Resurgence of membranous nephropathy in African Americans in inner city Chicago

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Abstract

Background. It is well established that the incidence of focal segmental glomerular sclerosis (FSGS) increased from 1970–1990 to become the leading primary glomerular disease in patients of African descent.

Methods. To determine whether this trend has continued in the past years in Chicago, adult, native kidney biopsies from January 2001 to December 2011 at our hospital were reviewed and collected relevant clinical information in patients with a primary glomerular disease including FSGS, membranous nephropathy (MN), minimal change disease (MCD), membranoproliferative glomerulonephritis (MPGN), and IgA nephropathy (IgAN).

Results. In the 204 patients analyzed, MN was the most prevalent (32.7%), followed by FSGS (29.7%), IgAN (15.8%), MCD (14.4%), and MPGN (4.5%). Patients with MN had the highest proteinuria (7.9 gms/d) and were significantly older, more edematous, hypoalbuminemic, and hypercholesterolemic than those with FSGS. In both African Americans and Hispanics, MN was the most prevalent primary glomerular lesion at 39.2% and 34% respectively.

Conclusions. Comparable in size to prior cohorts of African Americans and Hispanics, our report demonstrates a reversal in the incidence of FSGS and MN in both ethnic groups where MN is now more prevalent. To our knowledge, this is the first demonstration of a reverse in the upward trend of the prevalence of FSGS in African Americans.

Keywords: African American; focal segmental glomerulosclerosis; Hispanic; membranous nephropathy

Introduction

For over two decades, it has been widely accepted that focal segmental glomerular sclerosis (FSGS) was the most common cause of primary proteinuric renal disease in African Americans in Chicago [1] and in other areas of the USA [2–10]. Subsequently, researchers in other countries also noted a similar trend in the cause of renal disease in patients of African descent [11–14]. In most of these series, the incidence of FSGS by far surpassed that of membranous nephropathy (MN). In several reports, it was also suggested that the prevalence of FSGS in patients of African descent was increasing over the 20 years spanning the 1970s to 1990s, whereas the changes in other ethnic groups were less clear [5–11, 14]. The reason for the widespread increased incidence of FSGS in black individuals remains unknown but may reside in improved diagnostic testing including proteinuria screening or an expanded penetrance of recently described genetic risk factors [15, 16].

In recent years, we have had the impression that at our inner city hospital, MN was becoming more frequent in our African American patients and FSGS less so. To confirm this impression and to determine whether the ethnic predominance of FSGS originally noted at our institution in 1986 no longer applies, we reviewed the records of patients who underwent a renal biopsy between 1 January 2001 and 31 December 2011 at our institution.

Materials and methods

We retrospectively reviewed all adult, native kidney biopsies performed between 1 January 2001 and 31 December 2011 at the Stroger Hospital of Cook County. Data were collected on patients diagnosed with a primary glomerular disease including FSGS, MN, minimal change disease (MCD), membranoproliferative glomerulonephritis (MPGN) and IgA nephropathy (IgAN). Patients with secondary renal diseases were excluded including diabetic nephropathy, hypertensive nephrosclerosis, lupus nephritis, malignancy-associated glomerular disease, HIV-associated nephropathy, infection-related (including hepatitis B and C related as determined by the attending nephrologist) and deposition disorders including amyloidosis. We collected data for race, gender, blood pressure, presence of hematuria or pyuria, BUN, serum creatinine and albumin, total cholesterol and proteinuria. We also analyzed the degree of edema as noted by a nephrologist at the time a biopsy was scheduled. The results are
reported as mean ± SD. Statistical analysis was done using the unpaired Student’s t-test for continuous variables with P < 0.05 considered significant. Fisher’s exact test was performed on categorical data with P < 0.05 considered significant.

Results

Of the 738 renal biopsies performed, 204 cases of primary glomerular disease were diagnosed at our institution between 1 January 2001 and 31 December 2011. Of the 204 biopsies identified, 102 were from African Americans, 75 from Hispanics, 6 from Caucasians, and 21 from other ethnicities. Overall, 60% were male and the average age was 42.6 years (range 16–76 years) (Table 1). In our cohort, MN was the most frequent lesion (32.7%), followed by FSGS (29.7%), IgAN (15.8%), MCD (14.4%) and MPGN (4.5%) (Table 2). Of the 60 cases of FSGS, 48 (80%) had FSGS not otherwise specified (NOS), 6 (10%) had the FSGS tip variant and 6 (10%) had the collapsing FSGS variant. Among African American patients, MN was the most frequent lesion (39.2%), followed by FSGS (33.3%), and MCD (18.6%). There were only three cases of IgAN, four cases of MPGN and two cases of fibrillary glomerulonephropathy. Among the African Americans, FSGS NOS was the most common (28, 82%), followed by the collapsing FSGS variant (5, 14.7%), and one case of FSGS tip variant (2%). Between 2001 and 2005, 24 cases of FSGS were diagnosed; 20 were FSGS NOS, 3 (12.5%) were the collapsing variant and the 1 case of FSGS tip occurred in 2005. From 2006 to 2011, there were 10 cases of FSGS; 8 were FSGS NOS and 2 (20%) were the collapsing variant. In the Hispanic group, MN was also the most frequent lesion (34%), followed by FSGS (25.3%) and IgAN (22.7%). There were six cases of MCD, four cases of MPGN, one case each of thin basement membrane disease, fibronec tin nephropathy, and lipoprotein nephropathy, and two cases of Fabry disease (Table 2). Among the 19 cases of FSGS in Hispanics, 14 (74%) were FSGS NOS, 4 (21%) were the tip variant and only 1 case was the collapsing FSGS variant. In Caucasians, there were two cases of IgAN and one each of MN, FSGS, MPGN, and thin basement membrane disease, but none of MCD.

In the entire cohort, patients with MN were significantly older than patients with either FSGS or IgAN (Table 3). They also presented with significantly more edema than in either FSGS or IgAN and with significantly higher systolic BP’s than MCD. Proteinuria was greatest in MN (7.9 g/day) and was nearly significantly higher than FSGS (P = 0.065), but reached significance only in comparison to IgAN. Nonetheless, the nephrotic syndrome associated with MN resulted in more significantly prominent hypoalbuminemia and hypercholesterolemia than FSGS and IgAN. There was a wide range of proteinuria in the MN group (190 mg/day to 20.7 g/day); 29 (44%) were severely nephrotic over 8 g/day, 23 (35%) had moderate proteinuria between 4 and 8 g/day, and 14 (21%) were mildly proteinuric (<4 g/day) [17, 18]. Among the 40 African Americans with MN, severe proteinuria was seen in 15 (37.5%), moderate in 15 (37.5%) and mild in 10 (25%). Among the 24 Hispanics with MN, severe proteinuria was seen in 13 (54.1%), moderate in 7 (29.1%) and mild in 4 (16.67%). Histologically, 60.5% (40/66) of patients had Churg stage III or IV and the remaining 39.5% (26/66) had Churg stage I or II. Proteinuria was significantly lowest in IgAN (3.46 g/day). The patients with FSGS had the highest serum creatinine (212 µmol/L [2.4 mg/dL]) which was significantly higher than either MN or MCD. Serum albumin was significantly lowest in MCD and highest in IgAN. The serum albumin was significantly higher in FSGS compared with either MN or MCD. Total serum cholesterol was greatest in MCD (8.87 mmol/L [342.47 mg/dL], followed by FSGS and IgA (Table 3). And, clinic-based systolic and diastolic blood pressures prior to renal biopsy were comparable, except in MCD which had a significantly lower SBP than either MN or FSGS.

Of the three variants of FSGS noted in the entire cohort, the tip variant had a significantly lower BUN [6.84 ± 2.33 versus 10.19 ± 6.15 mmol/L (19.17 ± 6.49 versus 28.54 ± 17.24 mg/dL); P < 0.05] and creatinine [73.37 ± 19.45 versus 199.78 ± 166.19 µmol/L (0.83 ± 0.22 versus 2.26 ± 1.88 mg/dL); P < 0.01], and higher total cholesterol [9.25 ± 2.21 versus 6.15 ± 1.88 mmol/L (357 ± 85.49 versus 237.39 ± 72.44 mg/dL); P < 0.05] than FSGS NOS. The mean protein-to-creatinine ratio was similar in the NOS and tip variants (5.55 and 4.94 g/day, respectively). Although the collapsing variant had a higher mean creatinine [455.26 versus 199.78 µmol/L (5.15 versus 2.26 mg/dL)] and mean protein-to-creatinine ratio (10.76 versus 5.55 g/day) compared with the NOS variant, they were not significantly different. Only serum albumin was found to be significantly lower in the collapsing variant compared with FSGS NOS [18.6 ± 7.0 versus 32.8 ± 9.3 g/L (1.86 ± 0.7 versus 3.28 ± 0.93 g/dL); P < 0.01]. There was no significant difference between the tip and collapsing variants within the entire cohort.

Clinical characteristics of each of the primary glomerular diseases in African Americans are shown in Table 4. Patients with FSGS had a mean serum creatinine significantly higher than either MN or MCD. Other clinical parameters did not differ among African Americans with different diagnoses. Among the Hispanic patients (Table 5), serum creatinine did not differ significantly between diagnoses; however, those with MN had significantly more proteinuria, hypoalbuminemia, hypercholesterolemia and lower extremity edema than either FSGS or IgAN.

Discussion

In African Americans, the incidence of FSGS as a cause of the idiopathic nephrotic syndrome has been increasing

| Table 1. Baseline characteristics of patients with primary glomerular disease in the entire cohort and individual ethnicities|
|-----------------|-----------------|------------------|-----------------|-----------------|
|                  | Entire cohort   | African Americans| Hispanics        | Caucasians      |
|-----------------|-----------------|------------------|------------------|-----------------|
| n               | 204             | 102              | 75               | 6               |
| Age (years)     | 42.6 ± 14.1     | 44.8 ± 14.5      | 39.5 ± 12.6      | 44.7 ± 17.8     |
| Sex             |                 |                  |                  |                 |
| Male            | 125 (61.3%)     | 63 (61.8%)       | 44 (58.7%)       | 4 (66.6%)       |
| Female          | 79 (38.7%)      | 39 (38.2%)       | 31 (41.3%)       | 2 (33.3%)       |
|                  |                 |                  |                  |                 |
|                  |                  |                  |                  | 21              |
|                  | 42.7 ± 14.4     | 44.7 ± 17.8      | 4 (66.6%)        | 14 (66.6%)      |
|                  |                 |                  |                  |                 |
over the later decades of the 20th century [1–14]. Initially reported at our institution and then across the globe, not only did FSGS have a predilection for African Americans, but the incidence appeared to be increasing [5, 8, 10, 11, 12]. When first reported, Bakir et al. [1] demonstrated that 47% of African Americans biopsied had FSGS. Subsequently, other investigators confirmed these findings and reported lower rates in Caucasian patients [2–4, 6, 9], increasing the awareness of racial differences in the prevalence of primary glomerular diseases. Further, when analyzed over 20–30 years, it was reported that the incidence of FSGS had been increasing over the later decades of the 20th century [5–11, 14].

Haas et al. [8] and Braden et al. [5] have reported an increasing incidence of FSGS among nephrotic patients. Taken in 5-year increments, Braden et al. [5] reported an increase in FSGS in a rural setting. Of the 616 biopsies analyzed, nearly 420 were noted to have one of the primary glomerulopathies we analyzed. Within these biopsies, the overall prevalence of FSGS increased from 19 to 35.6% and MN decreased from 53.8 to 20.6% over the 20 years analyzed. However, there were few African Americans within this cohort. In the initial quartile spanning 1975–79, of the 12 African Americans biopsied, none had FSGS and 7 had MN. By the last quartile (1990–94), 53 African Americans were biopsied and 17 were found to have FSGS and 8 MN. Over nearly the same time period (1974–93), Haas et al. [8] also reported a surge in the annual incidence of primary FSGS from 4 ± 0.6% in 1974–79 to 12 ± 2.0% in 1987–93 among all renal biopsies analyzed. And, although information on race was limited, the authors were able to demonstrate a robust disproportionate predilection of FSGS for African Americans. Other cohorts have had similar limitations when it comes to racial analyses [10, 11, 14]. A more recent report from the FSGS Clinical Trial analyzed FSGS histological variants in several US cities from 2004 to 2008 in 138 participants aged 2–38 years [19]. Of the 35 adults included in this analysis, 23 (66%) had FSGS NOS, 6 (17%) had the tip variant and 6 (17%) had the collapsing variant. A Caucasian predominance of the tip lesion and African-American predominance of the collapsing variant was noted. Additional comparisons to the current report are limited, however, due to the predominance of younger patients in the D’Agati cohort, especially in light of the reported age-related differences within each of the FSGS variants.

Our results demonstrate a reversal in the trend of an increasing incidence of FSGS in adults, especially in African Americans in the first decade of the 21st century. In the initial series of 100 African-American biopsies in 1986, 47% had FSGS and only 14% had MN [1]. In the current series of 204 patients with primary glomerular disease, this distribution has changed dramatically, so that now, MN has become the leading cause of primary glomerular disease both overall and in African Americans at our institution. Compared with the earlier report by Bakir et al. [1] in 1989 at our institution, the incidence of MN in African Americans increased from 14 to 39.2%, while FSGS declined from 47 to 33.3%. Although the overall number of biopsies included in this cohort is limited, the proportion of African Americans analyzed (n = 102) is comparable or greater than previously published cohorts and thus permits comparison.

In our entire cohort, MN was the most frequent lesion diagnosed, had a prominent nephrotic syndrome and more peripheral edema than other etiologies (Table 3). Despite the high mean 24 h proteinuria (7.9 g), MN had a lower serum creatinine than FSGS and IgAN. Although this finding suggests that MN was diagnosed at an earlier stage of disease, it also indicates a higher preponderance of younger patients in the D’Agati cohort. In this regard, age-related differences in the definition of nephrotic syndrome (i.e., 3.5 g/24 h) may account for the higher prevalence of MN observed in both D’Agati and our study groups.

Table 2. Incidence of primary glomerular diseases in entire cohort and individual ethnicities

| Entire cohort | African Americans | Hispanics | Caucasians | Others |
|---------------|-------------------|-----------|------------|--------|
| n             | 60                | 60        | 29         | 6      |

Table 3. Baseline characteristics within the entire cohort for each primary glomerular disease studied

|        | MN        | FSGS      | MCD       | IgAN     | MPGN     |
|--------|-----------|-----------|-----------|----------|----------|
| n      | 66        | 60        | 29        | 32       | 9        |
| Age (years) | 45.9 ± 12.3 | 40.8 ± 14.9 | 41.3 ± 17.5 | 38.1 ± 11.2 | 52.2 ± 12.8 |
| Sex     | 43 (65.2%) | 41 (68.3%) | 17 (58.6%) | 15 (46.9%) | 6 (66.6%) |
| Male    | 23 (34.8%) | 19 (31.7%) | 12 (41.4%) | 17 (53.1%) | 3 (33.3%) |
| Female  | 20 ± 2.1   | 20 ± 2.1   | 20 ± 2.1   | 20 ± 2.1   | 20 ± 2.1   |
| Creatinine (µmol/L) | 172 ± 223.9 | 203.3 ± 150.28 | 229.8 ± 132.6 | 1,3,4,6 |
| Albumin (g/L)  | 2.4 ± 0.8   | 3.1 ± 1.0   | 2.3 ± 1.0   | 2.3 ± 1.0   | 2,7 ± 1.0   |
| Cholesterol (mmol/L) | 7.7 ± 2.7   | 6.54 ± 2.14 | 5.24 ± 1.81 | 5.19 ± 1.93 | 1,3,4,5,6 |
| Pyuria (#/HPF)  | 8.8 ± 16.2  | 17.3 ± 85.8 | 19.4 ± 7.6 | 34.4 ± 112.8 | 20.3 ± 2.1 |
| Hematuria (#/HPF) | 8.87 ± 3.36 | 5.24 ± 1.81 | 5.19 ± 1.93 | 1,3,4,5,6 |
| Periostitis (%) | 81.5% | 57.9% | 74.0% | 31.3% | 77.8% |

*Data presented as mean ± SD. Significant difference defined as *P* ≤ 0.05 by heteroscedastic T-test for continuous variables and by Fisher’s exact test for categorical data; 1, MN versus FSGS; 2, MN versus MCD; 3, MN versus IgA; 4, FSGS versus MCD; 5, FSGS versus IgA; 6, MCD versus IgA; 7, IgA versus MPGN; MN, membranous nephropathy; FSGS, focal segmental glomerulosclerosis; MCD, minimal change disease; IgAN, immunoglobulin A nephropathy and MPGN, membranoproliferative glomerulonephritis.*
stage of glomerular damage (Table 3), the majority of MN patients had stage 3 or 4 deposits (40/66, 60.6%). Also, when considering only the degree of proteinuria as a marker of severity [17], 44% of MN had a high risk of progression with ≥8 g/day proteinuria.

A male predominance of primary glomerular disease is also noted in our results. In the entire cohort, we noted that males were 1.5 times more likely than females to be diagnosed with a primary glomerular disease. This ratio persisted in both African Americans and Hispanics, 1.6 and 1.7, respectively. These gender differences are comparable with the increased incidence of end-stage renal disease in males in the USA [20] and most reports of adult primary glomerular disease cohorts [5, 7, 8, 14], but not all [2, 6]. Interestingly, within a cohort of 121 African Americans, Korbet et al. [6] noted a similar shift in primary glomerular disease from FSGS to MN when a subgroup older than 45 years of age was analyzed (n = 39). In this older subgroup, the proportion of patients with MN was significantly greater than younger African Americans and equaled that of FSGS in the older subgroup. In younger African Americans, the incidence of FSGS was far greater than MN. This can be more fully appreciated in our current cohort where the proportion of patients with MN was significantly greater than younger African Americans and equaled that of FSGS in the older subgroup. In younger African Americans, the incidence of FSGS was far greater than MN. This can be more fully appreciated in our current cohort where the proportion of patients with MN was significantly greater than younger African Americans and equaled that of FSGS in the older subgroup.

### Table 4. Baseline characteristics within African Americans for each primary glomerular diseases studied

|            | MN       | FSGS     | MCD     | IgAN     |
|------------|----------|----------|---------|----------|
| n          | 40       | 34       | 19      | 3        |
| Age (years)| 46.5 ± 11.5 | 41.8 ± 16 | 42.0 ± 18.8 | 3 |
| Sex        | Male     | Female   | Male    | Female   |
| BUN (mmol/L)| 7.89 ± 4.32 | 24 (70.6%) | 30 (29.4%) | 9 (47.4%) |
| Creatinine (µmol/L) | 159.12 ± 114.92 | 265.2 ± 256.36 | 106.08 ± 61.88 | 1,3 |
| Albumin (g/L) | 2.4 ± 0.8 | 2.8 ± 1.0 | 2.1 ± 0.9 | 3 |
| [g/dL] | [240 ± 80] | [280 ± 100] | [210 ± 90] | 3 |
| Cholesterol (mmol/L) | 6.99 ± 1.86 | 6.47 ± 2.04 | 6.63 ± 2.75 | 3 |
| [mg/dL] | [269.88 ± 71.81] | [249.81 ± 78.76] | [333.20 ± 106.18] | 3 |
| Urine Prot:Creat | 7.1 ± 4.6 | 7.2 ± 7.2 | 7.4 ± 5.4 | 3 |
| Hematuria (#/HPF) | 9.1 ± 17.8 | 25.9 ± 114.9 | 14.4 ± 23.1 | 3 |
| Pyuria (#/HPF) | 3.2 ± 4.0 | 4.3 ± 7.2 | 5.4 ± 8.0 | 3 |
| SBP (mmHg) | 135.0 ± 23.9 | 132.2 ± 22.4 | 130.4 ± 12.5 | 3 |
| DBP (mmHg) | 80.1 ± 11.9 | 85.8 ± 15.7 | 76.2 ± 10.6 | 3 |
| Peripheral edema (%) | 77.5% | 65.6% | 83.3% | 3 |

*Data presented as mean ± SD. Significant difference defined as P ≤ 0.05 by heteroscedastic T-test for continuous variables and by Fisher’s exact test for categorical data; 1, MN versus FSGS; 2, MN versus MCD; 3, FSGS versus MCD; 4, MN, membranous nephropathy; FSGS, focal segmental glomerulosclerosis; MCD, minimal change disease.*

### Table 5. Baseline characteristics within Hispanics for each primary glomerular diseases studied

|            | MN       | FSGS     | MCD     | IgAN     |
|------------|----------|----------|---------|----------|
| n          | 40       | 34       | 19      | 3        |
| Age (years)| 46.5 ± 11.5 | 41.8 ± 16 | 42.0 ± 18.8 | 3 |
| Sex        | Male     | Female   | Male    | Female   |
| BUN (mmol/L)| 7.89 ± 4.32 | 24 (70.6%) | 30 (29.4%) | 9 (47.4%) |
| Creatinine (µmol/L) | 159.12 ± 114.92 | 265.2 ± 256.36 | 106.08 ± 61.88 | 1,3 |
| Albumin (g/L) | 2.4 ± 0.8 | 2.8 ± 1.0 | 2.1 ± 0.9 | 3 |
| [g/dL] | [240 ± 80] | [280 ± 100] | [210 ± 90] | 3 |
| Cholesterol (mmol/L) | 6.99 ± 1.86 | 6.47 ± 2.04 | 6.63 ± 2.75 | 3 |
| [mg/dL] | [269.88 ± 71.81] | [249.81 ± 78.76] | [333.20 ± 106.18] | 3 |
| Urine Prot:Creat | 7.1 ± 4.6 | 7.2 ± 7.2 | 7.4 ± 5.4 | 3 |
| Hematuria (#/HPF) | 9.1 ± 17.8 | 25.9 ± 114.9 | 14.4 ± 23.1 | 3 |
| Pyuria (#/HPF) | 3.2 ± 4.0 | 4.3 ± 7.2 | 5.4 ± 8.0 | 3 |
| SBP (mmHg) | 135.0 ± 23.9 | 132.2 ± 22.4 | 130.4 ± 12.5 | 3 |
| DBP (mmHg) | 80.1 ± 11.9 | 85.8 ± 15.7 | 76.2 ± 10.6 | 3 |
| Peripheral edema (%) | 77.5% | 65.6% | 83.3% | 3 |

*Data presented as mean ± SD. Significant difference defined as P ≤ 0.05 by heteroscedastic T-test for continuous variables and by Fisher’s exact test for categorical data; 1, MN versus FSGS; 2, MN versus MCD; 3, FSGS versus MCD; 4, MN, membranous nephropathy; FSGS, focal segmental glomerulosclerosis; MCD, minimal change disease; IgAN, Immunoglobulin A nephropathy.*
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explanation for this, an age-related effect on etiology of primary glomerular disease is suggested by these two cohorts. Similarly, in the 75 Hispanics biopsied in our cohort, MN was the most common primary glomerular disease (32%), followed by FSGS (25.3%), IgAN (22.7%) and MCD (8%). In 2000, Braden et al. [8] had reported a 21.3% incidence of FSGS and a 8.1% incidence of MN in 65 Hispanics biopsied between 1990 and 1994. Our findings, in 75 Hispanics, corroborate these earlier results and suggest that Hispanics may have a predisposition to FSGS. However, unlike this earlier report, our results indicate that MN was more prevalent than FSGS, possibly reflecting a difference in populations; most of the Hispanics in our current report were originally from Central American countries, especially Mexico, compared with most of the Braden cohort from Puerto Rico. Alternatively, the smaller sample sizes may bias comparison. In a larger Brazilian cohort [11], FSGS was the most prevalent lesion among 943 biopsies of primary glomerular disease (32.1%) and increased from 1979 to 1999. Interestingly, unlike the earlier Braden report [8], MN was second most prevalent at 19.5% and did not change over the period of observation. Unfortunately, the ethnic makeup of this cohort was not reported which makes direct comparisons difficult. An alternate distribution was noted in a smaller cohort of children with nephrotic syndrome between 1978 and 1997 [21]. This retrospective study reported an 11% incidence of FSGS among the 37 Hispanic children included in the analysis. Within the entire cohort of 105 children, the incidence of FSGS was noted to have increased from 23 to 47% before and after 1990; however, the contribution of individual ethnicities to the changing incidence was not mentioned. Another large Spanish-speaking renal biopsy cohort, albeit not from Central America, was reported from Spain in which 7016 biopsies were analyzed between 1994 and 1999 [22]. Although FSGS and MN had similar overall frequencies at 10 and 9.7%, this analysis did not distinguish between primary and secondary glomerular diseases and included nonglomerular diagnoses (e.g., acute interstitial nephritis) in the analysis making comparisons difficult.

In our study, Hispanics with MN had a significantly lower serum creatinine and higher total cholesterol when compared with African Americans. Although not statistically significant, Hispanics with MN also had more extensive proteinuria and more edema. In fact, among patients with MN there were a greater proportion, albeit insignificant, of Hispanics with >8 g/day of proteinuria than African Americans (54 versus 32.5%, respectively). This trend suggests a propensity toward a more explosive form of MN with greater proteinuria and less tissue damage in Hispanics. However, Churg stage was similar in both ethnic groups suggesting another unknown factor. Conversely, Hispanics with FSGS, had significantly lower proteinuria and less edema than African Americans. Interestingly, these differences are not seen when only FSGS NOS is analyzed and a comparison was not possible in the collapsing variant since there was only one Hispanic with this lesion. No ethnic differences were noted in either MCD or IgAN.

This study demonstrates the prevalence of primary glomerular diseases seen in African Americans and Hispanics cared for at our institution from 2001 to 2011. We have described an increase in MN and a decline in the incidence of FSGS in African Americans in the first decade of the 21st century. Although seemingly contrary to prior reports of an increasing incidence of FSGS in the overall population, our cohort contains very few Caucasians and is comprised primarily of African Americans and Hispanics, unlike the majority of the earlier reports. In comparing African American cohorts, ours is similar in size to most, yet has the novel finding of an increase in MN and decline in FSGS. Although the causes of idiopathic MN and FSGS are beginning to be elucidated, it remains unclear why the incidence of FSGS increased in the last few decades of the 20th century [5, 7, 9–11, 14] only to decline in the first decade of the 21st century as seen in our cohort. Such changes in ethnic predilection for the primary glomerular diseases need to be studied further to determine whether there are important prognostic implications.

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(See Editorial Comment by M. Haas. Incidences of membranous nephropathy versus focal segmental glomerulosclerosis: increase in the former or decline in the latter? Clin Kidney J 2013; 6: 360–367)

References

1. Bakir AA, Bazilinski NG, Rhee HL et al. Focal segmental glomerulosclerosis. A common entity in nephrotic black adults. Arch Intern Med 1989; 149: 1802–1804
2. Pin-Wiggins VW. Nephrotic syndrome in adults. Clin Nephrol 1994; 42: 79–84
3. Ingulli E, Tejani A. Racial differences in the incidence and outcome of idiopathic focal segmental glomerulosclerosis in children. Pediatr Nephrol 1991; 5: 393–397
4. Pontier PJ, Patel TG. Racial differences in the prevalence and presentation of glomerular disease in adults. Clin Nephrol 1994; 42: 79–84
5. Haas M, Spargo BH, Coventry S. Increasing incidence of focal-segmental glomerulosclerosis among adult nephropathies: a 20-year renal biopsy study. Am J Kidney Dis 1995; 26: 740–450
6. Korbet SM, Genchi RM, Borak RZ et al. The racial prevalence of glomerular lesions in nephrotic adults. Am J Kidney Dis 1996; 27: 647–651
7. Haas M, Meehan SM, Karrison TG et al. Changing etiology of unexplained adult nephrotic syndrome: a comparison of renal biopsy findings from 1976–1997 and 1995–1997. Am J Kidney Dis 1997; 30: 621–231
8. Braden GL, Mulhern JG, O’Shea MH et al. Changing incidence of glomerular disease in adults. Am J Kidney Dis 2000; 35: 878–883
9. Dragovic D, Rosenstock JL, Wahl SJ et al. Increasing incidence of focal segmental glomerulosclerosis and an examination of demographic patterns. Clin Nephrol 2005; 63: 78–79
10. Swaminathan S, Leung N, Lager DJ et al. Changing incidence of glomerular disease in Olmsted County, Minnesota: a 30-year renal biopsy study. Clin J Am Soc Nephrol 2006; 1: 483–487
11. Bohiense-Oliveira M, Saldanha LB, Moto EL et al. Primary glomerular diseases in Brazil (1979–1999): is the frequency of focal and segmental glomerulosclerosis increasing?. Clin Nephrol 2004; 61: 90–97
12. Malofronte P, Mastroianni-Kirstoaj G, Betonico GN et al. Paulist registry of glomerulonephritis: 5-year data report. Nephrol Dial Transplant 2006; 21: 3098–3105
13. Narasimhan B, Chacko B, John GT et al. Characterization of kidney lesions in Indian adults: towards a renal biopsy registry. J Nephrol 2006; 19: 205–210
14. Hanko JP, Mullan RN, O’Rourke DM et al. The changing pattern of adult primary glomerular disease. Nephrol Dial Transplant 2009; 24: 3050–3054
15. Genovese G, Tonna SJ, Knob AU et al. A risk allele for focal segmental glomerulosclerosis in African Americans is located within a region containing APOL1 and MYH9. Kidney Int 2010; 78: 698–704
16. Kopp JB, Smith MW, Nelson GW et al. MYH9 is a major-effect risk gene for focal segmental glomerulosclerosis. Nat Genet 2008; 40: 1175–1184
17. Pei Y, Cattran D, Greenwood C. Predicting chronic renal insufficiency in idiopathic membranous glomerulonephritis. Kidney Int 1992; 42: 960–966
18. Cattran DC, Pei Y, Greenwood CM et al. Validation of a predictive model of idiopathic membranous nephropathy: Its clinical and research implications. Kidney Int 1997; 5: 901–907
19. D’Agati V, Alster JM, Jennette JC et al. Association of histologic variants in FSGS clinical trial with presenting features and outcomes. Clin J Amer Soc Neph 2012; 8: 1–8
20. US Renal Data System. USRDS 2009 Annual Data Report: Atlas of End-Stage Renal Disease in the United States. Bethesda, MD: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, 2009
21. Bonilla-Felix M, Parra C, Dajani T et al. Changing patterns in the histopahology of idiopathic nephrotic syndrome in children. Kidney Int 1999; 55: 1885–1890
22. Rivera F, Lopez-Gomez JM, Perez-Garcia R. Representing the Spanish Registry of Glomerulonephritis; frequency of renal pathology in Spain 1994–1999. Nephrol Dial Transplant 2002; 17: 1594–1602

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