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# Clinicopathologic Profile and Outcome of Adult Patients with Asymptomatic Urinary Abnormalities at National Kindey and Transplant Institute

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## Abstract

**Background:** Primary chronic glomerulonephritis which, can present initially with asymptomatic urinary abnormalities (AUA), may progress over time and lead to chronic kidney disease.

**Objectives:** To describe the clinicopathologic profile and renal outcome among patients who presented with AUA.

**Methods:** Charts of 1,550 patients admitted from January 2010 to December 2012 for kidney biopsy were reviewed. Clinicopathologic profile of patients with AUA was described and followed up at 36th month post-biopsy.

**Results**: Around 153 presented with AUA with 3-year incidence of 9.9%. The most common biopsy finding was IgA Nephropathy (52.3%). Thin membrane disease was common among IMH group, IgA Nephropathy in CHP group and FSGS in PA group at 67.7%, 70.8%, and 51.5%, respectively. Among the TBMD, supportive treatment such as ACE-I/ARB were given while prednisone ± ACE-I/ARB were given to IgA Nephropathy and FSGS. Due to the nature of the study in addition to high number of lost to follow-up and short duration of follow-up, treatment and renal outcome cannot be ascertained.

**Conclusion**: Asymptomatic urinary abnormality has incidence of 9.9% among Adult Filipino patients. There is a female preponderance among those who presented with IMH and CHP while PA is common among males. IgA Nephropathy, Thin Basement Membrane Disease and FSGS are the most common biopsy findings. Benign urinary findings as it may seem, renal graft biopsy is still indicated in AUA especially among patients with proteinuria since it can affect renal outcome over time.

**Keywords:** Benign urinary abnormalities; Asymptomatic proteinuria; Hematuria syndrome; Abnormal urinalysis

## Introduction

Urinalysis is a simple but very important laboratory test that plays a central role in the diagnosis of kidney disease. Abnormal findings, such as microscopic hematuria with or without proteinuria, on routine urinalysis in an otherwise healthy patient may be the first evidence of an underlying kidney problem. Primary chronic glomerulonephritis usually presents initially with asymptomatic urinary abnormalities (AUA), and chronic glomerulonephritis (GN) is among the leading cause of end-stage renal disease worldwide. Prevalence and natural history of proteinuria and/or hematuria in adults are poorly understood because of the high detection rate [1]. When genitourinary and non-renal diseases are eliminated in these patients, indication for renal biopsy is considered. Renal biopsy remains a useful diagnostic tool in patients with AUA. Because it is quite invasive, some nephrologists are reluctant to perform it especially when the renal function is normal. Thus, renal biopsy is not routinely indicated in these cases and the decision to do such varies in different parts of the country. Histological findings in patients with AUA are highly diverse and some may be more severe than those that may be expected from the clinical and laboratory findings [2-4].

Most of the previous studies done on AUA were focused on isolated microscopic hematuria and some were in both isolated microscopic hematuria with concomitant proteinuria. In both groups, the most common diagnoses on renal biopsy were: IgA nephropathy, thin-basement membrane disease (TBMD), minor glomerular disease, mesangioproliferative disease and focal segmental glomerulosclerosis [1,5,6].

Data from Toronto Registry indicates a ten-year risk of deterioration in renal function of zero in IgAN presenting with isolated microscopic hematuria [7,8]. A Spanish study showed excellent prognosis among IgAN patients presenting with normal renal function, persistent microhematuria, and minimal or negative proteinuria [9]. However, cohort study of IgAN from Hong Kong showed a significant risk of proteinuria, hypertension or renal impairment (44%) during a 7-year follow-up after presenting with microscopic hematuria and very low grade proteinuria [10]. In addition, several studies among adults with microscopic hematuria regardless of the exact diagnosis reported a 5-11% risk of proteinuria over approximately 5-year follow-up, and a 13-16% risk of new-onset hypertension [2, 12]. Conversely, 17-36% of the patients in those cohorts had complete regression of microscopic hematuria during the same follow-up period [13].

In one study of patients with microscopic hematuria and proteinuria <2.5 gm/day, 46% had IgN, 7% TBMD, and 26% other patterns of GN. Whereas those with isolated microscopic hematuria, 20% had IgAN and 43% TBMD with no other GN identified [11].

A study on the role of kidney biopsy among prospective kidney donors with AUA showed that TBMD was the most common cause of asymptomatic persistent microscopic hematuria followed by mild mesangiopathy, IgAN, focal segmental glomerulosclerosis (FSGS), and nonspecific interstitial changes. Patients with persistent microscopic hematuria with proteinuria revealed glomerulosclerosis and arteriosclerosis. Thus, patients with minor glomerular lesions, mesangial or interstitial changes were the only ones allowed to donate [6]. Another study showed that persistent hematuria before donation was sustained thereafter. The overall prevalence of persistent hematuria increased to 15.3%, with 8.3% of donors developing persistent proteinuria during a mean follow-up period of 2.3 years post-donation. It was then concluded that potential donors with persistent glomerular hematuria should be excluded from kidney donation [14].

Asymptomatic urinary abnormalities are quite common findings in routine urinalysis in which patients and even doctors would sometimes disregard. Chronic GN, the most common primary renal cause of end stage renal disease, can present with these benign urinary findings. There is paucity of data and studies concerning AUA. The issue whether to do kidney biopsy or not in a patient with AUA is still debatable and practices vary among different institutions.

## Objective

### General objective

» To describe the clinicopathologic profile and renal outcome of patients who presented with AUA.

### Specific objectives

» To determine the incidence of AUA among patients who underwent native kidney biopsies.

» To describe the demographic, clinical features and histopathologic profile.

» To describe the treatment modalities given and renal outcome at 3, 6, 12, 24 and 36-month follow-up.

## Methodology

A retrospective descriptive study which included Filipino patients aged >18 years old admitted as AUA and underwent kidney biopsy from January 1, 2010 to December 31, 2012. Excluded patients were as follows:

» (+) History and treatment of primary GN.

» Secondary causes of glomerular disease such as diabetes mellitus, systemic lupus erythematosus (SLE), Henoch-Schonlein purpura (HSP), etc.

» Evidence of non-glomerular, urologic or extra-renal cause of hematuria.

» eGFR < 60 ml/min.

» Incomplete baseline data.

Patients were divided into 3 groups based on urinalysis findings:

» Isolated microscopic hematuria (IMH).

» Concomitant hematuria with proteinuria (CHP).

» Proteinuria alone (PA).

For uniformity, patients with urine total protein: creatinine ratio (UTPCR) >150 mg/dl were grouped under proteinuria, regardless of dipstick result.

Patients' data were collected and reviewed from Philippine Renal Disease Registry (PRDR) and Medical Records Section. The following were collected: demographic characteristics, duration of symptoms of AUA prior to biopsy, co-morbidities, baseline blood pressure, mean serum creatinine, estimated glomerular filtration rate (eGFR) by CKD Epi, UTPCR and hemoglobin (Hgb).

Renal biopsy specimens were divided into light microscopy, indirect immunofluorescence, and electronic microscope, read by 3 renal pathologists. The amount of tissue was sufficient for all cases i.e., at least 10 glomeruli. The diagnostic criteria of WHO classification of renal diseases were used for pathologic diagnosis.

Patients were followed up from 3 to 36 months post-biopsy noting the treatment given and renal outcome (±1 month for the 3, 6, 12, 24 and 36-month post-biopsy follow-up). The outcome for hematuria was divided into 2 groups: resolution and persistence. The outcome for proteinuria was divided into 3 groups: remission, stable and increased by twofold.

### Definition of terms

- Asymptomatic urinary abnormality (AUA) is defined as urinalysis with microscopic hematuria, with or without proteinuria, or proteinuria only, with normal creatinine and absence of symptoms or systemic illness.
- Pre-biopsy duration is the time from the first abnormal urinary findings to renal biopsy.
- Hematuria is presence of red blood cells (RBC) >5 cells/hpf in routine urinalysis with at least +1 blood on dipstick obtained in at least 2 urinalysis.

### Proteinuria is the presence of protein in urine and classified in to 3

- Borderline proteinuria: trace protein on urine dipstick or urine protein: creatinine ratio 150-300 mg/dl.
- Overt proteinuria: +1 protein on urine dipstick and urine protein: creatinine ratio >300 mg/dl but <3 gm/dl
- Nephrotic range proteinuria: >3 gm/dl.
- Isolated microscopic hematuria is defined as urinalysis with presence of red blood cells >5 cells/hpf with at least +1 blood on dipstick in an asymptomatic patient with normal creatinine.
- Concomitant hematuria and proteinuria is defined as urinalysis with red blood cells of >5 cells/hpf and urine protein: creatinine ratio of >150 mg/dl in an asymptomatic patient with normal creatinine.
- Proteinuria alone is defined as presence of proteinuria with urine protein: creatinine ratio of >150 mg/dl in an asymptomatic patient with normal creatinine

### Treatment response

- Complete Remission as disappearance of microscopic hematuria or RBC <5 cells/hpf and disappearance or at least trace proteinuria on dipstick, or UTPCR <0.15.
- Partial Remission as at least 50% improvement in proteinuria or hematuria or both.

- Persistent as continuous RBC >5 cells/hpf and proteinuria >300 mg/dl despite optimal treatment.
- Relapse as recurrence of RBC >5 cells/hpf and/or proteinuria >300 mg/dl after 3 months of documented remission.
- Chronic kidney disease is eGFR <60 ml/min for >3 months.
- End stage renal disease (ESRD) is eGFR <15 ml/min or need for renal replacement therapy.

### Statistical analysis

Demographics and Clinical profile were described using estimates of central tendencies (mean, median). Means and standard deviation were computed for scale variables. Frequencies and percentages were used to show distribution of categorical variables

### Ethical consideration

This study was approved by NKTI Research Ethics Committee. Confidentiality and anonymity was maintained by assigning a specific number to each of the subjects and allowing only investigators to access the data.

## Results

Around 1,550 patients were admitted for native kidney biopsy during the study period and 153 patients presented with AUA.

### Incidence, demographic and baseline clinical characteristics

Asymptomatic urinary abnormality had a yearly incidence of 10.4%, 11.6% and 7.5%, in 2010, 2011 and 2012 respectively with a 3-year incidence of 9.9%.

Among these 153 patients, majority was female (55.6%) with mean age of 37 ± 11.2 years old and mean pre-biopsy duration of 22 ± 33.5 months. Around 58.2% had CHP, 21.5% had PA and 20.3% presented with IMH (Table 1).

There is a female preponderance among those who presented with IMH and CHP while PA is common among males. Patients who presented with PA had higher mean serum creatinine and lower eGFR compared with other groups.

### Renal histopathologic profile

The dominant pathologic diagnoses were IgAN, TBMD, and FSGS as shown in table 2. Thin Basement Membrane Disease was the most common biopsy finding in IMH group while IgAN among CHP group. The FSGS biopsy finding was dominant among the PA group.

Among patients with IgA nephropathy, majority were under sub-class III (focal proliferative disease) and sub-class IV (diffuse proliferative disease).

IgAN, TMBD, FSGS and Minor Glomerular Disease were the most common biopsy findings among middle age group (31-60 years old) while MCD among younger age group (>18 to 30 years old).

There was no difference in the incidence of biopsy findings between genders except TBMD which is commonly seen among female.

### Post-Biopsy course

Among the 153 patients included in the study, 74 (48.4%) were lost to follow-up post-biopsy. Around 79 (51.6%) had follow-up of ≥ 3 months hence only these patients' treatment modalities and renal outcome were reviewed. Table 3 shows the demographic characteristics.

For these 79 patients, 65 (82.3%), 54 (68.3%), 48 (60.8%), and 45 (29.4%) patients completed the 3, 6, 12, 24 and 36-month follow-up respectively. Lost to follow up were included in the non-responded to treatment group.

### Treatment Modalities and Renal Outcome

**Isolated microscopic hematuria**: Among the 15 patients in IMH group, 3 had IgAN, 10 TBMD, and 2 with FSGS. Majority was given ACE-I/ARB and the response was partial or persistence of the IMH as shown in table 4.

### Proteinuria

For patients with CHP, renal outcome was focused on proteinuria since it has more impact in the progression of chronic kidney disease. There were 64 patients with proteinuria (CHP and PA) that were followed-up.

However, due to poor follow-up, treatment outcome cannot be ascertained (Table 5).

Among 37 patients who completed the study follow-up period up for 36 months, 5 had renal insufficiency: 3 out of 5 had IgA nephropathy given prednisone and ACE-I/ARB, 1 had membranous nephropathy given prednisone alone and 1 FSGS given supportive therapy alone. None progressed to ESRD.

## Discussion

In our study, 1 out of 10 patients had AUA as indication for renal biopsy. Different centers and attending physicians have different practices as to the threshold of doing renal biopsy in patients presenting urinary abnormalities with or without accompanying symptoms. The mean pre-biopsy duration of 22 months may either reflect lack of regular screening, late referral to nephrologists or disregard of asymptomatic abnormalities.

Most patients with AUA were relatively young with a mean age of 37 and most common biopsy finding was IgAN (52.3%). These findings are similar with other studies [1,4,5,15]. However, this study, around 52.5% were female as opposed to the male predominance that occurs in IgAN [16,17]. Moreover, stratified according to age group, all of the 3 most common biopsy findings i.e. IgAN, TBMD and FSGS occurred in the middle age group, i.e. 31-60 years old.

TBMD, the second most commonly observed biopsy finding, was the dominant pathology in patients with IMH consistent with other studies [6,11,12]. FSGS, which usually presents with proteinuria more than hematuria, was consistent in this study at 48.5%.

Among those who presented with IMH in this study, TBMD was the most common biopsy finding (67.7%). Majority of patients with TBMD biopsy findings were not given treatment or just started on ACE-I/ARB and were noted to have lost to follow within 3 months post-biopsy. This might be due to the benign nature of the disease as explained by the attending physician. Despite no clear evidence-based treatment protocols available for TBMD, patients should be monitored for the appearance of hypertension, or proteinuria [18].

IgAN can also present as IMH, occurring around 25.8% in this study. Isolated persistent hematuria can also be associated with progressive disease over time. A study among patients with asymptomatic IMH found that ESRD occurred significantly more often than in those without hematuria on mean follow-up of 16 years [19]. This reiterates the importance of long-term monitoring.

For those patients having proteinuria in this study, the most common biopsy findings were IgAN (70.8%) for proteinuria with hematuria and FSGS (51.5%) for proteinuria alone. Despite having

**Table 1:** Baseline demographic and clinical profile of patients with AUA.

| Variables | | Total | IMH | CHP | PA |
|---|---|---|---|---|---|
| | | n=153 | 31 (20.3%) | 89 (58.2%) | 33 (21.5%) |
| Age (years) | Mean ± SD | 37 ± 11.2 | 42 | 36 | 35 |
| | Range | 18-70 | 23-70 | 18-62 | 18-60 |
| Gender | Male | 68 (44.4%) | 5 (16.1%) | 42(47.2%) | 21 (63.6%) |
| | Female | 85 (55.6%) | 26 (83.9%) | 47 (52.8%) | 12 (36.4%) |
| Pre-biopsy duration (months) | Mean ± SD | 22.7 ± 33.5 | 24.3 | 19 | 26.8 |
| Systolic Blood Pressure | Mean, SD | 121 ±11.3 | 112 | 120 | 125 |
| | Range | 90-150 | 100-140 | 90-140 | 100-150 |
| Diastolic Blood Pressure | Mean, SD | 79 ± 9.2 | 76 | 80 | 81 |
| | Range | 50-100 | 60-100 | 60-100 | 50-100 |
| UTPCR | Mean, SD | 1.35 ± 1.6 | 0.1 | 1.7 | 1.8 |
| | Range | 0.01-11.5 | 0.01-0.1 | 0.2-8.3 | 0.3-11.5 |
| Red blood cells/hpf on urinalysis | Mean, SD | 20.1 ± 45.1 | 16.5 ± 13.8 | 29.1 ± 52.2 | 1.7 ± 1.2 |
| | Range | 0-100 | May-54 | 5-486 | 0-4 |
| Scrum creatinine (mg/dl) | Mean, SD | 0.91 ± 0.2 | 0.8 | 0.8 | 1.1 |
| | Range | 0.4-1.5 | 0.4-1.2 | 0.5-1.4 | 0.4-1.5 |
| eGFR (ml/min) | Mean, SD | 93.8 ± 20.8 | 103 | 97 | 85.5 |
| | Range | 60.7-136.6 | 62.5-127.6 | 62.5-136.6 | 62.8-119 |
| Hemoglobin (gm/dl) | Mean, SD | 13.6 ± 1.4 | 12.9 | 12.7 | 14.3 |
| | Range | 9.9-17 | 11.3-14.6 | 9.9-16.2 | 11.2-17 |
| | Range | 0.6-2.8 | 0.9-2.5 | 0.9-2.5 | 0.6-2.8 |
| C3 level | Mean | 1.2 ± 0.3 | 1.3 | 1.3 | 1.4 |
| | Range | 0.6-2.8 | 0.9-2.5 | 0.9-2.5 | 0.6-2.8 |

**Table 2:** Renal Histopathologic Diagnosis.

| Biopsy Findings | | Total | WI | CHP | PA |
|---|---|---|---|---|---|
| | | N=153 | 31 (20.3%) | 89 (58.2%) | 33 (21.5%) |
| IgA Nephropathy | Total | 80 (52.3%) | 8 (25.8%) | 63 (70.8%) | 9 (27.3%) |
| | I | 4 (5%) | 1 (1.3%) | 3 (3.7%) | 0 (0) |
| | II | 8 (10%) | 2 (2.5%) | 5 (6.2%) | 1 (1.2%) |
| | III | 37 (46.3%) | 4 (5%) | 28 (35%) | 5 (6.3%) |
| | IV | 30 (37.5% | 1 (1.3%) | 23 (28.7%) | 6 (7.5%) |
| | V | 1 (1.2%) | 0 | 1 (1.3%) | 0 |
| Thin Basement Membrane Disease | | 29 (19%) | 21 (67.7%) | 8 (8.9%) | 0 |
| Focal Segmental Glomerulosclerosis | | 27 (17.6%) | 2 (6.5%) | 8 (8.9%) | 17 (51.5%) |
| Minor Glomerular Disease | | 8 (5.2%) | 0 | 4 (4.5%) | 4 (12.1%) |
| Immune Complex Glomerulonephritis | | 3 (2.0%) | 0 | 3 (3.4%) | 0 |
| Membranous Glomerulopathy | | 2 (1.3%) | 0 | 2 (2.2%) | 0 |
| Minimal Change Disease | | 2 (1.3%) | 0 | 0 | 2 (6.1%) |
| Mosangloprollierative Glornerulonaphritis | | 2 (1.3%) | 0 | 1 (1.1%) | 1 (3.0%) |

proliferative subclass III and IV with ≤ 22% global sclerosis on biopsy among the IgAN, 93% presented with non-nephrotic proteinuria. A study of 73 patients with persistent proteinuria and a normal or near-normal initial serum creatinine with repeat renal biopsies performed at five years, there was 4% histologic improvement, 41% were stable and 55% showed progressive glomerular and secondary vascular and tubulo interstitial injury [20]. Therefore, patients should be monitored because a stable creatinine does not necessarily indicate stable disease. In this study, most of the patients were given prednisone ± ACE-I/ARB with 54% remission but with high number of lost to follow up.

FSGS, the 3rd most common pathology in this study, is not a benign glomerulonephritis as it may present asymptomatically. The level of proteinuria has been known to have prognostic significance [21-23]. More than 50% of patients with nephrotic range proteinuria progress to ESRD over 5-10 years, however, entering remission have an excellent prognosis with a 10-year survival of >90% compared with <35% in patients not attaining remission [23-27]. Even partial remission portends a good prognosis with a 10-year survival of approximately 75%. Spontaneous remission is rare in nephrotic FSGS occurring in <5% of patients [26].

**Table 3:** Baseline Clinical profile of patient with follow up.

| Variables | | Total n=79 | IMH 15 (19.0%) | CHP 04 (55.7%) | PA 20 (25.3%) |
|---|---|---|---|---|---|
| Age (years) | Mean | 39 | 45 | 38 | 37 |
| | Range | 18.7 | 25.7 | 21-62 | 18.6 |
| Gender | Male | 39 (49.4%) | 4 (26.7%) | 21 (46.7%) | 14 (73.7%) |
| | Female | 40 (50.6% | 11 (73.3%) | 24 (53.3%) | 5 (26.3%) |
| Co-morbidities | HPN | 32 (66.7%) | 5 (15.6%) | 17 (53.1%) | 10 (31.3%) |
| | DM | 5 (10.4%) | 0 (0%) | 0 (0%) | 5 (19.2%) |
| | Asthma/ COPD | 3 (6.3%) | 0 (0%) | 0 (0%) | 3 (11.5%) |
| | CAD/HF | 2 (4.2%) | 2 (16.7%) | 0 (0%) | 0 (0%) |
| | None | 1 (2.1%) | 0 (0%) | 0 (0%) | 1 (3.8%) |
| | Others | 23 (47.9%) | 7 (58.3%) | 5 (50%) | 11 (42.3%) |
| Pre-biopsy duration (months) | Mean | 26.1 | 26.4 | 23.6 | 31.89 |
| Systolic Blood Pressure | Mean | 121 | 109 | 122 | 126 |
| | Range | 11-160 | 11-130 | 100-160 | 100-150 |
| Diastolic Blood Pressure | Mean | 80 | 78 | 79 | 82 |
| | Range | 50.1 | 70.9 | 60-100 | 50-100 |
| UTPCR | Mean | 1A | 0.1 | 1.6 | 2.2 |
| | Range | 0.02-11.5 | 0.02-0.1 | 0.2-5.3 | 0.3-11.5 |
| Serum creatinine (mg/d1) | Mean | 0.92 | 0.76 | 0.91 | 1.09 |
| | Range | 0.4-1.5 | 0.5-1.1 | 0.6-1.5 | 0.4-1.5 |
| eGFR (ml/min) | Mean | 94.2 | 101 | 95.4 | 86 |
| | Range | 45.2-133 | 74-118.2 | 61-133 | 45.2-119.8 |
| Hemoglobin (gm/d1) | Mean | 13.7 | 12.9 | 13.7 | 14.5 |
| | Range | 11.2-17 | 11.3-14.2 | 11.2-15.7 | 11.7-17 |
| | Range | 0.6-2.8 | 0.9-1.5 | 0.8-1.9 | 0.6-2.8 |

**Table 4:** Biopsy Findings of Isolated Microscopic Hematuria in Relation to Treatment Given and Outcome.

| Biopsy Findings | Treatment | Treatment Response | Follow-up Period (months) | | | | |
|---|---|---|---|---|---|---|---|
| | | | 3 | 6 | 12 | 24 | 36 |
| | | | (n=15) | (n=10) | (n=8) | (n=8) | (n=8) |
| IgA Nephropathy (n=3) | ACE-I/ARB (n=3) | Complete | 1 | 1 | 1 | 1 | 1 |
| | | Partial | 1 | | | | |
| | | Persistent | 1 | | | | |
| Thin Basement Membrane Disease (n=10) | ACE-I/ARB (n=4) | Complete | | 1 | 1 | 1 | 1 |
| | | Partial | 1 | | | | |
| | | Persistent | 3 | 2 | | | |
| | None (n=6) | Complete | 1 | 1 | 1 | 2 | 2 |
| | | Partial | 1 | 1 | 1 | 1 | 1 |
| | | Persistent | 4 | 2 | 2 | 1 | 1 |
| Focal Segmental Glomerulosclerosis (n=2) | Prednisone (n=1) | Partial | 1 | 1 | 1 | 1 | 1 |
| | ACE-I/ARB (n=1) | Persistent | 1 | 1 | 1 | 1 | 1 |

**Table 5:** Biopsy Findings of CHP and PA in Relation to Treatment Given and Outcome.

| Biopsy findings | Treatment | Treatment response | Follow-up Period (months) | | | | |
|---|---|---|---|---|---|---|---|
| | | | 3 | 6 | 12 | 24 | 36 |
| | | | (n=64) | (n=55) | (n=46) | (n=39) | (n=37) |
| IgA Nephropathy (n= 37) | Prednisone(n= 3) | Complete | | 1 | 1 | 1 | |
| | | Partial | | 1 | 2 | 2 | 2 |
| | | Persistent | 3 | 1 | | | |
| | ACE-I/ARB (n=17) | Complete | 4 | 2 | 2 | 3 | 3 |
| | | Partial | 2 | 8 | 5 | 5 | 5 |
| | | Persistent | 10 | 4 | 3 | 1 | 1 |
| | | Increased | 1 | 1 | 1 | 1 | 1 |
| | Prednisone + ACE–I /ARB (n=15) | Complete | | 2 | 2 | 3 | 3 |
| | | Partial | 8 | 7 | 6 | 5 | 6 |
| | | Persistent | 7 | 4 | 3 | 3 | 2 |
| | None (n=2) | Partial | 1 | 1 | 1 | 1 | 1 |
| | | Persistent | 1 | | | | |
| Thin Basement Membrane Disease (n=7) | ACE-I/ARB (n=4) | Complete | | 3 | 3 | 3 | 2 |
| | | Partial | 2 | | 1 | | |
| | | Persistent | 2 | 1 | | | |
| | Prednisone + ACE-I/ARB (n=1) | Partial | 1 | 1 | | | |
| | None (n=2) | Persistent | 2 | | | | |
| | | Increased | | 1 | | | |
| Focal Segmental Glomerulosclerosis (n=10) | Prednisone (n=1) | Partial | 1 | 1 | 1 | 1 | 1 |
| | ACE-I/ARB (n=3) | Complete | | | | | 1 |
| | | Partial | 3 | 2 | 2 | 2 | 1 |
| | Prednisone + ACE-I/ARB + others (n=5) | Complete | | 1 | 1 | 1 | |
| | | Partial | 3 | 3 | 1 | 1 | 2 |
| | | Persistent | 2 | 1 | 2 | 1 | 1 |
| | None (n=1) | Partial | 1 | 1 | 1 | | |
| Minor Glomerular Disease (n=5) | ACE-I/ARB (n=3) | Partial | 1 | | | | |
| | | Persistent | 2 | 2 | 2 | 2 | 2 |
| | Prednisone + ACE-I/ARB (n=2) | Partial | 1 | 1 | 2 | | |
| | | Persistent | 1 | 1 | | | |
| Membranous Glomerulopathy (n=2) | Prednisone (n=1) | Persistent | 1 | 1 | 1 | 1 | 1 |
| | Prednisone + ACE-I/ARB (n=1) | Complete | | | 1 | | |
| | | Partial | 1 | 1 | | | |
| Minimal Change Disease (n=1) | ACE-I/ARB (n=1) | Persistent | 1 | 1 | 1 | 1 | 1 |
| Mesangioproliferative Glomerulonephritis (n=2) | ACE-I/ARB (n=2) | Partial | 2 | 1 | 1 | 1 | 1 |

Proteinuria was proven as risk factor for progression of renal disease and blood pressure control affects the outcome [27].

Due to the nature of the study in addition to high number of lost to follow-up and short duration of follow-up, treatment and renal outcome cannot be ascertained.

## Conclusion

Asymptomatic urinary abnormality has incidence of 9.9% among Adult Filipino patients. There is a female preponderance among those who presented with IMH and CHP while PA is common among males.

IgA Nephropathy, Thin Basement Membrane Disease and FSGS are the most common biopsy findings. Benign urinary findings as it may seem, renal graft biopsy is still indicated in AUA especially among patients with proteinuria since it can affect renal outcome over time.

**Disclosure:** None

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