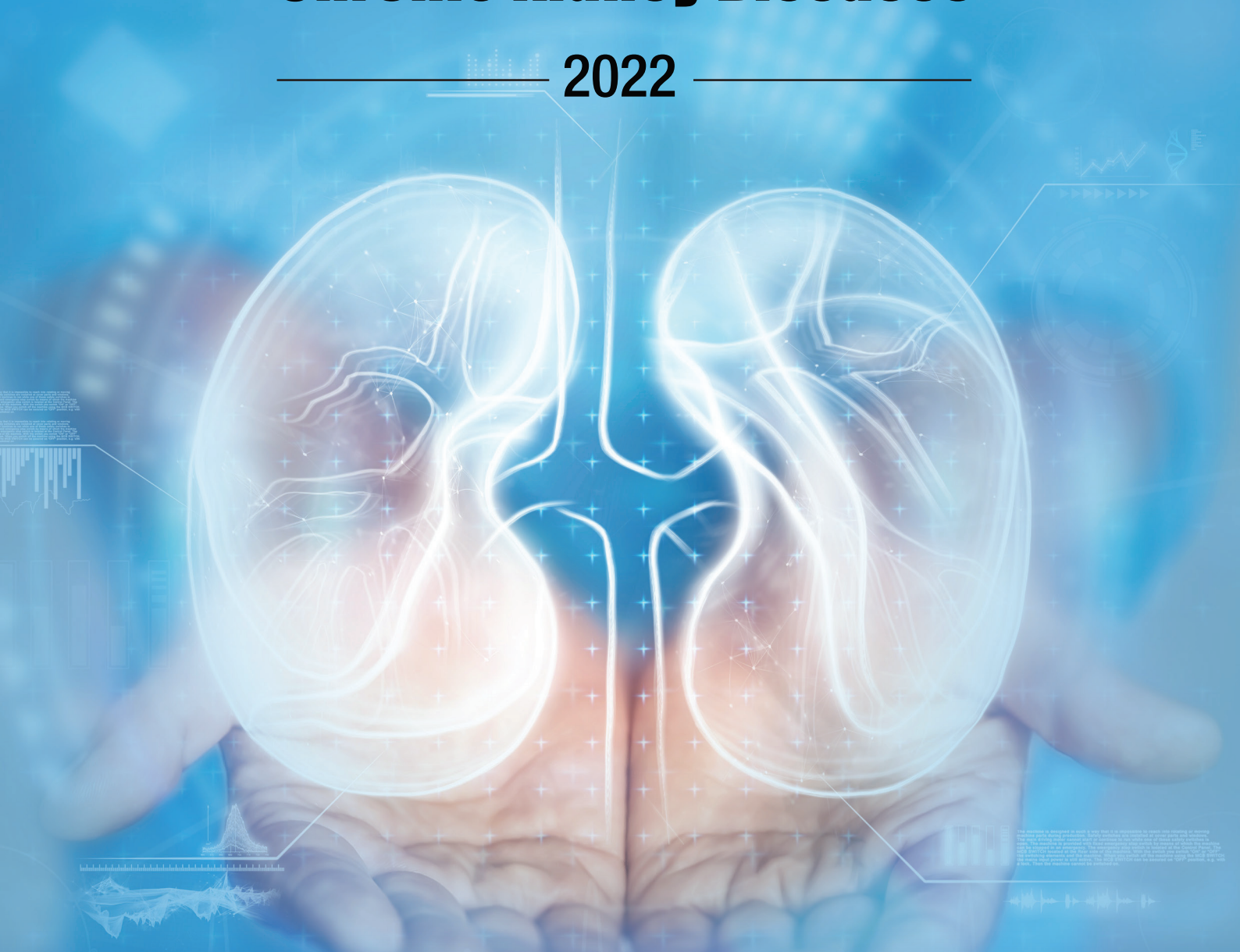
Ministry of Health & Family Welfare
Government of India

National Programme for Prevention and Control of Non-Communicable Diseases

Medical Officers' Manual for Prevention and Management of Chronic Kidney Diseases

2022



Directorate General of Health Services
Ministry of Health & Family Welfare
Government of India

National Programme for Prevention and Control of Non-Communicable Diseases

Medical Officers' Manual for Prevention and Management of Chronic Kidney Diseases

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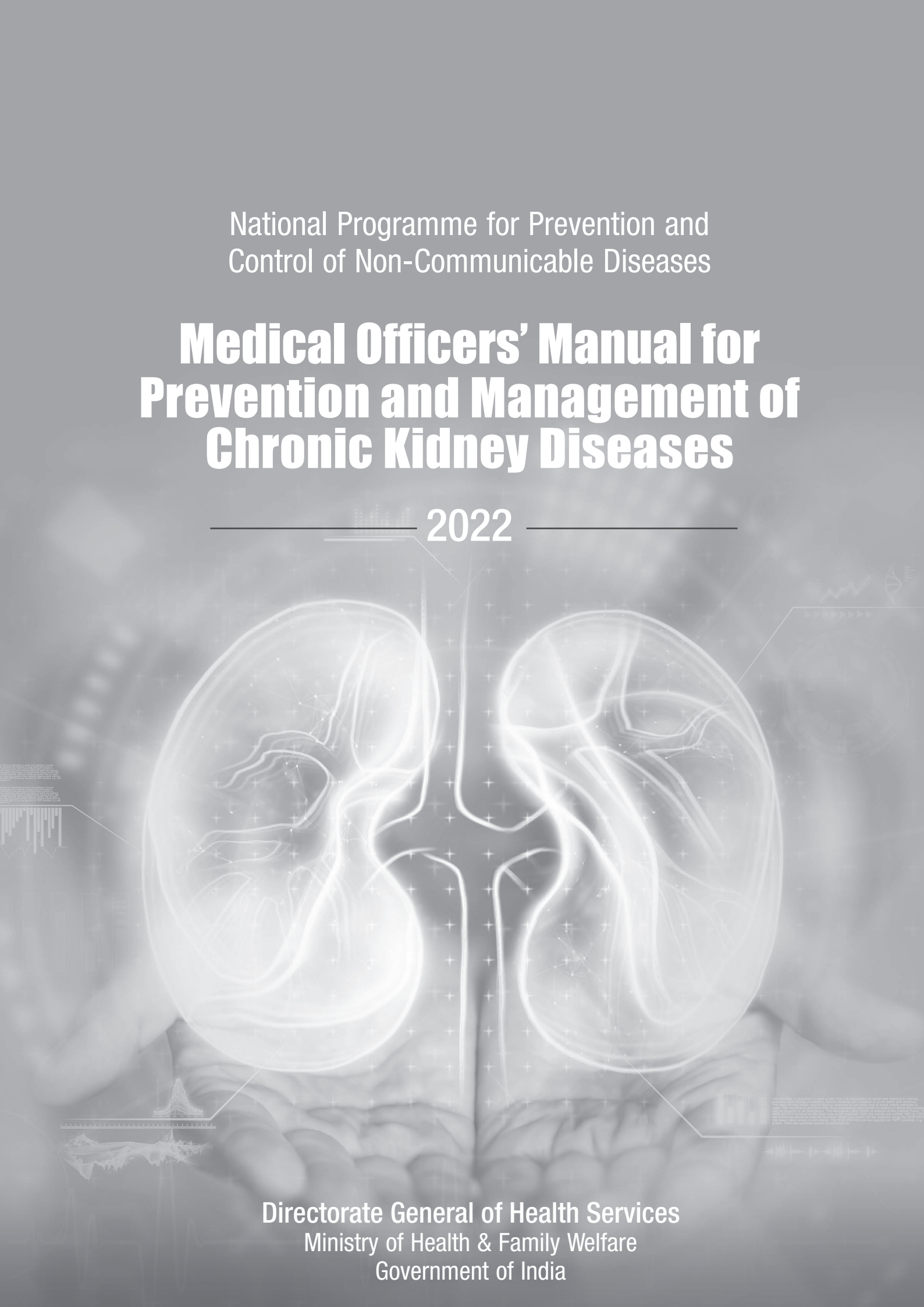

Directorate General of Health Services
Ministry of Health & Family Welfare
Government of India

Content

List of Abbreviation

ACEIs	ACE inhibitors
uACR	Urinary Albumin Creatinine Ratio
AKI	Acute Kidney Injury
ANM	Auxiliary Nurse Midwife
ARBs	Angiotensin-II Receptor Blockers
ASHA	Accredited Social Health Activist
AYUSH	Ayurveda, Yoga, Unani, Siddha and Homoeopathy
BMI	Body Mass Index
CBAC	Community Based Assessment Checklist
CHC	Community Health Center
CHO	Community Health Officer
CKD	Chronic Kidney Diseases
CPHC	Comprehensive Primary Health Care
CT	Computed Tomography
DALYs	Disability Adjusted Life Years
DH	District Hospital
Dte.GHS	Directorate General Health Services
DHS	District Health Society
DM	Diabetic Mellitus
eGFR	estimated GFR
ESA	Erythropoietin Stimulating Agents
ESRD	End Stage Renal Disease
ESKD	End Stage Kidney Disease
GFR	Glomerular Filtration Rate
H/o	History of
HBsAg	Hepatitis B virus surface antigen
HCV	Hepatitis C virus
HT	Hypertension
HWC	Health and Wellness Centers
IEC	Information Education Communication
IV	Intravenous

KFT	Kidney Function Test
MBD	Metabolic Bone Disorder
MO	Medical Officer
MPW (F/M)	Multipurpose Worker (Female/Male)
NCD	Non Communicable Diseases
NHM	National Health Mission
NHSRC	National Health System Resource Center
NPCDCS	National Programme for Prevention and Control of Cancer, Diabetes, Cardiovascular Diseases and Stroke
NPCBVI	National Programme for Control of Blindness and Visual Impairment
NPHCE	National Programme for Health Care of Elderly
NSAID	Non Steroidal Anti-Inflammatory Drugs
NTCP	National Tobacco Control Programme
uPCR	Urine Protein Creatinine Ratio
PHC	Primary Health Care
PIP	Program Implementation Plan
PMNDP	Pradhaan Mantri National Dialysis Programme
PMR	Physical Medicine & Rehabilitation
PPI	Proton Pump Inhibitors
PTH	Parathyroid Hormone
RKS	Rogi Kalyan Samiti
RRT	Renal Replacement Therapy
KRT	Kidney Replacement therapy
RT	Renal Transplant
SC	Sub-center
SHS	State Health Society
USG	Ultra-Sonography
UTI	Urinary Tract Infection
VHND	Village Health Nutrition Day
VHSNC	Village Health Sanitation & Nutrition Committee
VUR	Vesicoureteral Reflux
WHO	World Health Organization

डॉ. मनसुख मांडविया
DR. MANSUKH MANDAVIYA

सत्यमेव जयते
आज़ादी का अमृत महोत्सव

स्वास्थ्य एवं परिवार कल्याण
व रसायन एवं उर्वरक मंत्री
भारत सरकार
Minister for Health & Family Welfare
and Chemicals & Fertilizers
Government of India

MESSAGE

It is a well-known fact that many people suffer from Non-Communicable Diseases (NCDs) including Chronic Kidney Disease (CKD) for years and die prematurely due to NCDs' complications. This has long-term implications of increasing burden of sufferings on the patient, family, community, eventually on whole health care system.

As per available studies, prevalence of Chronic Kidney Disease ranges from 6-7% among adult population in India and CKD is the 9th frequent cause of cause of death in India.

I am confident that these Guidelines on Chronic Kidney Disease will focus on early detection, management, referral and Continuum of Care of CKD patients and also go a long way to enrich the knowledge of physicians across the county on standard treatment.

(Dr. Mansukh Mandaviya)

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Dr. Bharati Pravin Pawar

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MESSAGE

Non-Communicable Diseases (NCDs) like Chronic Kidney Disease (CKD) are rapidly increasing in most of the States/UTs due to the epidemiological transition that India is facing. Though all existing cases need to be detected early and managed properly, it is equally essential that we simultaneously work on preventing these diseases. Such an approach of making strong efforts towards prevention and appropriate management will go a long way in reducing the burden of CKD in India.

The current Guidelines deal on CKD incorporate the updated technical knowledge on CKD management and referral system, while also guiding on the resources needed at various levels of health facilities.

The Government of India, under the visionary leadership of Hon'ble Prime Minister Shri Narendra Modi ji, is committed to secure the health of the people of India and I strongly believe that the Programme Managers at State/District NCD Cells will find these guidelines useful and will successfully implement the requisite interventions for management of CKD in the country.

(Dr. Bharati Pravin Pawar)

"दो गज की दूरी, मास्क है जरूरी"

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16th November, 2022

MESSAGE

Kidney Diseases leads to approximately 150,000 to 200,000 new cases of chronic renal failure (CRF) in India. As a result, nearly 3.4 crore dialysis sessions are needed annually. In India, chronic kidney disease (CKD) ranks as the ninth leading cause of death.

The Ayushman Bharat - Health and Wellness Centers (AB-HWCs) are being established for the service delivery of commonly prevalent diseases in the community in order to expand comprehensive primary health care and improve community access to healthcare. More than 118,000 HWCs offer preventative, promotive, rehabilitative, and curative care for a various medical conditions including non-communicable diseases like hypertension, diabetes, cancer, COPD/asthma, CKD, etc.

Early detection, management, appropriate referral, and continuum of care are crucial given the growing prevalence of CKD. The National Health Mission's (NHM) Comprehensive Primary Health Care and Pradhan Mantri National Dialysis Program should be strengthened to provide better care for CKD patients.

These guidelines on CKD are intended to update and build capacities in health care providers across the nation regarding standard treatment. It will also aid medical officers in better understanding the condition and enhancing their capacity for decision-making for early detection and management of CKD at a primary healthcare level.

I am confident that these guidelines would help to manage CKD patients more effectively and decrease the burden of the disease.

(Vinod Paul)

स्वच्छ भारत
एक कदम स्वच्छता की ओर

I/34097847/2022

सत्यमेव जयते

भारत सरकार
स्वास्थ्य एवं परिवार कल्याण विभाग
स्वास्थ्य एवं परिवार कल्याण मंत्रालय
Government of India
Department of Health and Family Welfare
Ministry of Health and Family Welfare

राजेश भूषण, आईएएस
सचिव
RAJESH BHUSHAN, IAS
SECRETARY

MESSAGE

Chronic Kidney Disease (CKD), is a major non-communicable disease, and is also one of the leading causes of deaths in India. As per Global Burden of Disease (GBD) 2017 report, India contributes to nearly 115.1 million cases to the global case burden of CKD.

There are various primary causes of chronic kidney disease, with hypertension and diabetes being the most common. The link between kidney disease and other major non-communicable diseases highlights the importance of preventive care and public health policy measures in limiting the progression of chronic kidney disease.

The National Programme for Prevention and Control of Cancer, Diabetes, Cardiovascular Diseases and Stroke (NPCDCS) was launched in the year 2010, and a component of this programme for Chronic Kidney Disease (CKD) was added to it later on. With these Guidelines on CKD, we wish to further strengthen the core components of health promotion, early diagnosis, and appropriate treatment for Chronic Kidney Disease (CKD).

I congratulate all those involved in the development of these Guidelines and hope that they will prove to be useful for all.

Place : New Delhi
Date : 10-06-2022

(Rajesh Bhushan)

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DIRECTOR GENERAL OF HEALTH SERVICES

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Government of India
Ministry of Health & Family Welfare
Directorate General of Health Services

PREFACE

India adds almost 150,000 to 200,000 of newly diagnosed patients of chronic kidney disease resulting in chronic renal failure (CRF) every year. This results in an annual requirement of nearly 3.4 crore dialysis. Chronic Kidney Disease (CKD) happens to be the ninth most frequent cause of death in India.

Considering the rising burden of CKD, early detection, management, appropriate referral and continuum of care is of vital importance. The care for CKD patients under the National Health Mission (NHM) needs to be strengthened under Ayushman Bharat scheme including Comprehensive Primary Health Care, and Pradhan Mantri National Dialysis Programme (PMNDP). These guidelines on CKD, will help medical officers associated with the above-mentioned programs in understanding the disease and improve their decision-making ability for early diagnosis and management of CKD at a primary health care level.

These guidelines would also enable the states to put in place the required elements, such as human resource, drugs, equipment and diagnostics required at various health care levels for delivering the continuum of care required for chronic kidney disease.

(Atul Goel)

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FOREWORD

Chronic Kidney Disease (CKD) is a substantial cause of morbidity and mortality in India. It is an established fact that patients' access to diagnostic and management facilities, health awareness, drugs and treatment, treatment compliance and non-pharmacological interventions are cornerstones for an effective response.

In order to expand comprehensive primary health care to improve community access to healthcare, the Ayushman Bharat - Health and Wellness Centres (AB-HWCs) are being established for the service delivery of commonly prevalent diseases in the community. More than 118,000 HWCs are providing preventive, promotive, rehabilitative, and curative care for various health ailments including non-communicable diseases like Hypertension, Diabetes, Cancers, COPD/Asthma, CKD etc. Further, those needing tertiary care are being supported financially under the Ayushman Bharat – Pradhan Mantri Jan Arogya Yojana (AB PM-JAY).

Under the aegis of Pradhaan Mantri National Dialysis Programme (PMNDP), these guidelines have been developed for capacity building on awareness generation, early detection, appropriate management, and referral of patients suffering from CKD.

I believe that an earnest implementation of these guidelines would go a long way in reducing the burden of CKD, and better management of CKD patients.

Roli Singh

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MESSAGE

Non-communicable diseases, such as cardiovascular diseases, diabetes, cancer, Chronic Respiratory Diseases etc. account for over 50% of total burden of diseases. Chronic Kidney Disease (CKD) is one of the leading non-communicable diseases in India.

Comprehensive Primary Health Care reduces mortality and morbidity at much lower costs. It also significantly reduces the need for secondary and tertiary care, and the same holds true for CKD. Ayushman Bharat - Health and Wellness Centres (AB-HWCs) programme is being strengthened in the country to ensure Comprehensive Primary Health Care to the community.

CKD has been made an integral part of the National Programme for Prevention and Control of Cancer, Diabetes, Cardiovascular Diseases and Stroke (NPCDCS) under the aegis of National Health Mission. Pradhan Mantri National Dialysis Programme (PMNDP) was launched during year 2016-17 for chronic kidney disease to support in all district hospitals in a Public Private Partnership (PPP) mode under NHM to provide Haemodialysis and Peritoneal Dialysis services.

With these technical guidelines, we wish to further prioritize and strengthen critical components of health promotion, early diagnosis, management and appropriate treatment for CKD.

(Vishal Chauhan)

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Deputy Director General
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Chronic Kidney Disease (CKD) is one of the leading non-communicable diseases in India. According to ICMR report on "India: Health of the Nation's States" (ICMR, PHFI and IHME; 2017), CKD is 9th frequent cause of death in India and rose to 20th cause of DALY in 2016 from 30th in 1990, indicating the rising burden of CKD. The number of cases of kidney failure is expected to increase disproportionately in developing countries, such as India, where the number of elderly people and the NCD risk factors are increasing.

Major causes on CKD in India are uncontrolled diabetes, blood pressure and ageing of the population. CKD is associated with increased risk of complications and mortality at all stages in its natural course. End Stage Kidney Failure (ESKF) is the most advanced stage of kidney failure when survival without some form of renal replacement therapy, such as kidney transplant or maintenance dialysis is not possible.

Considering the rising burden of the CKD, its prevention and control guidelines are being included under the National Programme for Prevention and Control of Cancer, Diabetes, Cardiovascular Diseases and Stroke (NPCDCS) along with other non-communicable diseases.

To resolve the issues of ESRD patients like financial constraints, low service accessibility, Government of India has implemented Pradhan Mantri National Dialysis program under National Health Mission with an objective to ensure access to dialysis services in district hospitals. The States/UTs are being supported for provision of dialysis services to the poor.

Therefore, the current Guidelines have been prepared with an objective to provide the updated technical knowledge on CKD management and referral system.

I am sure that these Guidelines on Chronic Kidney Disease will update and build capacities in health care providers across the country on standard treatment, while also focusing on early detection, management, appropriate referral and continuum of care of CKD patients.

(Dr. Sudarsan Mandal)

1
Introduction

Rising burden of the behavioral and metabolic risk factors

Magnitude of CKD: Global and Indian Perspective

Integration with focus on continuum of care for CKD patients' facilities available/desired at different level

About the guidelines: What and for whom?

Non-Communicable diseases (NCDs) are increasing to such an extent that in one year almost 63% (6.028 million deaths) of all estimated deaths in India are due to these diseases. According to the NCD country profile report 2018, World Health Organization (WHO),[1] the major NCDs responsible for these deaths in India include cardiovascular diseases (2.58 million deaths, 27% of all estimated deaths), cancers (0.86 million deaths, 9% of all estimated deaths); chronic respiratory diseases (1.05 million deaths, 11% of all estimated deaths); and diabetes (0.28 million deaths, 3% of all estimated deaths). The risk of dying prematurely due to NCDs is between 30-70 years is 23 % in India.

Rising burden of the behavioral and metabolic risk factors

Driving force behind the rising level of NCDs are rising burden of four common behavioral risk factors (i.e., tobacco and alcohol consumption, physical inactivity, and unhealthy diet) which in turn increases the burden of the metabolic risk factors, i.e., raised blood sugar and blood pressure, obesity and high cholesterol.

Due to rising burden of both behavioral and metabolic risk factors, particularly high blood pressure (24%) and high blood sugar (8%), other NCDs which were not so much common earlier, also started increasing rapidly in the recent times, e.g., Chronic Kidney Diseases (CKD).

Magnitude of CKD: Global and Indian perspective

CKD constitutes to a major part of the NCDs burden and is a public health problem globally (1)amounting to >800 million individuals. Chronic kidney disease is more prevalent in older individuals, women, racial minorities, and in people experiencing diabetes mellitus and hypertension. Chronic kidney disease represents an especially large burden in low- and middle-income countries, which are least equipped to deal with its consequences. Chronic kidney disease has emerged as one of the leading causes of mortality worldwide, and it is one of a small number of non-communicable diseases that have shown an increase in associated deaths over the past 2 decades. The high number of affected individuals and the significant adverse impact of chronic kidney disease should prompt enhanced efforts for better prevention and treatment.","container-title":"Kidney International Supplements","DOI":"10.1016/j.kisu.2021.11.003","ISSN":"2157-1724","issue":"1","journalAbbreviation":"Kidney Int Suppl (2011. Though there has been a considerable improvement in health indicators like life expectancy, maternal and infant mortality rates in India, the burden of NCDs has been increasing exponentially over the years and they contribute to over 40% of all-cause mortality. The twin epidemic of hypertension and diabetes mellitus has largely driven the steady increase in CKD prevalence.

1 World Health Organization. (2018). Non-communicable diseases country profiles 2018. World Health Organization. http://apps.who.int/iris/handle/10665/274512 .

Global Magnitude of CKD

CKD has emerged as a major public health challenge worldwide. With a global prevalence of 13.5%., it is estimated that approximately 500 million people suffer from CKD, of which 80% live in low middle income countries (3).

According to the Global Burden of Diseases (GBD), CKD was one of the top ten most important drivers of increasing disability-adjusted life years (DALYs) burden with a 93.2% increase from 1990 to 2019. It was also estimated to be the eighteenth most common cause of DALYs in 2019. A modelling study based on the GBD data predicted that by 2040, the impact of CKD on global health would double and it would be the fifth leading cause of years of life lost (YLLs). (6) The increase in burden of CKD was also shown to be disproportionate in the GBD study with the African and Asian countries being more predominantly affected. Another point of concern from the GBD analysis is that the age-standardized mortality rate for CKD is increasing unlike that for other NCDs.

The International Society of Nephrology's Kidney Data Center (ISN-KDDC) cross-sectional study to assess CKD and cardiovascular risk in 12 countries (including India), reported a CKD prevalence of 14.3% in general population while it was 36.1% in high-risk populations. (7)and to investigate the risk of cardiovascular disease, in countries of low and middle income.\nMETHODS: We did a cross-sectional study in 12 countries from six world regions: Bangladesh, Bolivia, Bosnia and Herzegovina, China, Egypt, Georgia, India, Iran, Moldova, Mongolia, Nepal, and Nigeria. We analysed data from screening programmes in these countries, matching eight general and four high-risk population cohorts collected in the ISN-KDDC database. High-risk cohorts were individuals at risk of or with a diagnosis of either chronic kidney disease, hypertension, diabetes, or cardiovascular disease. Participants completed a self-report questionnaire, had their blood pressure measured, and blood and urine samples taken. We defined chronic kidney disease according to modified KDIGO (Kidney Disease: Improving Global Outcomes The CKD prevalence in India was 17% in this study. Moreover, this study also revealed that awareness of CKD was low in low-middle income countries and stressed the need for prospective screening programs.

Indian Magnitude of CKD

According to ICMR report on "India: Health of the Nation's States" (2017),[2]CKD is 9th frequent cause of death in India; it rose to 20th cause of DALYs in 2016 from 30th in 1990, indicating the rising burden of CKD. Major cause of this rise in the disease are uncontrolled diabetes, high blood pressure and ageing of the population.

Global Burden of Disease, has shown that crude prevalence of CKD has increased from 5.9% in 1990 to 8.3% in 2019, while during the same period, age standardized prevalence has increased from 8.6% to 9.1%.

True assessment of the burden of CKD is restricted by the lack of national registries in India and in part, due to use of different methodologies to diagnose CKD and heterogeneity in the definition of CKD. Estimation of CKD prevalence depends on measurements of glomerular filtration rate (GFR) and albuminuria, repeated over a period of 3 months for a definitive diagnosis of CKD, which is often missing in most studies.

Integration with focus on continuum of care for CKD patients

For optimization of the scarce resources and for ensuring the sustainability of the flawless services, the NPCDCS has been integrated in National Health Mission (NHM) framework. Sharing the NHM financial, administrative, and institutionalized structure, e.g., NCD Flexi pool, SHS, DHS, rationalization of human resources etc. all have become crucial program strategies for NPCDCS implementation.

For early detection, management, appropriate referral and continuum of care, the care for CKD patients under the NHM need to be streamlined with Ayushman Bharat including Comprehensive Primary Health Care, and Pradhan Mantri National Dialysis Programme (PMNDP).

2 Indian Council of Medical Research, Public Health Foundation of India, and Institute for Health Metrics and Evaluation. India: Health of Nation's States- The India State-level Disease Burden Initiative. New Delhi, India: ICMR, PHFI and IHME;2017 (source: https://bmjopen.bmj.com/content/12/2/e052525)

In order to ensure delivery of Comprehensive Primary Health Care (CPHC) services, existing Sub Health Centres covering a population of 3000-5000 in rural area and Urban Primary Health Centers or Urban Health Posts catering to about 50,000 populations in urban areas would be converted to Health and Wellness Centers (HWC) with the principle of "time to care" to be no more than 30 minutes. Primary Health Centers supported by outreach services, Mobile Medical Units, health camps; home visits and community-based interaction will provide the seamless continuum of care that ensures the principles of equity, quality, universality and no financial hardship[3].

To take care of rising burden of End Stage Kidney Diseases (ESKD) of which 2.2 lakh cases are being added every year in India, the PMNDP was announced in the Union Budget 2016-17. Under the programme there is a provision of the Renal Replacement Therapy (including hemodialysis and recently added peritoneal dialysis services) for CKD patient at district level under Public Private Partnership mode, inhouse mode and mixed mode.

As the treatment and rehabilitation for CKD patients are costly option, hence focus is also on to create awareness in the community for effective prevention and early detection, referrals and treatment strategies available through convergence with the ongoing interventions of National Health Mission (NHM), National Tobacco Control Programme (NTCP), National Programme of Health Care for Elderly (NPHCE) and Pradhan Mantri Dialysis Programme (PMNDP).

Facilities available/ desired at different levels

At SUB-CENTER / HEALTH and WELLNESS CENTRE (POPULATION= 5000)

Staff available	Indicative list of Equipment	Indicative list of Drugs	Lab investigations
• 1 Community Health Officer • 1 ANM / Health Worker (F) • 1 Health Worker (M) • 1 Part-time Safai Karamchari (Outsourced)	• Sahli's Hemoglobinometer • Weighing Scale • Height scale • BP Apparatus • Stethoscope • Measuring Tape • Glucometer (test marked as essential as per IPHS 2022)	• (Only dispensing / depot) • Tab Iron • Tab & Syrup Folic Acid • Other medicines can be indented as per need for various NCD National Program	• Hb by Sahli's method • Urine albumin & sugar by dipstick • Can use Point of care • Hemoglobinometer Blood Sugar (equipment marked as essential as per IPHS 2022)

3 Operational Guidelines: Ayushman Bharat- Comprehensive Primary Health Care through Health and Wellness Centres, Ministry of Health and Family Welfare, Government of India.

AT PHC / HEALTH and WELLNESS CENTRE (POPULATION= 30000)

Staff available	Indicative list of Equipment	Indicative list of Drugs	Lab investigations
1. 1 Medical Officer (MBBS) **2.** 1 Accountant cum DEO **3.** 1 Pharmacist **4.** 3 Staff Nurses **5.** 1 Health Assistant (Male) **6.** 1 Health Assistant (Fem) **7.** 1 Lab Technician **8.** 2 Multi-skilled Gr D staff **9.** 1 Health Educator **(Desirable)** 1. Logistic Assistant	**In addition to equipment listed in SC-HWC** **1.** Binocular Microscope **Desirable** **1.** ECG Machine- not recommended at PHC level in IPHS 2022 **2.** Nebulizer **3.** Blood cell counter* **4.** Glucometer **5.** Centrifuge **6.** Auto analyzer* for biochemistry and KFT **7.** Refrigerator * Equipment like blood cell counter and semi-automated analyzer are recommended at hub labs (CHC/SDH/DH) as per FDSI guidelines and IPHS 2022.	**In addition to above Anti hypertensive's** • ACEI/ARB • Beta Blocker • Calcium channel blocker • Alpha-blocker • Central Acting (Clonidine) • Diuretic • Methyldopa **Anti-diabetics** • Biguanides • Sulphonylureas • Insulin **Antibiotics & Others** • Beta lactams • Cephalexin, • Amikacin • Nalidixic Acid • Ciprofloxacilin • Nitrofurantoin • Tetracycline • Metronidazole/ Tinidazole others • Antiemetics • Calcium tablets, • Salbutamol for nebulizer **Desirable** • Sevelamer • Alpha D3 • Vitamin D, • Sodium bicarbonate tablets • K-Bind sachets • Cold Chain & Vaccines	**In addition to above** • Urine routine with microscopy • Complete Hemogram (Hb, platelet, RBC, WBC) • Gram staining • Blood sugar • Serum Creatinine • Serum Electrolyte • Blood Group **Desirable** • Blood Cholesterol • ECG Sample collection for tests like complete hemogram, Creatinine, blood cholesterol etc is recommended at PHC level, while the diagnostic tests are recommended at hub labs (CHC/SDH/DH) as per FDSI guidelines and IPHS 2022. Serum Electrolyte tests is placed in desirable category as per IPHS 2022.

AT COMMUNITY HEALTH CENTER (POPULATION ≈ 1.5 LAC)

Staff available	Indicative list of Equipment	Indicative list of Drugs	Lab investigations available
Block Public Health Unit **1.** 1 Block Medical Officer **2.** 1 Med. Superintendent **3.** 1 Public Health Specialist **4.** 1 Public Health Nurse **Specialty Services** • 1 General Surgeon • 1 Physician • 1 Obs. & Gynecologist • 1 Pediatrician • 1Orthopedic • 1 Anesthetist • 4 General Duty Officers • 12 Paramedics • Lab. Technicians, • Radiographer, • Rehabilitation Worker • Counselor • Dietician (Desirable)	**In addition to equipment listed in PHC** • Standard Surgical Sets • Laparoscope • Blood Transfusion Kit • OT Equipment • Radiology Equipment • Immunization **IPHS 2022 may be referred for categorization of equipment under essential and desirable category**	**In addition to above** • Wider range of oral and injectable antibiotics including • Doxycycline • Sulfonamides • Cefotaxime • Tinidazole • Chloramphenicol • Ciprofloxacin, etc. • Inj. Sodium bicarbonate • Inj. Calcium Gluconate • Inj. Potassium Chloride • Drugs under various National Programs	**In addition to Above** • Lipid profile • Iron studies • LFT and complete Biochemistry • X ray, • ECG • Ultrasonography **97 diagnostic tests are recommended at the CHC level in hub and spoke model in FDSI guidelines 2019.**

AT DISTRICT LEVEL HOSPITAL (POPULATION= 10 LAC)

Staff available	Indicative list of Equipment	Indicative list of Drugs	Lab investigations available
Specialty Med Manpower • 2 Medicine • 2 Surgery • 2 Obst. and Gynecology • 2 Pediatrics • 2 Anesthesia • 1 Radiology • 1 Pathology • 1 Eye • 1 Orthopedic • 1 ENT • 1 Dental • 1 Psychiatry • 1 AYUSH **Desirable** • Skin • Microbiology • Forensic • 11 Medical Officers • 31 Paramedical • 6 Lab Technicians • 4 Pharmacists (+1 AYUSH) • 2 Radiographers • 1 ECG • Technician • 1 Dietician • 2 Social Workers • 1 Counselor & • 13 others • 12 Administrative Staff **District Public Health Unit** • 1 Epidemiologist • 1 Entomologist • 1 Microbiologist • 1 IEC Officer • 1 District Public Health Nursing Officer • 1 District Data Analyst	**In addition to equipment listed in CHC** • Imaging equipment • X-Ray Room Accessories • Cardiopulmonary Equipment **Laboratory Equipment** • Fully automated auto analyzer • Semi auto analyzer • Centrifuge • Electronic cell counter • ELISA reader &washer • Blood gas analyzer • Electrolyte analyzer • Hematology analyzer • Histopathology equipment • Laminar flow, etc. • Surgical equipment sets • PMR equipment • Endoscopy Equipment • Anesthesia Equipment • ICU and its instruments • **IPHS 2022 may be referred for categorization of equipment under essential and desirable category**	**In addition to above** • Wider range of analgesics • ALL anti-pyretic • Wider anti-inflammatory • Wider antibiotics • Wider • chemotherapeutics • ALL infusion fluids • Drugs for hemopoietic system • Inj. Iron • Drugs for urogenital system • Hormonal preparation • ALL vitamins • Dialysis services (desirable) • Nephrology specialties **(Desirable)** • Urology Specialty **(Desirable)**	**In addition to above** • ELISA for HIV, HCV and HBsAg • Aspirated fluid cell count & cytology • Immuno-hematology • Histopathology - for biopsy • Culture & Sensitivity for blood, urine, pus, etc. • Glucose Tolerance Test • Glycosylated Hemoglobin • Cardiac Stress Test,2D ECHO • CT Scan • Endoscopy **Desirable** • PTH • Vitamin D 111/134 diagnostic tests are recommended at SDH/DH level respectively in Hub and spoke model in FDSI guidelines 2019.

About the guidelines: What and for whom?

Guidelines primarily are to be used by the medical officers for prevention, early detection and management of CKD patients reporting at Primary Health Center/ Health & Wellness Centre (HWC) level. The guidelines also briefly mention the roles and responsibilities of the supporting staff, e.g., ANM and ASHA worker in prevention and early detection of the CKD patients. The guidelines also state the epidemiology, risk factors, classification/ staging of the CKD, drug interactions for easy understanding and improving the decision-making capacity of medical officers at PHC/ HWC level. These guidelines also help primary care physician to decide when to refer CKD patient to higher level for diagnostic and/ or therapeutic decisions and actions.

The use of these guidelines would enable the states to put in place the necessary elements, i.e., human resources, drugs, equipment, and diagnostics required at various health care levels for delivering the continuum of care for CKD patients. However, states have the flexibility to make necessary modifications based on their specific needs and capacities. National screening programs and region-specific policy on CKD will ensure optimal management of patients with kidney disease and once integrated with the population-based health care system, will help in generating regional data on disease characteristics and health barriers.

2
Chronic Kidney Disease

Definition & Classifications

Causes, risk factors and risk multiplier for other NCDs

CKD progression and its risk factors

Presentation of CKD

Diagnosis of CKD and its pitfall

Principle of management of CKD

A. Definition

"Kidney disease" is manifested by evidence of urinary abnormality (Proteinuria and hematuria) and/or radiological abnormality and/or decrease in kidney function as assessed by increase in serum creatinine (corresponding to decrease in Glomerular Filtration Rate, GFR). These are the diseases where there is either evidence of kidney damage and/or evidence of low glomerular filtration rate below < 60 ml/min/1.74^2. Like any other organ kidney diseases are broadly divided into acute kidney injury (AKI) and chronic kidney disease (CKD).

Acute Kidney Injury (AKI) and Acute Kidney Disease (AKD)

AKI, earlier called as Acute Renal Failure (ARF), is defined as an abrupt (within hours) decrease in kidney function, which encompasses both injury (structural damage) and impairment (loss of function). The evidence suggests that even relatively mild injury or impairment of kidney function manifested by small changes in serum creatinine (S. Cr.) and/or decrease in urine output (U.O.), is likely to predict serious clinical consequences and the whole spectrum should be recognized in definition of AKI. Patients of AKI, who does not recover within one month, are then labelled as acute kidney disease (AKD) till the end of 3 months, after which if disease persist, it is called CKD.

By the latest classification of AKI proposed by the AKI Working Group of KDIGO (Kidney Disease: Improving Global Outcomes) AKI is diagnosed by -

A) An absolute increase in S. Cr., at least 0.3 mg/dl within 48 hours or
B) By a 50% increase in S. Cr., from baseline within 7 days, or
C) A urine volume of less than 0.5 ml/kg/h for at least 6 hours

AKI is staged into 3 stages for severity according to the following criteria -

Stage-1	1.5-1.9 times baseline OR >0.3 mg/dL (>26.5 µmol/L) absolute increase in S. Cr.	Urine volume <0.5ml/kg/hr for 6-12 hrs
Stage-2	S. Cr. >2.0-2.9 times baseline	Urine volume <0.5ml/kg/hr for> 12 hrs
Stage-3	Increase in S. Cr. >4.0 mg/dL or Initiation of renal replacement therapy or, In patients < 18 yrs decrease in eGFR by 35ml/min/1.73m^2	Urine volume <0.3ml/kg/hr for> 24 hrs Anuria for >12 hours

S. Cr. - serum creatinine, eGFR - estimated glomerular filtration rate

Chronic kidney disease

CKD is defined as evidence of kidney disease with/without decrease in GFR < 60 ml/min/1.74^2, persistent for more than 3 months in absence of reversible factors. Evidence of kidney disease is primarily defined by increase in urine protein beyond 30 mg/day. The Kidney Disease Outcomes Quality Initiative (KDOQI) of the National Kidney Foundation (NKF) established this definition and classification of CKD in 2002. CKD is classified based on cause, GFR category, and albuminuria category (CGA).

Criteria for CKD (either of the following present for >3 months)

Markers of kidney damage (one or more)	1. Albuminuria, Albumin excretion rate (AER) >30 mg/24 hours; ACR >30 mg/g of creatinine 2. Urine sediment abnormalities Electrolyte and other abnormalities due to tubular disorders Abnormalities detected by histology 3. Structural abnormalities detected by imaging 4. History of kidney transplantation
Decreased GFR	GFR < 60 ml/min/1.73 m^2

GFR categories in CKD - Based on degree of fall in GFR, there are GFR stages of CKD as shown in table below:

Table 1: GFR - glomerular filtration rate.

GFR category	GFR (ml/min/1.73 m^2)	Terms
G1	>90	High or normal
G2	60-89	Mild decrease
G3a	59-45	Mild to moderate decrease
G3b	44-30	Moderate to severe
G4	29-15	Severe decrease
G5	<15	Kidney failure

Albuminuria category in CKD - Based on degree of proteinuria, there are three stages of albuminuria category as shown below:

Table 2: AER- albumin excretion rate, ACR- albumin-to-creatinine ratio

Category	AER (mg/24 hrs)	ACR (mg/gm)	Terms
A1	<30	<30	Normal or mild increase
A2	30-300	30-300	Moderate increase
A3	>300	>300	Severe increase

B. Causes, Risk factors of CKD and Risk multiplier for other NCDs

Causes and Risk Factors of CKD - CKD is not a single disease. It is a syndrome which has many causes. Several general factors which contribute to the high prevalence of CKD are high prevalence of low birth weight, hypovitaminosis, maternal malnutrition and infections in pregnancy by producing congenital low nephron number and smaller kidney volumes at birth. The common causes of CKD are as below:

1. Diabetes mellitus (Commonest cause responsible for 35-40% CKD patients)
2. Hypertension (2nd common cause responsible for ~15% CKD patients)
3. Glomerulonephritis (~ 15% cases of CKD)
4. Tubulo-interstitial diseases including renal stone and Vesico-Urethral Reflux (VUR) (~15% CKD cases)
5. Cystic diseases of kidneys (~5% CKD patients)
6. CKD of undetermined cause (~10% CKD cases)
7. Other miscellaneous causes (AKI, CVD, Obesity, etc.)

Diabetes - It is both an important risk factor and the leading cause of CKD world over and in India. Type 1 diabetes (T1DM) is known to progress through a characteristic set of stages starting with glomerular hyper filtration, microalbuminuria, overt proteinuria, decreased renal function and ultimately ESKD over the next 25-30 years. Type 2 diabetes (T2DM) is more common as risk factor for CKD. T2DM has increasingly been known to present as non-proteinuric CKD. Recent studies have shown that nearly 30% patients present with raised serum creatinine without proteinuria. These patients tend to be older and have a greater burden of atherosclerosis and hypertension.

Hypertension - It is both a cause as well as consequence of CKD. The rampant ignorance and lack of awareness regarding the possible long term adverse impact of hypertension on the kidneys often results in CKD and other diseases as well.

Glomerulonephritis (GN) - Group of mostly immune mediated diseases which lead to progressive CKD when left untreated.

Tubulointerstitial diseases (TID) - These are group of kidney diseases which primarily affect tubules of kidney and finally leads to decrease in kidney function.

Cystic diseases of kidney - These are hereditary diseases of kidneys and runs in families which leads to CKD. One of the common diseases in this group is polycystic kidney disease (PKD).

Prior Acute Kidney Injury (AKI) - Patients with (AKI) have high in-hospital mortality and among those who survives, approximately 20% of them tend to progress to CKD. Recurrent AKI are associated with a much higher risk of CKD and its progression to ESRD.

Cardiovascular diseases (CVDs) - CVDs are often linked to CKD due to the underlying common risk factors.

Obesity - Obesity (BMI > 30 kg/m2) is now an established risk factor for CKD due to its close association with diabetes, hypertension, and obesity related glomerulopathy.

Other factors - Consanguinity can increase the risk of congenital anomalies of the kidney and urinary tract and several inherited disorders of the glomerulus. Rampant poverty, unsanitary living conditions, overcrowding, water and soil contaminants both known and unknown (including herbal remedies, heavy metals like mercury and lead) may lead to AKI and chronic diseases of the interstitium and of the tubules in the kidney. Low birth weight is associated with lesser nephron numbers and in later life are linked with development of CKD. In addition, low birth weight is also associated with diabetes and hypertension in later life, secondarily causing CKD.

Interplay with other Non-communicable diseases

The impact and burden of CKD on healthcare is substantial not only because of CKD itself but also because CKD affect other NCDs also. It often develops on a background of other NCDs like diabetes and hypertension contributing significantly to all cause morbidity and mortality. CKD itself results higher incidence of cardiovascular complications; coronary artery disease, heart failure and arrythmia and strokes. Also, patients with cardiovascular disease and strokes have higher chance of CKD. This makes it even more difficult to diagnose and treat. Therefore, CKD becomes a risk multiplier for other NCDs.

Further, Most NCDs share common metabolic and behavioral risk factors making them amenable to targeted interventions having benefits across the disease spectrum. Therefore, the need of the hour is to target the risk factors of CKD and strive for aggressive risk factor reduction, as this would not only aid in decreasing the burden of CKD and concurrently other NCDs, but it will also reduce the multiplier impact of CKD on other NCDs.

C. CKD progression and its factors

Once correctly diagnosed, CKD cannot be cured by any treatment. It is a progressive kidney disease and invariably progresses to ESKD irrespective of the cause of initial injury. Retardation progression of CKD and delaying the need for dialysis and/or renal replacement therapy (RRT) is one of the main purposes of conservative management of CKD.

Progression of CKD can occur in a linear and predictable pace or in a non-linear, rapid pace. Low nephron number and genetic factors are believed to contribute for rapid progression. Majority of the risk factor are potentially modifiable. Progression of CKD is mediated by multiple factors:

1. **Progression of initial diseases causing CKD**
   a. **Hypertension** - It accelerates glomerulosclerosis and other injurious cascades. Recommended target BP in CKD patients is <130/80 mmHg. A systolic BP of <120 mmHg is suggested if tolerated by the patient. Hypertension, resulting in increased intraglomerular pressure leads onto glomerulosclerosis.
   b. **Hyperglycemia** - A tight glycemic control has been demonstrated to prevent, minimize and retard microvascular and macrovascular complications of diabetes including diabetic nephropathy. Recommended target HbA1C is between 6-7. However, once diabetes has resulted into CKD, very tight control of diabetes should be avoided as in these patients' risk of hypoglycemia is common and very tight control does not add to benefit of retardation of CKD.
   c. **Proteinuria** - It augments renal injury by multiple mechanisms, including complement activation, induction of expression of several injurious cytokines and chemokines by tubular epithelial cells and promotion of interstitial inflammation.
2. **Intermittent acute factors causing AKI on pre-existing CKD**
   In a patient of CKD, any factor which can potentially cause AKI, usually results in progression of pre-existing CKD. Therefore, all such factors should be prevented and if they develop, then should be promptly treated. Some of the common acute factors are:
   a. **Volume depletion** - Any poor intake of fluid or extra loss of fluid due to vomiting, diarrhea and burns etc., will results into hypoperfusion of kidney and result AKI on CKD.
   b. **Hypotension** - Hypotension due to volume loss or due to excess anti-hypertensive medication, excess diuretics, poor cardiac function and decrease in effective circulatory volume will result ischemia, more so due to loss of autoregulation in failing kidney and will cause ischemic AKI.
   c. **Systemic infection** - Infections like pneumonia, bacteremia, cellulitis and foot infections in diabetics can result in progression of CKD by increasing inflammatory burden. Infectious episodes can cause AKI due to hypotension, cytokine release and nephrotoxicity of antimicrobials used.
   d. **Urinary tract infection** - In addition to mechanism related to infection, it also causes direct kidney damage resulting in AKI on CKD.
   e. **Nephrotoxin drugs** - Nephrotoxins can hasten CKD progression by causing AKI, chronic tubulointerstitial nephritis or glomerular disease. Hence, nephrotoxic drugs and agents must be avoided in CKD patients. Common nephrotoxic drugs include non-steroidal anti-inflammatory drugs (NSAIDs), aminoglycoside antibiotics, proton pump inhibitors, anti-cancer drugs (cisplatin, ifosfamide, gemcitabin, etc.,), tenofovir, colistin and amphotericin.
   f. **Urinary tract obstruction** - Any obstruction to urinary tract results into backpressure changes and decrease in GFR, resulting additional AKI to pre-existing CKD.
   g. **Hypercalcemia** - It is a known cause of AKI by causing volume loss, vascular constriction and intratubular deposition.
   h. **Contrast nephropathy** - Radiocontrast studies are an important cause of AKI. Pre-existing CKD is most important risk factor for radiocontrast induced AKI.
3. **Compensatory mechanism and complications of CKD**
   a. **Protein Restriction** - Dietary composition influences acid-base balance, with animal-derived proteins contributing to generation of hydrogen ions. In addition, protein causes hyperfiltration of remaining functioning nephron and leads to glomerular sclerosis. Current guidelines suggest consideration of dietary protein restriction to <1.0 g/kg/day for patients with Stage 3 CKD, and <0.8 g/kg/day for patients with Stage 4-5 CKD.
   b. **Secondary hyperparathyroidism** - Failing kidneys lead to phosphate retention, decrease in active vitamin-D3 and secondary hyperparathyroidism to compensate divalent abnormalities. Secondary hyperparathyroidism increases the CKD progression.
   c. **Acidosis** - Metabolic acidosis is a complication of CKD, particularly when glomerular filtration rate falls to <30 ml/min. With decreasing renal mass, there will be accumulation of acid. Metabolic acidosis contributes to a host of complications of CKD viz. growth retardation, bone disease, loss of muscle mass, enhanced protein catabolism and progression of CKD.

d. **Smoking** - It induces oxidative stress, causes endothelial dysfunction, enhances micro-inflammation, and favors a pro-thrombotic state. Rapid worsening of glomerulosclerosis and tubular atrophy has been demonstrated with smoking.

e. **Obesity** - It worsens CKD through direct (i.e., inflammation, and alternations of glomerular hemodynamics) and indirect (i.e., increased risk of diabetes, hypertension) mechanisms.

f. Obstructive Sleep Apnoea (OSA) - It is more prevalent among CKD patients & can cause and perpetuate kidney injury. OSA is associated with sleep fragmentation and hypoxemia with resultant activation of sympathetic system and renin-angiotensin-aldosterone axis. Independent of obesity, OSA, per se can cause glomerular hypertension and cause progression of CKD. Also, OSA is associated with hypertension which would enhance CKD progression.

g. **Hyperlipidemia** - Dyslipidemia contributes for the high cardiovascular morbidity and mortality in CKD and possibly for progression of CKD.

Figure 1: Risk Factors for Progression

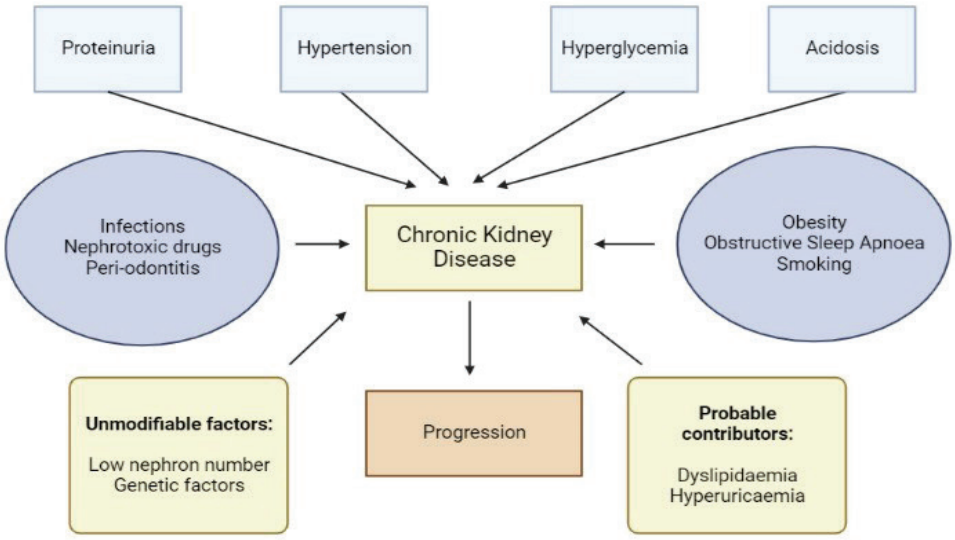


Table 3: actors causing progression of CKD and treatment measures

CKD Progression Factors	Management
Hypertension	BP control Target BP:<130/80 mmHg
Hyperglycaemia	Target HbA1C < 7
Proteinuria	RAAS inhibitors
Acidosis	Oral bicarbonate supplementation
Nephrotoxic agents	To be avoided
Obesity	Weight reduction,
Obstructive sleep apnoea	Weight reduction, CPAP if needed
Smoking	Cessation of smoking
Periodontitis	Antimicrobials
Other infections	Appropriate antibiotics
Protein consumption	Restriction of protein

D. Presentation of CKD

Patients of CKD present with different clinical presentations.

1. **Asymptomatic**

   In early stage of CKD (Stage 1-3), large number of patients may not have any symptoms related to CKD. Such patients are detected

   - On routine medical check-up

- During pre-insurance check-up
- On tests done before any surgery
- During ante-natal check-up
- On screening for CKD in high-risk population for CKD

2. Symptoms related to cause of CKD

Some patients may present with symptoms related to the cause of CKD

- Hypertension in CKD
- Edema in CKD due to glomerular disease
- Joint pains and fever due to vasculitis and connective tissue disorder
- Pain in abdomen and family history in patients with cystic disease
- Hematuria and oliguria in renal stone disease causing CKD

3. Acute Kidney Injury (AKI)

In a normal course, CKD is a slowly developing kidney disease. However, patients with CKD are at high-risk for AKI. If CKD patient develops AKI, then patient may present with symptoms with short duration of illness like any other AKI. In this situation one needs to differentiate true AKI from AKI superimposed on pre-existing CKD as treatment and prognosis of the two conditions will be different.

4. With Uremic symptoms

Uremic symptoms or symptoms of advance stage of CKD are mostly seen in stage 4 and 5, when GFR drops below 15-20 ml/min. Common uremic symptoms seen in advanced stage of CKD are:

- Anorexia
- Nausea/vomiting
- Weakness
- General feeling of being unwell
- Decreased effort tolerance
- Insomnia
- Loss of concentration

5. With Uremic complications

Patients of CKD are prone for complications in multiple organ system. Some of the common complications are:

- Pulmonary edema
- Heart failure
- Sepsis, commonly in chest and urinary system
- Acidosis
- Gastrointestinal bleeding
- Coronary artery disease

E. Diagnosis of CKD and its pitfalls

Evidence of kidney disease can be found on urine abnormality, radiological abnormalities, and evidence of kidney damage by increased serum creatinine. For diagnosis of CKD, both in hospital setting as well as in community, urine albuminuria and GFR measurements both are crucial and practical parameter. As 'measurement' of GFR is cumbersome process and impractical in community setting, GFR is 'estimated' by various formulas using S. Cr. value, so called estimated GFR (eGFR).

GFR and eGFR

GFR is used to estimate the burden of kidney disease at individual and population levels. Accurate GFR measurement is a difficult task without research setting as the golden method is the measured GFR with urinary clearances of inulin (which is an ideal filtration biomarker) being given in continuous IV infusions. Because of cost and inconveniences for using in large epidemiologic studies the concept of eGFR came

up. The two most used biomarkers for estimating eGFR in clinical practice are S. Cr. and plasma cystatin C. Of these two biomarkers, S. Cr. is more widely used and is commonly available in most places.

In terms of equation based eGFR derivation, over time 4 such equations came viz. Cockroft gault or CG (obsolete); Modified in diet in renal disease (MDRD) equation; CKD -EPI (2009); Race independent CKD-EPI 2021 eGFR. (***Equations at Annexure I***)

In large epidemiological studies, CKD EPI is being preferred since its inception in 2009. It applies a lower exponent to the S. Cr. value for low recorded creatinine concentrations (<0.7 mg/dl) for males and <0.9 mg/dl) for females. So, for GFR values >60 ml/min/1.73m2, CKD EPI is better and improves systemic bias by 67% compared with that of the MDRD equation in this subgroup.

National Kidney Foundation-American Society of Nephrology (NKF-ASN) Task Force recommends the adoption of the new eGFR 2021 CKD EPI creatinine equation that estimates kidney function without a race variable, however, it still has not come to practice in many countries.

Stages of CKD - National kidney foundation, USA has staged CKD into five stages based on GFR. Following table gives various stages of CKD (Table 4): In addition to stage number, if patient is on dialysis then "D" has to be added and if patient had transplant then "T" has to be added. For example, stage 3 CKD with transplant, needs to be written as Stage-3T.

Stage	Description	GFR ml/mt/1.73 m2
1	Evidence of Kidney disease with normal or ↑ GFR	> 90
2	Mild ↓ in GFR	60-89
3a	Mild to Moderate ↓ GFR	45-59
3b	Moderate to severe ↓ GFR	30-44
4	Severe ↓ GFR	15-29
5	Kidney failure	< 15 or on Dialysis

Albuminuria Measurement

By itself, eGFR is not sufficient for identifying stage 1 and stage 2 CKD, because in those patients the GFR may in fact be normal to near normal. In such cases, the presence of albuminuria is key measurement to define stage-1 and 2 CKD. Following are various other markers of kidney disease, though from a public health perspective in community setting, albuminuria is the only point of care marker which can be done for making diagnosis of CKD.

- Albuminuria (albumin excretion > 30 mg/24 hours or albumin creatinine ratio > 30 mg/g
- Urine sediment abnormalities
- Electrolyte and other abnormalities due to tubular disorders
- Histologic abnormalities
- Structural abnormalities detected by imaging

Finally, for labelling a patient of kidney disease as "chronic", the same tests need to be repeated after 3 months to qualify it as "chronic kidney disease".

Proteinuria Measurement

a. **Semiquantitative methods** - The commonest semiquantitative method available to screen patients for proteinuria is standard urine dipstick test.

   ➢ **Standard urine dipstick** - The standard urine dipstick primarily detects albumin and is relatively insensitive to non-albumin proteins. Thus, a positive dipstick usually reflects albuminuria (or indirectly glomerular proteinuria). The dipstick is very specific but not sensitive to low levels of albumin excretion. The lower limit of detection is a urine albumin concentration of approximately 10 to 20 mg/dL. Thus, patients with moderately increased albuminuria (formerly called "microalbuminuria") will usually not be identified by this method unless the urine is highly concentrated.

   Approximately corresponding value of UPCR with dipstick test is as below

   - Trace UPCR < 30 mg/g

- 1+ UPCR 30-300 mg/g
- 2+ UPCR > 300 mg/g
- 3+ UPCR 300 - 3500 mg/g
- 4+ UPCR > 3500 mg/g

False positive urine dipstick results may occur in the following settings:

- Use of iodinated radiocontrast agents (Test after 24 hours of agent use)
- With a highly alkaline urine (pH > 8)
- Presence of gross hematuria and a urocrit (the percent of urine volume comprised of red blood cells) greater than 1%
- Antiseptics use e.g., chlorhexidine and benzalkonium

b. **Quantitative methods** - The quantity of protein excretion is clinically important for several reasons:
   - Most benign forms of isolated proteinuria are < 1 g/day.
   - The higher degree of proteinuria is prognostically bad for glomerular diseases
   - The degree of proteinuria is used to monitor the response to therapy in glomerular diseases

   ➢ 24-hour versus spot urine collection - Patients with persistent proteinuria should have a quantitative measurement of total protein excretion. The gold standard for measurement of protein excretion is a 24-hour urine collection, with the normal value being less than 150 mg/day.

   ➢ Due to the limitations of a 24-hour urine collection, several alternatives have been proposed. These involve measuring the ratio of protein to creatinine (UPCR) (or urine albumin-to-creatinine ratio (UACR)) in urine specimens of less than 24 hours' duration. Most commonly, UPCR in a spot first or second morning urine sample after avoiding exercise is used to estimate 24-hour proteinuria. Usually, the urine protein and creatinine in a spot sample is measured in mg/dL and a ratio is obtained.

Table 5 Proteinuria and albuminuria

	Urine Collection Method	Normal	Micro Albuminuria	Clinical Proteinuria
Protein	24 Hour Urine	< 300 mg/day	Not Applicable	> 300 mg/day
	Dipstick	< 30 mg/dl	Not Applicable	> 30 mg/dl
	Spot Urine PCR	< 200mg/g	Not Applicable	> 200 mg/g
Albumin	24 Hour Urine	< 30 mg	30-300 mg	> 300
	Dipstick	< 3mg/dl	> 3 mg/dl	Not applicable
	Spot Urine ACR	< 17mg/g male < 25 mg/g Female	17-250 mg/g male 25-355 mg/g female	> 250 mg/g male > 355 mg/g female

PCR- Protein Creatinine Ratio, ACR-Albumin Creatinine Ratio

Radiological evidence of kidney disease

Features which suggest CKD due to common diseases includes hydronephrosis in patients with urinary tract obstruction or with vesico-ureteric reflux, cysts suggesting autosomal dominant or recessive polycystic kidney disease, increased cortical echoes as an indicator of little advanced glomerular, tubulointerstitial or vascular diseases. Depending on the anticipated abnormality, the imaging method could be chosen like Plain X-Ray abdomen, Ultrasonography of abdomen or CT scan.

Other abnormalities in urine for evidence of CKD are red cells, leucocytes or cellular casts. Dimorphic red cells accompany red cell casts in glomerulonephritis and pyuria in the context of pus cell casts

signifies tubulointerstitial nephritis. A single urine sediment examination may suffice but on occasion, when the index of suspicion is high, repeat examinations may be required. Urine dipsticks that are able to detect red blood cells (hemoglobin), neutrophils and eosinophils (leukocyte esterase) and bacteria (nitrites) are available. However, these dipsticks are incapable of detecting tubular epithelial cells, fat or casts in urine.

F. Principles of Management of CKD

CKD is usually a relentlessly progressive disease. There are four basic aims of management of any CKD -

i. Retarding progression of CKD to delay the need of RRT
ii. Keep the patient symptom free so that he/she can lead near normal life
iii. Prevent and treat complications
iv. Best prepare patient for most suitable RRT for him

The cornerstone of the conservative treatment of CKD is to identify the risk factors, delay the progression, manage the complications arising out of CKD progression, and smooth the transition from CKD stage 4 to stage 5 CKD, also called ESKD. The interaction between social and biological factors affecting the progression of CKD is shown in **Figure-1.** Early referral to nephrologists has better outcomes with the initiation of kidney replacement therapy (KRT), which include dialysis and/or kidney transplant (KT).

Step-1: Lifestyle management for patients with CKD

- **Smoking cessation**
- **Weight reduction**

  Obese and overweight people should be encouraged to reduce their BMI. Maintenance of healthy body weight of 18.5-22.9 kg/m^2; waist circumference <90 cm for men, and < 80 cm for women, is recommended to reduce blood pressure in those with hypertension.
- **Dietary protein control**

  A protein-controlled diet (0.8 g/kg/d) is recommended for adults with CKD. Protein < 0.80 g/kg/day should be carefully monitored for clinical and biochemical markers of nutritional deficiencies and malnutrition.
- **Alcohol intake**

  Alcohol consumption should be discouraged to control hypertension.
- **Exercise**

  A total of 30 minutes, 5 times a week exercise may be a helpful measure for protection from cardiovascular problems. Higher intensity exercises should be avoided.
- Dietary salt intake
- A dietary sodium intake of < 100 mmol/day (~ 5 g of table salt) is recommended to control hypertension. Patients with TID may not require reduction in salt intake.Normal salt should be decreased rather than using other alternate salt preparations.

Step 2: Intra-renal factors associated with progression of CKD and its management

Interventions to slow the progression of CKD to ESKD are cost-efficient approaches. It synergizes for the management of diabetes, hypertension, and vascular disease. It can be easily integrated with other programs aimed at the country's NCDs control programs.

Management of native kidney disease and identification of its reversibility:

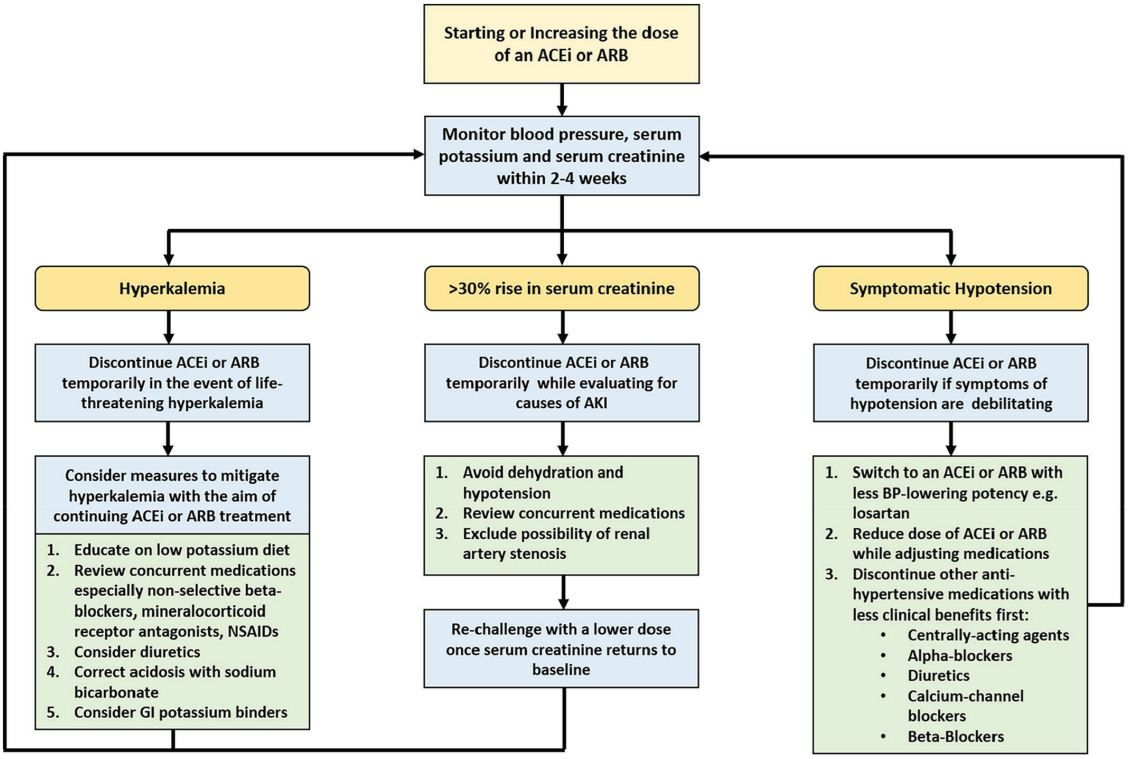
Ongoing active native kidney diseases, like early diabetic nephropathy, primary and secondary glomerular diseases, interstitial diseases, obstructive nephropathy, and cystic diseases need to be identified as there may be still some reversible component of native kidney disease, control of which can retard progression of CKD. Early referral of such patients to nephrologists may help identify reversibility of native kidney disease.

Step 3: Conservative management of clinically relevant factors

There are seven crucial clinically relevant factors which occur due to CKD and perpetuate the progression if not taken care of adequately. These factors are also accountable for major morbidities, hospitalization, and mortality of these patients. Most of them are modifiable risk factors and dealing with them can slow down the progress of the disease and reduce uremic complications -

3.1 Hypertension - ACEi and ARB retard CKD's progression through hemodynamic and non-hemodynamic ways by preventing fibrosis. CKD patients also require multiple classes of antihypertensive together to control their blood pressure. The choice of drugs depends on degree of proteinuria and other side effects. The broad guidelines of management of hypertension in CKD patients is as below:

Figure 2: Monitoring of serum creatinine and potassium levels during ACEi or ARB treatment-dose adjustment and monitoring of side effects



Patients with proteinuric CKD (UACR ≥ 30 mg/mmol) **or Diabetic CKD**

- Antihypertensive therapy should include either an ACEi or an ARB.
- Combinations should not be used.
- Blood pressure target should be < 130/80 mm Hg.

Patients with non-proteinuric CKD (UACR < 30 mg/g) **and Renovascular Disease**

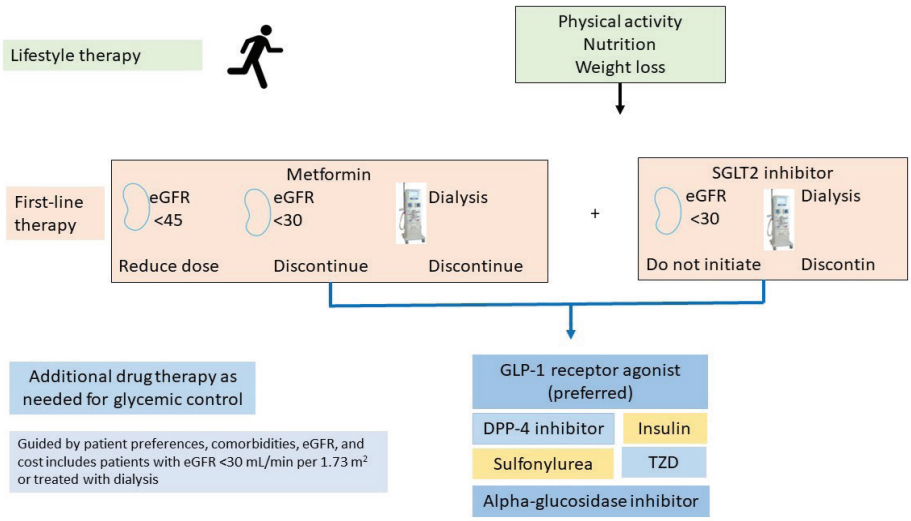
- Antihypertensive therapy should include either an ACEi or ARB
- A long-acting calcium-channel blocker or thiazide diuretic or β-blocker in various combination

3.2. Diabetes mellitus - Besides RAS blockers, the addition of SGLT2i, further slows the progression of DKD by reducing intraglomerular hypertension and hyperfiltration injuries. Approximately one-third of patients in diabetes may have non-albuminuric diabetic kidney disease. A proposed guideline for controlling diabetes in CKD with respect to retard the progression of CKD are as below:

Glycemic control

- Targets for glycemic control should be HbA1c 6.5 to 7.0%
- Glycemic control should be part of a multifactorial intervention strategy that addresses blood pressure control and cardiovascular risk prevention with the use of RAS blockers, statins, and acetylsalicylic acid.

Figure 3: Practice points for the use of metformin and other hypoglycemic agents in CKD patients



Antihyperglycemic Therapies

- Patients with eGFR ≥30 mL/min per 1.73 m^2 should be preferably treated with SGLT2i. with or without metformin as first-line therapy.
- Patients who have not achieved HbA1c targets despite the use of metformin and SGLT2i, or who are unable to use these medications, a long-acting GLP-1 RA can be safely used.
- The additional therapy of oral hypoglycemic agents (OHA) may be chosen from the cartoon wheel depicted in the figure- depending on the side effects of the medicines and comorbidities associated with the patient.

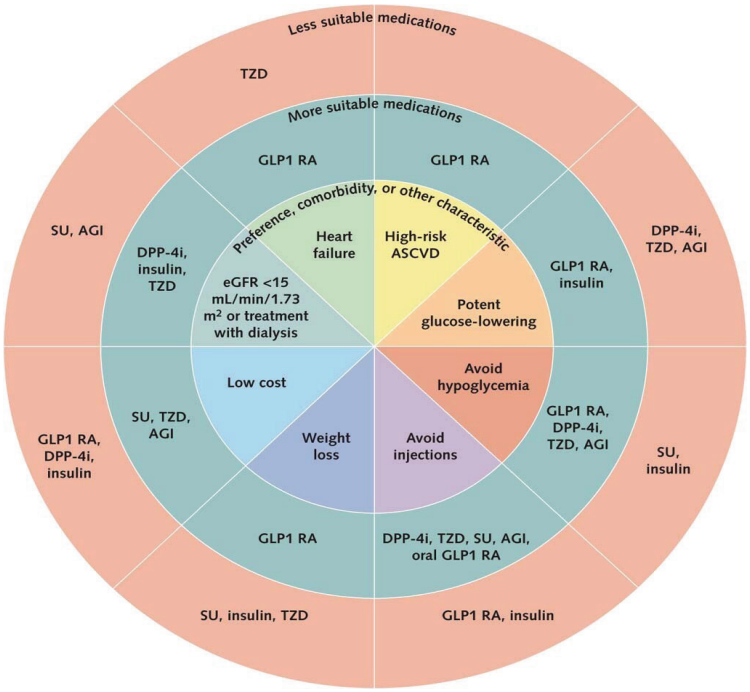
Use of metformin in T2DM (Fig.3)

- It is recommended for most patients with T2DM with stable stage 1 or 2 CKD.
- Metformin may be continued in patients with stable stage 3 CKD.
- Metformin should be stopped if there are acute changes in renal function. A particular care should be taken for patients taking RAS blocker, NSAID or diuretics, or after IV contrast administration because the risk of AKI and thus accumulation of lactic acid, is greatest in these patients.

Choice of other glucose-lowering agents

- Other glucose-lowering agents (including insulin) needs to be tailored to the individual patient, according to the level of renal function, and comorbidity. The choice of the other antihyperglycemic agents and risk factor of the patients in figure 4 below:

Figure 4: Patient factors influencing selection of glucose-lowering drugs other than SGLT2 inhibitors and metformin in T2DM and CKD.



AGI = α-glucosidase inhibitor; ASCVD = atherosclerotic cardiovascular disease; DPP-4i = dipeptidyl peptidase-4 inhibitor; GLP1 RA = glucagon-like peptide-1 receptor agonist; SU = sulfonylurea; TZD = thiazolidinedione. (Ref-KDIGO Diabetes Work Group. KDIGO 2020 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease. Kidney Int. 2020;98:S1- S115).

Risk of hypoglycemia

- It should be assessed regularly for patients taking insulin or insulin secretagogues.
- These patients should be taught how to recognize, detect and treat hypoglycemia.
- Short-acting sulfonylureas (e.g., gliclazide, glipizide) are preferred over long-acting agents in CKD.

3.3 Proteinuria evaluation and management

Proteinuria is marker of glomerular disease. Timely detection, evaluation, and treatment of the underlying diseases causing proteinuria is a valuable tool in slowing the progression and management of CKD.

Broad Guidelines for the measurement of proteinuria in CKD

- Adults with diabetes and persistent UACR > 2.0 mg/g should receive an ACEi or an ARB to delay the CKD progression.
- **Finerenone,** a nonsteroidal, selective mineralocorticoid receptor antagonist is shown to reduce proteinuria beyond the use of ACEi/ARB.
- Use of **SGLT2 inhibitors** has been found to lower albuminuria in patients with diabetic CKD by at least 30%, with favorable renal and cardiovascular outcomes
- Protein-controlled diet, as well as weight reduction in obese patients provide some benefit in decreasing proteinuria. Protein restriction in a diet of 0.6-0.8 g/kg per day may help reduce hyperfiltration and retard the progression of CKD

3.4 Dyslipidemia in CKD progression and management

The spectrum of lipid disorders in CKD is usually characterized by high triglycerides and reduced high-density lipoprotein (HDL), associated with normal or slightly reduced low-density lipoprotein (LDL)-cholesterol. This dyslipidemia is associated with an increased risk for atherosclerotic cardiovascular disease. The suggested guidelines for managing dyslipidemia in CKD are as below:

Screening and frequency of measurement

- A fasting lipid profile (total cholesterol, LDL, HDL, and triglyceride) should be measured with stage 1-3 CKD. Lipid profiles should be measured after an overnight fast (ideally ≥ 12 h duration).
- It should be measured no sooner than 6 weeks after initiation or change in pharmacologic therapy.
- It should be monitored every 6-12 months if the results could influence subsequent therapeutic decisions.

Treatment -

Population	CKD Stage	Treatment Recommendations
Adults ≥ 50 y	1-2	Statin
	3-5 (not on HD)	Statin plus Ezetimibe; Statin
Adults 18 - 49 y + ≥1 of the following: Known coronary disease (MI or coronary revascularization) Diabetes mellitus Prior ischemic stroke Estimated 10-y incidence of coronary death or non-fatal MI >10%	1-5 (not on HD)	Statin
Adults on dialysis	5 (HD or PD)	Statins or statin combinations should NOT be initiated; can be continued if already received at the time of dialysis initiation
Adult kidney transplant recipient	1-5	Statin

Monitoring for medication adverse effects

- Serial monitoring of creatinine phosphor-kinase (CPK) and alanine aminotransferase (ALT) is not required for asymptomatic patients with CKD taking a low to moderate dose of statin (≤ 20 mg/d atorvastatin, or an equivalent dose of another statin).
- CPK and ALT should be measured every 3 months for patients with CKD4, who are on moderate to high dose of statin (≥ 40 mg/d atorvastatin, or an equivalent dose of another statin).
- A statin and fibrate should not be co-administered in CKD4 because of the risk of rhabdomyolysis.
- Gemfibrozil is safe in CKD. Other fibrate preparations (e.g., fenofibrate) should be avoided or the dose significantly reduced in CKD 2 and beyond because of an increased risk of toxicity

3.5 Management of Anaemia in CKD

Anemia occurs in CKD because of multiple factors; a significant contributor is EPO deficiency which may be observed as early as CKD-stage 2. The guideline for the management of anemia is as below:

- **In CKD patients without anemia, measure Hb** when clinically indicated, at least annually in CKD3, twice per year in CKD 4-5
- **For CKD patients with anemia not being treated with an ESA,** measure Hb concentration when clinically indicated, at least every 3 months in CKD 3-5
- Anemia is defined as a Hb. < 13 g/dl for adult men and < 12 g/dl for adult women.
- Initial evaluation should include Hb, leukocyte count and differential, platelet count, erythrocyte indices, absolute reticulocyte count, serum ferritin and transferrin saturation.

Use of erythropoiesis-stimulating agents

- In patients with adequate iron stores, ESA should be initiated if Hb. Is < 10 g/dl.
- For patients on ESA, the target Hb. should be between 10-12 g/dl.
- ESA should be prescribed in consultation with specialist

Use of hypoxia-inducible factor- prolyl-hydroxylase inhibitor HIF-PHI (Roxadustat, Desidustat, daprodustat etc.):

- If target Hb is not achieved with the desired dose of ESA, in that case, we should search for the cause of ESA hypo responsiveness. If the cause of ESA hypo responsiveness is unknown or difficult to manage due to any reason, HIF-PHI should be considered.

- It should not be used in patients with malignancy, people with visual problems, and hepatic dysfunction.

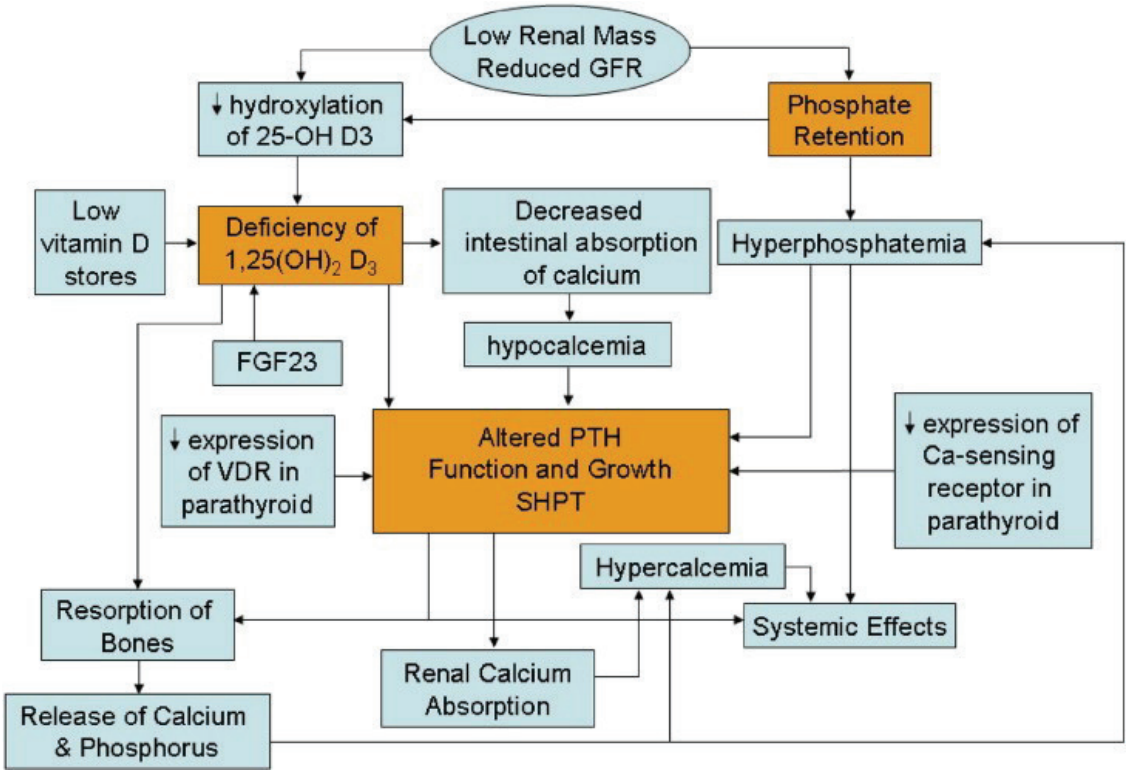
Use of iron therapy

- For patients on or not on ESA and who have a hemoglobin level < 11 g/dl, iron should be administered to maintain a level of ferritin > 200 ng/mL and transferrin saturation > 20%
- The oral form of iron is the preferred first-line therapy for patients with CKD.
- Patients who do not achieve serum ferritin or transferrin saturation targets or both while taking the oral iron or who do not tolerate the oral form should receive the intravenous form of iron

3.6 PO4 - calcium - vitamin D - PTH axis dysregulation in CKD

PO4-Ca-vitamin D-PTH axis dysregulation starts at a very early stage of CKD; however, clinically evident PO4 retention occurs when GFR usually falls below 30 ml/min/1.73 m2.

Figure 5: Mechanisms demonstrating Ca-PO4-PTH, calcitriol, and FGF-23 homeostasis in CKD.



Guidelines for the assessment and treatment of CKD-MBD -

Assessment and therapeutic targets

- Serum Ca, PO4, and iPTH should be measured for adults with CKD 3-5
- Serum Ca and PO4 should be maintained within the normal range.
- Intact PTH may be elevated above normal values; the target is unknown.

Frequency of monitoring of CKD-BMD components are as below:

Stage of CKD	Ca	PO4	Alk-PO4	PTH	25 (OH)Vit-D
Stage-3	6-12 month	6-12 Mon	6-12 month	Depend on baseline Value	Depend on baseline Value
Stage-4	3-6 month	3-6 month	6-12 month	6-12 month	Depend on baseline Value
Stage-5	1-3 month	1-3 month	3- 6month	3-6 month	Depend on baseline Value

Treatment options

- Dietary PO4 restriction should be used continuously to manage hyperphosphatemia
- Calcium-containing PO4 binders (Ca carbonate or acetate) should be initiated if dietary restriction fails to control hyperphosphatemia and Ca is not high.
- If hypercalcemia develops, the doses of calcium-containing phosphate binders or Vit. D analogues if being given should be reduced.
- Non-Ca PO4 binders Sevelamer carbonate, or Lanthanum carbonate are also used as a phosphate binder
- Hypocalcemia should be corrected if the patient has symptoms or if it is associated with increased iPTH levels.
- Vit. D analogues should be used if serum levels of iPTH are > 53 pmol/L. Therapy should be discontinued if hypercalcemia or hyperphosphatemia develops or if iPTH levels are < 10.6 pmol/L.
- Vit. D analogues should be used in consultation with a specialist.
- Cinacalcet, may be used to lower PTH. It activates the CSR of the parathyroid gland amplifying the glands' sensitivity to extracellular ionized Ca concentration resulting in suppression of PTH secretion, along with reducing Ca x PO4 product.

3.7 Metabolic acidosis

Metabolic acidosis (serum bicarbonate is consistently < 22 mEq/L) occurs as a complication with the decline in GFR and retention of organic and inorganic acids. It should be treated with oral alkali to mitigate these adverse consequences.

Potential Benefits of alkali therapy

- Reduce risk for CKD progression
- Increase skeletal muscle mass and strength
- Reduce bone buffering and preserve bone mineral
- Reduce serum potassium if hyperkalemic

Potential Risks of alkali therapy

- Increased blood pressure,
- Fluid retention,
- pulmonary and peripheral edema with sodium-based formulations
- Hypokalemia if bicarbonate is excessive
- Vascular calcification
- Kidney calcification
- Calcium phosphate nephrolithiasis

3.8 Hyperuricemia and CKD progression:

Kidneys are responsible for most of the daily uric acid excretion (65-75%), with the remaining (25-35%) being excreted through the gastrointestinal tract. Uric acid accumulates with the decline in renal function; however, its role in the initiation and progression of kidney disease is controversial and debatable. Uricosuric agents are not used and other urate lowering agents like allopurinol and Febuxostat needs dose modifications, based on GFR.

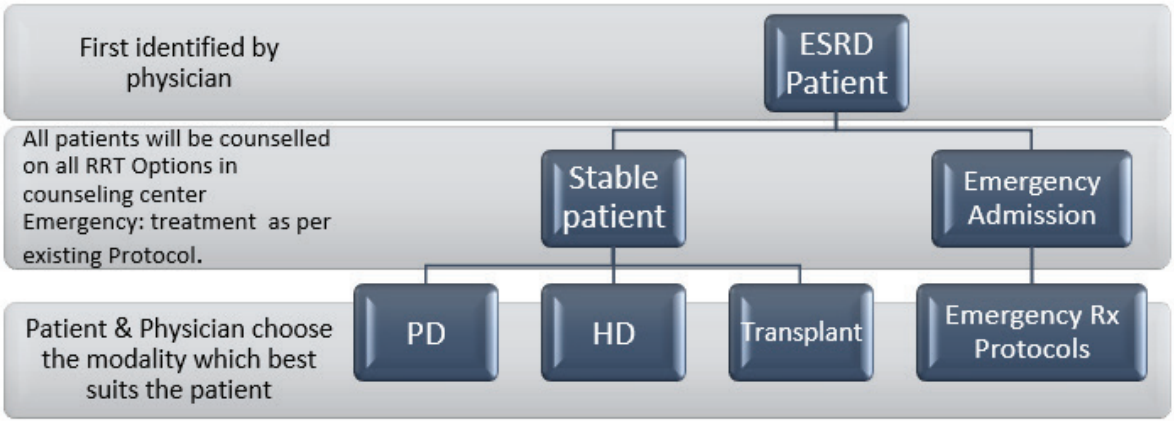
Step 4. Kidney replacement therapies

A well-managed CKD patients have a smooth transition to the stage of RRT. In the last stage of CKD, RRT is considered to improve longevity and quality of life. ESKD patients with uremic symptoms (usually with eGFR <5 to 10 ml.min/1.73m2) are managed by either KT or dialysis. The dialysis is usually provided broadly in the form of either HD or PD. These modalities are collectively called RRT. Supportive care involves with shared decision-making help in choosing the best RRT modality suitable for patient.

In the integrated care approach for KRT **(Figure 6**), KT, HD, and PD *are* complementary to each other and not competitive. The transfer of patient from one method to another should not be considered to

be a “failure” of the previous method. Among RRT modalities, KT in suitable patients is associated with the best clinical outcomes at the lowest cost. PD and HD are associated with almost similar outcomes.

Figure 6: Integrated approach for stable and emergency patients needing KRT



Emergency indication of dialysis:

i. Refractory pulmonary odema
ii. Refractory hyperkalemia
iii. Severe metabolic acidosis
iv. Uremic pericarditis
v. Uremic encephalopathy

These patients are stabilized with dialysis and counselled for the selection of modality for RRT.

Kidney transplantation (KT) - It is the process of placing a kidney in ESKD patients from a healthy, voluntary live kidney donor or deceased donor. KT is the modality of choice for ESKD patients with advantages of cost-effectiveness and longevity. A KT recipient requires lifelong immunosuppression to maintain the graft function and avoid acute rejection.

Peritoneal dialysis (PD) - It uses the peritoneal membrane as a filter to clear uremic wastes and extra fluid from the body. The PD process is based on an exchange with commercially available PD fluid. A patient undergoes between 3 to 4 exchanges each day. The PD preserves residual kidney function (RKF) better than HD, and RKF offers a better quality, nutrition, haemoglobin, and overall mortality benefit. PD can be provided at home with initial training to the patient helper or patient himself.

Most paediatric patients who require chronic dialysis worldwide are managed with PD, as vascular access is difficult to create and maintain in paediatric ESKD patients. The blood-borne infections are more common in HD patients; however, peritonitis, considered the Achilles heel for PD, occurs due to touch contamination in PD patients. PD is a more convenient modality to schedule into a daily routine.

Hemodialysis (HD) - HD is an extracorporeal treatment for ESKD that uses a machine to remove uremic waste through the dialyzer (called an artificial kidney). The ultrafiltration volume sets in the machine remove the water and also eliminates some amount of uremic toxins by convection methods. However, the newer modality hemodiafiltration (HDF) provides better outcomes than conventional HD at a higher cost. Continuous renal replacement therapy (CRRT) principles are based on convective clearance and provide more hemodynamic stability than conventional HD, offered to hemodynamically unstable and sick patients in intensive care units.

Under the Pradhan Mantri National Dialysis Program, many of the district hospitals in each state have been equipped with HD facilities, and dialysis is provided free of cost to the below poverty line patients. Now, PD has been proposed to be included under the PMNDP.

Principles of management of CKD by its stages

A patient with CKD will require life-long care. There are three major aims of CKD management:

1. Prevent any further damage to kidneys.

2. Retard the progression of CKD so that patient can avoid dialysis and/or renal transplant tillas late as possible.
3. During the time of management, patients remain symptom free with good quality of life.

Management in Stage 1-2

- In these earlier stages, patients are mostly asymptomatic as far as CKD is concerned. However, there may be symptoms related to cause of CKD or CKD may be accidently detected.
- Basic treatment in these stages is management of primary kidney disease so that renal function is preserved.
- Additional damage to kidney is also to be prevented in these stages.
- All potential acute factors of kidney damage should be prevented and promptly treated.
- Patients should be vaccinated for hepatitis B and other vaccination if not vaccinated already.

Management in Stage 3-4

- In the beginning of stage-3, patient should be referred to a nephrologist for planning further treatment.
- In these stages, patients start developing symptoms related to CKD, more so in stage - 4.
- In most kidney diseases, treatment of primary cause of CKD is not very useful in preserving renal function in these stages.
- Prevention and treatment of acute factors for kidney damage is still important.
- All factors for progression of CKD require regular investigations and management.
- Infections and cardiovascular complications are two major complications, which should be given close attention and treated promptly and adequately.
- Once in late stage-4, RRT should be discussed and preparation for vascular access for dialysis should be done.

Management in Stage 5

- In this stage, patient is close to need of RRT.
- Definite choice for RRT should be finalized and RRT started.
- If patient is still not started on RRT, then he should be closely followed to prevent any complication.

Drug handling in CKD

The kidneys excrete most medications and/or their metabolites. Patients with kidney disease have altered drug pharmacokinetics and pharmacodynamics, and consequently they are at risk of developing drug toxicities. In addition, these patients may have other co-morbidities (such as diabetes mellitus, coronary artery disease, congestive heart failure, etc.) and are on multiple drugs because of these co morbidities. This predisposes them for drug interactions and drug- related toxicities. Dose adjustments based on eGFR needs to be done for those medicines that are mainly excreted unchanged by the kidneys. Moreover, some medications can cause AKI due to their nephrotoxicity, which may further accelerate CKD progression. Hence, it is always advisable to exercise caution while prescribing any medicines in patients with CKD. Considering every drug is out of scope of these guidelines. It is always advisable to check the doses according to stage of CKD while prescribing in these patients. **(Table 6).**

Table 6: Commonly prescribed medicines and their dose adjustment in CKD

Commonly prescribed drug class	Precautions required in CKD
ANALGESICS	
NSAID eg. Ibuprofen, Diclofenac	Prolonged therapy is not recommended when GFR <60 mL/min/1.73 m^2 Safer alternative is Tab Paracetamol for fever and Tramadol for pain relief.
ANTIMICROBIALS	
Fluoroquinolones e.g., Levofloxacin, Ofloxacin	Reduce dose by 50% when eGFR <15 mL/min/1.73 m^2 Moxifloxacin does not require dose modification in CKD
Trimethoprim e.g., Septran / Bactrim	Reduce dose by 50% when GFR <30 ml/min/1.73 m^2 Risk factors for hyperkalemia include high doses, elderly, CKD, or with ACE-I and/or NSAIDs
Amino glycosides e.g., Amikacin, gentamicin	Use other alternative antibiotic if possible Monitor drug concentrations and serum creatinine levels and withdraw if creatinine rises Avoid multiple daily dosing
Azithromycin	No adjustment needed
Clindamycin	No adjustment needed
Antifungals	Reduce maintenance dose of fluconazole by 50% when GFR <45 mL/min/1.73 m^2
ANTIHYPERTENSIVES / CARDIAC MEDICATIONS	
Rennin angiotensin aldosterone antagonists e.g., ACE-I, ARB	Use with caution in patients with renal artery stenosis Start at lower dose in patients with GFR <45 mL/min/1.73 m2 Assess GFR and serum potassium 1-2 weeks after starting or escalating dose Consider temporarily holding during IV contrast administration, or any potential cause of volume depletion (bowel preparation prior to colonoscopy, acute illness, and surgery)
Beta blockers	Reduce dose of hydrophilic b-blockers (acebutolol, atenolol, bisoprolol, and nadolol) by 50% when GFR <30 mL/min/1.73m2 Metoprolol, labetalol and carvedilol do not require dose modification
Diuretics	Thiazide diuretics (eg. hydrochlorothiazide, chlorthalidone) generally become ineffective as diuretics when GFR is below 30 ml/min/1.73m2 Loop diuretics (furosemide and torsemide) remain effective at low GFR and are generally the preferred diuretics in renal impairment Potassium-sparing diuretics (eg triamterene) are the least effective diuretics and are often contraindicated in moderate to severe renal impairment because of the risk of life-threatening hyperkalemia
Calcium channel blockers	Pharmacokinetic parameters of calcium channel blockers are essentially unaltered in renal impairment.
e.g., Amlodipine, Nifedepine	Generally well tolerated and used in normal doses according to response.
Digoxin	Reduce dose according to plasma concentrations of drug
DIABETES MEDICATIONS	
Sulfonylureas	Avoid mainly renally excreted agents (eg, glyburide/glibenclamide) Agents mainly metabolized by the liver may need reduced dose when GFR
Metformin	May be continued in people with GFR ≥ 45 ml/min/1.73 m2 Use with caution for patients with a GFR of 30-45 mL/min/1.73 m2 Should be discontinued in people with GFR ≤ 30 ml/min/1.73 m2 Hold in patients during acute illness or before intravenous radio contrast
Insulin	Partly renally excreted and may need reduced dose when GFR <30

Commonly prescribed drug class	Precautions required in CKD
PROTON PUMP INHIBITORS (PPI)	
Pantoprazole, omeprazole	Recent evidence suggests that PPI exposure may be associated with increased risk of incident CKD, CKD progression, and ESRD. H2 receptor antagonists such as Ranitidine are safer
ANTI-TUBERCULAR DRUGS	
Rifampicin, Isoniazid, Pyrazinamide	No dose adjustment is required
Ethambutol	Should be used with particular caution in patients with renal impairment For GFR 10-20 ml/min/1.73m2 : 7.5-15 mg/kg/day For GFR < 10 ml/min/1.73m2 : 5 - 7.5 mg/kg/day Alternatively, for GFR < 30 ml/min/1.73m2 : 15-20 mg/Kg every 48 hours with monitoring of ethambutol plasma levels
Streptomycin	Should not be used in patients with any degree of renal
HERBAL MEDICINES: should be avoided in patients with chronic kidney disease.	

3
Role of Medical officer in prevention and management of CKD

Early detection of CKD

Reduce the risk of progression

Detecting the risk factors of other/concomitant NCDs diseases

Early detection and follow-up management of CKD associated complications

Referral to physician/nephrologists for consultation

Counseling and preparation for RRT for ESKD

Primary care physician and nephrologists need to work together to handle the public health problem of CKD. The great majority of CKD patients, particularly those up to stable stage 3 can be managed in primary care setting with guidance from Physician and nephrologists at higher centre. Medical officer at PHC is expected to have the following roles and responsibilities in prevention and management of CKD.

A. Early Detection of CKD
B. Reduce the risk of progression
C. Detecting the risk factors of other/Concomitant NCDs
D. Early detection, follow-up and management of CKD associated complications.
E. Referral for consultation at higher center
F. Counseling for Renal Replacement Therapy (RRT) for ESKD

A. Early Detection of CKD

Screening for CKD by urine dipstick for protein and sugar and blood test for serum creatinine

1. Opportunistic screening of all individuals > 30 years at higher risk for CKD
    a. Diabetes mellitus
    b. High blood pressure
    c. Family history of CKD
    d. Age > 60 years
    e. History of acute kidney injury (AKI)

B. Reduce the risk of progression

All patients of CKD have an inherent tendency to progress to more severe stage of CKD. One of the primary aims of treatment of these patients is to retard the progression of disease. Role of primary care physician in retarding the progression is as follows:

1. **Monitor and follow-up of patient in relation to treatment planned at higher centre**
   - Treatment of primary disease, causing CKD
   - Treatment in relation to factors causing CKD progression like proteinuria, hypertension, hyperparathyroidism, hyperlipidaemia etc.

2. **Prevent and manage acute factors responsible for CKD progression**
   - Any systemic infection
   - Fluid loss and dehydration
   - Avoiding nephrotoxic drugs (refer Chapter-1)

3. **Treatment and monitoring of hypertension**

Blood Pressure Target in CKD:

Blood pressure targets by degree of albuminuria		
	Degree of albuminuria	
BP Target (mm/Hg)	<300 mg/day	300 mg/day or more (dip stick method)
	140/90	130/80

Choice of Anti-hypertensive drug:

- The choice of antihypertensive therapy in CKD should be considered based on the level of proteinuria.
- ACE inhibitor or angiotensin II receptor blocker (ARB) as first-line therapy in patients with proteinuric CKD (i.e., albumin excretion greater than 300 mg/day by dipstick).
- Before starting on ACE-I/ARB therapy, S. Cr. and Potassium should be done. Significantly high creatinine (> 3.0 mg/dl) and high K (> 5.0 meq/L) may make patient unfit for these drugs.
- S. creatinine and serum K should be repeated after 1-2 weeks of starting ACE-I/ARB therapy and after every time dose is increased.

Note:

a. If serum creatinine rises > 30% or serum K rises > 5.5 meq/L, these drugs should be stopped and other drugs should be used.

4. **Treatment and monitoring of diabetes**

- Tight control of blood sugar in diabetic will delay the development and progression of the CKD.
- HbA1c between 6.5 to7 should be target of good control.
- The targets need to be relaxed in the advanced stage of CKD (Stage-4 and 5), individuals at high CVD risk and older individuals. (HbA1c ≥7).

C. Detecting the risk factors of other/concomitant NCDs

There are risk factors for CKD and other NCDs, which should be assessed for holistic care of CKD patients. Individuals with CKD even at earlier stages, i.e. 1, 2 and 3 are more likely to die from cardiovascular event than to reach the ESKD and need for RRT. Role of primary care physician in this regard is as follows:

1. Population based and opportunistic screening of all individuals > 30 years for DM & HT
2. Counseling for life-style modification
   - Weight control
   - Avoid smoking
   - Balanced healthy diet (Low salt and low cholesterol diet)
   - Regular exercise, at least 45 minutes, five days in a week. In case of heart disease, before starting exercise, consultation with expert is needed
3. Monitoring the treatment planned by higher centre for hyperlipidemia, cardiovascular disease etc.

D. Early detection and follow-up of management of CKD associated complications

The kidneys play a central role in maintaining normal Hb, bone and mineral metabolism and internal environment. With decline in GFR, there is disruption of these functions, which need to be investigated and managed. Role of primary care physician in this regard is as follows:

1. Monitoring of diet with an aim to control
    - Body fluid
    - High Blood pressure
    - Blood sugar
    - Hyperlipidemia
    - Hyperkalemia
    - Hyperphosphatemia,
2. Adjust doses of drugs as per eGFR
3. Screening and managing anemia due to other than erythropoietin deficiency like nutritional and blood loss

    (Given in Chapter 2 in detail)

E. Referral to physician/nephrologist for consultation

Patients of CKD can best be treated by a team of primary care physician and nephrologists. In many situation, treatment can be started by primary care physician. In other situations, it requires expertise of nephrologists. Although once treatment has been started by nephrologists, patient can be followed by primary care physician. Thus, it is important for primary care physician to know that in which setting, patient is to be referred to nephrologists. Primary care physician should broadly refer following patients to nephrologists/physician for consultation:

1. Patient of CKD, who need kidney biopsy for making original kidney disease
2. Patients of CKD with intrinsic renal disease for planning continuation of specific therapy like patients with
    - Primary glomerular diseases
    - Connective tissue disorders with kidney involvement
    - Polycystic kidney disease
    - Obstructive uropathy
3. All patients of CKD stage-3 onward at the time of diagnosis for planning long-term treatment
4. All CKD with complications like
    - Systemic infection
    - Cardiovascular disease
5. CKD with uncontrolled hypertension
6. CKD with uncontrolled diabetes
7. CKD who are expected to initiate following therapies
    - Erythropoietin stimulating agents
    - Vitamin-D
    - Calcimimetic drugs
    - ACE-I and ARB
    - IV Iron therapy
8. CKD patient scheduled for surgery
9. CKD patient planning for pregnancy
10. CKD patient require consultation and preparation for RRT.

Following is the heat map suggesting when a patient of CKD should be managed by primary care physician and when patient should be referred to nephrologist as per stage of eGFR and stage of albuminuria:

Figure - 7

GFR categories (ml/min/ 1.73 m²) Description and range				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–300 mg/g 3–30 mg/mmol	>300 mg/g >30 mg/mmol
	G1	Normal or high	≥90		Monitor	Refer*
	G2	Mildly decreased	60–89		Monitor	Refer*
	G3a	Mildly to moderately decreased	45–59	Monitor	Monitor	Refer
	G3b	Moderately to severely decreased	30–44	Monitor	Monitor	Refer
	G4	Severely decreased	15–29	Refer*	Refer*	Refer
	G5	Kidney failure	<15	Refer	Refer	Refer

F. Counselling and preparation for RRT for ESKD

A large number of patients of CKD, particularly stage-4 and 5 ultimately require RRT. From early stage of CKD, this issue should be kept in mind. Though primary care physician should be aware of pros and cons of different modalities of RRT, it is domain of nephrologists to finally discuss and decide specific type of RRT suitable for a particular patient in consultation with the patient.

Role of primary care physician in this aspect is as follows:

- Patients should be subjected to initial counseling for RRT once they are in stage-4 CKD.
- Update the list of the CKD patients in the need of RRT.
- Mapping of the District hospital registered for providing the RRT under the PMNDP.
- Develop referral linkages with the nephrologists and RRT under the PMNDP.
- Vaccination of CKD patients for Hepatitis B vaccine and other vaccinations as soon as diagnosis of CKD is made.
- CKD patients should be explained to prevent blood borne infection by avoiding high-risk behavior.
- Patients should be advised to preserve both fore-arm veins for arterio-venous fistula for possible future hemodialysis.

Role of Community Health Officer (CHO) related to CKD factors

- Population based screening for common NCDs including Hypertension and Diabetes mellitus.
- Assessment of patients diagnosed with hypertension and/or diabetes for risk of developing CKD, followed by appropriate referral to higher facilities. CHO can use the same checklist as suggested in the CKD guidelines for inclusion in CBAC form.
- Ensuring routine urine examination and serum creatinine of patients diagnosed with hypertension and diabetes and presenting with other risk factors.
- Counselling regarding lifestyle modification, including diet related advice, weight control, tobacco consumption.
- Ensuring treatment adherence of patients with hypertension and diabetes.

- Ensuring continuation of treatment of patients diagnosed with CKD and monitoring for complications.
- Follow up visits by primary healthcare team led by CHO to CKD patients, wherever required.
- Ensuring continuation of dialysis treatment (hemodialysis or peritoneal dialysis) for those identified with the requirement for same. CHO may facilitate peritoneal dialysis treatment at home through home visits.

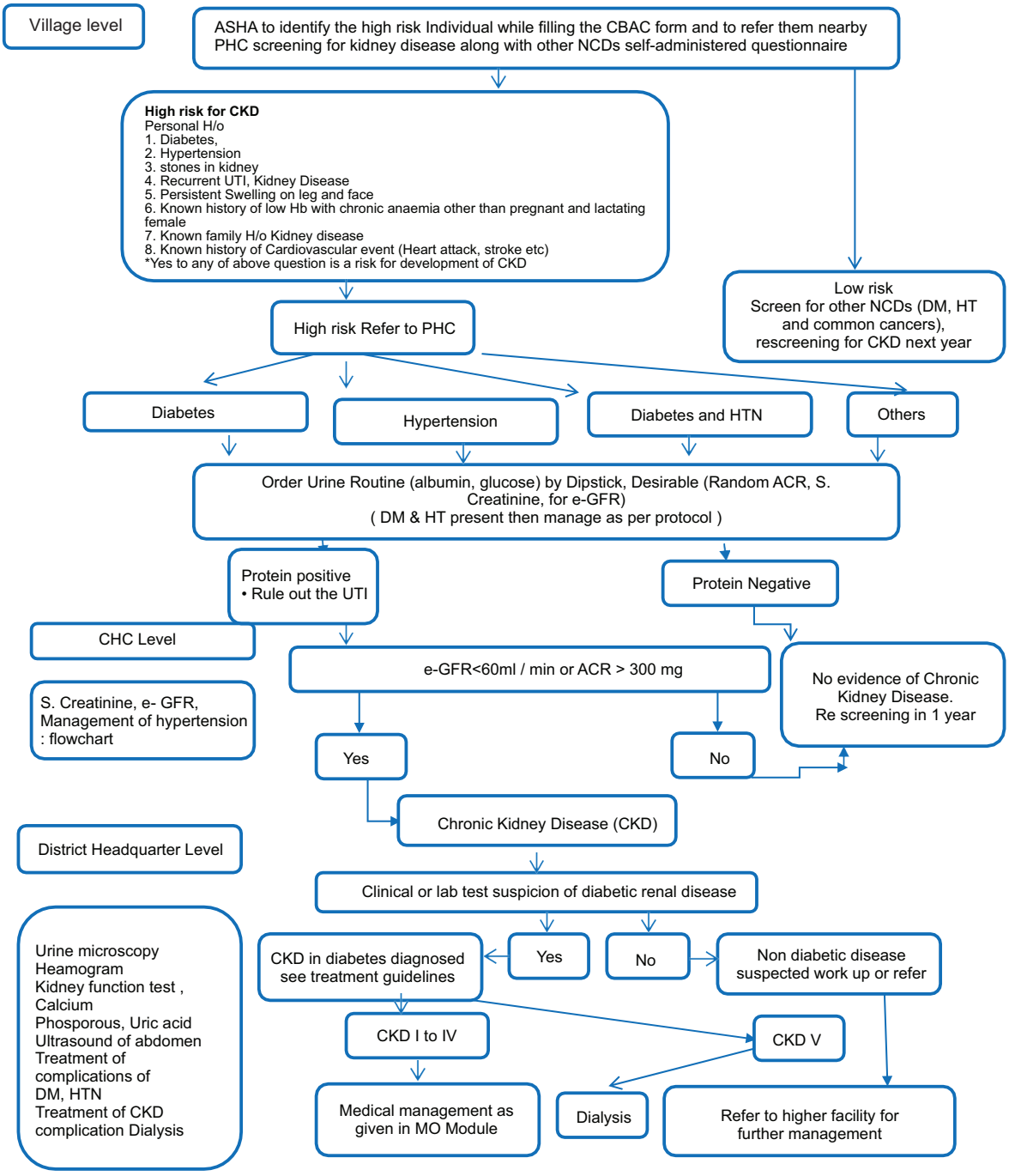
Role of ASHA/ANM at Community Level and sub centre / HWC level: Screening

While filling the CBAC (Community Based assessment Checklist) form, ASHA collects information on few common risk factors of NCD that exaggerate the risk for CKD as well, i.e., obesity (by measuring waist circumference), age >60 years. Few other risk factors for CKD may also be asked as a part of CKD screening. Personal H/o.

- Diabetes
- Hypertension
- Known of kidney stones
- Recurrent UTIs
- Swelling on legs and face
- Known of low Hb with chronic anemia other than pregnant and lactating female.
- Known family Kidney disease.
- Known Cardiovascular event
- Known kidney disease
- Past History of Acute Kidney Disease

All individuals with any risk factor for CKD may be referred to PHC for routine urine examination and serum creatinine and screening for other NCDs (DM, HT and three common cancers as mentioned in MO module).

Figure 8: Algorithm for management of CKD



Awareness generation

Home Visits, Village Health Nutrition Day (VHND), and meetings of Village Health Sanitation & Nutrition Committee (VHSNC), Mahila Mandal, and other platform should be used for discussion about AKI (Acute Kidney Injuries) and CKD and their risk factor along with other NCDs.

Undertaking follows up

- Follow up of referred patient by AMN/MO PHC.
- Follow up home visits for treatment adherence, enabling lifestyle changes and referring in case of any complications to MO (PHC).
- Patient support group (Figure 8)

Annexure: Phosphorus Handling through Diet in CKD patients

Phosphate Restricted Diet

Phosphorus (PO4) restriction is a very important nutritional treatment of mineral and bone disorders in chronic kidney disease (CKD-MBD). In CKD patients, PO4 retention occurs as a result of net intestinal absorption exceeding renal excretion. Therefore, PO4 should be controlled in diet, starting from the early CKD stages. PO4 is taken in diet in two ways; as a natural component and as a food additive.

As a natural food component, phosphorus is available as inorganic phosphate. On average, about 60% of dietary PO4 of animal origin is absorbed in the intestine, while it is less than 40% of PO4 of plant-origin. However, it is 100% if phosphate salts added as food preservatives.

There are four ways of PO4 restriction in diet

1. **Dietary protein restriction**

   PO4 goes with the protein. The restriction of protein intake is generally associated with a lower PO4 intake. on average, a mixed diet contains 12-14 mg of PO4 per gram of protein. Use of protein-restricted diets facilitates the reduction of dietary PO4 intake.

2. **Using foods with low PO4 content and/or low phosphorus bio-availability**

   PO4 content (mg/100 g edible part) in the various food varies. Knowing the PO4 content as mg/g of protein is useful for identifying foods which supply less phosphorus with the same amount of protein.

- High content: PO4 of more than 12 mg/g of protein is classified a “unfavourable” phosphorus to protein ratio.
- In general, intestinal absorption is lower for PO4 of plant origin than for PO4 of animal origin, such as from meat, fish, poultry and dairy products.

3. **Boiling foods**

   Boiling causes demineralization of food, thus reducing PO4 contents. The degree of mineral loss is proportional to the amount of boiling water used, the size of the pieces, the cooking time and the absence of the peel for plants. It is noteworthy that boiling reduces the PO4 content with a negligible loss of nitrogen, leading to a more favourable PO4 to protein ratio.

4. **Identifying and avoiding diet containing phosphate additives**

   PO4 is the main component of several additives (phosphoric acid, phosphates and polyphosphates) used in food processing to extend conservation, enhance colour or flavour, and retain moisture.

   There are various types of food items as per PO4 contribution in diet, which should be advised while taking other dietary precaution into consideration.

Tables having common food item with PO4 contents

PO4 content	Common food item
Highest	**Items with PO4 additives** Soft drinks Dehydrated milk Processed cheese and meat Dessert Instant Cappuccino
High	Hard cheese Nuts Yolk
Moderate	Meat Poultry Fish Soft cheese Milk Yogurt
Low	Cereals Legumes

PO4 content	Common food item
Very Low	Egg white Fruits and vegetables Vegetable fats Butter Sugar Protein free products

Annexure: potassium Handling through Diet in CKD patients

Potassium is an important component of diet and both low and high potassium can be risk factor for morbidity and mortality. Following is a guide to help physician to watch the diet for controlling potassium. The general guideline is to limit intake of high potassium foods, and to choose acceptable potassium foods. Serving sizes of food are important. If patient eat more than 1 serving size of foods that are low or moderate in potassium, patient can end up consuming a total higher amount of potassium. Actual values may vary depending on the product or processing. Patient can also refer to food labels for actual values.

High Potassium Foods (More than 200 mg/serving): Limit/Avoid

FRUITS	Serving Size	Potassium (mg)
Bananas, raw	1 medium	425
Cantaploupe, raw	½ cup	260
Figs, dried	2 each	240
Kiwi, raw	1 medium	325
Mango, raw	1 each	275
Nectarine, raw	1 each	240
Oranges, raw	1 each	235
Orange Juice	½ cup	400
Pomegranate, raw	1 whole	270
Raisins	¼ cup	
VEGETABLES		
Artichoke, cooked	1 medium	345
Avocado, raw	¼ each	245
Bok Choy, cooked	½ cup	316
Broccoli, cooked	½ cup	230
Greens, Beet, cooked	½ cup	655
Pumpkin, canned	½ cup	250
Spinach, cooked	½ cup	420
Sweet Potatoes, baked with skin	1 medium	450
Tomatoes, raw	1 medium	290
Tomato Juice	½ cup	275
White Potatoes, baked with skin	1 medium	925
White Potatoes, boiled	½ cup	255
White Potatoes, mashed	½ cup	330
Winter Squash, cooked	½ cup	250
Zucchini, cooked	½ cup	220
OTHERS		
Chocolate	1½ ounce bar	165
Coconut Milk	1 cup	497
Coconut Water, ready-to-drink	1 cup	404
French Fries, fast food	3 oz/small	470
Milk, chocolate	1 cup	420

FRUITS	Serving Size	Potassium (mg)
Milk, fat free, low fat, whole	1 cup	350-380
Nuts: Almonds, peanuts, hazelnuts, Brazil, cashew, mixed	1 oz	200
Nuts: pistachios	1 oz	295
Potato Chips	1 oz	465
Raisin Bran, dry	1 cup	385
Salt Substitute (i.e. MORTON(R) Salt Substitute)	¼ teaspoon	610
Seeds, sunflower or pumpkin	1 oz	240
Soy Milk	1 cup	300

Low Potassium Foods (less than 200 mg/serving): Acceptable

FRUITS		
Apples, raw/cooked	1 each	150
Applesauce	½ cup	90
Apple Juice	½ cup	150
Blackberries, raw or canned	½ cup	115
Blueberries, raw or canned	½ cup	60
Canned Fruit Cocktail	½ cup	97
Cherries, raw or Canned	10 each	150
Cranberry Juice Cocktail	½ cup	20
Grapes, raw	½ cup	155
Grape Juice	½ cup	170
Lemons and limes, raw	1 each	80
Mandarin Oranges, Canned	½ cup	99
Peaches, canned	½ cup	120
Peach nectar	½ cup	50
Peaches, raw	1 each	185
Pears, canned	½ cup	120
Pear nectar	½ cup	35
Pear, raw/cooked	1 each	193
Pineapple Juice	½ cup	165
Plums, raw	1 each	105
Raspberries, raw	½ cup	90
Strawberries, raw or canned	½ cup	125
Tangerines, raw	1 each	140
Watermelon, raw	½ cup	85
VEGETABLE (Serving size is ½ cup cooked or 1 cup raw)		
Asparagus, small spears, cooked	½ cup or 6 spears	155
Cabbage, cooked	½ cup	150
Carrots, cooked or raw	½ cup	180
Cauliflower, raw	½ cup	150
Celery, raw	½ cup	155
Corn, cooked	½ cup or 1 ear	195
Cucumbers, cooked or raw	½ cup	80
Eggplant, cooked	½ cup	60
Green Beans, cooked	½ cup	90
Lettuce, all types, raw	1 cup	100
Okra, cooked	½ cup	110

Onions, raw	½ cup	120
Peas, green, forzen	½ cup	90
Peppers, green/red	½ cup	130-160
Radishes, raw	½ cup	135
Turnips, cooked	½ cup	140

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