NUTRICION ARTIFICIAL EN EL PACIENTE RENAL

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Chronic kidney disease: the global challenge

A Meguid El Nahas, Aminu K Bello

Lancet 2005; 365: 331–40

	Incidence of ESRD (per million per year)	Prevalence of ESRF (per million)
Europe		
UK	101	626
European average	135	700
Russia	15	79
Australia		
White people	94	658
Aboriginal people	420	1895
USA		
Overall	336	1403
White people	256	1004
African-American people	982	4432
Less developed countries		
India	34–240	Unknown
Nigeria	Unknown	2·5

Most data are for the period 2000–2003.

Table 1: **Incidence and prevalence of ESRD in various parts of the world**

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In Europe, dialysis alone takes up about 2% of health-care budgets with only a small proportion (<0·1%) of the population needing treatment.

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Panel 1: **Stages of CKD according to the US National Kidney Foundation and the Kidney Disease Outcomes Quality Initiative**

Stage 1
Kidney damage (pathological abnormalities or markers of damage including abnormalities in blood or urine tests or in imaging studies) with normal or raised glomerular filtration rate (≥90 mL per min per 1·73 m^2)

Stage 2
Glomerular filtration rate 60–89 mL per min per 1·73 m^2 with evidence of kidney damage

Stage 3
Glomerular filtration rate 30–59 mL per min per 1·73 m^2

Stage 4
Glomerular filtration rate 15–29 mL per min per 1·73 m^2

Stage 5
End-stage renal failure; glomerular filtration rate <15 mL per min per 1·73 m^2

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Initiation factors

Many cohort studies in the USA[20–22] and Japan[14] have identified hypertension, diabetes, hyperlipidaemia, obesity, and smoking as risk factors or markers in the general population for the development of CKD.

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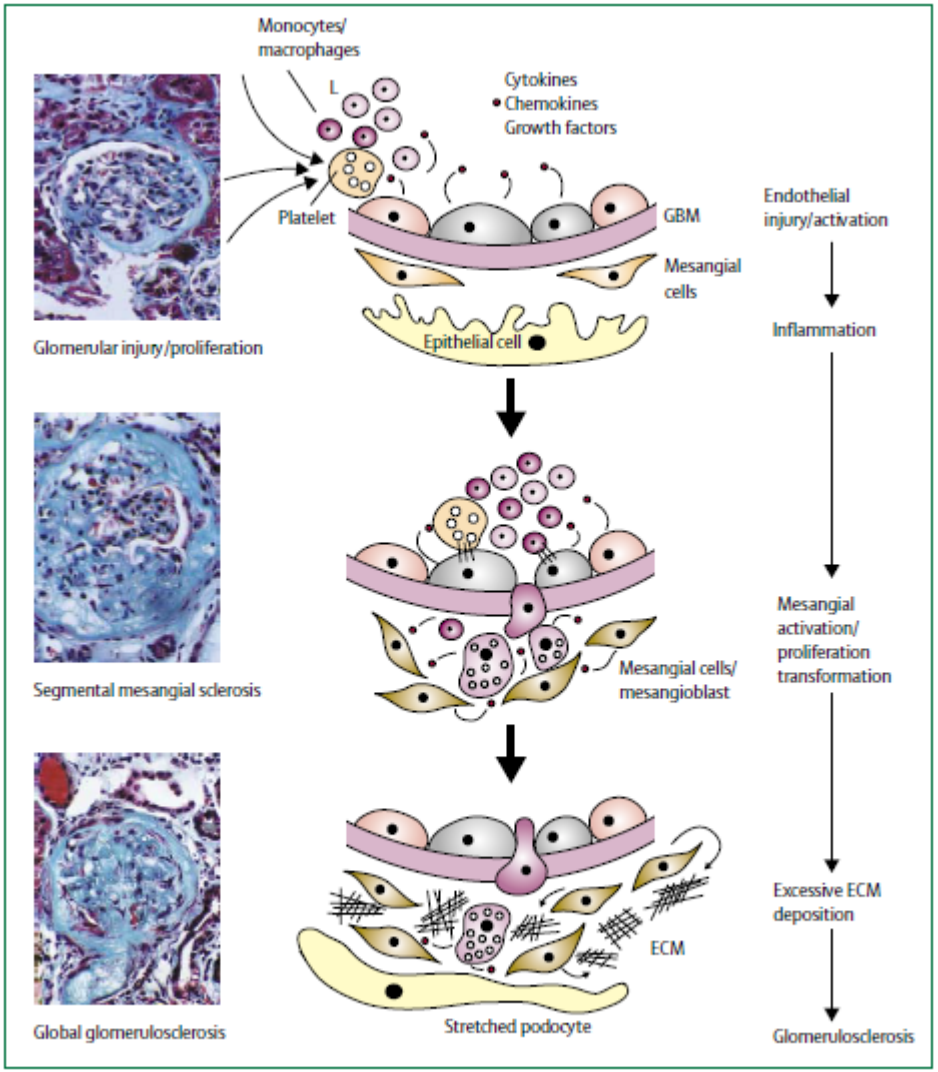

Figure 1: **Diagrammatic representation of the stages of glomerulosclerosis**
GBM=glomerular basement membrane; ECM=extracellular matrix.

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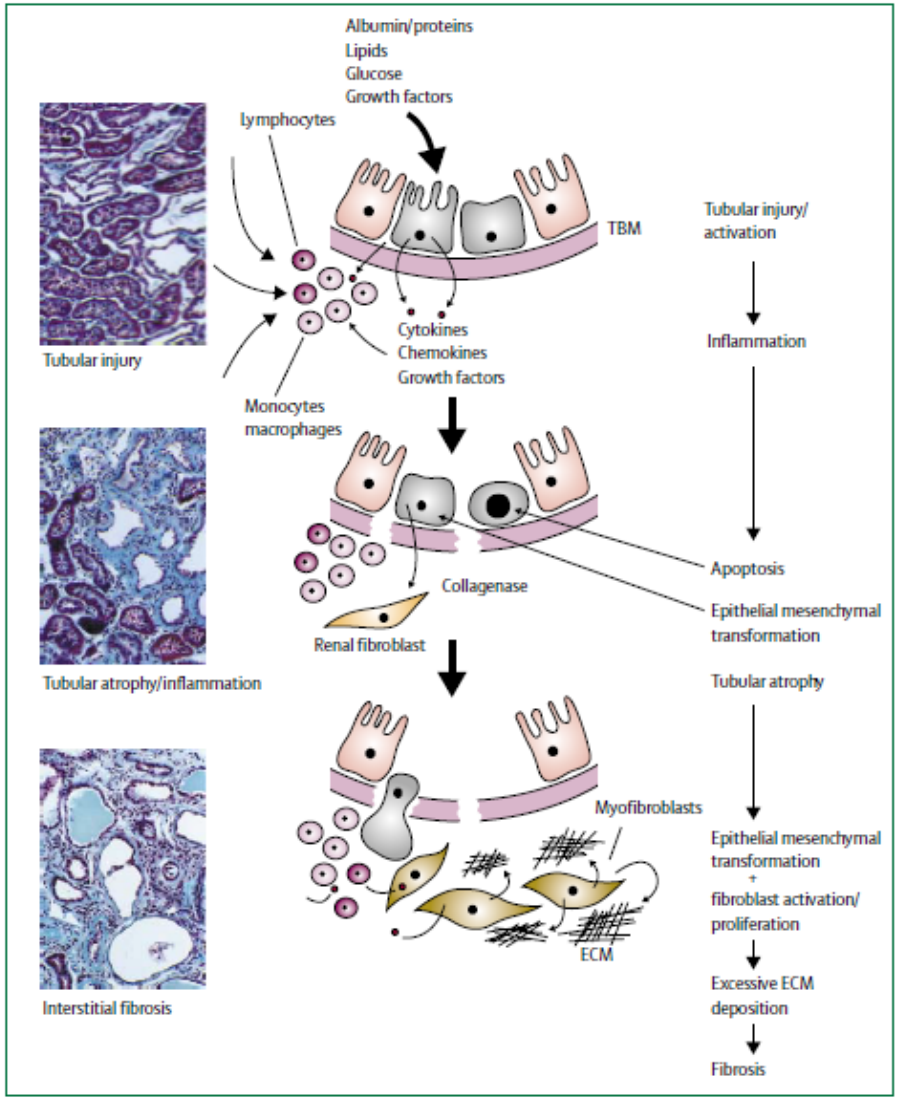

Figure 2: **Diagrammatic representation of the stages of tubulointerstitial fibrosis**
TBM=tubular basement membrane; ECM=extracellular matrix.

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Panel 2: Interventions and objectives to slow the progression of CKD

Diet

Moderate protein restriction: 0·60–0·75 g/kg daily

Low salt: 60–80 mmol/day (4–6 g sodium chloride) to optimise blood-pressure control

Blood-pressure control

For blood pressure <130–135/80–85 mm Hg (mean arterial pressure 92 mm Hg), if proteinuria <1 g in 24 h

For blood pressure <125/75 mm Hg (mean arterial pressure 90 mm Hg), if proteinuria >1 g in 24 h

Initially with an ACE inhibitor

- Add salt restriction/diuretic to maximise the effect of the ACE inhibitor
- Add: angiotensin-2-receptor blocker; or non-dihydropyridine calcium-channel blocker, because they are more effective in reducing proteinuria than dihydropyridine calcium-channel blockers; or β or α blocker

Proteinuria

<1 g in 24 h—use an ACE inhibitor or angiotensin-2-receptor blocker alone or in combination; titrate to control proteinuria even if blood-pressure target is achieved

Blood-glucose control in diabetes mellitus

Haemoglobin A_{1c} 7–8%

Dyslipidaemia

Total cholesterol <5·17 mmol/L,

LDL cholesterol <3·10 mmol/L: use a statin

Smoking

No cigarette smoking

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Panel 3: Interventions and objectives to limit complications of CKD

Cardiovascular

Keep left-ventricular hypertrophy to a minimum and avoid congestive heart failure
Control hypertension
Control dyslipidaemia
Control anaemia
Control hyperparathyroidism
Cessation of smoking

Anaemia

Maintain haemoglobin above 110 g/L; avoid fall below 100 g/L
Correct haematinic deficiencies
Supplement with parental iron in CKD 4–5
Treat with erythropoietin in CKD 4–5

Renal osteodystrophy/hyperparathyroidism

Maintain serum calcium >2·2 mmol/L, serum phosphorus <1·8 mmol/L, parathyroid hormone between normal and twice normal
Reduce phosphate intake to about 800 mg/day
Calcium and vitamin D supplementation

Malnutrition

Adequate protein/calories supplementation
Correct metabolic acidosis
Timely initiation of renal replacement therapy (glomerular filtration rate 10 mL/min)

Review Article

Chronic Kidney Disease Influences Multiple Systems: Describing the Relationship between Oxidative Stress, Inflammation, Kidney Damage, and Concomitant Disease

Patrick S. Tucker,[1,2] Aaron T. Scanlan,[1,2] and Vincent J. Dalbo[1,2]

Oxidative Medicine and Cellular Longevity
Volume 2015,

Conclusions

Nontraditional risk factors such as oxidative stress and inflammation are far more prevalent in chronic kidney disease (CKD) patients than in normal subjects. Malnourished predialysis patients have biochemical evidence of more oxidative stress than well-nourished ones. Therefore, assessing the nutritional intake of macro as well as micronutrients that have antioxidant properties or act as precursors of antioxidant enzymes should be given importance and efforts should be made to meet the RDA (recommended dietary allowance) through diet or supplementation if need arises to combat the antioxidant and other nutritional deficiencies, to minimize oxidative stress and improve the overall health status. Therefore, individualized and parametric counseling for dietary intake is mandatory in CKD patients to increase their awareness about the important nutrients related to their diseased state and how to get these nutrients from diet.

THE LANCET, MARCH 15, 1986

Occasional Survey

DIETARY TREATMENT OF CHRONIC RENAL FAILURE: TEN UNANSWERED QUESTIONS

A. M. EL NAHAS G. A. COLES

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DOES CHRONIC RENAL FAILURE ALWAYS PROGRESS?

HOW SHOULD WE ASSESS THE PROGRESSION OF CHRONIC RENAL FAILURE?

IS THERE A PLACEBO EFFECT IN DIET TRIALS?

HAVE THERE BEEN ANY CONTROLLED STUDIES?

WHEN SHOULD A LOW-PROTEIN DIET START?

THE LANCET, MARCH 15, 1986

Occasional Survey

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WHICH LOW-PROTEIN DIET?

HOW SHOULD COMPLIANCE BE ASSESSED?

WHAT ARE THE RISKS OF A LOW-PROTEIN DIET?

HOW SHOULD NUTRITIONAL STATUS BE ASSESSED?

WHAT IS THE COST OF A LOW-PROTEIN DIET?

SPECIAL COMMENTARY

J Am Soc Nephrol 15: 234–237, 2004

Diets For Patients With Chronic Kidney Disease, Still Worth Prescribing

WILLIAM E. MITCH* and GUISEPPE REMUZZI†

**Department of Medicine, University of Texas, Galveston, Galveston, Texas; and the †Mario Negri Institute for Pharmacological Research and the Unit of Nephrology, Ospedali Riuniti di Bergamo, Bergamo, Italy.*

The "protein intolerance" of CKI is another example of the relationship between dietary indiscretion and symptoms (1): foods rich in protein lead to metabolic acidosis and accumulation of uremic toxins, while an excess of salt will aggravate hypertension and phosphate-rich foods will accelerate secondary hyperparathyroidism.

Many of these complications can be prevented by manipulating the diet (6). It is not surprising, therefore, that Beale (7) concluded over 130 yr ago that a dietary plan should be a standard therapeutic approach for CKI patients.

SPECIAL COMMENTARY

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Diets For Patients With Chronic Kidney Disease, Still Worth Prescribing

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*Department of Medicine, University of Texas, Galveston, Galveston, Texas; and the †Mario Negri Institute for Pharmacological Research and the Unit of Nephrology, Ospedali Riuniti di Bergamo, Bergamo, Italy.

Table 1. Specific dietary requirements for CKI patients

Patients	Protein Requirement	Notes
Normal adults or those with uncomplicated CKI[a]	Minimum: 0.6 g of protein/kg per day	• 30 to 35 kcal/kg per day needed to utilize dietary protein efficiently • Adjustments for specific problems (diabetes, hyperphosphatemia)
CKI patients with muscle mass loss	0.8 g of protein/kg per day	
CKI patients with proteinuria	≤0.8 g of protein/kg per day plus 1 g protein per gram of proteinuria	• This is the maximum needed • Even less dietary protein may be sufficient

[a] CKI, chronic insufficiency of kidney function.

McClellan WM, Knight DF, Rubens DJ, Monk RD, Grossman EB: Early detection and treatment of renal disease in hospitalized diabetic and hypertensive patients: Important differences between practice and published guidelines. *Am J Kid Dis* 29: 368–375, 1997

EDITORIAL

Nutritional Intervention in Chronic Kidney Disease

Philippe Chauveau, MD
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In conclusion, nutritional interventions and specifically supplemented very low protein diets have many proven advantages in terms of the progression of renal failure, better metabolic and endocrine control, and decreased proteinuria. Patients are in need of a detailed nutritional survey by dietitians and nephrologists. This should be the case for all CKD patients, but especially for SVLPD patients, to avoid malnutrition. Toward this goal, all the data reported in this issue by international experts on this topic will help...

Long-term outcome on renal replacement therapy in patients who previously received a keto acid–supplemented very-low-protein diet[1,2]

Philippe Chauveau, Lionel Couzi, Benoit Vendrely, Valérie de Précigout, Christian Combe, Denis Fouque, and Michel Aparicio

Composition of tablets

Ketosteril

One tablet of Ketosteril consisted of ketoisoleucine, calcium salt (67 mg); ketoleucine, calcium salt (101 mg); ketophenylalanine, calcium salt (68 mg); ketovaline, calcium salt (86 mg); keto-DLmethionine, calcium salt (59 mg); L-Lysine acetate (105 mg); L-threonine (53 mg); L-tryptophan (23 mg); L-histidine (38 mg); L-Tyrosine (30 mg); and calcium (50 mg).

Cetolog

One tablet of Cetolog consisted of ketoisoleucine, L-ornithine (153 mg); ketoleucine, L-lysine (162 mg); ketoleucine, L-histidine H_2O (51 mg); ketovaline, L-ornithine (73 mg); ketovaline, L-lysine (77 mg); DL-hydroxymethionine, calcium salt (28 mg); L-threonine (74 mg); and L-tyrosine (151 mg).

Nephrol Dial Transplant (2013) 28: 2295–2305
doi: 10.1093/ndt/gft092
Advance Access publication 9 June 2013

With blog commentary

Vegetarian low-protein diets supplemented with keto analogues: a niche for the few or an option for many?

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Valentina Consiglio[1],
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Federica Fassio[2],
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Keywords: chronic kidney disease, nutrition, progression of chronic renal failure, vegetarian diet

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Review

Nutrition Prescription to Achieve Positive Outcomes in Chronic Kidney Disease: A Systematic Review

Susan Ash [1,*], Katrina L. Campbell [2], Jessica Bogard [3] and Anna Millichamp [3]

Table 3. Nutritional Parameter in International Guidelines with evidence.

Nutrient or Requirement	Most Current Equivalent Guideline Statement	Grade of Evidence Equivalent to GRADE [59]
	KDOQI (2000) [60], BDA (2013) [19]	
Energy-dialysis	The recommended daily energy intake for maintenance haemodialysis or chronic peritoneal dialysis patients is 35 kcal/kg ideal body weight/day (146 kJ/kg IBW/day) for those who are less than 60 years of age and 30 to 35 kcal/kg body weight/day (126–146 kJ/kg IBW/day) for individuals 60 years or older.	C
	CARI (2013) [15]	
Protein–pre-dialysis	We recommend for patients with early CKD consume a normal protein diet of 0.75–1.0 g/kg IBW/day with adequate energy. This is the Recommended Dietary Intake for the general population.	1C
	A low protein diet (≤0.6 g/kg IBW/day) to slow down CKD progression is not recommended because of the risk of malnutrition.	1C
	We suggest that patients with excess protein intakes reduce their intakes to the RDI levels as a high protein diet may accelerate renal function decline in mild renal insufficiency	2C
	ADA (2010) [18]	
Protein–pre-dialysis with keto acids	For adults with CKD without diabetes, not on dialysis, with an eGFR < 20 mL/min, a very low protein controlled diet providing 0.3 g–0.5 g dietary protein per kg of body weight per day with addition of keto acid analogs to meet protein requirements may be recommended. International studies report that additional keto acid analogs and vitamin or mineral supplementation are needed to maintain adequate nutrition status for patients with CKD who consume a very low protein controlled diet (0.3–0.5 g/kg/day)	Strong, conditional evidence

Table 3. *Cont.*

Protein-dialysis	**KDOQI (2000) [59] BDA (2013) [19]** The recommended dietary protein intake for clinically and weight stable maintenance HD patients is 1.1 g/kg ideal body weight/day. At least 50% of the dietary protein should be of high biological value. For clinically and weight stable PD patients, the recommended protein intake is 1.0–1.2 g/kg ideal body weight/day. Those who are not stable may need higher levels of protein.	C
Sodium-pre-dialysis	**CARI (2013) [15]** We recommend that early CKD patients restrict their dietary sodium intake to below 100 mmoL per day or less, as it reduces blood pressure and albuminuria in patients with CKD.	1C
Sodium-dialysis	**KDOQI (2000)[59]** Dietary sodium intake of less than 2.4 g/day (less than 100 mmol/day) should be recommended in most adults with CKD and hypertension.	A
Fluid-pre-dialysis	**CARI (2013) [15]** We suggest that patients drink fluids in moderation. For most patients with early CKD, a daily fluid intake of 2–2.5 L (including fluid content of foods) is sufficient, although this may need to be varied for individual circumstances.	2C
Phosphate-pre-dialysis	**CARI (2013) [15]** We suggest that early CKD patients (stages 1–3) should not restrict dietary phosphate intake as restrictions of dietary phosphate does not influence renal or cardiovascular outcomes in these patients.	2C
	KDIGO (2009) [17] In patients with CKD stages 3–5, we suggest maintaining serum phosphorus in the normal range.	2C
	In patients with CKD stages 3–5 we suggest using phosphate-binding agents in the treatment of hyperphosphatemia.	2D
	It is reasonable that the choice of phosphate binder takes into account CKD stage, presence of other components of CKD–MBD, concomitant therapies, and side-effect profile.	Not graded

Table 3. *Cont.*

	KDIGO (2009) [17]	
Phosphate-dialysis	In patients with CKD stage 5D, we suggest lowering elevated phosphorus levels toward the normal range.	2C
	In patients with CKD stages 5D we suggest using phosphate-binding agents in the treatment of hyperphosphatemia.	2B
	It is reasonable that the choice of phosphate binder takes into account CKD stage, presence of other components of CKD–MBD, concomitant therapies, and side-effect profile.	Not graded
	In patients with CKD stages 3–5D and hyperphosphatemia, we recommend restricting the dose of calcium-based phosphate binders and/or the dose of calcitriol or vitamin D analog in the presence of persistent or recurrent hypercalcemia.	1B
	In patients with CKD stages 3–5D and hyperphosphatemia, we suggest restricting the dose of calcium based phosphate binders in the presence of arterial calcification and/or adynamic bone disease and/or if serum PTH levels are persistently low.	2C
	In patients with CKD stages 3–5D, we recommend avoiding the long-term use of aluminum-containing phosphate binders and, in patients with CKD stage 5D, avoiding dialysate aluminum contamination to prevent aluminum intoxication.	1C
	In patients with CKD stages 3–5D, we suggest limiting dietary phosphate intake in the treatment of hyperphosphatemia alone or in combination with other treatments.	2D
	CARI (2103) [15]	
Fibre	We suggest patients with early CKD consume a diet rich in dietary fibre that is associated with reduced inflammation and mortality in CKD patients.	2D
	CARI (2013) [15]	
Potassium-pre-dialysis	We suggest that early CKD patients with persistent hyperkalaemia restrict their dietary potassium intake with the assistance of a qualified dietitian.	2D

Table 3. *Cont.*

Vitamin D-pre-dialysis	**CARI (2013) [15]**	
	We suggest Vitamin D deficiency (25 hydroxy vitamin D < 37.5nmol/L) and insufficiency (25 hydroxy vitamin D 35.5–75 nmol/L) if present be corrected using treatment strategies for the general population:	2C
	Daily oral intake 19–50 year: 5 μg; 51–70 year: 10 μg; >70 year: 15 μg (1 μg = 40 IU). It is very difficult to meet RDI with food intake alone.	2D
	A few minutes in Australian summer for fair skinned people and 2–3 h of sunlight/week in winter in southern regions.	2D
	We recommend a prescription of vitamin D therapy for early CKD patients with secondary hyperparathyroidism, as it has been shown to be effective in suppressing elevated levels of parathryroid (PTH) hormone. There is insufficient evidence to determine whether this improves patient-level outcomes and the potential benefits of vitamin D therapy must be weighed against its potential deleterious effects, including hypercalcaemia, hyperphosphataemia, vascular calcification, adynamic bone disease and accelerated progression of CKD.	1A
	We recommend that early CKD patients on vitamin D therapy have their calcium, phosphate, PTH, alkaline phosphate and 25(OH) vitamin D level monitored regularly.	1C
Vitamin D-dialysis	**KDIGO (2009) [17]**	
	In patients with CKD stage 5D and elevated or rising PTH, we suggest calcitriol, or vitamin D analogs, or calcimimetics, or a combination of calcimimetics and calcitriol or vitamin D analogs be used to lower PTH.	2B
Calorie restriction/weight loss	**CARI (2013) [15]**	
	We recommend that overweight/obese patients with CKD should be prescribed caloric restriction under the management of an appropriately qualified dietitian. A reduction in weight can mean an improvement of CKD.	1C
	We suggest, in the absence of specific recommendations for CKD, overweight and obese patients are encouraged to aim for a body mass index (BMI) of between 18.5 and 24.9 kg/m^2 and waist circumference of ≤102 cm for men and ≤88 cm for women.	2C
	CMA (2008) [11]	
	Obese (BMI > 30.0 kg/m^2) and overweight (BMI 25.0–29.9 kg/m^2) people should be encouraged to reduce their BMI to lower their risk of chronic kidney diseaseand end-stage renal disease.	D
	Maintenance of a health body weight (BMI 18.5–24.9 kg/m^2; waistcircumference < 102 cm for men, <88 cm for women) is recommended to prevent hypertension.	C
	Or to reduce blood pressure in those with hypertension.	B
	All overweight people with hypertension should be advised to lose weight.	B

Table 3. *Cont.*

Other dietary components	**CARI (2013) [15]** Fruit and vegetables—we suggest adults with early CKD consume a balanced diet rich in fruit and vegetables, as these appear to reduce blood pressure and have renoprotective effects comparable to sodium bicarbonate. Mediterranean diet—we suggest adults with CKD consume a Mediterranean style diet to reduce dyslipidemia and to protect against lipid peroxidation and inflammation.	2C 2C
Counselling	**CARI (2013) [15]** We suggest that patients with progressive CKD have individualised dietary interventions involving an appropriately qualified dietitian. **NICE (2008) [13]** Where the clinician in discussion with the patient has decided that dietary intervention to influence progression of CKD is indicated, an appropriately trained professional should discuss the risks and benefits of dietary protein restriction, with particular reference to slowing down the progression of disease *vs.* protein-calorie malnutrition. Where dietary intervention is agreed this should occur within the context of education, detailed dietary assessment and supervision to ensure malnutrition is prevented. Offer dietary advice to people with progressive CKD concerning potassium, phosphate, protein, calorie and salt intake when indicated.	2C Not graded
Conservative management	**CMA (2008) [11]** Renal programs and care providers for patients with progressive chronic kidney disease who choose not to pursue renal replacement therapies should ensure patients have access to an interdisciplinary team to provide comprehensive conservative management. • All chronic kidney disease programs and care providers should have a mechanism by which to develop documents and processes for advanced-care planning. • Comprehensive conservative management protocols should include symptom management, psychological care and spiritual care. • Coordinated end-of-life care should be available to patients and families.	Not graded