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End Stage Renal Disease in a 17-Year-Old Boy: A Case Report

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ABSTRACT

Introduction: Chronic Kidney Disease (CKD) in children is asymptomatic in early stages, which makes early diagnosis challenging. However, children with chronic kidney disease can progressively worsen and lead to end-stage renal disease (ESRD), the most severe form of kidney failure. ESRD is a rare condition in children but significantly affects their quality of life. ***Case Presentation:*** A 17-year-old boy went to the emergency department at Wangaya Hospital with a chief complaint a persistent vomiting accompanied by epigastric pain. The patient also reported generalized weakness, decreased appetite, and decreased fluid intake. Physical examination revealed stage 2 hypertension, pale conjunctiva in both eyes, tenderness in the epigastric region, and pallor of the skin. Laboratory investigations showed normochromic normocytic anemia with thrombocytopenia, elevated blood urea nitrogen, also creatinine serum, and hyperkalemia. Initial management included oxygen supplementation and intravenous fluid therapy. The patient was scheduled to receive a transfusion of packed red cells and was given symptomatic medications. The patient was diagnosed with End-Stage Renal Disease and referred for hemodialysis. ***Discussion:*** Chronic Kidney Disease (CKD) is defined by KDIGO as kidney damage or a glomerular filtration rate (GFR) <60 mL/min/1.73 m^2 lasting for ≥3 months. This case describes a 17-year-old boy with ESRD, confirmed by a glomerular filtration rate of 5.21 mL/min/1.73 m^2. The mother's history of Stage V CKD increases the patient's genetic risk, consistent with familial aggregation findings. Management includes supportive therapies, antihypertensives, and urgent referral for hemodialysis due to end-stage renal disease. Therefore, early recognition, comprehensive management, and timely planning for renal replacement therapy, either dialysis or kidney transplantation, are crucial to improve long-term outcomes. ***Conclusion:*** ESRD at such a young age reflects the progressive nature of chronic kidney disease and its profound impact on growth, development, and quality of life, emphasizing the importance of comprehensive care and early dialysis planning.

Keywords: end-stage renal disease; glomerular filtration rate; hemodialysis

INTRODUCTION

Chronic kidney disease (CKD) is a significant cause of morbidity and mortality, with its prevalence steadily increasing worldwide. It is projected that by the year 2030, approximately 5.4 million patients will require kidney transplantation. The Global Burden of Disease 2017 study ranks CKD as the 12th leading cause of death globally[1]. Comprehensive national data regarding the incidence of CKD in Indonesia, particularly among children, remains incomplete. However, the Indonesian Health Survey 2023 reported a prevalence of 0.18% among individuals over 15 years of age [2].

The most common cause of CKD in children is structural abnormalities. Congenital malformations such as renal hypoplasia, renal dysplasia (11%), and obstructive uropathy (22%) are the primary etiologies in children under 5 years old [3]. But mostly children older than 5 years, CKD is caused by acquired diseases, such as glomerulonephritis or inherited disorders like Alport syndrome[4].

The natural progression of CKD in its early stages is highly variable and often challenging to predict. Several risk factors influence the progression, including hypertension, proteinuria, genetics, race, age, and sex. The management of CKD aims to slow disease progression, manage complications effectively, support growth and development, also prepare for renal replacement therapies such as dialysis or kidney transplantation when necessary[5]. Therefore, early detection and timely intervention are essential to improving the quality of life for children with end-stage chronic kidney disease.

CASE PRESENTATION

A 17-year-old boy was brought by his parents to the emergency department with the main complaint of vomiting. Nothing made the symptoms better or

worse. He also experienced epigastric pain that felt like cramps. The patient was then taken to the emergency department at Wangaya General Hospital and was given treatment. However, his symptoms did not improve, he felt nauseous and experienced persistent vomitting with continuous epigastric pain, so he was brought back to Wangaya General Hospital emergency. Over the past month, the patient has felt weak, with a decreased appetite and reduced fluid intake. He has also noticed a decrease in urine output over the last week and had one episode of loose stool without blood or mucus. He denies fever, presence of blood in urine, pain during urination, back pain, or swelling. The patient admits consume packaged and carbonated beverages frequently.

The physical examination revealed a moderately ill. The patient with a body weight 60 kg and body height 174 cm showed a good nutritional status. He was alert, blood pressure 150/90 mmHg, pulse rate 92 beats per minute, respiratory rate 20 breaths per minute and oxygen saturation was 95% on room air. We found that pale conjunctiva in both eyes also pale skin and tenderness in the epigastric region.

Laboratory tests revealed the following results hemoglobin 4.6 g/dL, MCV 86.8 fL, MCH 30.5 pg, and PLT 84 x 10^3/µL. The peripheral blood smear showed a normochromic normocytic anemia along with thrombocytopenia. Kidney function tests indicated a blood urea level of 314 mg/dL and creatinine at 23.4 mg/dL. The Glomerular Filtration Rate (GFR), calculated using the Schwartz formula, was 5.21 mL/menit/1.73 m^2. Electrolyte analysis showed a potassium level of 6.2 mmol/L. Arterial blood gas analysis revealed a pH 7.22, PCO2 28 mmHg, PO2 35 mmHg, cHCO3 11 mmol/L, base excess -16 mmol/L, standard bicarbonate concentration at 12 mmol/L and oxygen saturation 59%.

The examination results showed that the patient suffers stage 2 hypertension and severe normochromic normocytic anemia, based on complete blood count and peripheral blood smear findings. Elevated blood urea and creatinine levels reveal uremia, accompanied by an electrolyte imbalance characterized by hyperkalemia. The arterial blood gas analysis results revealed acidosis metabolic.

The patient was initially treated with oxygen therapy via nasal cannula and intravenous fluid therapy with Ringer's lactate. Transfuse packed red cells (PRC) was scheduled 3 times (250 cc per day) with a premedication of Lasix. Symptomatic medications were administered including ranitidine 50 mg twice daily IV, ondansetron 4 mg three times daily IV and antihypertensive treatment with captopril 25 mg three times daily orally.

DISCUSSION

According to KDIGO, Chronic Kidney Disease (CKD) is defined as the presence of kidney damage or impaired kidney function lasting for three months or more. This includes a decrease in the glomerular filtration rate (GFR) below 60 mL/min/1.73 m^2, or indicators of kidney damage such as albuminuria, abnormal urine findings, electrolyte imbalances, or structural abnormalities of the kidneys detected through imaging studies or kidney biopsy[5]. The etiology of chronic kidney disease in children typically differ from those in adults. In children, chronic kidney disease is most often due to congenital and structural abnormalities, such as renal dysplasia, obstructive uropathy, and hereditary kidney disorders [3], [5].

The pathophysiology of chronic kidney disease begins with kidney damage caused by a reduction in the number of functional nephrons, which triggers hyperfiltration. This process increases pressure within the glomerulus, leading to damage of the endothelium and mesangium, and subsequently stimulates interstitial fibrosis and glomerulosclerosis [6]. As a results, kidney function deteriorates, characterized by a decreased glomerular filtration rate, which leads to the accumulation of metabolic waste products in the bloodstream. This impaired function also causes disturbances in electrolyte balance, acid-base homeostasis, fluid retention, and a reduction in the production of erythropoietin hormone, resulting in anemia [4].

The prevalence of chronic kidney disease in children ranges from 15 to 75 cases per million children. Comprehensive national data on the incidence of chronic kidney disease in Indonesia, particularly in children, is not yet fully available. However, Indonesian Health Survey 2023 reported that the prevalence of chronic kidney disease in children over 15 years old in Bali province was 0.19% [2]. A study conducted in 2017 reported that across 14 teaching hospitals with pediatric nephrology consultants, a total of 212 children were diagnosed with kidney failure and received renal replacement therapy [7].

The clinical manifestation that appeared in this patient are vomiting and pain in the epigastric region. Other case reports also indicate that patients with chronic kidney disease predominantly experience gastrointestinal symptoms, such as nausea and vomiting, which are suspected to be caused by uremia as well as congenital anomalies of the kidneys and urinary tract[8]. The accumulation of uremic toxins can stimulate the vomiting center in the brain and irritate the stomach, causing patients with chronic kidney disease to experience persistent vomiting and pain in the epigastric region [9].

Additionally, the patient has a family history of a mother diagnosed with Stage V CKD that undergoing hemodialysis. According to a study, individuals with a first-degree relative affected by chronic kidney disease have a threefold higher risk of developing the condition themselves. This finding is supported by earlier research focusing on familial aggregation of chronic kidney disease, particularly in its advanced stages or kidney failure. The risk of kidney failure among first-degree relatives has been estimated to be nine times higher in African and American populations [10].

The physical examination revealed stage II hypertension. Hypertension is an early indicator of

chronic kidney disease in children. The prevalence of hypertension in chronic kidney disease reaches up to 80% in the end stages [4]. Hypertension caused by fluid overload and activation of the renin-angiotensin-aldosterone system [11]. Anemia in children with chronic kidney disease often presents with symptoms such as weakness, fatigue and pallor accompanied by anemic conjunctiva. The complete blood count of this patient showed normochromic normocytic anemia, which is the most common form of anemia in chronic kidney disease patients. The anemia occurs due to inflammatory cytokines inhibiting erythropoiesis in the bone marrow, decreased erythropoietin production in the kidneys, and increased hepcidin production in the liver. Chronic kidney disease is often accompanied by chronic inflammation that increases the production of inflammatory cytokines such as IL-6, which disrupt iron metabolism and suppress erythropoietin production. The pathophysiology of anemia in CKD can also result from the accumulation of uremic toxins and oxidative stress, which induce changes in the erythrocyte membrane and cytoskeleton, promoting hemolysis and shortening the lifespan of red blood cells [12].

The patient also had electrolyte disturbances in the form of hyperkalemia, which can occur due to the kidneys' inability to excrete potassium [4]. Patients with end stage renal disease have a ninefold higher risk of developing hyperkalemia compared to those in the early stages [13]. Elevated serum urea and creatinine levels in the patient indicate uremia. Uremia is a clinical condition resulting from the accumulation of uremic toxins in the blood. The decline in kidney function reduces the effective excretion of urea, leading to its accumulation and toxicity in the body [9], [14]. The kidney function can be seen by the glomerular filtration rate (GFR) that calculated using the Schwartz formula. In this case, the GFR is approximately 5.21 mL/min/1.73 m^2. This finding confirms the patient is in stage V chronic kidney disease, indicating kidney failure [5]. The glomerular filtration rate (GFR) in this patient is approximately 5.21 mL/min/1.73 m^2. This indicates that the patient has reached end stage of chronic kidney disease, also known as kidney failure.

Furthermore, the patients with end-stage chronic kidney disease, the kidneys lose almost all of their ability to perform excretory and acid-base regulatory functions. The damaged kidneys are unable to effectively excrete acid, leading to acid accumulation in the blood, which causes metabolic acidosis. Blood gas analysis of the patient revealed compensated metabolic acidosis [15]. In this case, result of blood gas analysis showed compensated metabolic acidosis.

The patient received supplemental oxygen and intravenous fluid therapy to stabilize the condition. However, careful monitoring of fluid administration is necessary to prevent fluid overload. In addition, the patient was given symptomatic intravenous medications such as ranitidine and ondansetron. Ranitidine functions by reducing gastric acid secretion through the inhibition of H2 receptors, thereby decreasing the production of stomach acid [16], [17]. Ondansetron acts by blocking 5-HT3 receptors, which play a role in triggering the reflex of nausea and vomiting, thereby preventing the transmission of signals to the vomiting center in the brain [18].

Captopril is an angiotensin-converting enzyme (ACE) inhibitor, was administered as antihypertensive therapy. The aim is to reduce blood pressure, preventing progressive renal impairment and cardiovascular complications. Management of hypertension in children with chronic kidney disease should be conducted comprehensively with a target blood pressure below the 90th percentile according to age, gender, and body height[19]. The patient also scheduled to receive a packed red cell (PRC) transfusion because of the severe anemia, which must be conducted under strict monitoring [12].

The patient progressed to end stage renal disease (ESRD), requiring the initiation of renal replacement therapy. It is indicated when conservative management and pharmacologic interventions fail to maintain adequate renal function, fluid balance, or metabolic stability [20], [21]. In children, dialysis initiation is recommended when the estimated glomerular filtration rate (eGFR) falls below 10–15 ml/min/1.73 m^2 [22]. The patient was referred for hemodialysis in Prof. Ngoerah Hospital.

In children, hemodialysis poses unique challenges, including vascular access difficulties, hemodynamic instability, and the need for individualized dialysis prescriptions to accommodate growth and developmental considerations [21]. Despite these challenges, hemodialysis remains a life-sustaining therapy, effectively removing uremic toxins, correcting electrolyte imbalances and maintaining homeostasis until definitive management such as renal transplantation can be considered [23].

Therefore, early recognition, comprehensive management, and timely planning for renal replacement therapy either dialysis or kidney transplantation are crucial to improve long-term outcomes also reduce morbidity and mortality in pediatric patients [5].

CONCLUSION

This case highlights the serious burden of ESRD in a 17 years old male. ESRD at such a young age reflects the progressive nature of chronic kidney disease and its profound impact on growth, development, and quality of life, emphasizing the importance of comprehensive care and early dialysis planning.

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