MATHEWS

Case Report

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Chronic Kidney Disease-An Escalating Public Health Crisis! India Case Study

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ABSTRACT

Type 2 diabetes mellitus (T2DM) has become the main cause of end stage renal disease (ESRD) in the past decade followed by hypertensive nephrosclerosis (HN). Apart from diabetic nephropathy (DN) & hypertension Nephropathy (HN). Glomerulonephritis (GN) in India is frequently caused by post-infectious complications, autoimmune disorders, vasculitis, and hereditary factors. Prolonged use of Nonsteroidal anti-inflammatory drugs (NSAIDs) contributes significantly to the burden. In many cases, the exact cause remains unknown (Idiopathic crescentic GN). By 2040, CKD is projected to become a top five causes of death in India, with mortality potentially exceeding half a million annually. Strengthening diabetes and hypertension control, integrating an early CKD detection program, and ensuring equitable access to renal replacement therapy are urgently needed to bend the trajectory. However, very often young general practitioners struggle to diagnose and manage at least the top two causes of DN &HN leading to chronic kidney disease (CKD) and ESRD. This article is to equip such young professionals with requisite skills and knowledge. Materials & Methods: This article is an outcome of the challenges faced in managing three CKD cases one each due to Diabetes, Hypertension and DKD with MCD by the author in last one year. It is intended to update Family Physicians to distinguish DKD and HKD in their routine practice and minimize the challenge of Screening for underlying conditions and not to prematurely conclude that Diabetes or Hypertension are the sole cause of CKD, as other glomerular nephritides can co-exist. Outcome: All three anecdotal cases reported in this article were managed well with the support of Cardiac and Nephrology specialists. Aggressive reduction of Hb1Ac, FBS and Hypertension were the key approaches

Keywords & Abbreviation's: Type 2 diabetes mellitus =**T2DM, HT**= Hypertension, FBS= Fasting Blood Sugar, Hb1Ac= Glycolate Haemoglobin, **SBP**= Systolic BP, **DBP**= Diastolic BP, **CKD**= Chronic Kidney disease, **DKD**= Diabetic Kidney Disease, **HKD**=Hypertension kidney disease, **ESRD**= end stage renal disease, **GN** = Glomerular-Nephritis, NSAIDs= of Nonsteroidal anti-inflammatory drugs, **DKD with MCD**= Diabetic Kidney Disease (DKD)

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with Minimal Change Disease (MCD), **DS**= dialysis services, **KT**= Kidney Transplantation.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) has become the main cause of end stage renal disease (ESRD) in the past decade followed by hypertensive nephrosclerosis (HN), which typically presents as a slow, progressive decline in renal function caused by chronic, poorly controlled high blood pressure damaging renal vessels. Apart from diabetes and hypertension, glomerulonephritis (GN) in India is frequently caused commonly by post-infectious complications (such as post-streptococcal throat or skin infection GN), Infection-related GN (IRGN) including IgA-dominant IRGN, frequently caused by staphylococcal infections, autoimmune disorders (lupus nephritis, IgA nephropathy), vasculitis, and hereditary factors like Alport syndrome. Prolonged use of Nonsteroidal anti-inflammatory drugs (NSAIDs) also notoriously contributes significantly to the burden. In many cases, the exact cause remains unknown (Idiopathic crescentic GN) [1].

Common Symptoms to Watch are i) Cola-coloured or pink urine (haematuria) ii) Foamy urine (indicating excess protein) iii) Swelling (oedema) in the face, hands, feet, or abdomen iv) Unexplained high blood pressure [1,2].

Chronic Kidney Disease (CKD) is an escalating public health crisis in India, as it is expected to affect approximately 148 million (about 10% of estimated 1.48 billion) Indian mid-year population of 2026. As of July 1, 2026, the population is projected at 1,476,625,576, making India as the world's most populous country, comprising around 17.79% of the total global population [2,3]. Driven by rising diabetic nephropathy (36.8% of cases in those >40 years), hypertension, chronic glomerulonephritis, infections, and a significant percentage (16.2%) of cases with unknown causes, the prevalence is expected to reach 12-15% by 2040. The disease is rising rapidly, with an increase in prevalence from 11.12% (2011–2017) to 16.38% (2018–2023) among adults, with higher rates in rural areas [4].

A 2025 systematic review covering 2005–2025 found a pooled CKD prevalence of 14.4% in India, reinforcing the urgent need for national screening programs. In early 2025, the FDA approved new treatments, such as injectable semaglutide, for CKD in people with type 2 diabetes, which are relevant to India's high metabolic-driven burden. India has the second-highest number of CKD cases globally, trailing only China [4]. The disease is rapidly increasing, with some reports showing a prevalence up to 17.2%. The primary drivers are diabetic nephropathy (36.8% of cases in those >40 years) and hypertension. Other factors include chronic glomerulonephritis, infections, and a significant percentage (16.2%) of cases with unknown causes. Although often considered an urban lifestyle disease, a 2025 meta-analysis showed higher CKD prevalence in rural areas (15.34%) compared to urban areas (10.65%). High blood pressure, diabetes, high salt consumption, and prolonged use of NSAIDs are some of the risk factors. A 2025 study on indigenous populations in Kerala found a 22.6% prevalence of CKD, with a high proportion in early subclinical stages [2].

Most patients present at advanced stages (Stage 4 or 5). Less than 10% of end-stage renal disease patients have access to renal replacement therapy (dialysis or transplant). While 1.75 to 2 lakh patients start dialysis annually, only about 12,000–15,000 transplants are performed, failing to meet the massive demand. The Pradhan Mantri National Dialysis Program (PMNDP), launched on April 7, 2016, under the National Health Mission (NHM), provides free haemodialysis and peritoneal dialysis services to poor, BPL (Below Poverty Line) patients in district hospitals across India [3].

This article is an outcome of the challenges faced in managing three CKD cases in last one year. It is intended to update Family Physicians to distinguish DKD and HKD in their routine practice.

CASE REPORTS

Case 1. Diabetic Nephropathy: Ramesh an 85-year-old man presented to the author with 6 months of fatigue, burning pain in his toes, due to which he stopped his daily morning walk of about 3 Kms since a month. His Blood Sugar Fasting 145 mg/Dl and Hb1Ac -7.8%. showed hidden but late onset diabetes. He was put on oral antidiabetics in January 2026. In the first week of April 2026, he reported with lower extremity oedema gradually worsening. He has a 20-year history of border line diabetes managed by diet and exercises. Since he stopped walking about a year ago, due to age related fatigue it was branded as poorly controlled T2DM. He had undergone CABG in 2002. His medications include metformin, amlodipine, and atorvastatin. T2DM history was present in his mother, brother, and two of the 4 sisters. His father died of a myocardial infarction at age 95. He has no history of smoking,

alcohol, or recreational drug use and did not report any recent illnesses, fevers, weight loss, dysuria, polyuria, dyspnoea or orthopnoea. He had no eye exams in last 5 years.

Physical examination revealed the patient to be 178 cm tall and weighs 70kg. His body mass index (BMI) is 21 with blood pressure of 128/78 mmHg, heart rate of 80 bpm, respiratory rate of 14 bpm, and temperature of 37 °C. Cardiopulmonary, abdomen, CNS exam reveals normal features. He had 1+ pitting Oedema of the lower extremities without any rashes /erythema. His Lab Investigations report of 15 April 2026 showed i) A urine sample appears dark yellow with a foamy surface. Urine dipstick shows 2+ protein, no blood, ++ glucose ii) His HbA1c was 9%, Serum Creatinine 1.28 mg/dl. electrolytes like Sodium (1.3 mmol/l), Potassium (3.26 mmol/l) and Chlorides (90.7 mmol/l) were moderately low. EGFR was 50%. Ultrasound findings showed increased renal cortical echogenicity, reduced renal parenchyma thickness, and kidneys were enlarged a bit.

Case Report 2: A case of Diabetic Kidney Disease (DKD) with Minimal Change Disease (MCD): A 49-year-old male patient with a diabetes duration of 3 years was admitted to our secondary care hospital for "bilateral lower extremity oedema for 1 month. His creatinine was 1.4, and the GFR was 39 and dropping rapidly. His nephrologist announced that nothing to worry now but in 10 yrs from now he will be on dialysis or need Kidney transplantation. What really should have been done is to assess why the renal function was decreasing and then advised how to slow it down. The most common cause of renal disease is high blood pressure not well controlled and or diabetes. In such patients, if there is protein in the urine as large amounts of protein suggest more serious forms of renal disease. The clinical manifestations were nephrotic syndrome and diabetic nephropathy, which were confirmed by renal biopsy. According to the medical history, DKD with MCD was considered. The patient received glucocorticoid for 6 months and was completely relieved of proteinuria. Ultrasound findings showed increased renal cortical echogenicity, reduced renal parenchyma thickness marginally.

Case Report 3. Hypertension Induced CKD: A 75-year-Mr Sridhar visited an outpatient clinic for a routine Cardiac care for monitoring Hypertensiion in January 2025. Physical examination showed his BP was 180/128. He was taking NSAIDs for his Rheumatoid arthritis of both knees for over a decade. After a routine ECG and Tred-meal test, did not alarm much. As first stage he was put on Initial Core Therapy of combining an ACEI with a CCB and a potent diuretic (Lasix). Two weeks later the BP had not responded and read 167/112. Stage 2 the diuretic was changed to a long-acting thiazide-like diuretic such as chlorthalidone for better 24-hour control. A week later the BP was still high at 154/108 in Step 3, a mineralocorticoid receptor antagonist (MRA) namely spironolactone (12.5–50 mg/day) was added as the potassium levels were <4.5 mmol/L. After another fortnight BP was 132/101 still above expected level, a beta-blocker (bisoprolol) was added. As that also did not bring the BP to targeted level of 120/80, direct vasodilator minoxidil was added. After 3 months of multidrug therapy, an ultrasound was done as a precaution to check for any structural changes or complications, helping to ensure his kidneys remain stable and functioning as best as possible. From 4th month onwards slowly one drug after the other in the reverse order were withdrawn and BP Monitored. He is now on only an ACEI with a CCB for the last 6 months.

Discussions: Chronic Kidney Disease (CKD) in India is mostly caused by Diabetes, HT, post-infectious complications, autoimmune disorders, vasculitis, Hereditary factors and Prolonged use of NSAIDs. This article addresses only first three conditions.

Diabetic nephropathy: Type 2 diabetes mellitus (T2DM) has become the main cause of end stage renal disease (ESRD) in the past decade. Diabetic kidney disease (DKD) is one of the most serious complications of diabetes. In clinical practice, the diagnosis of DKD is based primarily on clinical criteria, therefore some non-diabetic kidney diseases (NDKDs) are misdiagnosed as DKD. Renal biopsy is helpful to differentiate diabetes with chronic kidney disease (CKD), however rarely resorted to! DKD and DKD with MCD can be differentiated in the early stage of DKD but are difficult to differentiate in the late stage of DKD [1,6].

Diabetic Nephropathy (DN) begins with structural kidney changes that can precede clinical markers like albuminuria or elevated creatinine. Hyperglycaemia causes nonenzymatic glycosylation of type 4 collagen in the GBM and activates the polyol, hexosamine, and protein kinase C (PKC) pathways. The polyol pathway depletes cellular nicotinamide adenine dinucleotide phosphate (NADPH), disrupting nitric oxide bioavailability. The hexosamine pathway promotes transforming growth factor β1 (TGF-β1) production, leading

to mesangial expansion through deposition of mesangial matrix. Hyperfiltration arises early in DN from increased glomerular capillary pressure due to decreased afferent and increased efferent arteriolar tone. Concurrent activation of vascular endothelial growth factor (VEGF) promotes vascular proliferation and hyaline arteriosclerosis, compounding renal injury. Increased capillary leakiness and proliferation result in plasma exudation, tubular obstruction, and interstitial fibrosis, culminating in nephron dropout and eventual global glomerulosclerosis.

Although albuminuria had traditionally been considered an early marker of DN, but it is now recognized that structural lesions & even impaired eGFR can occur without elevated urine albumin levels. The progression of DN varies significantly, and not all patients progress to end stage disease. T2DM patients with HbA1c below 7% have reduced microvascular complications like DN and retinopathy, especially with blood pressure below 140/90 mmHg. Patients with T2DM taking angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers have a slower rate of developing DN [5].

Diabetic kidney disease (nephropathy) progresses in five stages based on glomerular filtration rate (GFR) and albumin levels in the urine, ranging from hyperfiltration to end-stage renal disease (ESRD). The disease progresses silently over 10–25 years, often causing no symptoms until stages 3-4. Stages of Diabetic Kidney Disease as shown in the chart are:

Stage 1: Hyperfiltration & Early Damage GFR: 90 mg/mmol (Normal or high), Kidney size may increase; damage starts but function is normal or increased.

Stage 2: Mild Kidney Damage GFR: GFR= (60–89 mf/mmol (Mildly reduced) Damage exists, often with intermittent albuminuria but no symptoms.

Stage 3: Moderate Kidney Damage (Incipient Nephropathy) GFR: 30–59 mg mmol. Mild-to-moderate loss of function with persistent Microalbuminuria occurs. Symptoms may begin, such as high blood pressure and mild fluid retention.

Stage 4: Advanced Kidney Damage (Overt Nephropathy) GFR= 15–29 mg. mmol. There is severe reduction in function with high levels of proteinuria. Kidney function drops severely, leading to fatigue, high blood pressure, and potential nausea.

Stage 5: End-Stage Renal Disease (ESRD) / Kidney Failure GFR =< 15 mf/mmol/min. Kidneys fail with symptoms of severe swelling, fatigue, and itching.

Hypertensive Nephropathy (HN): Hypertension-induced chronic kidney disease (CKD), or hypertensive nephrosclerosis (HN), typically presents as a slow, progressive decline in renal function caused by chronic, poorly controlled high blood pressure damaging renal vessels. Key pathological features include benign nephrosclerosis, vascular thickening, moderate proteinuria, and end stage renal disease (ESRD in advanced cases. Management involves intensive BP control using multiple antihypertensive drugs like ACE inhibitors/ ARBs if required is resistant hypertension.

			mg/mmol	mg/g
1	Normal or high	≥90		
2	Mildly decreased	60–90		
3a	Mildly to moderately decreased	45–59		
3b	Moderately to severely decreased	30–44		
4	Severely decreased	15–29		
5	Kidney failure	<15		

Usual patient profile indicates an often older, though sometimes younger, patients with a long history of uncontrolled hypertension. Usually asymptomatic in early stages and 5-10 years after beginning signs like peripheral oedema, fatigue, and lethargy [2,4].

Differences between eGFR and mGFR

	Estimated GFR (eGFR)	Measured GFR (mGFR)
How it works	A calculation used to estimate how well your kidneys are filtering wastes produced by your body, such as: • creatinine (a waste product that comes from the normal wear and tear on muscles) • cystatin C (a protein that slows down the breakdown of other protein cells)	A measurement of how well your kidneys are filtering certain compounds not produced by your body, such as: • Inulin (a kind of fiber that is found in some plant foods) • Iohexol (contrast agent used in imaging tests)
Availability	Widely available	Not widely available
Cost	Less expensive	More expensive
Time to complete the test	Less time needed	More time consuming
Accuracy	**Possible inaccurate estimates** of GFR, especially in early stages of kidney disease (stages 1 and 2)*	**Accurate measures** of GFR, including early stages of kidney disease (stages 1 and 2)
Precision	Can **miss early GFR changes**, such as a rapid decrease in levels, which may be a sign of diabetic kidney disease	Can **identify early GFR changes**, such as a rapid decrease in levels, which may be a sign of diabetic kidney disease

Renal investigations show modest Proteinuria reduced eGFR (indicative of nephropathy), and nephrosclerosis. eGFR (Estimated Glomerular Filtration Rate) measures how well kidneys filter blood, with a normal rate above 90 mL/min/1.73. A persistent eGFR below 60 for over 3 months, or signs of kidney damage, indicates chronic kidney disease (CKD), which is staged from 1 (mild) to 5 (kidney failure, <15) based on the level of function. Pathology shows accelerated aging of renal vasculature, thickening & sclerosis of resistance vessels [6].

Management Strategies: i) The key approach is to maintain systolic BP <130 mmHg using multiple ACE inhibitors (ACEI) or angiotensin receptor blockers (ARB) to treat hypertension and reduce proteinuria. ii)Lifestyle modifications include sodium restriction, weight management, and smoking cessation are critical [6].

Magnitude of HT Burden: The World Health Organization (WHO) estimates that worldwide there are 1.28 billion adults aged 30-79 years with hypertension. In the India it is the second leading cause of CKD and kidney failure and a significant contributor to cardiovascular mortality and morbidity. Globally, hypertension is the leading preventable & modifiable risk factor for cardiovascular disease, stroke, disability, and premature mortality. Even though hypertension is a common and prevalent disease. The WHO estimates that increasing the percentage of controlled hypertension to 50% would prevent 76 million deaths between 2023 and 2050. As of now only 21% of individuals diagnosed with hypertension are estimated to have it under control. The reasons for this are i) limited resources, ii) physician's failure to initiate or intensify therapy when they encounter BP levels above goal, and adherence by patients.

Box 1. Definition of RH

Components of Definition

- BP that remains elevated above the patient's individualized target despite the concurrent use of 3 antihypertensive agents of different classes, ideally including a diuretic, administered at maximum or maximally tolerated doses and at the appropriate dosing frequency.
- BP that is controlled to the patient's individualized target with ≥4 antihypertensive medications, ideally including a diuretic, administered at maximum or maximally tolerated doses and at the appropriate dosing frequency (ie, controlled RH).

Differentiating Pseudoresistant Hypertension From True RH

- The BP threshold for diagnosis and treatment goals should be in accord with current clinical practice guidelines.
- Ensure proper technique in BP measurement.
- Exclude "white-coat hypertension" by performing out-of-office BP measurements.
- Exclude antihypertensive medication nonadherence.

Abbreviations: BP, blood pressure; RH, resistant hypertension.
Definition based on Carey et al.[5]

Resistant hypertension: Resistant hypertension is defined as uncontrolled hypertension despite 3 or 4 antihypertensive drugs of different classes, or controlled BP with 4 or more antihypertensive drugs; both definitions include a thiazide diuretic and all medications at maximally tolerated doses. Resistant hypertension prevalence ranges from 2%-40%, according to various studies.

Initial Core Therapy: Step 1- The foundation of RHTN therapy consists of combining an ACEI or ARB with a CCB and a potent diuretic. Step 2 - The diuretic should ideally be a long-acting thiazide-like diuretic such as chlorthalidone or indapamide rather than hydrochlorothiazide for better 24-hour control. Step 3 - The most effective fourth-line agent for resistant hypertension is a mineralocorticoid receptor antagonist (MRA) such as spironolactone (12.5–50 mg/day), to be used only when potassium levels are <4.5 mmol/L. Step 4 - Further Additions: If BP remains uncontrolled, options include beta-blockers (e.g., carvedilol or bisoprolol) or combined alpha-beta blockers. Step 5 - If still uncontrolled, direct vasodilators like hydralazine or minoxidil may be added.

New Therapies: ***a) Aprocitentan*** was approved in March 2024 as an endothelin receptor antagonist for resistant hypertension. **Fixed-Dose Combinations:** Using a single-pill combinations (e.g., ACEI+CCB+Diuretic) is highly recommended to improve adherence and reduce treatment failure since a year.

Critical Considerations: Before intensifying therapy, it is crucial to exclude i) white coat effect (using home/ambulatory monitoring) and poor patient adherence ii) Strict reduction of dietary sodium (<2000 mg/day), weight loss, and exercise are essential, as RHTN is often linked to volume expansion. In our case Drug-Induced hypertension was due to use of NSAIDs. Other drugs that can lead RHT are sympathomimetics, decongestants, and oral contraceptives. Sometimes Screening for underlying conditions like primary aldosteronism or obstructive sleep apnoea may be needed. It is crucial not to prematurely conclude that hypertension is the sole cause of CKD, as other glomerular nephritides can co-exist [6,7].

Diabetic Kidney Disease (DKD) with Minimal Change Disease (MCD) represents a rare, complex overlap where a patient with diabetes develops abrupt nephrotic syndrome (heavy protein leakage) due to superimposed MCD rather than solely from diabetic damage. Patients often present with sudden-onset proteinuria or full-blown nephrotic syndrome, which is atypical for simple, early-stage diabetic nephropathy as was in our second case. While DKD usually progresses gradually with retinopathy, this combination presents with sudden oedema. A renal biopsy is critical for diagnosis. Pathologically, shows classic features of DKD (mesangial expansion) plus widespread, diffuse foot process effacement characteristic of MCD. Clinical Presentation. Such cases respond to steroid (glucocorticoid) therapy, leading

to potential complete remission of the nephrotic syndrome. Therefore, it is often considered a "treatable" non-diabetic kidney disease (NDKD) occurring alongside diabetes, requiring immunosuppressive therapy [5].

Uddanam nephropathy" /chronic kidney disease of unknown aetiology (CKDu): The phrase "CKD of unknown etiology" (CKDu) has been used to refer to CKD that is not caused by a typical risk factor such as diabetes, high blood pressure, or HIV. Uddanam region of Srikakulam district, Andhra Pradesh, recently reported non-diabetic/non-hypertensive, public health crisis affecting thousands in rural, agricultural communities in over 500 villages. It was characterized by chronic tubulointerstitial nephritis, caused by prolonged heat stress, dehydration, agrochemicals, and or contaminated drinking water. The prevalence was as a high as 24% in some villages. It primarily affected agricultural labourers, farmers, and rural women. Unlike traditional CKD, this form presented without symptoms, diabetes, or hypertension in many cases and was often diagnosed in advanced stages [8]. While the exact cause remains unknown, investigations suggest a combination of Environmental & Occupational factors. Key suspected causes include i) Chronic dehydration from working in hot/humid climates (heat stress) ii) Exposure to pesticides, iii) heavy metals like cadmium/arsenic, or high silica/strontium in ground water iv) Consumption of contaminated water or high intake of sugary drinks.

Accredited Social Health Activists (ASHAs) and ANMs were trained to identify patients. Free diagnostic facilities and dialysis were provided, along with specialized research to identify root causes. A 2026 pilot study showed a high CKD prevalence with 61.8% of rural participants screened having some form of CKD, and only 16.5% being aware of their condition prior to screening. Ongoing studies are focusing on comprehensive testing of food, water, and socioeconomic factors to determine the exact cause of this condition [8].

Management & Treatment of CKDu: Lifestyle medication, less use of chemicals/pesticides, and proper hydration were encouraged among the population through awareness programs at the local level through ASHAs and ANMs. State Government and GOI rose to the occasion by providing for renal transplantation, for diseased people [9].

CONCLUSION

By 2040, CKD is projected to become a top five cause of death in India, with mortality potentially exceeding half a million annually. Strengthening diabetes and hypertension control, integrating an early CKD detection program, and ensuring equitable access to renal replacement therapy are urgently needed to bend the trajectory.

CKD is among the fastest-rising causes of mortality in India. By 2021, it caused over 175,000 deaths, and projections indicate that by 2040, annual deaths could exceed half a million. The surge is heavily driven by metabolic factors, with Type 2 Diabetes being the leading cause (34%), followed by hypertension (19%). Older age, presence of diabetes, lower baseline eGFR, and hyperuricemia increase the risk of developing new-onset CKD in hypertensive patients. The prevalence of CKD is higher in rural areas (15%) vs urban areas (11%). Roughly 220,000 new patients develop end-stage kidney disease (ESKD) annually, creating a massive demand for dialysis. As of early 2026 the demand for treatment far outstrips capacity, with only about 5,000 dialysis centres & 3,340 nephrologists in the country.

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