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Homoeopathic management of proteinuria in chronic kidney disease: A case series

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Abstract
A rising global public health concern, Chronic Kidney Disease (CKD) is characterised by rising incidence, prevalence with related morbidity and mortality. Diabetes mellitus, Hypertension and Glomerulonephritis have been recognised as the main contributing variables to the multifactorial aetiology of Chronic Kidney Disease (CKD). Other illnesses including Renal calculi, Hyperuricemia and Dyslipidaemia have also been identified as possible risk factors for the onset and advancement of chronic kidney disease. The estimated Glomerular Filtration Rate (eGFR) and Albuminuria are two important clinical indicators for diagnosis and disease monitoring. The primary predictive factor in this study was Albuminuria and all patients underwent a thorough evaluation both before and after receiving Homoeopathic treatment.
Individualised Homoeopathic medications were provided after a thorough case history, clinical examination and evaluation which was supported by necessary laboratory tests. All patients showed clinical improvement within the first month of treatment. Follow-up examinations were performed over the next six months and the urine Albumin-Creatinine Ratio was re-evaluated after treatment. Each patient's illness and post-intervention progress were thoroughly documented clinically and in the laboratory. The case report series adhered to the CARE Checklist guideline and the causal attribution of the Homoeopathic treatment effect was evaluated using the Modified Naranjo Criteria (MONARCH Criteria). However, because this study had a limited sample size and a short follow-up period, more large-scale, high-quality clinical investigations with long follow-up periods are needed to confirm these findings.

Keywords: Homoeopathy, proteinuria, albuminuria, Chronic Kidney Disease (CKD), Estimated Glomerular Filtration Rate (eGFR), Urine Albumin Creatinine Ratio (Urine ACR)

Introduction

Definition: Chronic Kidney Disease (CKD) is defined according to current international guidelines as either a reduction in Renal function specifically, a Glomerular Filtration Rate (GFR) of less than 60 mL/min/1.73 m² or the presence of markers of Kidney damage or both, persisting for a minimum duration of three months, irrespective of the underlying cause.
Diagnostic criteria: CKD is diagnosed when one or both of the following criteria are present for at least three months:

Reduced GFR

- GFR < 60 mL/min/1.73 m² (stages G3a-G5).

Markers of kidney damage (one or more)

- Albuminuria (Albumin-creatinine ratio ≥ 30 mg/g)
- Urinary sediment abnormalities
- Electrolyte or other abnormalities due to Tubular disorders
- Histological abnormalities
- Structural abnormalities detected by imaging
- History of Kidney transplantation [1]

Etiology: CKD results from various conditions that progressively impair Renal structure and function. The major etiological factors include:

- **Diabetes mellitus (diabetic nephropathy):** 40% of cases

- **Hypertension (hypertensive nephropathy):** 20% of cases
- **Chronic glomerulonephritis (particularly IgA nephropathy):** 20% of cases other causes encompass:
- **Chronic tubulointerstitial nephritis:** Resulting from chronic pyelonephritis, infections or prolonged drug use
- **Congenital and hereditary disorders:** Such as Polycystic Kidney Disease and Alport syndrome
- **Vascular and systemic diseases:** Including renal artery stenosis, vasculitis, amyloidosis, Systemic Lupus Erythematosus (SLE), rheumatoid arthritis, gout and multiple myeloma [2]

Symptom burden in CKD: One major public health problem is the substantial symptom burden seen in patients with chronic kidney disease (CKD). Low educational attainment and socioeconomic and cultural characteristics have been found to be independent predictors of greater symptom severity. Nonspecific symptoms such Excessive weariness, Oedema, Sleepiness, Hypertension, Muscle twitches and Foamy urine are prevalent in the early stages of chronic kidney disease (CKD). The main causes of the increased symptom burden in advanced stages (Stage 5 and Stage 5 on dialysis) include Uraemia and its related consequences, including as Anaemia, Metabolic abnormalities and Fluid retention [3].

Albumin as a marker of glomerular damage: In glomerular illnesses, Albumin makes up the majority of Proteinuria. It is easy to identify underlying glomerular problems when persistent Albuminuria is present. Diabetic kidney disease's first observable stage is Microalbuminuria, which is the excretion of trace amounts of Albumin in the urine. For detecting Microalbuminuria, the Albumin-to-Creatinine Ratio (ACR) is thought to be the best technique [4].

Pathophysiology of albuminuria: It is thought that limited Albumin filtration takes place under typical physiological conditions, while increased Albumin excretion is the consequence of the interaction between Glomerular filtration and Tubular reabsorption. Long-term increases in Albumin excretion are thought to contribute to Progressive Renal Injury by raising Glomerular Hydraulic Pressure, improving the Glomerular Filtration Coefficient and changing the glomerular membrane's size and charge selectivity [5].

Clinical significance of albuminuria: Albuminuria is a sign of endothelial dysfunction as well as a predictor of the onset and course of renal disease, both Diabetic and Non-diabetic. Even in patients with low estimated Glomerular Filtration Rate (eGFR), recent research has shown that proteinuria is independently linked to higher mortality. As a result, proteinuria is becoming more widely acknowledged as a crucial instrument for risk assessment in individuals suffering from chronic kidney disease (CKD) [6].

Albuminuria in CKD and diabetes: Patients with Type 2 Diabetes Mellitus often have elevated excretion of urine Albumin. Microalbuminuria, also known as moderately high Albuminuria, is a sign of increased cardiovascular morbidity and mortality risk and an early predictor of progressive renal impairment that results in Diabetic Nephropathy. The degree of proteinuria and the risk of developing cardiovascular and renal problems are directly correlated [7].

Hypertension and proteinuria in CKD: Uncontrolled Hypertension over an extended period of time raises Intraglomerular pressure, which hinders glomerular filtration. Microalbuminuria or Overt proteinuria are symptoms of unusually high protein levels caused by increased protein leakage into the urine due to damage to the glomeruli [8].

Detection and clinical implications of albuminuria: The recommended technique for identifying Microalbuminuria is the Albumin-to-creatinine ratio (ACR). End-Stage Renal Disease (ESRD) is more likely to develop in patients who go from Microalbuminuria to Macroalbuminuria (≥300 mg/24 hours). However, when started as soon as possible, early therapies have been demonstrated to lower this risk and decrease the progression of renal disease [4].

Diagnosis and investigation of proteinuria in CKD: In the study of Chronic Kidney Disease (CKD), proteinuria assessment is essential, albeit the best way to do so is still up for debate [9]. The most often utilised biomarkers for CKD evaluation are Albuminuria and estimated Glomerular Filtration Rate (eGFR), and proteinuria primarily indicates glomerular damage. Since measuring GFR directly takes time, endogenous filtration indicators like Cystatin C (CysC) and Serum Creatinine (SCr) are commonly used to estimate GFR [10]. The development of the disease and its clinical results have been closely linked to Albuminuria, which may occur before detectable deteriorations in renal function. One of the earliest signs of chronic kidney disease (CKD) caused by Diabetes, Hypertensive Nephrosclerosis, or other Glomerular abnormalities is increased excretion of Albumin in the urine. When possible, a first-morning urine sample is recommended, but a random, non-timed "spot" urine sample is a good place to start. The Albumin-to-creatinine ratio is the standard way to express the results (ACR) [11]. People with Diabetes, Hypertension, a family history of chronic kidney disease (CKD), or a personal or family history of cardiovascular disease are among the high-risk groups who should have regular Albuminuria screening [11].

Classification of albuminuria: Albuminuria is classified based on the Albumin-to-creatinine ratio (ACR) into three categories:

- **A1:** ACR < 30 mg/g (< 3.4 mg/mmol)
- **A2:** ACR 30-299 mg/g (3.4-34 mg/mmol)
- **A3:** ACR ≥ 300 mg/g (≥ 34 mg/mmol) [12].

Materials and Methods

The White Memorial Homoeo Medical College and Hospital in Kanniyakumari District's Inpatient and Outpatient Departments (IPD/OPD) were used to enrol and monitor all of the patients. Study participants were those with Albuminuria who satisfied the criteria for chronic kidney disease (CKD). Reductions in Albuminuria levels and ameliorations in clinical symptoms were methodically documented throughout the follow-up period.

Diagnostic procedure and assessment

- All cases were diagnosed as chronic kidney disease (CKD) based on the current international guideline criteria [1,13].

Table 1: Classification of Chronic Kidney Disease (CKD)

GFR Category	Description	GFR Range (mL/min/1.73 m²)	Albuminuria Category (ACR mg/g)	Albuminuria Description	Prognosis of CKD (Risk of Progression)
G1	Normal or high	≥ 90	**A1:** < 30	Normal to mildly increased	Low risk (if no other markers of kidney damage)
G2	Mildly decreased	60 - 89	**A2:** 30 - 300	Moderately increased (Microalbuminuria)	Mildly increased risk
G3a	Mild to moderate decrease	45 - 59	**A3:** > 300	Severely increased (Macroalbuminuria)	Moderate to high risk
G3b	Moderate to severe decrease	30 - 44	**A1-A3**	Varies with Albuminuria level	High risk of progression
G4	Severe decrease	15 - 29	**A1-A3**	Varies with Albuminuria level	Very high risk
G5	Kidney failure	< 15	**A1-A3**	Varies with Albuminuria level	Extremely high risk (End-stage renal disease)

- The cases were further classified according to the urine Albumin-to-creatinine ratio (ACR) assessment table [14].

Table 2: Assessment table of Albumin Creatinine Ratio (ACR)

ACR (mg/g)	Assessment	Score
< 30.97 (Female) < 22.12 (Male)	Normal	0
30.972 - 266.49 (Female) 22.12 - 265.49 (Male)	Moderately elevated Albuminuria	1
265.492 - 619.47	Dipstick test becomes positive from this point onwards	2
619.47 - 2654.87	Glomerular disease more likely more than 1 gram/24 hour	3
> 2654.87	Nephrotic range almost always glomerular disease more than 3.5 gm/24 hours	4

- The urine ACR test was repeated after six months to monitor disease progression and assess treatment response. It helped in evaluating the consistency of Albuminuria levels to confirm the improvement of renal damage.

Case presentation

The following eight cases were selected for this study, each exhibiting a urine Albumin-to-creatinine ratio (ACR) above 30 mg/g, representing Microalbuminuria or Macroalbuminuria. All selected patients were considered at risk for progression of chronic kidney disease.

Case 1: On March 5, 2024, a 55-year-old man arrived complaining of four months of nocturia, exhaustion, and generalised oedema. He had Hypertension for fifteen years, which was not well managed with Allopathic treatment. Blood pressure: afebrile, 160/100 mmHg. Lab results: urine Albumin ++, urine ACR 180 mg/g, and FBS 110 mg/dL. Upon physical examination, minor pedal oedema was found. 10 doses of Calcarea carbonica 200 were administered to him along with dietary management. Over the course of six months, the ACR dropped to 95 mg/g with Blood pressure: afebrile, 135/90 and the fortnightly follow-up revealed a progressive decrease in oedema.

Case 2: On April 10, 2024, a 62-year-old woman complained of weariness, dizziness, and polyuria that had persisted for six months. History of Osteoarthritis and type 2 Diabetes with FBS 210 mg/dL. 145/85 mmHg, Afebrile Blood pressure. Lab results: urine Albumin +, urine ACR 75 mg/g. Homoeopathic prescription: 8 doses of phosphorus 200 along with dietary management. The Urine ACR decreased to 28 mg/g over the six- months with follow-ups every fortnight showing FBS 110 mg/dL, 130/80 mmHg Afebrile Blood pressure and symptoms of weariness and light-headedness improved.

Case 3: On May 2, 2024, a 50-year-old man complained of minor oedema and three months of lethargy. 12 years of Hypertension with sporadic medication use. Blood pressure of 155/95 mmHg. Lab results: urine Albumin +, urine ACR 110 mg/g, and FBS 120 mg/dL. 11 doses of sulphur 200 were prescribed along with dietary management. The Urine ACR dropped to 45 mg/g throughout the six-months with follow-ups every fortnight showing Blood pressure 130/80 and energy levels improved.

Case 4: On June 15, 2024, a 45-year-old woman arrived complaining of five months of frothy urine, frequent headaches and exhaustion. Diabetic history and erratic use of Allopathic medications. Blood pressure of 150/90 mmHg. Lab results: urine Albumin +, urine ACR 60 mg/g, and FBS 220 mg/dL. 10 doses of Natrum muriaticum 200 were prescribed to her along with dietary management. The six-months treatment with follow- ups every fortnight revealed clinical improvement and a decrease in urine ACR to 20 mg/g with FBS 125 mg/dL and Blood pressure: 135/85 mmHg.

Case 5: On July 20, 2024, a 58-year-old man complained of minor oedema, generalised weakness, and nocturia. 15 years of history with Hypertension. Blood pressure of 165/100 mmHg. Lab results: urine Albumin ++, urine ACR 200 mg/g, and FBS 115 mg/dL. Prescription: 200 pills of Lycopodium clavatum in 12 doses along with dietary management. After six months of fortnightly follow-up, the oedema resolved and the ACR dropped to 90 mg/g with Blood pressure: 140/90 mmHg.

Case 6: On August 5, 2024, a 52-year-old woman complained of four months of frothy urine, mild dyspnoea, and exhaustion. History of high Blood pressure and Diabetes. 148/88 mmHg is the Blood pressure. Lab results: urine Albumin +, urine ACR 85 mg/g, and FBS 180 mg/dL. 10 doses of Arsenicum album 200 were recommended to

her along with dietary management. Follow-up over six months very fortnight showed ACR reduced to 35 mg/g with FBS 125 mg/dL, Blood pressure: 130/80 mmHg and improved energy levels.

Case 7: On September 12, 2024, a 60-year-old man arrived complaining of weariness, polyuria, and pedal oedema that had persisted for six months. Hypertensive Nephropathy history. Blood pressure of 160/95 mmHg. Lab results: urine Albumin ++, urine ACR 220 mg/g, and FBS 110 mg/dL. 12 doses of Kali phosphoricum 200 were prescribed along with dietary management. The ACR was reduced to 100 mg/g with FBS 99 mg/dL, Blood pressure: 140/80 mmHg and oedema improved during the fortnightly follow-up over six months.

Case 8: On November 25, 2024, a 57-year-old man arrived complaining of minor oedema, exhaustion, and nocturia that had persisted for six months. history of high Blood pressure and Diabetes. 158/96 mmHg is the Blood pressure. Lab results: urine Albumin ++, urine ACR 190 mg/g, and FBS 170 mg/dL. Phosphoric acid 200 was prescribed to him in 11 doses along with dietary management. Six months of fortnightly follow-up revealed an improvement in general health and a decrease in urine ACR to 80 mg/g with FBS 99 mg/dL, Blood pressure: 130/85 mmHg.

Table 3: Analysis of cases

Sl. No.	Presenting complaints	Comorbidities	ACR before (mg/g)	ACR before scoring	ACR after (mg/g)	ACR after scoring	Medicine given
1.	Edema, fatigue, nocturia	Hypertension (10 yrs)	180	A2	95	A2	Calcarea carbonica 200
2.	Polyuria, fatigue, dizziness	Diabetes, Osteoarthritis	75	A2	28	A1	Phosphorus 200
3.	Mild edema, lethargy	Hypertension (12 yrs)	110	A2	45	A1	Sulphur 200
4.	Fatigue, frothy urine, headache	Diabetes (irregular)	60	A2	20	A1	Natrum muriaticum 200
5.	Nocturia, weakness, mild edema	Hypertension (15 yrs)	200	A2	90	A2	Lycopodium clavatum 200
6.	Fatigue, breathlessness, frothy urine	Diabetes, Hypertension	85	A2	35	A1	Arsenicum album 200
7.	Pedal edema, fatigue, polyuria	Hypertensive Nephropathy	220	A3	100	A2	Kali phosphoricum 200
8.	Mild edema, fatigue, nocturia	Diabetes, hypertension	190	A2	80	A2	Phosphoric acid 200

Therapeutic intervention and assessment

As presented in Table 3, all eight cases were diagnosed with Chronic Kidney Disease (CKD) based on a comprehensive evaluation that included detailed medical history, clinical examination and relevant laboratory investigations. The degree of Albuminuria in each case was carefully graded and classified according to the criteria outlined in Table 2, following the standard CKD assessment guidelines.

Each patient was then prescribed an individualized Homoeopathic remedy, selected in accordance with their totality of symptoms, level of susceptibility, constitutional makeup and the pathological nature of the disease. The remedies were administered in appropriate potencies and repetitions, with close follow-up to assess therapeutic response.

Over the course of treatment, all patients reported a gradual improvement in general well-being, with notable relief in associated symptoms such as fatigue, edema, and urinary disturbances. Periodic clinical assessments and repeat laboratory evaluations demonstrated favourable changes in renal function parameters and stabilization of Albuminuria levels, indicating a positive therapeutic outcome with individualized Homoeopathic management.

Follow-up

All patients were closely followed up for a period of six months to monitor clinical progress and renal function. The eight CKD cases treated during 2024-2025 underwent regular evaluations at two-week intervals, during which their symptoms and general health status were carefully assessed. The Homeopathic medicines were repeated or modified as per the patient's response, following standard Homeopathic principles of individualization.

During each review visit, relevant laboratory investigations, including serum creatinine, fasting blood sugar (FBS) and routine urinalysis, were performed to assess systemic and renal parameters. In addition, the urine Albumin-to-Creatinine Ratio (ACR) was re-evaluated after six months to monitor any change in Albuminuria levels and disease progression.

After the six-month follow-up, as in table 4 the MONARCH Inventory was used to assess a causal relationship between Homoeopathic intervention and the outcome of this case. The total scores were 8, 8, 8, 9, 8, 9, 9, 8 in accordance to the case respectively. Thus, suggesting a 'possible' association between the medicine and the outcome. The patient expressed satisfaction with the treatment outcome and reported no adverse effects.

Table 4: Assessment by modified Naranjo criteria (Monarch) score

Domain	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8
1. Was there an improvement in the main symptom or condition for which the homeopathic medicine was prescribed?	+2	+2	+2	+1	+2	+1	+1	+2
2. Did the clinical improvement occur within a plausible time frame relative to the drug intake?	+1	+1	+1	+1	+1	+1	+1	+1

3.	Was there an initial aggravation of symptoms?	0	0	0	0	0	0	0	0
4.	Did the effect encompass more than the main symptom or condition (i.e., were other symptoms not related to main presenting complaint ultimately improved or changed)?	+1	+1	+1	+1	+1	+1	+1	+1
5.	Did overall well-being improve? (suggest using validated scale)	+1	+1	+1	+1	+1	+1	+1	
6A	Direction of cure: did some symptoms improve in the opposite order of the development of symptoms of the disease?	0	0	0	+1	0	+1	+1	0
6B	Direction of cure: did at least two of the following aspects apply to the order of improvement of symptoms:	0	0	0	0	0	0	0	0
•	From organs of more importance to those of less importance? • From deeper to more superficial aspects of the individual? • From the top downwards?								
7.	Did "old symptoms" (defined as non-seasonal and non-cyclical symptoms that were previously thought to have resolved) reappear temporarily during the course of improvement?	0	0	0	0	0	0	0	0
8.	Are there alternate causes (other than the medicine) that with a high probability could have caused the improvement? (Consider known course of disease, other forms of treatment, and other clinically relevant interventions)	0	0	0	+1	0	+1	+1	0
9.	Was the health improvement confirmed by any objective evidence? (e.g., laboratory test, clinical observation, etc.)	+2	+2	+2	+1	+2	+1	+1	+2
10.	Did repeat dosing, if conducted, create similar clinical improvement?	+1	+1	+1	+2	+1	+2	+2	+1
Total		**8**	**8**	**8**	**9**	**8**	**9**	**9**	**8**

Discussion

Proteinuria, particularly in the form of elevated urine Albumin-Creatinine Ratio (ACR), is a well- established marker of renal damage and a strong predictor of chronic kidney disease (CKD) progression. In the present case series, eight patients with Microalbuminuria or Macroalbuminuria and varying comorbidities including long-standing Hypertension, Diabetes Mellitus and lifestyle-related factors were treated with individualized Homoeopathic medicines alongside standard dietary guidance. Across all cases, a consistent reduction in ACR values was observed over a six-month follow-up period, accompanied by improvement in subjective symptoms such as fatigue, oedema, nocturia, and polyuria. The MONARCH criteria scores were indicating positive casual relationship between the medical intervention and the clinical outcome.

The positive response observed in these cases aligns with several earlier Homoeopathic reports that have suggested potential benefits of individualized prescribing in chronic systemic diseases. Individualized selection based on totality of symptoms remains a core principle of Homoeopathic practice. The present series demonstrates that such an approach may hold promise in patients presenting with proteinuria, especially when used as an adjunct to dietary regulation and ongoing conventional monitoring.

Another noticeable trend in this study was the improvement in Blood pressure and Fasting Blood Glucose in several patients. While these changes may partly reflect adherence to lifestyle advice or natural disease variability, stabilization of these parameters likely contributed to the reduction in Albuminuria, given their well-established role in renal hemodynamic. The observed symptomatic relief reduction in oedema, improved energy levels and decreased urinary complaints further supports the potential integrative value of Homoeopathy in managing early renal compromise.

However, the findings of this case series should be interpreted with caution. Case reports are inherently limited by the absence of control groups, small sample size and susceptibility to confounding factors. Spontaneous remission, improved compliance with dietary instructions or variations in underlying disease severity could have influenced outcomes. Additionally, the short follow-up period precludes conclusions regarding long-term renal protection or sustained reduction in proteinuria. Laboratory variations across cases are further limitations.

Despite these constraints, the consistent downward trend in ACR across all eight cases is noteworthy and warrants further exploration. These preliminary observations highlight the need for larger, well-designed clinical studies such as randomized controlled trials or prospective cohort studies to better evaluate the potential therapeutic role of individualized Homoeopathic treatment in early-stage CKD and proteinuria. Future research should also examine long-term renal outcomes, integration with conventional care and potential mechanistic pathways. In the present study, a notable reduction in urinary Albumin-to-Creatinine Ratio (ACR) was observed following Homoeopathic intervention, suggesting a potential role for individualized Homoeopathy in the management of CKD-related Albuminuria.

Conclusion

The use of individualized Homoeopathic medicines in the management of Albuminuria in CKD patients demonstrated notable therapeutic benefits. Homoeopathy was found to be safe, cost-effective and well-tolerated, with no adverse effects or complications observed during the study period. Additionally, patients experienced marked symptomatic improvement, contributing to an enhanced quality of life. These findings suggest that individualized Homoeopathic intervention may serve as a valuable approach in the supportive management of CKD, particularly in early stages or in patients seeking non-invasive treatment options.

Consent of the patient

The patient has provided written informed consent for the inclusion of their clinical information in this publication. All

identifying details have been omitted to ensure anonymity and maintain ethical standards.

Conflict of interest: Nil

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