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Article

Diagnostic Accuracy and Clinical Determinants of Urinary Podocyte Biomarkers in Paraprotein-Related Kidney Disease

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Abstract

Background/Objectives Kidney injury is a frequent complication of multiple myeloma (MM) and monoclonal gammopathies. Podocyte stress markers, such as urinary nephrin and podocin, have been studied in other renal diseases but their utility in paraprotein-related kidney disease remains unclear. This pilot study investigated the association of urinary nephrin and podocin levels with albuminuria and biopsy-proven podocytopathy in patients paraprotein-related diseases. **Methods** We retrospectively analyzed 75 patients with plasma cell dyscrasias including MM and MGRS and 11 healthy controls. Urinary podocin and nephrin mRNA levels were measured using qPCR, and urinary podocin protein levels were quantified via ELISA. Associations were assessed between these biomarkers and urinary protein-to-creatinine ratio (uPCR), albumin-to-creatinine ratio (uACR), and histologically confirmed podocytopathia. Diagnostic performance was evaluated using Receiver Operating Characteristic (ROC) analysis. **Results** Higher urinary podocin protein levels were significantly associated with lower uACR ($p=0.007$) and uPCR ($p=0.026$). Neither podocin nor nephrin mRNA showed significant associations with proteinuria metrics. ROC analysis indicated that podocin ELISA (AUC=0.350) and podocin mRNA (AUC=0.510) lacked diagnostic accuracy for predicting renal involvement. The presence of urinary tract infection (UTI) was a significant confounder, leading to increased levels of podocin and nephrin mRNA. **Conclusions** Urinary podocin shows a trend toward elevation in MM/MGRS patients with histological podocyte injury. The study revealed an unexpected inverse association between urinary podocin and albuminuria, suggesting complex release kinetics or stage mismatches in this population. Given the confounding effect of UTIs and the pilot nature of this study, further research is required to validate these podocyte proteins as prognostic biomarkers in paraprotein-related kidney disease.

Keywords: multiple myeloma; paraproteinemias; podocytes; intracellular signaling peptides and proteins; biomarkers

1. Introduction

Kidney injury is a well-recognized complication of monoclonal gammopathies and multiple myeloma (MM).[1,2] While tubular injury from free light chains and cast nephropathy is often the focus, glomerular lesions—especially podocyte injury, or podocytopathy—contribute to proteinuria and renal dysfunction in this context [3–6]. Distinguishing glomerular damage with podocyte injury

from purely tubular injury has diagnostic and prognostic implications and might allow for non-invasive longitudinal evaluation of renal response to treatment.

Podocyte-derived biomarkers detectable in urine, such as nephrin and podocin, offer a potential noninvasive window into glomerular injury.[7–9] Nephrin, a transmembrane protein of the slit diaphragm, plays a central role in filtration barrier integrity and intracellular signaling, and perturbations in nephrin expression or phosphorylation are linked to podocyte dysfunction.[10–12] Podocin, an integral membrane protein interacting with nephrin and cytoskeletal linkers, is critical for anchoring slit diaphragm complexes and has been studied in podocyte injury models and glomerular disease.[13–16] In non-myeloma glomerular diseases, detectable nephrinuria and podocinuria have been correlated with disease activity as well as chronic histological damage.[17,18] For example, urinary nephrin has been proposed as an early indicator of glomerular injury, sometimes preceding overt proteinuria.[19] A recent clinical study also measured urinary nephrin and podocalyxin in various glomerular diseases, finding correlations with disease severity, albeit with low sample size.[17] However, to date, there is very limited direct evidence whether urinary nephrin or podocin levels are associated with podocytopathy in the context of MM or monoclonal gammopathy.

Given the mechanistic plausibility and the unmet need for noninvasive glomerular biomarkers in paraprotein-related kidney disease, we here investigate urinary nephrin and podocin concentrations associated with albuminuria, proteinuria and the presence of biopsy-proven podocytopathy in patients with MM or monoclonal gammopathy of renal significance (MGRS). We test this hypothesis by comparing urinary podocin and nephrin levels in patients with and without histologically confirmed podocyte injury and exploring their relationships with proteinuria metrics such as albumin-to-creatinine ratios (uACR).

2. Materials and Methods

Patient Cohort

We retrospectively identified 75 patients with plasma cell dyscrasias, predominantly multiple myeloma and MGRS, and 11 healthy controls. Frozen urine samples were available from our biobank; for a subset of patients, samples were obtained on the day of diagnostic kidney biopsy. As this work represents a pilot study focusing on processes and methods rather than hypothesis testing or effect estimation, no detailed sample size calculation was planned. We included subjects of at least 18 years of age, who had provided informed consent for sample collection and subsequent storage in our biobank. Exclusion criteria were active participation in an interventional clinical trial within four weeks prior to sample collection. All subjects reported normal urine production and normal hydration levels at the time of sample collection.

Urine Samples

A total of 8 mL of morning urine was centrifuged at 3000 rpm for 10 minutes. The supernatant was immediately frozen at -80°C . The urinary sediment was lysed in 1000 μL Trizol reagent.

RNA Isolation

The RNAzol lysate was mixed with 250 μL chloroform, briefly mixed by frequent inverting, and phase separation was performed by centrifugation at 12,000 g for 15 minutes. The aqueous phase was transferred to a separate tube and RNA was precipitated by adding 500 μL isopropanol followed by centrifugation at 12,000 g for 20 minutes. After a brief wash with 70% ethanol, the RNA pellet was air-dried and resuspended in RNase-free water. The RNA was either used directly for reverse transcription or frozen at -80°C .

Reverse Transcription and Real-Time Quantitative PCR

For qPCR of podocin (Hs00387817_m1) and nephrin (Hs00190446_m1), a TaqMan assay from Applied Biosystems was used. As housekeeping control, a VIC-labeled GAPDH probe set (Applied Biosystems, Foster City, CA) was added to each reaction, with the water volume reduced accordingly. Gene expression levels were analyzed using the $\Delta\Delta CT$ method with normal kidney tissue as reference.

Podocin ELISA

A podocin-specific ELISA was obtained from Abcam (Human Podocin ELISA Kit, ab245708). Urine analysis for podocin concentration was performed according to the manufacturer's instructions.

Statistical Analysis

Categorical data were reported as absolute numbers and relative frequencies, and continuous data as mean \pm standard deviation or median and interquartile range, as appropriate. The main continuous predictors nephrin and podocin were modeled on their original scales.

Pearson correlations with Sidak-corrected p-values were used to assess relationships of the three predictors (nephrin mRNA, podocin mRNA, podocin ELISA) with urinary protein-to-creatinine (uPCR) and albumin-to-creatinine ratios (uACR). uPCR and uACR were log-transformed for normalization.

To test the null hypothesis of no difference between predictors in categorized outcome groups, we applied bootstrapped ANOVA or t-test, as appropriate. To quantify associations between predictors and continuous outcomes, nonparametric Epanechnikov regression with bootstrap confidence intervals was used, as assumptions for ordinary least squares regression were not met.

Receiver operating characteristic (ROC) analyses were performed to assess the diagnostic and discriminatory performance of the assessed podocyte markers in subjects affected with a form of plasma cell dyscrasia and albuminuria. Comparisons between AUCs of the different biomarkers were performed using paired DeLong test.

Data management and analysis were conducted using R version 4.5.1 (R foundation, Vienna, Austria) and Stata 17 (Stata Corp., College Station, TX). A two-sided p-value < 0.05 was generally considered statistically significant. Results are provided as mean \pm standard deviation unless specified otherwise.

Ethics Approval

Approval was obtained from the local ethics committee of the Medical University of Vienna (approval number: 2435/2020)

3. Results

A summary of the patient cohort included in this study is presented in Table 1.

Table 1. Baseline characteristics of the study cohort.

Characteristics	Total cohort (n=86)
Age (years)	67,2 \pm 12,0
sex- n (%)	
- male	49 (57 %)
- female	37 (43 %)
BMI	26,4 \pm 4,9

Hematological condition – n (%) - MM - MGRS - LC Amyloidosis - MGUS - healthy controls	56 (65,1 %) 3 (3,5 %) 8 (9,3 %) 8 (9,3 %) 11 (12,8 %)
Laboratory parameters: - uPCR (mg/g) - uACR (mg/g) - eGFR (mL/min/1.73m ²) - Serum total protein (g/l) - Serum Albumin (g/l) - HbA1c (%) - Presence of microhematuria (%)	1016,5 ± 2551,3 570,5 ± 2180,3 63,6 ± 31,3 65,4 ± 8,8 40,2 ± 5,8 5,5 ± 0,5 15 (17,9 %)
Comorbidities – n (%) - arterial hypertension - type II diabetes mellitus - coronary heart disease - heart disease - glomerulonephritis - kidney transplantation Histomorphology - n (%) - number of biopsies subjects - evidence of podocytopathia in >20 of glomeruli	38 (44,1 %) 10 (11,8 %) 9 (10,6 %) 3 (3,5 %) 3 (3,5 %) 6 (7,0 %) 23 (26,4 %) 11 (47,8 %)

Higher levels of podocin as assessed by ELISA were significantly associated with lower amounts of urinary protein- and albumin excretion (uPCR: $\beta = -0.158$, 95%CI -0,311 to 0.059, $p=0.026$; uACR: $\beta = -0.265$, 95%CI -0,487 to 0,094, $p=0.007$). Both podocin-mRNA and nephrin-mRNA did not show significant association with uPCR or uACR. (Table 2)

Table 2. Association between urinary podocyte biomarkers with urinary protein-creatinine-ratio and urinary albumin-creatinine-ratio.

variable	β (95% CI) Podocin-ELISA	p	β (95% CI) Podocin-mRNA	P	β (95% CI) Nephrin-mRNA	P
uPCR (mg/g)	-0,158 (-0,311; 0,059)	0,026	0,043 (-0,175; 0,103)	0,591	-0,045 (-0,233; 0,120)	0,643
uACR (mg/g)	-0,265 (-0,487; 0,094)	0,007	0,021 (-0,354; 0,125)	0,942	-0,026 (-0,398; 0,266)	0,878

ROC analysis, performed to evaluate the diagnostic performance of the podocyte markers in patients with plasma cell dyscrasia and albuminuria, demonstrated limited diagnostic accuracy for podocin RNA (AUC 0.510, 95% CI 0.354–0.666), podocin ELISA (AUC 0.350, 95% CI 0.202–0.498), and nephrin mRNA (AUC 0.350, 95% CI 0.202–0.498). No significant difference in predictive performance was observed between models ($p = 0.352$). (Figure 1).

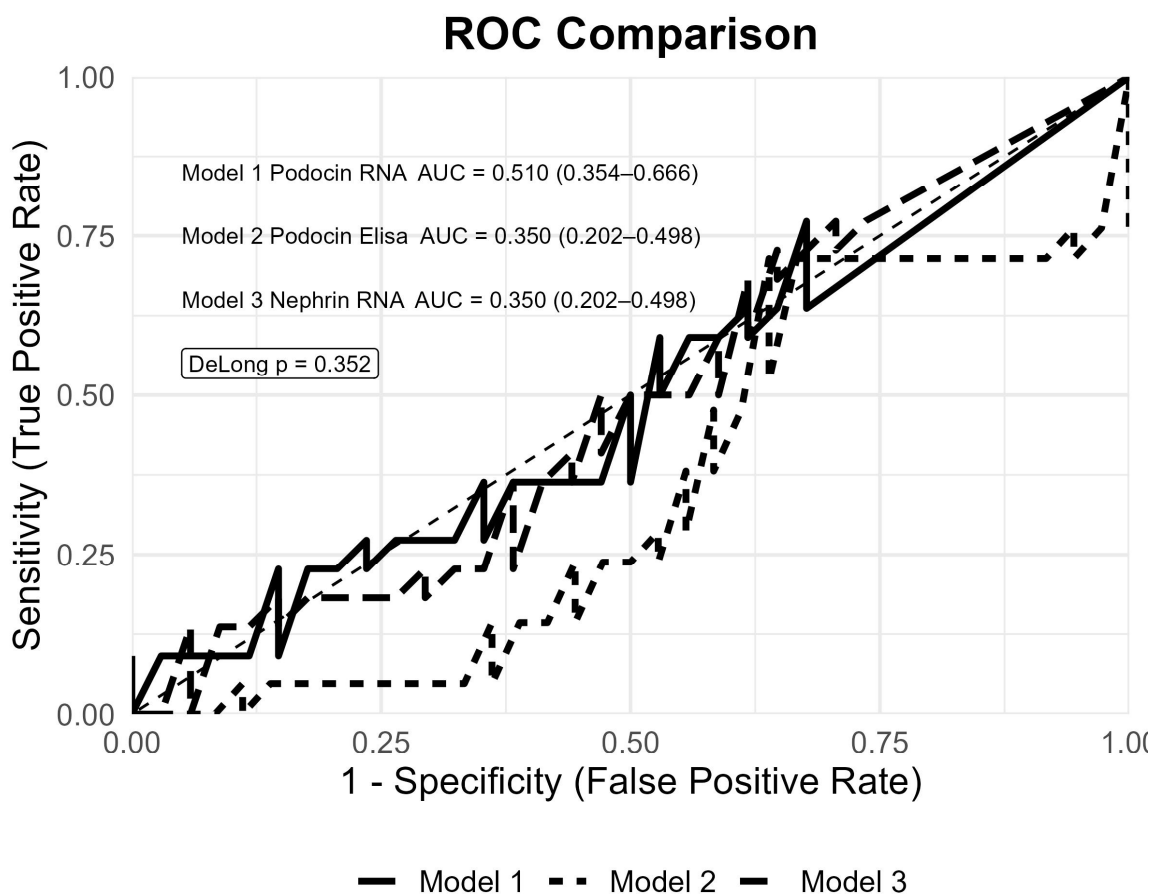


Figure 1. Diagnostic performance of urinary podocyte biomarkers for predicting kidney involvement in Multiple Myeloma.

Importantly, both podocin and nephrin mRNA were significantly increased when urinary tract infection (UTI) was present, while no significant differences in protein levels were shown for podocin ELISA ($p=0.007$, $p=0.080$ and $p=0.293$, respectively). (Table 3).

Table 3. Impact of concurrent urinary tract infection (UTI) on urinary podocyte biomarker controls.

	UTI excluded	UTI present	p-value
n	65	19	
Marker			
Podocin ELISA	8,85 ± 2,33	9,00 ± 4,38	0,293
Podocin mRNA	6,56 ± 4,72	9,83 ± 4,51	0,007
Nephrin mRNA	7,05 ± 4,30	9,82 ± 3,13	0,080

Protein levels of podocin and mRNA levels of both podocin and nephrin did not differ significantly between different forms of MGRS, MM or healthy controls (Table 4).

Table 4. Comparison of urinary podocyte biomarkers across the spectrum of plasma cell dyscrasias and association between urinary podocyte biomarkers and biopsy-proven podocytopathy.

	MM	MGRS	LC Amyloidosis	MGUS	Control	p-value
n	56	3	8	8	11	
Marker						

Podocin ELISA [pg/ml]	9,22 ± 2,62	8,27 ± 1,54	6,36 ± 4,53	9,82 ± 1,63	3,92 ± 3,92	0,183
Podocin mRNA*	7,27 ± 4,72	10,38 ± 1,81	8,67 ± 4,76	4,13 ± 5,19	7,67 ± 4,63	0,203
Nephrin mRNA*	6,90 ± 4,63	10,70 ± 0,65	9,91 ± 1,89	6,78 ± 4,53	9,32 ± 1,54	0,141

mRNA and protein levels did not differ significantly between other clinical conditions (Supplementary Table 1).

Regression analysis did not show significant association of the assessed parameters with histology confirmed podocytopathy, however, a trend towards significance was detected for podocin ELISA ($\beta=0.200$, 95%CI 0.017 to 0.414, $p=0.097$). (Table 5).

Table 5. Association between urinary podocyte biomarkers and biopsy-proven podocytopathy.

Variable	β (95% CI) Podocin-ELISA	p-value	β (95% CI) Podocin-mRNA	p-value	β (95% CI) Nephrin-mRNA	p-value
Podocytopathia	0,200 (0,017 to 0,414)	0,097	-0,106 (-0,396 to 0,097)	0,433	-0,334 (-1,164 to 0,430)	0,458

4. Discussion

In the present work we investigated whether urinary nephrin and podocin in terms of mRNA transcripts or protein are reflective of glomerular injury, as assessed by uACR levels and – where available – biopsy-proven podocytopathy in patients with MM or monoclonal gammopathy. Our results revealed a non-significant trend toward higher urinary podocin protein levels in subjects with histologic podocyte injury. Interestingly and counter-intuitively, we also found elevated podocin to be significantly associated with lower ACR.

Although the association did not reach statistical significance, likely due to lack of statistical power as a reflection of the highly experimental nature of our study, the direction of the effect is biologically credible as podocin release into urine might reflect podocyte stress, detachment, or slit diaphragm disruption prior to or concurrent with visible glomerular injury.[20] This is in line with previous reports, demonstrating podocyturia as an early marker of glomerular damage in various kidney diseases and is in agreement with previous findings by Jiminez et al regarding the prognostic role of urinary podocin and nephrin in diabetic kidney disease.[18] Immunoglobulin- or light chain depositions along glomerular structures can trigger complement activation and may thus preferentially affect podocytes in certain instances.[21–24] The inverse correlation between urinary podocin and ACR is counterintuitive, since albuminuria is often taken as a proxy for glomerular barrier injury.[25] Possible explanations include a temporal mismatch in disease stage, whereby podocin shedding and podocyte fragment release may precede overt glomerular albumin leakage, as well as differences in biomarker kinetics, with podocin being released early in disease progression and subsequently declining in advanced stages.[26] Thus, urinary podocin may represent a different phase of podocyte injury than ACR. Additionally, we found significantly increased levels of urinary podocin and nephrin mRNA where UTI was present despite the absence of convincing evidence for ascending UTI or pyelonephritis. While it is possible that these markers of podocyte stress were elevated as a consequence of systemic inflammation in this context, to the best of our knowledge these findings are novel and warrant further investigation and external confirmation.[27] Importantly, these associations with current UTI, together with low sample size, may explain our expected findings of ROC analysis, where podocin protein levels and nephrin mRNA levels showed worse-than-random predictive performance for renal involvement.

Urinary nephrin did not show robust associations in our analysis. This may reflect differences in release kinetics as experimental models suggest that nephrin shedding earlier in slit diaphragm disruption, while podocin release requires more advanced or severe podocyte detachment.[28] In conditions outside myeloma, nephrinuria correlates with severity of glomerular disease and proteinuria. [17,19]

Strengths of our study include the pairing of urinary podocyte stress marker measurements with histologic confirmation of podocytopathy in a subset of subjects, which is rare in this disease domain. Limitations include modest sample size, heterogeneity of underlying renal manifestations of monoclonal gammopathy or myeloma, cross-sectional design and non-availability of nephrin protein levels. Additionally, we kidney biopsies were performed only in a subset of patients as they were not deemed indicated in the remaining individuals due to clinical lack of evidence of kidney disease.

While our results do not provide definitive answers regarding the clinical usefulness of these biomarkers, our experimental data may inform future trials evaluating the feasibility of podocyte stress markers as prognostic biomarkers in MM and MGRS.

In summary, our study provides preliminary experimental evidence that urinary podocin may trend higher in subjects with biopsy-proven podocytopathy in MM and MGRS and shows an unexpected inverse association with albuminuria, while nephrin mRNA failed to show a clear relationship in this dataset. These results support further exploration of urinary podocyte proteins as noninvasive biomarkers of glomerular injury in paraprotein-related kidney disease in larger collectives or as components of diagnostic biomarker panels. However, our findings warrant caution when assessing podocyte markers in the presence of UTI. Alternatively, nephrin and podocin should be investigated as markers of UTI in specific investigations.[29] Future research should evaluate nephrin and podocin in larger cohorts, integrate multiple biomarkers, and assess longitudinal dynamics.

Supplementary Materials: The following supporting information can be downloaded at the website of this paper posted on Preprints.org. Table S1: P-value table for between-group comparisons of levels of urinary podocyte biomarkers between sexes and/or conditions.

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Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki, and approved by the Ethics Committee of the Medical University of Vienna (approval number 2435/2020).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: Primary data is available upon reasonable request addressed to the corresponding author (oliver.helk@meduniwien.ac.at).

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

LC	Light Chain
uACR	Urinary Albumin-to-Creatinine Ratio
MM	Multiple Myeloma
MGUS	Monoclonal Gammopathy of Unknown Significance

MGRS Monoclonal Gammopathy of Renal Significance

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