Ezra Science Bulletin

# EZRA SCIENCE BULLETIN

E-ISSN xxxx-xxxx | DOI prefix 10.58526 | https://ezra.krabulletin.com



## UNDERSTANDING HYPERVOLEMIA IN CHRONIC KIDNEY DISEASE PATIENTS

**[1]Ika Ainur Rofi'ah, [2]Yudha Hendrayanto Utomo**

[1]Medical Surgical Nursing, Emergency Care Nursing, and Critical Care Nursing Department, Faculty of Health Science, Bina Sehat PPNI University, Mojokerto City, East Java, Indonesia

[2]Nurse of Emergency Department Dr. Soetomo General Academic Hospital, Surabaya City, East Java, Indonesia

***Corresponding Author:***
Ika Ainur Rofi'ah
e-mail: ikaainur.ns@gmail.com
Faculty of Health Science, Bina Sehat PPNI University, Mojokerto City, East Java, Indonesia

**ABSTRACT**

**Introduction:** Chronic Kidney Disease (CKD) is a progressive and irreversible decline in kidney function and a decreased Glomerular Filtration Rate (GFR). Decreased kidney function contributes to decreased water excretion and fluid overload caused by decreased GFR. A progressive decrease in GFR in renal disease leads to hypertension, sodium retention, and fluid overload. **Discussion:** CKD is kidney damage or also defined as a decrease in glomerular filtration rate (GFR) of less than 60 ml/minute/1.73m$^2$ that occurs for 3 months or more. CKD is most caused by Diabetes Mellitus 64.1% and Hypertension 80.7%. One clinical manifestation in patients with CKD is an increase in fluid volume (hypervolemia). Hypervolemia is one of the nursing problems in the nursing care process. Based on the Indonesian Nursing Diagnosis Standard Book, hypervolemia is an increase in intravascular, interstitial, and/or intracellular fluid volume. The etiology of hypervolemia in CKD patients is a regulatory mechanism disorder associated with decreased GFR. Signs and symptoms both major and minor in hypervolemia nursing problems show that most patients have peripheral oedema of 53.8%, the prevalence of anemia is 52.3%, the prevalence of Haemoglobin 7-10 mg/dl is 68.3%, and the prevalence of oliguria is 16.3%. One of the actual nursing diagnosis in patients with CKD is hypervolemia related to the disturbance of regulatory mechanisms (a decrease in GFR). A progressive decrease in GFR can lead to sodium and fluid retention which causes fluid balance disturbances.

**Keywords: Hypervolemia, Chronic Kidney Disease, Fluid Overload**



### INTRODUCTION

The kidney is an organ that an important role in maintaining fluid and electrolyte balance in the body. One of the most common problems with the kidneys is chronic kidney failure. Chronic Kidney Disease (CKD) is a condition of progressive and irreversible decline in kidney function (Rahmah et al., 2022). CKD is also defined by a glomerular filtration rate (GFR) <60 ml/min/1.73m$^2$, albuminuria of at least 30 mg/24 hours, or markers of kidney damage such as haematuria, structural damage to the kidney (polycystic or dysplastic) for 3 months or more. CKD is more common in low-income and middle-income countries than

it is in high-income countries. CKD is most often associated with a patient's medical history such as Type 2 Diabetes Mellitus, Hypertension, or other causes (Glomerulonephritis, Infection, environmental exposures such as herbal remedies) (Chen et al., 2019).

Decreased kidney function contributes to decreased water excretion and excess fluid caused by decreased GFR. A progressive decrease in GFR of renal disease leads to Hypertension, sodium retention, and fluid overload. In anuric patients, fluid overload or hypervolemia because of kidney damage can be life-threatening. The average of GFR in CKD patients was 21.4 ml/minute/1.73m$^2$ with a standard deviation of 9.2 ml/minutes/1.73m$^2$ (the normal range of GFR is more than 90 ml/minute/1.73m$^2$) (Khan et al., 2016).

Hypervolemia is a common phenomenon in patients with End-Stage Chronic Kidney Disease or End Stage Renal Disease (ESRD) (Tsai et al., 2014). Hypervolemia is also a prognostic factor not only for patients undergoing kidney dialysis therapy, but also for patients who have pre-dialysis stage of CKD. This is an independent risk factor for cardiac dysfunction (Szymczak et al., 2021).

One of the clinical manifestations that occur in patients with CKD is an increase in fluid volume (hypervolemia). Hypervolemia can result in two clinical manifestations, such as increased blood volume and oedema. The results of the study by Aisara et al (2018), showed that patients with kidney failure experienced fluid balance disorders, that was most of the peripheral oedema was 53.8%, pleural effusion was 3.8%, and ascites was 4.8%. Based on this background, the researchers wanted to know how hypervolemia occurs in patients with Chronic Kidney Disease.

## DISCUSSION

### Chronic Kidney Disease

**Definition and Classification of CKD**. CKD is kidney damage or also defined as a decrease in GFR of less than 60 ml/minute/1.73m$^2$ that occurs for 3 months or more. CKD classification according to KDIGO (2012), recommends details about the causes of CKD and classifies it into six categories based on GFR values (G1, G2, G3a, G3b, G4, G5, and G6). It is also based on 3 levels of albuminuria (A1, A2, and A3) with each stage of CKD categorized based on the Albuminuria-Creatinine Ratio (mg/gm or mg/mmol) in the morning urine sample. The six stages of kidney failure include: 1) G1: GFR 90 ml/minute/1.73m$^2$ and above; 2) G2: GFR 60-89 ml/minute/1.73m$^2$; 3) G3a: GFR 45-59 ml/minute/1.73m$^2$; 4) G3b: GFR 30-44 ml/minute/1.73m$^2$; 5); G4: GFR 15-29 ml/min/1.73m$^2$; 6) G5: GFR <15 ml/minute/1.73m$^2$ or treatment by dialysis. While the 3 categories of levels based on Albumin-Creatinine Ratio (ACR) include: 1) A1: ACR <30 mg/gm (<3.4 mg/mmol); 2) A2: ACR 30-299 mg/gm (3.4-34 mg/mmol); 3) A3: >300 mg/gm (>34 mg/mmol) (Vaidya & Aeddula, 2023).

**The Etilogy of CKD.** The etiology of CKD varies widely globally. The following are the most primary common diseases that cause CKD, consist of 1) Diabetes Mellitus Type 2; 2)

hypertension; 3) Primary Glomerulonephritis; 4) Chronic Tubulointerstitial Nephritis; 5); Hereditary or Cystic Disease. This is in line with research of Khan et al (2016), that most patients with CKD had comorbid of Diabetes Mellitus was 64.1% and comorbid of Hypertension was 80.7%. Other studies also showed that comorbid of Hypertension in CKD patients was 93.3% (pre-hypertension of 29.8%, Stage I of Hypertension was 29.8%, and Stage 2 of Hypertension was 30.8%). (Aisara et al., 2018).

**Diabetes Mellitus.** Hyperglycemia is the stimulation of cell hypertrophy, extracellular matrix synthesis, and the production of TGF-β which is mediated by the activation of protein kinase-C (PKC) which is included in the serine-threonine kinase which has vascular functions such as contractility, blood flow, cell proliferation and capillary permeability. Chronic hyperglycemia can cause non-enzymatic glycation of amino acids and proteins or Mallard and Browning reactions. Glucose will bind to amino and non-enzymatic residues to become a Schiff glycation base, then rearrangement occurs to achieve a more stable but still reversible form and is referred to as an Amador product. If this process continues, irreversible Advanced Glycation End-Products (AGEs) are formed. AGEs are thought to mediate cellular activities such as the expression of adhesion molecules which a role in the withdrawal of mononuclear cells (the occurrence of cell hypertrophy), extracellular matrix synthesis and inhibition of Nitric Oxide synthesis. This process will continue until there is expansion of the mesangial and formation of nodules and tubulointerstitial fibrosis. High glucose levels cause glycosylation of basement membrane proteins, resulting in thickening of the basement membrane, and accumulation of basement membrane glycoprotein-like substances in the mesangial so that the glomerular capillaries are pushed, and blood flow is disrupted which can cause glomerulosclerosis. This condition causes a decrease in kidney function and causes kidney failure (Rivandi & Yonata, 2015).

**Hypertension.** Hypertension causes barotrauma stimulation of the glomerular capillaries and increases glomerular capillary pressure. If this condition lasts a long time, it will cause glomerulosclerosis. Glomerulosclerosis can stimulate chronic hypoxia which causes kidney damage. Hypoxia that occurs causes an increase in oxygen demand for metabolism and causes the release of vasoactive substances (endothelin, angiotensin, and norepinephrine) in local blood vessel endothelial cells resulting in increased vasoconstriction of Renin-Angiotensin-Aldosterone System (RAAS) activation. Besides that, it can also cause oxidative stress which increases oxygen demand and exacerbates hypoxia. Oxidative stress also causes a decrease in the efficiency of sodium transport and damage to DNA, lipids & proteins, which in turn causes tubulointerstitial fibrosis which exacerbates kidney damage (Kadir, 2018).

## Hypervolemia

**Hypervolemia.** Hypervolemia is one of the nursing diagnosis in the nursing care process. Based on the Indonesian Nursing Diagnosis Standard Book, hypervolemia nursing

diagnosis are included in the physiological category and the nutritional and fluid sub-categories. Hypervolemia is an increase in intravascular, interstitial, and/or intracellular fluid volume. The etiology of hypervolemia includes: 1) Impaired regulatory mechanisms; 2) Excess fluid intake; 3) Excess sodium intake; 4) Impaired venous return; 5) Effect of pharmacological agents. Disease conditions related to hypervolemia nursing diagnosis include: 1) Kidney disease: acute/chronic kidney failure, nephrotic syndrome; 2) Hypoalbuminemia; 3) Congestive heart failure; 4) Hormonal disorders; 5) Liver disease (e.g. cirrhosis, ascites, liver cancer); 6) Peripheral venous disease (e.g.: varicose veins, venous thrombus, phlebitis); 7) Immobility (PPNI, 2016).

**The Etiology of Hypervolemia.** The etiology of hypervolemia in CKD patients is a regulatory mechanism disorder, this is in line with a study conducted by Khan et al (2016) that the average of GFR in CKD patients were 21.4 ml/minute/1.73m$^2$ with a standard deviation of 9.2 ml/minute/1.73m$^2$. Meanwhile, the average of GFR in CKD patients who experienced hypervolemia were 16.4 ml/minute/1.73m$^2$ with a standard deviation of 9.6 ml/minute/1.73m$^2$. Further analysis in this study showed that there were differences in GFR values between patients with CKD who experienced hypervolemia and without hypervolemia (p value = 0.03; $\alpha$ <0.05). The results of other studies showed that the average of GFR in CKD patients with hypervolemia were 26.1 ml/minute/1.73m$^2$ with a standard deviation of 14.7 ml/minute/1.73m$^2$, while the average of GFR in CKD patients without hypervolemia were 31.5 ml/minute/1.73m$^2$ with a standard deviation of 14.8 ml/minute/1.73m$^2$. Further analysis showed that there was a significant difference between the GFR of CKD patients with hypervolemia and without hypervolemia (p value = 0.01; $\alpha$ <0.05) (p value=0.01; $\alpha$ <0.05) (Hung et al., 2014).

**Decrease of GFR.** When the kidney nephrons are damaged (including the glomeruli and renal tubules) it causes a decrease in GFR. A decrease of GFR causes retention of metabolic waste products, one of which is fluid and sodium retention in the extracellular space. Excess fluid in the body results in two clinical manifestations, such as increased blood volume and oedema. Fluid and sodium retention can increase circulatory load, oedema, and Congestive Heart Failure or Hypertension. In conditions of GRF <15 ml/minute/1.73m$^2$ it will cause more serious complications and the patient requires kidney replacement therapy, that is dialysis or kidney transplant (Narsa et al., 2022).

**Sign and Symptoms of Hypervolemia.** Signs and symptoms of hypervolemia nusing diagnosis can be classified into two categories, the first that major symptoms and the second that minor symptoms. Major symptoms and signs include: 1) Orthopnea; 2) Dipsnea; 3) Paroxysmal nocturnal dyspnea (PND); 4) Anasarca and/or peripheral oedema; 5) Body weight increases in a short time; 6) Jugular venous pressure (JVP) and/or central venous pressure (CVP) increases; 7) Positive hepatojugular reflex. One of the major symptoms and signs of hypervolemia nursing diagnosis is oedema, this is in line with the research of Khan et al (2016), that was CKD patients who experience leg edema with a score > 1, the majority

of CKD patients with hypervolemia was 41.4%. Further analysis of the study showed that there was a significant difference in leg oedema scores >1 in CKD patients with hypervolemia and CKD patients without hypervolemia (p value <0.01; α <0.05).

Minor signs and symptoms include: 1) Jugular vein distension; 2) Additional breath sounds are heard; 3) Hepatomegaly; 4) Decrease of Haemoglobin/Hematocrit levels; 5) Oliguria; 6) Intake is more than output (excessive fluid balance); 7) Pulmonary congestion (PPNI, 2016). This is in line with the study of Aisara et al (2018), which showed that most CKD patients had anemia (anemic conjunctiva) of 62.5%, most of the Haemoglobin values 7-10 gr/dl were 68.3%, and some patients had oliguria of 16.3% .

**Decrease of Haemoglobin Levels.** Decreased haemoglobin levels in CKD patients are caused by failure of kidney function in producing the Erythropoietin hormone (EPO). EPO hormone is a glyco-protein hormone which is a stimulant for erythropoiesis as a metabolic pathway that produces erythrocytes (red blood cells). The EPO hormone is produced by the kidneys to stimulate the formation of red blood cells by the bone marrow. **Oliguria**. Oliguria occurs due to impaired kidney function in maintaining body fluid homeostasis by controlling fluid volume which causes fluid to accumulate in the body. Oliguria is defined as reduced urine output, i.e. less than 0.5 ml/kilograms of body weight/hour (Aisara et al., 2018).

## CONCLUSION

One of the actual nursing diagnosis in patients with CKD is hypervolemia related to disruption of regulatory mechanisms (decrease of GFR). A progressive decrease in GFR can lead to sodium and fluid retention which causes fluid balance disturbances.


## ACKNOWLEDGMENT

Researchers would like to thank the team that contributed to the preparation of this article.


## CONFLICT OF INTEREST

The author reported no conflict in this research.

## DAFTAR PUSTAKA


Aisara, S., Azmi, S., & Yanni, M. (2018). Gambaran klinis penderita penyakit ginjal kronik yang menjalani hemodialisis di rsup dr. M. Djamil padang. *Jurnal Kesehatan Andalas*, *7*(1), 42. https://doi.org/10.25077/jka.v7i1.778

Chen, T. K., Knicely, D. H., & Grams, M. E. (2019). Chronic Kidney Disease Diagnosis and Management. *Journal of the American Medical Association*, *322*(13), 1294–1304. https://doi.org/10.1001/jama.2019.14745.Chronic

Hung, S. C., Kuo, K. L., Peng, C. H., Wu, C. H., Lien, Y. C., Wang, Y. C., & Tarng, D. C. (2014). Volume overload correlates with cardiovascular risk factors in patients with chronic

kidney disease. *Kidney International*, *85*(3), 703–709. https://doi.org/10.1038/ki.2013.336

Kadir, A. (2018). Hubungan Patofisiologi Hipertensi dan Hipertensi Renal. *Jurnal Ilmiah Kedokteran Wijaya Kusuma*, *5*(1), 15. https://doi.org/10.30742/jikw.v5i1.2

Khan, Y. H., Sarriff, A., Adnan, A. S., Khan, A. H., & Mallhi, T. H. (2016). Chronic kidney disease, fluid overload and diuretics: A complicated triangle. *PLoS ONE*, *11*(7), 1–13. https://doi.org/10.1371/journal.pone.0159335

Narsa, A. C., Maulidya, V., Reggina, D., Andriani, W., & Rijai, H. R. (2022). Studi Kasus: Pasien Gagal Ginjal Kronis (Stage V) dengan Edema Paru dan Ketidakseimbangan Cairan Elektrolit. *Jurnal Sains Dan Kesehatan*, *4*(SE-1), 17–22. https://doi.org/10.25026/jsk.v4ise-1.1685

PPNI. (2016). *Standar Diagnosis Keperawatan Indinesia: Definisi dan Indikator Diagnostik, Edisi*. DPP PPNI.

Rahmah, A. N., Sari, E. A., & Fitria, N. (2022). Management of Hypervolemia In Chronic Kidney Disease Stage 5 At Sumedang Hospital: A Case Study. *Padjajaran Acute Care Nursing Journal*, *3*(10). https://jurnal.unpad.ac.id/pacnj/

Rivandi, J., & Yonata, A. (2015). Hubungan Diabetes Melitus Dengan Kejadian Gagal Ginjal Kronik. *Jurnal Majority*, *4*(9), 27–34. http://juke.kedokteran.unila.ac.id/index.php/majority/article/view/1404/1246

Szymczak, A., Kusztal, M., & Krajewska, M. (2021). Overhydration: A cause or an effect of kidney damage and how to treat it. *Advances in Clinical and Experimental Medicine*, *30*(2), 219–227. https://doi.org/10.17219/ACEM/132035

Tsai, Y. C., Tsai, J. C., Chen, S. C., Chiu, Y. W., Hwang, S. J., Hung, C. C., Chen, T. H., Kuo, M. C., & Chen, H. C. (2014). Association of fluid overload with kidney disease progression in advanced CKD: A prospective cohort study. *American Journal of Kidney Diseases*, *63*(1), 68–75. https://doi.org/10.1053/j.ajkd.2013.06.011

Vaidya, S. R., & Aeddula, N. R. (2023). *Chronic Renal Failure*. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK535404/