Multidisciplinary care for poor patients with chronic kidney disease in Mexico

Guillermo Garcia-Garcia1, Yolanda Martinez-Castellanos1, Karina Renoirte-Lopez1, Alberto Barajas-Murguia1, Librado de la Torre-Campos1, Laura E. Becerra-Munoz1, Jaime A. Gonzalez-Alvarez2 and Marcello Tonelli3

1Division of Nephrology, Hospital Civil de Guadalajara, University of Guadalajara Health Sciences Center, Guadalajara, Jalisco, Mexico; 2OPD Hospitales Civiles de Guadalajara, Guadalajara, Jalisco, Mexico and 3Department of Medicine, University of Alberta, Edmonton, Alberta, Canada

Coordinated multidisciplinary care (MDC) could improve management and outcomes of patients with chronic kidney disease (CKD). We opened a nurse-led, MDC CKD clinic in Guadalajara, Mexico. We report the clinic’s results between March 2008 and July 2011. The records of 353 patients with CKD stage 3 and 4 were reviewed. Data were collected prospectively. Mean age was 59.1 ± 15.5 years; 54.4% were female and 63.7% were diabetic. We observed significant changes in the quality of care between baseline and follow-up. Compliance with practice guidelines for angiotensin II receptor blockers (ARB) and beta blockers increased from 30.6% to 46.6%, and from 11% to 19%, respectively; for statins from 41.4% to 80.3%; for erythropoietin and calcium binders from 10.5% to 23.4%, and from 41.9 to 82.6%, respectively. At last visit, 90% of patients were on ACE inhibitors/ARB. Blood pressure < 130/80 mm Hg increased from 23% to 38%. Serum glucose < 130 mg/dl increased from 54.4% to 67.7%. Serum cholesterol < 160 mg/dl decreased from 64.8% to 60.3%. At last visit, 70% of the patients had a serum Hgb ≥ 11.0 g/dl, and 80.1% and 65.1% had a normal serum calcium and serum phosphate, respectively. In conclusion, we observed a trend in the improvement of quality of care of CKD patients similar to those reported by other MDC programs in the developed world. Our study demonstrated that a nurse-led MDC program could be successfully implemented in developing countries.

Kidney International Supplements (2013) 3, 178–183; doi:10.1038/kisup.2013.9

KEYWORDS: chronic kidney disease; prevention; outcomes

INTRODUCTION

Chronic kidney disease (CKD) is a public health problem in Mexico and is associated with an increased mortality and substantial health care costs. Approximately 8% of the Mexican adult population has CKD, and the prevalence is twofold higher in high-risk populations. Given these exceedingly high prevalence rates, early detection and management of CKD could have a significant impact at the population level because timely intervention can decrease the incidence of cardiovascular disease and progressive kidney function loss in this population.

Owing to the complexity of care of patients with CKD, it has been recommended that a coordinated multidisciplinary approach could improve management and outcomes in this population. Multidisciplinary clinics have been shown to be associated with reduced morbidity and mortality (once on dialysis) in patients with CKD stages 4 and 5—potentially due to more intensive management of diabetes, hypertension, mineral metabolism and timely vascular access creation.

In a large study of 6978 elderly outpatients with CKD, multidisciplinary care (MDC) was associated with a significant reduction in the risk for all-cause mortality and a trend toward a reduction in risk for hospitalizations. Additionally, MDC has been shown to reduce costs without compromising the quality of life of patients with CKD.

In 2005, our institutions entered into a partnership aimed at preventing kidney failure in the poor of the state of Jalisco, Mexico. The objective of this collaboration is to reduce morbidity and mortality caused by kidney failure by identifying CKD and risk factors for cardiovascular disease in this population. Different strategies have been used to identify cases, including screening in public places on World Kidney Day and promoting awareness of kidney disease among local primary care physicians. Additionally, since 2006, we have screened people at risk of the presence of CKD using mobile units that travel to poor rural and urban communities. Patients with CKD identified in this way are referred to a nurse-coordinated, protocol-driven, MDC clinic. Established in 2008, the clinic provides subsidized care to eligible patients without social security or private medical
insurance. In this study, we report the results of this program between March 2008 and July 2011.

METHODS
The MDC clinic
Our clinic’s operating procedures (Table 1) are based on the multidisciplinary model of the University of Alberta’s MDC Clinic, Edmonton, Canada.10 The clinic is located at the Hospital Civil de Guadalajara, a tertiary care facility that offers comprehensive renal care to the uninsured population of the state of Jalisco. Since 2006, six nephrology fellows from our center have received training in MDC during rotations in Edmonton.

Patients are referred to the clinic by their physicians, other nephrologists, or by the staff of the Fundacion Hospitales Civiles’ mobile units. At the first visit, an education session is held with the patient and his or her family members and is attended by a specialized clinic nurse, registered dietician, and nephrologist. Patient education includes a discussion of CKD and its progression and complications, fluid and dietary restrictions, monitoring BP, effects of medications, and recommendations regarding exercise and diet. MDC patients undergo clinical evaluation and blood work every 1 to 3 months, as determined by nephrologist and nurse, to monitor kidney function and metabolic complications. Management in the MDC clinic is focused on medical management and lifestyle modification to delay progression of CKD and target cardiovascular risk factor reduction. Except for erythropoietin, all medications are provided free of cost by Seguro Popular,14 a national health insurance program for the uninsured (poor urban and rural communities, self-employed, and informal workers).

Table 1 | Operating procedures

| Team member | Intervention |
|-------------|-------------|
| Nurse (30 min) | Retrieval of the patient from waiting area |
| Dietician (30 min) | Review of clinic blood work for K⁺, Ca, PO4, albumin, hemoglobin, uric acid, vitamin D3, cholesterol, triglycerides, HgbA1c and electrolytes |
| Physician (30 min) | Review of clinic blood work for K⁺, Ca, PO4, albumin, hemoglobin, uric acid, vitamin D3, cholesterol, triglycerides, HgbA1c and electrolytes |
| Social worker (30 min) | Review of social and economic factors; employment; family support |

Definitions
Results of dipstick urinalysis demonstrating ≥1+ protein were considered to indicate proteinuria. Hypertension was classified according to the Joint National Committee 7 scheme.15 Patients were classified as having diabetes mellitus if they gave a history of diabetes or had a fasting blood glucose level >126 mg/dl. Levels of total cholesterol and fasting triglycerides were classified according to published guidelines.16 Serum creatinine was used to calculate estimated glomerular filtration rate (eGFR) using the Modification of Diet in Renal Disease (MDRD). Study equation and CKD was classified according to NKF-K/DOQI guidelines.17 Normal values of serum calcium and phosphate for adults in our program were 8.4–10.2 mg/dl and 2.7–4.6 mg/dl, respectively.

Stud outcomes
Assessment of quality of care parameters for management of blood pressure, anemia, glycemic, lipids, and mineral disorder in CKD was done following published NKF-K/DOQI and KDIGO guidelines.18–22
**Statistical analysis**

Characteristics of patients with CKD stages 3 and 4, as well as differences between baseline and follow-up variables were compared with χ²-tests for categorical variables and t-test for continuous variables. A P-value < 0.05 was considered statistically significant. All analysis were conducted with SPSS (version 15.0).

**RESULTS**

A total of 353 patients with CKD stage 3 (n = 175) and stage 4 (n = 178) were followed for a mean of 14.2 months (range 0.8–40.2 months) (Table 2). Mean age was 59.1 ± 15.5 years; 54.4% were female and 63.7% were diabetic; at baseline, serum creatinine and eGFR were 2.29 ± 0.86 mg/dl and 31.7 ± 11.9 ml/min per 1.73 m², respectively; 52.9% had urine protein ≥ 1 +; systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg was present in 56.3% of the patients and 23% had a blood pressure < 130/80 mm Hg. In all, 36.4% had a serum glucose > 126 mg/dl; HgbA1c levels measured at baseline were available in 86 diabetic patients and it was < 7.0% in 26.4% of them. Average hemoglobin was 11.8 ± 1.8 g/dl and 27.7% had hemoglobin < 11 g/dl. Hyperuricemia was present in 46.3% of the patients. Dyslipidemia was highly prevalent; serum cholesterol ≥ 160 mg/dl, and serum triglycerides > 500 mg/dl, were present in 66% and 2.1% of patients, respectively. Female gender (60.7% vs. 48%, P = 0.01), serum hemoglobin < 11.0 g/dl (37.5% vs. 17.6%, P = 0.001), hypocalcemia (26.3% vs. 15.1%, P = 0.03), and proteinuria (61% vs. 44%, P = 0.003) were more prevalent in patients with CKD stage 4 than in stage 3.

| Table 2 | Demographics and clinical characteristics of study participants |
|--------|---------------------------------------------------------------|
|        | All (n=353) | CKD stage 3 (n=175) | CKD stage 4 (n=178) | P-value |
| Age (years) | 59.1 ± 15.5 | 60.7 ± 15.8 | 57.5 ± 15.1 | 0.05 |
| Gender (%) | | | | 0.01 |
| Male | 141 (45.6) | 91 (52) | 70 (39.3) | |
| Female | 192 (54.4) | 84 (48) | 108 (60.7) | |
| Diabetics (%) | 225 (63.7) | 114 (65.1) | 111 (62.4) | 0.32 |
| Height (m) | 1.59 ± 0.9 | 1.6 ± 0.9 | 1.5 ± 0.9 | 0.65 |
| Weight (kg) | 66.8 ± 14.4 | 67.0 ± 14.9 | 66.7 ± 13.9 | 0.84 |
| Body mass index (kg/m²) | 26.0 ± 5.1 | 25.7 ± 5.0 | 26.4 ± 5.26 | 0.21 |
| Serum creatinine (mg/dl) | 2.29 ± 0.86 | 1.70 ± 0.55 | 2.86 ± 0.72 | 0.001 |
| eGFR (ml/min per 1.73 m²) | 31.7 ± 11.9 | 41.6 ± 8.6 | 21.9 ± 4.2 | 0.001 |
| Systolic blood pressure (mm Hg) | 146 ± 30 | 146 ± 31 | 146 ± 30 | 0.97 |
| Diastolic blood pressure (mm Hg) | 79 ± 14 | 78 ± 15 | 80 ± 14 | 0.36 |
| SystBP ≥ 140 or diastBP ≥ 90 mm Hg (%) | 197 (56.3) | 98 (56.0) | 99 (55.6) | 1.00 |
| Hemoglobin (g/dl) | 11.8 ± 1.8 | 12.5 ± 1.8 | 11.2 ± 1.7 | 0.001 |
| ≤ 11 g/dl | 96 (27.7) | 30 (17.6) | 66 (37.5) | 0.001 |
| Serum glucose (mg/dl) | 130 ± 68 | 129 ± 58 | 132 ± 77 | 0.72 |
| > 126 mg/dl (%) | 124 (36.4) | 65 (38.2) | 59 (34.5) | 0.50 |
| < 130 mg/dl (%) | 229 (67.2) | 109 (64.1) | 120 (70.2) | 0.25 |
| HgbA1c (%)a | 8.5 ± 2.3 | 8.6 ± 2.2 | 8.4 ± 2.4 | 0.82 |
| < 7.0 (%) | 20 (5.6) | 32 (86.5) | 26 (63.4) | 0.03 |
| Total cholesterol (mg/dl) | 188 ± 55 | 185 ± 52 | 186 ± 57 | 0.81 |
| ≥ 160 mg/dl | 182 (46.8) | 95 (66.0) | 87 (63.5) | 0.70 |
| Triglycerides (mg/dl) | 186 ± 128 | 190 ± 143 | 182 ± 110 | 0.59 |
| > 500 mg/dl | 5 (1.8) | 3 (2.1) | 2 (1.5) | 1.00 |
| Serum albumin (g/dl) | 3.5 ± 0.6 | 3.6 ± 0.6 | 3.5 ± 0.6 | 0.14 |
| Serum uric acid (mg/dl) | 6.85 ± 2.0 | 6.75 ± 1.89 | 6.97 ± 2.22 | 0.33 |
| Hyperuricemia (%)b | 150 (46.3) | 77 (46.4) | 73 (46.2) | 0.53 |
| Serum calcium (mg/dl) | 8.9 ± 0.8 | 8.9 ± 0.9 | 8.8 ± 0.7 | 0.21 |
| Within normal range (%) | 169 (76.8) | 85 (80.2) | 84 (73.7) | 0.26 |
| < 8.4 mg/dl | 46 (20.9) | 16 (15.1) | 30 (26.3) | 0.04 |
| Serum phosphate (mg/dl) | 4.3 ± 0.8 | 4.2 ± 0.9 | 4.4 ± 0.7 | 0.21 |
| Within normal range (%) | 125 (67.2) | 58 (66.7) | 67 (67.7) | 1.00 |
| > 4.6 mg/dl | 57 (30.6) | 25 (28.7) | 32 (32.2) | 0.63 |
| Proteinuria (%) | 171 (52.9) | 69 (44.2) | 102 (61.1) | 0.003 |

Abbreviations: diastBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; systBP, systolic blood pressure.

aHgbA1c was measured only in diabetic patients.

b > 6.5 mg/dl in females and > 7.5 mg/dl in males.
Pre- and post-intervention results are presented in Table 3. There were significant changes in the quality of care parameters between baseline and the final visit. Use of angiotensin II receptor blockers (ARB) and beta blockers increased from 30.6% to 46.6% (P = 0.001) and from 11% to 19% (P = 0.003), respectively; insulin and statin use increased from 65.0% to 74.3% (P = 0.001) and from 41.4% to 80.3% (P = 0.001), respectively; the use of hypoglycemic agents did not significantly differ (16.8% vs. 17.3%, P = 0.89); allopurinol, erythropoietin, and calcium binders use increased from 20.7% to 63.1% (P = 0.001), from 10.5% to 23.4% (P = 0.001), and from 41.9% to 82.6% (P = 0.001), respectively. At last visit, 66% of patients were taking ACE inhibitors (ACEI) and 90% were taking either ARB or ACEI.

Mean arterial blood pressure decreased from 101 ± 18 mm Hg at referral to 94 ± 18 mm Hg (P = 0.001) at

| Table 3 | Quality of care parameters |
|---|---|---|
| | Baseline (n = 353) | Last visit (n = 353) | P-value |
| **Systolic blood pressure (mm Hg)** | 146 ± 30 | 137 ± 30 | 0.0001 |
| **Diastolic blood pressure (mm Hg)** | 79 ± 14 | 71 ± 15 | 0.0001 |
| **MAP mm Hg** | 101 ± 18 | 94 ± 18 | 0.0001 |
| **SystBP ≥ 140 mm Hg or diastBP ≥ 90 mm Hg** | 197 (56.3) | 162 (46.4) | 0.01 |
| **Blood pressure < 130/80 mm Hg (%)** | 81 (23.1) | 132 (37.8) | 0.0001 |
| **HTN on treatment (%)** | 182 (91.5) | 199 (100) | 0.0001 |
| **HTN treatment and controlled (%)** | 52 (14.4) | 62 (31.5) | 0.0001 |
| **BMI (kg/m²)** | 26.0 ± 5.1 | 25.8 ± 5.4 | 0.17 |
| **Hemoglobin (g/dl)** | 11.8 ± 1.9 | 11.8 ± 1.8 | 0.98 |
| < 11 g/dl (%) | 96 (27.7) | 104 (30.0) | 0.57 |
| **Blood glucose (mg/dl)** | 149 ± 78 | 130 ± 73 | 0.005 |
| ≤ 130 mg/dl (%)a | 114 (50.4) | 153 (67.7) | 0.0001 |
| **HgbA1c (%)a** | 8.5 ± 2.0 | 7.8 ± 1.6 | 0.03 |
| < 7.0 (%) | 20 (25.6) | 27 (31.4) | 0.52 |
| **Total cholesterol (mg/dl)** | 188 ± 59 | 178 ± 53 | 0.003 |
| > 160 mg/dl (%) | 182 (64.8) | 190 (60.3) | 0.30 |
| **Triglycerides (mg/dl)** | 187 ± 130 | 174 ± 115 | 0.11 |
| > 500 mg/dl (%) | 5 (1.8) | 10 (3.1) | 0.44 |
| **Serum uric acid (mg/dl)** | 6.88 ± 2.06 | 6.43 ± 1.92 | 0.001 |
| Hyperuricemia (%)b | 150 (46.3) | 120 (37.0) | 0.02 |
| **Serum albumin (g/dl)** | 3.5 ± 0.6 | 3.5 ± 0.7 | 0.37 |
| **Serum calcium (mg/dl)** | 8.9 ± 0.9 | 8.9 ± 0.7 | 0.46 |
| Within normal range (%) | 169 (76.8) | 218 (80.1) | 0.43 |
| < 8.4 mg/dl | 46 (20.9) | 48 (17.6) | 0.42 |
| **Serum phosphate (mg/dl)** | 4.3 ± 0.8 | 4.4 ± 1.0 | 0.74 |
| Within normal range | 125 (67.2) | 166 (65.1) | 0.72 |
| > 4.6 mg/dl | 57 (30.6) | 82 (33.3) | 0.81 |
| **Serum creatinine (mg/dl)** | 2.29 ± 0.86 | 2.8 ± 1.70 | 0.000 |
| eGFR (ml/min per 1.73 m²) | 32.4 ± 13.0 | 32.4 ± 17.0 | 0.43 |
| eGFR (ml/min per 1.73 m²)a | 31.3 ± 11.3 | 27.5 ± 15.3 | 0.000 |
| **Proteinuria non-DM (%)** | 54 (47.8) | 54 (45.8) | 0.86 |
| **Proteinuria DM** | 117 (55.7) | 122 (56.2) | 0.99 |
| **Insulin use (%)a** | 147 (65.0) | 168 (74.3) | 0.02 |
| **Oral hypoglycemics use (%)a** | 38 (16.8) | 39 (17.3) | 0.89 |
| **ACEI use (%)** | 243 (68.8) | 232 (66.3) | 0.52 |
| **ARB use (%)** | 108 (30.6) | 163 (46.6) | 0.001 |
| **Beta blockers use (%)** | 39 (11.0) | 67 (19.0) | 0.003 |
| **Aspirin use (%)** | 94 (26.7) | 91 (26.0) | 0.89 |
| **Statin use (%)** | 146 (41.4) | 281 (80.3) | 0.001 |
| **Allopurinol use (%)** | 73 (20.7) | 221 (63.1) | 0.001 |
| **EPO use (%)** | 37 (10.5) | 82 (23.4) | 0.001 |
| **Calcitriol use (%)** | 10 (2.8) | 6 (1.7) | 0.41 |
| Calcium binders (%) | 148 (41.9) | 289 (82.6) | 0.001 |

Abbreviations: ACE, ACE inhibitor; ARB, angiotensin II receptor blockers; BMI, body mass index; BP, blood pressure; diastBP, diastolic blood pressure; DM, diabetes mellitus; eGFR, estimated glomerular filtration; EPO, erythropoietin; MAP, mean arterial blood pressure; systBP, systolic blood pressure.

aDiabetic patients only.
bNondiabetic patients.

Kidney International Supplements (2013) 3, 178–183
G Garcia-Garcia et al.: Multidisciplinary CKD care
last visit. Systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg decreased from 56.3% to 46.4% (P = 0.01) and blood pressure < 130/80 mm Hg increased from 23% to 38% (P = 0.001). In diabetic patients, serum glucose decreased from 149 ± 78 mg/dl to 130 ± 73 mg/dl (P = 0.005) and serum glucose ≤ 130 mg/dl increased from 54.4% to 67.7% (P = 0.0001). The proportion of HgbA1c < 7.0% did not significantly differ (25.6% vs. 31.4%, P = 0.52). Serum cholesterol decreased from 188 ± 59 mg/dl to 178 ± 53 mg/dl (P = 0.003), but the proportion with serum cholesterol > 160 mg/dl did not significantly differ (64.8% vs. 60.3%, P = 0.30). At last visit, 70% of the patients had hemoglobin ≥ 11.0 g/dl, and 80.1% and 65.1% had a serum calcium and phosphate within the normal range, respectively. Uric acid decreased from 6.88 ± 2.06 mg/dl to 6.43 ± 1.92 mg/dl (P = 0.001), and the proportion of patients with hyperuricemia decreased from 46.3% to 37.0% (P = 0.02). Proteinuria in nondiabetic (47.8% vs. 45.8%, P = 0.86) and diabetic patients (55.7% vs. 56.2%, P = 0.99) did not significantly differ.

**DISCUSSION**

Our findings suggest that a coordinated MDC could improve management and outcomes in poor CKD patients treated in a developing country. Blood pressure control is a major tool for improving outcomes in patients with CKD; our data demonstrate the potential value of the MDC care for achieving this objective. Despite the increase in the proportion of patients with controlled blood pressure, the majority (62%) remained above the target of <130/80 mm Hg as recommended by the NKF-K/DOQI, and this finding is similar to those reported by Thanamayooran et al. and demonstrate the difficulty in achieving target blood pressure even in an optimal clinic environment. The proportion of patients with proteinuria was similar at follow-up and baseline. However, as we did not use a quantitative method to measure protein excretion, we could not assess the impact of treatment on proteinuria. The average decline in eGFR was −3.8 ml/min per 1.73 m² in diabetic patients, but did not change in nondiabetic individuals, which compares favorably with the average rate of kidney function loss as reported by others.

Hemoglobin levels did not change over time. At last visit, mean Hgb was 11.8 ± 1.8 g/dl, and the proportion of patients meeting the NKF-K/DOQI target of Hgb 11.0–12.0 g/dl at baseline and at follow-up was 30%. Although erythropoietin prescription doubled at follow-up in comparison with the baseline, the majority (77%) of the patients did not receive treatment even after visiting the clinic—likely because of the high cost of this medication.

Calcium control was generally good. The proportion of patients with normal calcium levels rose from 76% at baseline to 80% at last visit. At baseline, hypocalcemia was present in 21% of patients and decreased to 17.6% at last visit, but it did not reach statistical significance. Phosphate control was more difficult to obtain; one-third of the patients had hyperphosphatemia at baseline and did not change at follow-up despite the increase in calcium-binders prescription. Glycemic control improved over time; 67.7% of diabetic patients reached the NKF-K/DOQI guideline target of serum glucose ≤ 130 mg/dl, compared with 50.4% at baseline. However, HgbA1c < 7.0% was achieved in only 30% of 86 patients at last visit. Uric acid levels decreased significantly at the end of the study. Targeting increased uric acid levels has been shown to slow CKD progression and decrease cardiovascular risk. Despite a significant increase in statin prescription, two-thirds of patients had serum cholesterol > 160 mg/dl at follow-up. Whether this was due to poor compliance with medications could not be ascertained.

A significant proportion of patients (23%) were lost to follow-up. Although we did not investigate the reason for dropping out of the program, one possibility is that they cannot afford the expense of traveling to the clinic. Indeed, we have previously reported that many of our dialysis patients abandon their treatment because of the expense of commuting to our hospital. Finally, six patients electively started CAPD, two hemodialysis, and two received living-donor kidney transplants. Five of our patients died at home on follow-up, but the cause of death could not be determined.

Our study has several limitations. First, the length of follow-up was relatively short between the intervention and evaluation of quality targets achieved; therefore, we could not assess the impact of intervention on decline of eGFR or other clinically relevant outcomes, such as mortality and cardiovascular events. Second, although data were collected prospectively, they may not be generalizable to all low-income settings or other regions of Mexico. Third, we used dipstick urinalysis rather than timed urine collection or albumin-creatinine ratios to assess proteinuria; therefore, the prevalence of proteinuria may have been overestimated and did not allow us to assess the impact of treatment. Fourth, the limited availability of HgbA1c measurement did not allow us to properly assess the impact of treatment on diabetes control. Fifth, the MDRD Study equation has not been validated specifically in an unselected Mexican population; therefore, some participants may have been misclassified with respect to the presence or absence of eGFR < 60 ml/min per 1.73 m² or ≥ 15 ml/min per 1.73 m². Sixth, the lack of a control (standard care) group did not allow us to determine if the results are attributable to the MDC alone or to other factors. Finally, although it is tempting to speculate that intervention in people found to have CKD alone or to other factors. Finally, although it is tempting to speculate that intervention in people found to have CKD alone or to other factors. Final...
study demonstrates that nurse-led MDC programs could be successfully implemented in developing countries, and could help to improve clinical outcomes. Although our results suggest that the components of MDC programs may improve care for patients with CKD, further research is needed to evaluate the program component effectiveness in CKD prevention and management.

ACKNOWLEDGMENTS
This work was supported by a grant from the University of Alberta. Publication of this article was supported in part by the National Health and Medical Research Council of Australia through an Australia Fellowship Award (no. 511081: theme Chronic Disease in Health and Medical Research Council of Australia through an investigation of the aetiology and prevention of chronic diseases).

REFERENCES
1. Garcia-Garcia G, Briseño-Rentería G, Luquin-Arellano VM et al. Survival among patients with kidney failure in Jalisco, Mexico. J Am Soc Nephrol 2007; 18: 1922–1927.
2. López-Cervantes M, Rojas-Russell ME, Tirado-Gómez LL et al. Enfermedad renal crónica y su atención mediante tratamiento sustitutivo en México. Facultad de Medicina, Universidad Nacional Autónoma de México: México, D.F., 2009.
3. Amato D, Alvarez-Aguilar C, Castañeda-Limones R et al. Prevalence of chronic kidney disease in an urban Mexican population. Kidney Int 2005; 68(Suppl 97): 11–17.
4. Gutierrez-Padilla JA, Mendoza-García M, Plascencia-Perez S et al. Screening for CKD and cardiovascular disease risk factors using mobile clinics in Jalisco, Mexico. Am J Kidney Dis 2010; 55: 474–484.
5. Obrador GT, García-García G, Villa AR et al. Prevalence of chronic kidney disease in the Kidney Early Evaluation Program (KEEP) Mexico and comparison with KEEP US. Kidney Int 2010; 77(Suppl 116): S2–S8.
6. Mendelsohn DC. Coping with the CKD epidemic: the promise of multi-disciplinary team-based care. Nephrol Dial Transplant 2005; 20: 10–12.
7. Ronksley PE, Hemmelgarn BR. Optimizing care for patients with CKD. Am J Kidney Dis 2012; 60: 133–138.
8. Curtis BM, Ravani P, Malberti F et al. The short- and long-term impact of multi-disciplinary clinics in addition to standard nephrology care on patient outcomes. Nephrol Dial Transplant 2005; 20: 147–154.
9. Goldstein M, Yassa T, Dacouris N et al. Multidisciplinary predialysis care and morbidity and mortality of patients on dialysis. Am J Kidney Dis 2004; 44: 706–714.
10. Hemmelgarn BR, Manns BJ, Zhang J et al. Association between multidisciplinary care and survival for elderly patients with chronic kidney disease. J Am Soc Nephrol 2007; 18: 993–999.
11. Hopkins RB, Garg AX, Levin A et al. Cost-effectiveness analysis of a randomized trial comparing care models for chronic kidney disease. Clin J Am Soc Nephrol 2011; 6: 1248–1257.
12. Koshi SM, Garcia G, Marquez I et al. Screening for kidney disease in children on World Kidney Day in Jalisco, Mexico. Pediatr Nephrol 2009; 24: 1219–1225.
13. Garcia-Garcia G, Marquez-Magaña I, Renoirete-Lopez K et al. Screening for kidney disease on World Kidney Day in Jalisco, Mexico. J Nephrol 2010; 23: 224–230.
14. Frenk J, Gonzalez-Pier E, Gomez-Dantes E et al. Comprehensive reform to improve health system performance in Mexico. Lancet 2006; 368: 1524–1534.
15. Chobanian AV, Bakris GL, Black HR et al. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA 2003; 289: 2560–2571.
16. National Cholesterol Education Program (NCEP) Expert Panel. Detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). National Institutes of Health: Bethesda, MD, 2002.
17. National Kidney Foundation. KDOQI Clinical Practice Guidelines for Chronic Kidney Disease. Am J Kidney Dis 2002; 39(suppl 1): S76–S11.
18. K/DOQI clinical practice guidelines on hypertension and antihypertensive agents in chronic kidney disease. Am J Kidney Dis 2004; 43(suppl 1): S1–S268. http://www.kidney.org/professionals/kdoqi/guidelines_bprcommend.htm. Last accessed June 30th, 2012.
19. KDOQI Clinical Practice Guidelines and Clinical Practice Recommendations for Anemia in Chronic Kidney Disease. Am J Kidney Dis 2006; 47(suppl 3): S1–S46.
20. KDOQI™ Clinical Practice Guidelines and Clinical Practice Recommendations for Diabetes and Chronic Kidney Disease. Am J Kidney Dis 2007; 49(suppl 2): S1–S180.
21. K/DOQI Clinical Practice Guidelines for Managing Dyslipidemias in Chronic Kidney Disease. Am J Kidney Disease 2003; 41(suppl 3): S1–S77.
22. Kidney Disease: Improving Global Outcomes (KDIGO) CKD–MBD Work Group. KDIGO clinical practice guideline for the diagnosis, evaluation, prevention, and treatment of chronic kidney disease-mineral and bone disorder (CKD-MBD). Kidney Int 2009; 76(Suppl 113): S1–S130.
23. Thanamayooran S, Rose C, Hirsch DJ. Effectiveness of a multidisciplinary kidney disease clinic in achieving treatment guideline targets. Nephrol Dial Transplant 2005; 20: 2385–2393.
24. Barrett BJ, Garg AX, Goeree R et al. A nurse-coordinated model of care versus usual care for stage 3/4 chronic kidney disease in the community: A randomized controlled trial. Clin J Am Soc Nephrol 2011; 6: 1241–1247.
25. Bayliss EA, Bhardwaja B, Ross C et al. Multidisciplinary team care may slow the rate of decline in renal function. Clin J Am Soc Nephrol 2011; 6: 704–710.
26. Golcoechea M, García de Vinuesa S, Verdalles U et al. Effect of allopurinol in chronic kidney disease progression and cardiovascular risk. Clin J Am Soc Nephrol 2010; 5: 1388–1393.
27. Carrero JJ, Stenvinkel P. Novel targets for slowing CKD progression. Nat Rev Nephrol 2011; 7: 65–66.
28. Garcia-Garcia G, Renoirete-Lopez K, Marquez-Magaña I. Disparities in renal care in Jalisco, Mexico. Semin Nephrol 2010; 30: 3–7.
29. Collister D, Rigatto C, Hildebrand A et al. Creating a model for improved chronic kidney disease care: designing parameters in quality, efficiency and accountability. Nephrol Dial Transplant 2010; 25: 3623–3630.