Kidney Disease: Personalized Lifestyle Health Care Makes a Big Difference

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Abstract

We cannot solve the kidney disease problem through the building of more dialysis centers or by providing a greater number of kidney transplants. We must find a way to implement effective lifestyle management programs if we truly want to bend the curve and decrease the prevalence of kidney disease. The solution to the chronic kidney disease challenge lies in the skilled application of personalized lifestyle health care. Achieving this goal represents a tremendous opportunity for multidisciplinary collaboration and integration.

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You never know when a conversation will open your mind to realities that you never before understood. I had this experience recently during a conversation—one that I expected to be casual—with my son. Kyle has worked as an executive in the biotechnology industry for many years and his specific area of focus has been therapeutics and kidney disease. This work requires him to spend a considerable amount of time in dialysis centers around the United States. Now, I have always considered myself to be reasonably familiar with kidney disease, and so when I opened the conversation by asking what the average life expectancy is for a person with end-stage renal disease who requires kidney dialysis, I found myself surprised—perhaps even shocked—by his answer: "5 years." My reaction was the result of my (false) impression that it was far longer. I definitely wanted to know more about the influence of kidney dialysis on the preservation of kidney function. One statistic in particular intrigues me, and that is the large standard deviation in the life expectancy of a dialysis patient.

According to the National Kidney Foundation, average life expectancy on dialysis is 5 to 10 years, but the foundation also indicates that many patients can live well for 20 or even 30 years. What accounts for this? Revisiting the conversation with my son, I felt that some personal observations he shared with me provided some insight. Following a decade spent in dialysis centers, Kyle told me he felt that he had developed an instinct for privately forecasting potential life expectancy based on lifestyle habits he took note of among patients. On many, many occasions, he indicated he has observed individuals arrive at a dialysis center with large containers of carbonated beverages and bags of fast food in hand. This tendency has become a marker for him, one that he has come to personally correlate with diminished success of treatment and an increased likelihood that he will see those patients at the centers he visits only a limited number of times in the future.

Kidney disease is the ninth leading cause of death in America. It accounts for close to 50 000 deaths annually. An estimated 10% of adults in the United States—more than 20 million people—are thought to have chronic kidney disease (CKD). Its annual cost to the medical system is more than $50 billion. The incidence in CKD in people 65 years and older more than doubled from 2000 to 2008.[1,2] The majority of individuals with CKD are older than 40 years, and the evidence from multiple studies indicate that for most of them their disease is directly related to lifestyle and diet variables—so much that CKD can be called a "lifestyle disease."[3,4,5] There is evidence that genetic factors play a role in CKD susceptibility; however, in a study of 68 monozygotic and 30 dizygotic twin pairs, it was found that heredity had a negligible role in the etiology of CKD and that lifestyle, diet, and environmental factors were the most significant contributors to the disease.[6,7] Given this understanding of the connection between genetic and lifestyle factors, it is possible—and very likely—that future efforts may focus on the development of a personalized lifestyle medical approach to the prevention and management of this epidemic.

Although my outlook for future possibilities is optimistic, there is no escaping the rather grim reality of the present. More than 450 000 people in the United States are presently undergoing kidney dialysis for the treatment of kidney failure. More than 120 000 people are awaiting kidney transplants in the United States with fewer than 17 000 receiving one each year. The annual cost of dialysis is nearly $90 000 for a single individual, contributing to total expenditures for various stages of kidney disease in excess of $99 billion in 2013. [8] Kidney disease is one of today's greatest economic burdens on the medical system.

Let us step away from the numbers now and examine the human face of kidney disease and the personal challenge it presents to the dialysis patient. Nausea, vomiting, cramps, dizziness, and constant fatigue are common problems. Most patients must be dialyzed 3 times per week, 4 hours each visit. Dialysis centers are often open 24 hours per day to accommodate patients who arrive at 4:00 AM to dialyze until 8:00 AM when they then go to work. Taking a business trip or vacation requires finding a dialysis center in the travel area. Most dialysis patients have their lives revolve around their treatments. All of this is superimposed upon the realization that the average life expectancy once dialysis is initiated may only be 5 years.

With these sobering facts, the question is: What can be done to change this upward trend in the prevalence of CKD to a downward direction? In my opinion, there are 2 important concepts that underlie an actionable approach to this question: early diagnosis and personalized lifestyle intervention.

There has been controversy as to what constitutes the best biomarker for the early detection of CKD. Guidelines developed by the National Kidney Foundation that are commonly referred to as the KDOQI guidelines recommend that CKD be classified based on the glomerular filtration rate (GFR) and degree of albuminuria.[9] Measuring GFR accurately is a complicated medical procedure involving an insulin challenge test, so the standard practice has become that medical laboratories provide an estimate of GFR (termed *eGFR*) calculated from serum creatinine, body size, age, and ethnicity.[10] Values of eGFR less than 60 are associated with increasing evidence of compromised kidney function. As the eGFR decreases, it indicates increasing kidney dysfunction, with values less than 30 associated with severe loss of kidney function.[11] It has been pointed out that the eGFR is not an accurate determination of kidney function, but rather an estimate and other diagnostic methods such as the measurement of microalbuminuria or urinary protein to creatinine ratio might provide improved early recognition of kidney disease.[12,13,14,15]

Other analyses used for measuring kidney function include serum cystatin-C and β-trace protein.[16,17] Cystatin C has been shown to provide a more accurate estimate of kidney function in older patients, especially when used in combination with measurements of serum creatinine.[18] The important takeaway from the literature surrounding the assessment of kidney function is that serial measurements in time are very important in tracking the trajectory of kidney dysfunction. It is the traditional view in medicine that once a person has CKD it can never be reversed, but that the rate of loss of kidney function can be reduced. The question I want to address is this: Once it has been determined that a person is losing kidney function at a rate that is concerning, what can be done to slow, if not stop, advancement of CKD? It starts with the control of hypertension, which represents the major contributing factor to kidney disease.[19] This is where personalized lifestyle health care comes to the head of the class. It is now well established that obesity and a sedentary lifestyle contribute to increasing blood pressure and the progression of CKD.[20] Intervention with a lower protein, low glycemic load diet plan[21] and regular programmed exercise has proven effective in managing CKD.

There is now persuasive evidence that a personalized approach to dietary and lifestyle intervention represents a primary treatment for CKD and can help to preserve kidney function, even in high-risk individuals. Lifestyle factors such as cessation of smoking, reduction in alcohol consumption, increased physical activity, and increased social support all correlated with improved GFR and microalbuminuria measured in the course of 5.5 years.[22] The combination of managing hypertension in conjunction with a personalized lifestyle and dietary program that encourages adherence and compliance was demonstrated in the See Kidney Disease (SeeKD) trial to reduce the rate of loss of kidney function as measured by eGFR.[23]

There are many review articles that describe the role of a personalized lower protein diet coupled with a regular walking program that describe the benefit in managing CKD.[24,25] These reviews emphasize the role that lifestyle health care has as a primary therapy for the management of kidney disease.

CKD and the transition to kidney failure requiring dialysis is a very good example of the challenges confronting the current medical system both in terms of patient management and cost for the treatment of age-related chronic diseases. This condition constitutes a major percentage of expenses attributed to disease care. Once the kidneys have failed, the quality of life of the patient is seriously compromised. Depression and dementia are common comorbidities found in people with kidney disease as are heart attacks and type 2 diabetes. Is there any bright light with which to view a condition that is largely associated with daily discomfort and difficulty? For me, it is the recognition that with early intervention using an appropriate personalized lifestyle medical program, the acceleration of loss of kidney function can be slowed. This program must take into account psychosocial factors that will encourage compliance with the recommendations. In addition, it may require health

coaching by a professional who is trained in the delivery of personalized lifestyle medicine programs.

What is obvious to me—and I hope to many—is that we cannot solve this problem through the building of more dialysis centers or by providing a greater number of kidney transplants. We must find a way to implement effective lifestyle management programs if we truly want to bend the curve and decrease the prevalence of kidney disease. The solution to the CKD challenge lies in the skilled application of personalized lifestyle health care. Achieving this goal represents a tremendous opportunity for multidisciplinary collaboration and integration.

References

1. National Kidney Foundation. Fact sheet. https://www.kidney.org/news/newsroom/factsheets/FastFacts. Retrieved August 11, 2016. Accessed November 12, 2016.
2. National Institute of Diabetes and Digestive and Kidney Diseases. Kidney disease statistics for the United States. https://www.niddk.nih.gov/health-information/health-statistics/Pages/kidney-disease-statistics-united-states.aspx. Retrieved August 11, 2016. Accessed November 12, 2016.
3. Foster MC, Hwang SJ, Massaro JM, Jacques PF, Fox CS, Chu AY. Lifestyle factors and indices of kidney function in the Framingham Heart Study. *Am J Nephrol*. 2015;41(4-5):267-274.
4. Teng HL, Yen M, Fetzer S, Sung JM, Hung SY. Effects of targeted interventions on lifestyle modifications of chronic kidney disease patients: Randomized controlled trial. *West J Nurs Res*. 2013;35(9):1107-1127.
5. Robinson-Cohen C, Littman AJ, Duncan GE, et al. Physical activity and change in estimated GFR among persons with CKD. *J Am Soc Nephrol*. 2014;25(2):399-406.
6. Luttropp K, Lindholm B, Carrero JJ, Glorieux G, Schepers E. Genetics/genomics in chronic kidney disease: Towards personalized medicine. *Semin Dial*. 2009;22(4):417-422.
7. Tarnoki DL, Tarnoki LD, Littvay L, et al. Genetic and environmental variance of renal parenchymal thickness: A twin study. *Croat Med J*. 2013;54(6):550-554.
8. National Kidney Foundation. Fact sheet. https://www.kidney.org/news/newsroom/factsheets/FastFacts. Retrieved August 11, 2016. Accessed November 12, 2016.
9. Wouters OJ, O'Donoghue DJ, Ritchie J, Kanavos PG, Narva AS. Early chronic kidney disease: Diagnosis, management and models of care. *Nat Rev Nephrol*. 2015;11(8):491-502.
10. Hsu CY, Bansai N. Measured GFR as "Gold Standard"—All that Glitters Is Not Gold? *CJASN*. 2011;6(8):1813-1814.
11. National Kidney Foundation. Glomerular filtration rate (GFR). https://www.kidney.org/atoz/content/gfr. Retrieved August 11, 2016. Accessed November 12, 2016.
12. Yamamoto K, Yamamoto H, Yoshida K, et al. The total urine protein-to-creatinine ratio can predict the presence of microalbuminuria. *PLoS One*. 2014;9(3):e91067.
13. Saha TK, Bhattarai AM, Batra HS, Banerjee M, Misra P, Ambade V. Correlation of microalbuminuria with estimated GFR (eGFR) by Cockcroft-Gault and MDRD formula in type 2 diabetics and hypertensives. *Indian J Clin Biochem*. 2015;30(3):271-274.
14. Hong DS, Oh IH, Park JS, et al. Evaluation of urinary indices for albuminuria and proteinuria in patients with chronic kidney disease. *Kidney Blood Press Res*. 2016;41(3):258-266.
15. von Scholten BJ, Reinhard H, Hansen TW, et al. Urinary biomarkers are associated with incident cardiovascular disease, all-cause mortality and deterioration of kidney function in type 2 diabetic patients with microalbuminuria. *Diabetologia*. 2016;59(7):1549-1557.
16. Stevens LA, Coresh J, Schmid CH, et al. Estimating GFR using serum cystatin C alone and in combination with serum creatinine: A pooled analysis of 3418 individuals with CKD. *Am J Kidney Dis*. 2008;51(3):395-406.
17. Hiffman A, Nimtz M, Conradt HS. Molecular characterization of beta-trace protein in human serum and urine: A potential diagnostic marker for remal diseases. *Glycobiology*. 1997;7(4):499-506.
18. Fan L, Levey AS, Gudnason V, et al. Comparing GFR estimating equations using cystatin c and creatinine in elderly individuals. *J Am Soc Nephrol*. 2015;26(8):1982-1989.
19. Collister D, Ferguson T, Komenda P, Tangri N. The patterns, risk factors, and prediction of progression in chronic kidney disease: A narrative review. *Semin Nephrol*. 2016;36(4):273-282.
20. Morales E, Praga M. The effect of weight loss in obesity and chronic kidney disease. *Curr Hypertens Rep*. 2012;14(2):170-176.
21. Steinman TI. Kidney protection: How to prevent or delay chronic renal failure. *Geriatrics*. 1996;51(8):28-35.
22. Dunkler D, Kohl M, Heinze G, et al. Modifiable lifestyle and social factors affect chronic kidney disease in high-risk individuals with type 2 diabetes mellitus. *Kidney Int*. 2015;87(4):784-791.
23. Galbraith L, Hemmelgarn B, Manns B, et al. *Can J Kidney Health Dis*. 2016;3:35.
24. Howden EJ, Coombes JS, Isbel NM. The role of exercise training in the management of chronic kidney disease. *Curr Opin Nephrol Hypertens*. 2015;24(6):480-487.
25. Piccoli GB, Capizzi I, Vigotti FN, et al. Low protein diets in patients with chronic kidney disease: A bridge between mainstream and complementary-alternative medicine? *BMC Nephrol*. 2016;17(1):76.