Original Article

Socio-Economic Impact on Presentation and Progression of Chronic Glomerulonephritis

Hossain SMM[1], Nahas AME[2]

Abstract

There are some literature describing the socio-economic impact on chronic kidney disease (CKD). Chronic Glomerulonephritis (CGN) is one of the major causes of CKD. But there is only a few literatures that show directly the socio-economic impact of CGN. We conducted a retrospective cohort study to evaluate wheather there is any impact of socio-economic status on CGN.

The medical records of 115 patients with CGN undertaking regular follow up in the Sheffield Kidney Institute Northern General Hospital, Sheffield, UK were reviewed. Information on age, sex, race, weight, occupation, living and GP's postcode, use of angiotensin converting enzyme inhibitors or angiotensin receptor blockers, statins, blood pressure, serum cholesterol, haemoglobin, and 24 hour urinary protein excretion were retrospectively collected from the computerized patient record system. Other information such as height and smoking were retrieved from patients' case notes. Socio-economic status of all patients was determined by postcode and Index of Multiple Deprivation-2004 (IMD-2004). Multivariate regression analysis was used to identify the predictors of CGN presentation and progression.

Comparison between the demographic variables between least deprived and most deprived group of the study population did not show any significant statistical differences. Patients presenting with CGN are mostly coming from the Low socio-economic group. There is no impact of socio-economic status on presentaion and progression of CGN. Prospective intervention trials rwarranted to find out the hidden risk factors of presentaion and progression of CGN.

Keywords: *Chronic Glomerulonephritis (CGN), Socio-economic Status (SES), Index of Multiple deprivation (IMD), Chronic kidney disease (CKD), Estimated GFR (e GFR), Proteinuria, Hypertension.*

1. Dr. S. M. Murad Hossain MBBS, MSc in Nephrology (University of Sheffield UK.)
Medical Officer Department of Medicine
General Hospital Khulna
2. Prof. A. Meguid El Nahas
Sheffield Kidney Institute
Northern General Hospital University of Sheffield UK.

Introduction:

Chronic kidney disease (CKD) is a worldwide public health problem. According to the World Health Report 2002 and Global Burden of Disease (GBD) project, diseases of kidney and urinary tract contribute to the global burden of diseases, with approximately 850,000 deaths every year and 15,010,167 disability-adjusted life years[1-3]. They are the 12th cause of death and the 17th cause of disability, respectively. CKD is increasing worldwide at an annual growth rate of 8%.Epidemiological studies have shown that the incidence and prevalence of kidney diseases is higher in the developing countries than in the industrialized world [4]. Data for much of the developing world are often unavailable due to lack of renal registries, but given the prevalence of poor socio-economic factors, the incidence is likely to be greater[5]. In the industrialized countries, the prevalence of CKD increases with age[6-8].

Although diabetic nephropathy increasing alarmingly in the recent years, the most common causes of CKD in the developing countries are chronic glomerulonephritis (CGN) and systemic hypertension, diabetic nephropathy being the most common cause in Europe, the United States and Japan. Renal disease, especially glomerular disease, is more prevalent in Africa and seems to be of a more severe form than is found in western countries[9]. The primary health problems in Africa are HIV/AIDS,tuberculosis, malaria, gastroenteritis and hypertension. CGN and hypertension are principal causes of CKD in tropical Africa and East Africa, together with diabetes mellitus and obstructive uropathy[10-11]. In North of Africa, the incidence of kidney diseases is much higher than that in West Africa. The principal causes of CKD are interstitial nephritis (14% to 32%), glomerulonephritis (11% to 24%), diabetes (5% to 20%) and nephrosclerosis (5% to 21%)[12]. In Indian Subcontinent, CGN is the most common cause, accounting for more than one third of patients, while diabetic nephropathy accounts for about one fourth of all patients[13]. In other countries of South-east Asia, the epidemiology of renal disease in this region is also poorly understood. CKD occurs most commonly from chronic glomerulonephritis and nephrolithiasis as well as from complicated acute renal failure due to a variety of stimuli[14]. In China Ig A nephropathy is the leading cause of CKD, among the primary causes of glomerulonephritis, while lupus nephritis is the most prominent among the cause of secondary glomerulopathy[15]. In South American Countries such as Brazil, chronic glomerulonephritis is the leading

cause of CKD[16]. Immunoglobulin A (Ig A) nephropathy is the most common form of primary glomerulonephritis world wide. The prevalence of Ig A nephropathy varies among racial groups, being more common in Native Americans from New Mexico[17]and rare in African Americans. While the rate of glomerulonephritis-related ESRD is second only to diabetes among indigenous Australians, there does not appear to be a specific increase in Ig A nephropathy[18]. In 2000, glomerulonephritis was a major cause of ESRD among Filipino patients in Hawaii undergoing dialysis, accounting for 24% of cases[19]. This figure was twofold higher than in the continental United States ESRD population. This higher prevalence of glomerulonephritis may be related to the higher rates of Ig A nephropathy among Asians worldwide.

Strong evidence indicates that socio-economic disadvantage is associated with increased risk of ESRD [17,20], a complex interaction that might directly precipitate renal damage or influence the quality of health care of those with kidney disease. Individual-level socio-economic status (SES) has been found to be associated inversely with progressive CKD. The effect of area level SES on progressive CKD is not well known. Few studies have examined the relationship between early stage of kidney disease (pre-ESRD) and SES. The recent literature suggests that neighbourhood or community socio-economic characteristics may have important roles in affecting an individual's health, independent of that person's individual SES[21-23]. In the specific case of kidney disease, relevant area characteristics associated with impoverished areas may include exposure to lead, stressful exposure to high crime levels, limited access to health information and resources, and limited access to healthy foods and recreational resources[24-26]. Area SES is likely to be a proxy for these features. Socio-economic factors, such as low income, poor education, residence in a low-income areas and consequently poor access to health care, are strong predictors for the development of ESRD[27, 28] (Fig.1).

Our knowledge and understanding of impact of socio-economic factors on CKD can be advanced by focusing on the properties of social (family and community) and physical environments and the mechanisms by which they influence health and health outcomes, and the magnitude and the mechanisms through which socio-economic factors of poverty, racism, and discrimination affect health. A long history of research shows that health-related problems vary systematically by community, often in conjunction with socio-economic characteristics[29]. Social characteristics vary across communities along dimension of socio-economic status (e.g., poverty, wealth, occupational attainment), cultural context, family structure and life cycle (e.g., female-headed households, child density), residential stability (e.g., home ownership and tenure), racial and ethnic composition (e.g., racial segregation), and language (e.g., linguistic minorities of any language). Many of these factors, the interaction among the factors, and their relation to health access and quality and to health outcomes are inadequately defined and understood.

Figure: 1

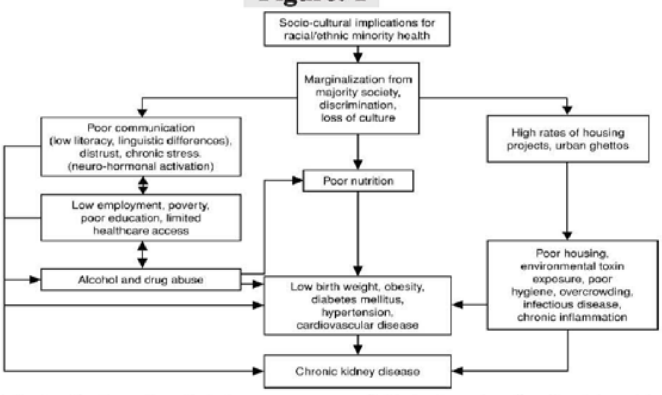

Fig.1. Socio-cultural influences on racial/ethnic minority health with implications for chronic kidney disease (CKD) [41]. Modified from Cunningham(BMJ 2003).

The relationship between socio-economic disadvantages and CKD has not frequently been reported. Research on patterns of incidence of CKD has generally been limited to a description of differences according to age, sex, race, and state or territory. One study was done by Alan Cass and his colleagues in Australia, published in NSW Public Health Bulletin July 2002, which described the relationship between the incidence of ESRD and indicators of socio-economic disadvantages at the indigenous Australians by Aboriginal and Torres Strait Islanders Commission (ATSIC) region. Strong associations were evident between the incidence of ESRD and indicators of socio-economic disadvantages[30].

Another study was done by the same group where they described the relationship between the incidence of ESRD and indicators of socio-economic disadvantages within the capital cities of Australia. They used the Index of Relative Socio-economic Disadvantage (IRSD), developed by ABS. The IRSD, constructed using principal-component analysis, is derived from attributes such as income, educational attainment, employment status, and occupation [31]. Thus they were able to show a strong association between incidence of ESRD and area-based markers of socio-economic disadvantage. In 2003, a population-based case-control study was done in Sweden to show the relationship of socio-economic status and chronic kidney disease where they used occupation and educational level independently to estimate SES[32]. My aim of this study is to find out the socio-economic impact on presentation and progression of CGN. In my study I will use the Index of Multiple Deprivation (IMD) 2004 UK to determine the social deprivation index of patients presenting with CGN.

Subjects and Methods:

Setting: In Sheffield, The National Health Service (NHS) UK provides health care at hospital and primary health care centres to all residents. This free service always tries to ensure equal opportunities to all residents. This study was done in the Sheffield Kidney Institute (SKI) which is a tertiary care specialized teaching hospital in the South Yorkshire region of United Kingdom(UK) during the period of 1st June to 20th September 2006

Subjects: List of patients with GN was provided by the SKI. Eligible as subject were the patients with biopsy proven CGN. A list of 500 patients was provided, out of which only 115 patients made my inclusion criteria. Inclusion criteria were patients from Sheffield town only-those received treatment for biopsy proven CGN and continuing their follow-up at SKI, age between 18 to 90 years. Chronic Glomerulonephritis was defined as GN for at least 3months or more {as evidenced by persistent proteinuria (24hours proteinuria >150 mg/day at or after 3 months of initial biopsy diagnosis. Patients living out-side the Sheffield city and who did not continue their follow-up at SKI, age <18 years or >90 years, GN that completely resolved (no proteinuria or 24 hours proteinuria <150 mg/day) within 3 months have been excluded from this study. Diagnosis of associated condition was based on routine clinical work-up.

Data collection: Sheffield Kidney Institute (SKI) maintains the NHS database system called "PROTON" which is well organised, continuously updating with all the information of each patient in their every visit to SKI. Data for this study was collected from the "PROTON" database system. Demographic information such as patients' age, sex, race, weight, their living and GP's postcode, occupation, marital status are taken first. Then clinical and laboratory parameters such as biopsy diagnosis along with dates, presence or absence of Hypertension, Systolic and Diastolic blood pressure, Use of Angiotensin Converting Enzyme (ACE) inhibitors or Angiotensin Receptor Blockers (ARBs), Statins, Serum cholesterol, Haemoglobin level, Serum creatinine, Serum urea, and 24hours urinary protein excretion estimated Glomerular Filtration Rate (e GFR) were retrospectively collected from the computerized patient record system in three phases-baseline(at the time of diagnosis), mean follow-up period and at present. Other information such as smoking and alcohol intake were retrieved from patients' case notes kept in medical record room.

Methodology: Socio-economic status can be determined either by individual level or by area level. All the previous studies in this ground were considered either individual level SES or community level SES alone which may not reflect the true SES of that population as neighbourhood or community socio-economic characteristics may have important influences in affecting an individual's health, independent of person's individual SES.[22-23].That is why I considered both for estimation of SES. To determine the area level socio-economic status of Sheffield town I relied on the "Index of Multiple Deprivation (IMD) 2004-PCT level"-published by the UK government on 28th of April 2004 which measures the multiple deprivations at small area level and local authority area in England. Previous researchers those who used the community level SES only considered few indicators of SES to determine the SES of study population due to absence of generally accepted area based index of socio-economic disadvantages. But the (English) Index of Multiple Deprivation, developed in 2004 ranks districts in several "domains" of deprivation relating to : Income, Employment, Health and disability, Education, skills and training, Barriers to Housing and Services, quality of Living environment and crime, which are given different weights to produce an overall, composite indicator of deprivation. From IMD-2004 by PCT level (based on national quantile) if we consider only Sheffield town, we see that North and South-east Sheffield is most deprived area and South-west and West Sheffield is least deprived area (Fig. 2). Primary care trust or GP's postcode of each patient has been matched with the above two regions of Sheffield town and thus the area level socio-economic status of each patient has been measured and finally classified as most deprived and less deprived groups. Here it is to be mentioned that patients GP postcode represents the same area of their living postcode in most cases and exceptional cases have been excluded from the study.

Occupation of each individual patient was considered to measure the SES at individual level. In case of individual level SES determination, I considered only occupation because of lack of information of other indicators such as individual patient's educational attainment. But educational level was considered as an important domain in IMD-2004.

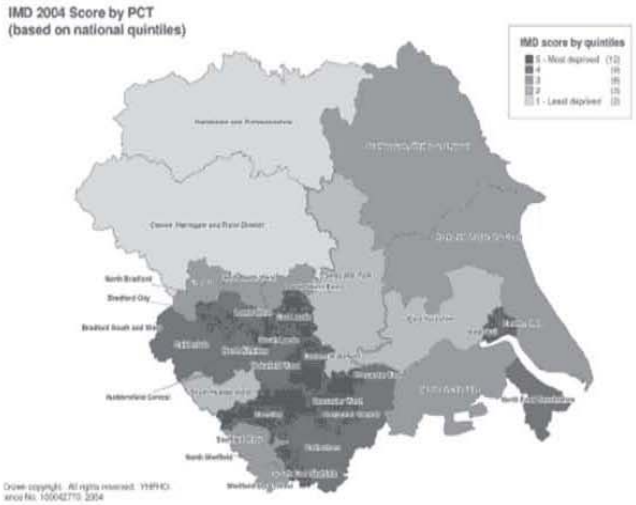

Fig.2. IMD 2004 Score by PCT (Sheffield Town in UK)

For the great variation in occupation, I classified them by Standard Occupational Classification 2000 (SOC 2000), produced by the Government Statistical Service (GSS). SOC-2000 has 9 major groups of occupation. But those groups are again modified and clustered into 3 classes because of purpose of description of limited number of study subjects. Each study subject was classified according to the modified SOC-2000 where Class-1 and Class-2 were considered as high SES and Class-3 was considered as low SES. For the purpose of description, patients are further classified according to their age–below 60years and ≥60years, according to race-White and Non-white. Progression rates are determined by decline in estimated Glomerular Filtration Rate (e GFR) per year. Decline in e GFR more than 2ml/min/1.73m2/year is considered as progressor and less than that as non-progressor.

Statistical Analysis:

All data obtained was collected into Microsoft Excel spreadsheets. Data were then transferred and analyzed using the Statistical Package for Social Sciences (SPSS, Inc., Chicago, IL, and Version 14.0.1). Descriptive statistics are presented as mean and standard deviation or number (percent). Data were analyzed using parametric T-test to compare the frequency distribution of the continuous variables and Chi-square (X2) test was used to compare the frequency distribution of categorical variables. Pearson's correlation coefficient (r) was used to examine the associations between social class and different variables of the study. Multivariate linear regression analysis was used to identify the predictors of presentation of CGN based on 24 hours proteinuria as the dependable variable. Multivariate logistic regression analysis was used to identify the predictors of progression of CGN based on progression rate as the dependable variable. Multivariate stepwise, forward, enters and backward regression analyses were also used to identify the predictors and produce the predictive models of progression of CGN. All analyses were done with 95% confidence intervals, all P values were two sided and $P < 0.05$ was considered as statistically significant. Finally a Binomial test was applied to find out the statistical significance of the difference of social class of patients presenting with CGN.

Result:

Demographic characteristics of study population: The study population was classified according to the age (below 60 years and 60 years and above), sex (male,female), race (white and non-white), occupational class {Class 1 (Senior Officials, Professionals, Technical), Class 2 (Secretarial, Skilled, Personal Service), Class 3 (Sales, Machine Operatives, Elementary Occupations)} and social class {most deprived and least deprived.The number of patients below 60 years {64 (55.7%) was nearly equal to number of patients above 60 years or more{51 (44.3%}. There were more than thrice as many men as woman. Among the study population 83.5% was white and only 16.5% was non-white. More than 50% of study subjects was having the occupational class-3 according to modified SOC-2000. The valid percentage of smoker among the study subjects was 31.3% where as non-smoker was 22%. The observed percentage of alcoholic and non-alcoholic person was nearly same as smoker and non-smoker.

Clinico-demographic characteristics of study population: Demographic characteristics such as age, sex, race, occupation, marital status, smoking, alcohol intake- all are compared between most deprived and least deprived group of study population. The mean age and body weight of both groups was similar. At the same time clinical parameters such as presence or absence of hypertension, use of ACE/ ARBs, use of statin, progression rate of CGN are also compared. Comparison between the above mentioned characteristics and clinical parameters between least deprived and most deprived group of the study population did not show any significant statistical differences. Details of this comparison are shown on Table.1. Comparison of other clinical parameters such as haemoglobin, serum creatinine, serum urea, e GFR, 24 hour proteinuria, serum cholesterol, systolic and diastolic blood pressure are also done in three phases-baseline(at the time of initial diagnosis), mean follow-up period and at present. There were no statistically significant differences between the two groups except present serum creatinine (P-value = 0.049) and present diastolic blood pressure (P-value = 0.054).

Correlation between social class and other variables in the study population: Present serum creatinine (P-value = 0.049) and present diastolic blood pressure (P-value = 0.054) have negative correlation with social class. Other than that there was no significant correlation between the

Table 1 Comparisons between Demographic Characteristics and Clinical Parameters (Chi-Square Test) between LD and MD groups:

Variable		LD N%	MD N%	P-value
Age group	Below 60 years	27 (65.9)	37 (50)	0.1
	60 years or above	14 (34.1)	37 (50)	
Sex	Male	34 (82.90	55 (74.3)	0.29
	Female	7 (17.1)	19 (25.70	
Racial Class	White	35 (85.4)	61(82.4)	0.68
	Non-white	6 (14.6)	13 (17.6)	
Occupational class	Class-1	7 (17.1)	4 (5.7)	0.09
	Class-2	13 (31.7)	19 (26.8)	
	Class-3	21(51.2)	48 (67.6)	
Marital Status	Single	13(31.7)	19(25.7)	0.69
	Married	23(56.1)	42(56.8)	
	Divorced	3(7.3)	5(6.8)	
	Widow	2(4.9)	8(10.8)	
Smoking	Smoker	12(29.3)	24(32.4)	0.56
	Non-smoker	10(24.4)	12(16.2)	
Alcohol Intake	Alcoholic	16(39)	22(29.7)	0.57
	Non-alcoholic	6(14.6)	14(18.9)	
Baseline use of Statin	Statin User	20(48.8)	27(36.5)	0.26
	Statin Non-user	13(31.7)	35(47.3)	

Baseline Hypertension	Present	20(48.8)	35(47.3)	0.98
	Absent	14(34.1)	26(35.1)	
Baseline use of ACEi/ARBs	ACEi/ARBs User	20(48.8)	36(48.6)	0.94
	ACEi/ARBs Non-user	14(34.1)	27(36.5)	
Mean Follow-up Hypertension	Present	21(51.2)	42(56.8)	0.73
	Absent	13(31.7)	23(31.1)	
Mean Follow-up Use of ACEi/ARBs	ACEi/ARBs User	22(53.7)	38 (51.4)	0.94
	ACEi/ARBs Non-user	12 (29.3)	24 (32.4)	
Hypertension at Present	Present	23 (56.1)	47 (63.5)	0.67
	Absent	13 (31.7)	21 (28.4)	
Progression Rate	Non-Progressor	12 (29.3)	29 (39.3)	0.25
	Progressor	28 (68.3)	45 (60.8)	
Mean Follow-up use of Statin	Statin User	21 (39.6)	32 (60.4)	0.38
	Statin Non-user	13 (28.3)	33 (71.7)	

social classes and other parameters. Correlation analyses between social class and different variables are shown in the Table.2.

Table 2 Correlation analysis between social Class and different variables

Variable		Social Class
Age	Pearson Correlation(r)	.092
	P-value	.331
Weight	Pearson Correlation(r)	.173
	P-value	.102
Baseline Serum Creatinine	Pearson Correlation(r)	.061
	P-value	.515
Baseline Serum Urea	Pearson Correlation(r)	.082
	P-value	.082
Baseline e GFR	Pearson Correlation(r)	-.076
	P-value	.417
Baseline Haemoglobn	Pearson Correlation(r)	-.115
	P-value	.223
Baseline Cholesterol	Pearson Correlation(r)	-.035
	P-value	.733
Baseline 24 hour Proteinuria	Pearson Correlation(r)	-.084
	P-value	.371
Baseline Systolic BP	Pearson Correlation(r)	.075
	P-value	.474
Baseline Diastolic BP	Pearson Correlation(r)	-.090
	P-value	.390
Mean Follow-up Sr. Cr	Pearson Correlation(r)	.006
	P-value	.948
Mean Follow-up Sr.Urea	Pearson Correlation(r)	.031
	P-value	.740
Mean Follow-up e GFR	Pearson Correlation(r)	.079
	P-value	.401
Mean Follow-up Hb	Pearson Correlation(r)	-.098
	P-value	.301
Mean Follow-up Cholesterol	Pearson Correlation(r)	.002
	P-value	.987
Mean Follow-up 24hr Proteinuria	Pearson Correlation(r)	-.098
	P-value	.345
Mean Follow-up SBP	Pearson Correlation(r)	.005
	P-value	.962
Mean Follow-up DBP	Pearson Correlation(r)	-.088
	P-value	.407
Present Serum Creatinine	Pearson Correlation(r)	-.184
	P-value	.049
Present Serum Urea	Pearson Correlation(r)	-.142
	P-value	.129
Present e GFR	Pearson Correlation(r)	.128
	P-value	.173
Present Haemoglobn	Pearson Correlation(r)	-.116
	P-value	.219
Present Cholesterol	Pearson Correlation(r)	.063
	P-value	.545
Present 24 hour Proteinuria	Pearson Correlation(r)	-.021
	P-value	.840
Present Systolic BP	Pearson Correlation(r)	-.018
	P-value	.864
Present Diastolic BP	Pearson Correlation(r)	-.203
	P-value	.054

Prediction analysis:

Predictors of CGN presentation: All demographic and baseline variables including social class and occupational class were entered into backwards selection models. A total of 9 regression models were generated using multivariate backward linear regression analysis. The regression model number (9) was taken as the predictive model since it contains few numbers of variables with a high R2 value. By backward elimination of variables, baseline Haemoglobin (P-value = 0.046) and baseline Serum Urea (P-value =. 0.015) was found to be significant predictors of CGN presentation based on 24 hours Proteinuria. But there is no impact of SES on presentation of CGN.

Prediction of CGN progression based on Progression rate (e GFR reduction per year): Multivariate stepwise, forward, enter and backward regression analyses were also used in three phases (baseline, mean follow-up, at present) to identify the predictors and produce the predictive models of progression. All demographic and baseline variables including social class and occupational class were entered into forwards selection models. One regression model was generated using multivariate forward logistic regression analysis. The regression model was taken as the predictive model. By forward elimination of variables, baseline Serum Urea (P-value = 0.005) was found to be significant predictor of CGN progression. To obtain the most robust model of mean follow-up parameters that could predict the CGN progressions, all mean follow-up variables were taken into enter selection model. One regression model was generated using multivariate enter logistic regression analysis. The regression model was taken as the predictive model. Mean follow-up serum urea (P-value = 0.032) was found to be significant predictor of CGN progression. Finally all present variables were taken into backward selection model. Five regression models were generated using multivariate backward logistic regression analysis. Present Serum Urea (P-value = 0.006) and DBP (P-value = 0.041) were found to be significant predictor of CGN progression. But there is no significant impact of SES on progression of CGN.

Result of Binomial Test: Table.3 shows that patient presenting with CGN are mostly coming from the most deprived group of study population and that is statistically significant (P-value = 0.003).

Table 3 Binomial Test.

		Category	N	Observed Prop.	Test Prop	Asymp. Sig. (2-tailed)	Exact Sig. (2-tailed)
Social Class of patients presenting with Chronic Glomeru lonephritis	Group 1	Most Deprived (North and South-East Sheffield)	74	.64	.50	.003(a)	.003
	Group 2	Least Deprived (West and South-West Sheffield)	41	.36			
	Total		115	1.00			

Discussion: In this retrospective study demographic pattern of my study population showed that there were no significant differences between the most and least deprived group. However, male were most commonly affected by CGN in comparison to female. This pattern was also seen in other studies and this could be supported by the fact that ESRD due to CKD is observed in higher proportion in male than in female[33]. The mean age of my study population in both groups was similar. This is similar to the fact that the incidence of renal disease increases with age. In the USA, the incidence of ESRD is around 117pmp per year in patients aged between 20-44 years and 542pmp between 45-64 years[33].

In my study it has been observed that most of the patients with CGN were white (83.5%) in comparison to non-white (16.5%). This may contrast the fact that a higher level of ESRD for all causes in African-American compare with white men at all levels of blood pressure [34]. But this finding of my study can be explained by the careful observation of ethnic diversity of Sheffield town. According to Wikipedia's list of English districts by ethnic diversity (based on the 2001 UK census) Sheffield's ethnic diversity shows- 91.2% are white and only 1.8% are Afro-Caribbean. This is why I have found more white patients. It is also worth mentioning here that racial class did not show any significant differences between the two study groups.

Among the study population most commonly observed histological pattern of glomerulonephritis was the Ig A nephropathy and Membranoproliferative glomerulonephritis (MPGN). Ig A nephropathy is common world wide as I mentioned earlier. Patient with MPGN in most deprived group was almost double than that of least deprived group. Higher rates of infection due to sub-optimal housing standard, lack of personal hygiene in most deprived area can be associated with increase number of infection related MPGN [35].

Comparison of clinical parameters between least deprived and most deprived group in three phases (baseline, mean follow-up and at present) did not show any significant differences except in present serum creatinine and present diastolic blood pressure. The mean present serum creatinine level in most deprived group was 194.17 µmol/lit where as in most deprived group it was 146.28 µmol/lit. The lower mean serum creatinine level in most deprived group can be explained by the fact that these group of people are more prone to malnutrition and malnutrition invariably lowers serum creatinine level. The mean DBP of the least deprived group was higher than that of most deprived group. The sedentary lifestyle, decreased physical inactivity, high fatty food in least deprived group could be the reason for that.

Linear regression analysis for predicting the factors of CGN presentation showed that baseline serum urea and baseline haemoglobin could be predicting factors for CGN presentation. But it did not show any impact of SES on the presentation of CGN. This is because SES does not plausibly affect renal function and can not be regarded as a specific exposure but a marker for general material and cultural circumstances. Associated biologically meaningful exposure are likely to explain most or all of the relationship with CKD and there by CGN[32].

Logistic regression analysis in my study to predict the progression of CGN showed that serum urea and DBP at present could predict the progression of CGN. But it did not show SES as a predicting factor for progression. All though level of serum urea is not a good measure of renal function as many factors such as volume status, dietary protein, and liver disease all are affecting the serum urea level but we can use initially serum urea level as a supportive clue for renal function. Strong evidence links the progression of CKD to systemic hypertension. It has been suggested that the rate of progression of CKD is twice as fast in patients with DBP in excess of 90 mm of Hg compared with those with lower level[36].

Conclusion: In summary, my study has shown that SES has no effect on presentation and progression of CGN although significant more people are coming from the most deprived group. Some previous researchers have shown that low socio-economic status is associated with an increased risk of CKD and ESRD[32, 37-39] in their studies where higher number of study population was having diabetic nephropathy, renal vascular diseases, and obstructive uropathy as a cause of CKD. So to conclude that CGN come from socio-economically most deprived group in Sheffield, I must know from where the rest of CKD patients are coming up. If all CKD patients in Sheffield are coming from most deprived group then there is no difference. Until now no such study has been conducted although one of my colleagues is working on it.

Events involved in the initiation of glomerular disease are still poorly understood. Most glomerular diseases develop as a result of immune dysregulation, either an inappropriate immune response to self-antigens occurring through a failure to tolerance ("autoimmunity") or an ineffectual response to foreign antigen[40]. So it is unlikely to have any impact of SES on CGN as immune mechanism is not known to be altered by SES. The reason why more people are coming from the most deprived group could be due to higher number of people are living in most deprived area because of lower living cost or migration of people (change of living places) from least deprived to most deprived area. The reason why SES does not have any impact on progression of CGN in my study may be due to higher standard of health care provided by the SKI. Early detection, regular follow-up by nephrologists, appropriate treatment measures all can slow the progression of CGN. Some other factors such as dietary and food behaviour, lifestyle, and educational level which are now known to affect the renal function and can be obtained best by questionnaire was not considered in my study due to shortage of time in this 3 months project. Data regarding smoking, alcohol intake, BMI were insufficient although every effort has been made to up to date my study. Result of this study, although mostly limited to white people, indicate a need for further study. The mediating factors that are considered and analyzed in this study did not explain much of the association between SES and CGN presentation and progression, suggesting that other factors may be involved. Thus the underlying hidden mechanisms warrant further research to identify possibly preventable risk factors for CGN.

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