Stability of Renal Function in Spite of Low Glomerular Filtration Rate: A Case Report

Mohammad Reza Tamadon 1,*; Seyed Seifollah Beladi Mousavi 2

1Department of Internal Medicine, Faculty of Medicine, Semnan University of Medical Sciences, Semnan, IR Iran
2Chronic Renal Failure Research Center, Jundishapur University of Medical Sciences, Ahvaz, IR Iran

*Corresponding Author: Mohammad Reza Tamadon, Department of Internal Medicine, Faculty of Medicine, Semnan University of Medical Sciences, Semnan, IR Iran. Tel: +98-9823105059. Fax: +98-2134309130. E-mail: mrt.tamadon@yahoo.com

Received: June 29, 2014; Revised: December 27, 2014; Accepted: December 31, 2014

1. Introduction

Chronic renal dysfunction is a progressive and irreversible process of kidney function, which often results in chronic kidney disease (CKD) or chronic renal failure (CRF). The most common causes are diabetes, hypertension, artery disease, glomerulonephritis (GN) congenital-inherited cystic diseases, interstitial nephritis, pyelonephritis, secondary GN and vasculitis. Chronic renal failure is the progressive and irreversible loss of kidney function. This progressive failure becomes manifested as stepwise increasing in the serum creatinine level in parallel to glomerular filtration declining. The modern treatment aims to establish the renal replacement therapy by dialysis or a kidney transplant before appearing of advanced uremia symptoms. Decreased renal function causes to end products of protein metabolites, which are usually excreted through urine accumulates in blood and thus result to uremia (1, 2). Stages of chronic kidney failure are as decreased renal reserve, renal failure and end stage renal disease (ESRD). Declining rate of renal function and progression of chronic renal failure depend on the underlying variations, the urinary protein excretion and hypertension. The disease is progressing faster in patients who excrete more protein in urine and have the blood pressure more than normal level (3-5). Five stages of CKD include: stage 1: kidney damage with normal glomerular filtration rate (GFR) of ≥ 90. In the first stage of kidney disease, the goal of treatment is to slow the progression of CKD and lower the cardiovascular disease risk. Stage 2 is like kidney damage with a mild decrease in GFR (between 60 and 89). Estimating of CKD progression accompanying by evaluation of declining process of glomerular filtration are important in diagnosis and effective treatment. Step 3: moderate decrease in GFR (between 30 and 59). When the CKD progresses to this stage, assessment and treatment of complications such as anemia and osteoporosis are needed. Stage 4 is a severe reduction in GFR (between 15 and 29). The continued follow-up and training techniques for the replacement therapy are required to treat problems. Any treatment requires preparation. If you choose hemodialysis as treatment, you may need to perform surgery on the patient's vein for vascular access. For peritoneal dialysis, patients require catheter insertion in peritoneal cavity. Kidney transplantation is required for a donor who can be a family member or friend. Step 5: end-stage renal disease (GFR < 15). At this stage, dialysis or a kidney transplantation is required (6, 7). Since many organs affected by uremia due to CRF, the patients have various signs and symptoms. Severity of these signs and symptoms depends on the disease progresses, the patient's age and other underlying conditions. Reduction in GFR can be diagnosed by a 24-hour urine collection analysis and measurement of the creatinine clearance. By reducing of GFR (due to glomerular dysfunction) creatinine clearance will be reduced, while serum creatinine and blood urea nitrogen (BUN) levels increase. Serum creatinine level is the most sensitive index for renal function, because it is constantly produced in the body (1, 2).

Keywords: Kidney; Glomerular Filtration Rate; Proteinuria
2. Case Presentation

The patient is a 78-year-old male patient with renal disease from 9 years ago (March 2005). He has lived in Semnan, Iran. His medical records were reviewed in October 2014. Laboratory findings in the patient at first were as follows: BUN = 22, Cr = 1.3, weight: 60 kg, height: 166 cm, blood pressure 145/95 mmHg. The patient was treated. The BUN and creatinine levels gradually increased and reached to BUN = 43 and Cr = 4 thirty months after the first referral. All laboratory tests were performed in a medical diagnostic laboratory using the calibrated equipment and common methods. The BUN and creatinine levels were measured by auto analyzer TC-6062 manufactured by Tecom Co, China. Blood pressure was measured by the Riester sphygmomanometer (Germany). At that time he had referred for arteriovenous graft and an arteriovenous fistula made by a surgeon. Six months later he had a functional fistula. Patients were treated with the conventional treatment and Eprex. Specific variables in this case were creatinine and GFR. Blood pressure controlled in the range of 110/80 mmHg. The patient’s GFR decreased from 20 to 14 cc/min and his last blood pressure was 140/80 mmHg. The mean serum creatinine level was 3, maximum 4.2 and minimum 1.5. At present, the patient is in stage 4 of CKD and under usual medical treatment.

3. Discussion

The worldwide prevalence of CKD has been reported between 8-16% (8). The prevalence in Norway has been 3.9%, 11% in the United States, 16% in Australia, 9.07% in Malaysia and 13.5% in the UK (9). Coresh J, et al. evaluated the prevalence of different stages of CKD. Based on these results, 3.3% of the patients were in stage 1, 3% in stage 2, 3.4% in stage 3, 0.2% in stage 4 and 0.2% in stage 5. Age has been as a predictive factor for CKD, so that 11% of patients older than 65 years without hypertension and diabetes were in stages 3 or higher (10). Chronic kidney disease is a progressive process; patients in the first stage have evidence of kidney damage despite of a GFR above 90. In the second stage, GFR is gradually declining, but the patient has no symptoms. In the third and fourth stages of the disease, symptoms start gradually and have sharp decline in GFR. In the fifth stage, the patient has a GFR less than 15 and will require to dialysis (10). The usual assumption is that patients with CKD have a progressive nephropathy, although the rate of progression is extremely variable. Proteinuria, hypertension, and black race are the risk factors in increasing the rate of disease progression (11). The majority of CKD patients have progressive loss of kidney function, although some clinical trials suggest that renal function in some patients had not any changes and had been constant (9). Experimental and histopathological evidences are proposed the possibility of improving the renal function (12). Of course, this matter is controversial that renal function will be really improved. However, there is no compelling evidence to suggest that renal function in patients with CKD can be improved based on the GFR measurement. In one study, it has been reported that renal function can be improved in some patients with hypertensive CKD (13). Although our patient had CKD stage 4, but his condition has not deteriorated and remained constant and stable for several years only by control of blood pressure and usual treatment which prescribed for patients at this stage.

Acknowledgements

We would like to thank Mr. Mehrdad Zahmatkesh for his help in preparation of this paper.

Authors’ Contributions

Dr. Mohamad Reza Tamadon: data collection and writing of the paper. Dr. Seyfollah Beladi Mousavi: Writing of the paper.

References

1. Nesrallah GE, Mustafa RA, Clark WF, Bass A, Barnieh I, Hemmelgarn BR, et al. Canadian Society of Nephrology 2014 clinical practice guideline for timing the initiation of chronic dialysis. CMAJ. 2014;186(2):132-7.
2. Hajivandia A, Amiri M. World kidney day 2014: Kidney disease and elderly. J Parathyrol Dis. 2014;2(1):3-4.
3. Nasir H. The awareness of chronic kidney disease and aging; the focus of world kidney day in 2014. J Nephropharmaco. 2014;3(1):1-2.
4. Nasir H. World kidney day 2013: acute kidney injury; a public health aware. Iran J Public Health. 2013;42(1):338-40.
5. Ardalan MR, Sanadgol H, Nasir H, Baradaran A, Rafieian-Kopaei M. Vitamin D therapy in diabetic kidney disease; current knowledge on a public health problem. J Parathyrol Dis. 2014;2(1):15-7.
6. Tamadon MR,. Secondary hyperparathyroidism and chronic kidney disease. J Parathyrol Dis. 2012;1(1):35-6.
7. Abboud H, Henrich WL. Stage IV Chronic Kidney Disease. N Engl J Med. 2010;362(1):56–65.
8. Assadi F. The epidemic of pediatric chronic kidney disease: the danger of skepticism. J Nephrol. 2012;25(2):66-4.
9. Roderick P, Roth M, Mindell J. Prevalence of chronic kidney disease in England: Findings from the 2009 Health Survey for England. J Epidemiol Community Health. 2011;65(Suppl 2):A12.
10. Coresh J, Astor BC, Greene T, Eknoyan G, Levey AS. Prevalence of chronic kidney disease and decreased kidney function in the adult US population: Third National Health and Nutrition Examination Survey. Am J Kidney Dis. 2003;41(1):1-12.
11. Jha V, Garcia-Garcia G, Iskeli K, Li Z, Naicker S, Plattner B, et al. Chronic kidney disease: global dimension and perspectives. Lancet. 2013;382(9888):260–72.
12. Gowda S, Desai PB, Kulkarni SS, Hull VV, Math AA, Venkata SN. Markers of renal function tests. N Am J Med Sci. 2010;2(4):170-3.
13. Amiri M, Nasir H. Secondary Hyperparathyroidism in chronic kidney disease patients. J Parathyrol Dis. 2014;2(1):1-2.