Oshada; Health Journal
https://nawalaeducation.com/index.php/O/index
Volume 2 Number 3, June 2025
e-ISSN : 3047-017X
DOI : https://doi.org/10.62872/ean83813

Severe Anemia Normocytic Normochromic EC CKD

Elvi Fitraneti [1], Alimurdianis [2]
Faculty of Medicine, Universitas Baiturrahmah, Indonesia [1], Faculty of Medicine, Universitas Baiturrahmah, Indonesia[2]
e-mail: * elvi_fitraneti@fk.unbrah.ac.id[1]
alimurdianis@fk.unbrah.ac.id[2]

Input	: May 22, 2025	Revised	: June 20, 2025
Accepted	: June 25, 2025	Published	: June 30, 2025

ABSTRACT

This study aims to clinically describe a case of severe normocytic normochromic anemia due to chronic kidney disease (CKD) and contribute to strengthening medical understanding of the relationship between anemia and kidney dysfunction. The method used is a descriptive case study with a clinical observation approach and literature review. Data were obtained from a 58-year-old male patient who came to the Emergency Department of M. Natsir Solok Regional Hospital complaining of weakness, pallor, joint pain, and decreased appetite. Laboratory examination results showed a hemoglobin level of 5.8 g/dL and creatinine of 8.13 mg/dL, indicating severe anemia due to stage V CKD. The patient underwent PRC transfusion therapy, intravenous fluids, and folate and antihypertensive administration. During four days of treatment, the patient's condition showed gradual clinical improvement. The discussion emphasized the importance of early detection of normocytic normochromic anemia in CKD patients because decreased erythropoietin production is a major factor. This study concluded that appropriate diagnosis and treatment of anemia due to CKD not only improve the clinical prognosis but also play a crucial role in preventing systemic complications and improving the patient's overall quality of life.

Keywords: Normocytic Normochromic Anemia; Chronic Kidney Disease (CKD), Erythropoietin, Hemoglobin, PRBC Transfusion, Case Study, Anemia Therapy.

INTRODUCTION

Anemia is a medical condition characterized by reduced hemoglobin levels in the body. Hemoglobin itself is an iron-containing metalloprotein found in red blood cells, functioning to transport oxygen from the lungs to all body tissues. When hemoglobin levels decrease, oxygen transport is disrupted, which then causes various clinical symptoms such as weakness, fatigue, lethargy, palpitations, increased pulse, and complaints such as tinnitus. A person is categorized as suffering from anemia if their blood hemoglobin level is less than 13 g/dL in men, less than 12 g/dL in women, and below 11 g/dL during

pregnancy, which is considered an abnormal condition. Based on data from the 2018 Basic Health Research (Riskesdas), the prevalence of anemia in Indonesia is quite high, reaching 48.9%. This figure is more worrying in the age group 5 to 24 years old at 84.6%, while in the age group 25–34 years old it is 33.7%, and 35–44 years old it is 33.6%. At ages 45–54, the prevalence is again high at 84.6%. Data from the Indonesian Ministry of Health (2018) states that the prevalence of anemia among adolescents aged 15–24 is 32%, meaning that approximately 3–4 out of every 10 adolescents experience anemia. Furthermore, the prevalence of anemia among women (27.2%) is also higher than among men (20.3%).

Anemia is a medical condition characterized by decreased hemoglobin levels in the blood, which impacts the body's oxygen-carrying capacity. One common form of anemia in patients with chronic kidney disease (CKD) is normocytic normochromic anemia. This form of anemia is characterized by normal red blood cell size (normocytic) and normal hemoglobin levels per red blood cell (normochromic), but a decreased total erythrocyte count (Fitraneti & Alimudiarnis, 2023). According to 2018 Basic Health Research (Riskesdas), the prevalence of anemia in Indonesia reached 48.9%, with the age groups 5–24 and 45–54 showing the highest incidence rates, at 84.6% (Basic Health Research, 2018). This prevalence is higher in women (27.2%) than in men (20.3%), and is particularly concerning among adolescents and women of childbearing age (Wahyuni, 2024).

Etiologically, anemia can be caused by various factors, including impaired red blood cell production, blood loss, and increased red blood cell destruction. In patients with CKD, the primary cause of anemia is decreased erythropoietin production by the kidneys. Erythropoietin is a crucial hormone that stimulates the bone marrow to produce red blood cells (Yuniarti, 2021). Chronic kidney dysfunction causes this hormone level to drop drastically, inhibiting erythropoiesis and leading to normocytic, normochromic anemia (Bitin, 2022).

Previous studies have confirmed that anemia is one of the most common and serious complications in patients with advanced CKD (Yuniarti, 2021). Furthermore, Bitin (2022) found a significant association between anemia and reduced quality of life in hemodialysis patients, reinforcing the importance of early diagnosis and treatment of anemia in this population.

Bakta (2017) also showed that anemia in CKD not only impacts clinical symptoms such as weakness, pallor, and tachycardia, but also increases the workload of the heart and increases the risk of cardiovascular complications such as heart failure and myocardial ischemia.

Other literature also highlights the importance of classifying anemia based on erythrocyte indices (MCV and MCH) in aiding diagnosis. The WHO classifies anemia into three categories based on hemoglobin levels: mild, moderate, and severe. In normocytic, normochromic anemia, MCV and MCH values remain within normal limits (MCV 80–95 fL and MCH 27–34 pg), despite a decreased total red blood cell count (Nugraha & Yasa, 2022).

Specifically, iron deficiency anemia remains the most common cause of anemia globally, particularly in women of childbearing age, children, and the

elderly (Fitriany & Saputri, 2018; Novita Sari, 2020). On the other hand, hemolytic, megaloblastic, and malignant anemias are also important to recognize due to their distinct pathophysiological mechanisms (Rouli & Amalia, 2016).

Thus, a literature review indicates that normocytic, normochromic anemia in CKD patients is a complex condition, requiring a comprehensive diagnostic approach and coordinated therapeutic interventions. Management of this anemia is aimed not only at increasing hemoglobin levels but also at improving quality of life and reducing the risk of broader systemic complications (Fitraneti & Alimudiarnis, 2023).

Clinically, one of the main signs of anemia is pallor, which occurs due to reduced blood volume, low hemoglobin levels, and vasoconstriction as the body's response to optimize oxygen delivery. In addition to pallor, other common symptoms include tachycardia, heart murmurs, headaches, dizziness, and blurred vision. In cases of severe anemia, more serious systemic symptoms can occur, such as lethargy, cognitive impairment, and cardiovascular complications such as heart failure, arrhythmias, myocardial infarction, and angina pectoris. One type of severe anemia is normocytic normochromic anemia, which can occur due to chronic diseases such as Chronic Kidney Disease (CKD), where decreased erythropoietin production plays a significant role in reducing red blood cell production.

This case report aims to provide a deeper understanding of severe normocytic normochromic anemia caused by chronic kidney disease (CKD). This research is also expected to serve as a source of information and scientific reference for medical students and healthcare professionals in recognizing, analyzing, and managing severe normocytic normochromic anemia, particularly in patients with a history of CKD.

METHODOLOGY

This article uses a descriptive method with a literature review and clinical observation approach. Data were obtained through a search of scientific literature from books, relevant national journals, and observations of traumatic hyphema cases during the clinical clerkship in the Ophthalmology Department of M. Natsir Solok Hospital. Primary data was not collected directly from patients; instead, analysis was conducted by comparing theories and literature review results with clinical findings observed during practice. This article uses a descriptive case study method in a patient with severe normocytic normochromic anemia due to CKD, based on direct observation, clinical data analysis, and relevant literature searches. The purpose of this method is to strengthen academic and clinical understanding of traumatic hyphema as an ocular emergency, as well as to contribute to medical education, particularly in early recognition and appropriate management of this condition.

RESULTS AND DISCUSSION

Epidemiology and Vulnerable Populations

Anemia is a very common disorder found both in clinics and in the field. It is estimated that more than 30% of the world's population, or 5,000 million people, suffer from anemia. The prevalence of anemia in Indonesia according to population groups is most common in pregnant adult women with a prevalence of 50-70%, followed by non-pregnant adult women 30-40%, school-age children 25-35%, and adult men 20-30%. Based on data from the 2018 Basic Health Research (Riskesdas), the prevalence of anemia in Indonesia was quite high at 48.9%, in the age range of 5 to 24 years at 84.6%, while the age range of 25 to 34 years was 33.7%, and the age of 35 to 44 years was 33.6% and anemia sufferers aged 45 to 54 years was 84.6%.

Etiology and Risk Factors

1. Anemia Due to Impaired Red Blood Cell Production

Several causes of anemia can be classified based on the mechanism by which red blood cell formation or loss occurs.

a. Iron deficiency is the most common cause of anemia worldwide. This usually occurs due to a diet low in iron or chronic blood loss, such as gastrointestinal bleeding or excessive menstruation.
b. Vitamin B12 or folic acid deficiency disrupts DNA synthesis during erythropoiesis, resulting in megaloblastic anemia, characterized by large, immature red blood cells.
c. Bone marrow disorders, such as aplastic anemia, leukemia, or myelofibrosis, can directly inhibit red blood cell formation due to hematopoietic dysfunction.
d. Chronic kidney disease (CKD) can cause anemia due to decreased production of erythropoietin, a hormone that stimulates the bone marrow to produce red blood cells.
e. Chronic inflammatory diseases, such as tuberculosis, systemic lupus erythematosus, rheumatoid arthritis, and various types of cancer, can cause anemia (anemia of chronic disease) through systemic inflammation that inhibits iron metabolism and the erythropoietin response.

2. Anemia Due to Blood Loss (Hemorrhage)

Blood loss is a major cause of anemia due to a decrease in the number of red blood cells circulating in the body. Blood loss can be acute or chronic, depending on the cause and duration of the bleeding.

a. Acute bleeding occurs suddenly and in large amounts, such as from physical trauma, surgery, or blood vessel rupture. This condition can cause a rapid decrease in blood volume and requires immediate treatment.
b. Chronic bleeding, even if it occurs in small amounts but continues, can cause iron deficiency, leading to anemia. Some common causes of chronic bleeding include:

1) Gastrointestinal disorders, such as peptic ulcers, gastritis, colorectal cancer, and hemorrhoids, which often go undetected until anemia develops.
2) Heavy menstruation (menorrhagia), which is common in women of reproductive age and can cause significant iron loss during each menstrual cycle.

3) Intestinal parasitic infections, particularly hookworms, which suck blood from the intestinal wall and are an important cause of iron deficiency anemia in tropical and subtropical regions.

3. Anemia Due to Increased Red Blood Cell Destruction (Hemolysis)

Hemolytic anemia occurs when the rate of red blood cell (erythrocyte) destruction exceeds the body's ability to produce new cells. This hemolysis process can be caused by various mechanisms, including immunological, infectious, and genetic.

a. Autoimmune hemolytic anemia is a condition in which the body's immune system mistakenly recognizes red blood cells as foreign and destroys them. This can occur idiopathically or in association with other autoimmune diseases such as systemic lupus erythematosus.

b. Certain infections, such as malaria caused by Plasmodium spp., damage red blood cells directly during the parasite's life cycle within the red blood cells. Severe infections such as sepsis can also trigger hemolysis through bacterial toxins or microcirculatory disorders.

1) Genetic disorders are also a major cause of hereditary hemolytic anemia. These include:
   Thalassemia, a disorder in the production of globin chains in hemoglobin, causes red blood cells to become fragile and easily destroyed in the spleen.
2) Sickle cell anemia, characterized by abnormally crescent-shaped red blood cells due to mutations in the β-globin gene. These cells rupture easily and block small capillaries, triggering hemolytic crises and pain.
3) G6PD (glucose-6-phosphate dehydrogenase) deficiency, an enzymatic disorder that makes red blood cells susceptible to oxidative stress, especially after exposure to certain drugs, infections, or foods such as fava beans.

Patofisiologi

Anemia occurs when the body lacks red blood cells (erythrocytes) or hemoglobin, the protein in red blood cells that carries oxygen throughout the body. This deficiency prevents the body from receiving enough oxygen, resulting in symptoms such as weakness, dizziness, and paleness. The causes of anemia can be divided into three main mechanisms:

1. Insufficient Red Blood Cell Production
   a. The body is unable to produce enough red blood cells due to a lack of iron, vitamin B12, or folic acid.
   b. An example is iron deficiency anemia, which occurs if we don't eat enough iron-rich foods (such as meat or green vegetables), or if the body can't absorb iron properly.
2. Excessive Destruction of Red Blood Cells
   a. Red blood cells are destroyed faster than the body can produce them.
   b. An example is hemolytic anemia, which can be caused by autoimmune diseases (where the body's immune system attacks red blood cells) or certain infections.
3. Blood Loss

a. Large blood loss, for example due to bleeding (a major wound, heavy menstruation, or bleeding in the digestive tract), causes the red blood cell count to drop drastically.

Hyphema Classification

The most common parameter used to indicate a decrease in red blood cell mass in the body is the hemoglobin (Hb) level. Besides hemoglobin, two other parameters that are also often used are hematocrit and red blood cell count. According to the World Health Organization (WHO), there are hemoglobin level limits used to determine anemia based on age group and gender. Men are said to have normal hemoglobin levels if they are in the range of 13.5 to 18.0 g/dL, while women are in the range of 12.0 to 15.0 g/dL. For children, normal hemoglobin levels range from 11.0 to 16.0 g/dL, and for pregnant women, hemoglobin levels are considered normal if more than 10.0 g/dL. Based on hemoglobin levels, WHO also classifies anemia into three degrees of severity, namely mild anemia with Hb levels between 8.0 to 9.9 g/dL, moderate anemia with Hb levels between 6.0 to 7.9 g/dL, and severe anemia if hemoglobin levels are below 6.0 g/dL. This classification is important to determine the severity of anemia and appropriate treatment.

Another classification for anemia is based on morphological features, such as erythrocyte indices or peripheral blood smears. In this classification, anemia is divided into three groups:

a. Microcytic hypochromic anemia, if MCV <80 fl and MCH <27 pg
b. Normocytic normochromic anemia, if MCV 80-95 fl and MCH 27-34 pg
c. Macrocytic anemia, if MCV >95 fl.

Clinical Manifestations

The symptoms of anemia can be grouped into three main types. First, general symptoms of anemia, or anemia syndrome, arise from organ ischemia and the body's compensatory mechanisms for decreased hemoglobin levels. These symptoms generally appear when Hb is <7 g/dL. These symptoms include weakness, lethargy, fatigue, ringing in the ears, blurred vision, cold feet, shortness of breath, and digestive problems, as well as pale conjunctiva, oral mucosa, palms, and nails. Second, there are specific symptoms based on the type of anemia, such as dysphagia, angular stomatitis, and spoon nails (koilonychia) in iron deficiency anemia; glossitis and neurological disorders in megaloblastic anemia; jaundice and an enlarged spleen and liver in hemolytic anemia; and bleeding and infection in aplastic anemia. Third, symptoms of the underlying disease depend on the cause of the anemia. For example, anemia due to hookworm infection causes abdominal pain and yellowing of the palms, while anemia due to chronic diseases such as rheumatoid arthritis is more dominated by symptoms of the underlying disease.

Diagnosis

Anemia is not a single disease, but rather a syndrome that can be caused by various underlying conditions. Therefore, in establishing a diagnosis of anemia, it is not sufficient to simply identify the presence of anemia; it is also crucial to

determine the type of anemia, the etiology of the underlying disease, and the presence of comorbidities that may affect treatment. This diagnostic process involves four stages: (a) confirming the presence of anemia, (b) determining the morphological type of anemia, (c) identifying the underlying cause, and (d) assessing the possibility of comorbidities.

Laboratory tests are crucial in supporting the diagnosis and fall into several categories. First, screening tests such as hemoglobin levels, erythrocyte indices, and peripheral blood smears are used to detect anemia and determine its morphological form. Second, a blood series for anemia, including leukocyte count, platelet count, reticulocyte count, and erythrocyte sedimentation rate, are now increasingly accurate thanks to the use of automated equipment. Third, bone marrow examination provides crucial information about the hematopoiesis process and is essential for aplastic anemia, megaloblastic anemia, and other hematologic disorders. Fourth, specific tests are performed as indicated, such as iron levels, ferritin, and iron staining for iron deficiency anemia; folate and vitamin B12 tests for megaloblastic anemia; and serum bilirubin and a Coombs test for hemolytic anemia. These tests help establish a more accurate diagnosis and determine appropriate therapy.

Governance

Treatment for anemia can include:

1. Emergency therapy, such as life-threatening acute bleeding due to aplastic anemia, or acute post-hemorrhagic anemia accompanied by hemodynamic compromise.
2. Supportive therapy.
3. Specific therapy for each anemia.
4. Causal therapy to treat the underlying disease causing the anemia.
5. In situations where a definitive diagnosis cannot be established, trial therapy (ex juvantivus therapy) is necessary. Close monitoring of the patient's response to therapy and changes in the course of the disease must be carried out, and continuous evaluation of the possibility of a change in the diagnosis is necessary.
6. Transfusions are given for acute post-hemorrhagic anemia with signs of hemodynamic compromise. In chronic anemia, transfusions are only given if the anemia is symptomatic or if there is a threat of heart failure. Packed red cells, not whole blood, are given. Increased blood volume is often present in chronic anemia; therefore, transfusions are given as a slow drip. A fast-acting diuretic such as furosemide may also be given before the transfusion.

Complications

Anemia can have various serious health impacts if not properly treated. First, fatigue is the most common symptom and can significantly limit daily activities. When hemoglobin levels are low, the body lacks oxygen, leaving sufferers feeling weak, lethargic, and unable to carry out normal activities, ultimately reducing quality of life. Second, anemia can also cause heart problems.

The heart is forced to work harder to circulate blood to meet the body's oxygen needs. This condition can trigger a rapid or irregular heartbeat and even increase the risk of heart muscle enlargement and heart failure. Third, in certain conditions, anemia can be fatal, particularly in cases of genetic anemia such as sickle cell anemia or acute anemia due to severe bleeding. Therefore, it is important to recognize and treat anemia early to prevent serious complications.

Prognosis

In general, the prognosis for anemia depends heavily on the specific cause and response to treatment. Prompt and appropriate treatment can significantly improve outcomes, while complications or underlying conditions can worsen the prognosis.[1]

Case study

Based on clinical data obtained, a 58-year-old male patient with a history of chronic illness came to the Emergency Department (ER) with the main complaint of weakness that had been felt since five days before admission, accompanied by pallor, pain in the knees, tingling in the hands and feet, and decreased appetite. Supporting examinations showed severe anemia with a hemoglobin level of 5.8 g/dL, hematocrit of 17%, and a very low erythrocyte count of 1.89 million/mm^3. In addition, increased levels of urea (170 mg/dL) and creatinine (8.13 mg/dL) were found, indicating the presence of chronic kidney disease (CKD) stage V. The patient's sodium was also below the normal limit, namely 129.8 mEq/L, which increases the risk of electrolyte complications. Based on the history and laboratory data, the diagnosis was established as severe normocytic normochromic anemia due to CKD. The patient also had a history of type 2 diabetes mellitus since 2005, which was previously treated with metformin and glimepiride, then switched to insulin.

Table 1. Laboratory Examination

P. Labor	Result	Reference	P. Labor	Result	Reference
Hemoglobin	5.8 g/dL (LL)	14.0-17.4	Blood Glucose	98 mg/dL	<200
Erythrocytes	1.89 $10^6/mm^3$(L)	4.5-5.5	Urea	170 mg/dL (H)	20-50
Hematocrit	17.0 % (L)	42-52	Creatinine	8.13 mg/dL (HH)	0.5 – 1.5
MCV	89.9 fL	84-96	Natrium (Na)	129.8 mEq/L (L)	135 – 145
MCH	30.7 pg/cell	28-34	Calium (K)	4.9 mEq/L	3.5 – 5.5
MCHC	34.1 g/dL	32-36	Chlorida (Cl)	104.6 mEq/L	98 – 108
RDW-CV	16.9 % (H)	11.5-14.5			
Leukocytes	7.8 $10^3/mm^3$	5.0-10.0			
Platelets	210 10^3/mL	150-400			

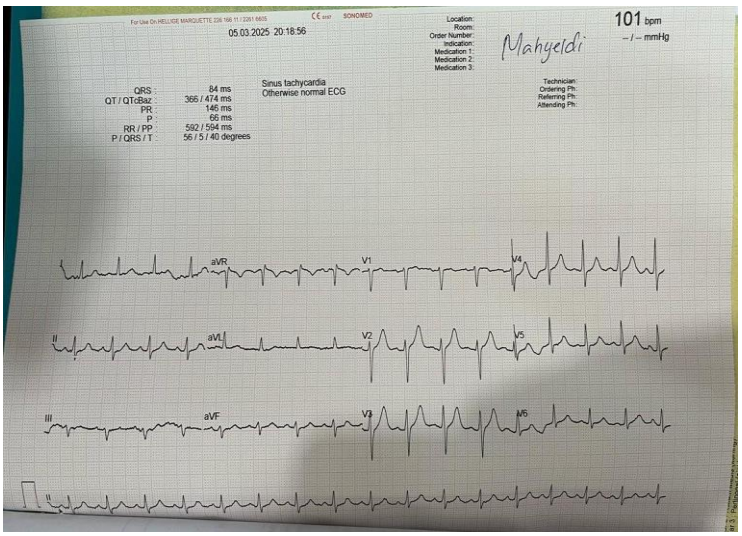

Figure 1. ECG examination

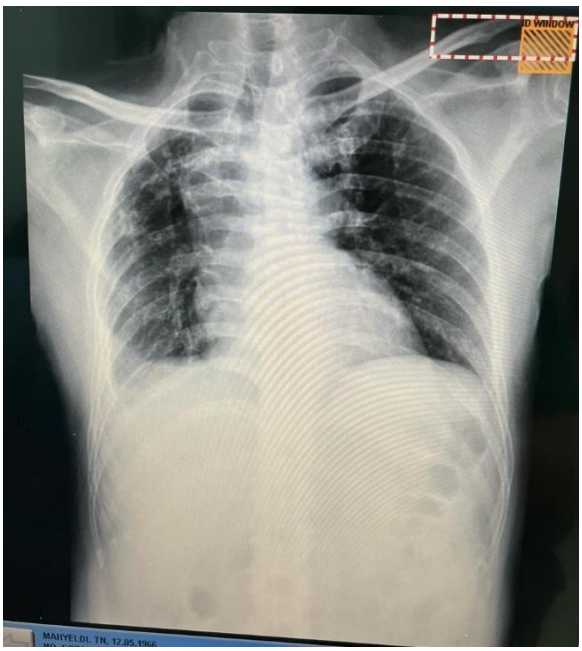

Figure 2. Thoracic X-ray

During the treatment period from March 11 to 15, 2025, the patient received therapy in the form of 4 bags of PRC transfusions (1 bag per day), IVFD NaCl 3% and RL, as well as pharmacological therapy such as folic acid, sodium bicarbonate, Lasix, and Candesartan. Clinically, the patient's condition showed gradual improvement, marked by reduced pallor and weakness, and improved appetite. On the fourth day of treatment, the patient's general condition was assessed as mild, with stable vital signs and oxygen saturation reaching 100%. Despite symptomatic improvement, the patient's prognosis was still considered cautious (dubia ad bonam for quo ad vitam and dubia ad malam for quo ad

fungsionam and sanationam) considering the condition of advanced chronic kidney disease and accompanying severe anemia. Discharge planning and education about anemia management as well as routine check-ups are crucial to prevent recurrence and improve the patient's long-term quality of life.

Table 2. Progress During Treatment

Date	Subyek	Object	Assessment	Plan
Wednesday 12/3/2025	Pale body, weak body, decreased appetite	KU = Medium Awareness = CMC TD : 158/95 HR : 90 RR : 20 SpO_2 : 98 T : 36,6	Severe anemia normocytic normochrom e EC CKD	IVFD RL kolf/12 hours PRC 1 Unit Folic Acid 1x5 Candesartan 1x16
Thursday 13/3/2025	Relatively reduced pale body Relatively reduced weak body Normal appetite	KU = Medium Awareness = CMC TD : 153/84 HR : 96 RR : 20 SpO_2 : 98 T : 36,8	Severe anemia normocytic normochrom e EC CKD	IVFD RL kolf/12 hours PRC 1 Acid Unit Folat 1x5 Candesartan 1x16
Friday 14/3/2025	Relatively reduced pale body Relatively reduced weak body Normal appetite	KU = Medium Awareness = CMC TD : 166/96 HR : 86 RR : 23 SpO_2 : 98 T : 36,3	Severe anemia normocytic normochrom e EC CKD	IVFD RL kolf/12 hours PRC 1 Unit Folic Acid 1x5 Candesartan 1x16
Saturday 15/3/2025	Good appetite Palace and relative weakness are reduced	KU = Mild Consciousnes s = CMC BP: 121/70 HR: 84 RR: 20 SpO2: 100 T: 36.7	Severe anemia normocytic normochrom e EC CKD	IVFD RL kolf/12 hours PRC 1 Unit Folic Acid 1x5

KESIMPULAN (Book Antiqua, 12, bold, spasi 1)

A 55-year-old patient was admitted to M Natsir Solok Regional Hospital on March 11, 2025, with a diagnosis of Severe Normocytic Normochromic Anemia EC CKD. The diagnosis was made based on history, physical examination, and supporting findings.

The patient's history included complaints of weakness for 5 days on admission, worsening after 1 day on admission. These complaints were also accompanied by pallor and decreased appetite. Physical examination revealed a blood pressure of 139/77 mmHg, pulse rate of 73 beats/minute, respiratory rate of 20 breaths/minute, SpO2 of 100%, temperature of 36°C, and anemic conjunctiva. Routine hematology examination revealed hemoglobin 5.8 g/dL, erythrocyte count 1.89 106/mL, hematocrit 17.0%, MCV 89.9 fL, MCH 30.7 pg/cell, RDW-CV 16.9%, urea 170 mg/dL, creatinine 8.13 mg/dL, and sodium 129.8 mEq/L.

The patient was given IVFD RL 12 hours/cold, 4 units of PRBC, sodium bicarbonate 3x1, folic acid 1x5, Lasix injection 1x1 amp, and discontinued his blood sugar medication. The patient was scheduled for monthly internal checkups. He was educated on anemia prevention, the importance of maintaining a healthy diet and taking medications regularly, and regular light exercise such as walking.

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