Case Report

Chronic kidney disease in young age: A case report of early diagnosis and comprehensive management

Randolph Serep Marantuan[1], Theza Elizianno Andrew Pellondo'u[2], Ganda Pariama[3]

From [1]Anesthesiologist, Department of Anesthesiology, Faculty of Medicine, Universitas Kristen Indonesia, [2]Forensic, Department of Forensic Medical Science and Legal Medicine, Universitas Kristen Indonesia, [3]Neurologist, Department of Neurology, Universitas Kristen Indonesia, Jakarta, Indonesia

ABSTRACT

Chronic kidney disease (CKD) is characterized by progressive and irreversible deterioration of kidney function. Although it is more commonly found in older individuals, CKD can also occur at a young age and may result in significant long-term complications. A 17-year-old female with Stage 5 CKD undergoing regular hemodialysis presented with shortness of breath for 2 days, difficulty sleeping, cough with initially pink-colored sputum, fever, and decreased urine output. Physical examination revealed bilateral conjunctival pallor and bilateral rhonchi. Laboratory examination showed elevated urea (99 mg/dL) and creatinine (6.77 mg/dL), decreased hemoglobin (10.4 g/dL) and hematocrit (31.7%), as well as decreased bicarbonate levels (17.6 mmol/L), suggesting metabolic acidosis. Renal ultrasonography demonstrated bilateral small kidneys with increased parenchymal echogenicity and indistinct corticomedullary differentiation, consistent with a chronic renal process. Chest radiography revealed cardiomegaly. The patient was managed with regular hemodialysis, fluid management, antihypertensive therapy, and supportive treatment. This case highlights the occurrence of advanced CKD in a young patient and its associated complications, including respiratory symptoms, anemia, metabolic acidosis, severe hypertension, and cardiomegaly.

Key words: Anemia, Chronic kidney disease, Hemodialysis, Hypertension, Young patient

Chronic kidney disease (CKD) is a pathological condition characterized by a gradual, progressive, and permanent decline in kidney function. In this condition, the kidneys are unable to perform their functions properly, particularly in maintaining fluid and electrolyte balance, which may result in elevated levels of urea in the blood. CKD is generally defined as a condition in which the estimated glomerular filtration rate is <60 mL/min/1.73 m² [1-4]. Globally, CKD is estimated to affect approximately 10–16% of the world's population. In Indonesia, the prevalence of CKD has also increased over the past several years. According to data from the 2018 Basic Health Research, the prevalence of CKD increased from 2% in 2013 to approximately 3.8% in 2018. This increase has occurred not only among older adults but has also begun to be observed in younger populations [5-9].

The risk of developing CKD also increases with age. For example, the prevalence is approximately 4% among individuals younger than 40 years but can reach up to 47% among those aged 70 years and older. In general, CKD is more commonly found in older populations due to degenerative processes and the higher prevalence of chronic diseases, such as hypertension and diabetes mellitus [1,3,5,10,11]. In addition, risk factors, such as hypertension, obesity, the use of nephrotoxic medications, and delays in the diagnosis and management of kidney disease may accelerate the development of kidney damage at a young age [12].

CKD at a young age is an important concern because it can have long-term impacts on the patient's quality of life, including the risk of cardiovascular complications, metabolic disorders, such as anemia, electrolyte disturbances and abnormalities in bone metabolism, as well as the need for kidney replacement therapy, such as hemodialysis or kidney transplantation at a productive age. Therefore, early identification, evaluation of risk factors, and appropriate management are very important to prevent disease progression and more severe complications. In addition, CKD at a young age often does not show specific symptoms in the early stages, so the diagnosis is often established late. Therefore, this case report is expected to provide a clinical description of CKD at a young age and emphasize the importance of

Access this article online	
Received - 02 September 2026 **Initial Review** - 18 September 2026 **Accepted** - 14 October 2026	**Quick Response code**
DOI: ***	

Correspondence to: Randolph Serep Marantuan, Department of Anesthesiology, Faculty of Medicine, Universitas Kristen Indonesia, Jakarta, Indonesia. E-mail: randolph.marantuan@uki.ac.id

© 2026 Creative Commons Attribution-NonCommercial 4.0 International License (CC BY-NC-ND 4.0).

early detection and appropriate management to prevent disease progression [13].

Based on the aforementioned background, this case report aims to describe the clinical presentation, risk factors, and management of CKD in a patient at a young age. It is expected to provide a better understanding of the characteristics and management of CKD in this age group.

CASE REPORT

A 17-year-and-6-month-old female patient presented to the Emergency Department of RSUD CAM Bekasi on February 10, 2026, with a chief complaint of shortness of breath for 2 days before admission. She also reported difficulty sleeping for 1 day, coughing with pink-colored sputum for 1 day, and fever. The patient initially experienced a dry cough, followed by pink-colored sputum that subsequently became white. She developed fever that night. The patient had undergone hemodialysis at 12:00 PM, and after completing the session at 5:00 PM, she experienced worsening shortness of breath. Chest pain, dizziness, rhinorrhea, nausea, vomiting, and numbness were denied. Urine output was minimal, while bowel movements were normal. She was under routine follow-up with an internal medicine specialist. In September 2025, the patient first developed bilateral leg swelling. She was taken to a primary healthcare facility, where she received medication and was referred to RSCM. However, the edema resolved after medication, and the family did not proceed with the referral. On October 21, 2025, the patient developed severe weakness and was taken to the Emergency Department. Laboratory examination revealed hemoglobin of 5.8 g/dL, potassium of 6.2 mmol/L, and urea of 227 mg/dL. Radiological examination demonstrated bilateral chronic kidney changes, and the patient was subsequently diagnosed with Stage 5 CKD.

The patient had a history of hypertension, anemia, and Stage 5 CKD. She had been receiving hemodialysis for approximately 3 months on a regular schedule every Tuesday and Friday, with each session lasting 4.5 h.

On physical examination, the patient appeared severely ill. Her Glasgow Coma Scale (GCS) score was E4M6V5, and she was fully conscious (*compos mentis*). Her body weight was 38 kg, and her height was 145 cm. The patient's body temperature was 36°C, blood pressure was 127/95 mmHg, pulse rate was 90 beats/min, and respiratory rate was 21 breaths/min. Oxygen saturation was 100% while receiving oxygen through a non-rebreathing mask (NRM) at 10 L/min. On cardiac examination, no visible apical impulse was observed on inspection. On palpation, the apical impulse was palpable at the fifth left intercostal space along the midclavicular line. Percussion revealed the right cardiac border at the fourth intercostal space along the right parasternal line and the left cardiac border at the fifth intercostal space along the left midclavicular line. Heart sounds I and II were regular, with no murmurs or gallops. A central dialysis line (CDL) was in place in the upper extremity region. Both upper and lower extremities were warm, with capillary refill time of <2 s and no edema.

Laboratory investigations are mentioned in Table 1. Abdominal ultrasonography (October 23, 2025) showed that the liver was not enlarged, with a smooth surface, normal echoparenchyma and vascularity, and no space-occupying lesion. The bile ducts and portal vein were not dilated. The gallbladder had a normal shape and size, with no wall thickening and a smooth surface. No gallstones or sludge were observed. The pancreas was normal in shape and size, with homogeneous parenchyma and no dilatation of the pancreatic duct. The spleen was normal in shape and size, with normal hilum and echoparenchyma. Both kidneys were small in size, with increased parenchymal echogenicity. The corticomedullary differentiation and renal sinus were indistinct, and no renal stones were observed bilaterally. The urinary bladder was normal in size, with a thickened and irregular wall, and no stones were observed. The uterus was normal in size, with homogeneous parenchyma. Minimal ascites was observed in the lower abdomen. The ultrasonographic impression was bilateral chronic kidney changes, minimal ascites, and cystitis.

Thyroid Ultrasonography (December 24, 2025) of the right and left thyroid lobes and isthmus showed normal

Table 1: Laboratory examination (February 25, 2026)

pH	7,409—Normal (N 7,35–7,45)
PCO_2	27,3 mmHg—Decreased (hypocapnia) (N 35–45 mmHg)
PO_2	156,4 mmHg—Increased (N 83–108 mmHg)
BE blood	−5,8 mmol/L—Decreased (N−2–3)
BE ecf	−7,5 mmol/L—Decreased (N−2–3)
HCO_3	17,6 mmol/L—Decreased (N 22–26)
Std HCO_3 (SBC)	19,6 mmol/L—Decreased (N 22–26)
TCO_2	18,5 mmol/L—Decreased (N 23–27)
O_2 Saturation (SO_2%)	99,5%—Slightly increased (N 95–98)
$AaDO_2$	383,1 mmHg—Markedly increased (N rendah)
Ureum	99 mg/dL—Increased (N 20–40 mg/dL)
Creatinine	6,77 mg/dL—Increased (N 0,5–1,5 mg/dL)
Hemoglobin	10,4 g/dL—Decreased (N 12–14 g/dL)
Hemoglobin (AGD)	7,5 g/dL—Decreased
Hematocrit	31,7%—Decreased (N 37–47%)
Leukocyte	10,3 ribu/µL—Slightly increased (N 5–10 ribu/µL)
Platelets	277 ribu/µL—Normal (N 150–400 ribu/µL)
Sodium (Na)	138 mmol/L—Normal (N 135–145 mmol/L)
Potassium (K)	4,3 mmol/L—Normal (N 3,5–5,0 mmol/L)
Chloride (Cl)	97 mmol/L—Normal (N 94–111 mmol/L)
Random blood glucose	74 mg/dL—Normal (N 60–110 mg/dL)

size and homogeneous parenchyma, with no nodules or masses identified. In the bilateral cervical, submandibular, and supraclavicular regions, soft tissue was observed in the bilateral cervical and submandibular subcutaneous areas, with minimal surrounding fluid collection. No oval hypoechoic lesions were observed in the bilateral cervical regions. The impression was a thyroid gland of normal size with homogeneous parenchyma and no nodules or masses.

Chest radiography (January 21, 2026) was performed in the AP view with adequate inspiration and examination conditions. The bronchovascular markings were normal, and both costophrenic angles were sharp. The diaphragm was flattened. The cardiothoracic ratio was >0.56. The skeletal structures were intact. The tip of the CDL was located at the level of T9. The radiological impression was: (1) Lungs within normal limits; (2) Cardiomegaly; (3) Tip of the CDL located at the T9 level.

Based on the clinical history, laboratory findings, imaging findings, and established need for maintenance hemodialysis, the patient was diagnosed with Stage 5 CKD on hemodialysis.

The available documentation does not establish the underlying etiology of the patient's Stage 5 CKD. Given her young age, identification of the cause would be clinically important. The available record does not document a systematic evaluation for congenital or structural renal abnormalities, hereditary kidney disease, glomerular disease, nephrotic syndrome, autoimmune disease, recurrent urinary tract infection, or other potential causes of advanced CKD.

Pharmacological management included omeprazole, ondansetron, furosemide, nicardipine, paracetamol, cefoperazone, candesartan, nifedipine OROS, clonidine, Channa striata extract, and inhaled budesonide and salbutamol.

Non-pharmacological management included intensive monitoring of vital signs, body weight, fluid balance, electrolyte status, and laboratory parameters. Hemodynamic monitoring was performed during hemodialysis. Fluid intake was restricted according to tolerance and urine output. The patient was prescribed a diet restricted in sodium, potassium, and phosphorus with adequate protein intake for patients undergoing hemodialysis. Infection prevention, maintenance of dialysis access sterility, psychosocial support, and patient and family education were also provided.

The patient was admitted to the Emergency Department (February 10, 2026) with worsening shortness of breath following hemodialysis. Her previous medical history included Stage 5 CKD on hemodialysis for approximately 3 months and hypertension. A CDL was present in the right clavicular region, and routine hemodialysis was performed every Tuesday and Friday. Her previous medications included sodium bicarbonate 500 mg 3 times daily, folic acid 1 mg 3 times daily, sucralfate suspension 3 times daily, candesartan 16 mg once daily in the afternoon, codeine 10 mg twice daily, rebamipide 100 mg 3 times daily, Osteocal 3 times daily, vitamin B12 50 mcg 3 times daily, cefixime 200 mg twice daily, Adalat OROS 30 mg once daily at night, omeprazole 20 mg once daily, and furosemide, two tablets in the morning. On examination, the patient's body weight was 38 kg, height was 145 cm, and body mass index (BMI) was 18.1 kg/m², categorized as normal weight. The patient appeared moderately ill and had a GCS score of E4M6V5. Her blood pressure was 181/126 mmHg, heart rate was 122 beats/min, respiratory rate was 22 breaths/min, oxygen saturation was 90%, and body temperature was 36.6°C.

On February 11, 2026, at 08:00, the patient complained of severe shortness of breath. She appeared moderately ill and remained conscious with a GCS score of E4M6V5. Her blood pressure was 225/130 mmHg, heart rate was 108 beats/min, respiratory rate was 22 breaths/min, oxygen saturation was 95%, and body temperature was 36.6°C. Bilateral conjunctival pallor was noted. Pulmonary auscultation revealed bilateral rhonchi, and epigastric tenderness was present. At 15:00, the patient continued to experience severe shortness of breath and became restless. Her condition appeared severely ill, with a GCS score of E4M6V5. Her blood pressure was 213/155 mmHg, heart rate was 141 beats/min, respiratory rate was 32 breaths/min, oxygen saturation was 94% while receiving oxygen through an NRM at 15 L/min, and body temperature was 36.5°C. Bilateral conjunctival pallor, bilateral rhonchi, and epigastric tenderness remained present.

Laboratory examination performed on February 11, 2026, showed a hematocrit of 28.0%, which was decreased. Hemoglobin was 6.9 g/dL and was also decreased. Blood base excess was 9.5 mmol/L and extracellular fluid base excess was 9.4 mmol/L, both increased. HCO_3 was 32.6 mmol/L, which was increased. Oxygen saturation was 99.3%, PO_2 was 167.3 mmHg, pH was 7.528, indicating alkalosis; standard HCO_3 was 33.3 mmol/L, and TCO_2 was 33.8 mmol/L.

Chest radiography showed lungs within normal limits, cardiomegaly, and the tip of the CDL located at the T9 level. The assessment at this stage was dyspnea in a patient with Stage 5 CKD on hemodialysis. The patient received furosemide injection, 0.9% NaCl infusion, nifedipine 10 mg 3 times daily, Budesma suspension, Adalat OROS 30 mg once daily, nicardipine injection at a rate of 0.5 mg/h, candesartan 16 mg twice daily, clonidine 0.15 mg 3 times daily, ondansetron injection, nitroglycerin injection, and omeprazole injection.

Due to the patient's clinical condition, she was transferred to the intensive care unit (ICU) at 16:30 on February 11, 2026. At 16:30, the patient complained of nausea and vomiting. She appeared severely ill but remained conscious with a GCS score of E4M6V5. Her blood pressure was 222/170 mmHg, heart rate was 108 beats/min, respiratory rate was 22 breaths/min, oxygen saturation was 95%, and body temperature was 36.6°C. Bilateral conjunctival pallor and bilateral rhonchi were

noted, and epigastric tenderness was present. At 21:00, the patient's blood pressure was 241/169 mmHg, heart rate was 127 beats/min, respiratory rate was 20 breaths/min, oxygen saturation was 100%, and body temperature was 36°C. At 23:59, her blood pressure was 200/133 mmHg, heart rate was 108 beats/min, respiratory rate was 29 breaths/min, oxygen saturation was 100%, and body temperature was 36°C. Bilateral conjunctival pallor, bilateral rhonchi, and epigastric tenderness remained present; the assessment in the ICU remained Stage 5 CKD on hemodialysis. Management included ranitidine injection twice daily, omeprazole injection 40 mg twice daily, ondansetron injection 8 mg 3 times daily, furosemide injection at a rate of 20 mg/h, nitroglycerin injection, nicardipine injection (Perdipine) 10 mg/50 cc, candesartan 16 mg orally twice daily, Adalat OROS, clonidine, inhalation therapy every 8 h with Budesma and Ventasal, and continuous positive airway pressure at a setting of 10.

DISCUSSION

Stage 5 CKD requiring maintenance hemodialysis in a 17-year-old patient is clinically unusual and should prompt careful consideration of the underlying cause. In this case, the most important finding supporting chronic rather than acute kidney injury is the imaging evidence of bilaterally small kidneys with increased parenchymal echogenicity and indistinct corticomedullary differentiation. These findings are compatible with chronic structural renal disease. However, they do not identify the etiology of the CKD.

The episode of bilateral leg edema in September 2025 may have represented an early manifestation of renal disease, although the available information is insufficient to determine whether the edema was caused by renal, cardiovascular, nutritional, or another process. The subsequent presentation in October 2025, with severe anemia, hyperkalemia, markedly elevated urea, and imaging evidence of chronic bilateral renal changes, indicates that substantial renal impairment was already present at that time. The rapid recognition of Stage 5 CKD therefore occurred after the disease had already progressed to an advanced stage. In an adolescent with advanced CKD and small kidneys, the differential diagnosis should include congenital or structural kidney abnormalities, hereditary tubulointerstitial or cystic kidney diseases, chronic glomerular disease, reflux or other chronic urinary tract abnormalities, chronic tubulointerstitial disease, and other acquired causes. A definitive etiological diagnosis would require information that is not documented in the present case, such as previous renal function measurements, urinalysis and urine protein assessment, detailed renal and urinary tract imaging, assessment of kidney size and structural abnormalities over time, relevant autoimmune and infectious investigations when clinically indicated, and genetic evaluation when hereditary disease is suspected. Kidney biopsy may also be considered in selected patients when the kidneys are not markedly scarred and when the result would alter management; however, the present case does not provide sufficient information to determine whether biopsy would have been appropriate [14].

The patient's hypertension is an important complication of advanced CKD and may also have contributed to cardiovascular remodeling. The markedly elevated blood pressure documented during the acute admission and the presence of cardiomegaly suggest a substantial cardiovascular burden. However, the available information does not establish whether hypertension preceded the CKD, resulted from the CKD, or contributed to its progression. In a young patient, distinguishing these possibilities is important because severe or longstanding hypertension may itself be a manifestation of an underlying renal disorder [15].

Anemia was another important complication. The patient had a hemoglobin concentration of 5.8 g/dL at the initial October 2025 presentation and 6.9 g/dL on February 11, 2026. On February 25, the laboratory panel reported hemoglobin of 10.4 g/dL, whereas arterial blood gas analysis simultaneously reported hemoglobin of 7.5 g/dL. These discrepant measurements should be verified against the original laboratory records. Regardless of the discrepancy, the longitudinal record clearly documents clinically significant anemia in the setting of advanced CKD. Anemia in CKD is multifactorial and may involve reduced erythropoietin production, iron deficiency, inflammation, blood loss, nutritional factors, and other causes. The available case does not contain sufficient information regarding iron studies, reticulocyte count, erythropoiesis-stimulating agent therapy, or transfusion history to further characterize the anemia [16,17].

The respiratory presentation was characterized by acute dyspnea, bilateral rhonchi, and initially pink-colored sputum following hemodialysis. In advanced CKD, impaired fluid and sodium excretion can contribute to volume overload and pulmonary congestion. However, the clinical presentation should not automatically be attributed to fluid overload, particularly because the chest radiograph described the lungs as within normal limits. Other cardiopulmonary or infectious causes may therefore need to be considered based on the complete clinical evaluation.

The BMI of 18.1 kg/m² should also be interpreted cautiously. Because the patient was 17 years old, adult BMI categories are not the preferred method for nutritional classification. BMI-for-age and sex-specific percentiles should be used instead. The available report does not provide a BMI-for-age percentile; therefore, classifying the patient as having normal weight based solely on adult BMI criteria would be inappropriate. This distinction is relevant because nutritional status is an important component of care in adolescents with advanced CKD and chronic hemodialysis.

Overall, the central clinical issue in this case is not only the management of Stage 5 CKD but also the

unexplained development of advanced kidney disease at a very young age. The imaging findings establish chronicity but do not establish etiology. Consequently, the principal limitation of this case is the absence of a documented comprehensive etiological work-up. Future evaluation should focus on reconstructing the patient's prior renal history and investigating congenital, hereditary, structural, glomerular, autoimmune, infectious, obstructive, and other potential causes as clinically appropriate.

This case emphasizes the importance of considering CKD in adolescents presenting with non-specific manifestations, such as edema, severe anemia, hypertension, or reduced urine output. Early identification of kidney disease and timely investigation of its underlying cause may provide an opportunity to slow progression and appropriately address complications before kidney function reaches Stage 5.

CONCLUSION

This case reports a 17-year-old female patient with Stage V CKD undergoing regular hemodialysis who presented with shortness of breath, suggesting fluid overload and possible pulmonary congestion due to fluid retention in advanced renal failure. This case emphasizes the importance of multidisciplinary management and the appropriate selection of anesthetic techniques and agents in patients with end-stage CKD to minimize complications and improve patient safety.

REFERENCES

1. Sudoyo AW, Setiyohadi B, Alwi I, Simadibrata M, Setiati S. Buku Ajar Ilmu Penyakit Dalam Edisi v. Indonesia: Pusat Penerbitan Ilmu Penyakit Dalam; 2009. p. 1-25.
2. Lohia S, Vlahou A, Zoidakis J. Microbiome in chronic kidney disease (CKD): An omics perspective. Toxins (Basel) 2022;14:176.
3. Romagnani P, Agarwal R, Chan JC, Levin A, Kalyesubula R, Karam S, *et al*. Chronic kidney disease. Nat Rev Dis Prim 2025;11:8.
4. Kalantar-Zadeh K, Jafar TH, Nitsch D, Neuen BL, Perkovic V. Chronic kidney disease. Lancet 2021;398:786-802.
5. Rizka AS, Kardiwinata MP. lysis of the incidence of chronic kidney disease in individuals with diabetes mellitus in Indonesia: An analysis of data from the 2018 Basic Health Research (Riskesdas). Arch Commun Health 2023;10:632-44.
6. Chen TK, Knicely DH, Grams ME. Chronic kidney disease diagnosis and management: A review. JAMA 2019;322:1294-304.
7. Poznyak AV, Sadykhov NK, Kartuesov AG, Borisov EE, Sukhorukov VN, Orekhov AN. Atherosclerosis specific features in chronic kidney disease (CKD). Biomedicines 2022;10:2094.
8. Xie K, Cao H, Ling S, Zhong J, Chen H, Chen P, *et al*. Global, regional, and national burden of chronic kidney disease, 1990-2021: A systematic analysis for the global burden of disease study 2021. Front Endocrinol (Lausanne) 2025;16:1526482.
9. Meola M. Chronic kidney disease. In: Meola M, editor. Clinical Ultrasound in Nephrology. Cham, Switzerland: Springer Nature; 2026. p. 525-66.
10. Behera TK, Sahoo S, Behera JR, Acharya SS, Saint SK, Acharya A. Clinicoetiological profile of chronic kidney disease in young population in a tertiary care hospital. C J Geriatr Med 2026;18:1-8.
11. Lowe KM, Cruz JB, Jones KM. Complications in patients with chronic kidney disease. Crit Care Nurs Clin 2022;34:395-407.
12. Bikbov B. Global, regional, and national burden of chronic kidney disease, 1990 - 2017 : A systematic analysis for the global burden of disease study 2017. Lancet 2017;395:709-33.
13. Hill NR, Fatoba ST, Oke JL, Hirst JA, Callaghan AO, Lasserson DS, *et al*. Global prevalence of chronic kidney disease - a systematic review and meta-analysis. CKD Glob Preval 2016;11:e0158765.
14. Gropper MA, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Cohen NH, Leslie K. Miller's Anesthesia. 9th ed. Amsterdam: Elsevier; 2024.
15. Aitkenhead AR. Textbook of Anaesthesia. London: Churchill Livingstone Elsevier; 2007. p. 858.
16. Thompson JP, Bhatt S. Renal ultrasound. Ultrasound Clin 2014;9:653-81.
17. Lee C, Kim SH, Kim B. Renal parenchymal disease. In: Kim SH, editor. Uroradiology. Singapore: Springer Nature; 2026. p. 467-500.

Funding: Nil; Conflicts of interest: Nil.

How to cite this article: Marantuan RS, Pellondo'u TE, Pariama G. Chronic kidney disease in young age: A case report of early diagnosis and comprehensive management. Indian J Case Reports. 2026; September 22 [Epub ahead of print].