# Management of aboriginal and nonaboriginal people with chronic kidney disease in Quebec

## *Quality-of-care indicators*

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## Abstract

***Objective*** To compare quality-of-care indicators for management of patients with chronic kidney disease (CKD) and type 2 diabetes among the James Bay Cree of Northern Quebec with those among residents of Montreal, Que.

***Design*** A cross-sectional survey using medical records from patients seen between 2002 and 2008.

***Setting*** Predialysis clinics of the McGill University Health Centre in Montreal.

***Participants*** Thirty Cree and 51 nonaboriginal patients older than 18 years of age with type 2 diabetes mellitus and estimated glomerular filtration rates of less than 60 mL/min/1.73 $m^2$.

***Main outcome measures*** Rates of anemia, iron deficiency, obesity, and renoprotective medication use among aboriginal and nonaboriginal patients.

***Results*** Overall, the Cree patients were younger (59 vs 68 years of age, $P<.0035$) and weighed more (101 vs 77 kg, $P<.001$). The 2 groups were prescribed medication to control blood pressure, lipids, and phosphate levels at similar rates, but the Cree patients were more likely to receive renoprotective agents (87% vs 65%, $P=.04$). Despite similar rates of erythropoietin supplementation, the Cree patients were at greater risk of anemia, with an adjusted risk ratio of 2.80 (95% CI 1.01 to 7.87).

***Conclusion*** Cree patients with CKD were younger, weighed more, and were more likely to receive renoprotective agents. With the exception of the management of anemia, quality of CKD care was similar between the 2 groups. Anemia education for family physicians and continuous monitoring of quality indicators must be implemented in northern Quebec.

### EDITOR'S KEY POINTS

- Management of Cree patients with chronic kidney disease and diabetes was equivalent to management of nonaboriginal patients.
- The only area of discrepancy was in the management of anemia. Despite similar rates of erythropoietin supplementation, the Cree patients were at greater risk of anemia.
- After adjusting for severe obesity, erythropoietin supplementation, low albumin levels, serum ferritin levels, iron saturation, and stage of kidney disease, the Cree cohort still had a higher risk of anemia compared with the nonaboriginal cohort.

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# Traitement des Autochtones et des non-Autochtones souffrant de maladie rénale chronique au Québec

## *Indices de la qualité des soins*

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## Résumé

***Objectif*** Comparer les indices de qualité pour le traitement des malades souffrant de maladie rénale chronique (MRC) et de diabète de type 2 chez les Cris de la Baie James du Nord du Québec par rapport aux malades vivant à Montréal, Qué.

***Type d'étude*** Enquête transversale à partir des dossiers médicaux des patients ayant consulté entre 2002 et 2008.

***Contexte*** Cliniques de prédialyse du centre de santé de l'Université McGill à Montréal.

***Participants*** Trente Cris et 51 patients non-Autochtones de plus de 18 ans souffrant de diabète de type 2 et ayant un taux de filtration glomérulaire estimé à moins de 60 mL/min/1,73 $m^2$.

***Principaux paramètres à l'étude*** Taux d'anémie, de déficience en fer, d'obésité et de prise d'agents de protection rénale chez les Autochtones par rapport aux patients non autochtones.

***Résultats*** Dans l'ensemble, les patients Cris étaient plus jeunes (59 c. 68 ans, $P<,0035$) et plus lourds (101 c. 77 kg, $P<,001$). Les 2 groupes prenaient, à des taux comparables, des médicaments pour contrôler la tension artérielle et les niveaux des lipides et de phosphate, mais les Cris étaient plus susceptibles de prendre des agents de protection rénale (87% c. 65%, $P=,04$). Même s'ils recevaient des suppléments d'érythropoïétine à des taux semblables, les Cris étaient plus à risque d'anémie, leur taux de risque ajusté s'élevant à 2,80 (IC à 95% 1,01 à 7,87).

***Conclusion*** Les patients cris souffrant de MRC étaient plus jeunes et plus lourds, et ils étaient plus susceptibles de prendre des agents de protection rénale. Sauf pour le traitement de l'anémie, la qualité du traitement de la MRC était semblable dans les 2 groupes. Il y a lieu d'instaurer des séances de formation sur l'anémie pour les médecins de famille et une surveillance continue des indices de qualité dans le Nord du Québec.

### POINTS DE REPÈRE DU RÉDACTEUR

- Le traitement des Cris souffrant de maladie rénale chronique et de diabète était équivalent à celui des patients non autochtones.
- La seule différence observée concernait le traitement de l'anémie. Même s'ils recevaient des suppléments d'érythropoïétine en quantités identiques, les Cris étaient plus à risque d'anémie.
- Après ajustement pour l'obésité sévère, les suppléments d'érythropoïétine, les bas taux d'albumine, les niveaux de ferritine sérique, la saturation en fer et le stade de la maladie rénale, la cohorte des Cris avaient toujours un risque d'anémie plus grand que la cohorte des non-Autochtones.

Cet article a fait l'objet d'une révision par des pairs.
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Quality-of-care indicators are increasingly being used to highlight care parameters that need improvement and to provide optimal treatment of patients with chronic kidney disease (CKD) who are seen in predialysis clinics. It is particularly important to monitor the care received by the James Bay Cree of Northern Quebec. First, the Cree have high rates of type 2 diabetes mellitus.[1] As well, aboriginal people with diabetes appear to be at elevated risk of developing CKD as a complication of diabetes: in 2005, 53% of the Cree people diagnosed with diabetes also had renal complications.[2] There are also differences in how CKD care is provided. The 9 Cree communities are located in the northwestern part of the province, far from urban centres. Consequently, unlike other residents of the province, Cree patients receive most of their CKD care from primary care physicians in the community. At the physicians' discretion, patients with considerable renal disease might be referred to the predialysis or nephrology clinics at the McGill University Health Centre (MUHC) in Montreal, Que.

Aboriginal patients' access to specialized care might be limited by distance and language barriers, and sometimes by distrust of modern-day medical care. Past studies have shown that aboriginal patients generally have less access to nephrologists and to renal transplantation than do nonaboriginal patients in Canada.[3,4] In the case of the James Bay Cree, patients needing specialized care must be transferred by air to the MUHC or to the largest of the Cree communities, Chisasibi, Que, where a nephrologist visits every 3 months. Both sites use the NephroCare electronic medical charting system to record clinical and laboratory data. In Montreal, an interface with the laboratory system allows automatic download of laboratory values into patients' medical charts. In Chisasibi, however, the data must be entered manually.

In short, differences in disease prevalence, primary provider of care, access to specialized care, and record-keeping systems all combine to create a risk that predialysis care will be less satisfactory for Cree patients than for residents of Montreal. Our objective was to compare quality-of-care indicators for Cree and nonaboriginal patients with CKD and type 2 diabetes at the MUHC. We hypothesized that management of blood pressure, lipid levels, bone and mineral metabolism, and anemia was suboptimal for the Cree patients.

To our knowledge, our study is the first to compare quality-of-care indicators for the management of CKD among the James Bay Cree of northern Quebec with those among residents of Montreal.

## METHODS

A cross-sectional survey based on existing health records was performed. Research ethics approval was received from the Director of Professional Services of the MUHC. Adult patients (older than 18 years of age) seen at the MUHC predialysis clinics between 2002 and 2008 who had estimated glomerular filtration rates (eGFR) of less than 60 mL/min/1.73 $m^2$ were included in the study. Only patients with type 2 diabetes were included in order to have a more homogeneous group with respect to the causes of kidney disease. Cree living off reserve, non-Cree patients living on reserve, and patients without type 2 diabetes were excluded from the study.

Two sources of data were used for abstraction of study variables: NephroCare and MUHC medical charts (inpatient and outpatient). The following variables were abstracted: patient demographic characteristics (age, sex, aboriginal status); comorbidities (hypertension, cardiac and peripheral vascular disease, retinopathy, and peripheral neuropathy); weight and body mass index (BMI); blood pressure at the most recent clinic visit; and laboratory values at the most recent clinic visit (serum creatinine, hemoglobin, hemoglobin $A_{1c}$, ferritin, albumin, phosphate, calcium, alkaline phosphates, low-density lipoprotein). Finally, we abstracted data on prescriptions as of the last clinic visit, whether written by the patient's family physician or by a nephrologist. This included prescriptions for antihypertensive agents, antihyperglycemic drugs, insulin, statins, fibrates, erythropoiesis-stimulating agents, and anticoagulants.

Body mass index was calculated as weight/height$^2$ in kg/$m^2$. Patients with BMIs of 25.0 to 29.9 kg/$m^2$ were considered to be overweight, those with BMIs of 30.0 kg/$m^2$ or higher were considered to be obese, and those with BMIs of 35.0 kg/$m^2$ or greater were considered to be severely obese.[5] The eGFR was calculated using the 4-variable MDRD [Modification of Diet in Renal Disease] formula[6] based on serum creatinine measurement, age, race, and sex. This formula has been validated in another aboriginal population.[7] Anemia was defined as hemoglobin concentration below 110 g/L.[8]

Statistical analyses were performed using $\chi^2$ tests for proportions and Wilcoxon rank tests for continuous variables. Multivariate analyses were conducted using the Cox hazard model, with the relevant time period being the elapsed time between first referral to the nephrology clinic and the date when the patient's clinical and laboratory data were abstracted. Statistical significance was set at the .05 level. All statistical analyses were performed with SAS software, version 9.1.

## RESULTS

Thirty Cree and 51 non-Cree patients met the criteria for inclusion in the study. **Table 1** describes patient demographic characteristics. Fifty percent of Cree

and nearly 55% of non-Cree patients were male. Cree patients were significantly younger than their non-Cree counterparts, with median ages of 59.1 and 67.9 years, respectively ($P=.0035$). There were no significant differences between Cree and non-Cree patients with respect to time of follow-up or to diagnoses of hypertension, cardiac disease, peripheral vascular disease, retinopathy, or peripheral neuropathy noted in the medical charts.

**Table 2** describes the clinical characteristics of renal function of the study population. Most notable, the Cree individuals demonstrated a significantly higher median weight than non-Cree patients (101 vs 77 kg, $P<.001$) with a much higher proportion (80%) of Cree patients classified as obese (BMI ≥30 kg/m²). The mean systolic blood pressure was 143 mm Hg for Cree patients and 147 mm Hg for non-Cree patients, and approximately 30% of both groups had systolic pressure less than 130 mm Hg. **Table 2** also shows mean creatinine values of 315 and 291 µmol/L for Cree and non-Cree groups, respectively, as well as the range of severity of renal failure based on eGFR. At the time of the chart review, 37% of Cree patients were classified as having stage-5 kidney failure (eGFR <15 mL/min/1.73 m²), compared with only 27% of non-Cree patients.

The laboratory results and prescription medications for both groups in the study population are presented in **Table 3**. Interestingly, a statistically significant difference in median albumin levels was observed, with Cree patients having a lower median albumin level than non-Cree patients (33.0 vs 38.0 g/L, $P=.029$). All other laboratory results showed no significant differences between the groups. However, the 2 groups differed with respect to the medications prescribed by family physicians or nephrologists. Compared with the non-Cree cohort, the Cree patients were significantly more likely to have been prescribed angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) and phosphate binders ($P=.04$). No statistically significant differences were observed with respect to lipid-lowering agents, oral hypoglycemics, insulin, erythropoietin agents, calcium channel blockers, or β-blockers.

Upon comparing the 2 groups, a potential association between anemia and CKD was suggested. Interestingly, in an unadjusted Cox hazard model, an increased risk of anemia among Cree patients was seen: hazard ratio 2.36 (95% CI 1.1 to 5.1). After adjusting the model for severe obesity (BMI ≥35 kg/m²), erythropoietin supplementation, low albumin, serum ferritin, iron saturation, and stage-5 CKD (eGFR <15 mL/min/1.73 m²), the Cree cohort had a risk ratio of 2.80 (95% CI 1.01 to 7.87) for anemia when compared with nonaboriginal patients (**Table 4**).

**Table 1. Patient demographic characteristics**

| CHARACTERISTICS | CREE* (N = 30) | NON-CREE* (N = 51) | *P* VALUE |
|---|---|---|---|
| Male sex, n (%) | 15 (50.0) | 28 (54.9) | .81 |
| Median age (range), y | 59.1 (35-81) | 67.9 (33-84) | .0035 |
| Median time of follow-up (range), mo | 1.5 (0-20) | 2.0 (0-14) | .45 |
| Median duration of diabetes (range), y† | 16.0 (3-30) | 15.0 (5-50) | .61 |
| Comorbidities noted in chart, n (%) | | | |
| • Hypertension | 29 (96.7) | 42 (82.4) | .25 |
| • Cardiac disease | 11 (36.7) | 21 (41.2) | .81 |
| • Peripheral vascular disease | 6 (20.0) | 12 (23.5) | .79 |
| • Retinopathy | 13 (43.3) | 16 (31.4) | .28 |
| • Peripheral neuropathy | 5 (16.7) | 12 (23.5) | .46 |

*Data were based on the last clinical visit noted in the medical chart.
†Values calculated based on 25 Cree and 49 non-Cree participants.

**Table 2. Clinical characteristics and renal function of the study population**

| CHARACTERISTICS | CREE* (N = 30) | NON-CREE* (N = 51) | *P* VALUE |
|---|---|---|---|
| Median weight (range), kg | 101 (64-163) | 77 (36-121) | <.001 |
| BMI, kg/m², n (%) | | | |
| • Normal | 1 (3.3) | 10 (19.6) | .001 |
| • Overweight | 5 (16.7) | 19 (37.2) | |
| • Obese | 24 (80.0) | 22 (43.1) | |
| Systolic pressure | | | |
| • Mean (SD), mm Hg | 143 (21) | 147 (25.6) | .28 |
| • <130 mm Hg, n (%) | 9 (30.0) | 15 (29.4) | .95 |
| Diastolic pressure | | | |
| • Mean (SD), mm Hg | 73 (14) | 77 (15) | .26 |
| • <80 mm Hg, n (%) | 23 (76.7) | 38 (74.5) | .83 |
| Mean (SD) creatinine, µmol/L | 315 (137) | 291 (119) | .40 |
| eGFR with 4 variables | | | |
| • Mean (SD), mL/min/1.73 m² | 19 (9) | 21 (10) | .40 |
| • <15 mL/min/1.73 m², n (%) | 11 (36.7) | 14 (27.5) | .18 |
| • 15-29 mL/min/1.73 m², n (%) | 16 (53.3) | 26 (51.0) | |
| • 30-59 mL/min/1.73 m², n (%) | 3 (10.0) | 11 (21.6) | |

BMI—body mass index, eGFR—estimated glomerular filtration rate.
*Data were based on the last clinical visit noted in the medical chart.

**Table 3. Laboratory results and prescriptions for the study population**

| TESTS AND MEDICATIONS | CREE* (N = 30) | NON-CREE* (N = 51) | *P* VALUE |
|---|---|---|---|
| **Laboratory tests** | | | |
| Anemia, Hb < 110 g/L, n (%) | 18 (60.0) | 21 (41.2) | .10 |
| Median Hb concentration (range), g/L | 106 (72-142) | 112 (77-151) | .50 |
| Ferritin | | | |
| • Median level (range), µg/L | 103 (5-427) | 127 (16-407) | .93 |
| • < 200 µg/L, n (%) | 18 (60.0) | 38 (74.5) | .17 |
| Iron saturation < 0.21%, n (%) | 17 (56.7) | 26 (51.0) | .62 |
| Albumin | | | |
| • Median level (range), g/L | 33.0 (22-45) | 38.0 (22-50) | .029 |
| • < 30 g/L, n (%) | 6 (20.0) | 5 (9.8) | .31 |
| Inorganic phosphate | | | |
| • Median level (range), µmol/L | 1.45 (0.16-2.36) | 1.39 (0.85-2.8) | .75 |
| • < 1.45 µmol/L, n (%) | 15 (50.0) | 29 (56.9) | .55 |
| Calcium-phosphate product < 2 $mg^2/dL^2$, n (%) | 25 (83.3) | 40 (78.4) | .59 |
| Median alkaline phosphate level (range), IU/L | 104 (59-166) | 100 (50-180) | .27 |
| LDL | | | |
| • Median (range) level, mmol/L | 2.1 (1-4.6) | 2.0 (0.11-4.7) | .29 |
| • < 2.0 mmol/L, n (%) | 17 (56.7) | 29 (56.9) | .98 |
| Median $HbA_{1c}$ level (range), %† | 14 (5-50) | 6.8 (5.5-11.5) | .86 |
| **Medications, n (%)** | | | |
| Lipid-lowering agents | 21 (70.0) | 26 (51.0) | .09 |
| ACEIs or ARBs | 26 (86.7) | 33 (64.7) | .04 |
| Oral hypoglycemics | 10 (33.3) | 17 (33.3) | .83 |
| Insulin | 11 (36.7) | 13 (25.5) | .42 |
| EPO agents | 7 (23.3) | 11 (21.6) | .85 |
| Calcium channel blockers | 14 (46.7) | 17 (33.3) | .33 |
| β-Blockers | 9 (30.0) | 23 (45.1) | .11 |
| Phosphate binders | 10 (33.3) | 6 (11.8) | .04 |

ACEI—angiotensin-converting enzyme inhibitor, ARB—angiotensin receptor blocker, EPO—erythropoietin, Hb—hemoglobin, LDL—low-density lipoprotein.
*Data were based on the last clinical visit noted in the medical chart.
†Data on $HbA_{1c}$ levels were not available for 12 of the Cree participants.

## DISCUSSION

Our findings suggest that management of Cree individuals with CKD and diabetes is equivalent to management for non-Cree individuals. The current system, in which family physicians provide primary care and nephrology consultations, provides similar quality of care among Cree and non-Cree patients, with more Cree patients receiving renoprotective medications. The only area of discrepancy was in the management of anemia. Despite similar rates of erythropoietin supplementation, Cree patients with CKD and diabetes were at greater risk of anemia. Compared with nonaboriginal patients, Cree patients were also significantly younger and had higher BMIs. Management of CKD was otherwise similar between the Cree and nonaboriginal populations.

This study points out one area of concern in medical management of a chronic illness in aboriginal communities. Remote aboriginal communities have higher prevalence of hypertension, diabetes, ischemic heart disease, chronic obstructive pulmonary disease, and kidney disease.[9] Aboriginal populations in Canada and Australia have insufficient access to medical care for chronic illness management.[9-11] Regarding CKD, aboriginal people have a higher burden of comorbidity, but are less likely to obtain nephrology referrals and are less likely to receive kidney transplant.[3,4]

Previous research has shown an increasing trend for erythropoietin requirements in aboriginal patients with CKD.[12] Chou et al compared the quality of care for aboriginal and nonaboriginal hemodialysis patients and observed no significant differences in anemia management between the 2 groups. However, they pointed out that aboriginal hemodialysis patients received increased erythropoietin yet were unable to achieve target hemoglobin levels. Interestingly, our study found that, despite similar rates of erythropoietin supplementation, Cree patients with CKD and diabetes were at greater risk of anemia. One potential explanation is that Cree patients have a decreased sensitivity to erythropoietins. Erythropoietin hyporesponsiveness in CKD is associated with older age, obesity, diabetes, and use of ACEI or ARB medications.[13] While Cree patients were younger than their nonaboriginal counterparts, the Cree were more likely to be prescribed ACEI or ARB medications, and nearly all Cree individuals in the population were obese or overweight (96.7%). In a previous cohort study, obesity was found to be associated with increased inflammatory markers,

**Table 4. Multivariate Cox hazard model:** *Association between anemia and aboriginal background (N = 81).*

| VARIABLE | HR | 95% CI | *P* VALUE |
|---|---|---|---|
| Cree (nonaboriginal is the referent) | 2.80 | 1.01–7.87 | .049 |
| BMI ≥ 35 kg/m$^2$ | 1.53 | 0.64–3.67 | .338 |
| ESA use | 1.15 | 0.36–3.61 | .815 |
| eGFR (< 15 mL/min/1.73 m$^2$) | 1.18 | 0.48–2.89 | .717 |
| Serum albumin (per 1 g/L) | 1.01 | 0.93–1.09 | .879 |
| Ferritin level < 200 µg/L | 1.00 | 0.99–1.01 | .194 |
| Iron saturation < 0.21% | 4.13 | 1.83–9.34 | .001 |

BMI—body mass index, eGFR—estimated glomerular filtration rate, ESA—erythropoiesis-stimulating agent, HR—hazard ratio.

such as C-reactive protein and interleukin-6.[14] In turn, the inflammatory state can increase resistance to erythropoietin, as noted in 64 hemodialysis patients by Biesenbach et al.[15] Our data seemed to imply that the Cree patients had higher rates of low-grade inflammation than their nonaboriginal counterparts did, as indicated by their lower serum albumin levels. Moreover, iron deficiency represents another possible cause of anemia. While the Cree group had a lower median ferritin level, after adjusting for serum ferritin and iron saturation, as well as obesity, erythropoietin supplementation, serum albumin levels, and stage-5 CKD, there was still a higher risk of anemia among the Cree. This suggests a need for more frequent patient monitoring and dose adjustments of erythropoiesis-stimulating agents in northern Cree patients with CKD.

Although anemia was not optimally managed, other modifiable risk factors were well managed. Quality-of-care indicators such as management of blood pressure, lipid levels, and calcium phosphate levels were similar between the Cree and nonaboriginal cohorts. Furthermore, prescription rates were similar for both groups, except for renoprotective medications and phosphate binders, which were prescribed more often in the Cree group.

## Limitations

The limitations of this study include its small sample size, limiting the power of the study, and its cross-sectional design, preventing a thorough examination of the evolution of the patients' conditions over time. Hemoglobin $A_{1c}$ levels were not available for 40% of the Cree individuals; this is mostly owing to patient noncompliance. Moreover, data on erythropoiesis-stimulating agent dosing would have helped clarify whether anemia was due to underdosing or to hyporesponsiveness. A prospective study is necessary to investigate the relationship between anemia and inflammation, diabetes, and obesity in aboriginal populations.

## Conclusion

This study compared quality-of-care indicators for Cree and nonaboriginal patients with CKD and diabetes and demonstrated that although overall management was similar in the 2 groups, management of anemia was suboptimal for Cree patients. To improve this, a direct interface between the laboratory and the NephroCare system in the Chisasibi and Chibougamau hospitals will be developed.

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**Competing interests**

None declared

**Contributors**

**Mr Patapas** contributed to the concept of the study, statistical analyses, interpretation of results, and writing the manuscript. **Ms Blanchard** contributed to the concept of the study, writing the ethics proposal, and preparation of the manuscript. **Dr Iqbal** contributed to the concept of the study, statistical analyses, interpretation of results, and writing and revising the manuscript. **Dr Vasilevsky** contributed to the concept of the study, provided the study population, and helped with preparation of the manuscript. **Dr Dannenbaum** contributed to interpretation of results and writing and revising the manuscript for important intellectual content.

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**References**

1. Hanley AJ, Harris SB, Gittelsohn J, Wolever TM, Saksvig B, Zinman B. Overweight among children and adolescents in a Native Canadian community: prevalence and associated factors. *Am J Clin Nutr* 2000;71(3):693-700.
2. Kuzmina E, Dannenbaum D. *Diabetes in Eeyou Istchee: 2005 annual update.* Chisasibi, QC: Cree Board of Health and Social Services of James Bay; 2005.
3. Tonelli M, Hemmelgarn B, Manns B, Pylypchuk G, Bohm C, Yeates K, et al. Death and renal transplantation among aboriginal people undergoing dialysis. *CMAJ* 2004;171(6):577-82.
4. Gao S, Manns BJ, Culleton BF, Tonelli M, Quan H, Crowshoe L, et al. Access to health care among status aboriginal people with chronic kidney disease. *CMAJ* 2008;179(10):1007-12.
5. Mei Z, Grummer-Strawn LM, Pietrobelli A, Goulding A, Goran MI, Dietz WH. Validity of body mass index compared with other body-composition screening indexes for the assessment of body fatness in children and adolescents. *Am J Clin Nutr* 2002;75(6):978-85.
6. Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, Roth D. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group. *Ann Intern Med* 1999;130(6):461-70.
7. Nelson RG, Greene T, Beck GJ, Van Lente F, Wang XL, Knowler WC. Estimating GFR by the MDRD and Cockroft-Gault equations in Pima Indians. *J Am Soc Nephrol* 2003;14:134a.
8. Levin A, Hemmelgarn B, Culleton B, Tobe S, McFarlane P, Ruzicka M, et al. Guidelines for the management of chronic kidney disease. *CMAJ* 2008;179(11):1154-62.
9. Zhao Y, Connors C, Wright J, Guthridge S, Bailie R. Estimating chronic disease prevalence among the remote aboriginal population of the Northern Territory using multiple data sources. *Aust N Z J Public Health* 2008;32(4):307-13.
10. Rowe BH, Voaklander DC, Wang D, Senthilselvan A, Klassen TP, Marrie TJ, et al. Asthma presentations by adults to emergency departments in Alberta, Canada: a large population-based study. *Chest* 2009;135(1):57-65. Epub 2008 Aug 8.
11. Glasgow NJ, Goodchild EA, Yates R, Ponsonby AL. Respiratory health in aboriginal and Torres Strait Islander children in the Australian Capital Territory. *J Paediatr Child Health* 2003;39(7):534-9.
12. Chou SH, Tonelli M, Bradley JS, Gourishankar S, Hemmelgarn BR. Quality of care among aboriginal hemodialysis patients. *Clin J Am Soc Nephrol* 2006;1(1):58-63. Epub 2005 Nov 9.
13. Rossert J, Gassmann-Mayer C, Frei D, McClellan W. Prevalence and predictors of epoetin hyporesponsiveness in chronic kidney disease patients. *Nephrol Dial Transplant* 2007;22(3):794-800. Epub 2007 Jan 8.
14. Ramkumar N, Cheung AK, Pappas LM, Roberts WL, Beddhu S. Association of obesity with inflammation in chronic kidney disease: a cross-sectional study. *J Ren Nutr* 2004;14(4):201-7.
15. Biesenbach G, Schmekal B, Eichbauer-Strum G, Janko O. Erythropoietin requirement in patients with type 2 diabetes mellitus on maintenance hemodialysis therapy. *Wien Klin Wochenschr* 2004;116(24):844-8.