Chapter 12

# Fluid Overload in Peritoneal Dialysis

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**Abstract**

The prevalence of end-stage renal disease (ESRD) has increased globally to 10% due to diabetes mellitus, hypertension, and stroke. When chronic kidney disease (CKD) maintenance therapy fails, patients require renal replacement therapy (RRT) to survive, such as peritoneal dialysis (PD), hemodialysis, and renal transplantation. The most common therapy in Mexico is PD because it is a feasible, low-cost, and easy-to-perform procedure; however, fluid overload is a frequent condition in patients with this RRT modality. The usual adverse comorbidities in patients with PD are cardiovascular diseases (CVD) associated to atherosclerosis, uremia, inflammation, and oxidative stress. Fluid overload is intimately associated to hypertension, left ventricular hypertrophy, heart failure, and worsening of kidney failure, leading to increased hospital admissions, higher cardiovascular mortality, and reduced life expectancy. Two main pathologies are involved in the deterioration of both heart and kidney functions, namely, cardiorenal syndrome and uremic cardiomyopathy. Along with these phenomena, patients in PD with rapid peritoneal transport have reduced ultrafiltration, increased glucose absorption, and albumin loss in the dialysate, which lead to overhydration, hypertension, dyslipidemia, and malnutrition. This review focuses on the clinical, physiological, and biochemical mechanisms involved in fluid overload of patients with CKD undergoing PD.

**Keywords:** end-stage renal disease, peritoneal dialysis, fluid overload, cardiorenal syndrome, uremic cardiomyopathy, ultrafiltration failure

# 1. Introduction

## 1.1. Chronic kidney disease

The prevalence of chronic degenerative diseases has recently increased due to aging of the society and advances in medical technology [1]. In Mexico, there is a significant increase in the prevalence and incidence of non-transmittable diseases, such as diabetes mellitus (DM), hypertension, and stroke, which raises the prevalence of end-stage renal disease (ESRD) up to 10% globally [2–5].

Chronic kidney disease (CKD) is defined as a severe, irreversible kidney damage, measured by the level of proteinuria and reduced glomerular filtration rate that prevents the kidneys from functioning properly and removing toxins and waste products from the blood [6, 7]. Among the many traditional risk factors for CKD, DM is the leading cause of kidney dysfunction in the developed world. CKD induce vascular damage and therefore a raise in cardiovascular mortality; it is considered an independent risk factor for cardiovascular events, even from the early stages of the disease [8–13].

When CKD maintenance therapy fails, patients will require renal replacement therapy (RRT) to survive; among the alternatives of RRT, there is peritoneal dialysis (PD), hemodialysis (HD), and renal transplantation (TR) [14, 15]. The most common cause of ESRD in the world is type 2 DM (38%) according to the study of Kidney Early Evaluation Program (KEEP) [16–18]. The second most common cause is hypertension, and the combination of DM and hypertension raises the prevalence up to 42% [3, 19]. However, ESRD of unknown origin is one of the most prevalent diagnoses due to a lack of prompt recognition of the disease, and is unclear whether hypertension is a cause or consequence of CKD [20]. Furthermore, a study including 3564 healthy subjects reported a prevalence of deteriorated creatinine clearance <60 mL/min of 37% [21].

The United States Renal Data System (USRDS), in 2013, reported that Mexico, USA, and Portugal had a rate of 63, 58, and 54 patients per million of habitants (ppmh), respectively. The overall prevalence of CKD in adults varies between 6% and 69% [22]. The Registry of Dialysis and Transplant in Jalisco and Morelos has reported an increase of patients who require a RRT in the last decade [23, 24]. In 2013, Mendez et al. reported an incidence of 421 per million and a prevalence of 1653 per million of habitants, which places Mexico in the second country with more ESRD incident cases and the sixth most prevalent [25].

Health systems are taking emergency measures to control the burden of the disease due to the health impact, elevated costs, overall risk of developing CKD, and its consequences. Thanks to the National Kidney Foundation (NKF) that produces clinical practice guidelines through the NKF Kidney Disease Outcomes Quality Initiative (NKF K/DOQI), in 2002, it has been possible to establish an early diagnosis, risk stratification, and well-defined action plans to mitigate the progression of the disease and its cardiovascular complications [26].

## 1.2. Chronic kidney disease and renal replacement therapy

In Mexico, PD is the most frequent RRT implemented, followed by HD, which has increased rapidly over the last years [5]. The PD is a feasible, low-cost, and easy-to-perform procedure,

reason for why in Latin America is gaining popularity. Chile is the more prevalent country with PD as the first-line RRT, and Mexico has the second place in Latin America and eighth place around the world [3, 27]. Hemodialysis is mainly available in social security and private institutions; however, HD is more expensive for governmental institutions, and hence it is not an open resource for all patients with ESDR [28]. Renal transplant is the RRT associated with better long-term survival rates and is considered the best RRT for ESDR patients; immunosuppressive drugs have reduced mortality and improved the viability of the graft [29]. The highest proportion of renal transplantation in the world is in Jalisco, Mexico, according to the USRDS [22]. However, the waiting lists for a RT are increasing exponentially, in spite of the fact that in Mexico the donation of live donors is privileged [5].

Unfortunately, RRT generate high costs and are limited to treating certain populations with social security, leaving the so-called disadvantaged populations in abandonment, generating a high rate of morbidity and mortality in younger populations. In 2005, the Mexican Institute of Social Security (IMSS) reported that treating ESDR represented 21% of the total expenditure of its main program, with only 0.7% of the beneficiaries' investment [22].

HD increase chronic inflammation by different mechanisms. A continuous contact with artificial filter dialysis membranes that induce complement activation, cytokines, and nitric oxide production characterizes HD. There also may be exposure to dialysate contaminants, which cross the dialysis membranes with monocyte stimulation and activation. Another deleterious process contributing inflammation in HD is local or systemic infections through contamination of vascular accesses, such as endovascular catheters, synthetic grafts, and arteriovenous fistulas. Fluid overload also occurs in HD due to extracellular fluid expansion and ventricular growth, which enhances CVD risk [30].

### 1.3. Peritoneal dialysis and cardiovascular mortality

ESDR is among the leading causes of death worldwide; morbidity and mortality in this group of patients are mainly due to CVD [31]. CVD in patients with PD is associated to traditional risk factors, such as atherosclerosis, DM, and hypertension, in addition to uremia, inflammation, and oxidative stress [12]. Cardiorenal syndrome (CRS) is a manifestation of CVD in patients with ESRD and is manifested by acute and chronic conditions where the primary dysfunction may be renal or cardiac. Among the five categories of CRS, type 4 is characterized by pre-existing CKD that leads to ESRD with progressive worsening of cardiac function [32].

Fluid overload is one of the main characteristics of patients with late CKD. The abnormal state of fluid in the disease correlates with hypertension, left ventricular hypertrophy (LVH), and other adverse cardiovascular sequels [33]. There is evidence that fluid overload is associated with significant increased risk of mortality from all cardiovascular causes in dialysis patients [34], which makes strict volume control imperative to improve the survival of patients undergoing dialysis [35]. A previous study showed the positive relationship between fluid overload with an increased risk of initiating dialysis and decrease in rapid renal function in late CKD, which means that fluid overload is not a feature in CKD, but also a prognostic marker of rapid progression of late CKD [36]. Adverse progression of kidney disease in patients with DM is associated with changes for fluid, thus contributing to fluid overload [37]. There appears

to be a complex interaction between DM, fluid overload, and progression of kidney disease [38]. Dialysis procedure by itself plays an important role in the pathogenesis of accelerated atherosclerosis in patients with ESRD [39] (**Figure 1**).of fluid retention in patients undergoing peritoneal dialysis are shown

*1.3.1. Cardiorenal syndrome*

There is a close relationship between cardiac and renal functions. It is bidirectional and has physical, chemical, and biological implications. Primary disorders of one of these two organs often result in secondary dysfunction or injury to the other [40]. Over the last decade, cardiovascular mortality in patients with CKD has remained strikingly elevated. CKD is a recognized risk factor for the development of CVD [41] and increases 10- to 20-fold the risk of cardiac death compared to non-CKD subjects, after adjusting for age and gender [42]. A glomerular filtration rate (GFR) below 60 mL/min/1.73 $m^2$ is associated with cardiovascular risk; therefore, patients with CKD should be thoroughly evaluated in the search for cardiovascular risk factors that may require aggressive management [43].

Cardiorenal syndrome is a pathophysiological disturbance of the interaction between the heart and the kidneys caused by acute or chronic dysfunction in one of the two organs, capable of inducing acute or chronic dysfunction in the other organ. In 2008, Ronco et al. proposed five subtypes according to the temporal sequence of organ failure and the clinical context [31]:

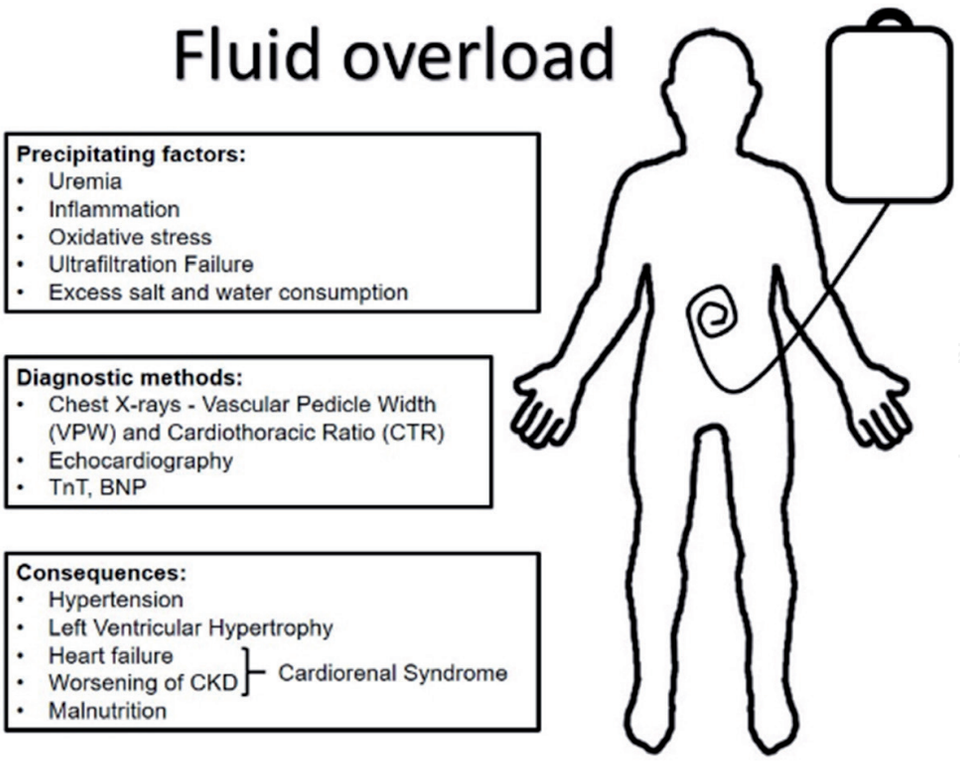


**Figure 1.** Fluid overload. The mechanisms of fluid retention in patients undergoing peritoneal dialysis are shown schematically.

An acute cardiac disease that leads to acute kidney injury (AKI) [33] or worsening of a chronic kidney failure characterizes acute cardiorenal syndrome or *CRS type 1*. It is also a consequence of low cardiac output due to acute coronary syndrome. When renal function worsens, it is possible to predict significantly higher rates of hospitalization and mortality from acute heart failure [44].

In *CRS type 2*, a chronic heart failure leads to CKD due to a hemodynamic imbalance. It is often manifested as a chronic renal dysfunction associated to chronic heart failure [45]. An episode of AKI that leads to acute heart failure characterizes *CRS type 3*. Retention of uremic solutes and/or volume overload may contribute to cardiac injury. According to experimental data, it is suggested that cardiac dysfunction may be related to activation of the immune system, release of inflammatory mediators, oxidative stress, and cellular apoptosis [46]. Other proposed mechanisms include electrolyte and fluid imbalance, metabolic acidosis, and uremia [47].

Chronic cardiorenal syndrome or *CRS type 4* is defined as a primary CKD that induces heart failure, ventricular hypertrophy, diastolic dysfunction, and/or greater risk of major cardiovascular events. Clinically, it is very difficult to distinguish between *CRS types 2* and *4*, since the first insult is not often recognized [39]. The prototype of *CRS type 4* is polycystic renal disease, an autosomal dominant genetic disease that leaves no doubt of the primary event. Increased fluid retention characterizes *CRS type 4*, found in approximately 70–80% of patients with ESRD [48, 49].

*CRS type 5* comprises simultaneous heart and kidney dysfunction due to a systemic disease. Given the broad spectrum of diseases that contribute to this syndrome, there are several pathophysiological mechanisms consequence of the systemic disease: an overwhelming insult leads to the simultaneous development of AKI and acute cardiac dysfunction [50]. Sepsis and drug-induced toxicity are the most common causes leading to *CRS type 5*. It may develop in a patient with previously impaired organ function or when there is no discernible evidence of prior abnormality. The sequence of organ involvement may vary depending upon the acuity and nature of the underlying disorder. Other known systemic diseases that lead to *CRS type 5* are autoimmune disorders, such as lupus, Wegener's granulomatosis, and sarcoidosis. It is difficult to identify the underlying pathophysiological mechanisms in order to develop a diagnostic and therapeutic intervention strategy; thus, to identify the underlying mechanism, it is essential to consider the temporal events that initially lead to this syndrome. *CRS type 5* has the following phases: hyperacute (0–72 h after the diagnosis), acute (3–7 days after), subacute (7–30 days), and chronic (>30 days). Most of the evidence of hyperacute stage comes from clinical trials of sepsis, and patients with cirrhosis support the research from chronic stage. A precipitating event usually contributes to the development of *CRS type 5* in a chronic patient, for example, a spontaneous bacterial peritonitis in a patient with cirrhosis. Therefore, we may find superimposition of acute *CRS type 5* on an indolent chronic process with immediate relevance for intensive care physicians, nephrologists, and cardiologists [51].

Almost 75% of the patients with ESRD have a cardiovascular pathology [31]. Kidney failure worsens the short- and long-term prognosis due to several comorbid cardiovascular conditions. Acute myocardial infarction survival is lower as the deterioration of renal function increases, and the chance for survival is even worst in patients with ESRD and congestive heart failure [52]. CKD patients have 10–20 times higher risk for cardiovascular mortality than healthy subjects; even small reductions in kidney function can induce a significant increase

of cardiovascular risk: patients with stage 1–3 of CKD have 25–100 times higher risk of CVD, and stage 5 has a similar kidney and heart morbidity and mortality [31]. Almost half of the patients with ESRD in PD have cardiac arrhythmias (especially atrial fibrillation) [53]. Other risk factors for cardiovascular mortality in PD users are cardiac valvulopathies, water retention, hypertension, DM, vascular calcifications, altered oxidative status, bone mineral disorders, and uremic cardiomyopathy [54].

#### *1.3.2. Uremic cardiomyopathy*

Uremic cardiomyopathy (UC) is a suitable example for *CRS type 4*, as it is characterized by cardiac dysfunction leading to fluid overload and hypertension, accentuated by the presence of high levels of myocardial urea [55, 56]. UC is found at early stages of CKD and leads to structural and functional cardiovascular damage as the kidney dysfunction progresses [57, 58]. UC can predict CVD mortality at the beginning of PD [30]. The main feature is LVH, considered as a primary manifestation of UC, but it also induces left ventricular dilation and both systolic and diastolic cardiac dysfunctions [28].

The first-line treatment for UC is conventional HD, since it leads to a reduction in LVH. HD can also reverse systolic dysfunction by improving the left ventricular ejection fraction. The earlier HD is initiated in patients with PD and fluid retention, the more damage to the myocardium induced by UC can be avoided [59]. Angiotensin-converting enzyme inhibitors decrease LVH even in normotensive subjects. Likewise, RT confers remodeling to the myocardium affected by UC in patients with ESRD undergoing PD during short or medium time lapses, although some data are contradictory because of the dyslipidemia, hypertension, and DM associated to immunosuppressants in RT recipients [60].

### 1.4. Diagnostic methods and cardiovascular disease biomarkers

Chronic intravascular hypervolemia in patients with PD is an important contributor to CVD. There is no simple and reliable method to assess the volume status in patients with PD [61]; ankle edema or elevated jugular venous pressure is not accurate because they can only detect abnormal body water volume. Traditionally, body fluid compartment is measured by dilution methods for solutes or isotopes, but the tests are cumbersome and rarely used in routine clinical practice. More recently, measurement of vascular pedicle width (VPW) and cardiothoracic ratio (CTR) on chest radiographs is a noninvasive surrogate marker of intravascular volume status in critically ill patients [62]. Moreover, temporary changes in fluid balance are reflected in simple chest X-rays. The objective radiographic findings of intravascular volume may be more appropriate for fluid balance than subjective measurements; the VPW is the most sensitive determination. When systematically quantified, sequential chest radiographs provide substantial information to other clinically available data to help handle fluids in patients with water retention [63]. In patients with long-standing PD, CTR is an independent predictor of hospitalization-free patient survival; this radiological parameter can be used for risk stratification of patients undergoing PD [64].

Echocardiography is the most reliable, noninvasive, diagnostic procedure, capable to identify UC-related findings. Its capacity to quantify ventricular mass, ejection fraction, valvular disease,

pericardial effusion, or pulmonary arterial hypertension [65] makes it useful to predict the damage extent of ESRD patients with recent RRT [66, 67]. Diastolic dysfunction and LVH have been described in three out of four patients, and systolic dysfunction in half of the patients who undergo initial PD. It is also very common to find aortic and mitral valve alterations, almost in one third of these patients. These findings have repercussion in the prognosis of patients with ESDR who start a PD program [68].

There are several useful biomarkers already evaluated in patients with CKD: troponin (TnT), plasminogen activator inhibitor type 1 (PAI-1), homocysteine, brain natriuretic peptide (BNP), C-reactive protein (CRP), serum amyloid-A protein, ischemia-modified albumin, and advanced glycation products (AGEs) have been shown to correlate with adverse cardiovascular (CV) events in patients with CKD [31].

Troponin T (TnT) and BNP have a good predictive value in this population [69, 70]. They were both elevated in patients with hypervolemia and were able to identify asymptomatic patients with CKD who have 2–5 times increased CV risk. BNP is also a useful marker in patients with left ventricular dysfunction and cardiovascular congestion. Increased levels of TnT represent a strong independent predictor of overall cardiovascular mortality in asymptomatic patients with HD [31]. Renal biomarkers, such as cystatin C (CysC) and neutrophil gelatinase-associated lipcalin (NGAL), have recently been studied as prognostic and diagnostic markers of cardiovascular outcomes in CKD patients [71]. There are increased levels of CysC in atherosclerotic processes and LVH; it has association with the latter, independently of renal function. Researchers found increased levels of NGAL expression in the atherosclerotic plaque of patients with heart failure due to coronary heart disease [31, 72].

### 1.5. Peritoneal transport

A useful tool for the management of patients in PD is the peritoneal equilibration test (PET). This method has proved to be effective in assessing peritoneal function. In this test, the saturation curves of the solutes in the peritoneum with respect to the plasma are evaluated; thus, it is possible to classify the peritoneal functioning in an easy and reproducible way [73, 74]. PET has been shown to have a prognostic value in patients undergoing PD [75] and allows patients to be classified according to the ratio of solute concentrations in dialysate and plasma (D/P ratio) 4 h after the test. Creatinine, urea, electrolytes, phosphate, and proteins are the commonly tested solutes, and it classifies patients in different types of transporters: (a) high or fast, (b) average high, (c) low average, and (d) low. PET allows the clinician to determine the best dialysis modality for each individual who will undergo continuous ambulatory PD or automated DP (continuous cyclic DP or intermittent nocturnal DP) [76].

Patients with high peritoneal solute transport rates often have inadequate transport of the peritoneal fluid. It is not known whether inadequate transport of fluids is solely due to a rapid drop in osmotic pressure or if the reduction in the efficiency of liquid transport is also a contributing factor. The difference in fluid transport between the abovementioned groups is apparently due to variances in the rate of disappearance of the total osmotic pressure of the dialysate, resulting from the transport velocity of glucose and other small solutes [77]. Although glucose gradient is the main factor influencing the rate of ultrafiltration, other solutes,

such as urea, are also important [78]. However, there is a relationship between comorbid states that lead to an elevated mortality and the rapid transport of solutes [79].

Patients in PD with rapid peritoneal transport have reduced ultrafiltration, increased glucose absorption, and albumin loss in the dialysate. This phenomenon induces fluid overload, hypertension, dyslipidemia, and malnutrition, along with increased mortality. In addition, systemic vascular disorders observed in DM, hypertension, atherosclerosis, sepsis, and smoking contribute to survival deterioration in these patients; vascular and endothelial disorders are closely related to malnutrition-inflammation-atherosclerosis syndrome [80]. By its own, this syndrome can explain the high mortality rate observed in patients with rapid peritoneal transport [81].

The rapid transport of solutes at the beginning of PD is closely associated with genetic, inflammatory, and structural factors of the peritoneal membrane [82]. The clinical consequences of these alterations are CVD, metabolic disturbances of glucose and lipids, hypoalbuminemia, and malnutrition. In order to treat adequately patients with rapid solute transport, it is necessary to improve their comorbidities and modify their dialytic solutions with better osmotic substances different from glucose, as well as dialysis modalities that optimize ultrafiltration [60].

### *1.5.1. Ultrafiltration failure*

Peritoneal transport dysfunction is usually associated with ultrafiltration failure (UF) or deficit. Ultrafiltration failure is defined by the Society of Peritoneal Dialysis as the impossibility to maintain a stable dry weight in spite an adequate fluid restriction, and the total ultrafiltration volume is less than 400 mL after two or more hypertonic dialytic exchange with at least 4 h inside the peritoneal cavity using dextrose solution of 3.86% [83]. The prevalence of UF increases with duration of PD, so that 30–50% of patients with PD develop UF, many patients abandon PD due to UF, and dropout increases depending on the type of PD. It has been reported up to 3% of dropouts during the first year and 31% after the next six years [59, 84].

There are four ultrafiltration failure causes:

- Type I, due to an increase of the effective peritoneal surface with increase in solute transport. It appears in the acute phase of peritonitis episodes associated to PD and is characterized by an early recovery after 30 days [85].
- Type II is characterized by a reduced effective peritoneal surface with irreversible peritoneal involvement due to peritoneal adhesions or sclerosing peritonitis secondary to previous surgical scars or repetitive bacterial peritonitis [86].
- Type III is due to the increased rate of peritoneal lymphatic reabsorption [87].
- Type IV, also known as transcellular UF, is the most recently described and illustrated by a cellular dysfunction or disruption of aquaporins in the cellular wall [88].

Some mechanical problems should be discarded if fluid overload is suspected: lost from dialysate fluid due to herniation or history of multiple abdominal surgeries, poorly positioned catheters secondary to migration of the original catheter, inadequate placement during the surgical procedure, and abdominal adhesions from previous surgeries [89].

The functional study of the peritoneal membrane is useful to guide the prescription of PD, predict the response of standard exchanges, and diagnose ultrafiltration disturbances during PD treatment. Knowing the pathophysiological mechanisms can help determine the underlying etiology of UF to ensure prompt actions that can help preserve peritoneal membrane for longer periods [90].

### 1.6. Salt restriction and volume status

Salt is an ionic component composed of sodium chloride (60% chloride and 40% sodium), with a molar mass of 58,433 g/mol. Sodium is an essential nutrient for the correct functioning of nerves and muscles, as well as water self-regulation and fluid balance. Salt is widely used to preserve processed foods, cooking, and seasoning. Processed foods have higher amounts of salt than natural foods, such as meats, fruits, and vegetables, which have a significant impact on a higher daily intake of sodium derived from the consumption of these foods [91]. Excessive salt intake stimulates thirst and promotes water intake, which contributes to fluid overload and hypertension [92]; therefore, a common strategy for patients with ESRD is salt and water restriction. In patients with PD, the salt balance can be improved by different strategies, among them the reduction in dietary intake, the use of diuretics to increase urinary secretion, and the increase of extraction by peritoneal ultrafiltration. The appropriate salt intake is the first treatment option for proper maintenance of the volume state [93]. The recommendation according to the Cardiovascular and Metabolic Guidelines of the International Society for Peritoneal Dialysis is to reduce intake to <2 g of sodium or <5 g of salt per day [94]. The lack of adherence to these recommendations is an important cause of fluid gain in patients undergoing PD [95].

Although at the beginning of PD excessive ingestion of salt and liquids is not usually a problem due to the preservation of residual renal function, as renal function decreases, it is imperative to advise patients to decrease salt and water intake [96]. The advice of diet salt and water restriction in patients with PD leads to a decrease in body weight of 2.8 ± 0.5 kg and consequently to a reduction of blood pressure from 158.2 ± 17.0/95.7 to 119.7 ± 16.0/77.9 mmHg, in addition, a decrease in CTR from 48.0% ± 5.6% to 42.9% ± 4.5%. The role of salt and water restriction for the management of volume overload is highlighted due to the impact on the maintenance of volume status in patients with PD, which makes it fundamental for the adequate control of volume status [97]. However, some contradictory studies like the one by Fine et al. found that administration of 60 mEq/day of sodium chloride was significantly associated with an increase in blood pressure. The raise in systolic blood pressure was from 135 ± 19 to 144 ± 21 and diastolic blood pressure from 77 ± 8 to 82 ± 12 mmHg, without body weight gain (72 ± 10 to 72 ± 11 kg) in 20 patients undergoing PD enrolled to a double-blinded crossover clinical trial. They concluded that patients tolerate a diet with normal sodium intake and does not lead to volume overload [98]. Nevertheless, salt restriction in these patients has been widely recommended for adequate maintenance of volume status.

There is no gold standard for assessing dietary salt intake in PD patients. The tools used for the evaluation are food diaries, 24-h reminders, consumption frequency questionnaires, and urine analysis for 24 h. The limitations of these tools include variation in day-to-day sodium

intake, errors related to memory lapses, patient motivation, false perception of diet, difficulty in measuring salt use, over-/under-collection of urine, among others [99].

Urine 24-h sodium determination does not reflect the current sodium intake in patients with PD, since the elimination of sodium occurs through urine and dialysate. In addition, the removal of sodium from the dialysate depends on the convection through the peritoneal membrane, so it cannot reflect the current sodium intake in these patients [100, 101]. The measurement of total sodium withdrawal during dialysis adequacy assessment might be a simple and effective method of estimating sodium ingestion in patients with PD. Total sodium withdrawal during dialysis adequacy assessment may be a simple and effective method to estimate sodium intake [102]:

$$\text{Sodium intake (mg/dL)} = 15.64 \times \text{total sodium withdrawal (mEq/d)} + 646 \quad (1)$$

For example, a sodium intake of 2000 mg would correspond to a total sodium removal of approximately 87 mEq/d [102].

In a cohort of 305 PD incident patients, Dong et al. reported that low sodium ingestion was significantly associated with nutrient deficiency and poor muscle reserve and an independent predictor for mortality. It is necessary to consider whether salt restriction in the diet would improve outcomes in patients with low calorie and protein intake [100].

A correct nutritional advice can achieve a decrease in salt intake, minimizing processed foods and avoiding salt in food preparation. Certain strategies to reduce salt ingestion can be useful to improve the taste of food, such as substitution with flavor enhancers like pepper, paprika, curry, thyme, and oregano; also changing to salt substitutes, which contain potassium chloride in patients who do not require potassium restriction [103]. An advantage of salt substitutes compared to flavor enhancers is that the former have a higher salty taste, but the disadvantage is the risk of hyperkalemia [104].

Finally, another strategy is to prepare a diet containing 2 g of sodium (88 mM NaCl), by allowing to add 1/3 of tablespoon of salt for each meal during that day. It is worth noting the impact of salt intake on patients with PD. Therefore, under this context, the previously mentioned strategies to maintain a low ingestion of salt in the diet could help to avoid a deficit in the consumption of nutrients and the maintenance of the state of volume.

## 2. Conclusions

The incidence and prevalence of ESRD are increasing, making the need for PD necessary as a demanding RRT for patients with CKD. The main morbidity and mortality cause in patients with CKD is still primarily due to CVD. It is important to start an early approach to fluid overload by performing and interpreting different assessments, such as echocardiography, PET, UF test, and an adequate food survey for the identification of factors that contribute to poor adherence to dietary recommendations in water and saline intake. Fluid overload is an important cause for hospital admissions; thus, clinicians must have this in mind for the early identification of the causes of cardiac decompensation, besides attending the individual disorders of each patient with PD.

## Conflict of interest

There are no conflicts of interest.

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