Renal function and cardiovascular risk markers in a remote Australian Aboriginal community

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Abstract

Background. Australian Aborigines living in remote areas have exceedingly high rates of renal failure together with increased cardiovascular morbidity and mortality. To examine the basis of this association, we studied markers of renal function and cardiovascular (CV) risk in a coastal Aboriginal community in a remote area of the Northern Territory of Australia. End-stage renal disease (ESRD) incidence rates in that community are 15 times the national non-Aboriginal rate and CV mortality rates in the region are increased 5-fold.

Methods. A cross-sectional community survey was conducted. Markers of early renal disease examined included urine albumin/creatinine ratio (ACR), serum creatinine concentration and calculated glomerular filtration rate (GFR). CV risk markers included blood pressure as well as measures of glycaemia, diabetes and serum lipids.

Results. The study group included 237 people, 58% of the adult population of the community. The crude prevalence of microalbuminuria (urine ACR: 3.4–33.9 g/mol, 30–299 mg/g) was 31% and of overt albuminuria (urine ACR: ≥34 g/mol, ≥300 mg/g), 13%. The prevalence of overt albuminuria increased with age, but the prevalence of microalbuminuria was greatest in the 45–54 year age group. Microalbuminuria was associated with increasing body mass index, whereas overt albuminuria was associated with increasing glycated haemoglobin (HbA1c) and systolic blood pressure and a history of diabetes. The prevalence of elevated serum creatinine concentration (≥120 µmol/l) was 10%. GFR (calculated using the MDRD equation) was <60 ml/min/1.73 m² in 12% and 60–79 ml/min/1.73 m² in a further 36% of the study population. Although many people with albuminuria had well preserved GFRs, mean GFR was lower in people with higher levels of albuminuria.

Conclusions. The high prevalence of markers of renal disease in this community was consistent with their high rates of ESRD. The distribution of microalbuminuria suggested a ‘cohort effect’, representing a group who will progress to overt albuminuria. The powerful association of renal disease markers with CV risk factors confirms a strong link between renal and CV disease in the early, asymptomatic stages of each. Thus, pathologic albuminuria, in part, might be a manifestation of the metabolic/hemodynamic syndrome and both conditions might arise out of a common menu of risk factors. Hence, a single agenda of primary and secondary intervention may benefit both.

Keywords: albuminuria; Australian Aborigines; cardiovascular disease; cardiovascular markers; end-stage renal disease

Introduction

A relationship between end-stage renal disease (ESRD) and cardiovascular disease has been well documented. More recently, this association has been extended to groups with reduced glomerular filtration rate (GFR) [1,2] and albuminuria in the ‘microalbuminuria’ [3–6] and even ‘high-normal’ [7] ranges, although this has not been universally observed [8]. In one Australian Aboriginal (AA) community with a strikingly high rate of ESRD, an association between albuminuria and subsequent cardiovascular mortality was demonstrated [9]. The degree to which the high prevalence of markers of early renal disease and their association with cardiovascular risk factors can be generalized to other AA communities is unclear.

On a national level, a high incidence of treated ESRD among AA groups has been demonstrated.
from registry data. Rates differ between regions and communities, with a 10-fold or greater variation in ESRD incidence [10]. High but variable prevalence rates of albuminuria demonstrated in several Aboriginal communities are compatible with this excess of ESRD [11–13]. Rates of cardiovascular disease and cardiovascular death are also considerably higher among AA people. Observed cardiovascular death rates range from two to five times the national average [14] and are increased by >10-fold in young adults.

The AA community in which the association between early renal disease and cardiovascular mortality was studied previously had an ESRD incidence rate of 2700 per million and a 3–4-fold increase in cardiovascular deaths. There, albuminuria was shown to be inversely correlated with Cockcroft–Gault estimated creatinine clearance and directly correlated with body mass index (BMI), blood pressure, insulin and lipids on cross-sectional survey [11]. Albuminuria predicted not only all cases of ESRD [15], but also most cases of cardiovascular death on longitudinal study [9]. These findings suggested that, in that community, renal and cardiovascular diseases were intimately related, even in their early and asymptomatic stages.

Challenges exist, however, when assessing early renal disease among AA communities. The validity of formulae used to calculate creatinine clearance or GFR have not been tested in this racial group. Differences in body composition between Aboriginal and non-Aboriginal Australians have been identified [16], which might affect the validity of such estimates. Popular formulae used to predict GFR, such as the MDRD formula [17] and that of Cockcroft and Gault, are empirically derived; hence, one cannot readily control for potentially differing body composition between different racial groups, although the MDRD equation includes a correction factor for African Americans. Neither formula has been validated among Aboriginal Australians, in whom there may be a lower proportion of lean muscle mass for a given BMI [16].

To further explore the relationship between markers of renal disease and cardiovascular risk, we performed a cross-sectional survey in another remote Aboriginal community. This community was located in a remote coastal area of the Northern Territory (NT) of Australia, 600 km from Darwin (and a similar distance from the community previously reported [11,15]). This community was typical of remote Aboriginal communities, with high rates of poverty, household crowding and unemployment. In the 1996 Australian Census, in this community 82% of adult residents reported a weekly income <$A 200, only 6% were part of the labour force and an average of 7.8 people per occupied dwelling. Over the 10 year period 1991–2001, crude ESRD incidence rate in this community was 590 per million per year, in contrast to the Australia average of 70–90 per million per year over this time. Age at commencement of renal replacement therapy was also younger; the indirectly age-standardized ESRD rate was 15 times the non-indigenous rate. Cardiovascular mortality was also elevated, with an age-adjusted regional rate of 700 per million per year for AA males and 540 per million per year for females for the period 1986–95, compared with the NT non-Aboriginal rates of 300 and 170 per million per year for males and females, respectively [14].

Subjects and methods

Members of the community of 18 or more years of age were invited to take part in the project by local advertising, word of mouth and personal invitation from members of the study team. After consent was given, a questionnaire was administered (which included self-reported histories of cigarette smoking and diabetes), a brief physical examination was performed and blood and urine specimens were obtained. Resting blood pressure was measured twice at rest (5 min apart) by a single observer (S.M.) using a mercury sphygmomanometer and Korotkoff phases I and V for systolic (SBP) and diastolic blood pressures, respectively. A 14 × 28 cm bladder in a Velcro-fastened cuff (Spiedel & Keller, Jungingen, Germany) was used, except where the indicated range for arm circumference was exceeded or the width appeared to be less than two-thirds the upper arm, when a 17 × 34 cm bladder and a larger cuff was used. Weight was measured to the nearest 0.2 kg with an electronic scale and standing height was measured using a wall-mounted stadiometer. BMI was calculated by dividing the square of the height (m) into the weight (kg).

Blood and urine specimens were non-fasting and collected without regard for time of day. Serum was separated immediately after clotting, within 20 min in all cases. All specimens were then refrigerated until transfer to the pathology laboratory by air, within 24 h.

Serum was analysed for electrolytes, urea, creatinine, liver function tests, total and high-density lipoprotein (HDL) cholesterol, Lp(a) and triglycerides using a standard automated analyser (Hitachi 917 Autoanalyser, Boehringer-Mannheim, Rotkreuz, Germany).

The proportion of glycated haemoglobin (HbAlc) was used as the measure of glycaemia for all participants and was analysed on whole venous blood. Low-density lipoprotein (LDL) cholesterol concentrations were calculated using the Friedewald equation if serum triglyceride concentration was <4.5 mmol/l. Given the non-fasting nature of the serum specimens, triglyceride concentrations were not further analysed. Albumin and creatinine concentrations were analysed in the spot urine sample and an albumin/creatinine ratio (ACR) calculated. This was classified into ‘normal’ (ACR: <3.4 g/mol, <30 mg/g), ‘microalbuminuria’ (ACR: 3.4–33.9 g/mol, 30–299 mg/g) and ‘overt albuminuria’ (ACR: ≥34 g/mol, ≥300 mg/g) [18].

Analyses were carried out using standard statistical software (Intercooled Stata version 7.0, Stata Corporation, College Station, TX, USA). Serum creatinine was analysed using an inverse transformation and urine albumin and creatinine concentration were log-transformed. Where urine ACR was analysed as a continuous variable, a shifted log transformation [ln(ACR−0.217)] g/mol] was used to...
correct the right skew. Means and confidence intervals (CIs) for all transformed variables are presented after back-transformation to their original scale. For multivariate analyses, ACR was analysed as categories of normal, microalbuminuria and overt albuminuria. Multinomial logistic regression was used to analyse the relationship of the albuminuria categories. This allowed derivation of separate odds ratios (ORs) for micro- and overt albuminuria, compared with the ‘normal’ ACR group. Goodness of fit of continuous predictor variables in logistic models was examined with the Hosmer–Lemeshow test; predictors were analysed as categories where this test suggested a poor fit as a continuous variable.

GFR was estimated from serum creatinine, urea and albumin concentrations using the MDRD Equation 7 [17]. A second estimate was made using the Cockcroft–Gault equation. This was corrected for body surface area (BSA) to allow comparison with the MDRD calculation. Other formulae were not examined here as the Cockcroft–Gault and MDRD formulae are those commonly in clinical use. Agreement between the MDRD and Cockcroft–Gault estimates was then examined using the ‘limits of agreement’ method of Bland and Altman.

The study protocol was approved by the Joint Institutional Ethics Committee of Menzies School of Health Research and Royal Darwin Hospital.

Results

A total of 237 people (133 female) were recruited, ~58% of eligible people in the community, as estimated in the 1996 Australian Census of Population and Housing. Smoking, diabetes, hypertension, obesity and especially albuminuria were all common (Table 1). Significantly more males than females reported current cigarette smoking, but more females were both under- and over-weight (BMI: <20 and ≥25 kg/m², respectively).

The prevalence of micro- and overt albuminuria rose with age (Figure 1). Within this increase in albuminuria, however, there were differing trends with age for microalbuminuria (which was most common in the 45–54 year age group) compared with overt albuminuria (which steadily increased with age). The distribution of ACR categories did not vary significantly with gender ($\chi^2_{2df} = 3.1, P = 0.2$), but when analysed as a continuous variable, mean ACR was significantly greater among females (Table 1). Neither urine albumin nor creatinine concentrations differed significantly with gender, although there was a trend towards higher urine albumin concentrations and lower urine creatinine concentrations among females (Table 1).

Table 1. Characteristics of study population

<table>
<thead>
<tr>
<th></th>
<th>Females ($n = 133$)</th>
<th>Males ($n = 104$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age [median (IQR), years]</td>
<td>35.8 (28.8–46.0)</td>
<td>38.5 (28.9–48.7)</td>
</tr>
<tr>
<td>Tobacco smokersa</td>
<td>77/129 (60%)</td>
<td>84/104 (81%)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>33/133 (25%)</td>
<td>26/104 (25%)</td>
</tr>
<tr>
<td>BP ≥140/90 mmHg</td>
<td>18/125 (14%)</td>
<td>16/102 (16%)</td>
</tr>
<tr>
<td>Obesity (BMI ≥30 kg/m²)</td>
<td>12/125 (10%)</td>
<td>10/103 (10%)</td>
</tr>
<tr>
<td>Overweight (BMI 25–29 kg/m²)b</td>
<td>14/125 (11%)</td>
<td>21/103 (20%)</td>
</tr>
<tr>
<td>Underweight (BMI &lt; 20 kg/m²)c</td>
<td>35/125 (28%)</td>
<td>23/103 (22%)</td>
</tr>
<tr>
<td>HbA1c [median (IQR), %]</td>
<td>5.8 (5.3–6.6)</td>
<td>5.8 (5.4–6.4)</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/l)</td>
<td>2.5 (2.3–2.6)</td>
<td>2.8 (2.6–3.0)</td>
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<tr>
<td>HDL cholesterol (mmol/l)d</td>
<td>0.82 (0.78–0.89)</td>
<td>0.83 (0.78–0.89)</td>
</tr>
<tr>
<td>Overt albuminuria (ACR ≥34g/mol)</td>
<td>19/120 (16%)</td>
<td>10/98 (10%)</td>
</tr>
<tr>
<td>Microalbuminuria (ACR 3.4–33.9g/mol)</td>
<td>40/120 (33%)</td>
<td>27/98 (28%)</td>
</tr>
<tr>
<td>Urine albumin (g/l)d</td>
<td>0.33 (0.31–0.35)</td>
<td>0.40 (0.38–0.42)</td>
</tr>
<tr>
<td>Urine creatinine (mmol/l)d</td>
<td>6.1 (5.2–7.1)</td>
<td>7.4 (6.2–8.8)</td>
</tr>
<tr>
<td>ACR (g/mol, median (IQR))</td>
<td>3.2 (0.9–14.9)</td>
<td>1.3 (0.5–8)</td>
</tr>
<tr>
<td>Serum creatinine (µmol/l)e</td>
<td>83.4 (81.1–85.8)</td>
<td>107.2 (103.7–111.0)</td>
</tr>
<tr>
<td>Serum creatinine ≥120 µmol/l</td>
<td>6/130 (5%)</td>
<td>17/96 (18%)</td>
</tr>
<tr>
<td>MDRD GFR (ml/min/1.73 m²)</td>
<td>78.8 (75.7–82.0)</td>
<td>79.3 (76.1–82.6)</td>
</tr>
<tr>
<td>Cockcroft–Gault GFR (ml/min/1.73 m²)e</td>
<td>83.2 (79.1–87.2)</td>
<td>76.2 (72.3–80.1)</td>
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</table>

Although 237 people were recruited, some investigations were not performed on some people, hence, the numbers do not always add to 237. For ACR the ‘geometric’ mean is calculated using a shifted log transformation. The Cockcroft–Gault GFR estimate was normalized to 1.73 m² BSA.

aP < 0.001, bP < 0.001, cP < 0.05, dgeometric mean (95% CI), eP = 0.02 and hharmonic mean (95% CI).
In addition to age and gender, a number of other cardiovascular risk factors were significantly associated with albuminuria. A history of diabetes was associated with albuminuria, independently of age and gender (Table 2). Furthermore, each quartile of HbA1c was associated with a progressively increasing proportion of people with an abnormal albuminuria (Figure 2). This was independent of age category and gender; for microalbuminuria the OR was 1.4 (1.0–2.0) per quartile of HbA1c \( P = 0.04 \) and for overt albuminuria the OR was 2.2 (1.3–3.8) per HbA1c quartile \( P = 0.002 \). Higher blood pressure and higher BMI were similarly associated with increased rates of albuminuria over a continuum (Table 2). In contrast, current tobacco smoking was associated with lower rates of pathologic albuminuria. Neither total nor LDL- or HDL-cholesterol concentrations were significantly associated with ACR (Table 2). In contrast, current tobacco smoking was associated with lower rates of pathologic albuminuria. Neither total nor LDL- or HDL-cholesterol concentrations were significantly associated with ACR category after adjustment for age and gender.

Diabetes, BMI and SBP were combined with age and gender in a multivariate multinomial logistic model. Of the predictors of microalbuminuria, BMI \[ \text{OR} \; 1.6 \; (1.1–2.3) \; \text{per} \; 5 \; \text{kg/m}^2; \; P = 0.03 \], age and gender remained significant in this model, whereas for overt albuminuria, diabetes \[ \text{OR} \; 3.5 \; (1.1–11.1); \; P = 0.03 \] and SBP \[ \text{OR} \; 1.4 \; (1.0–1.8) \; \text{per} \; 10 \; \text{mmHg} \]

<table>
<thead>
<tr>
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<th>Microalbuminuria</th>
<th>Overt albuminuria</th>
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<tbody>
<tr>
<td>Diabetes</td>
<td>2.3 (1.0–5.3), ( P = 0.05 )</td>
<td>5.6 (1.0–15), ( P = 0.001 )</td>
</tr>
<tr>
<td>BMI (per 5 kg/m(^2))</td>
<td>1.6 (1.1–2.3), ( P = 0.007 )</td>
<td>2.0 (1.2–3.4), ( P = 0.01 )</td>
</tr>
<tr>
<td>SBP (per 10 mmHg)</td>
<td>1.1 (0.87–1.3), ( P = 0.5 )</td>
<td>1.5 (1.1–1.9), ( P = 0.003 )</td>
</tr>
<tr>
<td>HbA1c (per quartile)</td>
<td>1.4 (1.0–2.0), ( P = 0.04 )</td>
<td>2.2 (1.3–3.8), ( P = 0.002 )</td>
</tr>
<tr>
<td>Tobacco smoking</td>
<td>0.50 (0.24–1.1), ( P = 0.06 )</td>
<td>0.33 (0.12–0.89), ( P = 0.03 )</td>
</tr>
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ORs are shown with 95% CI and are adjusted for age category and gender.

Harmonic mean serum creatinine concentration was significantly higher among males (Table 1). There was a significant negative correlation between age and serum creatinine \( r = -0.31, \; P < 0.001 \). Height \( r = 0.44, \; P < 0.001 \) and weight \( r = 0.18, \; P = 0.007 \) were also correlated with serum creatinine. The MDRD GFR and the creatinine clearance from the Cockcroft–Gault calculations were significantly correlated (Pearson \( r = 0.8, \; P < 0.001 \)). The values from each equation were similar on average [mean difference 0.62 (−1.10, 2.34) ml/min/1.73 m\(^2\)]. Although the MDRD GFR did not differ with gender, the mean Cockcroft–Gault GFR was lower among males (Table 1). The Bland–Altman limits of agreement were broad (−24.6 to 25.9 ml/min/1.73 m\(^2\)) and were similar in males and females. This suggests substantial variation when the results from each formula are compared in an individual. The MDRD formula was used for subsequent comparisons, except where otherwise stated.

There was no significant difference in calculated GFR with gender, but there was significant variation with age, with a steadily decreasing GFR with age (Figure 3). After adjustment for age, calculated GFR was significantly lower in those with diabetes \[ \text{in whom the mean GFR was 6.4 (2.0–10.9) ml/min/1.73 m}^2 \text{ lower; } P = 0.005 \] and inversely with SBP \[ \text{GFR decreased } 1.2 \; (0.1–2.2) \; \text{ml/min/1.73 m}^2 \; \text{per} \; 10 \; \text{mmHg increase in SBP; } P = 0.03 \]. Calculated GFR was not associated with smoking or BMI, but did vary significantly across the quartiles of HbA1c \[ \text{(ANOVA; } P < 0.0001) \]. When adjusted for age and gender, however, the variation of GFR with HbA1c quartile was not significant \( P = 0.5 \). Calculated GFR fell with increasing serum cholesterol concentration and decreasing serum HDL cholesterol concentration, but was not significantly associated with calculated LDL cholesterol concentration. After adjustment for age and gender, calculated GFR remained significantly associated with HDL cholesterol concentration \[ \text{[mean GFR increased } 4.2 \; (0.0–8.5) \; \text{ml/min/1.73 m}^2 \; \text{per doubling of HDL cholesterol concentration; } P = 0.05 \].

The prevalence of abnormal GFR was sharply higher in those with micro- and overt albuminuria (Figure 4), but even in the group with overt albuminuria, 31% had a ‘normal’ calculated GFR \( \geq 80 \; \text{ml/min/1.73 m}^2 \) and a further 24% had a GFR \( 60–79 \; \text{ml/min/1.73 m}^2 \). Using the same threshold values with creatinine clearance derived from the Cockcroft–Gault equation, the prevalence of ‘normal’ creatinine clearance \( \geq 80 \; \text{ml/min/1.73 m}^2 \) in the overt albuminuria group was 33%, with a further 26% of the overt albuminuria group in the 60–79 min/min/1.73 m\(^2\) range.
Finally, in the group with serum creatinine ≤120 μmol/l (1.4 mg/dl), chosen as a clinically relevant indicator of potentially 'normal' renal function, 31% had microalbuminuria and 11% had overt albuminuria. If a calculated GFR value of ≥80 ml/min/1.73 m² is taken to indicate 'normal' renal function, then 26% of this group had microalbuminuria and 8% overt albuminuria.

Discussion

This study showed high rates of pathologic albuminuria among members of a remote AA community with a high incidence of treated ESRD. Higher levels of albuminuria were associated with lower levels of calculated GFR. Albuminuria was also associated with increasing BMI, increasing blood pressure,
increasing levels of HbA1c and with a history of diabetes, while calculated GFR was related to HDL cholesterol concentration and inversely related to HbA1c level and increasing SBP (but not to BMI). These relationships applied over a continuum of GFR and over a continuum of blood pressure, BMI and HbA1c, not just over ‘hypertensive’, ‘obese’ and ‘diabetic’ ranges, and suggest a role for renal markers as indices of cardiovascular risk, although a cross-sectional study cannot answer the question of whether renal impairment is itself a causal factor in cardiovascular disease.

These observations confirm and extend the associations of albuminuria with renal function and cardiovascular risk reported in one other AA community [11] and support the predictive value of albuminuria for CV death [9]. It appears likely, therefore, that these associations can be generalized to other AA communities with a high prevalence of renal disease and possibly to other indigenous groups. The degree of albuminuria and reduced GFR represent a large asymptomatic ‘base’ to a pyramid of renal disease, the tip of which is manifest as ESRD. Among the large proportion of the population with asymptomatic renal disease, there were strong correlations between renal and most cardiovascular risk factors. Only one urine ACR estimation was obtained in this study. Since there is substantial biological variability in albumin excretion [19], this will have led to non-differential misclassification of albuminuria category. This will, however, obscure associations rather than lead to bias.

The prevalence of albuminuria was strongly age-related, although the patterns for micro- and overt albuminuria differed. This probably represents a combination of cohort effects. Firstly, older people have experienced more nephropathic risk factors than younger people (e.g. low birth weight, infections) and, secondly, the progression of albuminuria in individuals over time, as has been shown in another Aboriginal community [15]. The lower GFRs in people with micro- and overt albuminuria are consistent with a role of albuminuria as a predictor of declining renal function. However, the high prevalence of micro- and overt albuminuria in the group with ‘normal’ creatinine and GFR emphasizes the utility of ACR as a marker of risk of renal disease when renal excretory function is still well preserved.

The ‘GFR’ values calculated from the Cockcroft–Gault and MDRD equations were ‘significantly’ correlated. There was, however, poor agreement between the two methods in individuals when quantified in the Bland–Altman approach. Although there was not a trend of either method to systematically under- or over-estimate GFR compared with the other, the weighting of gender led to difference comparisons between formulae. GFR estimates from both formulae had similar relationships to ACR and to cardiovascular risk factors. However, the applicability of these formulae to Aboriginal people still needs to be evaluated. The lower muscle mass for a given BMI in Aboriginal Australians [16], however, suggests that creatinine-based formulae of any sort may underestimate the degree of renal insufficiency in these groups.

The distribution of renal markers can now be compared with studies based in non-indigenous populations. The recent Australian study ‘AusDiab’ measured spot urine ACR on a stratified sample representative of the non-indigenous Australian population. The prevalence of pathological albuminuria, using the same definitions as this study, was much lower with microalbuminuria detected in 6.1% and overt albuminuria in 0.7% [20]. This study also reported a median GFR (from the Cockcroft–Gault formula standardized to BSA) of 89 ml/min/1.73 m², similar to the mean value observed here. In the AusDiab study, however, a Cockcroft–Gault GFR value <60 ml/min/1.73 m² was seen in 3.4% in contrast to the present study where 16% had a Cockcroft–Gault GFR value in this range. This is primarily attributable to differences in creatinine: the AusDiab prevalence of serum creatinine >120 µmol/l was 1.1% [21]; in contrast, 10% of the cohort studied here had serum creatinine ≥120 µmol/l. Thus, the prevalence rates of both albuminuria and higher creatinine are increased compared with available Australian non-Aboriginal data. This suggests that the high rates of ESRD observed among this AA group arise from a widespread prevalence of milder degrees of renal impairment.

The rates of diabetes, smoking, hypertension and overweight described here are similar to those in the previous AA community studied with high ESRD rates [11]. The HbA1c levels were consistent with the high prevalence of diabetes. Rates of diabetes and smoking were considerably higher than those in the contemporary non-Aboriginal Australian population; although rates of overweight and hypertension were lower [21]. The associations of BMI, diabetes, HbA1c and blood pressure with albuminuria and of diabetes, blood pressure and lower HDL cholesterol with lower GFRs, underscore the associations between renal disease and cardiovascular risk. There is a quantitative dimension to the association between renal and cardiovascular risk also; diabetes and higher SBP, representing later stages of metabolic and vascular disease, were associated with overt albuminuria, whereas increasing BMI can be seen as an earlier marker of metabolic changes and was associated with an earlier stage of renal disease. This is compatible with the predictive value of albuminuria for cardiovascular death demonstrated in another remote Aboriginal community [9,15], in non-indigenous communities elsewhere [22] and the high prevalence of diabetes and cardiovascular disease in both the indigenous and non-indigenous ESRD populations.

The lack of a positive relationship between smoking and renal disease is contrary to reports on smoking and renal disease in other populations, but compatible with findings in the other community
studied [11]. Perhaps, in a cash-poor environment, cigarette smoking marks relative affluence in these populations, which itself partially protects against renal and other diseases.

The links between the indices of impaired renal function and diabetes and hypertension shown here emphasize that in this environment, chronic non-communicable ‘lifestyle’ diseases do not occur in isolation, but cluster together with the attendant increased mortality risks. The widely reported association of cardiovascular disease with renal impairment reflects an intimate relationship in early and asymptomatic disease, related to a common menu of risk factors. Whether there is also a specific atherosclerotic effect of mild renal impairment (perhaps by alterations in LDL oxidation [23,24] or other means) in addition to the associated risk factors is not clear. The close associations with cardiovascular risk factors have important implications for screening and treatment programs, which need to address multiple health issues simultaneously.

References


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