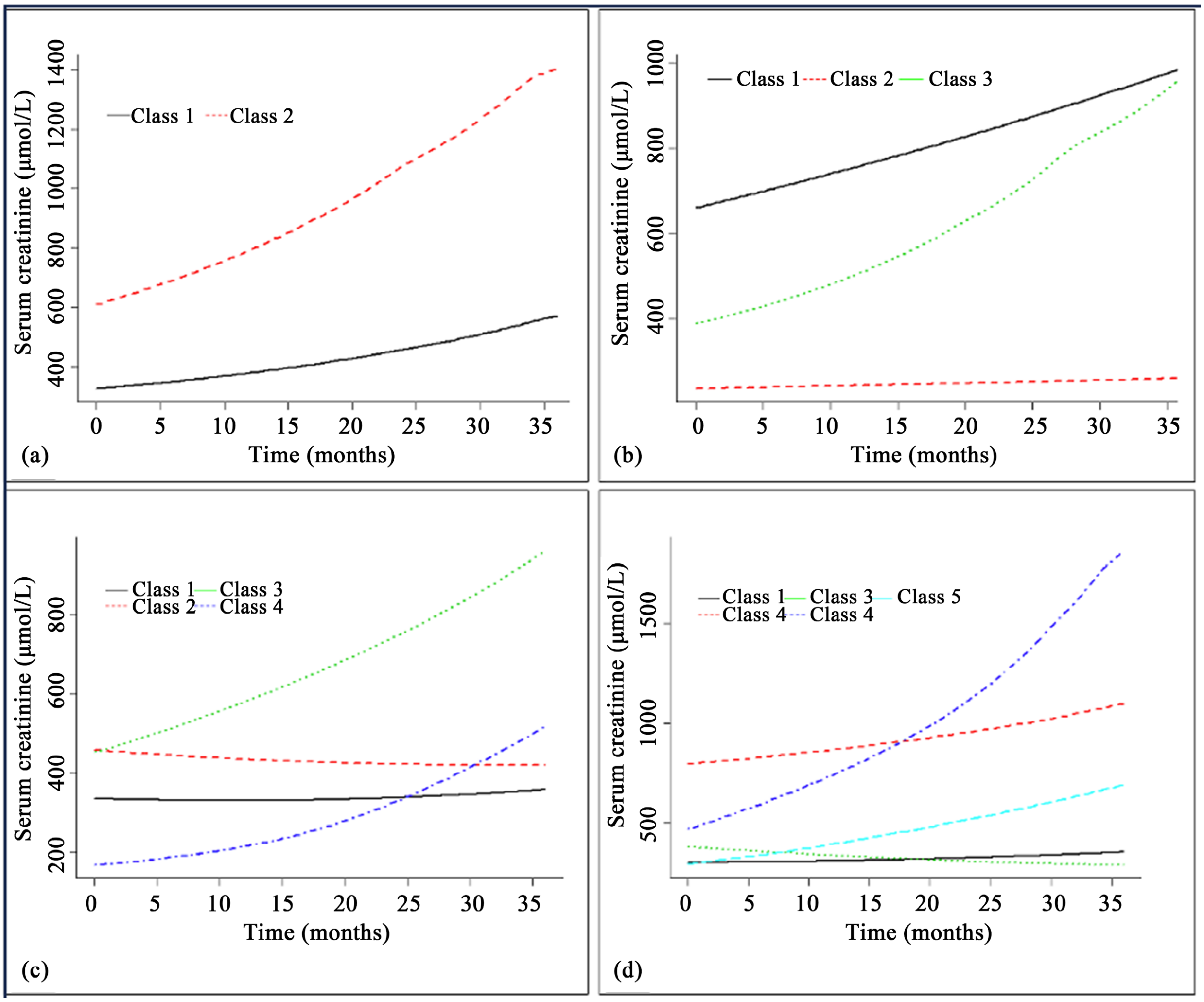

Figure 2. Evolution of serum creatinine according to the two-class (a), three-class (b), four-class (c) and five-class (d) hypothesis.

Table 3. Model comparison according to number of classes.

Number of classes	Log-likelihood	Number of parameters	AIC	Proportion				
				Class 1	Class 2	Class 3	Class 4	Class 5
2	−3756.815	30	7573.63	70.49	29.51			
3	−3730.984	44	**7549.97**	15.57	31.97	52.46		
4	−3743.107	54	7594.21	27.87	19.677	39.34	13.11	
5	−3714.909	66	7561.82	33.61	23.78	9.84	18.85	13.93

AIC: Akaike information criterion.

Table 4. Patients' characteristics by identified trajectory class.

Variables	Class 1 (N = 19)	Class 2 (N = 39)	Class 3 (N = 65)
Age (years) mean ± sd	73 ± 34.55	62 ± 38	59.6 ± 34
Sex			

Continued

Female	9 (47.4)	9 (23.1)	25 (38.5)
Male	10 (52.6)	30 (76.9)	40 (61.5)
Geographical origin n (%)			
Rural	5 (26.3)	4 (10.3)	9 (13.9)
Semi-urban	4 (21.1)	11 (28.2)	22 (33.8)
Urban	10 (52.6)	24 (61.5)	34 (52.3)
Education level n (%)			
None	11 (57.9)	17 (43.6)	21 (32.3)
Primary	2 (10.5)	9 (23.1)	16 (24.6)
Secondary	5 (26.3)	10 (25.6)	24 (36.9)
University	1 (5.3)	3 (7.7)	4 (6.2)
Income level			
Low	13 (68.4)	24 (61.5)	44 (67.7)
Middle	6 (31.6)	15 (38.5)	20 (30.8)
High	0	0	1 (1.5)
Comorbidities			
Hypertension n (%)			
Yes	15 (78.9)	34 (87.2)	46 (70.8)
No	4 (21.1)	5 (12.8)	19 (29.2)
Diabetes mellitus n (%)			
Yes	6 (31.6)	8 (20.5)	12 (18.5)
No	13 (68.4)	31 (79.5)	53 (81.5)
Gout n (%)			
Yes	2 (10.5)	5 (12.8)	8 (12.3)
No	17 (89.5)	34 (87.2)	57 (87.7)
Urogenital schistosomiasis			
Yes	0	3 (7.7)	1 (1.5)
No	19 (100)	36 (92.3)	64 (98.5)
Herbal medicine consumption n (%)			
Yes	6 (31.6)	15 (38.5)	14 (21.5)
No	13 (68.4)	24 (61.5)	51 (78.5)
NSAIDs use n (%)			
Yes	6 (31.6)	15 (38.5)	20 (30.8)
No	13 (68.4)	24 (61.5)	45 (69.2)
Viral hepatitis n (%)			
Yes	0	1 (2.6)	1 (1.5)
No	19 (100)	38 (97.4)	64 (98.5)

Continued

HIV n (%)			
Yes	1 (5.3)	0	2 (3.1)
No	18 (94.7)	39 (100)	63 (96.9)
BMI (kg/m^2)	23.3 ± 5.2	24.2 ± 5.1	23.2 ± 5.1
SBP (mmHg)	153.5 ± 29.1	149 ± 29.2	155.7 ± 30.2
DBP (mmHg)	91.2 ± 16.2	90.3 ± 16.3	92.3 ± 16.6
Proteinuria (g/24 hours)	1.1 ± 1.1	1.1 ± 1.3	1.3 ± 1.2

BMI: body mass index; DBP: diastolic blood pressure; HIV: human immunodeficiency virus; NSAIDs: non-steroidal anti-inflammatory drugs; SBP: systolic blood pressure. Values are presented as mean ± standard deviation (sd) or number (percentage). For each latent class, the values represent the estimated conditional means, which are based on the posterior membership probabilities for all 123 patients and are estimated by the model. N is the number of patients a posteriori classified in each class.

3.6. Predictive Factors for Class Membership According to the Final Multivariate Model

Table 5 presents the predictors of class membership, with Class 2 (stable trajectory) as the reference. A longer time since inclusion was associated with a higher probability of belonging to Class 1 (rapid progression; coefficient = 0.053; p = 0.0006) and Class 3 (moderate progression; coefficient = 0.083; p = 0.0001) compared to Class 2. Advanced age was not significantly associated with Class 1 membership (coefficient = 0.002; p = 0.8009). However, age decreased the probability of belonging to Class 3 (coefficient = -0.050; p = 0.0016), suggesting that older patients were more likely to have a stable trajectory. Patients from semi-urban areas were more likely to belong to Class 3 than rural residents (coefficient = 1.727; p = 0.0359). Conversely, urban residents were less likely to belong to Class 3 (coefficient = −2.128; p = 0.0032). No significant association was observed between geographic origin and Class 1 membership (p > 0.05). Urogenital schistosomiasis was associated with a lower probability of belonging to Class 1

Table 5. Predictive factors for class membership (multivariate analysis).

Predictors	Class 1 *vs* Class 2		Class 3 *vs* Class 2	
	Adjusted coefficient	p-value	Adjusted coefficient	p-value
Time since inclusion (months)	0.053	0.0006	0.083	0.0001
Age (years)	0.002	0.8009	−0.050	0.0016
Geographic origin				
Semi-urban *vs* rural	−0.487	0.1410	1.727	0.0359
Urban *vs* rural	−0.715	0.0722	−2.128	0.0032
Urogenital schistosomiasis, yes vs no	−4.269	0.0003	−5.322	0.0038
NSAID use, yes *vs* no	−0.188	0.4524	2.125	0.0006
Anemia, yes *vs* no	1.741	0.0001	3.522	0.0001

NSAIDs: non-steroidal anti-inflammatory drugs.

(coefficient = −4.269; p = 0.0003) and Class 3 (coefficient = −5.322; p = 0.0038) compared to Class 2. However, only four patients (3.3%) had this history, so these estimates should be interpreted with caution. NSAID use increased the probability of belonging to Class 3 (coefficient = 2.125; p = 0.0006) but was not associated with Class 1 (coefficient = −0.188; p = 0.4524). Anemia was associated with a higher probability of belonging to Class 1 (coefficient = 1.741; p = 0.0001) and Class 3 (coefficient = 3.522; p = 0.0001) compared to Class 2. The effect was more pronounced for the moderate progression trajectory.

4. Discussion

In this study, we examined serum creatinine trajectories rather than eGFR trajectories. This choice was guided by several factors. First, creatinine was assayed using the Jaffe method, which overestimates creatinine levels compared to enzymatic methods [22]. Second, inter-laboratory variability in creatinine assays is well documented and can substantially affect eGFR accuracy without standardized calibration [12] [22]. Using non-standardized values to calculate eGFR would have introduced significant bias, with observed variations reflecting calibration differences rather than true CKD progression. By studying serum creatinine trajectories, we mitigated this potential bias. This approach is supported by Onuigbo *et al.* [11], who demonstrated that the prognostic relevance of serum creatinine trajectories is comparable to that of GFR trajectories.

However, this methodological choice is not without limitations. Serum creatinine remains an imperfect marker of renal function, influenced by several physiological factors. Therefore, it cannot replace eGFR calculated under standardized conditions, which remains the reference indicator. Consequently, our results are not directly comparable to studies examining eGFR trajectories. Nevertheless, they offer a practical approach for studying CKD progression in settings where assay standardization is not always possible.

We identified three distinct serum creatinine trajectories, consistent with previous studies describing slow, moderate and rapid profiles of CKD progression [5] [23] [24]. Previous studies have varied in the number of classes identified, with some reporting up to five trajectories based on population characteristics and methodology [5] [23] [24].

In a study including 1,967 patients with a median age of 58 years, Boucquemont *et al.* [5] used latent class analysis to identify five distinct trajectories of measured GFR: a first class characterized by rapid progression with a sharp decline in eGFR; a second marked by high baseline eGFR with non-linear improvement; and three classes (3, 4, and 5) showing near-linear progression, with slow, moderate-slow, and severe-slow eGFR decline, respectively. Patients in the rapid progression class (Class 1) were, on average, younger than those in the moderate-decline classes (Classes 3, 4, and 5) [5].

Santos *et al.* [23] investigated CKD progression in 378 patients with a mean age of 75.4 years. They identified four eGFR trajectories: slow decline; gradual decline, early rapid decline, and rapid decline. Women, diabetic subjects, those with significant proteinuria, and those hospitalized in the year prior to inclusion were more likely to belong to the rapid decline class. In a cohort of patients with stage 4 CKD, Xie *et al.* [24] identified three evolutionary trajectories: a slow-decline class comprising 72% of patients (Class 1), a rapid-decline class (Class 2), and a class with initial stable eGFR followed by rapid decline (Class 3).

Age is a well-established risk factor for CKD, with approximately 44% of individuals over 65 affected [7] [25] [26]. Aging is associated with a higher prevalence of renal risk factors including diabetes, hypertension, and cardiovascular disease [26] [27]. Physiological renal decline begins around age 30, with average eGFR loss of 0.9 mL/min/year [25]. Consequently, healthy individuals may exhibit an eGFR of less than 60 ml/min/1.73m^2 after the age of 60 due to this natural process [26]. However, factors such as hypertension, smoking, dyslipidemia, cardiovascular disease and environmental exposures can accelerate this physiological decline [28] [29].

Our multivariate analysis revealed an unexpected relationship between age and chronic kidney disease (CKD) progression, with older patients being more likely to have a stable trajectory. This could be due to a subgroup of older individuals with few comorbidities or slowly progressive nephropathies, who maintain stable renal function. This could reflect either normal physiological decline or indolent disease.

In our cohort, patients in the rapid progression class had a longer follow-up duration, likely due to the onset of complications requiring closer monitoring [5].

Although CKD is generally more prevalent in women, its progression to end-stage renal disease is typically faster in men, attributed to the pro-inflammatory effects of testosterone and a higher prevalence of hypertension and smoking [30] [31]. However, in resource-limited settings, women face barriers to healthcare access due to economic and socio-cultural factors, worsening their renal and overall prognosis [31]. This may explain the higher proportion of women in the rapid progression group in our study compared to the stable and moderate progression groups.

Anemia, present in 65.9% of patients at baseline and 71.9% at 36 months, is a marker of worsening CKD. Its prevalence increases with disease progression, rising from 8.4% at stage 1 to 53.4% at stage 5 [32]. It is associated with a two-fold faster eGFR decline [33] and was a strong independent predictor of progression in our study.

Several studies have demonstrated an association between NSAIDs use and CKD risk. Chronic NSAIDs use was specifically associated with moderate progression (class 3), consistent with its link to slowly progressive tubulointerstitial nephropathy. This nephrotoxic effect is cumulative, resulting from episodes of acute kidney injury with incomplete recovery due to the inhibition of prostaglandins involved in renal autoregulation during hypoperfusion [34] [35].

Healthcare access disparities exist based on residential area. Patients in rural areas often face poverty, lower education levels, exposure to environmental toxins, and lack of specialized healthcare infrastructure. This leads to inadequate comorbidity management and delayed CKD diagnosis [36]. CKD prevalence is generally higher in rural than urban areas [37] [38]. However, socioeconomic inequalities also exist in urban areas where poor populations may live in unsanitary conditions, without access to clean water, exposed to environmental threats and facing healthcare access difficulties [39]. In Pakistan, rural residents had lower risk of moderate to severe CKD (stages 3 and 4) than urban residents [40]. This suggests that inequities in healthcare access (economic, infrastructural, or geographic), rather than the residential area per se, underlie the increased CKD prevalence and faster progression observed in some rural populations.

Urogenital schistosomiasis is a neglected tropical disease that is endemic in sub-Saharan Africa and is responsible for chronic tubulointerstitial and glomerular nephropathies [41] [42]. However, a history of urogenital schistosomiasis is associated with a lower probability of belonging to progressive trajectories (Classes 1 and 3). Nevertheless, this result should be treated with prudence, as it was observed in only four patients.

Study limitations

This study has several limitations. The retrospective design and modest sample size (n = 123) restrict the study's generalizability. Serum creatinine was measured only every six months, which could have resulted in underestimation of fluctuations. Behavioral factors such as treatment adherence and lifestyle were not assessed. Furthermore, the latent class analysis was not adjusted for baseline kidney disease severity (creatinine, eGFR or CKD stage). Therefore, it cannot be excluded that the identified trajectories partly reflect differences in baseline severity rather than purely longitudinal changes. Further research involving larger sample sizes is needed to test the robustness of these trajectories when adjusted for baseline kidney function. Finally, the scarcity of CKD progression studies in developing countries limits comparative analysis.

Perspectives

These preliminary results provide a foundation for developing a decision-support tool to predict CKD evolution in patients in Burkina Faso. Such modeling would enable personalized care, facilitating the early identification of patients at high risk of rapid progression and enabling targeted intervention. While we acknowledge the methodological limitations of this preliminary work, we believe it provides a basis for future research.

5. Conclusion

CKD progresses towards end-stage renal disease at variable rates. Our study identified three CKD progression trajectories in patients attending nephrology outpatient clinics. The results show that anemia is a strong predictor of progression, while semi-urban residence and NSAID use are associated with moderate progression trajectories. The relationship with age was complex, highlighting the role of comorbidities. Further studies on larger populations are needed to develop a decision-making tool that will ensure personalized, optimal care for CKD patients in Burkina Faso.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Supplementary

Table S1. Clinical and biological parameters during follow-up.

Parameters	Baseline (N = 123)	6 months (N = 123)	12 months (N = 123)	18 months (N = 74)	24 months (N = 72)	30 months (N = 57)	36 months (N = 57)
SBP (mmHg)	153.2 ± 29.5	148.9 ± 26.3	152.9 ± 26.7	153 ± 30.4	153.6 ± 23.6	153.1 ± 33.6	146.1 ± 25.4
<140	36 (29.3)	56 (45.6)	34 (27.7)	26 (35.2)	17 (23.6)	21 (36.8)	20 (35.1)
≥140	87 (70.7)	67 (54.4)	89 (72.3)	48 (64.8)	55 (76.4)	36 (63.2)	37 (64.9)
DBP (mmHg)	91.6 ± 16.3	86.8 ± 14.1	88.1 ± 14.5	87.6 ± 17	86.6 ± 14.7	87.2 ± 16.7	85.7 ± 13.9
<90	44 (35.8)	63 (51.2)	62 (50.4)	40 (54.1)	40 (55.6)	28 (49.1)	33 (57.9)
≥90	79 (64.2)	60 (48.8)	61 (49.6)	34 (45.9)	32 (44.4)	29 (50.9)	24 (42.1)
BMI (kg/m^2)	23.6 ± 5.1	23.5 ± 5	23.5 ± 5	23.6 ± 4.8	23.5 ± 4.8	23.8 ± 4.5	23.9 ± 4.9
<18.5	11 (8.9)	14 (11.4)	11 (8.9)	7 (9.6)	11 (15.3)	7 (12.2)	6 (10.5)
18.5 - 24.9	83 (67.5)	79 (64.2)	80 (65.1)	46 (62.1)	39 (54.2)	31 (54.4)	32 (56.1)
25-29.9	16 (13)	17 (13.8)	18 (14.6)	12 (16.2)	15 (20.8)	14 (24.6)	12 (21.1)
≥30	13 (10.6)	13 (10.6)	14 (11.4)	9 (12.1)	7 (9.7)	5 (8.8)	7 (12.3)
Blood urea nitrogen (mmol/L)	14 ± 9.4	14.3 ± 11.5	16.1 ± 11.5	12.1 ± 8	14 ± 9.6	14.6 ± 10	16.6 ± 11.9
Serum creatinine (µmol/L)	369.3 ± 333.7	367.3 ± 361.3	429.8 ± 428.7	307.6 ± 211	352.3 ± 255.9	388.3 ± 313.8	524.7 ± 569.6
eGFR (ml/min/1173m^2)	30.4 ± 18	30.1 ± 16.5	26.8 ± 16.4	30.4 ± 17	27.6 ± 14.8	26.3 ± 16.4	23.5 ± 16.1
CKD stage n (%)							
1	0	0	0	0	0	0	0
2	8 (6.5)	5 (4.1)	4 (3.3)	3 (4.1)	1 (1.4)	2 (3.5)	1 (1.8)
3a	16 (13)	18 (14.6)	15 (12.2)	8 (10.8)	8 (11.1)	6 (10.5)	4 (7)
3b	36 (29.3)	35 (28.5)	26 (21.1)	23 (31.1)	22 (30.6)	14 (24.6)	12 (21.1)
4	33 (26.8)	39 (31.7)	44 (35.8)	28 (37.8)	25 (34.7)	19 (33.3)	21 (36.8)
5	30 (24.4)	26 (21.1)	34 (27.6)	12 (16.2)	16 (22.2)	16 (28.1)	19 (33.3)
Serum uric acid (µmol/L)	494.4 ± 136.7	485.8 ± 134	514.7 ± 147.3	478 ± 110	497 ± 109.7	484.3 ± 128	511.8 ± 136.9
Serum sodium (mmol/L)	137.6 ± 6.1	137.9 ± 5.8	138.1 ± 4.4	138.1 ± 6	137.1 ± 5.8	136.7 ± 5.7	135.8 ± 6.1
Serum potassium (mmol/L)	4.2 ± 0.7	4.4 ± 0.7	4.5 ± 0.8	4.3 ± 0.8	4.3 ± 0.6	4.2 ± 0.6	4.3 ± 0.8
Serum calcium (mmol/L)	2.2 ± 0.2	2.2 ± 0.3	2.2 ± 0.3	2.3 ± 0.2	2.2 ± 0.2	2.2 ± 0.3	2.2 ± 0.3
Serum phosphate (mmol/L)	1.4 ± 0.4	1.4 ± 0.4	1.6 ± 1	1.3 ± 0.3	1.4 ± 0.3	1.4 ± 0.4	1.5 ± 0.6
Serum bicarbonate (mmol/L)	22.3 ± 4.6	22.1 ± 4.7	20.6 ± 5	23.5 ± 4.2	21.8 ± 4	21 ± 5.1	20.4 ± 5

Continued

Blood glucose (mmol/L)	5.5 ± 2.3	5.5 ± 1.9	5.5 ± 1.6	5.9 ± 2.5	5.6 ± 2.5	6,2 ± 2,5	6.3 ± 3.3
Hemoglobin (g/dL)	10.7 ± 2.3	10.8 ± 2.3	10.4 ± 2.5	11.3 ± 2.1	11 ± 2	11.1 ± 2.3	10.4 ± 2.5
Anemia n (%)	81 (65.9)	77 (62.6)	85 (69.1)	46 (62.2)	47 (65.3)	34 (59.7)	41 (71.9)
White blood cells (cells/mm^3)	6528.5 ± 3039	6740.2 ± 5297.1	6346.7 ± 1846.7	6751.5 ± 2760.1	6546.5 ± 1895.6	6726.3 ± 2191.5	6582.5 ± 1818.9
Platelets (cells/mm^3)	257015.5 ± 91491.6	252006.5 ± 66,144	257292.7 ± 75648.1	236,027 ± 59456.9	240972.2 ± 70295.2	246684.2 ± 72601.2	248649.1 ± 75326.1
24-hour proteinuria (g/24h)	1.2 ± 1.5	1.1 ± 1.1	1.2 ± 1.3	1.2 ± 1.4	1.1 ± 1	1.2 ± 1.4	1.5 ± 1.8
Proteinuria ≥ 0.5 g/24h n (%)	18 (14.6)	23 (18.7)	24 (19.5)	11 (14.9)	18 (25)	13 (22.8)	14 (24.6)

BMI: body mass index; CKD: chronic kidney disease; DBP: diastolic blood pressure; eGFR: estimated glomerular filtration rate; SBP: systolic blood pressure. Values are presented as mean ± standard deviation (sd) or number (percentage).