

Chronic Pruritus in Predialysis Chronic Kidney Disease: Prevalence, Phenotype and Temporal Patterns in a Prospective Cohort

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CKD-associated pruritus (CKD-aP) is a burdensome symptom with incompletely understood pathophysiology and limited predialysis data. In this prospective observational study, 95 adult nondialysis CKD patients (stages G1–G5) were followed for up to 36 months (232 observations), 86 patients were analysed after exclusion of chronic dermatological conditions. Chronic pruritus (≥6 weeks) was assessed using a numerical rating scale (NRS) and anatomical mapping. Chronic pruritus occurred in 26 of 86 patients (30.2%), with numerically higher prevalence in advanced CKD (G3b–5:32.8% vs G1–3a: 22.7%), whereas intensity was numerically higher in early CKD (median 8 vs 5). The back, legs and head/face were most frequently affected, consistent with the bilateral, nondermatomal pattern described in dialysis populations. Among patients with repeated assessments, 19.0% showed an episodic trajectory and 7.6% had persistent pruritus. Multivariable logistic regression and generalized estimating equations (GEE) were used to identify independent predictors of chronic pruritus. No laboratory parameter, including PTH, was independently associated with pruritus. Dyslipidaemia was the only clinical characteristic significantly associated with pruritus status (69.2% vs 43.3%, $p=0.035$). Given the modest sample size, smaller effect estimates may have been missed. CKD-aP affects approximately 1 in 3 predialysis patients, follows an episodic course, and is not explained by any single biochemical marker, supporting multifactorial pathophysiology.

Key words: pruritus; chronic kidney disease; itch; parathyroid hormone; pre-dialysis.

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Chronic pruritus is defined as itch persisting for more than 6 weeks (1). In patients with chronic kidney disease (CKD), it is a frequent and

SIGNIFICANCE

Many patients with chronic kidney disease experience persistent itching – a symptom that can severely affect sleep, concentration and overall well-being. Research has focused on patients on dialysis, mostly leaving out earlier stages of kidney disease. This study therefore followed 95 kidney patients over 3 years and found that roughly 1 in 3 developed chronic itching. The symptom was frequently episodic rather than constant, and no blood test could reliably predict who would be affected. These findings suggest that kidney patients should be asked for itching well before dialysis begins.

burdensome symptom (1, 2). Commonly referred to as CKD-associated pruritus (CKD-aP) or uremic pruritus (3–7), it is associated with impaired quality of life and patient-reported outcomes (4–6) as well as adverse clinical outcomes, including hospitalization, cardiovascular events and mortality (6–8).

Prevalence estimates of CKD-aP vary widely depending on study design, diagnostic criteria and patient populations (4, 8). While its epidemiological burden has been well documented in dialysis-dependent populations, with reported prevalence rates ranging from 25% to 55% (9–11) – data on earlier CKD stages remain limited (4, 12–14). Consequently, our understanding of when pruritus first emerges during renal disease progression, how it evolves over time and whether its phenotype differs before dialysis initiation remains incomplete. The pathophysiology of CKD-aP is complex and incompletely understood. Multiple interacting mechanisms have been proposed, including disturbances in mineral metabolism, accumulation of uremic toxins, systemic inflammation, altered peripheral and central neural processing, dysregulation of opioid signalling and xerosis-related skin barrier dysfunction (1, 2). However, clinical studies have yielded inconsistent associations, particularly with mineral metabolism, raising questions regarding causal relevance (2, 13, 15, 16).

A key limitation of the current literature is the predominance of cross-sectional studies in dialysis populations, which are less suited to clarifying symptom onset, longitudinal dynamics and early-stage disease

correlates (3, 6, 9). Moreover, detailed data on anatomical distribution and concomitant skin findings in affected predialysis patients remain sparse (4, 6, 12, 13). Predialysis cohorts allow assessment with less confounding from kidney replacement therapy and end-stage uremic burden.

A more granular characterization of CKD-aP across the CKD continuum, especially in predialysis stages, may therefore help distinguish early drivers from downstream consequences of kidney failure. This study primarily aimed to characterize the prevalence, severity, anatomical distribution and longitudinal pattern of chronic pruritus across the CKD spectrum in a non-dialysis population. Multivariable analyses explored potential biochemical correlates.

METHODS

Study design and participants

This prospective observational study investigated the prevalence and clinical correlates of chronic pruritus in adults with chronic kidney disease (CKD) not receiving dialysis. Consecutive patients aged 18 years or older were recruited from the outpatient nephrology

clinic in Wiesbaden, Germany, between 2013 and 2015 and followed at 6-month intervals for up to 36 months. Participation was voluntary, and patients could withdraw at any time.

Kidney function was classified using estimated glomerular filtration rate (eGFR), calculated with the race-free CKD-EPI 2021 creatinine equation (17). For the main analyses, patients were stratified into 2 groups: early-to-moderate CKD (G1-G3a; $\text{eGFR} \geq 45 \text{ mL/min/1.73 m}^2$) and advanced CKD (G3b-G5; $\text{eGFR} \leq 44 \text{ mL/min/1.73 m}^2$). This stratification corresponds to the established KDIGO boundary between G3a and G3b (18) and was chosen to distinguish patients with mild-to-moderate versus more advanced impairment of kidney function. Further, from a statistical standpoint, a lower threshold, such as $\text{eGFR} < 30 \text{ mL/min/1.73 m}^2$, would have left only 21 patients in the advanced group, precluding meaningful stratified analysis given the cohort size.

Eligibility criteria

Eligible participants were adults with CKD and sufficient clinical and laboratory data to determine CKD stage and pruritus status. Exclusion criteria were

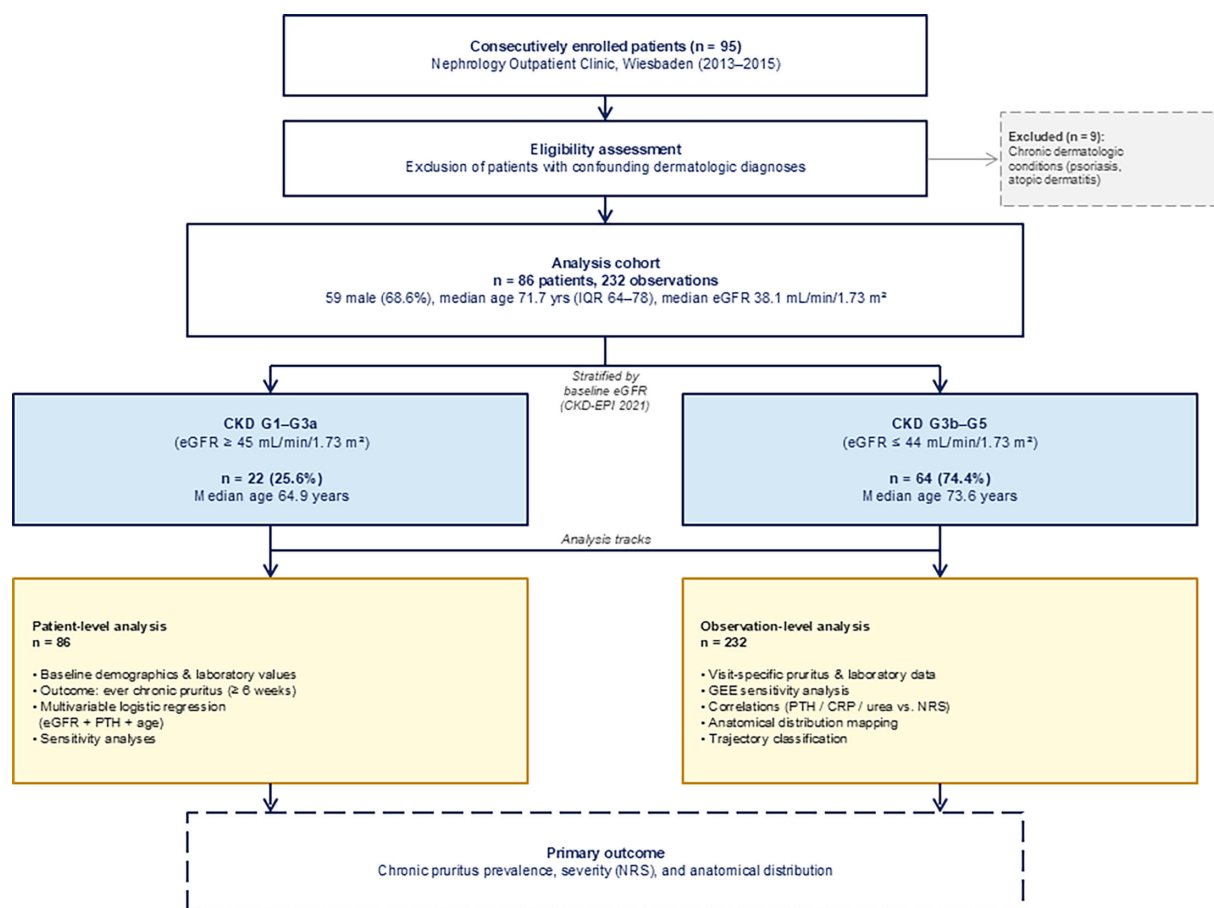


Fig. 1. Study flow diagram of the study. CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate (CKD-EPI 2021 equation); GEE, generalized estimating equations; NRS, numerical rating scale; PTH, parathyroid hormone; CRP, C-reactive protein.

chronic dermatological conditions likely to confound pruritus assessment (e.g. atopic dermatitis, psoriasis, cutaneous lymphoma), acute kidney injury, renal replacement therapy (dialysis or kidney transplantation) or missing key data required for CKD staging or pruritus classification.

Outcomes and data collection

The primary outcome was chronic pruritus, defined as itch lasting 6 weeks or longer. Pruritus intensity was assessed at each visit using a numerical rating scale (NRS, 0–10). Secondary and descriptive outcomes included pruritus duration, anatomical distribution, seasonal variation, skin phenotype (dry, scaly, oily, normal) and pruritus-related healthcare utilization. Skin phenotype was recorded at each visit by patient self-report through a structured questionnaire administered by a dermatologically trained study nurse. Patients were asked directly whether they suffered from dry, scaly or oily skin; “normal” was recorded when none of these were affirmed, and categories were not mutually exclusive. The nurse administered the structured questionnaire but did not perform a formal clinical skin examination (e.g. Overall Dry Skin Score or instrumented skin barrier measurement, e.g., transepidermal water loss). At each visit, standardized data collection included serum creatinine, eGFR, urea, phosphate, calcium, parathyroid hormone (PTH), 25-hydroxyvitamin D, C-reactive protein (CRP), alkaline phosphatase, haemoglobin, comorbidities, and current medication.

Statistical analysis

All analyses were performed in R version 4.3.3. Continuous variables are presented as medians with IQR; categorical variables as counts and percentages; group comparisons used Wilcoxon rank-sum and χ^2 or Fisher exact tests. Two complementary datasets were used: a patient-level dataset (baseline characteristics:

medication, diagnosis) and an observation-level dataset (questionnaires, laboratory and measurements). A prespecified multivariable logistic regression model including PTH, eGFR and age was fitted at the patient level; discrimination was assessed by AUC and multicollinearity by variance inflation factors. Sensitivity analyses additionally incorporated phosphate, binary CKD stage, diabetes mellitus or statin use. Generalized estimating equations (GEE; exchangeable correlation structure, robust standard errors) were applied at the observation level using the same predictors. Among patients with ≥ 2 visits, pruritus trajectories were classified as never, episodic or persistent. Spearman correlations were calculated between NRS and PTH, CRP, urea and number of affected body regions.

In a supplementary exploratory analysis, Random Forest and XGBoost models were applied at the observation level to identify potential nonlinear predictors of pruritus above the prespecified threshold of NRS>3 (Fig. S1).

RESULTS

Study population

The final cohort comprised 86 patients, of whom 59 (68.6%) were male (for a flow chart see Fig. 1). Median age was 71.7 years (IQR 64.2–78.3), and median eGFR was 38.1 mL/min/1.73 m². Twenty-two patients (25.6%) had early-to-moderate CKD (G1-G3a), and 64 (74.4%) had advanced CKD (G3b-G5). A total of 232 observations were available, corresponding to a mean of 2.7 visits per patient.

Baseline demographic characteristics and key laboratory variables stratified by CKD stage are shown in Table I.

Compared with CKD G1-G3a, patients with G3b-G5 were older and had higher phosphate, urea, creatinine, PTH and CRP; COPD was more frequent in advanced CKD. Sex, BMI, haemoglobin, calcium and vitamin D

Table I. Baseline demographic characteristics and key laboratory variables stratified by CKD stage group

Variable	Overall (n=86)	CKD G1-G3a (n=22)	CKD G3b-G5 (n=64)	p-value
Age, years	71.73 (64.22–78.28)	64.91 (61.98–74.69)	73.64 (66.31–79.00)	0.041
Male sex, n (%)	59 (68.6)	17 (77.3)	42 (65.6)	0.4
BMI, kg/m ²	27.00 (25.00–31.80)	27.05 (25.05–31.28)	27.00 (24.98–31.90)	0.8
Haemoglobin, g/dL	13.40 (12.10–14.20)	14.10 (12.90–14.58)	13.25 (12.05–13.83)	0.068
Calcium, mmol/L	2.37 (2.29–2.46)	2.41 (2.29–2.46)	2.37 (2.29–2.45)	0.5
Phosphate, mmol/L	1.12 (1.00–1.20)	1.03 (0.97–1.12)	1.15 (1.02–1.23)	0.016
Urea, mg/dL	63.50 (54.25–83.50)	50.50 (42.25–57.00)	68.50 (59.50–95.00)	<0.001
Creatinine, mg/dL	1.76 (1.51–2.09)	1.42 (1.27–1.62)	1.92 (1.67–2.15)	<0.001
eGFR (mL/min/1.73 m ²)	37.05 (31.45, 44.88)	48.95 (48.03, 55.30)	34.80 (29.85, 38.05)	<0.001
25-hydroxyvitamin D, ng/mL	23.00 (15.00–36.00)	23.50 (16.00–32.00)	23.00 (15.00–38.50)	0.8
Parathyroid hormone, pg/mL	69.50 (51.50–107.25)	62.00 (36.00–69.00)	82.00 (54.00–112.50)	0.003
C-reactive protein, mg/L	2.65 (1.10–5.93)	1.10 (0.50–2.30)	3.70 (1.45–6.50)	0.022

Values are shown as median (interquartile range) unless otherwise stated. p-values were calculated using the Wilcoxon rank-sum test for continuous variables and χ^2 or Fisher exact test for categorical variables, as appropriate; bolt when p<0.05. CKD Stages G1-G3a = eGFR ≥ 45 (mL/min/1.73 m²), CKD G3b-G5 = eGFR < 45 (mL/min/1.73 m²).

did not differ. Full stage-stratified characteristics are provided in Table SI.

Prevalence and severity of chronic pruritus

At the patient level, chronic pruritus was reported by approximately one third of participants during follow-up, with a numerically but not significantly higher prevalence in advanced CKD (**Fig. 2A**). Visit-level prevalence remained broadly stable across available follow-up assessments, consistent with a fluctuating rather than progressively accumulating pattern (**Fig. 2B**). Among visits with chronic pruritus, NRS intensity was numerically higher in early-to-moderate CKD; however, the small subgroup size limits interpretation (**Fig. 2C**).

Pruritus phenotype

Among all reported pruritus localisations, the back was the most frequently affected region (20.2%), followed by the legs (17.6%), head/face (15.1%) and arms (13.4%) (**Fig. 3**). Overall, the distribution pattern appeared nondermatomal.

Among observations with available symptom-duration data ($n=50$), pruritus had been present for months in 44%, years in 38% and days in 18%. Thus, in most observations with available data, symptoms had persisted for months or years.

Among affected observations with skin report available ($n=50$), dry skin was recorded in 72%, scaly skin in 8%, oily skin in 4% and normal skin in 24%; categories were not mutually exclusive, identifying patient-perceived xerosis as the dominant skin-related finding. Seasonal variation data were available for 29 observations from 19 patients; winter and summer were the most frequently

reported aggravating seasons (each 31%), with a constant nonseasonal pattern in 28%.

Pruritus trajectories

Among 78 patients with at least 2 evaluable visits, three trajectory patterns were identified: 57 (73.1%) never, 15 (19.2%) episodic and 6 (7.7%) persistent chronic pruritus (**Fig. 4a**).

A heatmap of individual NRS trajectories is shown for all patients with at least one pruritic visit ($n=26$; **Fig. 4b**). Patients with peak $\text{NRS} \geq 7$ tended towards persistent or recurrent symptoms, whereas lower-intensity pruritus was more often episodic.

Descriptive comparisons across trajectory groups showed numerically higher PTH concentrations in the persistent group (median 107 pg/mL, IQR 87–118) than in the episodic (73 pg/mL, IQR 57–102) and never-pruritus groups (67 pg/mL, IQR 48–104), but this difference was not statistically significant (Kruskal–Wallis $p=0.249$). No significant differences were observed for eGFR, urea, phosphate, calcium, haemoglobin or vitamin D.

Clinical and biochemical correlates of pruritus

Comparison of baseline characteristics between patients with and without chronic pruritus at any visit showed no significant differences in laboratory variables, CKD aetiology or most comorbidities and medications Table SII. Two characteristics differed significantly: dyslipidaemia was more frequent in patients with chronic pruritus (69.2% vs 43.3%, $p=0.035$), and overall comorbidity burden was higher (median 3 vs 2; $p=0.042$). In the primary multivariable logistic regression model at the patient level ($n=84$ with complete data; 26 events), no

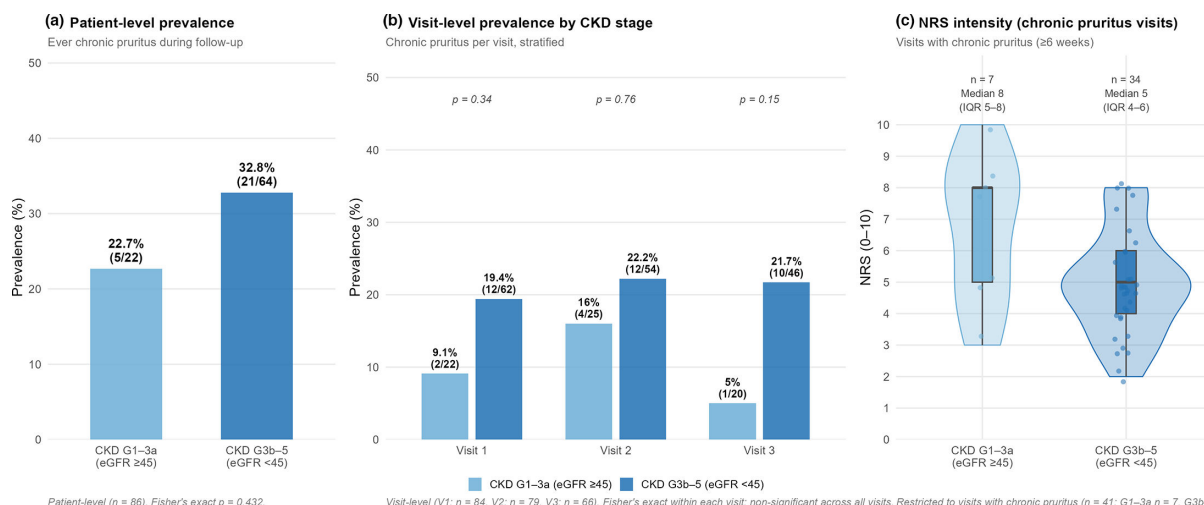


Fig. 2. A Violin plots showing NRS score distribution for CKD G1–3a and CKD G3b–5 on patient level and B on observation level. C Distribution of pruritus intensity (NRS) across CKD stages (observation-level) for chronic pruritus for NRS > 0 . CKD, chronic kidney disease, NRS, numerical rating scale.

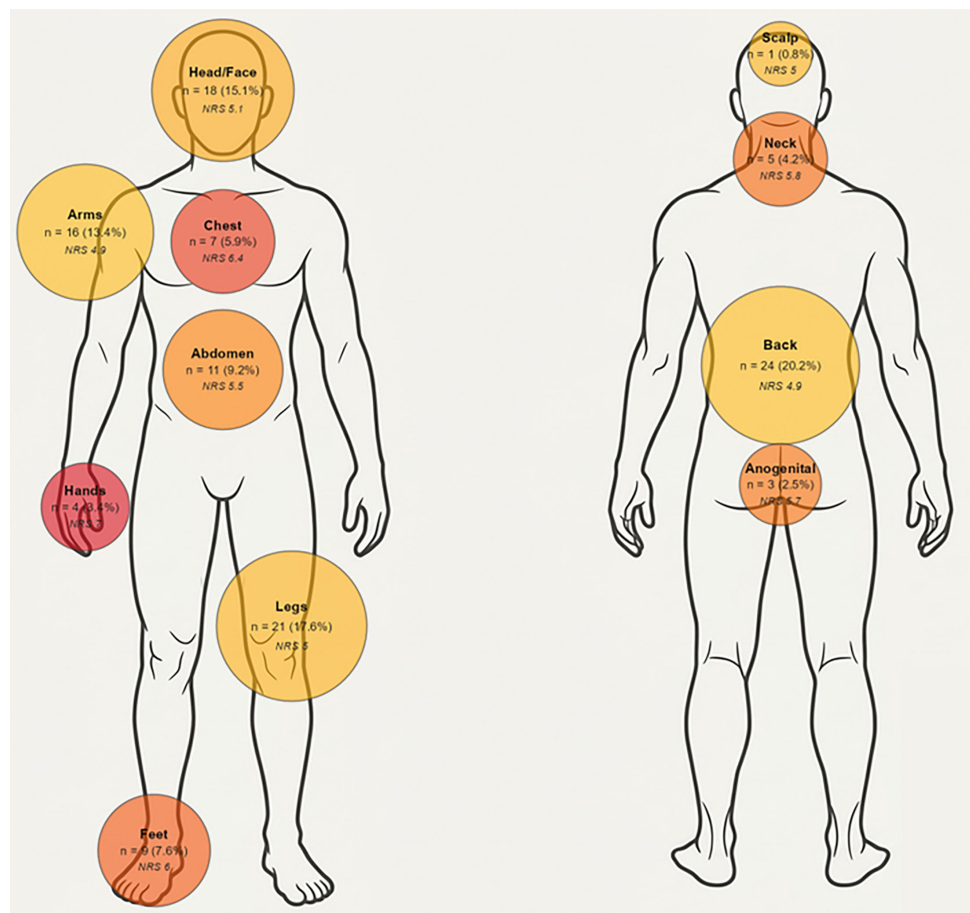


Fig. 3. Anatomical distribution of chronic pruritus in predialysis CKD patients. Data are displayed on anterior (left) and posterior (right) body silhouettes. Circle size is proportional to the number of study visits in which the respective body region was reported as affected (N); circle colour represents the mean pruritus intensity (numerical rating scale (NRS), NRS 0–10) among visits where that region was involved, ranging from green (Mild: 1 to 3) through yellow/orange (Moderate: 4 to 6) to red (Severe: 7 to 10). Labels indicate the body region, absolute count with percentage of total localisations, and mean NRS. Colour gradient maps linearly to mean NRS (0–10). Multiple body regions could be reported per visit; percentages therefore sum to more than 100%. Observation-level analysis.

predictor was independently associated with ever having chronic pruritus.

The estimates were as follows: PTH, OR 1.006 per pg/mL (95% CI 0.995–1.017; $p=0.283$); eGFR, OR 1.011 per mL/min/1.73 m² (95% CI 0.958–1.068; $p=0.683$); and age, OR 1.026 per year (95% CI 0.979–1.081; $p=0.307$). Model discrimination was poor (AUC=0.603), and all variance inflation factors were below 1.25.

Sensitivity analyses additionally adjusting for phosphate, binary CKD stage, or diabetes mellitus yielded comparable PTH estimates (OR range 1.004–1.006, all $p>0.25$; AUC $\leq$ 0.645). In the observation-level GEE model ($n=219$; 40 events), PTH was likewise not associated with pruritus (OR 1.005, 95% CI 0.998–1.010; $p=0.14$), nor were eGFR ($p=0.929$) or age ($p=0.239$).

Exploratory analyses

Among medications, statin use showed the strongest univariate association with chronic pruritus (OR 2.31, 95% CI 0.90–6.20; $p=0.086$) and remained directionally

similar after adjustment for age and eGFR (OR 2.05, 95% CI 0.77–5.78; $p=0.160$). No other medication showed a notable association.

At the observation level, NRS did not correlate significantly with PTH (Spearman rho=0.118, $p=0.14$, $n=157$), CRP, eGFR, or urea (all $p>0.35$). No significant correlations were observed between NRS and CRP ($\rho=-0.154$, $p=0.493$), eGFR ($\rho=0.131$, $p=0.361$), or urea ($\rho=-0.111$, $p=0.437$).

In supplementary Random Forest and XGBoost models, age and haemoglobin as ranked highest, followed by BMI, urea and PTH (Fig. S1). These analyses are hypothesis-generating only.

DISCUSSION

This prospective study provides a longitudinal and detailed clinical characterization of chronic pruritus in a predialysis CKD cohort followed for up to 3 years: Chronic pruritus is common across the nondialysis CKD spectrum, with approximately one in 3 patients reporting symptoms at least once during follow-up.

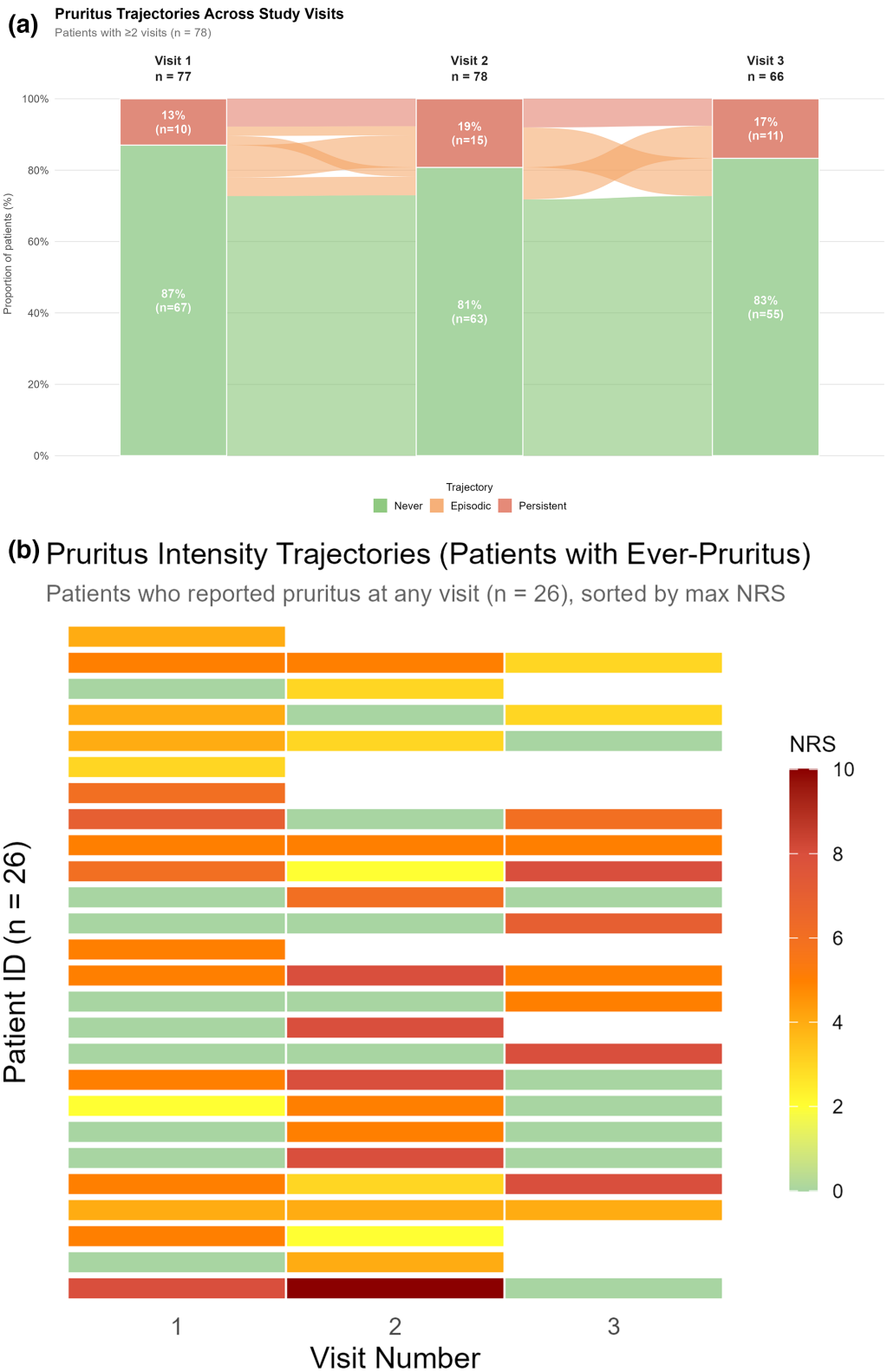


Fig. 4. a Pruritus prevalence trajectories. Alluvial plot - Stacked bars represent the proportion of patients with and without chronic pruritus at each visit among patients with at least two documented visits ($n=78$). Flows between visits are colour-coded by trajectory group: green=Never (no chronic pruritus at any visit), orange=Episodic (chronic pruritus at some but not all visits), red=Persistent (chronic pruritus at every visit). **b** Pruritus intensity trajectories: Each row represents one patient, sorted by maximum NRS score across visits. Patients who reported chronic pruritus at any visit ($n=26$), sorted by max NRS. NRS=numerical rating scale

Further, the clinical phenotype already closely resembled that described in dialysis populations, with a bilateral, nondermatomal distribution and frequent concomitant xerosis. Additionally, pruritus showed a fluctuating rather than uniformly progressive course. No single biochemical marker was independently associated, supporting a multifactorial pathophysiology.

The overall prevalence is consistent with contemporary nondialysis CKD data: moderate-to-extreme pruritus affects 24% of nondialysis patients in the CKD Outcomes and Practice Patterns Study (CKDopps) (3, 4), and incident moderate-to-severe pruritus increases with declining eGFR in the Chronic Renal Insufficiency Cohort (CRIC) (19). Taken together, these data argue against viewing CKD-aP as a problem confined to end-stage renal disease and support a broader awareness in predialysis CKD care.

The distribution was bilateral and nondermatomal, closely resembling the phenotype in haemodialysis cohorts (20, 21), suggesting that key features of CKD-aP may already be established before dialysis and are unlikely to be explained solely by dialysis-related factors.

Patient-reported xerosis was present in 72% of affected observations and was the dominant skin-related finding. This is consistent with current pathophysiological models, in which skin dryness and barrier dysfunction are regarded not merely as epiphenomena, but as potential amplifiers of itch in CKD (3, 14–16, 22, 23). As a directly assessable and modifiable component, xerosis supports targeted topical therapy regardless of dialysis status.

A particularly relevant finding is the temporal pattern of symptoms. Among patients with at least 2 visits, episodic pruritus was more common than persistent pruritus, and overall visit-level prevalence remained relatively stable over time. This argues against a simple model in which itch accumulates linearly as kidney function worsens. Instead, our data suggest a waxing-and-waning course, possibly modulated by intercurrent illness, skin condition, medication changes or short-term metabolic and inflammatory fluctuations. This interpretation is biologically plausible and fits with longitudinal dialysis data showing that pruritus severity is dynamic over time rather than fixed, with substantial within-patient variability across repeated assessments (20, 24). To our knowledge, such trajectory data have been largely lacking for predialysis CKD.

In contrast to some prior reports, we found no independent association between chronic pruritus and PTH, phosphate, calcium, CRP, urea or eGFR in multivariable models. The broader literature remains inconsistent: no single laboratory marker has emerged as a robust predictor of CKD-aP across populations (2, 19, 21, 23, 25). In prospective nondialysis cohort data, lower eGFR and several comorbid factors were

associated with incident pruritus, whereas mineral metabolism markers were not consistently predictive (12). Our findings do not exclude a contribution of PTH in patients with more pronounced secondary hyperparathyroidism but do not support a dominant role for mineral metabolism in unselected predialysis patients with moderate biochemical abnormalities.

Clinically, this reinforces an important message: in predialysis CKD, the absence of a “typical” biochemical profile should not deter clinicians from recognizing CKD-aP as a real and burdensome symptom.

An additional exploratory finding was the association with dyslipidaemia and a directionally consistent but nonsignificant trend for statin use. Although drug-induced pruritus has been described for several medication classes (26), this observation may reflect confounding by comorbidity burden rather than a drug-specific effect.

The higher overall comorbidity burden in patients with pruritus is consistent with prior work linking CKD-aP to worse outcomes and impaired quality of life (4–7), suggesting that pruritus may mark a phenotype of increased systemic illness complexity.

Strengths and limitations

Strengths of this study include the prospective design with repeated assessments, anatomical mapping, and the focus on a predialysis CKD population that remains underrepresented in the CKD-aP literature. Of note, pruritus was assessed with an NRS and structured interviews rather than currently discussed instruments such as the WI-NRS (19, 26).

Several limitations should also be acknowledged. Although the data were collected between 2013 and 2015, eGFR was re-calculated using the current CKD-EPI 2021 race-free (24), and KDIGO eGFR staging categories have remained broadly comparable (18, 27).

The clinical phenotype, anatomical distribution and longitudinal trajectory of predialysis CKD-aP have not been systematically re-examined since; recent large analyses focus either on incidence in CRIC (19) or clinical outcomes in CKDopps (4, 28), while longitudinal trajectory data remain largely dialysis-based (25, 29). Large databases such as CKDopps and CRIC relied on brief self-reported itch questions rather than detailed anatomical and dermatological phenotyping (4, 7, 12).

Our descriptive findings therefore retain relevance although the sample size was modest, limiting statistical power for subgroup analyses and for detecting smaller effect sizes. A true association of limited magnitude – particularly for PTH or other biochemical variables – therefore cannot be excluded. Second, the temporal resolution was

limited by the relatively small number of visits per patient, so the trajectory analysis should be understood as descriptive rather than definitive. Third, mechanistic domains including formal skin barrier assessment, itch-related cytokines, and emollient documentation were not captured, since the assessment of skin phenotype was based on nurse administered questionnaires.

Conclusion

Chronic pruritus affects approximately 30% of patients with predialysis CKD and presents with a bilateral, nondermatomal distribution pattern that is phenotypically consistent with CKD-aP as described in dialysis populations. The temporal course is frequently episodic rather than uniformly progressive. No individual laboratory parameter - including PTH, eGFR, phosphate, calcium, CRP or urea - independently predicted pruritus occurrence, reinforcing current multifactorial pathophysiology models. These findings suggest that systematic clinical phenotyping - documenting pruritus distribution, intensity, temporal patterns and associated skin characteristics - may be more valuable for early recognition and monitoring of CKD-aP than biomarker-based risk stratification.

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Data availability statement: The data that support the findings of this study are not publicly available due to data protection requirements and the absence of participant consent for open data sharing. De-identified datasets are available from the corresponding author upon reasonable request and subject to approval by the local ethics committee.

Ethics committee: The study was approved by the Institutional Review Board of the Gesellschaft zur Förderung der Forschung an der DKD (approval no. IRB-2012-2) and by the local ethics committee of the Nephrology Center Wiesbaden. All participants provided written informed consent prior to inclusion. The study was conducted in accordance with the Declaration of Helsinki. No trial registry identifier applies to this observational study.

The authors have no conflicts of interest to declare.

REFERENCES

- Mettang T, Kremer AE. Uremic pruritus. *Kidney Int* 2015; 87: 685–691. <https://doi.org/10.1038/ki.2013.454>
- Schricker S, Kimmel M. Unravelling the pathophysiology of chronic kidney disease-associated pruritus. *Clin Kidney J* 2021; 14: i23–i31. <https://doi.org/10.1093/cckj/sfab200>
- Scherer JS, Tu C, Pisoni RL, Speyer E, Lopes AA, Wen W, et al. CKD-associated pruritus and clinical outcomes in nondialysis CKD. *Kidney Med* 2024; 6: 100754. <https://doi.org/10.1016/j.xkme.2023.100754>
- Sukul N, Speyer E, Tu C, Bieber BA, Li Y, Lopes AA, et al. Pruritus and patient reported outcomes in non-dialysis CKD. *Clin J Am Soc Nephrol* 2019; 14: 673–681. <https://doi.org/10.2215/CJN.09600818>
- Weiss M, Mettang T, Tschulena U, Weissshaar E. Health-related quality of life in haemodialysis patients suffering from chronic itch: results from GEHIS (German Epidemiology Haemodialysis Itch Study). *Qual Life Res* 2016; 25: 3097–3106. <https://doi.org/10.1007/s11136-016-1340-4>
- Narita I, Alchi B, Omori K, Sato F, Ajiro J, Saga D, et al. Etiology and prognostic significance of severe uremic pruritus in chronic hemodialysis patients. *Kidney Int* 2006; 69: 1626–1632. <https://doi.org/10.1038/sj.ki.5000251>
- Wang T, Goh JX, Seth S, Do LL, Tesfaye W, Sud K, et al. Chronic kidney disease-associated pruritus and patient-centred outcomes: A systematic review. *J Nephrol* 2025; 38: 371–391. <https://doi.org/10.1007/s40620-025-02221-9>
- Hu X, Sang Y, Yang M, Chen X, Tang W. Prevalence of chronic kidney disease-associated pruritus among adult dialysis patients: A meta-analysis of cross-sectional studies. *Medicine (Baltimore)* 2018; 97: e10633. <https://doi.org/10.1097/MD.00000000000010633>
- Pisoni RL, Wikström B, Elder SJ, Akizawa T, Asano Y, Keen ML, et al. Pruritus in haemodialysis patients: International results from the Dialysis Outcomes and Practice Patterns Study (DOPPS). *Nephrol Dial Transplant* 2006; 21: 3495–3505. <https://doi.org/10.1093/ndt/gfl461>
- Weiss M, Mettang T, Tschulena U, Passlick-Deetjen J, Weissshaar E. Prevalence of chronic itch and associated factors in haemodialysis patients: A representative cross-sectional study. *Acta Derm Venereol* 2015; 95: 816–821. <https://doi.org/10.2340/00015555-2087>
- Makar M, Smyth B, Brennan F. Chronic kidney disease-associated pruritus: A review. *Kidney Blood Press Res* 2021; 46: 659–669. <https://doi.org/10.1159/000518391>
- Khanna D, Singal A, Kalra OP. Comparison of cutaneous manifestations in chronic kidney disease with or without dialysis. *Postgrad Med J* 2010; 86: 641–647. <https://doi.org/10.1136/pgmj.2009.095745>
- Wulczyn KE, Rhee EP, Myint L, Kalim S, Shafi T. Incidence and risk factors for pruritus in patients with nondialysis CKD. *Clin J Am Soc Nephrol* 2023; 18: 193–203. <https://doi.org/10.2215/CJN.09480822>
- Solak B, Acikgoz SB, Sipahi S, Erdem T. Epidemiology and determinants of pruritus in pre-dialysis chronic kidney disease patients. *Int Urol Nephrol* 2016; 48: 585–591. <https://doi.org/10.1007/s11255-015-1208-5>
- Duque MI, Thevarajah S, Chan YH, Tuttle AB, Freedman BI, Yosipovitch G. Uremic pruritus is associated with higher kt/V and serum calcium concentration. *Clin Nephrol* 2006; 66: 184–191. <https://doi.org/10.5414/cnp66184>
- Welter E de Q, Frainer RH, Maldotti A, Losekann A, Weber MB. Evaluating the association between alterations in mineral metabolism and pruritus in hemodialysis patients. *An Bras Dermatol* 2011; 86: 31–36. <https://doi.org/10.1590/s0365-05962011000100003>
- Inker LA, Eneanya ND, Coresh J, Tighiouart H, Wang D, Sang Y, et al. New Creatinine- and Cystatin C-Based Equations to Estimate GFR without Race. *N Engl J Med* 2021; 385: 1737–1749. <https://doi.org/10.1056/NEJMoa2102953>
- Chou FF, Ho JC, Huang SC, Sheen-Chen SM. A study on pruritus after parathyroidectomy for secondary hyperparathyroidism. *J Am Coll Surg* 2000; 190: 65–70. [https://doi.org/10.1016/s1072-7515\(99\)00212-4](https://doi.org/10.1016/s1072-7515(99)00212-4)
- Rigatto C, Collister D, Granger-Vallée A, Girard L, Hingwala J, Karaboyas A, et al. Pathways for diagnosing and treating CKD-associated pruritus: a narrative review. *Can J Kidney Health Dis* 2024; 11: 20543581241238808. <https://doi.org/10.1177/20543581241238808>
- Mathur VS, Lindberg J, Germain M, Block G, Tumlin J, Smith M, et al. A longitudinal study of uremic pruritus in

hemodialysis patients. Clin J Am Soc Nephrol 2010; 5: 1410–1419. <https://doi.org/10.2215/CJN.00100110>

21. Martin CE, Clotet-Freixas S, Farragher JF, Hundemer GL. Have we just scratched the surface? A narrative review of uremic pruritus in 2020. Can J Kidney Health Dis 2020; 7: 2054358120954024. <https://doi.org/10.1177/2054358120954024>
22. Ständer S, Weisshaar E, Mettang T, Szepietowski JC, Carstens E, Ikoma A, et al. Clinical classification of itch: A position paper of the International Forum for the Study of Itch. Acta Derm Venereol 2007; 87: 291–294. <https://doi.org/10.2340/00015555-0305>
23. Brennan F. The pathogenesis of CKD-associated pruritus: A theoretical model and relevance for treatment. Kidney360 2024; 5: 1727–1738. <https://doi.org/10.34067/KID.0000000573>
24. Vervloet MG, Tu C, Fuller S, Fotheringham J, Pisoni RL, Schaufler T, et al. Longitudinal trajectories of CKD-associated pruritus. Kidney Int Rep 2026; 11: 86–93. <https://doi.org/10.1016/j.ekir.2025.10.023>
25. Verduzco HA, Shirazian S. CKD-associated pruritus: New insights into diagnosis, pathogenesis, and management. Kidney Int Rep 2020; 5: 1387–1402. <https://doi.org/10.1016/j.ekir.2020.04.027>
26. Vernon M, Ständer S, Munera C, Spencer RH, Menzaghi F. Clinically meaningful change in itch intensity scores: An evaluation in patients with chronic kidney disease-associated pruritus. J Am Acad Dermatol 2021; 84: 1132–1134. <https://doi.org/10.1016/j.jaad.2020.06.991>
27. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int 2024; 105: S117–S314. <https://doi.org/10.1016/j.kint.2023.10.018>
28. Ståhle-Bäckdahl M, Hägermark O, Lins LE, Törring O, Hilliges M, Johansson O. Experimental and immunohistochemical studies on the possible role of parathyroid hormone in uraemic pruritus. J Intern Med 1989; 225: 411–415. <https://doi.org/10.1111/j.1365-2796.1989.tb00104.x>
29. Huang AH, Kaffenberger BH, Reich A, Szepietowski JC, Ständer S, Kwatra SG. Pruritus associated with commonly prescribed medications in a tertiary care center. Medicines (Basel) 2019; 6: 84. <https://doi.org/10.3390/medicines6030084>