Published in final edited form as:

J Ren Nutr. 2008 July ; 18(4): 323–331. doi:10.1053/j.jrn.2008.04.002.

Trends and Outcomes Associated With Serum Albumin Concentration Among Incident Dialysis Patients in the United States

George A. Kaysen, MD, PhD^{*}, Kirsten L. Johansen, MD^{†,‡,§}, Su-Chun Cheng, ScD[‡], Chengshi Jin, PhD[‡], and Glenn M. Chertow, MD, MPH[¶]

^{*}Division of Nephrology, Department of Medicine and Department of Biochemistry and Molecular Medicine, University of California at Davis, Davis, California, and VA Northern California, Sacramento, California.

[†]Division of Nephrology, San Francisco VA Medical Center, San Francisco, California.

[‡]Department of Epidemiology and Biostatistics, University of California at San Francisco, San Francisco, California.

[§]Department of Medicine, University of California at San Francisco, San Francisco, California.

[¶]Division of Nephrology, Department of Medicine, Stanford University School of Medicine, Stanford, California, and the United States Renal Data System Special Studies Center in Nutrition/Malnutrition.

Abstract

Objective and Methods: Serum albumin concentrations are associated with mortality, and respond to nutritional and inflammatory states. To explore whether changing demographics and practice patterns in dialysis have influenced serum albumin concentrations, we analyzed trends in serum albumin among incident patients on dialysis from 1995 through 2004.

Results: Mean serum albumin concentrations declined significantly over time, even after accounting for changes in age, diabetes, body size, and other factors. Although laboratory assays were not uniform within or across years, serum albumin declined over time, regardless of the reported laboratory lower limit of normal. Moreover, serum albumin retained its potent association with mortality over time. Lower serum albumin was especially hazardous among younger patients and blacks, and was less hazardous among persons with diabetes as a primary cause of kidney disease.

Conclusions: Despite higher body weights and the initiation of dialysis earlier in the course of progressive chronic kidney disease, hypoalbuminemia remains common and hazardous to persons starting dialysis.

THE DEMOGRAPHIC characteristics of incident dialysis patients have evolved over time.¹ The mean age of incident dialysis patients has increased from 61 to 65 years over the decade spanning 1995 to 2005, with increasing proportions of elderly and very elderly patients,² and a corresponding increase in the fraction of patients with type II diabetes mellitus and obesity. This change has occurred in the context of changing patterns of care. The use of

erythropoietic stimulating agents has increased. The estimated glomerular filtration rate (eGFR) at which patients begin dialysis has also been described as increasing steadily, a practice supported by clinical practice guidelines, in part to avoid the declining nutritional status that may accompany worsening kidney function.³

Albumin serves as a surrogate for nutritional status and for inflammation. In the absence of inflammation or the type of catabolic stress associated with dialysis,⁴ albumin concentrations remain near normal, even in the face of significant calorie and protein malnutrition.⁵ Dialysis patients, however, are subject to several forces that disrupt albumin homeostasis, including urinary or dialytic albumin loss,⁶⁻⁸ chronic or intercurrent inflammatory events,^{9,10} and the inadequate dietary intake that may result from advanced age or underlying illness. All of these factors contribute to reduced serum albumin concentrations. Moreover, serum albumin tends to decline the most in individuals who are both malnourished and inflamed.^{10,11}

Hypoalbuminemia, regardless of its cause, is associated with mortality and impaired functional status.^{9,12-16} Other nutritional proxies and inflammatory markers are also associated with mortality and poorer physical functioning, yet albumin remains the most commonly measured marker of health and well-being in the dialysis population. We conducted the current analysis to determine whether changes in the dialysis population or selected practice patterns had influenced the frequency or consequences of hypoalbuminemia.

Methods

In considering trends in serum albumin, we first identified all patients who initiated dialysis between April 1995 and December 2004. We used available data on the Centers for Medicare and Medicaid Services Medical Evidence Form (referred to as the "2728"). We compared selected demographic, clinical, and other laboratory factors among patients with missing and nonmissing serum albumin determinations. In analyses of trends of serum albumin, we included only patients with nonmissing serum albumin values, and made no attempt to impute missing values. We compared the mean level of serum albumin over time, using general linear models. Because the mean age and the fraction of elderly patients starting dialysis have increased over the past decade, and because mean serum albumin concentrations are known to differ by age, sex, and race in the general population^{17,18} as well as in end-stage renal disease (ESRD),¹⁹ we performed regression analyses adjusted for case mix (herein defined as age, sex, race, and geographic region, the latter defined by the ESRD Network, one of 18 organizations that service geographic areas based on the number and concentration of ESRD beneficiaries.). We performed a multivariable linear regression, adjusting for other clinical characteristics significantly associated with serum albumin concentration. To evaluate whether the trend in serum albumin differed among major demographic groups, we evaluated time $\times$ age, sex, race, and primary kidney disease interaction terms in the general linear models.

For analyses of the association of serum albumin with mortality, we compared risk by categories of serum albumin compared with the reference group (serum albumin ≥ 3.9 g/dL). In these analyses, we restricted the population to patients with at least 1 year of potential follow-up (patients who initiated dialysis through December 31, 2004). Unadjusted, case mix-adjusted, and multivariable-adjusted analyses were conducted using logistic regression with 1-year mortality as the outcome of interest. We calculated odds ratios (ORs) and 95% confidence intervals (95% CIs) from model parameter coefficients and standard errors, respectively. Where comorbid conditions were missing, we considered them absent. For patients with non-missing serum albumin values but missing values for key covariates (e.g.,

body weight and hemoglobin), the latter were imputed to the population mean. To determine whether the association between serum albumin and mortality was dependent on demographic characteristics, we evaluated selective multiplicative interaction terms (e.g., serum albumin $\times$ age, or serum albumin $\times$ race), using serum albumin as a continuous variable. Because albumin was determined in local laboratories using multiple methods and devices, we could not dismiss the possibility that changes in serum albumin concentrations over time might be related to a change in the assay used to measure albumin. To explore whether changes in serum albumin concentration over time might be related to changes in assay (e.g., bromocresol purple [BCP] versus bromocresol green [BCG]), we determined whether there was any trend in the median "laboratory lower limit" for serum albumin. We used the reported lower limit of albumin as a surrogate for the type of assay utilized, and analyzed trends in serum albumin for patients in whom the lower limit of albumin was reported to be 3.2 g/dL and 3.5 g/dL.

The eGFR was calculated using both the 4-variable (incorporating age, sex, race (black vs. nonblack), serum creatinine, urea nitrogen, and albumin) and 6-variable Modification of Diet in Renal Disease equations:

$$4- \text{ variable:}^{20} \left[\text{GFR} = 186 \times (\text{P}_{\text{Cr}})^{(-1.154)} \right] \\ \times \text{Age}^{(-0.203)} \times [0.742 \text{ if patient is female}] \\ \times (1.212) \text{ if patient is black.}$$

$$6- \text{ variable:}^{21} \left[\text{GFR} = 170 \times (\text{P}_{\text{Cr}})^{(-0.999)} \right] \\ \times \text{Age}^{(-0.176)} \times [0.762 \text{ if patient is female}] \\ \times (1.180) \text{ if patient is black} \\ \times [\text{SUN}]^{-0.170} \times [\text{Alb}]^{+0.318}.$$

Given the very large sample size, we considered two-tailed $P < .001$ to be statistically significant. Statistical analyses were conducted using SAS 9.1 (SAS, Inc., Gary, NC).

Results

Correlates of Serum Albumin Concentration

Table 1 shows the demographic and clinical characteristics of patients stratified by incident serum albumin concentration. One fourth of the patients did not have their serum albumin recorded (the 2728 form calls for documentation of laboratory values within 45 days of dialysis initiation). No clinical characteristics were particularly common among patients with missing serum albumin concentrations. In general, serum albumin concentrations were lower among women compared with men, and among blacks and Native Americans compared with whites. Among primary causes of kidney disease, diabetes mellitus was associated with lower serum albumin concentrations, and polycystic kidney disease was associated with higher serum albumin concentrations. As expected, lower serum albumin concentrations were associated with major comorbid conditions. There were no unexpected associations observed among persons with missing serum albumin concentrations.

Time Trends in Serum Albumin

Table 2 shows the demographic, clinical, and laboratory data stratified by incident year. The serum albumin concentration of incident dialysis patients declined annually between 1995 and 2002 (Fig. 1), after which the decline appeared to level off. Over the 9 years of

observation, serum albumin declined by approximately 0.15 to 0.20 g/dL. The decline was steepest between 1998 and 2001. While the mean age and the fraction of elderly dialysis patients have been increasing over time^{1,2}(Table 2), the decline in Serum albumin was independent of age, sex, race, primary kidney disease, insurance status, comorbid conditions, previous transplantation, and other laboratory parameters (Fig. 1). The decline in serum albumin was relatively uniform across major demographic and clinical variables. Only among the most elderly of patients (>80 years) was the temporal decline in albumin not observed ($P < .0001$ for time $\times$ age category interaction). The temporal decline in serum albumin was observed, regardless of the albumin laboratory lower limit of normal (Fig. 2).

Serum Albumin and Mortality

Incident serum albumin concentration was inversely proportional to the relative risk of death, even after adjustment for age, sex, race or ethnicity, and comorbidities (Fig. 3). The risk varied little over time, with a slightly more pronounced association between albumin and mortality in more recent years (Fig. 4, interaction $P < .0001$). The fully adjusted OR associated with a 0.2-g/dL decline in albumin was roughly 1.1, or a 10% increase in the odds of death ($P < .0001$), suggesting that the decline observed over the past decade in the level of population mean serum albumin is clinically meaningful.

Whereas younger patients had lower absolute mortality rates, the relative risks associated with lower serum albumin concentrations were more pronounced (interaction $P < .0001$) among younger patients. The association between albumin and mortality risk was similar among men and women (interaction $P = .12$). Lower serum albumin concentrations were also associated with more pronounced risk among blacks compared with nonblacks (interaction $P < .0001$). Patients whose primary cause of ESRD was diabetes mellitus experienced a less pronounced relative risk of death associated with lower serum albumin concentrations (interaction $P < .0001$). Finally, the relative risk of death associated with lower serum albumin concentrations was similar among patients who were obese (Quetelet's index ≥ 30 kg/m²) and nonobese (interaction $P = .51$).

Serum Albumin and eGFR

Serum albumin was directly correlated with serum creatinine and blood urea nitrogen (BUN) at the initiation of dialysis, despite their association with poorer kidney function. The complex confounding of parameters of kidney function by nutritional status is reflected by the relatively large differences observed between GFR estimated using the abbreviated (4-variable) versus 6-variable equation, with the latter including terms for serum albumin and BUN. Indeed, whereas the differences observed between the two estimating equations were modest when the level of serum albumin was 3.0 g/dL or greater, the mean eGFR differed by roughly 20% when the serum albumin was below 3.0 g/dL (Table 1). Nevertheless, whereas eGFR differed by albumin strata when calculated using either the 4-variable or 6-variable method, the trend of increasing eGFR among incident patients was one of significant increase over time, regardless of which model was used for the calculation (Table 2).

Discussion

Using nationally representative data on incident dialysis patients, we determined that the mean serum albumin concentration decreased slightly, and the proportion of patients with hypoalbuminemia increased slightly over the past decade. Serum albumin concentration remains one of the most important prognostic indicators in this population. The changes in albumin concentration observed over time may in part reflect a shift in assay procedures during that period, because values obtained using BCP may be somewhat lower than those

obtained using BCG.^{22,23} The BCP assay is more precise and accurate than BCG when compared using immunologic methods.^{24,25} Because the 2728 form did not ask that the albumin assay be specified, an unknown component of the change in albumin concentration observed may be attributable to a shift in the assays used. However, no change was observed in the reported minimum or median value over this time period, and the decrease in serum albumin concentration observed over time was similar among patients regardless of the laboratory lower limit of serum albumin, suggesting that clinical laboratories (and the reported results) might have adjusted for any changed assay procedures. More importantly, the association of albumin with mortality was not attenuated over time, as might have been anticipated, had the observations resulted from a widespread shift in laboratory assay rather than from biological processes. The association of albumin with mortality was actually slightly more pronounced in more recent years, the opposite of what would be anticipated had the change in albumin levels been a consequence of a drift in the assay employed.

Although the age of incident dialysis patients has increased, it is not possible to attribute the declining serum albumin concentration to advanced age, because the trend was evident across most age groups (as well as other demographic and major clinical categorizations). The actual reason for the declining serum albumin level is unknown. Lower serum albumin concentrations could reflect more significant (yet inadequately measured or documented) comorbidity, despite an earlier initiation of dialysis.

Undernutrition could also explain the decline, although as reported here and by others, there is also a trend toward higher body weight and Quetelet's (body mass) index over the same timeframe. Obesity is associated with inflammation^{26,27} as well as direct liver injury,^{28,29} and may also be associated with hypoalbuminemia, rather than serving as evidence of adequate nutritional status.³⁰ Other processes, such as those associated with acute and chronic inflammation not associated with increased visceral adiposity, may also be contributing to the decline in albumin. It is troubling that serum albumin concentrations are declining despite a trend toward earlier initiation of dialysis (defined as the eGFR at dialysis initiation), because one would believe (and hope) that the undernutrition or inflammation associated with incipient uremia would be less common when fewer patients are left undialyzed with eGFRs in the range of 5 to 10 mL/min/1.73 m².

There are several important limitations to this analysis. First, one quarter of our patients did not have a recent serum albumin level recorded. Although there was no evidence that missing serum albumin values were associated with any particular clinical characteristics, we would have had more confidence in our results if serum albumin concentrations had been obtained and recorded in a larger fraction of the population. Of note, survival rates for the "missing" serum albumin group were intermediate between those of higher and lower serum albumin categories (data not shown). Second, there was no central laboratory for the determination of serum albumin concentrations. As noted, different laboratories employ different assays. Moreover, test characteristics (e.g., coefficients of variation) may differ across laboratories using the same assay. Nevertheless, declining serum albumin concentrations were observed across all major demographic groups, as well as by primary cause of ESRD and geographic region. Third, serum albumin was measured anytime within 45 days of dialysis initiation. Given the fluid shifts and acute conditions that may develop in the weeks before the start of dialysis, the level of serum albumin may change abruptly during this period. Ideally, the timing of a "preinitiation" laboratory study would be uniform. However, a mandate to measure chemistries uniformly would not be possible across the population. Fourth, we had no follow-up serum albumin concentrations after the initiation of dialysis; additional laboratory determinations would have enhanced the predictive power of serum albumin. Moreover, additional laboratory studies, such as C-reactive protein or

prealbumin (transthyretin), might be helpful in teasing out the reasons for abnormal serum albumin concentrations.

In conclusion, serum albumin concentrations have declined over time among patients initiating dialysis in the United States. This trend appears to be independent of the aging of the dialysis population, as well as of changes in other demographic and clinical characteristics. A single serum albumin concentration, determined within several weeks of dialysis initiation, remains a potent and independent risk factor for mortality. Lower serum albumin concentrations are particularly ominous among younger persons and blacks. Finally, these findings suggest that trends in predialysis care, including earlier initiation of dialysis, have not abrogated the visceral protein component of malnutrition in patients with advanced chronic kidney disease.

Acknowledgments

This project was supported by contract N01-DK- 7-5007 from the National Institute of Diabetes, Digestive and Kidney Diseases of the National Institutes of Health.

References

1. Kazmi WH, Gilbertson DT, Obrador GT, et al. Effect of comorbidity on the increased mortality associated with early initiation of dialysis. *Am J Kidney Dis.* 2005; 46:887–896. [PubMed: 16253729]
2. Kurella M, Covinsky KE, Collins AJ, Chertow GM. Octogenarians and nonagenarians starting dialysis in the United States. *Ann Intern Med.* 2007; 146:177–183. [PubMed: 17283348]
3. National Kidney Foundation. K/DOQI clinical practice guidelines for hemodialysis adequacy. *Am J Kidney Dis.* 2001; 37(Suppl 1):S7–S64. update 2000.
4. Ikizler TA, Wingard RL, Sun M, Harvell J, et al. Increased energy expenditure in hemodialysis patients. *J Am Soc Nephrol.* 1996; 7:2646–2653. [PubMed: 8989743]
5. Rigaud D, Hassid J, Meulemans A, et al. A paradoxical increase in resting energy expenditure in malnourished patients near death: the king penguin syndrome. *Am J Clin Nutr.* 2000; 72:355–360. [PubMed: 10919927]
6. Kaysen GA, Schoenfeld PY. Albumin homeostasis in patients undergoing continuous ambulatory peritoneal dialysis. *Kidney Int.* 1984; 25:107–114. [PubMed: 6727122]
7. Yeun JY, Kaysen GA. Acute phase proteins and peritoneal dialysate albumin loss are the main determinants of serum albumin in peritoneal dialysis patients. *Am J Kidney Dis.* 1997; 30:923–927. [PubMed: 9398142]
8. Kaysen GA, Stevenson FT, Depner TA. *Am J Kidney Dis.* 1997; 29:658–668. [PubMed: 9159298]
9. Zimmermann J, Herrlinger S, Pruy A, et al. Inflammation enhances cardiovascular risk and mortality in hemodialysis patients. *Kidney Int.* 1999; 55:648–658. [PubMed: 9987089]
10. Kaysen GA, Dubin JA, Muller HG, et al. The acute-phase response varies with time and predicts serum albumin levels in hemodialysis patients. *Kidney Int.* 2000; 58:346–352. The HEMO Study Group. [PubMed: 10886581]
11. Kaysen GA, Chertow GM, Adhikarla R, et al. Inflammation and dietary protein intake exert competing effects on serum albumin and creatinine in hemodialysis patients. *Kidney Int.* 2001; 60:333–340. [PubMed: 11422769]
12. Lowrie EG, Lew NL. Death risk in hemodialysis patients: the predictive value of commonly measured variables and an evaluation of death rate differences between facilities. *Am J Kidney Dis.* 1990; 15:458–482. [PubMed: 2333868]
13. Yeun JY, Levine RA, Mantadilok V, Kaysen GA. C-reactive protein predicts all-cause and cardiovascular mortality in hemodialysis patients. *Am J Kidney Dis.* 2000; 35:469–476. [PubMed: 10692273]

14. Honda H, Qureshi AR, Heimbürger O, et al. Serum albumin, C-reactive protein, interleukin 6, and fetuin A as predictors of malnutrition, cardiovascular disease, and mortality in patients with ESRD. *Am J Kidney Dis.* 2006; 47:139–148. [PubMed: 16377395]
15. Cereda E, Vanotti A. The new geriatric nutritional risk index is a good predictor of muscle dysfunction in institutionalized older patients. *Clin Nutr.* 2007; 26:78–83. [PubMed: 17067726]
16. Schalk BW, Visser M, Deeg DJ, Bouter LM. Lower levels of serum albumin and total cholesterol and future decline in functional performance in older persons: the Longitudinal Aging Study Amsterdam. *Age Ageing.* 2004; 33:266–272. [PubMed: 15082432]
17. Gardner MD, Scott R. Age- and sex-related reference ranges for eight plasma constituents derived from randomly selected adults in a Scottish new town. *J Clin Pathol.* 1980; 33:380–385. [PubMed: 7400337]
18. McPherson K, Healy MJ, Flynn FV, et al. The effect of age, sex and other factors on blood chemistry in health. *Clin Chim Acta.* 1978; 84:373–397. [PubMed: 639328]
19. Kaysen GA, Greene T, Daugirdas JT, et al. Longitudinal and cross-sectional effects of C-reactive protein, equilibrated normalized protein catabolic rate, and serum bicarbonate on creatinine and albumin levels in dialysis patients. *Am J Kidney Dis.* 2003; 42:1200–1211. [PubMed: 14655192]
20. Hillege HL, Nitsch D, Pfeffer MA, et al. Candesartan in Heart Failure: Assessment of Reduction in Mortality and Morbidity (CHARM) Investigators. Renal function as a predictor of outcome in a broad spectrum of patients with heart failure. *Circulation.* 2006; 113:671–678. [PubMed: 16461840]
21. Levey AS, Bosch JP, Lewis JB, et al. Modification of Diet in Renal Disease Study Group. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. *Ann Intern Med.* 1999; 130:461–470. [PubMed: 10075613]
22. Joseph R, Tria L, Mossey RT, et al. Comparison of methods for measuring albumin in peritoneal dialysis and hemodialysis patients. *Am J Kidney Dis.* 1996; 27:566–572. [PubMed: 8678068]
23. Blagg CR, Liedtke RJ, Batjer JD, et al. Serum albumin concentration-related Health Care Financing Administration quality assurance criterion is method-dependent: revision is necessary. *Am J Kidney Dis.* 1993; 21:138–144. [PubMed: 8430673]
24. Duly EB, Grimeson S, Grimeson P, et al. Measurement of serum albumin by capillary zone electrophoresis, bromocresol green, bromocresol purple, and immunoassay methods. *J Clin Pathol.* 2003; 56:780–781. [PubMed: 14514785]
25. Carfray A, Patel K, Whitaker P, et al. Albumin as an outcome measure in haemodialysis in patients: the effect of variation in assay method. *Nephrol Dial Transplant.* 2000; 15:1819–1822. [PubMed: 11071971]
26. Bahceci M, Gokalp D, Bahceci S, et al. The correlation between adiposity and adiponectin, tumor necrosis factor alpha, interleukin-6 and high sensitivity C-reactive protein levels. Is adipocyte size associated with inflammation in adults? *J Endocrinol Invest.* 2007; 30:210–214. [PubMed: 17505154]
27. Winkler G, Kiss S, Keszthelyi L, et al. Expression of tumor necrosis factor (TNF)-alpha protein in the subcutaneous and visceral adipose tissue in correlation with adipocyte cell volume, serum TNF-alpha, soluble serum TNF-receptor-2 concentrations and C-peptide level. *Eur J Endocrinol.* 2003; 149:129–135. [PubMed: 12887290]
28. Motrin C, Moretto M, Padoin AV, et al. The role of ultrasound in the diagnosis of hepatic steatosis in morbidly obese patients. *Obes Surg.* 2004; 14:635–637. [PubMed: 15186630]
29. Brunt EM. Pathology of fatty liver disease. *Mod Pathol.* 2007; 20(Suppl 1):S40–S48. [PubMed: 17486051]
30. Torun D, Micozkadioglu H, To run N, et al. Increased body mass index is not a reliable marker of good nutrition in hemodialysis patients. *Ren Fail.* 2007; 29:487–493. [PubMed: 17497474]

Figure 1.

Time trends in serum albumin stratified by age group (<45, 45 to 64, 65 to 79, and >80 years), sex, race (black versus nonblack), and primary disease (diabetes versus other) and status (A, B, C, and D, respectively). Mean serum albumin concentrations are fully adjusted (all other demographic, clinical, and laboratory factors are available on the 2728 form).

Figure 2. Time trends in serum albumin stratified by a reported laboratory lower limit of 3.2 g/dL and 3.5 g/dL. Mean serum albumin concentrations are fully adjusted.

Figure 3.
Odds ratio of death at 1 year for albumin concentrations less than or equal to 3.9 g/dL
(values are unadjusted, case mix-adjusted, and multivariable adjusted).

Figure 4. Odds ratio of death by year per 0.2 g/dL decrease in serum albumin (values are unadjusted, case mix-adjusted, and multivariable adjusted).

Table 1

Selected Clinical Characteristics of Incident Dialysis Patients by Serum Albumin Concentration

Variable (Mean ± SD)	Serum Albumin Concentration					
	<2.5 g/dL (n = 94,844)	2.5 to 2.9 g/dL (n = 133,221)	3.0 to 3.4 g/dL (n = 181,488)	3.5 to 3.9 g/dL (n = 146,137)	4.0 g/dL (n = 69,189)	Missing (n = 215,469)
Age (y)	60.1 ± 15.9	62.9 ± 15.3	63.6 ± 15.2	62.8 ± 15.5	60.0 ± 16.3	62.5 ± 15.5
Sex (% male)	50.4%	51.0%	53.6%	56.4%	59.4%	53.6%
Race (%)						
White	59.3%	63.7%	65.4%	66.3%	67.9%	64.7%
Black	33.5%	29.8%	28.7%	28.2%	26.5%	29.0%
Asian	3.6%	3.5%	3.5%	3.4%	3.5%	3.3%
Native American	2.0%	1.4%	1.0%	0.7%	0.6%	1.3%
Other	1.7%	1.5%	1.4%	1.3%	1.4%	1.7%
Ethnicity (% Hispanic)	14.0%	12.9%	11.6%	10.6%	10.4%	13.3%
Weight (kg)	72.1 ± 21.7	72.7 ± 22.0	73.5 ± 22.1	74.0 ± 22.3	73.6 ± 21.9	74.1 ± 22.0
BMI (kg/m ²)	26.3 ± 11.4	26.5 ± 11.3	26.7 ± 11.8	26.7 ± 11.5	26.4 ± 11.4	26.9 ± 12.1
Serum creatinine (mg/dL)	7.4 ± 3.6	7.5 ± 3.6	7.6 ± 3.7	7.8 ± 3.7	8.1 ± 3.7	7.5 ± 3.6
BUN (mg/dL)	84.6 ± 34.2	88.4 ± 34.3	90.0 ± 33.9	90.2 ± 33.2	91.1 ± 33.2	87.6 ± 33.6
Hemoglobin (g/dL)	9.4 ± 1.7	9.5 ± 1.7	9.7 ± 1.7	10.0 ± 1.8	10.4 ± 1.8	9.8 ± 1.8
GFR 6-variable *	7.8 ± 4.3	8.2 ± 4.2	8.4 ± 4.2	8.6 ± 4.2	8.7 ± 4.3	9.2 ± 4.7
GFR 4-variable *	9.6 ± 5.5	9.2 ± 5.0	9.0 ± 4.9	8.8 ± 4.7	8.5 ± 4.6	9.3 ± 5.1
EPO use (%)	23.4%	26.7%	29.6%	33.0%	34.7%	27.7%
Primary kidney disease (%)						
Diabetes	60.6%	58.4%	53.3%	45.3%	33.4%	51.5%
Hypertension	22.7%	27.5%	31.4%	35.2%	38.1%	32.6%
Glomerulonephritis	12.8%	10.0%	10.3%	12.5%	15.9%	10.3%
PKD	0.7%	1.1%	1.7%	3.5%	8.6%	2.4%
Other	3.2%	3.1%	3.2%	3.5%	4.0%	3.2%
Initial dialysis modality (% PD)	4.4%	5.7%	8.0%	12.4%	19.5%	8.1%
Comorbidities (%)						
Heart failure	33.6%	37.1%	35.4%	29.8%	22.0%	30.9%
Ischemic heart disease	22.2%	26.5%	27.1%	24.8%	20.6%	22.8%
Peripheral arterial disease	16.2%	16.7%	15.7%	13.6%	10.6%	13.0%
Inability to ambulate	7.6%	5.7%	4.2%	2.6%	1.7%	3.6%
1 other comorbidity	29.2%	31.4%	30.4%	27.8%	23.5%	26.2%
Employed (%)	13.5%	14.6%	15.6%	18.6%	24.2%	16.3%
Insurance (% Medicaid)	29.9%	26.5%	23.7%	21.3%	19.3%	24.1%

BMI, body mass index; BUN, blood urea nitrogen; GFR, glomerular filtration rate; eGFR, estimated glomerular filtration rate; EPO, erythropoietin; PKD, polycystic kidney disease; PD, peritoneal dialysis; MDRD, Modification of Diet in Renal Disease.

All comparisons are significant at $P < .0001$.

* GFR is expressed as mL/min/1.73m².

Table 2

Selected Clinical Characteristics of Incident Dialysis Patients by Year

Variable (Mean ± SD)	Year									
	1995 (n = 51,448)	1996 (n = 74,662)	1997 (n = 79,826)	1998 (n = 86,219)	1999 (n = 89,637)	2000 (n = 93,205)	2001 (n = 96,448)	2002 (n = 97,994)	2003 (n = 100,184)	2004 (n = 70,725)
Age (y)	60.6 ± 15.8	61.1 ± 15.8	61.7 ± 15.6	62.0 ± 15.6	62.3 ± 15.6	62.6 ± 15.6	62.8 ± 15.5	63.2 ± 15.5	63.2 ± 15.4	63.0 ± 15.4
Sex (% male)	52.4%	53.4%	53.4%	53.3%	53.5%	53.6%	53.8%	54.5%	54.2%	55.2%
Race (%)										
White	63.6%	63.9%	64.6%	64.3%	64.7%	65.2%	65.1%	64.8%	64.5%	65.0%
Black	30.6%	30.2%	29.7%	29.9%	29.3%	28.6%	28.7%	28.9%	29.0%	28.2%
Asian	3.3%	3.3%	3.2%	3.4%	3.5%	3.5%	3.5%	3.6%	3.6%	3.3%
Native American	1.2%	1.2%	1.2%	1.2%	1.3%	1.2%	1.2%	1.1%	1.1%	1.2%
Other	1.3%	1.4%	1.3%	1.2%	1.2%	1.5%	1.4%	1.5%	1.9%	2.3%
Ethnicity (% Hispanic)	12.3%	11.6%	11.3%	11.2%	11.8%	13.3%	12.0%	12.6%	13.1%	13.1%
Weight (kg)	66.5 ± 22.3	67.7 ± 22.3	68.2 ± 22.5	69.4 ± 22.5	72.1 ± 21.9	75.7 ± 20.7	76.7 ± 21.0	77.2 ± 21.0	77.8 ± 21.2	78.5 ± 21.5
BMI (kg/m ²)	24.3 ± 12.5	24.6 ± 11.9	24.8 ± 11.8	25.2 ± 11.6	26.5 ± 13.2	28.0 ± 15.2	27.8 ± 12.5	27.7 ± 9.7	27.7 ± 7.9	27.8 ± 7.7
Serum creatinine (mg/dL)	8.7 ± 3.9	8.5 ± 3.8	8.2 ± 3.7	7.9 ± 3.7	7.7 ± 3.7	7.4 ± 3.6	7.3 ± 3.6	7.1 ± 3.6	7.0 ± 3.6	6.9 ± 3.5
eGFR 6-variable *	7.2 ± 3.4	7.4 ± 3.5	7.7 ± 3.7	8.1 ± 4.0	8.4 ± 4.2	8.7 ± 4.4	9.0 ± 4.6	9.2 ± 4.7	9.4 ± 4.9	9.6 ± 4.8
eGFR 4-variable *	7.5 ± 3.5	7.7 ± 3.6	8.1 ± 3.8	8.4 ± 4.0	8.8 ± 4.2	9.1 ± 4.4	9.4 ± 4.5	9.6 ± 4.6	9.8 ± 4.7	10.0 ± 4.7
BUN (mg/dL)	94.4 ± 33.3	94.6 ± 33.6	92.9 ± 33.7	90.9 ± 33.7	89.1 ± 33.8	88.2 ± 33.9	87.0 ± 33.7	85.9 ± 33.6	84.8 ± 33.7	83.5 ± 33.1
Hgb (g/dL)	9.3 ± 1.8	9.4 ± 1.8	9.5 ± 1.8	9.5 ± 1.7	9.7 ± 1.8	9.8 ± 1.8	9.9 ± 1.8	10.0 ± 1.7	10.0 ± 1.7	10.1 ± 1.7
EPO use (%)	22.1%	23.8%	24.6%	26.4%	27.9%	29.6%	31.5%	32.7%	32.7%	33.7%
Primary kidney disease (%)										
Diabetes	48.8%	49.8%	50.4%	50.9%	51.0%	52.2%	52.4%	51.9%	52.1%	52.3%
HTN	30.6%	30.6%	31.1%	30.9%	31.2%	31.0%	31.4%	32.0%	32.7%	32.1%
GN	14.1%	13.5%	12.6%	12.2%	11.6%	10.9%	10.5%	10.3%	9.7%	10.0%
PKD	3.0%	2.7%	2.7%	2.7%	2.7%	2.6%	2.5%	2.5%	2.4%	2.4%
Other	3.4%	3.3%	3.3%	3.2%	3.5%	3.3%	3.3%	3.3%	3.2%	3.1%
Modality (% PD)	14.3%	12.8%	11.0%	9.4%	8.7%	8.0%	7.8%	7.1%	6.9%	6.9%
Comorbidities (%)										
Heart failure	31.5%	32.7%	33.7%	32.7%	31.8%	32.4%	32.0%	31.4%	32.1%	32.0%

Variable (Mean ± SD)	Year									
	1995 (n = 51,448)	1996 (n = 74,662)	1997 (n = 79,826)	1998 (n = 86,219)	1999 (n = 89,637)	2000 (n = 93,205)	2001 (n = 96,448)	2002 (n = 97,994)	2003 (n = 100,184)	2004 (n = 70,725)
Ischemic heart disease	21.7%	22.8%	24.0%	23.9%	24.0%	24.9%	25.4%	25.2%	25.4%	25.3%
Peripheral arterial disease	14.2%	14.5%	15.0%	14.8%	14.4%	14.6%	14.5%	14.1%	14.3%	13.9%
Inability to ambulate	5.1%	4.6%	4.3%	4.0%	3.9%	4.1%	3.9%	4.2%	4.3%	4.1%
1 other comorbidity	26.4%	27.0%	28.0%	27.9%	27.6%	28.5%	29.2%	29.4%	29.2%	28.9%
Employed (%)	18.4%	17.9%	17.6%	17.2%	17.0%	16.6%	16.3%	15.9%	15.2%	15.5%
Insurance (% Medicaid)	25.5%	24.2%	23.9%	23.8%	22.9%	24.0%	23.7%	24.5%	25.0%	24.9%

BMI, body mass index; eGFR, estimated glomerular filtration rate; BUN, blood urea nitrogen; EPO, erythropoietin; HTN, hypertension; GN, glomerulonephritis; PKD, polycystic kidney disease; PD, peritoneal dialysis; MDRD, Modification of Diet in Renal Disease.

All comparisons are significant at $P < .0001$.

* eGFR is expressed as mL/min/1.73m².