Role of Peritoneal Ultrafiltration in Heart Failure Treatment

Kalp Yetmezliği Tedavisinde Peritoneal Ultrafiltrasyonun Yeri

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Abstract

Cardiorenal syndrome (CRS) is a general term that can reflect different clinical conditions in which cardiac and renal dysfunctions coexist. The main pathogenetic mechanisms playing a role in heart failure (HF) and CRS are neurohumoral adaptation, right ventricular dilatation and dysfunction and systemic inflammation. Persistence of these factors cause focal and segmental glomerulosclerosis, and tubulointerstitial fibrosis in the renal parenchyma. Diuretics, beta blockers, renin-angiotensin-aldosterone system inhibitors, and vasodilators are the main medical treatments besides conventional approach, such as salt and water restriction and quitting smoking, in HF treatment. Diuretic resistance is the main problem emerging during diuretic treatments. Two renal replacement treatments have become prominent for removal of excess fluids via ultrafiltration in HF patients with diuretic resistance extracorporeal ultrafiltration with hemodialysis and peritoneal dialysis (PD). Herein, the role of these two ultrafiltration modalities, especially peritoneal ultrafiltration (PUF) in the treatment of HF is discussed. The main studies and advantages of PUF in HF treatment were discussed. Moreover, effects of PD on glomerular filtration rate, hospitalization and mortality were investigated. In conclusion, PD is an alternative cheap, practical and convenient therapy in reducing cardiac volume burden in HF patients who do not respond well to standard treatments and/or require frequent hospitalization.

Keywords: Cardiorenal syndrome, heart failure, peritoneal dialysis, ultrafiltration

Anahtar Sözcükler: Kardiorenal sendrom, kalp yetmezliği, periton diyalizi, ultrafiltrasyon

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Introduction

Heart failure (HF) is a medical condition with high mortality rate affecting quality of life adversely with accompanying symptoms and frequent hospitalization. According to the classification by ejection fraction (EF) in current cardiology guidelines, an EF of ≥50% is defined as HF with preserved EF, an EF <40% as HF with reduced EF and an EF between 40-49% is classified as “grey zone” (1).

The prevalence is 1-2% in adult population in developed countries, however, this rate is above 10% in individuals older than 70 years of age (2-4). In the HF prevalence and predictors in Turkey (HAPPY) study performed in people aged ≥35 years in our country, the prevalence of HF was found to be higher than in western countries (absolute 2.9%, estimated 6.9%) (5).

The heart-kidney interaction has bidirectional nature. Cardiac diseases are associated with decreased kidney functions and progression in kidney diseases, whereas chronic kidney disease is an independent risk factor for cardiovascular events (6,7). Glomerular filtration rate (GFR) is <60 mL/min in 30-60% of patients with HF (8-11). Renal dysfunction has a greater impact on mortality than impaired cardiac function [EF and New York Heart Association (NYHA) class] in advanced HF patients (10,12).

Cardiorenal Syndrome

Cardiorenal syndrome (CRS) is a general term that can reflect different clinical conditions in which cardiac and renal dysfunctions coexist. CRS which is an interaction between cardiovascular and renal systems was described by the National Heart, Lung, and Blood Institute in 2004 as worsening of the renal function due to deterioration in left ventricular function (13). However, the classification was made with consideration of chronicity of disease and affected organs, because this description do not include details about different clinical conditions affecting the heart and kidney (14).

- Type 1: Acute worsening of cardiac function leading to renal dysfunction,
- Type 2: Chronic abnormalities in cardiac function leading to renal dysfunction,
- Type 3: Acute worsening of renal function causing cardiac dysfunction,
- Type 4: Chronic abnormalities in renal function leading to cardiac disease (coronary artery disease, heart failure (HF), arrhythmia, etc.),
- Type 5: Systemic conditions (sepsis, diabetes mellitus, systemic lupus erythematosus, etc.) causing simultaneous dysfunction of the heart and kidney.

The main pathogenetic mechanisms playing role in HF and CRS are listed below;

a) Neurohumoral Adaptation

The stroke volume decreases as a result of left ventricular dysfunction. As a response, systemic arterial vasoconstriction occurs to protect the perfusion to two vital organs (heart and brain). Whenever afterload increases with vasoconstriction, deterioration in renal perfusion occurs.

Neurohumoral adaptation mechanisms, such as renin-angiotensin-aldosterone system (RAAS) and sympathetic nervous system (SNS) activation, and compensatory elevation of antidiuretic hormone (ADH) occur as a response to decreased EF. Blood urea nitrogen (BUN) level might be an indicator of neurohumoral activation in HF and, a high BUN level is related with mortality in HF (15).

Renin-angiotensin-aldosterone system activation:

Tubuloglomerular feedback occurs as a result of decreased amount of sodium reaching the macula densa as a result of decreased renal perfusion pressure in HF. Then vasodilatation of the afferent arteriole occurs while RAAS is activated with secretion of renin from the macula densa. Vasoconstriction of the efferent arteriole occurs via activated RAAS and water and sodium reabsorption increases in the kidney as well.

Antidiuretic hormone: With increase ADH secretion by stimulation of non-osmotic receptors which is response to decreased effective systemic arterial pressure; free water reabsorption increases in collective tubules; simultaneously arterial and venous vasoconstriction occurs via V1a receptors. At the same time, arterial and venous vasoconstriction occurs via vascular V1a receptors. As a result of these mechanisms, the cardiac preload and afterload increase (16).

Sympathetic nervous system activation:

Baroreceptors located in the aorta are stimulated by decreased stroke volume as a result of HF and by this way, SNS becomes activated and systematic vasoconstriction occurs.

Ongoing RAAS and SNS activation, increased ADH level and systemic inflammation with HF cause focal and segmental glomerulosclerosis and tubulointerstitial fibrosis in the renal parenchyma (17,18).

b) Right Ventricular Dilatation and Dysfunction

Two pathogenic mechanisms causing left ventricular filling restriction are discussed in this topic. The first one is increased renal venous pressure and the other one is increased right ventricular pressure (reverse Bernheim phenomenon) (19). Increased systemic congestion due to right ventricular dysfunction independent of left ventricular EF has a negative effect on the kidney as well as other organs. There have been studies reporting that increased right atrial and central venous pressure were related with loss of renal function and, renal function recovery.
was observed with decreased systemic congestion (20-26). Although it is not clear, proinflammatory cytokines secreted as a result of endothelial stretch might be responsible (27).

**Conventional Treatment of Heart Failure**

Diuretics, beta blockers, RAAS inhibitors, and vasodilators are the main medical treatments besides conventional approach such as salt and water restriction and quitting smoking in patients with HF. Intravenous inotrope therapy, cardiac resynchronization treatment, mechanical ventilation support, and cardiac transplantation are more aggressive approaches (28).

Diuretics are effective in relieving congestive symptoms by removing excess fluid from the body. They activate RAAS and SNS and lower systemic blood pressure and, GFR. Rebound sodium reabsorption from the tubules decreasing the efficiency of the treatment can be seen in diuretic users. Combined diuretic use is needed to prevent this condition, however, side effects such as decreased GFR, hyponatremia, and hyperkalemia may develop (29-34). Although the frequency of diuretics use is high, mineralocorticoid-receptor antagonists are the only diuretic effective on survival (35).

Diuretic resistance is another problem emerging during diuretic treatments. Weight loss is not observed in approximately 20% of patients having intravenous diuretic treatments who are hospitalized because of acute decomposed HF and these patients are accepted as diuretic resistant. None compliance with sodium restriction, RAAS activation secondary to intravascular volume decrease following diuretic treatment, subsequence Na-Cl cotransporter hyperplasia and distal tubule hypertrophy, following sodium blockage in the thin ascending limb of the Henle’s loop may be the mechanism contributing to diuretic resistance (36,37).

Two renal replacement treatments become prominent for removal of excess fluids via ultrafiltration (UF) in HF patients with diuretic resistance: extracorporeal (EC) UF and PD.

**Extracorporeal Ultrafiltration with Hemodialysis in Heart Failure**

EC-UF with hemodialysis (HD) is thought to be an alternative or supportive to conventional treatment in HF. EC-UF has been studied in type 1 and 3 CRS patients who were admitted with acute decomposed congestive HF. In addition to this, the importance of EC-UF for type 2 and 4 CRS patients, who do not need renal replacement treatment, is less known than that for type 1 and 3 CRS patients.

There have been three large randomized studies [Ultrafiltration versus Intravenous Diuretics for Patients Hospitalized for Acute Decompensated Congestive HF (UNLOAD), Relief for Acutely-Fluid Overloaded Patients with Decompensated Congestive HF (RAPID-CHF) and Cardiorenal Rescue Study in Acute Decompensated-HF (CARRRESS-HF)] which compared diuretic treatment and EC-UF in acute decomposed HF. Mean creatinine levels in patients who were included in those studies were 1.5, 1.7 and 2.0 mg/dL, respectively. One hundred eighty-eight patients were enrolled in CARRRESS-HF study and more intensive diuretic treatment was administered compared to that in the other two studies (thiazide diuretic was added to the intravenous diuretic treatment after bolus administration of vasodilator and/or intravenous inotropes were given to selected cases). Weight loss via treatment was similar between the two groups (p>0.05) whereas worsening in renal function and frequency of side effects were found to be significantly high in UF group (p=0.003 and p=0.003, respectively). UNLOAD study consisted of 200 patients; weight loss was greater in UF group whereas the duration of hospitalization was found to be shorter than in other group (p=0.003, p=0.022, respectively). In RAPID-CHF study that had a smaller sample size (40 patients), weight loss was found to be greater in UF group.

When the follow-up durations in the three large randomized studies, UNLOAD, RAPID-CHF, and CARRRESS-HF, are analyzed there is not enough information to discuss the effect of EC-UF on long-term morbidity and mortality (90, 30, 60 days, respectively).

In the literature, there are not enough studies which compared HD and peritoneal dialysis (PD) in HF patients having renal replacement treatment because of end-stage renal disease. In a study including 107.922 patients with the diagnosis of HF between 1995 and 1997, newly diagnosed end-stage renal disease patients receiving renal replacement treatment were followed up for two years. At the end of the follow-up period, the mortality rate was found to be higher in PD group than in HD patients (38). However, the PD technique was not well-developed at the time of the study; thus, patients receiving PD were prone to complications. In addition, ‘icodextrin’ which was especially chosen for HF patients, and low glucose degradation product solutions were not available in these years. In another study in which ‘French Renal Epidemiology and Information Network Registry’ data for the years between 2002 and 2008 were used, mortality rate among patients, who were followed up with HF diagnosis and were started PD and HD (933 and 3468 patients, respectively) as renal replacement treatment, when needed, was compared and survival was found to be significantly higher in HD group than in PD group (HD group 36.7 months, PD group 20.4 months; HF: 1.48) (39). However, the average age of the patients and NYHA
stage in the PD group were higher than in HD group in this study. Further randomized controlled trials are needed to clarify this issue.

**Why Peritoneal Dialysis is Chosen in the Treatment of Heart Failure?**

The use of PD in HF dates back to the 1940’s (40). When PD is compared with diuretic treatment, neurohumoral activation (SNS activation, RAAS activation, endothelin, ADH, increase in atrial natriuretic peptide secretion), which occurs because of abrupt volume depletion with diuretics, causes vicious cycle in HF pathogenesis and increases rebound water and sodium absorption. The peritoneal dialysis does not introduce this vicious cycle because it removes the excess fluid from the body continuously and in a slow manner (41). However, there are no abrupt changes in renal hemodynamics with UF which is made slowly and controlled by PD and, by this way, PD has superiority to diuretics by preserving residual renal function (42).

Filtration in PD is isosmotic to the plasma, but fluid loss with diuretic treatment is more hypotonic (43,44). When the amount of sodium removal is compared between peritoneal UF (PUF) and diuretic treatment, 130-150 mmol/L sodium is removed by PUF and 50-100 mmol/L sodium is removed by diuretic treatment (45). In the light of this information, PUF seems to be more efficient than diuretic treatment to excrete excess fluid and sodium from the body in patients with HF.

RAAS blockers, which are proven to decrease mortality in HF patients, can not be used in some patients due to the side effects associated with hyperkalemia. Since potassium is not present in peritoneal dialysate, better potassium control can be provided and this gives opportunity to maximum congestive HF treatment which is also important with regard to survival.

Cytokines such as atrial natriuretic peptide, tumor necrosis factor-α, interleukin-1, and interleukin-6 are known to increase apoptosis of cardiac myocyte and to have negative inotropic effect. These mediators whose molecular weight ranges between 500 and 30000 Dalton can penetrate from the peritoneal membrane, by this way PD allows clearance of these agents while contributing to the support for the heart directly (46,47).

Unlike extracorporeal UF, PD feasible at home and offers additional advantages in terms of cost, other advantages of PD in HF. Especially in patients with HF who are planned to undergo PD for UF the use of ‘icodextrin’ can provide long-term UF, reduces the number of fluid exchange, thus reducing the load on fluid exchange (48,49). The advantages of PUF in HF are summarized in Table 1.

### Peritoneal Dialysis in Heart Failure

#### The Effects on Hospitalization

HF is one of the diseases in which recurrent hospitalization is frequently required. It has been shown that frequent and long-term hospitalization is related with high mortality (50-54). In the CHARM study, 7572 patients with preserved or low EF were analyzed for hospitalization frequency and mortality. Mortality after hospitalization was increased [HF 3.2; 95% confidence interval (CI) 2.8-3.5] and this risk was the highest in the first month and then decreased gradually (50).

Decrease in the number of hospital admissions is quite important for protection of life quality of patient and also for national economy. Size of patient population and cost of treatment become an important part of medical expenditures of countries estimated economical cost of HF in the United State of America) was about 30.7 billion dollars in 2012, and this amount was expected to increase to 69.7 billion dollars in 2030 (55).

It is thought that by decreasing hospitalization rate in HF patients, life quality will get better, economical burden will get reduced, and mortality rate will decrease in long term. PUF was found to decrease the rate of hospitalization because of acute decomposed HF in several studies (56-63).

#### Can Peritoneal Dialysis Decrease Mortality Rate?

Mortality rates are quite high in patients followed up for HF. In a study, five-year mortality after diagnosis was found to be 52.6% for HF patients, 24.4% in patients under sixty years old, and 54.4% in patients above 80 years old (64). This rate is higher in NHYA III-IV group patients. When expected survival in this patient group and the mean age of patient who were included in the study are taken into consideration, the effect of treatment regimen which was started during follow-up is hardly determined. Besides, heterogeneity of low number patients makes difficult randomization when the presence of additional diseases

<table>
<thead>
<tr>
<th>Table 1. The advantages of peritoneal ultrafiltration in heart failure treatment</th>
</tr>
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<tbody>
<tr>
<td>Slow and controlled in PD</td>
</tr>
<tr>
<td>Effective in protecting residual renal functions</td>
</tr>
<tr>
<td>Prevents rebound neurohumoral activation</td>
</tr>
<tr>
<td>Higher amount of removed sodium besides diuretic treatment</td>
</tr>
<tr>
<td>Eliminates cytokines that may have negative effects on the heart</td>
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<tr>
<td>Conventional treatment is more efficient in terms of cost</td>
</tr>
<tr>
<td>Reduces hospitalization</td>
</tr>
<tr>
<td>Regression in heart failure related symptoms and providing increase in ejection fraction</td>
</tr>
</tbody>
</table>

PD: Peritoneal dialysis
in HF patients, number and variety of medical treatments, and the number of patients in the studies are considered. In a prospective study by Núñez et al. (65) in 2012, it was reported that the mortality rate was lower in PD group than in conventional treatment group (HF = 0.40; 95% CI, 0.21-0.75; p=0.005).

There is a need for further randomized controlled studies to show the relationship between mortality and PUF precisely.

**How does Peritoneal Dialysis Affect Ejection Fraction or New York Heart Association Class?**

An increase in ventricular preload promotes increases in end-diastolic volume, the length of muscle fiber; leading to increase in ventricular contraction force. Than the stroke volume and cardiac output rapidly increases (Frank-Starling law). Cardiac output falls down due to decreased contractility in HF, the body increases end-diastolic volume by increasing water and sodium absorption in the framework of the Frank-Starling law to increase stroke volume. The diastolic end volume, which is increased compensatory beyond the physiological limits, is responsible for pulmonary venous congestion and congestive symptoms.

Excess fluid is cleared slowly from the body by PD without rebound mechanisms are enabled, thus, an increase in EF and a decrease in symptoms are expected according to the Frank-Starling law. One reason for the increase in EF is thought to be the removal of cardio depressant agents by PD (66). In many of these studies, the exercise capacities of HF patients were assessed by the NYHA classification and the results shows that NHYA class regresses and EF increases with PUF (58,67-73).

**How does Peritoneal Dialysis Affect Glomerular Filtration Rate?**

An increase in serum creatinine level is observed in 21-45% of patients admitted with acute decompensated HF (74-77). Activation of RAAS and SNS due to reduced cardiac output volume and the effects of increased intra-abdominal pressure (IAB) on the kidney are thought to be the causative mechanisms (24). One of the mechanisms of the effect of the IAB on the kidney is the increase of the interstitial pressure in the kidney by the reflex of the increased IAB to the renal veins resulting in renal ischemia. This condition can be similar to ischemic hepatitis occurring in HF. It has been showed that lowering IAB in HF patients improves renal function (20-26). On the other hand, unlike CARRESS, which was one of the three major studies comparing diuretic treatment with extracorporeal UF in patients with acute decompensated HF, weight loss was similar in both groups while the rate of deterioration in renal function in the extracorporeal UF group was greater than in the UF group (p=0.003) (78).

In two prospective studies, the study by Kunin et al. (63) with 37 patients and the study of Koch et al. (69) with 118 patients in which PUF and diuretic therapy used in the long-term treatment of patients with HF diagnosis were compared in the same patient groups, and the patients were observed before and after PUF, a decrease in GFR was found after P9). In many of the other studies that examined the effect of PUF on GFR, PUF was found to be ineffective on GFR (42,56,68,79). In a meta-analysis including six studies with a total of 282 patients without indications of renal replacement therapy, there was no significant difference in GFR before and after PUF (80). However, the analysis of the effect of GFR before and after treatment on mortality is quite difficult because of the small and heterogeneous group of patients, the lack of controlled studies, and the high expected mortality rates in patients. In addition, single-centered, non-randomized studies including small patient groups do not seem to be sufficient to predict this issue since adjustment of the volume status in patients with HF is highly sensitive and renal ischemia and decline in GFR might be thought except for the conditions in the case of hypervolemia in which pre-renal failure is seen. Possible loss of GFR during the follow-up in these patients is another question mark. The main studies on PUF in patients with HF are summarized in Table 2.

**In Which Patient Group Peritoneal Ultrafiltration may be Recommended?**

Mental and physical capacity of the patient to be able to pay attention to PD independent of the disease, is essential for performing PD. UF is not recommended for routine treatment by the cardiac failure guidelines, and is recommended for a group of patients unresponsive to diuretic therapy and for those with resistant volume loading (1).

Most of the studies about the use of PUF in diuretic-resistant HF patients found promising results, such as a reduction in hospitalization and mortality rates, increase in EF, and decrease in NHYA grade. In the light of the data in the literature, we recommend PUF for patients with NHYA class 3-4 who are hospitalized with the diagnosis of acute decompensated HF 2-3 times per year despite standard medical care. However, there is a need for further randomized studies in these groups of patients. The main issues about which patient group followed up with HF should be considered for PUF are summarized in Table 3.
**Peritoneal Dialysis Catheter Placement Procedure and Peritoneal Dialysis Prescription**

The use of PD for UF in patients with HF is a very practical and easy-to-use treatment method for units having the necessary equipment and devices. In patient-based assessments, good results are obtained in experienced centers. One of the most important issues that should not be overlooked here is that cardiologists should have a high awareness of the fact that PD is a treatment options for refractory HF patients.

Patients should be trained appropriately about technical and hygienic details and, the role of these details should be discussed carefully with the patient.

After the patient is selected, percutaneous insertion of the PD catheter with local anesthesia may be preferred in this group of patients because of the high risk of anesthesia. Here, a different strategy can be considered for each patient, taking into account the experience of the center, the experience of the surgeon and the clinical situation of the patient. It should also be noted that this group of patients has a high rate of antiaggregants or anticoagulants use. Bleeding is less likely to occur in midline cuts, and also there are centers that do this procedure with local anesthesia and without stopping anticoagulation or antiaggregan therapy. Beginning to use the catheter immediately after insertion increases the rate of mechanical complications such as leakage from the catheter site. Stegmayr et al. (81) from Sweden proposed in their prospective study of 61 patients in which using a

![Table 3. In which conditions should peritoneal ultrafiltration be recommended for patients with heart failure?](image)

<table>
<thead>
<tr>
<th>Study</th>
<th>Type of study, years</th>
<th>Number of the patients</th>
<th>Mean of age (years)</th>
<th>Follow up period (month)</th>
<th>Compared situations</th>
<th>Effect of PD; on hospitalization*, on GFR</th>
<th>Effect of PD; on NYHA, on EF, on PAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Courivaud et al. (58)</td>
<td>Retrospective two center 2014</td>
<td>126</td>
<td>72±11</td>
<td>16±16</td>
<td>Before and after PD at the same patient group</td>
<td>Decrease in hospitalization</td>
<td>N/A on GFR</td>
</tr>
<tr>
<td>Bertoli et al. (56)</td>
<td>Retrospective multicenter 2014</td>
<td>48</td>
<td>74±9</td>
<td>At least 6 months</td>
<td>Before and after PD at the same patient group</td>
<td>Decrease in hospitalization non effective on GFR</td>
<td>Decrease on NYHA increase in EF N/A on PAP</td>
</tr>
<tr>
<td>Kunin et al. (63)</td>
<td>Prospective 2013</td>
<td>37</td>
<td>66 (median)</td>
<td>42</td>
<td>Before and after PD at the same patient group</td>
<td>Decrease in hospitalization decrease in GFR</td>
<td>Decrease on NYHA non effective on EF N/A on PAP</td>
</tr>
<tr>
<td>Núñez et al.** (65)</td>
<td>Prospective 2012</td>
<td>62</td>
<td>74</td>
<td>16 (median)</td>
<td>PD and control group</td>
<td>Decrease in hospitalization</td>
<td>N/A on NYHA non effective on EF N/A on PAP</td>
</tr>
<tr>
<td>Koch et al. (69)</td>
<td>Prospective single center 2012</td>
<td>118</td>
<td>73±11</td>
<td>13.3±14</td>
<td>Before and after PD at the same patient group</td>
<td>N/A on hospitalization decrease in GFR</td>
<td>Decrease on NYHA non effective on EF non effective on PAP</td>
</tr>
<tr>
<td>Sánchez et al. (68)</td>
<td>Prospective single center 2010</td>
<td>17</td>
<td>64±9</td>
<td>15±9</td>
<td>Before and after PD at the same patient group</td>
<td>Decrease in hospitalization non effective on GFR</td>
<td>Decrease on NYHA non effective on EF non effective on PAP</td>
</tr>
<tr>
<td>Nakayama et al. (42)</td>
<td>Prospective single center 2010</td>
<td>12</td>
<td>81±5</td>
<td>26.5 (median)</td>
<td>Before and after PD at the same patient group</td>
<td>N/A on hospitalization non effective on GFR</td>
<td>Decrease on NYHA non effective on EF N/A on PAP</td>
</tr>
<tr>
<td>Gotloib et al. (47)</td>
<td>Prospective single center 2005</td>
<td>20</td>
<td>65±7</td>
<td>19.8±7.3</td>
<td>Before and after PD at the same patient group</td>
<td>Decrease in hospitalization N/A on GFR</td>
<td>Decrease in NYHA N/A on EF N/A on PAP</td>
</tr>
</tbody>
</table>

*Admission to acute decomposed heart failure

**Mortality was reduced with peritoneal dialysis

PD: Peritoneal dialysis, GFR: Glomerular filtration rate, NYHA: The New York Heart Association, EF: Ejection fraction, PAP: Pulmonary artery pressure, N/A: Not available
Exposed to glucose toxicity (56,83). Channels is not observed and also the patient is not that can be seen in glucose-based fluids using aquaporin from pores, the so-called 'sodium sieving' phenomenon allows single change, appears to be a more practical and appropriate option.

Because fluid passage through 'icodextrin' is mostly from pores, the so-called 'sodium sieving' phenomenon that can be seen in glucose-based fluids using aquaporin channels is not observed and also the patient is not exposed to glucose toxicity (56,83).

Conclusion

In conclusion, PD is an alternative therapy that can be used to reduce cardiac volume burden in patients who do not respond well to standard treatments and require frequent hospitalization. It is a cheap, practical and convenient method when appropriate treatment is scheduled to appropriate patient at the appropriate center.

Authorship Contributions


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