# CHAPTER II

# LITERATURE REVIEW

## 2.1 Concept of End Stage Renal Disease

### 2.1.1 Definition

Chronic Kidney Disease (CKD) is a condition characterized by structural and functional abnormalities of the kidneys lasting more than 3 months, with or without a decrease in glomerular filtration rate (eGFR <60 mL/min/1,73m$^2$) (Anggraini & Fadila, 2023). CKD is progressive and irreversible, where the kidneys` ability to maintain fluid and electrolyte homeostasis in the body gradually declines over time and cannot recover. The kidneys` inability to perform their functions optimally can lead to various systemic complications that seriously impact the patient's quality of life (Narsa et al., 2022).

Kidney damage is indicated by one of the following signs: kidney pathological abnormalities, persistent proteinuria, urine abnormalities such as renal hematuria, radiological imaging abnormalities, and a glomerular filtration rate or eGFR <60 mL/min/1,73 m$^2$ (Kalantar-Zadeh et al., 2021). CKD is categorized into 5 stages according to eGFR, and 3 categories according to albuminuria (Ammirati, 2020).

Table 2.1 CKD Classification Based on eGFR

| Stage | Classification | eGFR |
|---|---|---|
| I | Normal or increase | ≥90 |
| II | Mild decrease | 60 – 89 |
| IIIA | Mild to moderate decrease | 45 – 59 |
| IIIB | Moderate to severe decrease | 30 – 44 |

| Stage | Classification | eGFR |
|---|---|---|
| IV | Severe decrease | 15 – 29 |
| V | ESRD | <15 or dialysis |

Table 2.2 CKD Classification Based on Albuminuria

| Category | Classification | Albuminuria 24 hours | Albumin/Creatinine Ratio |
|---|---|---|---|
| A1 | Normal | <30 | <30 |
| A2 | Moderate | 30-300 | 30-300 |
| A3 | Severe | >300 | >300 |

A diabetic nephropathy adult patient with an eGFR of approximately 42 mL/min and albuminuria of 200 mg/24 hours for more than 3 months can be classified as a CKD stage IIIB A2 patient.

End Stage Renal Disease (ESRD) is a condition of CKD that has reached stage V with an estimated glomerular filtration rate (eGFR) of <15 mL/min/1,73 $m^2$. ESRD occurs when the kidneys are no longer adequate to sustain life without kidney transplantation or hemodialysis. (Wouk, 2021).

### 2.1.2 Etiology

End Stage Renal Disease (ESRD), or end stage kidney disease, can be caused by various medical conditions that lead to permanent kidney damage. Some of the most common etiological factors include diabetes mellitus and hypertension, which are primary causes of chronic kidney failure. Additionally, chronic glomerulonephritis significantly contributes to the progression of this disease. Long-term use of non-steroidal anti-inflammatory drugs (NSAIDs), autoimmune disorders such as systemic lupus erythematosus, and genetic abnormalities like polycystic kidney disease and Alport syndrome also serve as important risk factors. Moreover,

congenital malformations of the urogenital system, as well as acute kidney diseases that are not well-managed and persist over a long period, can also develop into ESRD. All these causes, both chronic and acute, require special attention in management to prevent progression to the end stage of kidney disease (Ammirati, 2020). Some risk factors that can also cause ESRD according to Kidney Disease Improving Global Outcomes (KDIGO) (Stevens et al., 2024) yaitu:

Table 2.3 ESRD Risk Factors

| Domain | Example Condition |
|---|---|
| Common risk factors | 1. Hypertension<br>2. Diabetes<br>3. Cardiovascular disease (including heart failure)<br>4. Previous AKI/AKD (Acute Kidney Injury/Acute Kidney Disease) |
| People living in geographical areas with high CKD prevalence | 1. Areas with endemic CKDu (CKD of unknown etiology)<br>2. Areas with high prevalence of APOL1 genetic variants<br>3. Environmental exposure |
| Genitourinary disorders | 1. Structural urinary tract disease<br>2. Recurrent kidney stones |
| Multisystem diseases/chronic inflammation | 1. Systemic lupus erythematosus<br>2. Vasculitis<br>3. HIV |
| Latrogenic (Related to treatment and medication procedures) | 1. Drug-induced nephrotoxicity and radiation nephritis |
| Family history or known genetic variants associated with CKD | 1. Kidney failure, regardless of known cause<br>2. Known kidney diseases associated with genetic abnormalities (Polycystic Kidney Disease/PKD, APOL1-mediated kidney disease, and Alport syndrome) |
| Gestational conditions | 1. Premature birth<br>2. Small for gestational age<br>3. Preeclampsia/eclampsia |

| Domain | Example Condition |
|---|---|
| Occupational exposure that increases CKD risk | 1. Exposure to cadmium, lead, and mercury<br>2. Polycyclic hydrocarbons<br>3. Pesticides |

### 2.1.3 Signs and Symptoms

Common signs and symptoms typically experienced by patients with ESRD include fatigue (70% prevalence), poor mobility (56% prevalence), bone/joint pain (55% prevalence), drowsiness (53% prevalence), pain (53% prevalence), insomnia (49% prevalence), sexual dysfunction (48% prevalence), itching (46% prevalence), heartburn (46% prevalence), muscle cramps (46% prevalence), leg swelling (45% prevalence), decreased appetite (42% prevalence), and shortness of breath (42% prevalence) (Stevens et al., 2024).

Signs and symptoms that may also appear in patients with ESRD are (Kusumawardani, 2018):

1. Gastrointestinal System Disorders

   The presence of protein metabolism disorders in the intestines leads to the formation of toxic substances, triggering anorexia, nausea, and vomiting. Excessive urea in saliva is converted into ammonia by bacteria in the mouth, resulting in ammonia-scented breath.

2. Integumentary System Disorders

   Pale skin due to anemia, sometimes yellowish skin due to urochrome accumulation, itching due to uremic toxins and calcium deposition in

skin pores, ecchymosis due to hematological disorders, and urea frost due to urea crystallization in sweat.

3. Hematological System Disorders

   Disruption in erythropoietin hormone production causes anemia; sometimes, platelet and leukocyte disorders also occur.

4. Nervous and Muscular System Disorders

   Patients may feel leg aches, leading to constant leg movement, commonly known as restless leg syndrome. Tingling sensations and a burning feeling, especially on the soles of the feet, are often referred to as burning feet syndrome. Myopathy, weakness, and muscle hypotrophy can also occur.

5. Cardiovascular System Disorders

   In ESRD patients, fluid and salt retention occur, increasing renin-angiotensin-aldosterone activity, which leads to elevated blood pressure. Electrolyte disturbances can cause cardiac arrhythmia.

6. Endocrine System Disorders

   Sexual dysfunction in men is caused by decreased testosterone production and spermatogenesis, resulting in reduced libido, fertility, and erectile function. Meanwhile, in women, this condition leads to menstrual irregularities, ovulation disorders, and amenorrhea. Additionally, glucose metabolism disorders, insulin resistance, and issues with insulin secretion also occur.

7. Other Systemic Disorders

   In bones, patients may experience renal osteodystrophy, which refers to abnormal changes in bone growth and formation. The accumulation of organic acids as a result of metabolism can also lead to metabolic acidosis. Electrolyte disturbances include hyperphosphatemia, hyperkalemia, and hypocalcemia.

### 2.1.4 Pathophysiology

The onset of ESRD depends on the underlying disease (A. Lestari, 2021). Some diseases that cause ESRD include hypertension and diabetes. In patients with uncontrolled hypertension, prolonged exposure leads to thickening of the renal arterial walls, resulting in reduced blood flow to the kidneys. The amount of blood and O2 entering the kidneys also decreases. Reduced oxygen supply to kidney cells damages the glomerulus and causes renal ischemic injury.

Damage to the glomerulus activates immune cells to repair it. During this process, immune cells release growth factors such as TGF-β1. These growth factors cause mesangial cells to revert to an immature or primordial form, namely mesangioblasts. Mesangioblasts themselves produce extracellular matrix; an excessive amount of extracellular matrix leads to glomerulosclerosis, or the formation of scar tissue in the glomerulus. The characteristic scar tissue is stiff and inflexible, thereby reducing the nephron's ability to filter blood. Over time, this progresses to ESRD.

In patients previously suffering from diabetes, ESRD can also develop. High glucose levels in the blood trigger non-enzymatic glycation, a process of glycation without enzymes. During the filtration process in the glomerulus, excessive glucose attaches to proteins, producing byproducts called Advanced Glycation End Products (AGEs), which can eliminate cell elasticity, leading to cell damage.

In cases of ESRD, non-enzymatic glycation occurs in the efferent arterioles (arteries carrying blood out of the kidney), causing the efferent arterioles to stiffen. If the efferent arterioles are stiff, less blood exits the kidney, and pressure within the glomerulus increases. This high pressure in the glomerulus leads to hyperfiltration, resulting in significant blood wastage. Furthermore, high pressure in the glomerulus activates mesangial cells, causing glomerulosclerosis and a decline in nephron function. This ultimately results in a reduced ability to filter blood, leading to ESRD.

### 2.1.5 Diagnostic Examinations

To aid in strengthening the diagnosis of ESRD, several diagnostic/supportive examinations will be conducted. These diagnostic examinations can also be used to determine the underlying cause of ESRD in the patient. Some diagnostic examinations that can be performed include (Stevens et al., 2024):

Table 2.4 ESRD Diagnostic Examinations

| Examination | Type of Examination | Result |
|---|---|---|
| Radiology | Ultrasonography, intravenous urography, CT scan of | Evaluates kidney structure (shape, size, symmetry, and evidence of obstruction) |

| Examination | Type of Examination | Result |
|---|---|---|
| | the kidney-ureter-bladder, nuclear medicine studies, MRI | |
| Kidney biopsy | Percutaneous with USG guidance | Used for accurate diagnosis, treatment planning, assessment of disease activity and chronicity, potential treatment response, and can also evaluate genetic diseases |
| Laboratory examinations | Serology, urinalysis | The presence of persistent hematuria or albuminuria is crucial in determining differential diagnoses |
| Genetic testing | APOL 1, COL4A3, COL4A4, COL4A5, NPHS1, UMOD, HNF1B, PKD1, PKD2 | Genetic causes are more common and may appear without a family history |

### 2.1.6 Complications

In ESRD, several complications may occur, with the first being cardiac arrhythmia. The kidneys function is to maintain the body's electrolyte balance. One electrolyte that should be secreted is potassium ($K^+$), however, in ESRD patients, the kidneys are unable to secrete this potassium. The potassium remains in the body, leading to hyperkalemia. Excessively high potassium levels are very dangerous, especially for the heart. Although cardiac muscles require potassium for every contraction, they do not need it in large amounts. High potassium levels can affect heart rhythm, leading to cardiac arrhythmia.

Another complication is renal osteodystrophy. Not only is potassium not secreted, but calcium ($Ca^{2+}$) electrolytes also cannot be properly secreted. Under normal conditions, the kidneys help activate vitamin D into

calcitriol. Active vitamin D aids the body in increasing calcium absorption from food. If the kidneys are damaged, vitamin D is not activated, leading to decreased calcium absorption from food. The body will experience calcium deficiency, known as hypocalcemia. In response, the body will release parathyroid hormone. Parathyroid hormone functions in calcium storage in bones and calcium breakdown from bones. If parathyroid hormone is active, calcium stored in bones will be broken down to meet the body's needs. Bones will lose calcium, becoming weak and brittle; this condition is termed renal osteodystrophy.

Furthermore, hypertension is a common complication. If the kidneys detect insufficient fluid intake, they will secrete the hormone renin to increase fluid entry into the kidneys by elevating blood pressure. The increased blood pressure is expected to enhance fluid flow into the kidneys. However, in ESRD patients, the glomerular filtration rate decreases, leading to increased renin secretion and consequently hypertension.

Another hormone secreted by the kidneys is erythropoietin. Erythropoietin stimulates the production of red blood cells in the bone marrow. In ESRD, erythropoietin levels decrease, resulting in reduced red blood cell production. Therefore, a common complication that can occur is anemia.

### 2.1.7 Management

Management of patients with ESRD, according to (Kronik, 2023) is as follows:

1. Dialysis

   Dialysis is a medical procedure that functions as a replacement for kidney function by excreting toxins and waste products from the body, maintaining electrolyte balance, and filtering blood. Dialysis is recommended upon reaching stage V with an eGFR <15 mL/min/1,73 $m^2$. There are two main types of dialysis wich is hemodialysis (HD) and peritoneal dialysis (PD).

2. Kidney transplantation

   Kidney transplantation, or kidney grafting, is the process of replacing a damaged kidney organ. The transplanted kidney can originate from either a living or deceased donor.

## 2.2 Concept of Hemodialysis Therapy

### 2.2.1 Definition

Hemodialysis is a therapeutic procedure that functions as a kidney replacement and only substitutes a portion of the kidneys' excretory function. This therapeutic procedure aims to correct biochemical imbalances in the blood caused by impaired kidney function. Hemodialysis is performed on patients with CKD stage V (ESRD) whose eGFR is <15 mL/min/1,73 $m^2$ (Liani, 2016). Hemodialysis only replaces excretory function, not endocrine function. Therefore, hemodialysis merely removes waste products and does not aid in hormone production.

The working principles of hemodialysis involve diffusion and ultrafiltration. Diffusion is the process of solute movement that occurs due

to a concentration difference between the blood and dialysate compartments, or the movement of molecules from a high concentration solution to a low concentration solution. High concentration solutes in the blood, such as potassium and urea, will move to the dialysate. Conversely, low concentration solutes in the blood will move from the dialysate compartment to the blood. Ultrafiltration is the movement of water molecules through a semi-permeable membrane, driven by a pressure difference. When hydrostatic pressure is increased, water will move (Kusumawardani, 2018).

### 2.2.2 Indications and Contraindications

Timely administration of therapy can prevent complications in ESRD patients. Renal replacement therapies such as hemodialysis carry various risks. When eGFR decreases, the decision for hemodialysis therapy must consider its risks and benefits. This decision takes into account various factors, such as the patient's social factors. According to the Decree of the Minister of Health of the Republic of Indonesia concerning the National Guidelines for Medical Services in Chronic Kidney Disease Management (2023), the implementation of hemodialysis therapy is recommended to begin if the patient experiences one or more specific clinical conditions indicating advanced kidney failure. These conditions include the emergence of symptoms directly related to kidney dysfunction, such as serositis, acid-base or electrolyte imbalance, and persistent pruritus. Furthermore, other indications include the body's inability to adequately maintain fluid volume

and blood pressure stability, a progressive decline in nutritional status despite interventions, and the onset of cognitive dysfunction. The decision to initiate hemodialysis is based on a comprehensive evaluation of the patient's clinical condition and not solely on the glomerular filtration rate value. Some of these conditions sometimes occur in patients with an eGFR between 5-10 mL/min/1,73 $m^2$ (Kronik, 2023).

According to the Decree of the Minister of Health of the Republic of Indonesia concerning the National Guidelines for Medical Services in Chronic Kidney Disease Management (2023), there are several contraindications for hemodialysis procedures, such as the absence of vascular access, problems with vascular access, heart failure, coagulopathy, unstable hemodynamics, advanced organ failure conditions (dementia and advanced cirrhosis with encephalopathy), advanced malignancy, and also advanced stage AIDS (Kronik, 2023).

### 2.2.3 Interdialytic Weight Gain (IDWG)

During hemodialysis therapy, patients are advised to consistently control their fluid intake in accordance with fluid output. Between two hemodialysis sessions, patients will experience an increase in body weight, which is referred to as Interdialytic Weight Gain (IDWG). IDWG is the difference between the pre-dialysis body weight and the post-dialysis body weight from the previous session. IDWG represents an increase in fluid volume, manifested as weight gain (Febrianti, 2023).

An acceptable IDWG is no more than 1.0–1.5 kg or not exceeding 3% of dry body weight (Isnaini, 2020). Dry body weight is the lowest weight achieved by a patient after dialysis where there is no indication of fluid retention (Febrianti, 2023). IDWG is grouped into 3 categories: an increase of <4% is mild IDWG, an increase of 4–6% is moderate IDWG, and an increase of >6% is high IDWG (Haloho, 2017). Meanwhile, (Koizer, 2014 dalam Hakim et al., 2023) divides IDWG into 3 categories: mild IDWG 2%, moderate 5%, and high 8%.

In each hemodialysis session, the patients' weight will be measured before and after dialysis. IDWG itself is measured by calculating the current pre-hemodialysis body weight minus the previous post-hemodialysis body weight, then dividing by the previous post-hemodialysis body weight, and multiplying by 100%.

$$IDWG = \frac{BW\ Pre\ HD\ 2 - BW\ Post\ HD\ 1}{BW\ Post\ HD\ 1} \times 100\%$$

There are several factors that can influence IDWG, originating from both internal (self-related) and external (physical and psychosocial) factors. The factors influencing an increase in IDWG include (Rahayu, 2023):

1. Fluid intake

   In ESRD patients, the kidneys are unable to adequately excrete and reabsorb fluid. This impairment can lead to fluid volume overload. Fluid intake is considered excessive in ESRD patients undergoing hemodialysis if fluid intake is greater than fluid output. Therefore,

ESRD patients undergoing hemodialysis are advised to maintain fluid intake consistent with the amount of fluid excreted.

2. Thirst

   Despite experiencing hypervolemia, ESRD patients often report increased thirst. This stimulates patients to increase their fluid intake. The common response to thirst is drinking. This thirst is caused by electrolyte disturbances, such as hypernatremia, hypokalemia, increased ureaplasma, as well as several psychological factors.

3. Self-efficacy

   Self-efficacy is an inherent strength that can generate positive energy through cognitive, motivational, affective, and selection processes (Haloho, 2017). High self-efficacy is necessary to cultivate intrinsic motivation to adhere to the prescribed therapy program. Effective adherence to the therapy program can prevent an increase in IDWG and enhance body fluid control.

4. Stress

   During stress, the body releases elevated levels of aldosterone and glucocorticoid hormones. Excessive levels of these hormones lead to sodium and salt retention. The stress response ultimately increases fluid volume, consequently reducing cardiac output, blood pressure, and tissue perfusion. Stress in ESRD patients can cause them to cease monitoring fluid intake, leading to an increase in IDWG (Ulum, 2023).

5. Age

As age advances, the function of bodily organs declines. Similarly, kidney function experiences a reduction in its ability to respond to changes in fluid and electrolytes.

### 2.2.4 Complications

#### 2.2.4.1 Intradialytic Hypertension

Hypertension is an increase in blood pressure above normal levels. The normal blood pressure range varies depending on age. Hypertension occurring after hemodialysis is termed intradialytic hypertension. According to Kidney Disease: Improving Global Outcomes (KDIGO) 2024, intradialytic hypertension is defined as a systolic blood pressure increase of ≥10 mmHg from pre- to post-hemodialysis (Stevens et al., 2024). Intradialytic hypertension occurs when the systolic blood pressure increases by ≥10 mmHg from pre- to post-hemodialysis (Inrig et al., 2007 cited iin Theofilis et al., 2023). Various studies define intradialytic hypertension differently, and its prevalence also varies depending on the definition used (Susanto, 2020).

The mechanism of intradialytic hypertension is attributed to the kidneys' failure to regulate blood pressure in ESRD patients. The following are several factors suspected to cause intradialytic hypertension:

1. Extracellular fluid volume overload

   Excess extracellular fluid is a common finding in patients with intradialytic hypertension. In an uncontrolled study, 6 hemodialysis patients underwent echocardiograms both before and during

hemodialysis. At the beginning of ultrafiltration, blood pressure and cardiac index appeared to increase. However, after further fluid removal, blood pressure normalized, and the cardiac index returned to baseline. Therefore, Buren & Inrig (2017) concluded that the increase in intradialytic blood pressure is caused by overhydration and the effects of changes in cardiac index.

2. Vasoconstriction

The relationship between extracellular fluid volume overload and intradialytic hypertension is strong but not sufficient to establish causality. A study by (Chou et al., 2006 cited in P. Buren & Inrig, 2017) on patients routinely undergoing hemodialysis, with pre- and post-hemodialysis echocardiogram examinations, showed a significant increase in vascular resistance in intradialytic hypertension patients.

This research identified a connection between increased blood pressure and endogenous vasoconstrictors. Researchers revealed an imbalance in endothelial cell mediators, specifically an increase in endothelin-1 (ET-1) and a decrease in the nitric oxide (NO) to ET-1 ratio. ET-1 is a vasoconstrictor, while NO is a vasodilator. ET-1 is produced by vascular endothelial cells in the heart, lungs, spleen, pancreas, and kidneys. High ET-1 levels bind to ETA and ETB receptors, both of which play a role in contraction (Kowalczyk et al., 2015).

3. Activation of Renin-Angiostenin-Aldosterone System (RAAS)

   In ESRD patients undergoing hemodialysis, there is an increase in plasma renin activity (PRA) and plasma aldosterone from pre- to post-hemodialysis. This indicates the presence of residual nephron function still utilized by hemodialysis patients to maintain their ability to respond to changes in intravascular volume in response to ultrafiltration. Rapid intravascular volume depletion during ultrafiltration stimulates RAAS activation, leading to increased peripheral resistance and elevated blood pressure (Sarafidis et al., 2017).

4. Overactivity of the sympathetic nervous system

   When measured using microneurography, hemodialysis patients exhibit excessive sympathetic nervous activity. This is due to increased plasma catecholamine concentrations in ESRD patients. Catecholamines are hormones produced in response to stress. Another mechanism believed to increase sympathetic nervous activity is the inhibition of NO (nitric oxide) by ADMA (asymmetric dimethylarginine). ADMA is an endothelial vasoconstrictor, while NO is an endothelial vasodilator (Bucharles et al., 2019).

5. Endothelial dysfunction

   In patients experiencing intradialytic hypertension, there is an increase in serum ET-1 during the hemodialysis process. This results in an imbalance between vasoconstrictors and vasodilators. This

imbalance occurs when there is an increase in serum endothelin-1 (ET-1) during dialysis, while nitric oxide (NO) remains at low levels. ET-1 acts as a vasoconstrictor and NO as a vasodilator. This imbalance leads to increased peripheral resistance (Assimon et al., 2018).

6. Dialysate sodium

   Dialysate contains serum sodium, which functions to maintain hemodynamic stability, electrolyte balance, and reduce muscle cramps. Intradialytic hypertension can be caused by the use of dialysate sodium at excessively high concentrations. This leads to high post-hemodialysis sodium levels and triggers thirst. Consequently, there is an increase in body weight during the interdialytic period.

7. Erythropoietin Agents (EPO) therapy

   EPO therapy is administered to ESRD patients who experience anemia. Intravenous EPO administration in patients can trigger an increase in endothelin-1 (ET-1) levels, a peptide that acts as a potent vasoconstrictor. This response causes an increase in mean arterial pressure (MAP) by approximately 20 mmHg, with an onset time of about 30 minutes post-injection, and the effect can last up to three hours. High-dose EPO use is known to contribute to increased blood pressure, caused by increased arterial stiffness and increased blood viscosity. This combination of hemodynamic changes can worsen hypertension in patients with chronic kidney disease who receive routine EPO therapy (Sebastian et al., 2016).

8. Arterial stiffness

   Arterial stiffness can be measured by pulse wave velocity. In a study consisting of 47 hemodialysis patients without a history of cardiovascular disease, this research compared pulse wave velocity between patients with HD-responsive blood pressure (defined as a MAP decrease of >5% during HD) and HD-nonresponsive blood pressure (defined as failure to decrease MAP >5% during HD). The results showed that 45% of patients were classified as having HD-responsive blood pressure with a 17% decrease in MAP, while the remainder had HD-nonresponsive blood pressure with a 6% increase in MAP. Pulse wave velocity was higher in patients with HD-nonresponsive blood pressure, suggesting that unrecognized arteriosclerosis can contribute to intradialytic hypertension (Georgianos et al., 2015).

9. Ultrafiltration (UF)

   During hemodialysis, ultrafiltration is performed to withdraw fluid. The amount of UF depends on the weight gain during the interdialytic period. The higher the weight gain, the higher the ultrafiltration rate. Conversely, the lower the weight gain, the lower the ultrafiltration rate. A faster ultrafiltration rate carries a greater risk of mortality and cardiovascular disease (CVD) (Susanto, 2020).

10. Drug elimination during hemodialysis

During the hemodialysis process, some antihypertensive drugs can be eliminated, increasing the risk of intradialytic hypertension. Antihypertensive drugs that can be eliminated during dialysis include ACE inhibitors and beta-blockers. However, it is still uncertain whether the elimination of these antihypertensive drugs definitively causes intradialytic hypertension.

#### 2.2.4.2 Intradialytic Hypotension

Intradialytic hypotension lacks standardization, and several studies provide differing definitions. According to the National Kidney Foundation Kidney Disease Outcomes Quality Initiative guidelines from 2005, intradialytic hypotension is defined as a systolic blood pressure decrease of ≥20 mmHg or a MAP decrease of ≥10 mmHg accompanied by hypotensive symptoms. The European Best Practice Guidelines define intradialytic hypotension as a systolic blood pressure decrease of ≥20 mmHg (Davenport, 2023).

There are several risk factors that can cause intradialytic hypotension, namely (Hamrahian et al., 2023):

1. Ultrafiltration (UF)

   If the patient's IDWG is high, the ultrafiltration rate will also be high. Rapid fluid removal during ultrafiltration can lead to a relative decrease in circulating volume. If the body cannot respond adequately, this can result in intradialytic hypotension.

2. Dialysis fluid concentration

Low concentrations of dialysate fluids such as sodium, magnesium, and calcium can increase the risk of intradialytic hypotension. A low sodium concentration in the dialysate increases the risk of intradialytic hypotension because a significant portion of sodium is removed during the hemodialysis process, thereby lowering the patient's plasma sodium levels. Therefore, switching from low to high sodium concentrations is done to maintain sodium balance and hemodynamic stability during dialysis. In ESRD patients, renal magnesium excretion is reduced, leading to low magnesium concentrations and causing intradialytic hypotension. Higher potassium concentrations can improve cardiac function and intradialytic systolic blood pressure compared to low potassium (Sidiq, 2021).

## 2.3 Theoretical Framework

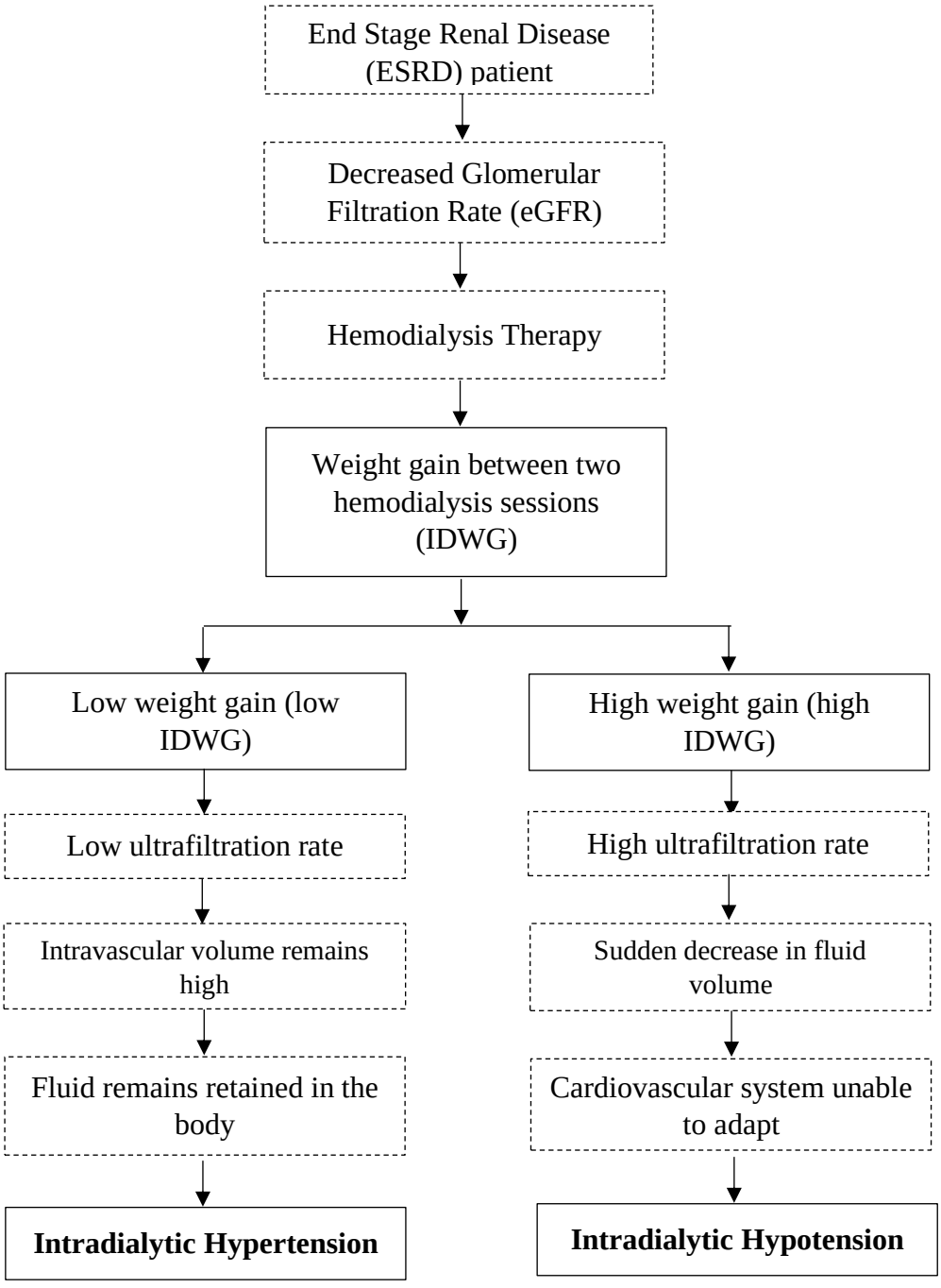


Figure 2. 1 Theoretical Framework

## 2.4 Hypothesis

Based on the description above, the hypotheses for this study are as follows:

1. $H_0$: There is no relationship between Interdialytic Weight Gain (IDWG) and intradialytic blood pressure changes in End Stage Renal Disease patients undergoing hemodialysis at Lavalette Hospital.
2. $H_1$: There is a relationship between Interdialytic Weight Gain (IDWG) and intradialytic blood pressure changes in End Stage Renal Disease patients undergoing hemodialysis at Lavalette Hospital..