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# An Observational Study on Clinical Profile and Drug Prescription Pattern in CKD Patients

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#### **ABSTRACT**

Chronic kidney disease (CKD) is characterized by progressive condition characterized by gradual loss of kidney function over a period of months to years, depending on the underlying an etiology. Among patients with CKD, we aimed to determine the characteristics of etiology, drug use in patients for a period of 6 months. The aim of the study is to evaluate drug prescription patterns in CKD patients. The main objective is to evaluate cause of CKD and to determine commonly prescribed drugs. This is an observational study done after approval from the IEC. A total of 150 patients were included in the study. Out of 150 patients 100 were males and 50 were females. Out of 150 patients which clearly indicates that occurrence of CKD in male gender is high. The age group of 61-70 were more commonly affected by CKD. Hypertension, diabetic nephropathy, glomerular nephritis, anemia were the most common risk factors. ESA's such as erythropoietin (70%) and Darbepoetin (13.33%) were given to treat anemia. Phosphate binders such, sevelamer carbonate (47.05%) were given to treat hyperphosphatemia, statins were given to treat cardiovascular diseases and anticonvulsants such as Pregabalin (85.71%) and gabapentin (14.28%) was prescribed for treating neuropathic pain. Xanthine oxidase inhibitors such as Febuxostat (96.15%) and Allopurinol (3.84%) were given to treat hyperuricemia. This study demonstrates that antihypertensives was the most frequently prescribed for treating hypertension and calcium channel blockers were most commonly prescribed in all the stages of CKD.

Keywords: Chronic kidney disease, erythropoietin stimulating agents, calcium channel blockers, glomerulonephritis

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## **INTRODUCTION**

Kidney damage lasting more than three months is characterized by either functional or structural defects of the kidney, which may or may not be accompanied by a reduced glomerular filtration rate (GFR). Anomalies related to pathology: or Signs of kidney injury, such as anomalies in the makeup of the blood or urine or variations in imaging studies [1]. GFR<60ml/min/1.73m2 for at least three months, whether renal impairment is present or not. [2]. The GFR is between 120 and 130ml/min. When GFR is less than 60ml/min, at least 50% of normal kidney function is lost, and the likelihood of developing problems from CKD is higher [3]. In the US, the most prevalent cause of kidney failure is diabetic renal disease. It represented about 45 percent of all new patients of ESRD starting renal replacement therapy between 1996 and 2006. A global public health concern, chronic kidney disease (CKD) affects individuals of nearly all ages, nationalities, and socioeconomic backgrounds [4,5]. According to estimates, India's rental units treat 80 to 100 new patients with end-stage renal illness per million inhabitants per year. When defined as a creatinine concentration in serum is more than 1.2 - 1.5 mg/dL, 3 nationwide surveys have indicated that 5% of adult community members in the United States suffer with CKD [6].12 million people globally lost their lives to CKD in 2017. kidney disease caused 1.2 million deaths worldwide, ranking it as the 12th leading cause of death. Furthermore, 7.6% of all CVD fatalities (1.4 million) were attributable to impaired renal function. A total of 4.6 percent of deaths were attributable to CVDs or CKD-related deaths<sup>9</sup>, Diabetes, hypertension, other kidney illnesses such as glomerulonephritis, and polycystic kidney disease are the

main four causes of stage 5 chronic kidney disease in the United States. Some of the causes and rates are polycystic kidney disease (5 cases/million), glomerulonephritis (22 cases/million), diabetic nephropathy (150 cases/million), and hypertensive nephropathy (80 cases/million) [7].

Its first sign is microalbuminuria with a regular or doubled GFR. Renal headaches associated with diabetes are less likely when blood pressure and blood sugar levels are properly controlled. It has been conclusively shown that close blood glucose monitoring reduces the development of microalbuminuria by 35% in both type 1 diabetes (Diabetes Control and Complications Trial) and type 2 diabetes (United Kingdom Prospective Diabetes Study) [8]. Anemia associated with chronic renal disease begins to develop when the glomerular filtration rate is between 30 and 35 percent of normal and is both normochromic and normocytic. This is typically caused by the failing kidney producing less erythropoietin; additional causes of anemia include erythropoiesis inhibitors, shortened red blood cell life spans, platelet dysfunction, reduced iron consumption, and secondary hyperparathyroidism [9]. Among the first signs of reduced renal function is hyperparathyroidism, which is present in patients with a glomerular filtration rate of 60 ml/min. The primary uremic toxin responsible for bone disease, hyperlipidaemia, encephalopathy, pruritus, peripheral neuropathy, anemia, and sexual dysfunction is parathormone [10]. Under the medical term glomerular nephritis, or glomerulonephritis (GN), the disease is defined by inflammation of the glomeruli, which are microscopic kidney filters that eliminate waste and surplus fluid from the bloodstream to produce urine. The regular filtration mechanism may be interfered with by this inflammation, resulting in several symptoms and problems. An important part of glomerulonephritis' pathophysiology is the immune system. The glomeruli are frequently the unintentional target of immune system mistargeting, which results in immunological-mediated inflammation. Proteinuria, or elevated protein levels in the urine, is a defining feature that indicates damage to kidneys. Although they do occur in stages 3 and 4, anomalies in the metabolism of calcium and phosphorus are more common in Stage5 CKD. When the GFR is 80 mL/min per 1.73 m2 or less, secondary hyperparathyroidism can occur despite normal serum calcium and phosphorus levels.

Abnormal aldosterone synthesis: The hormone that the adrenal glands produce, aldosterone, is essential for controlling the equilibrium of sodium and potassium. Abnormalities in aldosterone synthesis in chronic kidney disease (CKD) can cause the kidneys to retain excessive amounts of water and salt, which can exacerbate volume expansion and hypertension. Over time, this may cause more renal damage [11]. Elevated tubular salt and water reabsorption: The renal tubules, particularly the collecting ducts and distal convoluted tubule (DCT), are where the RAAS promotes their absorption of water and sodium. Dysregulation of the RAAS in chronic kidney disease (CKD) can result in increased reabsorption of water and salt, which can exacerbate the expansion of volume and hypertension. Promotion of ADH hormone production: Water reabsorption is facilitated by the action of antidiuretic hormone (ADH) or vasopressin on the kidney's collecting ducts, which is produced in response to RAAS stimulation. This can worsen volume expansion and hypertension in CKD patients, as well as cause water retention [11].

Increased fluid and water retention: Significant fluid and retained water in chronic kidney disease (CKD) can be caused by the interaction of aberrant aldosterone synthesis, enhanced tubular reabsorption, and a higher level of ADH. Increased blood pressure, oedema (swelling), and volume overload can result from this, all of which can worsen kidney injury [11]. In CKD stages 4 and 5, abnormalities in sodium and potassium levels often occur due to the impaired ability of the kidneys to regulate these electrolytes. This can lead to hyperkalaemia (high potassium levels) and hyponatremia (low sodium levels), which can be serious if not properly managed. The glomeruli suffer damage and lose their capacity to operate normally. Numerous symptoms may result from this, such as haematuria (blood in the urine), proteinuria (extra protein in the urine), and oedema (swelling), especially in the limbs [12].

The management of chronic kidney disease (CKD) aims to slow its progression, control symptoms, and reduce the risk of complications. Here's a more in-depth look at the management strategies Controlling hypertension is crucial in managing CKD [13]. Target blood pressure levels are generally <130/80 mmHg, but they may vary based on individual patient factors. A renal dietitian can help create a meal plan that considers the patient's kidney function, nutritional needs, and other health conditions. Dietary modifications often include limiting protein, sodium, potassium, and phosphorus. For patients with chronic kidney disease (CKD), choosing the right medications is crucial to preventing side effects and achieving the best possible results. Because CKD patients require complicated therapy regimens that require regular monitoring and dose modifications, rational medication prescription is challenging for these patients owing to a greater risk of drug-related issues. The issue becomes more complex when multiple medical conditions including diabetes mellitus, hypertension, coronary artery disease, and infections are present. Adverse drug effects may be exacerbated by inappropriate pharmaceutical usage, which can lead to longer hospital admissions, higher health care consumption, and higher expenses.

Chronic kidney disease is a progressive condition that effects >10% of general population worldwide amounting to >800 million individuals. It has emerged as one of the leading cause of mortality worldwide. There are many conditions like Hypertension, obesity, CVD, which contribute to CKD an its progression. Hence an attempt to focus on prescription pattern of CKD and effectiveness of treatment and to slowdown the progression of disease and also to ascertain the cause of CKD.

The aim of the study is to evaluate drug prescription pattern in CKD patients. The objectives include to obtain demographic information of the patient, to assess the risk factors of various stages of CKD, to determine commonly prescribed drugs and to evaluate causes of CKD.

#### **MATERIAL AND METHODS**

#### **Study Protocol**

It is an observational study being conducted for a period of 6 months. Patients who meet the study criteria will be included in the study. The required data will be collected through the patient's interview and their prescriptions. The data obtained shall be analysed to know the prescription pattern in CKD patients.

**Study design:** It is an observational study.

**Study site:** The study is to be conducted at the Nephrology Department, Yashoda Hospital, Secunderabad.

**Study period:** The study is conducted for 6 months from October, 2023 to March, 2024.

**Study population:** About 150 patients will be included in the study.

# Study criteria Inclusion criteria

- Patients of age groups (18-70) years.
- Patient diagnosed with CKD.
- Both Inpatient and Outpatient were included.

#### **Exclusion criteria**

- Pregnancy and lactating women.
- Paediatric group of patients.
- Patients with Acute kidney disease.

# Statistical analysis:

- Software used: SPSS version 24
- P value less than 0.05 is considered significant since the CI is 95%. Test performed: Chi-square.

### **RESULTS AND DISCUSSIONS**

## Demographic data

150 patients with various stages of chronic renal disease were involved in our study. Based on the data, out of the 150 patients, 50 (33%) female and 100 (67%) were male. This demonstrates unequivocally that men are more likely than women to develop chronic kidney disease (CKD)as shown in **table1**. The reason behind this could be that men are more prone to poor lifestyle choices, such as smoking, drinking too much alcohol, and eating a diet heavy in processed foods and sodium, all of which can harm the kidneys. Renal function may decline in males more quickly than in women due to men's higher testosterone levels, unhealthy lifestyles, and the protective effects of oestrogen. These outcomes agree with those [14]. The age range of 61 to 70 years old (46%) comprises the vast majority of study participants as shown in **table 2**. A deterioration in advanced age-related cognitive function and homeostatic reserve was associated with a decrease in eGFR in addition to a decline in physical performance and activity. This may be the direct outcome of CKD or the consequence of comorbidity clustering with aging, according to [15].

Table 1: Demographic data Age distribution

| GENDER | NO. OF PATIENTS | PERCENTAGE |
|--------|-----------------|------------|
| MALE   | 100             | 67%        |
| FEMALE | 50              | 33%        |

Table 2: Age distribution

| AGE   | No. of patients | Percentage | male | female |
|-------|-----------------|------------|------|--------|
| 21-30 | 5               | 3.39%      | 3    | 2      |
| 31-40 | 5               | 3.39%      | 5    | 0      |
| 41-50 | 22              | 14.60%     | 13   | 9      |
| 51-60 | 48              | 32.00%     | 33   | 15     |
| 61-70 | 69              | 46.00%     | 45   | 24     |
| 71-80 | 1               | 0.610%     | 1    | 0      |

#### Stages of CKD and associated with comorbidities

In our study the distribution of CKD stages was as all follows CKD stage 1 accounted for 4%, CKD stage 2 accounted for 3.30%, CKD stage 3 was found to be 41.30%, CKD stage 4 accounted for 24% and CKD stage 5 was found to be 27.30% respectively. Majority of the patients were diagnosed with CKD stage 3 as shown in **table 3**. Greater correlations were discovered between the management of comorbidity, functional and cognitive impairment, and the possibly reversible effects of kidney loss, including, hyperkalaemia, uraemia, anemia, phosphate retention, low serum albumin, secondary hypertension, and anaemias. Majority of the patients with CKD stage 3 are having the comorbidity hypertension followed by diabetes as shown in table **4**.

Table 3: Stages of CKD

| DIAGNOSIS | NO. OF PATIENTS | PERCENTAGE |
|-----------|-----------------|------------|
| CKD1      | 6               | 4.00%      |
| CKD2      | 5               | 3.30%      |
| CKD3      | 62              | 41.3%      |
| CKD4      | 36              | 24.0%      |
| CKD5      | 41              | 27.3%      |

Table: 4 Distribution of Patients based on Comorbidities

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|---|------|-------|-------|-------|-------|--|
| Distribution of Patients based on Comorbidities(N)                          |      |       |       |       |       |  |
| MEDICALHISTORY  | CKD1 | CKD 2 | CKD 3 | CKD 4 | CKD 5 |  |
| Hypertension  | 6    | 5     | 55    | 34    | 39    |  |
| Diabetes  | 6    | 2     | 41    | 20    | 28    |  |
| Hypothyroidism  | 0    | 1     | 4     | 2     | 7     |  |
| CAD   | 1    | 0     | 17    | 2     | 7     |  |
| Diabetic Nephropathy  | 4    | 1     | 21    | 11    | 4     |  |
| Diabetic Retinopathy  | 2    | 0     | 1     | 1     | 0     |  |
| CVA   | 1    | 0     | 4     | 1     | 1     |  |
| Other kidney disorders (uropathy, kidney polycysts, urecemia, nephritisetc) | 1    | 2     | 20    | 20    | 8     |  |

# **DISTRIBUTION BASED ON CAUSES**

Table 5 shows distribution of patients based on causes of CKD stage1 where Diabetic nephropathy i s the major leading because which constitutes 50.0% followed by diabetic neuropathy (16.63%), CVA (16.63%), polycystic kidney disease (16.63%) respectively. Causes of stage2 CKD in study population where Diabetic nephropathy accounts for 40.00% followed by chronic tubule interstitial disease 20.00%, hyperuricemia 20.00% and hypertension 20.00% respectively. Causes of stage3CKD in study population where Hypertension 36.9%, CAD 19.5%, diabetes 13.0%, renalcalculus 10.8%, CVA 8.60%, Smoking 6.50%, and CTID 4.34%. Causes of CKD stage4 in study population. Hypertension is the main cause accounting for 30.5%, diabetic nephropathy 25.7%, coronary artery disease 13.8%, renal calculus 8.30%, smoking 5.50%, CTID5. 50%, CVA2. 70% respectively. Cause of stage5 CKD in study population. Hypertension is the main cause found in our study which accounts for 29.2%, diabetes 24.0%, CAD14.0%, diabetic nephropathy 12%, smoking 7.39%, CVA 7.30% and renal calculus 4.87% respectively. In stage1 CKD, the most common causes are hypertension, diabetic nephropathy, and hyperuricemia. Sodium retention can indeed play a significant role in hypertension, both through volume-dependent and volumeindependent mechanisms. Volume dependent mechanisms involve an increase in extracellular fluid volume, which leads to increased peripheral tissue perfusion and can induce vasoconstriction raising blood pressure [16]. Diabetic patients found to have albuminuria considering that early levels or ranges of organ damage, it's also taken into consideration as a totally realistic marker of kidney progression Table 5 [17]. Autosomal polycystic kidney disease caused by mutation in PKD1 [18]. Hyperuricemia can be caused by diet high in purine-rich foods and fructose, as well as lead exposure, can increase uric acid levels, with fructose uniquely depleting ATP and promoting uric acid production [19]. Anaemia increases the risk of CVD in patients with chronic kidney disease (CKD), which is characterized by improper metabolism of calcium and phosphate [20]. Smoking appears to be a substantial contributor to kidney injury since it causes an occasional rise in blood pressure [21]. As renal damage increases, glomerulonephritis becomes salt sensitive. The resulting hypoxia and reduction in interstitial blood flow stimulate the intra renal RAAS, which in turn leads to the development of salt sensitive hypertension [22]. Kidney impairment and cerebral small vessel diseases share similarities in their vascular susceptibilities, making CKD predictive of the presence and severity of these brain conditions, highlighting the importance of managing CKD to potentially reduce the risk of cerebrovascular complications [23]. Urine calcium excretion decreases as a result of the GFR decline linked to chronic kidney disease (CKD), potentially reducing the risk of kidney stone development [24].

| Table 5: Distribution of patients based on causes of CKD stage |       |            |  |  |
|--|-------|------------|--|--|
| CKD stage1   |       |            |  |  |
| CAUSES   | N(6)  | PERCENTAGE |  |  |
| Diabetic Nephropathy   | 3     | 50%        |  |  |
| Diabetic Neuropathy  | 1     | 16.60%     |  |  |
| CVA  | 1     | 16.60%     |  |  |
| Polycystic kidney disease                                      | 1     | 16.60%     |  |  |
| CKD stage 2  |       |            |  |  |
| CAUSES   | N(5)  | PERCENTAGE |  |  |
| Diabetic Nephropathy   | 2     | 40.00%     |  |  |
| Chronic tubule interstitial disease                            | 1     | 20.00%     |  |  |
| Hyperuricemia  | 1     | 20.00%     |  |  |
| Hypertension   | 1     | 20.00%     |  |  |
| CKD stage 3  |       |            |  |  |
| CAUSES   | N(46) | PERCENTAGE |  |  |
| Diabetes   | 6     | 13.0%      |  |  |
| CAD  | 9     | 19.5%      |  |  |
| CVA  | 4     | 8.60%      |  |  |
| Hypertension   | 17    | 36.9%      |  |  |
| Smoking  | 3     | 6.50%      |  |  |
| Renal calculus   | 5     | 10.8%      |  |  |
| CTID   | 2     | 4.34%      |  |  |
| CKD stage 4  |       |            |  |  |
| CAUSES   | N(36) | PERCENTAGE |  |  |
| Diabetes   | 3     | 8.00%      |  |  |
| CAD  | 5     | 13.8%      |  |  |
| CVA  | 1     | 2.70%      |  |  |
| Hypertension   | 11    | 30.5%      |  |  |
| Smoking  | 2     | 5.50%      |  |  |
| Renal calculus   | 3     | 8.30%      |  |  |
| CTID   | 2     | 5.50%      |  |  |
| DIABETICNEPHROPATHY  | 9     | 25.7%      |  |  |
| CKD stage 5  |       |            |  |  |
| CAUSES   | N(41) | PERCENTAGE |  |  |
| Diabetes   | 10    | 24.0%      |  |  |
| CAD  | 6     | 14.0%      |  |  |
| CVA  | 3     | 7.30%      |  |  |
| Hypertension   | 12    | 29.2%      |  |  |
| Smoking  | 3     | 7.39%      |  |  |
| Renal calculus   | 2     | 4.87%      |  |  |
| DIABETIC NEPHROPATHY   | 5     | 12%        |  |  |

## Distribution based on treatment

Patients with chronic kidney disease (CKD) need polypharmacy since they are more likely to have numerous comorbidities. Most of these patients were prescribed more than 5 medications per prescription. These findings aligned with the research carried out by [25]. The most often prescribed medications in this study were antihypertensive, which were followed by vitamins and minerals,

erythropoietin stimulating agents, and proton pump inhibitors. found that the most commonly prescribed medications were cardiovascular drugs (16.48%), gastrointestinal tract drugs (14.07%), and nutritional supplements (10.88%). Since most patients have many comorbid conditions related to hypertension, cardiovascular medications are mostly needed for their management. Thiazides are helpful in patients with severe CKD, diuretics like torsemide (15%) and MRAs like finer none (7.50%) and beta blockers like carvedilol (11.5%) were recommended more frequently to ESRD patients as shown in **Table 6 and 7**. In observational studies, they result in an adverse sodium balance, raising sodium excretion by 10%–15% and causing weight loss of 1-2 kg.

Table 6: Anti-hypertensive classes prescribed in different stages of CKD

|                   | Antihypertensives prescribed in different stages of CKD |      |      |      |      |
|-------------------|---|------|------|------|------|
| ANTIHYPERTENSIVES | CKD1  | CKD2 | CKD3 | CKD4 | CKD5 |
| Thiazides         | 1   | 0    | 3    | 2    | 1    |
| Loop diuretics    | 1   | 2    | 14   | 21   | 21   |
| ACEi              | 0   | 0    | 1    | 1    | 0    |
| Beta blockers     | 2   | 1    | 12   | 16   | 19   |
| CCB               | 2   | 3    | 23   | 20   | 24   |
| ARB               | 5   | 2    | 9    | 3    | 4    |
| alpha blockers    | 2   | 1    | 21   | 5    | 12   |
| MRAS              | 4   | 0    | 19   | 4    | 0    |
| CCB+ARB           | 0   | 0    | 2    | 2    | 4    |

Table 7: Anti-hypertensive drugs prescribed in different stages of CKD

| Antihypertensive Drugs  | N  | %      |
|-------------------------|----|--------|
| Metolazone              | 7  | 2%     |
| Furosemide              | 8  | 2%     |
| Torsemide               | 51 | 15%    |
| Ramipril                | 3  | 0.90%  |
| Lisinopril              | 1  | 0.30%  |
| Spironolactone          | 3  | 0.90%  |
| Finerenone              | 24 | 7.50%  |
| Bisoprolol              | 1  | 0.30%  |
| Carvedilol              | 37 | 11.50% |
| Metoprolol              | 10 | 3.10%  |
| Nebivolol               | 3  | 0.90%  |
| Propranolol             | 1  | 0.30%  |
| Amlodipine              | 33 | 10.30% |
| Benidipine              | 1  | 0.30%  |
| Cilnidipine             | 17 | 5.30%  |
| Nifedipine              | 27 | 8.40%  |
| amlodipine+atenolol     | 1  | 0.30%  |
| amlodipine+telmisartan  | 1  | 0.30%  |
| clinidipine+telmisartan | 6  | 1.80%  |
| telmisartan+ amlodipine | 2  | 0.60%  |
| Losartan                | 1  | 0.30%  |
| Olmesartan              | 1  | 0.30%  |
| Telmisartan             | 16 | 5%     |
| Metoprolol succinate    | 5  | 1.50%  |
| Tamsulosin              | 13 | 4%     |
| Prazosin                | 18 | 5.60%  |
| Sildosin                | 11 | 3.40%  |
| Clonidine               | 7  | 2.10%  |
| Moxonidine              | 3  | 0.90%  |
| Minoxidil               | 1  | 0.30%  |
| Hydralazine             | 3  | 0.90%  |
| Isolazine               | 1  | 0.30%  |
| Amlodipine and atenolol | 2  | 0.60%  |

As shown in **table 8**, when compared to darbepoetin (13.33%), erythropoietin (70%) and other erythropoietin-stimulating medications were given more frequently. When compared to other trials, there was a high rate of ESA use. Because erythropoietin is not created enough in CKD patients, which results in anemia, ESAs and iron supplements are given to these patients. In addition to lowering cardiovascular morbidity and mortality, treating anemia in people with chronic kidney disease (CKD) enhances

functioning, cognitive function, and quality of life [26]. Proton pump inhibitors, like omeprazole (7.69%), pantoprazole (77%), and esome prazole (76%) were prescribed to treat peptic ulcer disease, which is more common in people with chronic kidney disease (CKD), and to reduce gastric acid output. If PPI use is linked to poor renal outcomes or patient's survival in CKD patients [27]. In the long-term care of patients with chronic kidney disease (CKD), pre-clinical studies on HMG co-a reductase inhibitor such as atorvastatin (84.61%) and rosuvastatin (15.38%) suggest a protective effect of statins on the reduction of proteinuria mostly via accelerating renal fibrosis [28]. While phosphate binders were used and detrimental effect less frequently in the trial by [13]. Sevelamer carbonate was used in our study (47.05%). Selvamer carbonate is an efficient and well-tolerated treatment for controlling phosphorus levels in hyperphosphatemic patients with chronic kidney disease. [29]. Anticonvulsants such as pregabalin (85.71%) and gabapentin (14.28%) which is prescribed to treat neuropathic pain which is caused by diabetic neuropathy, secondary hyperparathyroidism which leads to abnormalities in calcium, phosphorus and parathyroid hormone which can contribute to nerve damage and neuropathic pain. research has proven the anticonvulsant gabapentin to be powerful in painful diabetic neuropathy, combined neuropathies [30].

Table 8: Distribution of different class of drugs for treatment of CKD

| . Distribution of uniterent class of arags  | 101 | ti cutiliciit |
|---|-----|---------------|
| ESAs  | N   | %             |
| Erythropoietin                              | 21  | 70%           |
| darbepoietinalfa                            | 5   | 16.66%        |
| Darbepoetin                                 | 4   | 13.33%        |
| Iron replacement drugs                      |     |               |
| Elemental iron                              | 1   | 7.142%        |
| Ferriccarboxy maltose                       | 3   | 21.4%         |
| Ferriccarboxy maltose                       | 1   | 7.142%        |
| Ferrous ascorbate, folic acid zinc sulphate | 1   | 7.142%        |
| Iron sucrose                                | 5   | 35.71%        |
| Isomaltoside                                | 3   | 21.4%         |
| Folic acid                                  | 9   |               |
| PPI   |     |               |
| Esomeprazole                                | 1   | 7.69%         |
| Omeprazole                                  | 1   | 7.69%         |
| Pantoprazole                                | 10  | 77%           |
| Rabeprazole                                 | 1   | 7.69%         |
| calcium supplements                         |     |               |
| calcium acetate                             | 2   | 10%           |
| Calcitriol                                  | 11  | 55%           |
| calcium carbonate                           | 2   | 10%           |
| Calcium carbonate and calcitriol            | 2   | 10%           |
| calciumcarbonateandvitd3                    | 1   | 5%            |
| Calcium citrate                             | 1   | 5%            |
| Calcium gluconate                           | 1   | 5%            |
| Statins                                     |     |               |
| Atorvastatin                                | 11  | 84.61%        |
| Rosuvastatin                                | 2   | 15.38%        |
| Antiplatelets                               |     |               |
| Aspirin                                     | 23  | 71.87%        |
| Clopidogrel                                 | 7   | 21.87%        |
| Rosuvastatin+clopidogrel                    | 2   | 6.25%         |
| Anticonvulsants                             |     |               |
| Gabapentin                                  | 1   | 14.28%        |
| Pregabalin                                  | 6   | 85.71%        |
| Nutritional supplements                     | 22  |               |
| phosphate binders                           |     |               |
| Sevelamer                                   | 8   | 47.058%       |
| sevelamer acetate                           | 1   | 5.88%         |
| sevelamer carbonate                         | 8   | 47.058%       |
| xanthineoxidase inhibitors                  |     |               |
| Allopurinol                                 | 1   | 3.84%         |
| Febuxostat                                  | 25  | 96.15%        |
| L   |     | L             |

In patients with established cardiovascular disease (CKD), the antiplatelet medications clopidogrel (21.87%) and aspirin (71.87%) are frequently used for secondary prevention of cardiovascular events. Antiplatelet medication should be used in the early stages of CKD (stages 1-3) after a thorough evaluation of each patient's cardiovascular risk, with the advantages and disadvantages of bleeding carefully considered. Because of the increased risk of bleeding, the decision to utilize antiplatelet medication is more complicated in patients with advanced chronic kidney disease (CKD Stage 4-5), especially those receiving dialysis. Allopurinol (3.84%) and febuxostat (96.15%)were administered. Allopurinol slows the course of renal disease, as demonstrated by RCTs. Additionally, useful in reducing uric acid, Febuxostat that actions that protect against Reno [31].

## **CONCLUSION AND LIMITATIONS**

We found that occurrence of CKD was higher in males (66.60%) than females (33.30%). Age groups of 61-70 are at higher risk of CKD. Stage3CKDwas most seen than other stages of kidney disease. Hypertension, diabetic nephropathy, glomerulonephritis, anemia was most common risk factors seen in our study. Antihypertensivemedicationsweremostcommonlyprescribedinourstudycalcium channel blockers, diuretics and ACE inhibitors were largely given to patients. ESAs, folic acid, vitamin supplements were given to treat nutritional deficiency and anemia. Antiplatelet medications were given to treat cardio vascular diseases which is the common cause of CKD. Because this study was only done for six months, results from longer-term research may differ. As more male patients were seen, no proper information regarding the gender prevalence was obtained.

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#### **Competing Interest:** None

## **Ethical Approval**

The study was approved by the Institutional Review Board of Anurag university bearing the research proposal number: IRB-AU/2023-2024/05.

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