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Managing Pulmonary Edema and Electrolyte Imbalance in Advanced Chronic Kidney Disease: A Case Report

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Abstract

Background : *Chronic kidney disease (CKD) is a progressive disorder that leads to fluid overload and electrolyte imbalances, increasing the risk of pulmonary edema, hyperkalemia, and hyponatremia. These complications contribute to high morbidity and mortality, necessitating a multidisciplinary approach for optimal management.*
Case Descriprion : *A 74-year-old female with stage V CKD presented with acute respiratory distress and severe dyspnea due to pulmonary congestion. Laboratory tests revealed hyperkalemia (6.76 mEq/L), hyponatremia (129 mEq/L), and metabolic acidosis. Chest X-ray confirmed bilateral pulmonary edema, while ECG showed peaked T waves, indicating potassium-induced cardiac instability. Initial management with loop diuretics failed, requiring urgent hemodialysis for fluid removal and electrolyte correction. Calcium gluconate, insulin-dextrose, and potassium binderswere used to control hyperkalemia, while fluid restriction and slow sodium correction were implemented for hyponatremia. The patient showed significant improvement within 48 hours, with stabilization of respiratory function and electrolyte levels.*
Discussion : *Pulmonary edema in CKD results from volume overload, RAAS activation, and endothelial dysfunction, while electrolyte imbalances arise from impaired renal excretion. Management involves diuretics, dialysis, and targeted electrolyte correction. Emerging therapies such as SGLT2 inhibitors and novel potassium binders offer promising outcomes.*
Conclusion : *This case highlights the importance of early intervention, hemodialysis, and precise electrolyte management in CKD patients with pulmonary edema. Future research should focus on personalized nephroprotective strategies to enhance patient outcomes.*
Keywords : *Chronic Kidney Disease, lectrolyte imbalance, hemodialysis, hyperkalemia, hyponatremia, pulmonary edema*

Introduction

Chronic Kidney Disease (CKD) has been ranked as the 10th leading cause of death. Early detection, and diagnosis, along with proper management by primary care physicians are particularly important to minimize the complications, related to CKD that include cardiovascular, diseases and also end-stage renal disease (ESRD) and mortality (KDIGO, 2024).

According to the Indonesian Nephrology Association (PERNEFRI) in 2020, the prevalence of chronic kidney disease (CKD) in Indonesia was reported at 0.38%. However, the actual prevalence is likely much higher, as 9 out of 10 individuals are unaware they have kidney failure (PERNEFRI, 2021).

Chronic kidney disease (CKD) is a major global health burden affecting millions of individuals worldwide, characterized by a progressive decline in renal function that ultimately leads to end-stage renal disease (ESRD). CKD is associated with severe complications, including pulmonary edema and electrolyte imbalances, which significantly contribute to increased morbidity and mortality among affected patients (Farha et al., 2020). The increasing prevalence of CKD is driven by rising incidences of diabetes mellitus, hypertension, and aging populations, making its management a critical priority in modern nephrology (Chen et al., 2020). Managing these complications effectively is crucial in improving patient outcomes and reducing the burden of the disease.

Pulmonary edema in CKD is primarily caused by fluid overload due to impaired sodium and water excretion, as well as increased capillary permeability. These disturbances lead to pulmonary congestion and alveolar flooding, which result in respiratory distress, reduced oxygenation, and increased risk of cardiovascular complications

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(Mallamaci & Zoccali, 2022). Pulmonary edema is further exacerbated by heart failure, uremic toxin accumulation, and systemic inflammation, highlighting the complex pathophysiology of CKD-related respiratory dysfunction (Rossignol et al., 2021).

In addition to fluid overload, electrolyte imbalances are a common and life-threatening complication of CKD, including hyperkalemia, hyponatremia, hyperphosphatemia, and hypocalcemia. Hyperkalemia, defined as serum potassium levels exceeding 5.5 mEq/L, is a major concern due to its potential to induce fatal cardiac arrhythmias and neuromuscular dysfunction (Sterns & Silver, 2021). Hyponatremia, the most common electrolyte disorder in CKD, results from excessive free water retention, leading to cerebral edema and neurological symptoms such as confusion, seizures, and coma (Chiu & Hsu, 2021). Hyperphosphatemia occurs due to impaired phosphate excretion, stimulating excessive parathyroid hormone (PTH) secretion, leading to secondary hyperparathyroidism, vascular calcification, and increased cardiovascular mortality (Goraya & Wesson, 2020). Hypocalcemia often accompanies hyperphosphatemia due to impaired vitamin D metabolism, contributing to bone demineralization and an increased risk of fractures (Kovesdy et al., 2021).

The proper diagnosis of pulmonary edema and electrolyte imbalances in CKD patients involves a thorough clinical assessment, laboratory investigations, and imaging studies. Serum creatinine, blood urea nitrogen (BUN), and estimated glomerular filtration rate (eGFR) are essential for evaluating kidney function, while electrolyte panels help detect abnormalities (Chen et al., 2020). Early diagnosis and management of these complications are crucial for improving patient outcomes and preventing progression to ESRD. A comprehensive diagnostic approach includes serum electrolyte measurements, renal function tests, electrocardiography (ECG) for detecting hyperkalemia-induced arrhythmias, and chest imaging to assess pulmonary congestion (Palmer et al., 2022). Chest X-rays and lung ultrasounds provide valuable insights into pulmonary congestion and alveolar fluid accumulation, confirming the presence of pulmonary edema (Mallamaci & Zoccali, 2022). The treatment of pulmonary edema in CKD involves fluid restriction, diuretics, dialysis, and supportive oxygen therapy, while electrolyte imbalances are managed through dietary modifications, pharmacologic interventions, and renal replacement therapy in severe cases (Jager et al., 2021).

Recent advancements in CKD management have led to the development of novel therapeutic strategies, including sodium-glucose cotransporter-2 (SGLT2) inhibitors, which have shown promise in reducing fluid overload and delaying CKD progression (Kovesdy et al., 2021). Additionally, selective potassium binders, such as patiromer and sodium zirconium cyclosilicate, provide more effective and safer control of hyperkalemia compared to conventional potassium-exchange resins (Palmer et al., 2022). Furthermore, emerging technologies, such as bioengineered kidneys and wearable dialysis devices, aim to revolutionize renal replacement therapy and improve the quality of life for CKD patients (Kirkman et al., 2023).

This paper explores the pathophysiology, diagnostic approaches, and treatment strategies for pulmonary edema and electrolyte imbalances in CKD, emphasizing recent advancements in therapy and future perspectives in nephrology. Understanding the interplay between fluid balance, electrolyte homeostasis, and renal function is essential for developing effective management strategies to mitigate complications and enhance patient outcomes. This case report presents a 74-year-old woman with stage V CKD who developed severe pulmonary edema and multiple electrolyte imbalances, requiring urgent medical intervention. The discussion will explore the pathophysiology, diagnostic approach, and management strategies for these complications, with an emphasis on emerging treatment options and future perspectives in nephrology.

Case Description

A 74-year-old woman was brought to the Emergency Room of PKU Muhammadiyah Solo Hospital on October 16, 2024, after being referred from Surakarta General Hospital with the primary complaint of shortness of breath. Initially, the shortness of breath occurred during strenuous activities or fatigue, particularly at night. However, over the past week, it had become more frequent and was accompanied by chest discomfort. The condition worsened when lying down but improved when sitting upright or in a tilted position. No cold sweats were reported.

Additional symptoms included dizziness, nausea, vomiting, and coughing with thick yellow phlegm. The patient also experienced loss of appetite and a weight loss of 5 kg. During the five-day hospitalization, the patient had a urine output of 2500 ml, which was clear yellow in appearance, and swelling in both legs and the right hand.

The patient had no prior history of hemodialysis (HD) and was referred for HD initiation. Her medical history included diabetes mellitus (DM), hypertension, and stage 4 chronic kidney disease (CKD) diagnosed at Surakarta General Hospital. Previous medications included Candesartan, N-Acetylcysteine, and Folic Acid.

On physical examination, the patient was in a moderate general condition with a compos mentis level of consciousness. Vital signs were as follows: blood pressure 117/78 mmHg, pulse rate 59 beats per minute, respiratory rate 20 breaths per minute, temperature 36.7°C, and oxygen saturation of 98% on room air. General status was within normal limits. Examination of the head showed brown skin, symmetrical facial structure, slightly icteric sclera (+/+), and anemic conjunctiva (+/+). The pupils were isochoric, indicating intact autonomic innervation in both eyes. No lymphadenopathy was detected, the thyroid was not enlarged, and there was no increased jugular venous pressure (JVP).

Thoracic examination revealed symmetrical chest expansion, reduced fremitus in both lung fields, dull percussion, and the presence of bilateral moist rhonchi. Heart sounds were normal without murmurs. Abdominal examination was unremarkable, capillary refill time was less than two seconds, acral areas were warm, and there was edema in the extremities.

The patient was treated with IV Paracetamol 500 mg, IV Omeprazole twice daily, IV Ondansetron 8 mg, oral Acetylcysteine 200 mg three times daily, and oral Episan three times daily. After treatment, the patient's symptoms, including fever, cough, nausea, and vomiting, showed improvement.

Laboratory tests on October 16, 2024, revealed several abnormal values: urea 143 mg/dL (H), creatinine 7.8 mg/dL (H), potassium 6.76 mEq/L (L), erythrocytes 1.96 million/µL (L), hemoglobin 6.2 g/dL (L), hematocrit 17.8% (L), neutrophils 75.3% (H), lymphocytes 20.2% (L), eosinophils 0.3% (L), and an NLR value of 3.73 (H). ECG findings showed a sinus rhythm, heart rate of 68 bpm, and a normal axis without abnormalities.

A follow-up laboratory test on October 17, 2024, showed persistent abnormalities, including urea 120 mg/dL (H), creatinine 3.6 mg/dL (H), and hemoglobin 9.1 g/dL (L).

The EKG results were normal. The patient also underwent a chest X-ray, which showed no enlargement of the heart and no signs of lung exhaustion.

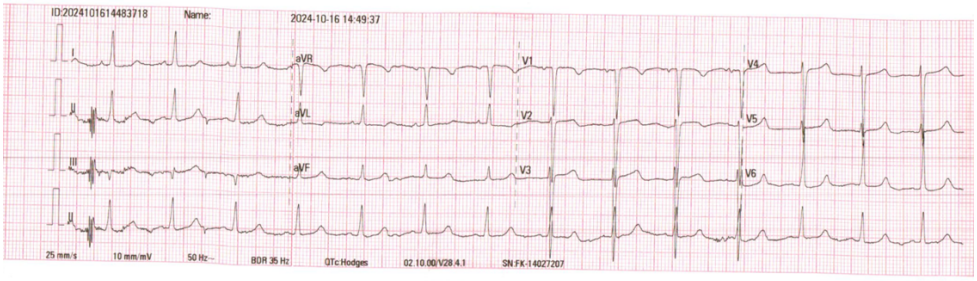

Figure 1. ECG examination

Initial chest X-ray findings revealed an increased vascular pattern without cardiac enlargement or signs of lung exhaustion. However, a follow-up chest X-ray showed increased vascular markings, right pericardial infiltration, cardiomegaly, pulmonary edema, bronchopneumonia, and bilateral pleural effusion.

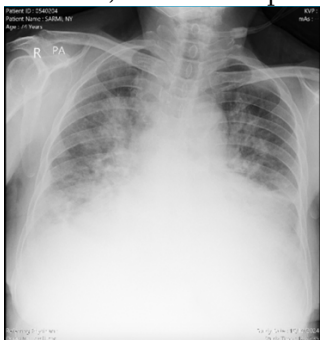

Figure 2. Thorax X-ray Examination

Due to the critical condition of the patient, electrolyte analysis was performed, revealing a potassium level of 6.76 mEq/L. Additional tests for urea and creatinine showed values of 143 mg/dL and 7.8 mg/dL, respectively, indicating kidney dysfunction. A chest X-ray was conducted to investigate the cause of shortness of breath, confirming findings

of increased vascular markings, right pericardial infiltration, cardiomegaly, pulmonary edema, bronchopneumonia, and bilateral pleural effusion.

Based on laboratory and radiological findings, the patient was started on several treatments, including IV NaCl 0.9%, parenteral Kleum, nasal cannula oxygen at 5 LPM with a target saturation of 94%, Nebu Combiven (Budesma) every 12 hours, IV Sanmol 1 gram every 8 hours, IV Furosemide 2 ampoules every 8 hours, IV Ca Gluconate 1 ampoule + D40% flash + IV Novorapid 10 IU, PRC 2 Kolf, IV Santagesic 1 ampoule every 8 hours, IV Anbacim 1 gram every 12 hours, and IV Fluomucyl 1 ampoule every 12 hours.

The patient was also prescribed oral medications based on their condition, including ISDN 3x5 mg, Folic Acid 2x1, Bicnar 3x1, Kalquest 3x1, Nifedipine 1x10 mg, and Respira 1x1. Monitoring included 24-hour urine collection, vital sign assessments, dyspnea evaluation, PRC 2 Kolf 1K every 12 hours, and fluid balance checks every 6, 10, and 12 hours. A dialysis catheter was placed in preparation for the HD program, with HD sessions scheduled accordingly.

The patient was placed on a dietary regimen with an energy intake of 30-35 kcal/kg body weight per day, protein intake of 1.25 g/kg body weight per day, fiber intake of 30-35 g per day, sodium intake of less than 2300 mg per day, and calcium intake of 1000 mg per day. Potassium intake was carefully monitored, keeping urine potassium below 400 ml/day or serum potassium below 5.5 mEq/L. Fluid intake was restricted to approximately 500 ml/day.

Discussion

Chronic Kidney Disease (Stage 5) is a progressive condition marked by a long-term decline in kidney function, typically occurring over months to years, and is defined by a decrease in Glomerular Filtration Rate (GFR) to less than 60 ml/min/1.73m². Several factors contribute to chronic kidney failure, including metabolic disorders (such as diabetes mellitus), infections (like pyelonephritis), urinary tract obstructions, immunological disorders, hypertension, primary tubular disorders (e.g., nephrotoxins), and congenital conditions that lead to reduced GFR (KDIGO, 2024).

CKD is classified based on both the stage of the disease and its underlying cause. The staging is determined by the level of kidney function, as measured by the estimated GFR (eGFR), using the Cockcroft-Gault formula (Table 1). This classification helps to assess the severity of kidney function decline (KDIGO, 2024).

Table 1. Classification helps to assess the severity of kidney function decline (KDIGO, 2024)

Stage	GFR	Kategori	Keterangan
G1	>= 90	normal atau sedikit berkurang	albuminuria persisten
G2	60-89	penurunan ringan	serum kreatinin naik dan albuminuria
G3	30-59	penurunan sedang	serum kreatinin naik dan albuminuria
G4	15-29	penurunan berat	persiapan terapi ginjal
G5	<15	gagal ginjal end stage	terapi ginjal permanen

Chronic kidney disease (CKD) is a progressive disorder that significantly impacts fluid balance and electrolyte homeostasis. One of the most critical complications in advanced CKD (stage IV-V) is pulmonary edema, primarily due to volume overload, increased capillary permeability, and reduced sodium and water excretion (Mallamaci & Zoccali, 2022). Furthermore, electrolyte imbalances such as hyperkalemia, hyponatremia, and hyperphosphatemiafrequently occur, increasing the risk of cardiac arrhythmias, neurological dysfunction, and cardiovascular mortality (Palmer & Clegg, 2020). The interplay between impaired renal clearance, hormonal dysregulation, and systemic inflammation exacerbates these complications, necessitating a multidisciplinary approach to management.

Chronic kidney disease (CKD) is a complex and progressive disorder that significantly disrupts fluid and electrolyte homeostasis, leading to severe complications such as pulmonary edema and electrolyte imbalances. These complications are among the leading causes of morbidity and mortality in CKD patients, making their management a key aspect of nephrology care (Jager et al., 2021). Pulmonary edema in CKD arises primarily from volume overload, increased capillary permeability, and left ventricular dysfunction, all of which contribute to respiratory distress and hypoxemia (Mallamaci & Zoccali, 2022). Simultaneously, electrolyte disturbances, including hyperkalemia,

hyponatremia, and hyperphosphatemia, further exacerbate the clinical burden and require careful monitoring and intervention (Palmer et al., 2022). Understanding the pathophysiology, diagnostic approaches, and emerging therapeutic strategies is essential for optimizing the management of these conditions.

Pulmonary edema in CKD occurs due to volume overload and increased hydrostatic pressure in the pulmonary capillaries. The inability of the kidneys to excrete excess sodium and water leads to fluid retention, increasing pulmonary capillary wedge pressure and promoting fluid leakage into the alveolar spaces, impairing oxygen diffusion and gas exchange (Bansal & Pergola, 2020). Pulmonary edema is a severe complication in patients with advanced chronic kidney disease (CKD) and is primarily caused by fluid overload and increased capillary permeability in the pulmonary vasculature. As kidney function deteriorates, the ability to excrete excess fluid is impaired, leading to increased venous pressure and transudation of fluid into the alveolar spaces. This process disrupts gas exchange, causing dyspnea and hypoxia (Mallamaci & Zoccali, 2022). Additionally, activation of the renin-angiotensin-aldosterone system (RAAS) and sympathetic nervous system (SNS) contributes to vasoconstriction, sodium retention, and systemic hypertension, exacerbating left ventricular overload and pulmonary congestion (Rossignol et al., 2021). Chronic inflammation in CKD also plays a role in endothelial dysfunction, increasing pulmonary vascular permeability and worsening pulmonary edema (Kumar & Berl, 2021).

Electrolyte imbalances are another major concern in advanced CKD due to impaired renal regulatory functions. Hyperkalemia is one of the most life-threatening disturbances, as reduced renal potassium excretion leads to increased serum potassium levels, increasing the risk of cardiac arrhythmias and neuromuscular dysfunction. Metabolic acidosis and the use of RAAS inhibitors further exacerbate hyperkalemia (Khan et al, 2020). Additionally, CKD patients often experience hyponatremia due to excessive water retention, leading to dilutional effects that may cause neurological symptoms such as confusion, seizures, and coma (Chiu & Hsu, 2021). Hyperphosphatemia is another common electrolyte disorder caused by reduced renal phosphate excretion, which triggers secondary hyperparathyroidism, leading to bone demineralization and vascular calcification. This is often accompanied by hypocalcemia due to impaired vitamin D activation in CKD (Goraya & Wesson, 2020). The diagnosis of pulmonary edema and electrolyte imbalances in CKD patients requires a combination of clinical assessment, laboratory investigations, and imaging studies. Laboratory tests reveal elevated blood urea nitrogen (BUN), creatinine, and decreased estimated glomerular filtration rate (eGFR), confirming CKD progression. Serum electrolyte levels must be monitored closely for hyperkalemia, hyponatremia, and metabolic acidosis (Mount, 2020).

The diagnosis of pulmonary edema and electrolyte imbalances in CKD patients requires a combination of clinical assessment, laboratory investigations, and imaging studies. Laboratory tests reveal elevated blood urea nitrogen (BUN), creatinine, and decreased estimated glomerular filtration rate (eGFR), confirming CKD progression. Serum electrolyte levels must be monitored closely for hyperkalemia, hyponatremia, and metabolic acidosis (Mount, 2020). Electrocardiography (ECG) plays a crucial role in detecting hyperkalemia-induced cardiac abnormalities, such as peaked T waves, widened QRS complexes, and prolonged PR intervals, which require urgent intervention (Sterns & Silver, 2020). Chest radiography and lung ultrasound are essential tools for evaluating pulmonary congestion and alveolar fluid accumulation, aiding in the diagnosis of pulmonary edema and volume overload (Mallamaci & Zoccali, 2022).

The management of pulmonary edema in CKD primarily focuses on fluid removal, oxygenation support, and hemodynamic stabilization. Loop diuretics (e.g., furosemide, bumetanide) remain the first-line treatment, promoting sodium and water excretion to relieve volume overload (Palmer et al., 2022). However, diuretic resistance is common in advanced CKD, necessitating higher doses or combination therapy with thiazide diuretics (Jager et al., 2021). In refractory cases, ultrafiltration via hemodialysis is required for rapid fluid removal and electrolyte correction (Kirkman et al., 2023). Oxygen therapy and non-invasive ventilation (e.g., CPAP, BiPAP) are beneficial in patients with severe hypoxemia and respiratory distress (Rossignol et al., 2021). In patients with concurrent heart failure, the use of RAAS inhibitors, beta-blockers, and SGLT2 inhibitors has been shown to improve cardiovascular and renal outcomes (Kovesdy et al., 2021).

Electrolyte imbalance management in CKD requires a targeted approach. Hyperkalemia is managed with calcium gluconate to stabilize cardiac membranes, insulin-dextrose infusion to shift potassium intracellularly, and potassium-binding agents such as sodium polystyrene sulfonate or patiromer (Bansal & Pergola, 2020). In severe cases, hemodialysis is required for potassium removal. Hyponatremia is addressed through fluid restriction to prevent dilutional effects, while hypertonic saline infusion is cautiously administered in symptomatic cases to avoid rapid correction and the risk of osmotic demyelination syndrome (Hoorn & Zietse, 2021). Hyperphosphatemia is controlled using phosphate binders such as sevelamer and lanthanum carbonate to reduce phosphate absorption, and vitamin D analogs are used to correct hypocalcemia and prevent secondary hyperparathyroidism (Wang & Hu, 2020).

Recent advances in CKD management have introduced new therapeutic options. SGLT2 inhibitors (e.g., dapagliflozin, empagliflozin) have been shown to reduce fluid retention, lower blood pressure, and improve clinical outcomes in CKD patients (Kovesdy et al., 2021). Selective potassium binders (patiromer, sodium zirconium cyclosilicate) are more effective in controlling hyperkalemia than traditional potassium binders, with fewer side effects (Palmer et al., 2022). Emerging technologies, such as bioengineered kidneys and portable dialysis systems, aim to revolutionize renal replacement therapy, improving the quality of life for CKD patients (Kirkman et al., 2023).

The prognosis of CKD patients with pulmonary edema and electrolyte imbalances largely depends on early intervention and appropriate management. If untreated, these complications significantly increase the risk of cardiovascular

morbidity, hospitalization, and mortality (Shigeo, 2020). Future advancements in CKD management include the use of sodium-glucose cotransporter-2 (SGLT2) inhibitors to improve fluid balance regulation, bioengineered kidneys as a potential alternative to dialysis, and artificial intelligence-based predictive models for early detection of electrolyte imbalances. Multidisciplinary care, personalized treatment strategies, and the implementation of emerging therapies can enhance patient outcomes and improve quality of life.

Conclusion

This case highlights the multifaceted challenges in managing pulmonary edema and electrolyte imbalances in patients with advanced CKD. Early diagnosis, individualized fluid and electrolyte management, and appropriate use of renal replacement therapy are key factors in improving patient outcomes. The integration of emerging therapies and predictive tools will further refine treatment protocols, reducing morbidity and mortality in CKD patients. Timely interventions, including the judicious use of diuretics, initiation of hemodialysis, and careful correction of electrolyte disturbances, are crucial in improving patient outcomes. Ongoing research and clinical vigilance are essential to refine management strategies and enhance the quality of care for this vulnerable population.

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References

Bansal, S., & Pergola, P. E. (2020). Current management of hyperkalemia in patients with chronic kidney disease. *Clinical Journal of the American Society of Nephrology*, 15(8), 1258–1260.

Chiu, Y. W., & Hsu, C. C. (2021). Hyponatremia in chronic kidney disease: Epidemiology, pathophysiology, and clinical management. *Kidney International Reports*, 6(5), 1232–1242.

Chen, T. K., Knicely, D. H., & Grams, M. E. (2020). Chronic kidney disease diagnosis and management: A review. *JAMA*, 322(13), 1294-1304.

Farha, N., & Munguti, C. (2020). A Dramatic Presentation of Pulmonary Edema Due to Renal Failure. *Kansas Journal of Medicine*, 13, 56–57.

Goraya, N., & Wesson, D. E. (2020). Management of the metabolic acidosis of chronic kidney disease. *Advances in Chronic Kidney Disease*, 27(5), 377–383.

Hoorn, E. J., & Zietse, R. (2021). Hyponatremia and chronic kidney disease: A detrimental combination. *Clinical Kidney Journal*, 14(6), 1556–1564.

Jager, K. J., Kovesdy, C., Langham, R., Rosenberg, M., Jha, V., & Zoccali, C. (2021). A single number for advocacy and communication—Worldwide more than 850 million individuals have kidney diseases. *Kidney International*, 96(5), 1048-1050.

KDIGO. (2024). KDIGO 2024 Clinical Practice Guideline For The Evaluation And Management Of Chronic Kidney Disease. *Kidney International*, *105*(4). Https://Doi.Org/10.1016/S0085-2538(24)00110-8

Khan, Z. A., Khan, R. A., Najmuddin, A. M., Khan, A. M., Fahad, S., & Safir, S. R. (2024). Electrolyte Imbalances in Kidney Disease: Hyponatremia, Hyperkalemia, and Hypocalcemia. *Journal of Population Therapeutics and Clinical Pharmacology*, 29(02), 623–628

Kirkman, D. L., et al. (2023). Advances in bioengineered kidneys for renal replacement therapy. *Nature Reviews Nephrology*, 19(2), 120-134.

Kovesdy, C. P., & Lott, E. H. (2021). Hyponatremia in chronic kidney disease: An update. *Nephrology Dialysis Transplantation*, 35(8), 1429–1435.

Kovesdy, C. P., Matsushita, K., Sang, Y., Brunskill, N. J., Carrero, J. J., Chodick, G.,Coresh, J. (2023). Serum potassium and adverse outcomes across the range of kidney function: A CKD Prognosis Consortium meta-analysis. *European Heart Journal*, 44(1), 31–40.

Kumar, S., & Berl, T. (2021). Chronic Kidney Disease, Fluid Overload, and Diuretics. *American Journal of Kidney Diseases*, 77(2), 282–292.

Mallamaci, F., & Zoccali, C. (2022). Detecting and Treating Lung Congestion with Kidney Failure. *Clinical Journal of the American Society of Nephrology*.

Mount, D. B. (2020). Fluid and electrolyte disturbances in chronic kidney disease. *Medical Clinics of North America*, 104(4), 649–660.
Palmer, B. F., & Clegg, D. J. (2022). Hyperkalemia across the continuum of kidney function. *Clinical Journal of the American Society of Nephrology*, 15(10), 1555–1561.
PERNEFRI. 11th report Of Indonesian renal registry 2021. Indones Ren Regist. 2021;14–5.
Rossignol, P., Legrand, M., Kosiborod, M., & Pitt, B. (2021). Emergency management of severe hyperkalemia: Guideline for best practice and opportunities for the future. *Pharmacology Research & Perspectives*, 9(1), e00699.
Shigeo, A. (2023). Fluid Management Strategies for Pulmonary Edema: Balancing Risks and Benefits. *Journal of Clinical Respiratory Medicine*, 7(2), 145
Sterns, R. H. (2021). Disorders of plasma sodium—causes, consequences, and management. *New England Journal of Medicine*, 372(1), 55–65.
Sterns, R. H., & Silver, S. M. (2020). Complications and management of hyponatremia: The role of dialysis. *Seminars in Dialysis*, 33(1), 41–46.
Wang, H., & Hu, J. (2020). Management of hyperkalemia in patients with chronic kidney disease. *International Journal of Nephrology and Renovascular Disease*, 13, 233–243.