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Case Report

The Beauty and Simplicity of Automated Peritoneal Dialysis in a Patient with Refractory Congestive Heart Failure and Severe Pulmonary Hypertension: Case Report

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Abstract

The prevalence of pulmonary hypertension (PH) in patients with end stage renal disease (ESRD) is about 25-60%. It carries high risk of morbidity and mortality. Here we report a 65-year-old Saudi lady, a known case of ESRD secondary to diabetes mellitus, hypertension and CHF functional class IV, due to dilated cardiomyopathy with severe PH. She was initially treated with hemodialysis for three months with no improvement. After shifting her to automated peritoneal dialysis, her pulmonary artery pressure significantly decreased. PH, however, recurred after a failed renal transplant. In our opinion, peritoneal dialysis is a very effective therapeutic modality in patients with refractory CHF and severe PH associated with renal impairment.

Key Words: Refractory congestive heart failure, pulmonary hypertension, renal failure, hemodialysis, automated peritoneal dialysis.

Introduction

The prevalence of pulmonary hypertension (PH) in patients with end stage renal disease (ESRD) is about 25-60% [1-7]. It is defined as a sustained elevation of pulmonary artery pressure (PAP) to > 25 mmHg at rest or to >30 mmHg with exercise. It has high risk for morbidity and mortality [8]. The presence of PH post renal transplant carries high risk for early allograft dysfunction and lower survival rate [9,10]. The survival rate of patients with PH at 1, 3 and 5 years is 79%, 63% and 30% respectively, and the main cause of death is cardiovascular disease [11]. It is characterized by endothelial dysfunction and remodeling of pulmonary vasculature mainly medial and intimal layers, as a consequence of high cardiac output, increased pulmonary blood flow and pulmonary resistance leading to diffuse constriction and occlusion of pulmonary blood vessels [6,7]. PH is usually classified to primary or secondary based on the presence of underlying etiologies, such as congestive heart failure, obstructive and restrictive pulmonary diseases, portal hypertension, collagen vascular disease, recurrent thromboembolism, human immunodeficiency virus infection and drugs [12-16]

Multiple risk factors have been associated with PH in ESRD including uremia, size and location of arterio-venous fistula (AVF), left ventricular dysfunction, low ejection fraction, anemia, volume overload, microbubbles created by exposure to dialysis membrane and hyperparathyroidism. The best choice of renal replacement therapy for patients with ESRD and PH is controversial. Multiple studies have advocated pre-emptive renal transplant and peritoneal dialysis but without a high quality of evidence supporting this assumption [5,6,10,12]. To the best of our knowledge, the outcomes of patients with PH and ESRD started on automated peritoneal dialysis (APD) have not been previously reported. Here we report a 65-year-old Saudi lady, a known case of refractory CHF with severe PH and ESRD, initially treated with hemodialysis through right internal jugular permcath (IJP) with no improvement in terms of the pulmonary artery pressure. After shifting to APD, her PAP dramatically improved but raised after failed renal transplant. In our opinion, PD is an effective therapeutic modality in patients with refractory CHF and severe PH with ESRD.

Case Presentation

This is a 65-year-old Saudi lady, a known case of ESRD secondary to diabetes mellitus and hypertension, dilated cardiomyopathy, CHF class IV, and severe pulmonary hypertension (PAP > 85). She was admitted to the intensive care unit due to pulmonary edema and hyperkalemia with metabolic acidosis. Initially, she was started on hemodialysis (HD) through right IJP. With catheter manipulation in each HD session, she developed ventricular tachycardia (VT) that once required cardiopulmonary resuscitation. The IJP was removed and a femoral permcath was inserted with no more VT. She was then discharged in a stable condition to be followed at our hemodialysis center. Six weeks later, she developed catheter related deep vein thrombosis and septicemia. Femoral catheter was then removed and anticoagulation therapy was started. Subsequently, she was referred to PD at our center. Her initial investigation: hemoglobin 7.7 gm/dl , platelet count 497 x 109 per liter, blood urea nitrogen 85 mg/dl, serum creatinine 6.0 mg/dl, serum potassium 5.5mmol/l, serum sodium 134mmol/l, calcium 8.3mg/dl, phosphorous 6.5 mg/dl, albumin 2.5 gm/dl, creatinine clearance 9 ml/min and PTH 25 pmol (normal range 1.3-6.8). Arterial blood gas: pH 7.2, carbon dioxide (PCO_2) 34mmHg and bicarbonate (HCO_3) 17 mEq/L. Cardiac enzymes, iso enzymes, hepatitis profile and HIV were all negative. Prothrombin time (PT) and partial thromboplastin time (PTT) were within normal levels. Chest x-ray (Figure 1) showed bilateral pulmonary congestion and bilateral pleural effusion. Electrocardiogram (ECG) showed biventricular enlargement, renal ultrasound revealed bilateral normal-size kidney and no mass or cystic lesion could be detected. Echo-cardiograph revealed severe left ventricular enlargement and severe PAP >85 mmHg. On January 2008 the Saudi PD catheter [42] was inserted uneventfully and she was started on Tidle 70% APD ,1.36% Physioneal 5 liters, 2.27% Physioneal 5 liters over 9 hours, each fill 1.5 liters gradually increased to 2 liters. Four weeks later icodextrin 2 liters as the last fill was added. Initial examination: elderly lady looked ill, in respiratory distress, weight 65 kg, body surface area 1.6 m^2. Vital signs: blood pressure (BP) 120/70 mmHg, pulse 95/min, respiratory rate 22/min and temperature 36.8 C. Jugular venous pressure was raised and chest auscultation revealed bilateral crepitation up to the middle zone. Cardiovascular system: normal S1 and loud S2 with S3 gallop and grade 3/6 pansystolic murmur best heard at the mitral area. Abdomen was soft and lax with moderate ascites and grade 3 bilateral lower limb edema. Neurological examination revealed

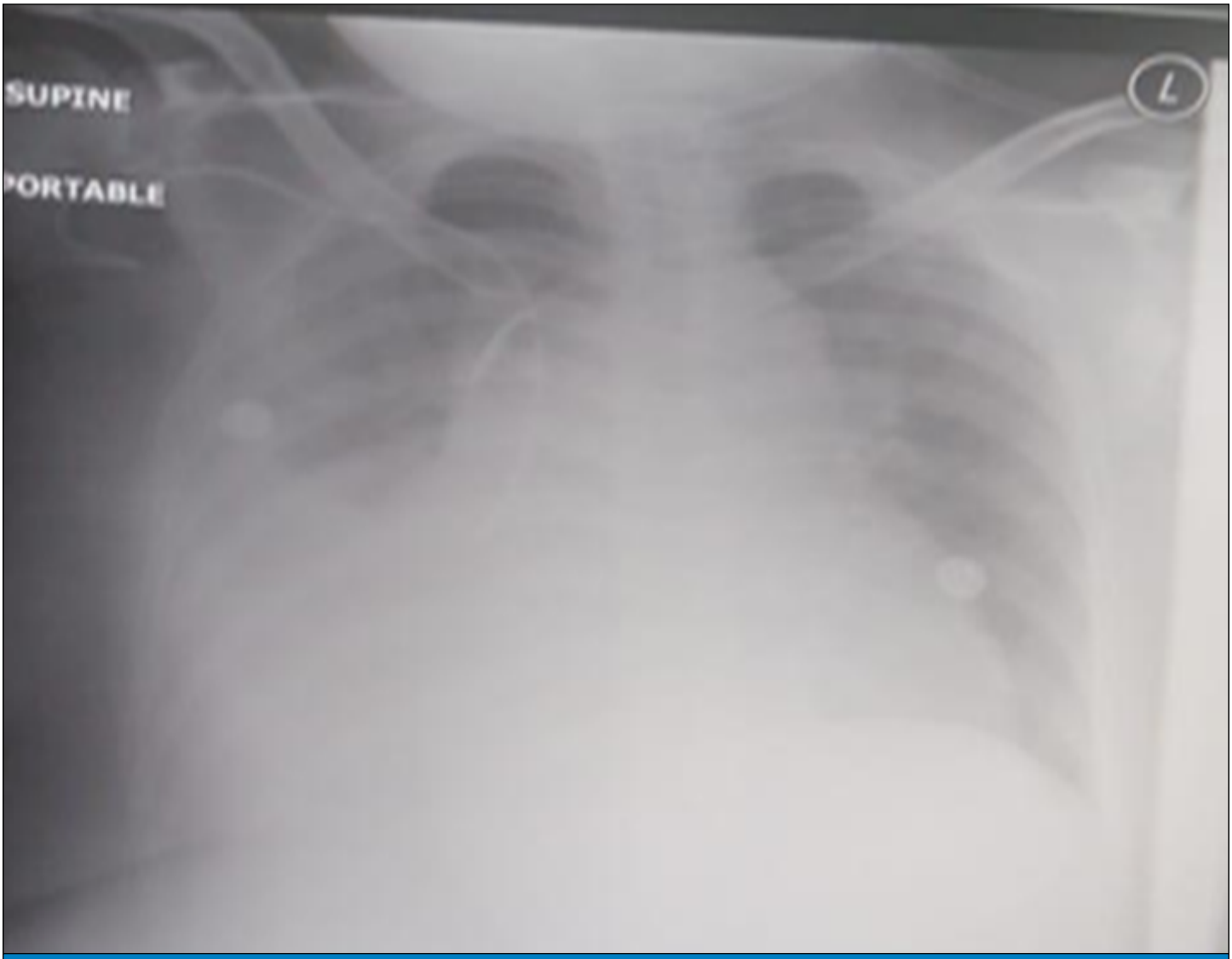

Figure 1: Chest x-ray showed bilateral pleural effusion with pulmonary congestion.

peripheral sensory neuropathy. Her course at PD was uneventful with regular follow up at PD center on monthly bases with no history of admission, peritonitis or exit site infection. Her KT/V was 2 and the average urine output was 900-1100 ml per day. Her PAP dramatically improved to 25 mmHg over one year and the New York heart association (NYHA) class dropped to class-II. On February 2009, she underwent an uneventful living related kidney transplant and she was discharged on mycophenolic acid, tacrolimus, prednisolone, trimethoprim/sulfamethoxazole and valacyclovir with regular follow up at transplant clinic. On 2012, due to the deterioration of her renal function renal biopsy was done and it was consistent with chronic graft rejection. Moreover, after the escalation of the immune suppression medication, her condition deteriorated more and she developed Kaposi sarcoma of both lower limbs, cerebrovascular stroke and renal failure. She succumbed to her illness in February 2013. Figure 2 illustrates the levels of PAP during HD, PD and post renal transplant. Figure 3 shows the course of the patient during HD, PD and post renal transplant.

Discussion

Diuretics remain the most effective means of fluid removal in CHF patients, and their use allows rapid relief of the congestion. The prevalence of PH in patients with ESRD is about 25-60% [1-7]. The definition of PH is sustained elevation of PAP to > 25 mmHg at rest or to >30 mmHg with exercise. It has a high risk for

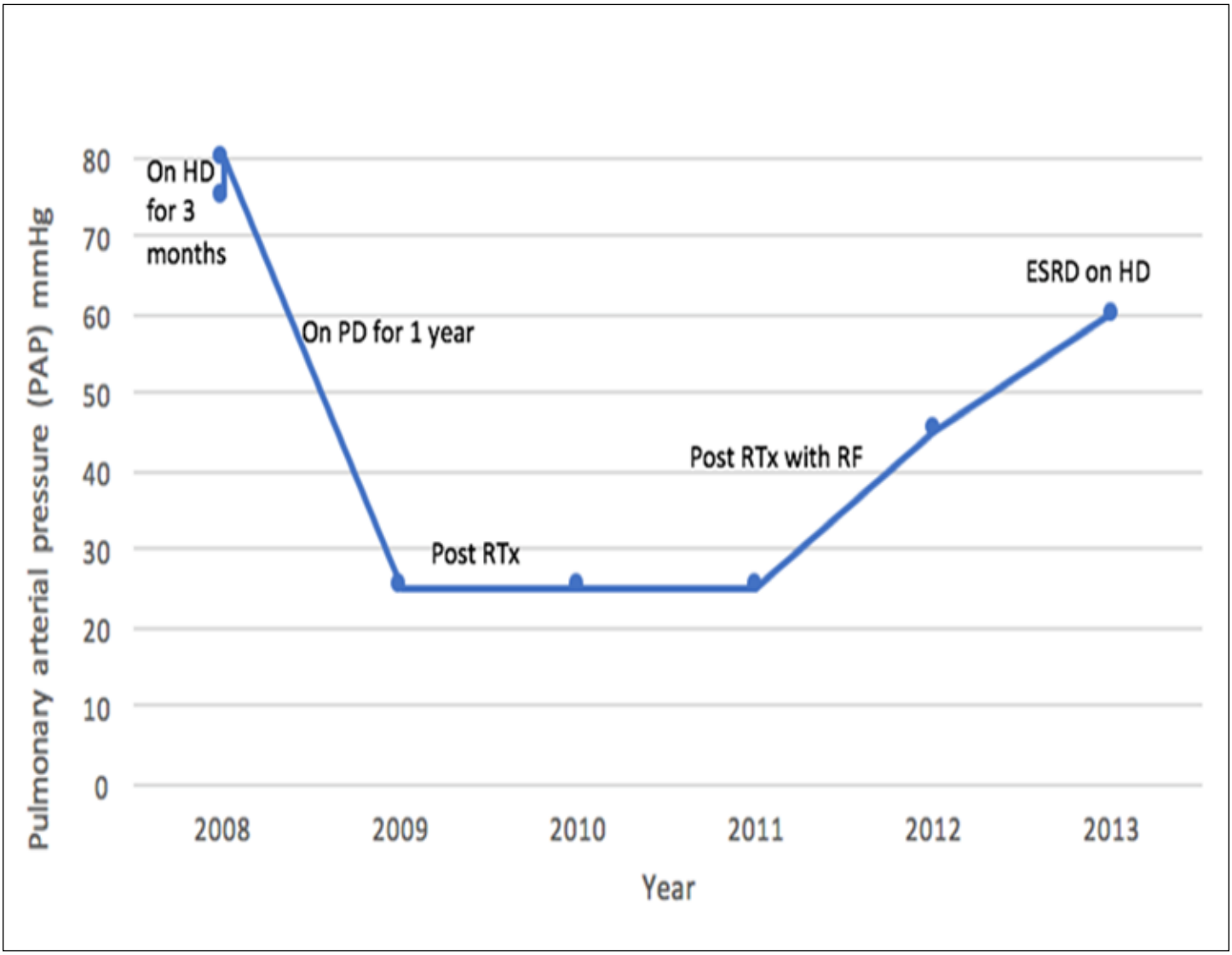

Figure 2: Course of pulmonary arterial pressure during hemodialysis, peritoneal dialysis and post renal transplant.

morbidity and mortality [8]. The presence of PH post-renal transplant carries high risk for early allograft dysfunction and lower survival rate [9,10]. The fact that PAP improved after successful kidney transplantation with intact AVF indicates that uremia and high cardiac output are not the only contributing factors in the pathogenesis of PH in chronic renal failure population [18]. The cytokines in particular tumor necrosis factors alpha, endothelial-1, atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP) and interleukin-1 have been shown to induce pulmonary angiogenesis, fibroblast proliferation and apoptosis of cardiac myocytes [22]. BNP released from ventricular myocytes is correlated with cardiovascular morbidity and mortality [23]. A high level of BNP has also been reported by various studies as a poor prognostic factor in patients with PH [23,24]. There is a high body of evidence indicating that PD has the capability to remove small and middle size molecules [25]. The molecular weight of TNF alpha is about 17KDa, and that of myocardial depressant factors is nearly 700-800Da [26]. Thus, the removal of these small and middle molecular weight cytokines by PD may contribute to reduction in PAP. In addition, PD imitates the normal physiological process with no hemodynamic disturbances and no arteriovenous (A-V) access that can augment PH in dialysis patients. This explains the

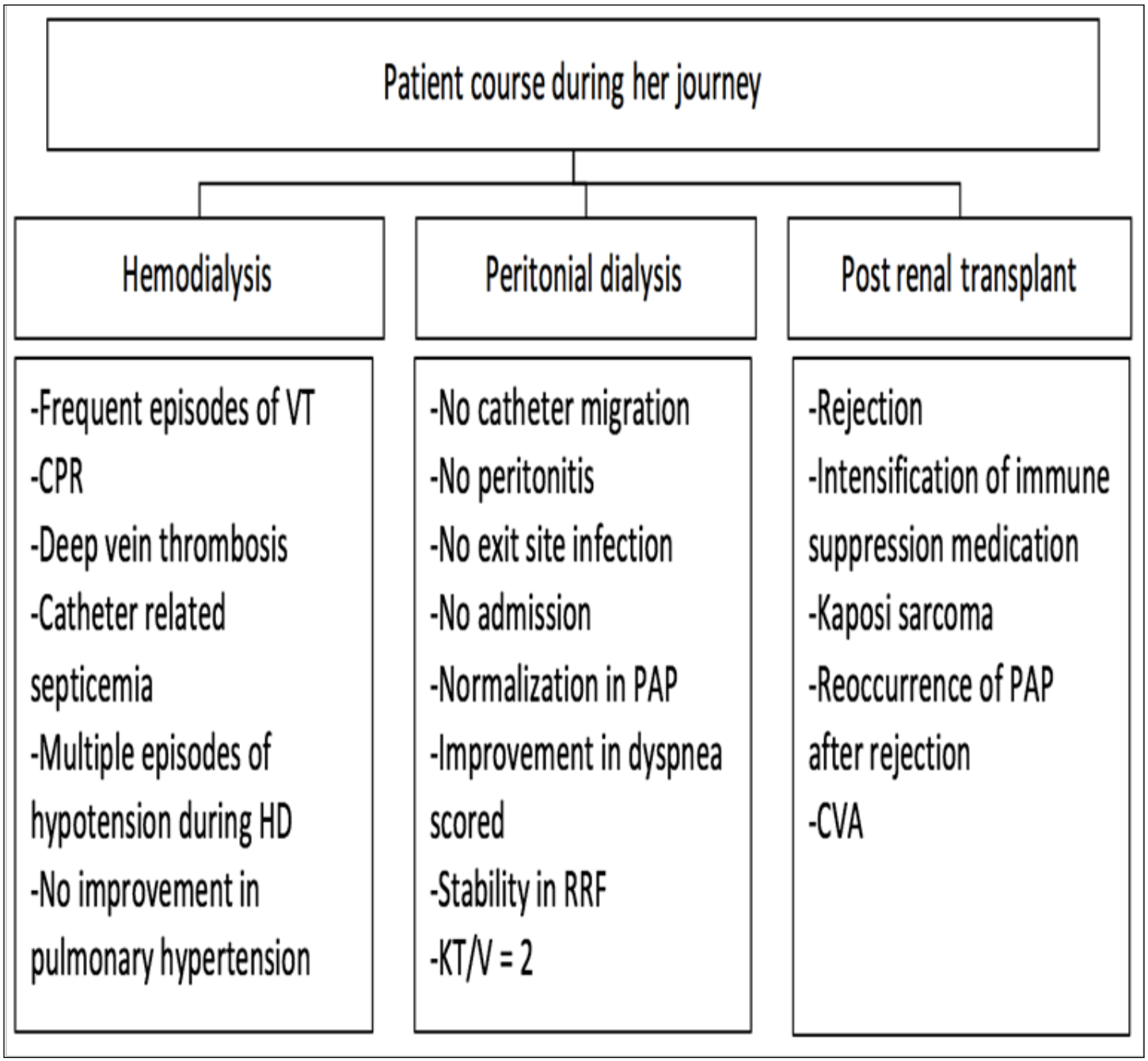

Figure 3: Patient's course during her illness. VT= Ventricular Tachycardia; CPR = Cardiopulmonary Resuscitation; HD = Hemodialysis; PAP= Pulmonary Arterial Pressure; RRF = Rresidual Renal Function; CVA= Cerebrovascular Accident.

low prevalence of PH in PD patients and the reduction of PAP after commencing PD [27]. Cécile, et al. [28] has shown a significant improvement of left ventricular ejection fraction and reduction of the number of hospitalization days in a cohort of 126 CHF patient refractory to diuretics with moderate renal impairment after PD initiation. Kogan and Raport reported a 19-year-old patient with severe PH and dilated cardiomyopathy with renal failure refractory to conventional therapy and HD. After starting continuous ambulatory peritoneal dialysis (CAPD) her PAP dramatically dropped from 68 mmHg to 25 mmHg [29] similar to our case.

The explanation of re-occurrence of PH after chronic rejection in our case highly reaffirms the role of uremia and inflammatory markers as important causes of PH in renal failure patients. In patients with high PAP, the prevalence of supraventricular arrhythmia and ventricular arrhythmia is 31% and 24% respectively [30]. Multiple risk factors have been reported to precipitate cardiac arrhythmia in patients with high PAP including, uremia, left ventricular hypertrophy, electrolytes disturbance, guide wire and catheter length, sympathetic hyperactivity and myocardial fibrosis [31] .Our patient developed frequent episodes of VT during each of HD sessions and this was attributed to severe PH and catheter length. VT completely resolved after catheter removal.

PD offers gentle Ultrafiltration (UF), while UF through HD is associated with myocardial stunning [32-35]. UF-induced myocardial stunning is associated with progression to fixed systolic dysfunction [36]. The minimal impact of PD on hemodynamics would theoretically result in a lower degree of neurohumoral stimulation secondary to fluid removal, compared to HD. As such, in theory, PD will more likely not stimulate the maladaptive neurohumoral responses in heart and kidney cross-talk already present in CHF. Several studies indeed suggested PD, compared to HD as being associated with a positive impact on residual kidney function, a factor known to be associated with survival [37,38]. Because it is a daily or continuous treatment, PD also allows for effective continuous solute clearance, including sodium and potassium, allowing better up-titration of pharmacological treatment for CHF. The peritoneal cavity access allows for the draining of ascites in the setting of right-sided heart failure. Although, it is speculative, providing a permanent outlet for ascitic fluid might reduce intra-abdominal pressure, which has been demonstrated to improve renal function in CHF [39]. Mullens W, et al. [40] reported that elevated intra-abdominal pressure (IAP), defined as ≥ 8 mm Hg can be associated with renal dysfunction in patients with CHF. They hypothesized that in the setting of persistently elevated IAP and progressive renal insufficiency refractory to intensive medical therapy, mechanical fluid removal (ascetic fluid in patients with CHF) that can be achieved by PD can be associated with improvements in IAP and renal function. This may be a reasonable additional explanation to why our patient PAP dramatically improved after PD. In a recent study, 24 of 40 patients admitted for CHF (mean left ventricular ejection fraction, 19%) had an IAP ≥ 8 mm Hg [41]. None of the 40 patients in the cohort complained of abdominal symptoms at the study entry. Patients with elevated IAP had significantly lower baseline glomerular filtration rate (GFR) compared with those with normal IAP, and the degree of reduction in IAP after diuresis predicted an improvement in renal function.

Conclusion

To the best of our knowledge the outcome of CHF patient and severe PH with ESRD started on APD has not been previously reported. Here we report a 65-year-old Saudi lady, a known case of refractory CHF with severe PH and ESRD, initially treated with HD through right IJP with no improvement. After shifting to APD, her PAP dramatically improved and reoccurs after failed renal transplant. In our opinion PD is a very effective therapeutic modality in patients with refractory CHF, severe PH and ESRD.

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