

ORIGINAL ARTICLE

Evaluation of risk factors of chronic kidney disease and major contributing factors of autoimmunity in chronic kidney disease

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ABSTRACT

The progressive decrease of kidney function over time is the hallmark of chronic kidney disease (CKD), a common and incurable medical disorder. The purpose of this study is to evaluate the risk factors linked to the onset and course of CKD and to determine the primary autoimmune contributing factors to the disease. Through a comprehensive literature review, we examined numerous studies and clinical reports to identify the risk factors for CKD. The primary risk factors identified include diabetes mellitus, hypertension, cardiovascular disease, family history, age, obesity, smoking, ethnicity, and chronic urinary tract infections. The role of specific medications in exacerbating CKD risk was also explored. Furthermore, we investigated the major autoimmune factors contributing to CKD. Autoimmune kidney conditions, like systemic lupus erythematosus (SLE), antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis, IgA nephropathy, membranous nephropathy, and Goodpasture syndrome, have been identified as substantial contributors to the emergence of chronic kidney disease associated with autoimmunity. The results from this evaluation emphasize the importance of understanding the diverse etiologies of CKD, ranging from non-immune mediated causes to autoimmune-related pathologies. Early identification of risk factors and autoimmune contributions could aid in implementing targeted preventive strategies and personalized treatment approaches for patients at risk of or already affected by CKD.

Keywords: Chronic Kidney Disease, Acute Kidney Injury and Risk factors.

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INTRODUCTION

The kidneys are retroperitoneal organs essential for maintaining systemic homeostasis. They regulate fluid balance, electrolyte composition, acid-base equilibrium, and blood pressure, while also excreting metabolic waste products such as urea, creatinine, and uric acid [1-4]. Structurally, each kidney contains approximately one million nephrons composed of glomeruli and tubular segments that filter blood, reabsorb essential solutes, and secrete waste products to form urine [5-9]. In addition to excretory functions, the kidneys synthesize key hormones including erythropoietin, renin, and calcitriol, thereby contributing to erythropoiesis, vascular regulation, and bone metabolism [4,10,11]. These functions are tightly regulated through complex interactions among nephrons, hormonal pathways, and neurohumoral mechanisms [12]. Kidney diseases represent a broad spectrum of disorders that impair renal structure and function, including infections, inflammatory conditions, genetic abnormalities, autoimmune diseases, and drug-induced nephrotoxicity [13-15]. Among these, chronic kidney disease (CKD) and Acute Kidney

Injury (AKI) are the most clinically significant entities. CKD is characterized by persistent structural or functional kidney abnormalities lasting more than three months and progressive decline in glomerular filtration rate (GFR) [16,17]. In contrast, AKI refers to a sudden deterioration in renal function due to causes such as sepsis, toxins, or hemodynamic instability [18].

CKD has emerged as a major global public health challenge, affecting approximately 13.4% of the world's population—nearly 750 million individuals—and accounting for more than 1.2 million deaths annually [19,20]. In India, the reported prevalence ranges from 7.2% to 17.2%, with a rising burden attributed to demographic transitions, lifestyle modifications, and increasing prevalence of non-communicable diseases [21–24]. The disease is associated with significant morbidity, including cardiovascular complications, anemia, mineral and bone disorders, and increased mortality risk [17,25]. The principal risk factors for CKD include diabetes mellitus and hypertension, which together account for nearly two-thirds of cases worldwide [19,26,27]. Additional contributors include obesity, smoking, family history, aging, ethnicity, chronic medication use, and environmental toxin exposure [28–31]. Other etiologies such as glomerulonephritis, polycystic kidney disease, obstructive uropathy, and drug-induced nephrotoxicity also play important roles in disease progression [32–35]. Diabetes-related kidney disease affects nearly 30% of diabetic individuals, while approximately 20% of hypertensive patients may develop CKD [36,37]. The pathophysiology of CKD involves persistent inflammation mediated by pro-inflammatory cytokines, activation of fibrogenic pathways, oxidative stress, endothelial dysfunction, and dysregulation of the renin-angiotensin-aldosterone system (RAAS) [38–42]. These mechanisms promote renal fibrosis, structural damage, and progressive nephron loss. Disturbances in calcium-phosphate metabolism further contribute to vascular calcification and renal deterioration [43]. RAAS inhibition using angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) has been shown to slow disease progression, particularly in patients with diabetes and hypertension [44]. Diagnosis of CKD relies on estimated glomerular filtration rate (eGFR) and assessment of albuminuria, as recommended by the kidney disease: Improving Global Outcomes (KDIGO) guidelines [45–47]. Additional investigations, including serum biochemical markers, urinalysis, imaging studies, and occasionally kidney biopsy, aid in determining etiology and staging [48–50]. Management strategies are stage-dependent and include lifestyle modification, strict blood pressure and glycemic control, pharmacotherapy, dialysis for advanced disease, and kidney transplantation as the definitive treatment for end-stage renal disease (ESRD) [51–53]. Despite advancements in diagnosis and treatment, CKD remains underdiagnosed and inadequately managed, particularly in low- and middle-income countries due to limited resources and lack of awareness. Early identification and aggressive management of modifiable risk factors remain the cornerstone of prevention and improved clinical outcomes. Complications like anemia, bone disease, and cardiovascular issues are managed with medication and lifestyle adjustments [54,55].

MATERIAL AND METHODS

This hospital-based retrospective observational study was conducted to evaluate the risk factors of chronic kidney disease (CKD) and to identify major contributing autoimmune factors associated with CKD in a tertiary care setting. The study was carried out at Parul Sevashram Hospital, Parul University, Vadodara, Gujarat, India, over a period of six months from September 2022 to February 2023. Ethical approval for the study was obtained from the Institutional Ethics Committee of Parul Sevashram Hospital (Approval No: PUIECHR/PIMSR/00/081734/5711) prior to data collection. A total of 101 patient records were included in the study. Data were collected from the Medical Record Department using a specially designed and validated patient data collection form, which included demographic details (age, sex), admission and discharge dates, reason for admission, medical history, social history, laboratory findings, and relevant clinical parameters. The inclusion criteria comprised patients diagnosed with CKD, autoimmune kidney disease (AIKD), and acute kidney injury (AKI), while records with incomplete or missing data were excluded from the study. The collected data were systematically tabulated and analyzed using Microsoft Excel. Descriptive statistical methods, including counts and percentages, were used to summarize categorical variables, and graphical representations such as tables, charts, and figures were employed to present the findings clearly and effectively.

RESULTS

A total of 101 patients with renal disease such as CKD and AKI were included in the study.

Disease wise Distribution

In the study of 101 numbers of patients were observed reported according to inclusion and exclusion criteria, majority were suffering from CKD (70.3%) and AKI (29.7) (Figure.1) was found in minor numbers.

Gender wise Distribution (CKD)

A total of 71 patients were enrolled in the study out of them 45(63.38%) were found to be of male gender (Table 1) of the total CKD patients' population and female were found to be 26(36.62%) after analyzing this it was found that majority were belongs to the male gender.

Gender	Number of Patients(n=71)	Percentage (%)
Male	45	63.38
Female	26	36.62

Table.1 Gender wise distribution

Risk Factors wise Distribution (Male)

The result of distribution according to risk factors of male shows most of the patients were suffering from HTN 28(62.22%), 8(17.77%) were found of DM, 5(11.11%) were found of DM & HTN BOTH, 3(6.67%) were found of Obstructive Uropathy and 1(2.22%) was found with Glomerulonephritis (Table 2).

Risk Factors	Number of Patients	Percentage(%)
DM	8	17.77
DM & HTN BOTH	5	11.11
GLOMERULONEPHRITIS	1	2.22
HTN	28	62.22
OBSTRUCTIVE UROPATHY	3	6.67

Table 2: Risk factors wise distribution

Risk Factors wise Distribution (Female)

The result of distribution according to risk factors of female shows most of the patients were suffering from HTN 19(73.08%), 3(11.54%) were found of Polycystic Kidney Disease, 2(7.69%) were found of DM & HTN BOTH, 1(3.84%) were found of Obstructive Uropathy (Table 3).

Risk Factors	Number of Patients	Percentage (%)
DM	1	3.85
DM & HTN	2	7.69
HTN	19	73.08
OBSTRUCTIVE UROPATHY	1	3.84
POLYCYSTIC KIDNEY DISEASE	3	11.54

Table 3: Risk factors wise distribution

Age wise Distribution (Male)

For this study of CKD, total 45 numbers of male patients were observed, According to age wise distribution with inclusion and exclusion criteria age of 77 patients 0-20 year (2.22%), 21-40 year(35.55%), 41-60 year(42.22%) and 60 year above (20%). Majority of the patients were found in the age group of 41- 60 years i.e. 19(42.22%) (Table 4).

Age	Number of Patients	Percentage (%)
0-20 YEARS	1	2.22
21-40 YEARS	16	35.55
41-60 YEARS	19	42.22
60 ABOVE	9	20

Table 4: Age wise distribution (male)

Age wise Distribution (Female)

For this study, total 26 numbers of female patients were observed, According to inclusion and exclusion criteria age of 26 patients 0-20 year (3.85%), 21-40 year(42.31%), 41-60 year(34.62%) and 60 year above (19.23%).Majority of the patients were found in the age group of 21-40 years i.e. 11(42.31%) (Table 5).

Age	Number of Patients	Percentage (%)
0-20 YEARS	1	3.85
21-40 YEARS	11	42.31
41-60 YEARS	9	34.62
60 ABOVE	5	19.23

Table 5: Age wise distribution (female)

Social History wise Distribution (Male)

For this study, total 45 numbers of male patients were observed, out of which 18(40%) Alcoholic, 2(4.44%) Smokers,4(8.89%) smokers & alcoholic both and 21(46.67%) have no addiction (Table. 6).

Social History	Number of Patients	Percentage
ALCOHOL	18	40
NONE	21	46.67
SMOKING	2	4.44
SMOKING & ALCOHOL BOTH	4	8.89

Table 6: Social history wise distribution

Social History wise Distribution (Female)

For this study, total 26 numbers of female patients were observed, out of which 1(3.85%) Alcoholic and 25(96.15%) have no addiction (Table. 7).

Social History	Number of Patients	Percentage
ALCOHOL	1	3.85
NONE	25	96.15
SMOKING	0	0
SMOKING & ALCOHOL BOTH	0	0

Table 7: Social history wise distribution

Gender wise Distribution (AKI)

A total of 30 patients were enrolled in the study, out of them 22(73.33%) were found to be of male gender of the total AKI patients population and female were found to be 8(26.67%) after analyzing this it was found that majority were belongs to the male gender (Table. 8).

Gender	Number of Patients (n=30)	Percentage (%)
Male	22	73.33
Female	8	26.67

Table. 8 Gender wise Distribution (AKI)

Risk Factors wise Distribution (Male)

The result of distribution according to risk factors of AKI in male shows most of the patients were suffering from HTN 8(36.36%), 6(27.27%) were found of DM & HTN BOTH, 5(22.73%) were found of DM, 1(4.55%) were found of Obstructive Uropathy and 1(9.09%) was found with Glomerulonephritis.

Risk Factor	Number of Patients	Percentage (%)
DM	5	22.73
DM & HTN BOTH	6	27.27
GLOMERULONEPHRITIS	2	9.09
HTN	8	36.36
OBSTRUCTIVE UROPATHY	1	4.55

Table 9: Risk factors wise distribution

Risk Factors wise Distribution (Female)

The result of distribution according to risk factors of female shows most of the patients were suffering from HTN 4(50%), 1(12.5%) were found of POLYCYSTIC KIDNEY DISEASE, 1(12.5%) were found of DM & HTN BOTH, 1(12.5%) were found of OBSTRUCTIVE UROPATHY (Table. 10).

Risk Factors	Number of Patients	Percentage (%)
DM	1	12.5
DM & HTN BOTH	1	12.5
HTN	4	50
OBSTRUCTIVE UROPATHY	1	12.5
POLYCYSTIC KIDNEY DISEASE	1	12.5

Table 10: Risk factors wise distribution

Age wise Distribution (Male)

For this study, total 22 numbers of male patients were observed, According to inclusion and exclusion criteria age of 22 patients were 0-20 year (4.54%), 21-40 year (36.36%), 41-60 year(50%) and 60 year above (9.09%). Majority of the patients were found in the age group of 41-60 years i.e. **11** (50%). (table.11)

Age	Number of Patients	Percentage (%)
0-20 YEARS	1	4.54
21-40 YEARS	8	36.36
41-60 YEARS	11	50
60 ABOVE	2	9.09

Table 11: Age wise distribution (male)

Age wise Distribution (Female)

For this study, total 8 numbers of female patients were observed, According to inclusion and exclusion criteria age of 8 patients were 0-20 year (0%), 21-40 year (37.5%), 41-60 year (50%) and 60 year above (12.5%). Majority of the patients were found in the age group of 41-60 years i.e. **4**(50%) (Table.12).

Age	Number of Patients	Percentage (%)
0-20 YEARS	0	0
21-40 YEARS	3	37.5
41-60 YEARS	4	50
60 ABOVE	1	12.5

Table 12: Age wise distribution (female)

Social History wise Distribution (Male)

For this study, total 22 numbers of male patients were observed, out of which 4(18.18%) Alcoholic, 1(4.54%) Smokers,3(13.64%) smokers & alcoholic both and 14(63.64%) have no addiction (Table.13).

Social History	Number of Patients	Percentage
ALCOHOL	4	18.18
NONE	14	63.64
SMOKING	1	4.54
SMOKING & ALCOHOL BOTH	3	13.64

Table 13: Social history wise distribution

DISCUSSION

The findings indicating that the majority of the study participants had CKD are consistent with global epidemiological trends demonstrating that CKD is a major global public health concern [56]. CKD is a chronic progressive disorder characterized by gradual loss of renal function and is associated with

significant complications including cardiovascular disease, anemia, and bone disorders. The high prevalence observed in this study underscores the need for effective preventive and management strategies to reduce disease burden. The observation that AKI was identified in 29.7% of patients aligns with previous studies reporting that although AKI is less prevalent than CKD, it carries high morbidity and mortality [57]. AKI is defined by a sudden decline in kidney function and is often precipitated by sepsis, dehydration, nephrotoxic drugs, or underlying comorbidities. Early identification and timely management are crucial for improving outcomes. CKD and AKI frequently coexist, and CKD patients are at increased risk of developing AKI [58]. Thus, optimal CKD management may reduce AKI incidence and improve overall patient prognosis. The predominance of male patients (63.38%) in this study is consistent with prior evidence suggesting higher CKD prevalence among males [59]. Hormonal, genetic, and lifestyle-related differences may contribute to this gender disparity [60]. Hypertension was the most common risk factor among male patients (62.22%), supporting established evidence that hypertension is a leading cause and progression factor of CKD [61]. Chronic elevated blood pressure damages renal vasculature, leading to progressive nephron loss. Diabetes, another established CKD risk factor, was less frequently observed among male participants in this study; however, hyperglycemia remains a key contributor to long-term renal damage [61]. Obstructive uropathy and glomerulonephritis were less common etiologies. Glomerulonephritis, characterized by inflammation of glomeruli, impairs filtration, whereas obstructive uropathy causes structural and functional kidney damage. Among female participants, hypertension (73.08%) was also the most common risk factor, reinforcing its dominant etiological role in CKD [61]. A notable proportion of females (11.54%) had polycystic kidney disease (PKD), a hereditary disorder marked by multiple renal cyst formation and progressive renal impairment [62]. Previous literature supports PKD as an important contributor to CKD, particularly among women. Age distribution data revealed higher CKD prevalence among middle-aged individuals (41–60 years), consistent with epidemiological studies demonstrating rising CKD incidence after age 40 [60,63]. Similar patterns have been observed globally, with peak prevalence between 45–64 years [63]. Among females, a considerable proportion fell within 21–40 years, consistent with reports suggesting hormonal influences in younger women may impact renal disease patterns [64,65]. Alcohol consumption was noted in a significant proportion of CKD patients. Previous studies have demonstrated an association between alcohol intake and increased CKD risk, including elevated proteinuria [66]. Chronic alcohol consumption contributes to oxidative stress and hypertension, thereby accelerating renal damage. Smoking prevalence was relatively low in this study; however, both smoking and alcohol have been independently associated with CKD progression [66,67]. Gender differences were also observed in AKI prevalence, with males constituting 73.33% of AKI cases. Earlier studies suggest men may be at higher AKI risk due to physiological, hormonal, and exposure-related differences [68,69]. Comorbid conditions such as diabetes and hypertension further increase AKI susceptibility [70,71]. In this study, hypertension was the most frequent risk factor among male AKI patients (36.36%), consistent with prior reports [71,72]. Among female AKI patients, hypertension remained the most common risk factor (50%), followed by obstructive uropathy and diabetes with hypertension. These findings align with earlier research emphasizing hypertension as a major AKI risk determinant [72]. Age-related analysis demonstrated increased AKI incidence in middle-aged and elderly populations, consistent with studies reporting higher AKI prevalence with advancing age due to declining renal reserve and comorbid conditions [73,74]. Lifestyle factors such as alcohol and smoking were observed among male AKI patients. Previous population-based studies have identified alcohol abuse and smoking as significant contributors to AKI risk [75,76]. These substances may induce renal hypoperfusion, oxidative stress, and inflammatory injury, increasing susceptibility to acute renal damage. Overall, this study reinforces that hypertension and diabetes are the primary modifiable risk factors for both CKD and AKI. Early detection, lifestyle modification, and aggressive management of comorbidities are critical to preventing disease progression and improving patient outcomes.

CONCLUSION

Upon assessing the risk factors for CKD and AKI in the population, it becomes apparent that multiple factors have a substantial impact on the occurrence of these conditions. Typical risk factors, including diabetes, hypertension, smoking, a family history of kidney disease, and advancing age, raise the probability of developing CKD or AKI. In addition to these common risk factors, autoimmune disorders have also been identified as contributing factors to CKD. Autoimmune kidney disease, also known as autoimmune glomerulonephritis, is a type of kidney disease that occurs when the immune system attacks the kidneys. Contributing factors to autoimmune kidney disease may include genetics, environmental triggers, and other autoimmune disorders such as lupus or rheumatoid arthritis. AKI can be caused by a variety of factors such as dehydration, infections, medications, and toxins. If left untreated, AKI can lead to

permanent kidney damage and the development of CKD. Overall, the data suggests that a multifaceted approach is needed to prevent and manage CKD and AKI. This includes addressing common risk factors such as diabetes and high blood pressure, while also considering factors such as autoimmune disorders and AKI. Identification of contributing factors to autoimmune kidney disease is also essential for the development of targeted treatment strategies. With a comprehensive approach, it is possible to prevent and manage these conditions effectively, leading to improved outcomes and quality of life for those affected.

REFERENCES:

1. Hall JE. (2021). Guyton and Hall Textbook of Medical Physiology. 14th ed. Philadelphia: Elsevier.
2. Boron WF, Boulpaep EL. (2017). Medical Physiology. 3rd ed. Philadelphia: Elsevier.
3. Barrett KE, Barman SM, Brooks HL, Yuan JXJ. (2019). Ganong's Review of Medical Physiology. 26th ed. New York: McGraw Hill.
4. Taal MW, Chertow GM, Marsden PA, Skorecki K, Yu ASL, Brenner BM. (2020). Brenner & Rector's The Kidney. 11th ed. Philadelphia: Elsevier.
5. Ross MH, Pawlina W. (2019). Histology: A Text and Atlas. 8th ed. Philadelphia: Wolters Kluwer.
6. Junqueira LC, Carneiro J. (2018). Basic Histology. 15th ed. New York: McGraw Hill.
7. Young B, O'Dowd G, Woodford P. (2014). Wheater's Functional Histology. 6th ed. Philadelphia: Elsevier.
8. Guyton AC, Hall JE. (2016). Formation of urine by the kidneys. In: Textbook of Medical Physiology. 13th ed. Philadelphia: Elsevier.
9. Koeppen BM, Stanton BA. (2013). Renal Physiology. 5th ed. Philadelphia: Elsevier.
10. Koury MJ, Haase VH. (2015). Anaemia in kidney disease: harnessing hypoxia responses for therapy. *Nat Rev Nephrol.* 11(7):394-410.
11. Kumar V, Abbas AK, Aster JC. (2020). Robbins & Cotran Pathologic Basis of Disease. 10th ed. Philadelphia: Elsevier.
12. Skorecki K, Chertow GM, Marsden PA, Taal MW, Yu ASL. Pathogenesis of kidney disease. In: Brenner & Rector's The Kidney. 11th ed. 2020.
13. Jha V, Garcia-Garcia G, Iseki K, et al. (2013). Chronic kidney disease: global dimension and perspectives. *Lancet.* ;382(9888):260-272.
14. Webster AC, Nagler EV, Morton RL, Masson P. (2017). Chronic kidney disease. *Lancet.* 389(10075):1238-1252.
15. Levey AS, Coresh J. (2012). Chronic kidney disease. *Lancet.* 379(9811):165-180.
16. KDIGO. KDIGO (2012). Clinical Practice Guideline for the Evaluation and Management of CKD. *Kidney Int Suppl.* 3(1):1-150.
17. Levin A, Stevens PE, Bilous RW, et al. (2013). KDIGO 2012 clinical practice guideline. *Kidney Int Suppl.* 3(1):1-150.
18. Kellum JA, Lameire N. (2012). KDIGO Clinical Practice Guideline for Acute Kidney Injury. *Kidney Int Suppl.* 2(1):1-138.
19. Mark, Patrick B et al. (2023). Global, regional, and national burden of chronic kidney disease in adults, 1990–2023, and its attributable risk factors: a systematic analysis for the Global Burden of Disease Study. *The Lancet*, Volume 406, Issue 10518, 2461 - 2482
20. Bikbov B, Purcell CA, Levey AS, et al. (2020). Global burden of CKD 1990–2017. *Lancet.* 395:709–733.
21. Singh AK, Farag YM, Mittal BV, et al. (2013). Epidemiology of CKD in India. *BMC Nephrol.* 14:114.
22. Varma PP. (2015). Prevalence of CKD in India. *Indian J Nephrol.* 25(3):133–137.
23. Rajapurkar MM, John GT, Kirpalani AL, et al. (2012). What do we know about CKD in India. *BMC Nephrol.* 13:10.
24. Anand S, Bitton A, Gaziano T. CKD in developing countries. *BMJ Glob Health.* 1:e000078.
25. Go AS, Chertow GM, Fan D, et al. (2004). CKD and mortality risk. *N Engl J Med.* 351:1296–1305.
26. Afkarian M, Sachs MC, Kestenbaum B, et al. (2013). Kidney disease in diabetes. *J Am Soc Nephrol.* 24(2):302–308.
27. Mills KT, Xu Y, Zhang W, et al. (2020). Global burden of hypertension. *Circulation.* 141(7):e1–e33.
28. Hsu CY, McCulloch CE, Iribarren C, et al. (2006). Body mass index and CKD risk. *Ann Intern Med.* 144:21–28.
29. Orth SR, Hallan SI. (2008). Smoking and kidney disease. *Kidney Int.* 73:397–404.
30. Coresh J, Selvin E, Stevens LA, et al. (2007). Prevalence of CKD in US. *JAMA.* 298:2038–2047.
31. Peralta CA, Katz R, Sarnak MJ, et al. (2006). Ethnicity and kidney disease. *J Am Soc Nephrol.* 17:289–296.
32. Floege J, Amann K. (2016). Primary glomerulonephritis. *Lancet.* 387:2036–2048.
33. Harris PC, Torres VE. (2009). Polycystic kidney disease. *Annu Rev Med.* 60:321–337.
34. Klahr S. (2000). Obstructive nephropathy. *Intern Med.* 39:355–361.
35. Perazella MA. (2009). Drug-induced nephrotoxicity. *Clin J Am Soc Nephrol.* 4:1275–1283
36. Alicic RZ, Rooney MT, Tuttle KR. (2017). Diabetic kidney disease. *Clin J Am Soc Nephrol.* 12:2032–2045.
37. Ku E, Lee BJ, Wei J, Weir MR. (2019). Hypertension in CKD. *J Am Coll Cardiol.* 73:126–139.
38. Eddy AA. (2005). Progression of CKD. *Kidney Int.* 68:S2–S7.
39. Ruiz-Ortega M, Rodrigues-Diez R, Lavoz C, Rayego-Mateos S. (2020). Renal inflammation. *Nat Rev Nephrol.* ;16:269–285.
40. Vaziri ND. (2004). Oxidative stress in CKD. *Kidney Int.* 65:1563–1576.
41. Ruster C, Wolf G. (2006). RAAS and kidney disease. *J Am Soc Nephrol.* 17:2985–2991.
42. Remuzzi G, Benigni A, Remuzzi A. (2006). Pathophysiology of CKD. *N Engl J Med.* 354:997–1008.
43. Moe SM, Chen NX. (2008). CKD-mineral bone disorder. *Am J Kidney Dis.* 52:1130–1144.

44. Lewis EJ, Hunsicker LG, Clarke WR, et al. (2001). ACE inhibition in diabetic nephropathy. *N Engl J Med*;345:851–860.
45. Levey AS, Stevens LA, Schmid CH, et al. (2009). CKD-EPI equation. *Ann Intern Med*. 150:604–612.
46. Matsushita K, van der Velde M, Astor BC, et al. (2010). GFR and albuminuria prognosis. *Lancet*.375:2073–2081.
47. Stevens PE, Levin A. (2013). Evaluation of CKD. *Kidney Int Suppl*.3:1–150.
48. Fogazzi GB. (1996). Urinalysis and kidney disease. *Am J Kidney Dis*. 28:1–11.
49. Moghazi S, Jones E, Schroeppe J, et al. (2005). Renal biopsy indications. *Am J Kidney Dis*.46:1034–1039.
50. O'Neill WC. (2000). Sonographic evaluation of renal disease. *Am J Kidney Dis*. 35:1021–1038.
51. Wright JT Jr, Williamson JD, Whelton PK, et al. (2015). Blood pressure control. *N Engl J Med*. 373:2103–2116.
52. Singh, M., Nayak, S. S., Tiwari, P., Shaikh, S., Gautam, V., & Ghatol, P. W. (2023). Review article on hemodialysis and its complications. *Asian Journal of Pharmaceutical Research and Development*, 11(2), 60-64..
53. Boddula, H., Shivani, R., Srinivas Nayak, S. P., Vaghasia, J., Chakraborty, G. S., Mandal, S. D., & Ghatol, P. (2023). The Effectiveness of SGLT2 Inhibitors in CKD Patients. *Journal of Drug Delivery & Therapeutics*, 13(3).22:209–217
54. Lokhandwala Z, et al. (2025). Assessment of prevalence of breast cancer in patients with obesity and evaluating benefits of statin therapy in breast cancer treatment. *Asian J Pharm Clin Res*. 18(5):80-86. doi:10.22159/ajpcr.2025v18i5.54356
55. Chang MY, Ong AC. (2013). New treatments for autosomal dominant polycystic kidney disease. *Br J Clin Pharmacol*. 76(4):524-35. doi: 10.1111/bcp.12136. PMID: 23594398; PMCID: PMC3791976.
56. Levey AS, Coresh J, Tighiouart H, et al. (2020). Chronic kidney disease. *Lancet*. 395(10225):709-733. doi:10.1016/S0140-6736(20)30045-3
57. Hoste EAJ, Kellum JA, Selby NM, et al. (Global epidemiology and outcomes of acute kidney injury. *Nat Rev Nephrol*. 2018;14:607-625. doi:10.1038/s41581-018-0052-0
58. Chawla LS, Eggers PW, Star RA, Kimmel PL. Acute kidney injury and chronic kidney disease as interconnected syndromes. *N Engl J Med*. 2014;371:58-66. doi:10.1056/NEJMra1214243
59. Zhang L, Wang F, Wang L, et al. (2012). Prevalence of chronic kidney disease in China. *Lancet*. 379:815-822. doi:10.1016/S0140-6736(12)60033-6
60. Gansevoort RT, Correa-Rotter R, Hemmelgarn BR, et al. (2013). Chronic kidney disease and cardiovascular risk. *Lancet*.382:339-352. doi:10.1016/S0140-6736(13)60595-4
61. Jha V, Garcia-Garcia G, Iseki K, et al. (2013). Chronic kidney disease: global dimension. *Lancet*. 382:260-272. doi:10.1016/S0140-6736(13)60687-X
62. Grantham JJ. (2008). Autosomal dominant polycystic kidney disease. *N Engl J Med*. 359:1477-1485. doi:10.1056/NEJMcp0804458
63. Hill NR, Fatoba ST, Oke JL, et al. (2016). Global prevalence of chronic kidney disease. *PLoS One*. 11:e0158765. doi:10.1371/journal.pone.0158765
64. Bhargava R, Jasuja S, Tangri N. (2017). Gender disparities in kidney disease. *Clin J Am Soc Nephrol*. 12:201-209. doi:10.2215/CJN.07370716
65. Fogo AB. (2018). Causes and pathogenesis of focal segmental glomerulosclerosis. *Nat Rev Nephrol*.14:76-92. doi:10.1038/nrneph.2017.181
66. Chen N, Hsu CC, Yamagata K, Langham R. (2017). Alcohol consumption and risk of CKD. *Kidney Int*. 92:114-122. doi:10.1016/j.kint.2017.02.018
67. Sood MM, Rigatto C, Bueti J. (2015). Lifestyle factors in CKD progression. *Clin J Am Soc Nephrol*. 10:151-159. doi:10.2215/CJN.04510514
68. Liu KD, Goldstein SL, Vijayan A, et al. (2020). Sex differences in acute kidney injury. *Clin J Am Soc Nephrol*. ;15:1173-1181. doi:10.2215/CJN.00690120
69. Xu X, Nie S, Liu Z, et al. (2018). Gender differences in AKI. *Kidney Int Rep*. 3:563-571. doi:10.1016/j.ekir.2018.01.010
70. Grammes F, Voigt A, Bergmann C, et al. (2016). Epidemiology of acute kidney injury. *Dtsch Arztebl Int*. 113:379-386. doi:10.3238/arztebl.2016.0379
71. Kovesdy CP, Kalantar-Zadeh K. (2018). Risk factors for acute kidney injury. *Nat Rev Nephrol*. 14:153-166. doi:10.1038/nrneph.2017.191
72. Sharma A, Mucino MJ, Ronco C. (2017). Hypertension and risk of AKI. *Clin Exp Nephrol*. 23:439-446. doi:10.1007/s10157-018-1655-2
73. Yessayan L, Yee J, Frinak S, Szamosfalvi B. (2016). Acute kidney injury in the elderly. *Clin Geriatr Med*. 2016;32:285-297. doi:10.1016/j.cger.2016.01.001
74. Liaño F, Pascual J. (1996). Epidemiology of acute renal failure. *Kidney Int*. 50:811-818. doi:10.1038/ki.1996.380
75. Wong L, Liew A, Weng C. (2015). Smoking and risk of acute kidney injury. *Nephrology*. 20:632-639. doi:10.1111/nep.12460
76. Azzopardi D, Verheggen R, De Meersman A, et al. (2017). Alcohol misuse and acute kidney injury. *Crit Care*. ;21:12. doi:10.1186/s13054-016-1597-9

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