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Excess prevalence of non diabetic renal disease in native American children in Manitoba

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Abstract. We undertook a 1-year prospective point prevalence study to test the hypothesis that there is an excess of non-diabetic renal disease in native American children; 29.6% (73/247) of the population attending the only regional pediatric nephrology clinic in 1993 were native compared with 8.2% of the Manitoba population in this age group (odds ratio = 4.4, $P < 0.001$). Patients were classified as low risk (normal renal function, no deterioration expected), high risk (normal renal function, deterioration probable), or established chronic renal failure (creatinine clearance chronically low or post renal transplant). Patients were further classified as suffering from congenital renal anomalies, genetic or metabolic disease, or acquired renal disease. Odds ratios were calculated based on data from the Aboriginal Peoples' Population Survey and Statistics Canada census data. The odds ratios for low-risk renal disease, high-risk renal disease, and chronic renal failure were 3.8, 5.6, and 6.3, respectively ($P < 0.001$ in all categories). The odds ratios for congenital, genetic, or acquired disease were 4.5 ($P < 0.001$), 0.9 (P = ns), and 6.1 ($P < 0.001$), respectively. Native American children in Manitoba demonstrate increased prevalence of serious congenital and acquired renal disease. These children are also more likely to live in medically underserviced communities, long distances from tertiary care centers. This study emphasizes the importance of considering factors other than diabetes mellitus when considering the problem of renal disease in native Americans.

Key words: Native American – Kidney disease – Epidemiology

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Introduction

Rates of renal disease in the native American population are high and rates of end-stage renal disease (ESRD) are rising [1–5]. The prevalence of renal disease in general and ESRD in adult native Americans has been documented to be higher than national averages in several studies. Most ESRD in native American adults has been attributed to excessive diabetic nephropathy in the adult population [6–8]. An increased rate of glomerulonephritis has also been reported in adults [9]. The prevalence and type of renal disease in native American children has never been studied. It has been our experience that native American children make up a disproportionate number of patients in the Pediatric Nephrology Clinic at the Winnipeg Children's Hospital. We therefore performed a 1-year prospective point prevalence study to test the hypothesis that there is an excess of non-diabetic renal disease in native American children referred to the Winnipeg Children's Hospital.

Patients and methods

A prospective point prevalence study of all patients aged 16 years or less attending the pediatric nephrology clinic of the Winnipeg Children's Hospital, Winnipeg, Manitoba, Canada, between January 1 1993 and December 31 1993 was carried out. This is the only clinic of its type servicing Manitoba, Northwestern Ontario, Saskatchewan, and the Central Arctic. Native children were identified on the basis of treaty numbers, geographic locations, physical characteristics, and family self-reporting consistent with the Aboriginal People's Survey [10]. The etiology of renal disease was determined by a pediatric nephrologist and the disease categorized according to severity as low risk, defined as normal renal function with minimal risk of progression to chronic renal failure (e.g., persistent microscopic hematuria, minimal change disease), high risk, defined by normal creatinine clearance by measurement or calculation, but at high risk of progression (e.g., IgA nephropathy with persistent proteinuria), or chronic renal failure, defined as abnormal creatinine clearance, patients on dialysis, or renal transplant recipients. Each diagnosis was classified as congenital (e.g., renal dysplasia), inherited/metabolic (e.g., oxalosis, polycystic kidney disease), or acquired (e.g., IgA nephropathy). Odds ratios were calculated based on data from the Aboriginal Peoples' Population Survey (1991)

Table 1. Renal diseases in native and non-native American children in Manitoba

Disease	Native	Non-native	Odds ratio
Congenital			
– hypoplasia/dysplasia	11	25	
– vesicoureteric reflux	3	9	
– obstructive uropathy	4	8	
Total	18	42	6.5*
Genetic/metabolic			
– PKD	1	9	
– RTA	1	1	
– oxalosis	–	1	
– cystinosis	–	3	
– Alport's	–	12	
– others[a]	1	13	
Total	3	39	0.9
Acquired			
– nephrotic syndrome	5	16	
– IgA nephropathy	16	16	
– MPGN	8	6	
– post-streptococcal GN	5	2	
– HUS	–	7	
– UTIs	5	12	
– others[b]	12	34	
Total	51	93	6.1**
Unknown	1		
Total	73	174	

PKD, Polycystic kidney disease; RTA, renal tubular acidosis; MPGN, membrano-proliferative glomerulonephritis; HUS, hemolytic uremic syndrome; UTIs, urinary tract infections

* $P < 0.001$

[a] Riley-Day syndrome, tuberous sclerosis, nephronophthisis, Bartter's syndrome, Vater association, hemangiomas, benign familial hematuria

[b] Hypertension, Henoch-Schonlein purpura, perinatal ischemia, systemic lupus erythematosus, orthostatic proteinuria, Goodpasture's syndrome, renal artery stenosis, renal vein thrombosis, sepsis, rapidly progressive GN, post-chemotherapy

Table 2. Categories of disease severity in native American and non-native American children with renal disease

Class	Low risk	High risk	CRF
Native American (*n*)	34	19	20
Other (*n*)	100	38	36
Odds ratio	3.8	5.6	6.3
P	<0.001	<0.001	<0.001

CRF, Chronic renal failure

and Statistics Canada census data [10]. Statistical analysis was performed by comparing the distribution within any diagnostic or severity category with the number of native and non-native children in Manitoba by chi-squared test with the Yate's correction using the Instat software package (Graphpad, San Diego, Calif., USA).

Results

A total of 247 patients were seen during the study period and 73 of these were native American (29%). From the Aboriginal Peoples' Survey, native Americans aged 16 years or less make up only 8.2% of the total population of the study area. The proportion of native American children was significantly more than expected (odd ratio = 4.4, $P < 0.001$). Three native American children had type II diabetes mellitus presenting in adolescence and 3 non-native patients had type I diabetes mellitus. Diabetes mellitus was not the reason for presentation in any case. One diabetic native American presented with hypertension while 2 presented with IgA nephropathy. One non-native American presented with proteinuria with a normal biopsy, 1 presented with minimal change disease, and the third presented with posterior urethral valves.

Native American children demonstrated an excess of both congenital and acquired renal disease, but no excess of single gene inherited disorders (Table 1). This excess was seen in all categories of disease severity, with a trend towards a higher proportion of native American children in the more severe disease categories (Table 2).

Discussion

Native American children are over-represented in the pediatric renal clinic at the Winnipeg Children's Hospital. This population is composed of a diverse group of tribes, but mainly Cree, Saulteaux, and Dakota. No single disease, but rather a combination of both congenital and acquired renal problems, forms the basis for this over-representation. A high proportion of native American patients reside on reserves that may be as far as 2,000 km from Winnipeg rather than in the metropolitan area, making a geographic referral bias unlikely. Referral biases based on access to other levels of medical care, such as community pediatricians who might either adequately manage mild disease or limitations to access to such physicians that might increase referral for one group of people, would seem unlikely. Either of the above situations might influence the proportion of minor disease referred. Our clinic is, however, the only center assessing and treating ESRD in children of all ethnic and socioeconomic groups in this region; if our results are based on such referral biases, the excess of ESRD in native American children would not be observed.

Genetic factors may contribute to the observed increases in renal disease prevalence. Previous studies from this institution have reported an excessive and unique profile of recessively inherited metabolic disease in native Americans in this region [11]. Other studies have implicated genetic factors for renal disease of unknown etiology in natives [12]; however, specific genetic mechanisms have never been confirmed [13]. If genetic factors are relevant, they would seem to be expressed as increased susceptibility to renal injury or malformation, as prevalence of primary single gene mutation renal disorders was not increased.

High rates of child poverty and associated low standards of living may lead to exposure to, or deficiency of, environmental factors that are relevant to renal disease [12]. The native population of Manitoba represent a group with a very high rate of child poverty [10]. Geography and lifestyle may increase risk of exposure to potential nephrotoxins such as organic mercury. In Quebec, organic mercury levels have increased secondary to flooding from hydroelectric projects [14]. Similar projects with similar

environmental problems exist in our region. Quotas have been placed throughout the region on the number of fish safe to consume before the risk of mercury poisoning becomes unacceptable. This introduces the risk of intoxication if the recommendation is ignored and deficiency of a food that has been dietary staple of these people for thousands of years if it is followed. Several communities still use extracts from many plants for medicinal purposes and one could speculate that perhaps there is a form of nephrotoxin present in some of those extracts [12].

ESRD therapy is one of the most expensive burdens within health care in developed countries. The humanitarian burden of chronic ill health is one that can be ill afforded by economically struggling native American communities. Our results identify native American children within this region as a group at high risk of renal disease and mandate further community-based studies to explore the etiology of this unique and significant risk.

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