We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,300

Open access books available

172,000

International authors and editors

190M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Fluid Overload in Peritoneal Dialysis

Leonardo Pazarin-Villaseñor,
Francisco Gerardo Yanowsky-Escatell,
Jorge Andrade-Sierra,
Luis Miguel Roman-Pintos and
Alejandra Guillermina Miranda-Diaz

Additional information is available at the end of the chapter

<http://dx.doi.org/10.5772/intechopen.69324>

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 m² 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 ESRD 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 lipocalin (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 of 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 $158.2 \pm 17.0/95.7$ to $119.7 \pm 16.0/77.9$ mmHg, in addition, a decrease in CTR from $48.0\% \pm 5.6\%$ to $42.9\% \pm 4.5\%$. 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 cross-over 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.

Author details

Leonardo Pazarin-Villaseñor¹, Francisco Gerardo Yanowsky-Escatell¹, Jorge Andrade-Sierra¹, Luis Miguel Roman-Pintos^{2,3} and Alejandra Guillermina Miranda-Diaz^{4*}

*Address all correspondence to: kindalex1outlook.com

1 Department of Nephrology, Hospital Civil de Guadalajara “Dr. Juan I. Menchaca”
Guadalajara, Jalisco, México

2 Department of Sickness and Health as an Individual Process, Centro Universitario de
Tonalá, Universidad de Guadalajara, Guadalajara, Jalisco, México

3 Department of Internal Medicine, Hospital Ángeles del Carmen, Guadalajara, Jalisco,
México

4 Department of Physiology, Centro Universitario de Ciencias de la Salud, Universidad de
Guadalajara, Guadalajara, Jalisco, México

References

- [1] Choi ES, Lee J. Effects of a face-to-face self-management program on knowledge, self-care practice and kidney function in patients with chronic kidney disease before the renal replacement therapy. *Journal of Korean Academy of Nursing*. 2012;**42**(7):1070-1088
- [2] Correa-Rotter R, González-Michaca L. Early detection and prevention of diabetic nephropathy: A challenge calling for mandatory action for Mexico and the developing world. *Kidney International*. 2005;**(98)**:S69-S75
- [3] Parfrey PS, Foley RN. The clinical epidemiology of cardiac disease in chronic renal failure. *Journal of the American Society of Nephrology*. 1999;**10**:1606-1615
- [4] Shastri Sh, Sarnak MJ. Cardiovascular disease and CKD: Core curriculum 2010. *American Journal of Kidney Disease*. 2010;**56**(2):399-417
- [5] Alonso Gómez AM. ¿Qué debe de conocer el nefrólogo de la afectación cardiaca del paciente en diálisis peritoneal? *Nefrología*. 2008;**28**(6):105-111
- [6] White SL, Chadban SJ, Jan S, Chapman JR, Cass A. How can we achieve global equity in provision of renal replacement therapy? *Bulletin of the World Health Organization*. 2008;**86**:229-237
- [7] Jin DC, Han JS. Renal replacement therapy in Korea, 2012. *Kidney Research and Clinical Practice*. 2014;**33**(1):9-18

- [8] Weiner DE, Tighiouart H, Stark PC, et al. Kidney disease as a risk factor for recurrent cardiovascular disease and mortality. *American Journal of Kidney Disease*. 2004;**44**:198-206
- [9] Zhang L, Zuo L, Wang F, et al. Cardiovascular disease in early stages of chronic kidney disease in a Chinese population. *Journal of the American Society of Nephrology*. 2006;**17**:2617-2621
- [10] Chronic Kidney Disease Prognosis Consortium. Association of estimated glomerular filtration rate and albuminuria with all-cause and cardiovascular mortality in general population cohorts: A collaborative meta-analysis. *Lancet*. 2010;**375**:2073-2081
- [11] Van der Velde M, Matsushita K, Coresh J, et al. Lower estimated glomerular filtration rate and higher albuminuria are associated with all-cause and cardiovascular mortality. A collaborative meta-analysis of high-risk population cohorts. *Kidney International*. 2011;**79**:1341-1352
- [12] Clase CM, Gao P, Tobe SW, et al. Estimated glomerular filtration rate and albuminuria as predictors of outcomes in patients with high cardiovascular risk: A cohort study. *Annals of Internal Medicine*. 2011;**154**:310-318
- [13] Fox CS, Matsushita K, Woodward M, Bilo HJ, Chalmers J, et al. Chronic Kidney Disease Prognosis Consortium: Associations of kidney disease measures with mortality and end-stage renal disease in individuals with and without diabetes: a meta-analysis. *Lancet*. 2012;**380**:1662-1673
- [14] Garcia-Garcia G, Briseño-Rentería G, Luquín-Arellano VH, Gao Z, Gill J, Tonelli M. Survival among patients with kidney failure in Jalisco México. *Journal of the American Society of Nephrology*. 2007;**18**:1922-1927
- [15] Kim EJ, Chung CH, Park MY, Choi SJ, Kim JK, Hwang SD. Mortality predictors in patients treated with continuous renal replacement. *Korean Journal of Nephrology*. 2011;**30**:73-79
- [16] Kidney Disease: Improving Global Outcomes (KDIGO) CKD Group. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Summary of Recommendation Statements. *Kidney International*. 2013;**3**(1):5-14
- [17] Cueto-Manzano A, Quintana-Piña E, Correa-Rotter R. Long term CAPD survival and analysis of mortality risk factors: 12-year experience of a single Mexican center. *Peritoneal Dialysis International*. 2001;**21**:148-153
- [18] Cueto-Manzano A, Rojas-Campos E. Status of renal replacement therapy and peritoneal dialysis in Mexico. *Peritoneal Dialysis International*. 2007;**27**:142-148
- [19] Obrador GT, García-García G, Villa AR, et al. Prevalence of Chronic kidney disease in the Kidney early Evaluation Program (KEEP) Mexico and comparison with KEEP US. *Kidney International*. 2010;**77**(Suppl 116):S2-S8
- [20] Vasavada N, Agarwal R. Role of excess volume in the pathophysiology of hypertension in chronic kidney disease. *Kidney Int*. 2003 Nov;**64**(5):1772-9

- [21] Amato D, Alvarez-Aguilar C, Castañeda-Limones R, et al. Prevalence of chronic kidney disease in an urban Mexican Population. *Kidney International*. 2005;**68**(Suppl 97): S11-S17
- [22] The US Renal Data System. Available at: <https://www.usrds.org/adr.aspx>. Accessed: February 12, 2017
- [23] Saran R, Li Y, Robinson B, et al. US Renal Data System 2015 Annual Data Report: Epidemiology of kidney disease in the United States. *American Journal of Kidney Diseases*. 2016;**67**(3 suppl 1):S1-S434
- [24] Guía de Práctica Clínica. Prevención, Diagnóstico y Tratamiento de la Enfermedad Renal Crónica Temprana. México; Secretaría Nacional de Salud; 2009
- [25] Mendez DA, Mendez-Bueno F, Tapia YT, Muñoz MA, Aguilar SL. Epidemiología de la insuficiencia renal crónica en México. *Diálisis y Trasplante*. 2010;**31**(1):7-11
- [26] National Kidney Foundation. K/DOQI Clinical Practice Guidelines for chronic kidney disease: Evaluation, classification and stratification. *American Journal of Kidney Disease*. 2002;**39**(Suppl 1):S1-S266
- [27] Garcia GG, Monteon RF, Garcia BH, et al. Renal replacement therapy among disadvantaged populations in Mexico a report from the REDTJAL. *Kidney International*. 2005;**68**:s58-s61
- [28] Durán-Arenas L, Avila-Palomares PD, Zendejas-Villanueva R, Vargas-Ruiz MM, Tirado-Gómez LL, López-Cervantes M. Direct cost analysis of hemodialysis units. *Salud Pública de México*. 2011;**53**(4):516-524
- [29] Garcia GG, Harden P, Chapman J. El papel global del trasplante renal. *Nefrología*. 2012; **32**(1):1-6
- [30] Clementi A, Virzi GM, Go ChY, Cruz D, Granata A, Vescovo G, et al. Cardiorenal syndrome type 4: A review. *CardioRenal Medicine*. 2013;**3**:63-70
- [31] Ronco C, Haapio M, House AA, Anavekar N, Bellomo R. Cardiorenal syndrome. *Journal of the American College of Cardiology*. 2008;**52**:1527-1539
- [32] Shah BN, Greaves K. The cardiorenal syndrome. *International Journal of Nephrology*. 2011;**2011**:920195
- [33] Wizemann V, Schilling M. Dilemma of assessing volume state—The use and the limitations of a clinical score. *Nephrology, Dialysis, Transplantation*. 1995;**10**:2114-2117
- [34] Paniagua R, Ventura MD, Avila-Díaz M, Hinojosa-Heredia H, Méndez-Durán A, et al. NT-proBNP, fluid volume overload and dialysis modality are independent predictors of mortality in ESRD patients. *Nephrology, Dialysis, Transplantation*. 2010;**25**:551-557
- [35] Ozkahya M, Ok E, Toz H, Asci G, Duman S, et al. Long-term survival rates in haemodialysis patients treated with strict volume control. *Nephrology, Dialysis, Transplantation*. 2006;**21**:3506-3513

- [36] Tsai YC, Tsai JC, Chen SC, Chiu YW, Hwang SJ, Hung CC, Chen TH, Kuo MC, Chen HC. Association of fluid overload with kidney disease progression in advanced CKD: A prospective cohort study. *American Journal of Kidney Disease*. 2014;**63**(1):68-75
- [37] Tucker BJ, Collins RC, Ziegler MG, Blantz RC. Disassociation between glomerular hyperfiltration and extracellular volume in diabetic rats. *Kidney International*. 1991;**39**: 1176-1183
- [38] Forbes JM, Fukami K, Cooper ME. Diabetic nephropathy: Where hemodynamics meets metabolism. *Experimental and Clinical Endocrinology and Diabetes*. 2007;**115**:69-84
- [39] Li PK, Kwan BC, Ko GT, Chow KM, Leung CB, Szeto CC. Treatment of metabolic syndrome in peritoneal dialysis patients. *Peritoneal Dialysis International*. 2009;**29**(2): S149-S155
- [40] Stevenson LW, Nohria A, Mielniczuk L. Torrent or torment from the tubules? Challenge of the cardiorenal connection. *Journal of the American College of Cardiology*. 2005;**45**(12): 2004-2007
- [41] Chertow GM, Normand S-LT, Silva LR, McNeil BJ. Survival after acute myocardial infarction in patients with end-stage renal disease: Results from the cooperative cardiovascular project. *American Journal of Kidney Disease*. 2000;**35**(6):1044-1051
- [42] Herzog CA. Dismal long-term survival of dialysis patients after acute myocardial infarction: can we alter the outcome? *Nephrology, Dialysis, Transplantation*. 2002;**17**(1):7-10
- [43] Clementi A, Virzì GM, Brocca A, de Cal M, Vescovo G, Granata A, Ronco C. Cardiorenal syndrome type 4: Management. *Blood Purification*. 2013;**36**(3-4):200-209
- [44] Damman K, Navis G, Voors AA, Asselbergs FW, Smilde TD, Cleland JG, van Veldhuisen DJ, Hillege HL. Worsening renal function and prognosis in heart failure: Systematic review and meta-analysis. *Journal of Cardiac Failure*. 2007;**13**:599-608
- [45] De Vecchis R, Baldi C. Cardiorenal syndrome type 2: From diagnosis to optimal management. *Therapeutics and Clinical Risk Management*. 2014;**10**:949-961
- [46] Bagshaw SM, Cruz DN. Epidemiology of cardiorenal syndromes. *Contributions to Nephrology*. 2010;**165**:68-82
- [47] Clementi A, Virzì GM, Brocca A, de Cal M, Pastori S, Clementi M, Granata A, Vescovo G, Ronco C. Advances in the pathogenesis of cardiorenal syndrome type 3. *Oxidative Medicine and Cellular Longevity*. 2015;**2015**:1-8
- [48] Cheung AK, Sarnak MJ, Yan G, Berkoben M, Heyka R, Kaufman A, Lewis J, Rocco M, Toto R, Windus D, Ornt D, Levey AS; HEMO Study Group. Cardiac diseases in maintenance hemodialysis patients: Results of the HEMO study. *Kidney International*. 2004;**65**:2380-2389
- [49] Corradi GMVV, Panagiotou A, Gastaldon F, Cruz DN, de Cal M, et al. ADPKD: Prototype of cardiorenal syndrome type 4. *International Journal of Nephrology*. 2011;**2011**:490795

- [50] Mehta RL, Rabb H, Shaw AD, Singbartl K, Ronco C, McCullough PA, Kellum JA. Cardiorenal syndrome type 5: Clinical presentation, pathophysiology and management strategies from the eleventh consensus conference of the Acute Dialysis Quality Initiative (ADQI). *Contributions to Nephrology*. 2013;**182**:174-194
- [51] Ronco C, McCullough PA, Anker SD, Anand I, Aspromonte N, Bagshaw SM, et al. Cardiorenal syndromes: An executive summary from the consensus conference of the Acute Dialysis Quality Initiative (ADQI). *Contributions to Nephrology*. 2010;**165**:54-67
- [52] Schrotten NF, Damman K, Valente MA, Smilde TD, van Veldhuisen DJ, Navis G, Gaillard CA, Voors AA, Hillege HL. Long-term changes in renal function and perfusion in heart failure patients with reduced ejection fraction. *Clinical Research in Cardiology*. 2016;**105**(1):10-16
- [53] McCullough PA, Assad H. Diagnosis of cardiovascular disease in patients with chronic kidney disease. *Blood Purification*. 2012;**33**(1-3):112-118
- [54] Cuba M, Batista R. Cardiac calcification in patients with terminal chronic renal insufficiency and kidney transplant patients. *Nefrología*. 2004;**24**(2):196-197
- [55] Chinnappa S, Hothi SS, Tan LB. Is uremic cardiomyopathy a direct consequence of chronic kidney disease? *Expert Review of Cardiovascular Therapy*. 2014;**12**(2):127-130
- [56] Alhaj E, Alhaj N, Rahman I, Niazi T, Berkowitz R, Klapholz M. Uremic cardiomyopathy: An underdiagnosed disease. *Congestive Heart Failure*. 2013;**19**(4):E40-E45
- [57] Kunz K, Dimitrov Y, Muller S, Chantrel F, Hannedouche T. Uraemic cardiomyopathy. *Nephrology, Dialysis, Transplantation*. 1998;**13**(4):39-44
- [58] Riggato C, Parfrey PS. Uraemic Cardiomyopathy. *Journal of Clinical and Basic Cardiology*. 2001;**4**:93-95
- [59] Codognotto M, Piccoli A, Zaninotto M, Mion M, Plebani M, Vertolli U, Tona F, Ruzza L, Barchita A, Boffa GM. Renal dysfunction is a confounder for plasma natriuretic peptides in detecting heart dysfunction in uremic and idiopathic dilated cardiomyopathies. *Clinical Chemistry*. 2007;**53**(12):2097-2104
- [60] Rosales BG, Sotelo CM, Monteon RF, Quirarte JA, Zuñiga G, Cueto-Manzano A. Metabolic and echocardiographic changes during dialysis and after renal transplantation. *Nefrología Mexicana*. 2002;**23**(1):5-10
- [61] Szeto CC, Li PK. Salt and water balance in PD. In: Molony D, Craig J, editors, *Evidence-Based Nephrology*. Oxford: Wiley Blackwell (BMJ Books); 2009. pp. 488-499
- [62] Ely EW, Haponik EF. Using the chest radiograph to determine intravascular volume status: The role of vascular pedicle width. *Chest*. 2002;**121**(3):942-950
- [63] Martin GS, Ely EW, Carroll FE, Bernard GR. Findings on the portable chest radiograph correlate with fluid balance in critically ill patients. *Chest*. 2002;**122**(6):2087-2095

- [64] Gao N, Kwan BC, Chow KM, Chung KY, Leung CB, Li PK, Szeto CC. Longitudinal changes of cardiothoracic ratio and vascular pedicle width as predictors of volume status during one year in Chinese peritoneal dialysis patients. *Kidney and Blood Pressure Research*. 2009;**32**(1):45-50
- [65] Stewart GA, Gansevoort RT, Mark PB, Rooney E, McDonagh TA, Dargie HJ, Stuart R, Rodger C, Jardine AG. Electrocardiographic abnormalities and uremic cardiomyopathy. *Kidney International*. 2005;**67**(1):217-226
- [66] Hung KC, Lee CH, Chen CC, Chu CM, Wang CY, Hsieh IC, Fang JT, Lin FC, Wen MS. Advanced left ventricular diastolic dysfunction in uremic patients with type 2 diabetes on maintenance hemodialysis. *Circulation Journal*. 2012;**76**(10):2380-2385
- [67] Pecoits-Filho R, Berberato SH. Echocardiography in chronic kidney disease: Diagnostic and prognostic implications. *Nephron. Clinical Practice*. 2010;**114**:c242-c247
- [68] Pazarín-Villaseñor L, Reyes LU, León-Flores AM, Miranda-Díaz AG, Andrade-Sierra J. Miocardiopatía urémica y transporte peritoneal en pacientes incidentes con diálisis peritoneal en el occidente de México. *Nefrología*. 2016 (in press). <http://dx.doi.org/10.1016/j.nefro.2016.11.005>
- [69] Daniels LB, Maissel AS. Natriuretic peptides. *Journal of the American College of Cardiology*. 2007;**50**:2357-2368
- [70] deFillipi CR, Fink JC, Nass CM, Chen H, Christenson R. N-terminal Pro B-type natriuretic peptide for predicting coronary disease and left ventricular hypertrophy in asymptomatic CKD not requiring dialysis. *American Journal of Kidney Disease*. 2005;**46**:35-44
- [71] Iwanaga Y, Miyazaki S. Heart Failure, chronic kidney disease, and biomarkers—An integrated viewpoint. *Circulation Journal*. 2010;**74**(7):1274-1282
- [72] Ynstead A, Landro L, Uelan T, Dahl CP, Flo TH, Vinge LE, et al. Increased Systemic and Myocardial expression of neutrophil gelatinase-associated lipocalin in clinical and experimental heart failure. *European Heart Journal*. 2009;**30**(10):1229-1236
- [73] Twardowski ZJ, Nolph KDA, Prowant LBF, Moore HL, Nielsen MP. Peritoneal equilibrium test. *Peritoneal Dialysis Bulletin*. 1987;**7**(3):138-147
- [74] Cueto-Manzano AM. Peritoneal dialysis in Mexico. *Kidney International*. 2003;**63**(83):s90-s92
- [75] Scott BK, Walker M, Margetts PJ, Kundhal KK, Rabbat CG. Metanalysis: Peritoneal membrane transport, mortality and technique failure in peritoneal dialysis. *Journal of the American Society of Nephrology*. 2006;**17**:2591-2598
- [76] Chávez Valencia V, Orizaga de la Cruz C, Pazarin Villaseñor HL, Fuentes Ramírez F, Parra Michel R, Aragaki Y, et al. Frequency of peritoneal transport in a population of the Hospital General Regional No. 46, Instituto Mexicano del Seguro Social. *Gaceta Médica de México*. 2014;**150**(2):186-193

- [77] Waniewski J, Debowska M, Lindholm B. Water and solute transport through different types of pores in peritoneal membrane in CAPD patients with ultrafiltration failure. *Peritoneal Dialysis International*. 2009;**29**(6):664-669
- [78] Sobiecka D, Waniewski J, Weryński A, Lindholm B. Peritoneal fluid transport in CAPD patients with different transport rates of small solutes. *Peritoneal Dialysis International*. 2004;**24**(3):240-251
- [79] Cueto-Manzano AM. Rapid solute transport in the peritoneum: Physiologic and clinical consequences. *Peritoneal Dialysis International*. 2009;**29**(2):s90-s95
- [80] Jeznach-Steinhagen A, Słotwiński R, Szczygiał B. Malnutrition, inflammation, atherosclerosis in hemodialysis patients. *Roczniki Panstwowego Zakladu Higieny*. 2007;**58**(1):83-88
- [81] Abe M, Okada K, Maruyama T, Maruyama N, Matsumoto K, Soma M. Relationship between erythropoietin responsiveness, insulin resistance, and malnutrition-inflammation-atherosclerosis (MIA) syndrome in hemodialysis patients with diabetes. *International Journal of Artificial Organs*. 2011;**34**(1):16-25
- [82] Chung SH, Stenvinkel P, Bergström J, Lindholm B. Biocompatibility of new peritoneal dialysis solutions: What can we hope to achieve? *Peritoneal Dialysis International*. 2000;**20**(5):S57-S67
- [83] Teixidó-Planas J, Troya-Saborido MI, Pedreira-Robles G, Del-Rio-Lafuente M, Romero-Gonzalez R, Bonet-Sol J. Measuring peritoneal absorption with the prolonged peritoneal equilibration test from 4 to 8 hours using various glucose concentrations. *Perit Dial Int*. 2014;**34**(6):605-11
- [84] Aguirre AR, Abensur H. Protective measures against ultrafiltration failure in peritoneal dialysis patients; *Clinics (São Paulo, Brazil)*. 2011;**66**(12):2151-2157
- [85] Fusschoeller A. Histomorphological and functional changes of the peritoneal membrane during long-term peritoneal dialysis. *Pediatric Nephrology*. 2008;**23**:19-25
- [86] La Milia V, Di Filippo S, Crepaldi M, Del Vecchio L, Dell'Oro C, Andrulli S, Locatelli F. Mini-peritoneal equilibration test: A simple and fast method to assess free water and small solute transport across the peritoneal membrane. *Kidney International*. 2005;**68**:840-846
- [87] Kawaguchi Y, Kawanishi H, Mujais S, Topley N, Oreopoulos DG. Encapsulating peritoneal sclerosis: Definition, etiology, diagnosis, and treatment. International Society for Peritoneal Dialysis Ad Hoc Committee on Ultrafiltration Management in Peritoneal Dialysis. *Peritoneal Dialysis International*. 2000;**20**:S43-S55
- [88] Flessner MF. Peritoneal ultrafiltration: Mechanisms and measures. *Contributions to Nephrology*. 2006;**150**:28-36
- [89] Prasad N, Gupta S. Ultrafiltration failure in peritoneal dialysis: A review. *Indian Journal of Peritoneal Dialysis*. 2012;**22**(1):15-24

- [90] Heimbürger O, Waniewski J, Werinski A, Tranaeus A, Lindholm B. Peritoneal transport in CAPD patients with permanent loss of ultrafiltration capacity. *Kidney International*. 1990;**38**:495-506
- [91] Ha SK. Dietary salt intake and hypertension. *Electrolyte Blood Press*. 2014;**12**:7-18
- [92] Inal S, Erten Y, Akbulu G, et al. Salt intake and hypervolemia in the development of hypertension in peritoneal dialysis patients. *Advances in Peritoneal Dialysis*. 2012;**28**:10-15
- [93] Biesen WV, Vanholder R, Veys N, Lameire N. Improving salt balance in peritoneal dialysis patients. *Peritoneal Dialysis International*. 2005;**25**(S3):S73-S75
- [94] Wang AY, Brimble KS, Brunier G, et al. ISPD Cardiovascular and Metabolic Guidelines in Adult Peritoneal Dialysis Patients. Part I—Assessment and management of various cardiovascular risk factors. *Peritoneal Dialysis International*. 2015;**35**:379-387
- [95] Tzamaloukas AH, Saddler MC, Murata GH, et al. Symptomatic fluid retention in patients on continuous peritoneal dialysis. *Journal of the American Society of Nephrology*. 1995;**6**:198-206
- [96] Ates K. Salt and water in PD. The Turkish contribution. *Peritoneal Dialysis International*. 2008;**28**(3):224-228
- [97] Inal S, Erten Y, Tek N, et al. The effect of dietary salt restriction on hypertension in peritoneal dialysis patients. *Turkish Journal of Medical Sciences*. 2014;**44**(5):814-819
- [98] Fine A, Fontaine B, Ma M. Commonly prescribed salt intake in continuous ambulatory peritoneal dialysis patients is too restrictive: results of a double-blind crossover study. *Journal of the American Society of Nephrology*. 1997;**8**:1311-1314
- [99] McMahon EJ, Campbell KL, Mudge DW, Bauer JD. Achieving salt restriction in chronic kidney disease. *International Journal of Nephrology*. 2012;**2012**:720429
- [100] Dong J, Li Y, Yang Z, Luo J. Low dietary sodium intake increases the death risk in peritoneal dialysis. *Clinical Journal of the American Society of Nephrology*. 2010;**5**:240-247
- [101] Cheng LT, Wang T. Changes in total sodium intake do not lead to proportionate changes in total sodium removal in CAPD patients. *Peritoneal Dialysis International*. 2006;**26**(2):218-223
- [102] Bae SY, Kim SB, Kim SM. Simple method to estimate daily sodium intake during measurement of dialysis adequacy in chronic peritoneal dialysis patients. *Clin Nutr*. 2016;**35**(1):S191-S192
- [103] Haddad N, Shim R, Hebert LA. Nutritional management of water, sodium, potassium, chloride, and magnesium in kidney disease and kidney failure. *Nutr Manag Renal Dis* Chapter 22: 323-338, 2013
- [104] Yip T, Wan W, Hui PC, Lui SL, Lo WL. Severe hyperkalemia in a peritoneal dialysis patients after consumption of salt substitute. *Perit Dial Int*. 2012;**32**(2):206-208