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RESIDUAL RENAL FUNCTION AND CARDIOVASCULAR COMPLICATIONS IN PATIENTS ON PERITONEAL DIALYSIS

SUMMARY

Cardiovascular diseases are the leading cause of death in patients on maintenance dialysis, accounting for more than half of all deaths in patients on peritoneal dialysis. The patients on PD are at increased risk of cardiovascular morbidity and mortality due to the presence of traditional risk factors and non-traditional, uremic factors. The prevalence and impact of non-traditional risk factors is more pronounced with decline of residual renal function (RRF). Patients on PD without RRF had worse metabolic and cardiovascular profile (high grade anemia, resistance to erithropoietin, high Ca^{2+} x PO_4^{3-} product, worse inflammation and malnutrition, severe hypertension and left ventricular hypertrophy (LVH), and higher cardiovascular mortality compared to patients with preserved RRF. Preservation of the residual renal function could improve the cardiovascular outcome in patients on PD.

Key words: **residual renal function, peritoneal dialysis, cardiovascular complications**

INTRODUCTION

According to U.S. Renal Data System (USRDS*)* and European Renal Association-European Dialysis and Transplantation Association registry, the mortality in patients in the end-stage renal disease is 10-20 times higher compared to the general population (1). Cardiovascular diseases are the leading cause of death in patients on maintenance dialysis, accounting for more than half of all deaths in patients on peritoneal dialysis (PD) (1). The patients on PD are at increased risk of cardiovascular morbidity and mortality due to the presence of traditional risk factors (diabetes melitus, obesity, hypertension, hyperlipidemia, smoking habits, insulin resistence) as well as non-traditional, uremic factors (anemia, extracellular fluid overload, hyperphosphatemia, secondary hyperparathyroidism, hyperhomocystinemia, oxidative stress, micro-inflammation, endothelial disfunction, sympatetic overactivity) (1, 2). The prevalence and impact of non-traditional risk factors is more pronounced with decline of residual renal function (1). Preservation of the residual renal function could improve the cardiovascular outcome in patients on PD (1).

Early detection and refferal of the patients with increased cardiovascular risk on PD, as well as up-to-date therapic strategy could improve the quality of life of these patients and reduce cardiovascular morbidity and mortality (1, 2).

Residual renal function in patients on peritoneal dialysis

Residual renal function (RRF) is a very important parameter to monitor in patients on chronic renal replacement therapy. The loss of RRF is defined as exponential decline in glomerular filtration rate after commencing with dialysis

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treatment (3). Many factors are known to have some impact on decline of RRF (Table 1): the disease of the native kidneys, nephrotoxicity of the drugs used and radiocontrast media, blood pressure control (hypertension and hypotension), fluid balance of the patient (hypervolemia or hypovolemia), biocompatibility of peritoneal dialysis solutions, peritonitis rate, etc. (3).

Other factors related to the rapid decline of GFR were supposed to be: being older than 60 years of age, automated peritoneal dialysis (APD) rather than continuous ambulatory peritoneal dialysis, mean blood pressure higher than 110 mmHg, and serum atrial natriuretic peptide level higher than 60 pg/dL (4).

Table 1. Risk factors for RRF decline in peritoneal dialysis

1. Primary kidney disease
2. Comorbidities: diabetes melitus, congestive heart failure
3. Cardiovascular (hemodynamic) instability:
   - arterial hypertension
   - arterial hypotension
   - dehidration
4. High body mass index (BMI)
5. Peritoneal dialysis vintage
6. High peritoneal membrane transport on start of PD treatment
7. High net peritoneal ultafiltration
8. Nefrotoxic agents:
   - aminoglycosides
   - non-steroid antiinflammatory drugs
   - radiocontrast media
9. High peritonitis rate
10. Microinflammation: bioincompatibility of peritoneal dialysis solutions
11. Heavy proteinuria
12. Non-compliant patient: high protein intake

RRF estimation in patients on peritoneal dialysis

Residual renal function in patients on PD is estimated to be an average value of the sum of urea and creatinine clearances normalized to the body surface area (BSA), and express as liters/week (3). The equation for BSA is:

$$BSA = 0.007184 x BW(kg)^{0.425} x H(cm)^{0.725}$$

The equation for RRF is then:

$$RRF = \frac{(Curea + Ccreatinine)}{2} x 1.73 m^2 / BSA \quad \text{(L/week)}$$

In patients with minor daily urine output (less then 300 ml/24h), a 48-hours urine specimen collection is recommended. The placement of the urinary catheter is obligatory in patients with neurogenic bladder or m. detrusor disfunction (3). Another equation for GFR estimation is based on MDRD formula (mL/min/1,73m^2) (5):

$$GFR = -0.70 + 22 \text{ x} \left(\frac{1}{\text{Cystatin C}} \right)$$

Periodic estimation of RRF is important in delivering the adequate dose of peritoneal dialysis to the patient and is related to the clinical outcome (3-5).

The importance of RRF preservation in patients on PD

RRF is better preserved in patients on PD than on hemodialysis (3). Cardiovascular instability during hemodialysis along with release of inflammatory mediators due to bioincompatibility of the arteficial kidney are responsible for the observed differences in preservation of RRF (6).

Residual renal function is an independent predictor of survival in PD patients (6). The aim of the clinical study of CANUSA was to evaluate the relationship of peritoneal dialysis adequacy and nutritional status to mortality, technique failure, and morbidity in a prospective manner with 680 patients included commencing continuous peritoneal dialysis in 14 centers around Canada and United States during 28-month period (7). A clear association was found between the value of the whole clearance (sum of peritoneal and renal clearance) and survival with indexes based on urea kinetic modeling (a value of 2.1 for weekly Kt/V and a weekly CCr of 70 L/ 1.73 m2) each of them was associated with an expected 2-year survival of 78% (7). No differences regarding patients' survival related to the peritoneal or renal clearance were reported (7). Reanalysis of the data from the CANUSA study (8) indicated that the predictive power for mortality in PD patients was attributable to RRF and not to the whole dose of PD delivered. Every increase in RRF of 5L/week/ 1,73m^2 reduced the relative risk of death for 12%, and every increase of residual urine volume for 250 ml/day reduced the relative risk of death for 36% in patients on PD (8). Such a significant association was not confirmed separetely for peritoneal clearance (8). Similar results related to the impact of RRF to PD dialysis adequacy were shown in the NESCOSAD (Netherlands Cooperative Study on the Adequacy of Dialysis) (9), and later, in ADEMEX study (ADEquacy of peritoneal Dialysis in MEXico) (10).

In the first year on PD, the impact of RRF in archieving adequate dose of PD could be as much as 50% (6). Besides better clearance of uremic toxins in the range of middle molecules, preserved RRF provides better clearances of uremic toxins in the higher molecular weight range ($_2$-microglobulin, p-cresol, AGE-advanced glycation end products i AOPP-advanced oxidised protein products) (6). It seems that preserved RRF does have other important metabolic effects besides clearance of small molecules of urea and creatinine on better survival in patients on PD (6, 11). It was already shown that patients on PD without RRF had worse metabolic and cardiovascular profile (high grade anemia, resistance to erithropoietin, high Ca^{2+} x PO_4^{3-} product, worse inflammation and malnutrition, severe hypertension and left ventricular hypertrophy (LVH), and higher cardiovascular mortality compared to patients with preserved RRF (6, 11).

High blood pressure

RRF plays an important role in fluid balance in patients on PD (6, 12-14). Expanded extracellular volume (ECT) on PD could result from diminished RRF, ultrafiltration failure and non-compliant fluid intake (12-16). Patients with RRF less than 2,0 mL/min/1,73m^2 have statistically higher ECT compared to patients with RRF higher than 2,0 mL/min/1,73m^2 (3). In one study (17), the link between extracellular fluid expansion and systolic blood pressure was highlightened. Increased sodium pool due to the peritoneal membrane ultrafiltration failure, and/or reduced RRF is also important in maintaining expanded ECT and elevated blood pressure (18-24). Expanded ECT on the basis of reduced RRF, burden left ventricle with volume leading to eccentric left ventricle hypertrophy in PD patients (6, 18).

Left ventricle hypertrophy (LVH)

LVH is the most frequent cardiac alteration in ESRD, resulting from a combined pressure and volume overload *(Figure 1)*.

Cardiac work (LV minute work) is the product of stroke work (equals the product of LV pressure and stroke volume) and heart frequency (25). Myocardial oxygen consumption and energy expenditure increase with stroke work. Changes in LV wall stress during the cardiac cycle are, according to Laplace's law, result of the tensile stress (r) which is directly proportional to intraventricular pressure (P) and radius (r) and inversely proportional to ventricular wall thickness (h) according to the formula:r ¼ Pr/2h (25). That tensile stress is the major mechanical signal for LV remodeling (25). The consequence of pressure-volume-tension

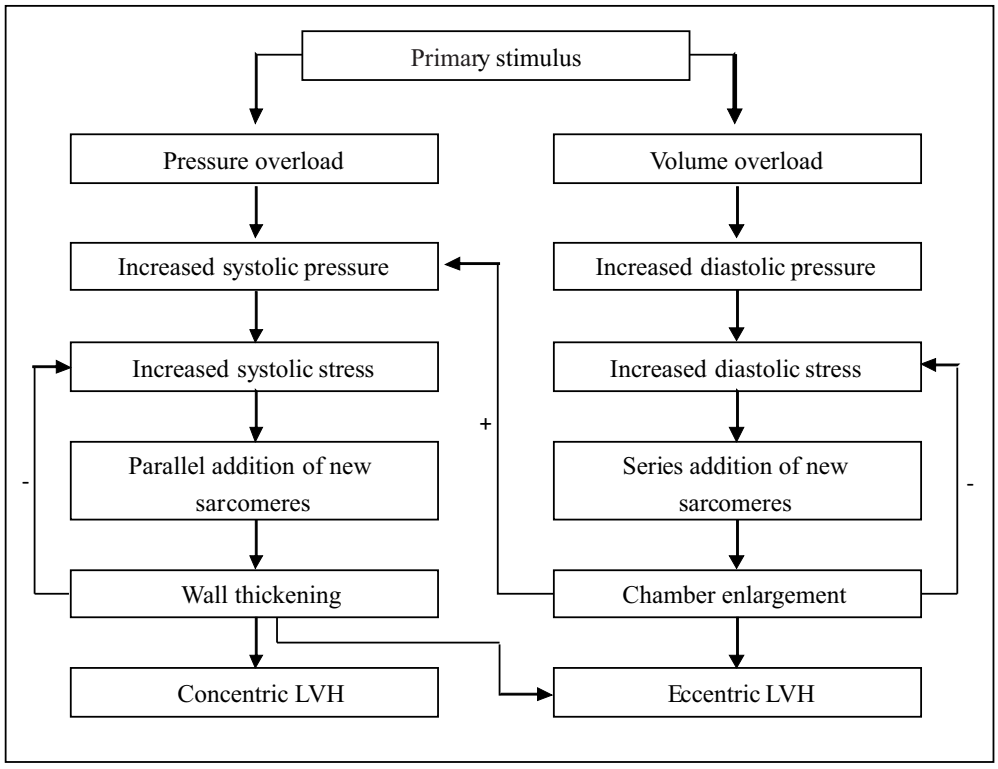

Figure 1.Hypothesis on patogenetic mechanisms of left ventricle hypertrophy

relationships is an increase in wall thickness (25). An increase in pressure or volume workload of the left ventricle is frequently associated with an increased release of neurotransmitters, hormones, and vasoactive substances that have a direct or permissive effect on the growth of cardiomyocyte and cardiac interstitium (25). LVH is both beneficial and detrimental. By distributing tension among a larger number of sarcomeres, LVH reduces the load of each individual muscle fiber and regulates cardiac efficiency and oxygen consumption, increasing the working capacity of the left ventricle (25). The beneficial effect permits maintenance of normal systolic function during the phase of compensated adaptive hypertrophy. While the beneficial effects of LVH dominate in the initial adaptation to overload, the sustained overload leads progressively to a maladaptive hypertrophic response (25).

The prevalence of LVH is already increased in early renal disease and progresses with a decrease in renal function (6). On starting dialysis, 75% of adults have LVH, with concentric hypertrophy in 42% of patients and eccentric hypertrophy in 44% of patients (26). In patients on maintenance hemodialysis, it is difficult to classify LVH into eccentric or concentric type because of the absence of steady-state conditions. Contraction of blood volume during hemodialysis decreases the left ventricle diameter and induces "acute" changes in relative wall thickness (26). The expansion of volume status to the left ventricle diameter leads to an overestimation of LV mass by echocardiography. It was also suggested to index LV mass to body height, because it provides better prediction of mortality and cardiovascular outcome (27). The increase in LV mass in ESRD patients results from a mild enlargement of LV end-diastolic diameter and/or an increase in LV wall thickness, and combines the features of eccentric and concentric hypertrophy (23).

RRF in patients on PD is longer well-preserved than on hemodialysis. Patients on PD with preserved RRF (RRF > 2,0 mL/min/1.73m^2) had a significantly smaller LV mass index, higher ejection fraction and fractional shortening of LV then patients without RRF (13).

Loss of RRF on PD is associated with high peritoneal membrane transport characteristics and expanded ECT, worsening of blood pressure control, diminished clearences of small, middle and high molecular weight uremic toxins, high grade anemia syndrome, inflammation-malnutrition syndrome and worsening in calcium phosphate balance with heart valves calcifications (6), which are all risk factors for further LVH progression.

The impact of anemic syndrome on cardiovascular function is also of paramount importance. Along with declining of RRF, anemia in PD patient is getting worse due to the deficit of erythropoietin. Anemia is associated with functional alterations that develop to maintain optimal oxygen delivery to tissues and organs and is achieved both by nonhemodynamic and hemodynamic adaptations. Nonhemodynamic adaptations include a lower affinity of hemoglobin for oxygen and increased oxygen extraction and AV difference. When the hemoglobin concentration declines to less than 100–120 g/L, a hemodynamic adaptation occurs since nonhemodynamic factors are insufficient to compensate (27). The most typical hemodynamic change observed is increased cardiac output due to high stroke volume and increased heart rate (27). Several mechanisms are responsible: reduction in arterial resistance due to arteriolar dilation and decreased blood viscosity, increased preload due to increased venous return, and increased left ventricle contractility attributed to sympathetic activity and noncatecholamine inotropic factors (27, 28). Each 10 g/L decrease in hemoglobin level was independently associated with left ventricle dilation (28). Finally, a similar relationship between hemoglobin level and LV mass has been demonstrated (28). Several studies in CRF and ESRD have shown that partial or complete correction of anemia with erythropoietin decreases the cardiac output and heart rate, and induces a partial regression of the LVH, principally in relation with decreased left ventricular end-diastolic diameter, while the effects on wall thickness are less evident (29-33).

Cardiovascular calcifications are increasingly recognized as a frequent and important complication in patients on dialysis. It is largely attributed to deranged mineral metabolism with resulting abnormal calcium–phosphorus control. Valvular calcification was recently identified as an important predictor of mortality and cardiovascular death in PD patients (34). PD patients exhibiting both valvular calcification and atherosclerotic vascular disease (as compared with patients having either or neither of these complications) had the highest mortality and cardiovascular death (35). Those results, together with recent findings of an association between heart valve calcification and carotid atherosclerosis (36) provide important evidence that valvular calcification represents a marker of atherosclerosis as well as a reflection of poor calcium and phosphorus control in PD patients. A recent study showed that serum fetuin-A, a circulating calcification inhibitory protein and a negative acute-phase reactant, is inversely associated with valvular calcification independent of CRP and a high Ca×P product (36). Furthermore, serum fetuin-A is predictive of mortality and cardiovascular death in PD patients. However,

no association was observed between RRF and serum fetuin-A (36).

A previous survey showed that, in at least 40% of chronic PD patients, serum phosphorus was elevated above 1.78 mmol/L (37) the target currently recommended by the Kidney Disease Outcomes Quality Initiative. More importantly, that study observed a strong inverse relationship between RRF and phosphorus control even when the average RRF of the PD patients fell below 2 mL/min/1.73 m2 (37). Another survey reported that at least one third of prevalent PD patients had heart valve calcification (38). Apart from the important association with Ca×P product, heart valve calcification was demonstrated to be closely linked to inflammation and malnutrition in PD patients. Even among patients without an excessive Ca×P product, the presence of inflammation and malnutrition was associated with an increased risk of valvular calcification (38).

The degree of inflammation is not only closely linked with atherosclerotic vascular disease, it is also associated with RRF, in which anuric PD patients show the greatest inflammatory response as measured either by CRP (39) or by soluble vascular cell adhesion molecule 1 (1). The exact mechanism underlying this latter association remains unclear. Loss of RRF (or uremia per se) has been suggested to possibly enhance an inflammatory response by increasing oxidative stress—a response that may lead to monocyte activation and cytokine production (40). On the other hand, inflammation has been closely linked with arterial stiffening, LVH and dilatation, and systolic dysfunction in PD patients (1). Importantly, loss of RRF, inflammation, and LVH are not only closely interrelated, they also combine adversely to increase the mortality and cardiovascular death risk in chronic PD patients (1). Similarly, circulating soluble vascular cell adhesion molecule 1 is also associated with RRF and LVH in PD patients. In addition, the association between loss of RRF and increased mortality and cardiovascular event risk is partly mediated via the close associations of RRF with inflammation and endothelial activation (1). In PD patients, the combination of inflammation and endothelial activation confers a higher risk of mortality and of cardiovascular event occurrence than either factor does alone (1). In contrast to published literature describing three forms of uremic cardiomyopathy – left ventricular hypertrophy (LVH), dilation, and systolic dysfunction, it was also shown that LVH is the predominant cardiomyopathy specific to uremia, while LV dilation and systolic dysfunction are due to underlying (possibly silent) ischemic heart disease (41).

In conclusion, residual renal function is the keystone in preserving cardiovascular function in peritoneal dialysis patients. Every therapeutic effort is needed to prolong the period with diuresis in patients on PD, taking into account the cardiovascular well-being of these patients in the first place.

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REZIDUALNA RENALNA FUNKCIJA I KARDIOVASKULARNE KOMPLIKACIJE KOD BOLESNIKA NA PERITONEUMSKOJ DIJALIZI

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SAŽETAK

Kardiovaskularne bolesti su vodeći uzrok smrti bolesnika na peritoneumskoj dijalizi. Razlog leži u visokoj prevalenciji tradicionalnih, a posebno netradicionalnih faktora rizika. Opadanje rezidualne renalne funkcije povećava prevalenciju i težinu faktora rizika i doprinosi razvoju kardiovaskularnog morbiditeta i mortaliteta ovih bolesnika. Rano otkrivanje bolesnika sa povećanim kardiovaskularnim rizikom omogućava pravovremenu primenu terapije, koja treba prvenstveno da uključi terapijsku strategiju očuvanja rezidualne renalne funkcije. Očuvanje rezidualne renalne funkcije popravlja kardiovaskularni ishod bolesnika koji se leče peritoneumskom dijalizom.

Ključne reči: **rezidualna renalna funkcija, peritoneumska dijaliza, kardiovaskularne komplikacije**