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# KIDNEY BIOPSY IN ADULT IRAQI PATIENTS WITH RENAL IMPAIRMENT. A RETROSPECTIVE STUDY IN SINGLE CENTER IN MEDICAL CITY IN BAGHDAD – IRAQ 2024

**[*1]Dr. Abbas Fadhil Shakir Alshalah, [2]Dr. Adnan Abduladheem Aljber, [3]Dr. Yasir Saad Jasim**

[1]C. A. B. M. S. (Medicine), C. A. B. M. S. (Nephro), M. B. Ch. B.
[2]FICM med. FICM Neph, Consultant Nephrology.
[3]M.B.Ch.B., C.A.B.M.S (Med), C.A.B.M.S. (Nephro).

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***Corresponding Author: Dr. Abbas Fadhil Shakir Alshalah**
C. A. B. M. S. (Medicine), C. A. B. M. S. (Nephro), M. B. Ch. B.
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## ABSTRACT

**Introduction:** Acute and progressive chronic kidney diseases are subject to a variety of inflammatory and autoimmune processes, which are often accompanied by degenerative lesions or are also genetically determined. In many cases, the underlying causes cannot be distinguished clinically, nor can they be identified by advanced laboratory tests because they remain confined to the renal parenchyma. Therefore, a renal biopsy is indicated when knowledge of the histological diagnosis is essential for appropriate therapy. Despite advances in non-invasive biochemical and imaging investigations, kidney biopsies play a pivotal role in the diagnosis of kidney disease, so the available evidence suggests that the histological diagnosis of both native and transplanted kidney biopsies has a direct therapeutic impact or significantly influences the patient's further treatment in about 40–60% of cases. **Method:** A retrospective study design includes the native and transplanted kidney biopsies performed at the Renal disease and kidney Transplant center in medical city during the 2024. A total of 100 cases of different age groups were biopsied and included in this study. The indications for renal biopsy included patients with, nephritic syndrome, renal insufficiency (failure) due to an unknown etiology, nephrotic syndrome and asymptomatic urinary abnormality. The biopsy procedure followed an established operative protocol**. Results:** Distribution of diagnosis of patients according to histological finding, FSGS was the highest pathological lesion (18%) encountered in our study. Immunoglobulin A glomerulonephritis (IGA GN) was found to be the second frequent pathology in our study 14%. Lupus nephritis (LN) (12%) and Membranous glomerulonephritis (MGN)(12%) were represents the third most frequent diagnosis among patients with primary glomerular diseases in our study.. The other pathological causes of primary renal disease in our study were: diabetic nephropathy (DN)(3%), Arteriosclerosis (2%), minimal change disease (MCD)(2%), light chain deposition disease (LCDD), (2%)Arterionephroseclrosis (2%), Pausi immune (2%), Amyloidosis (2%),CNI toxicity(2%), Acute tubular injury (ATI)(2%), Normal(2%), Collagen fibrotic glumerularpathy (1%0), Crystalline nephropathy (1%), C3 glumerulopathy (C3GN)(1%). While in transplanted kidney patients the first pathological cause was chronic active antibody mediated rejection (5%) then the second cause was acute T-cell mediated cellular rejection ATMR(4%), while the Focal segmental glumeruloseclorosis (FSGS) was the third cause (3%), and the other pathological causes were: Acute tubular necrosis (ATN) (2%), Immunoglobulin A glomerulonephritis (IGA GN) (2%), CNI toxicity (1%), B.K nephropathy (1%), Acute tubular injury (ATI)(1%), Acute T-cell mediated cellular rejection ATMR+ Immunoglobulin A glomerulonephritis (IGA GN) (1%). **Conclusion:** Renal biopsy still a gold stander in the diagnosis and management of different renal diseases. It has an essential role in taking histopathological information that cannot be obtained from clinical examination or imaging alone, by renal biopsy we know classification of glomerular, tubular, interstitial, and vascular pathologies, also can follow appropriate treatment strategies and guess prognosis.

**KEYWORDS:** renal biopsy, glomerulonephritis, proteinuria, hematuria, acute kidney injury.

## 1-INTRODUCTION

Acute and progressive chronic kidney diseases are subject to a variety of inflammatory and autoimmune processes, which are often accompanied by degenerative lesions or are also genetically determined. In many cases, the underlying causes cannot be distinguished clinically, nor can they be identified by advanced laboratory tests because they remain confined to the renal parenchyma. Therefore, a renal biopsy is indicated when knowledge of the histological diagnosis is essential for appropriate therapy.[1,2] Nevertheless, there is general agreement that the biopsy findings should always be viewed and interpreted in the context of clinical and historical data. In addition to histological diagnosis, a renal biopsy also allows the prognosis of underlying renal disease to be assessed. However, the advantages of histological diagnosis must always be weighted against the possible risks due to the invasive nature of the procedure.[3,4] Despite advances in non-invasive biochemical and imaging investigations, kidney biopsies play a pivotal role in the diagnosis of kidney disease, so the available evidence suggests that the histological diagnosis of both native and transplanted kidney biopsies has a direct therapeutic impact or significantly influences the patient's further treatment in about 40–60% of cases.[5,6] Biopsy can be used to diagnose relapses or progression of disease. Histological transformation can occur with relapses, potentially changing the treatment required and prognosis.[7,8] Furthermore, relapses are an independent predictor of progression to ESKD.[9] In patients where relapse is suspected, there is a low threshold for repeat biopsy.[10]. There are no accurate clinical predictors of class transformation, reinforcing the value in histologically restaging the disease to guide immunosuppression and inform the risk of progression to ESKD.[11,12] Refinement of biopsy technique and interpretation skills led to major development and a paradigm shift toward incorporation of modern molecular techniques. This was due to the explosion in kidney disease research in the last 20-30 years that brought new knowledge from bench to bedside and resulted in new molecular and genetic tools that enhance the diagnostic and prognostic power of the renal biopsy. These genomic technologies show a useful adjunct to the renal biopsy, and provide examples of how these may transform pathologic interpretation into molecular disease phenotypes.[13]

### Clinical Presentations of Glomerular Disease[14]

**1-Asymptomatic**

Proteinuria 150 mg to 3 g per day, Hematuria >2 red blood cells per high-power field in spun urine or >10 × 106 cells/L (red blood cells usually dysmorphic).

**2-Macroscopic Hematuria**

Brown/red painless hematuria (no clots); typically coincides with intercurrent infection, Asymptomatic hematuria ± proteinuria between attacks.

**3-Nephrotic syndrome Proteinuria:** adult >3.5 g/day; child >40 mg/h/m2, Hypoalbuminemia <3.5 g/dL Edema, Hypercholesterolemia Lipiduria.

**4-Nephritic syndrome Oliguria**

Hematuria: red cell casts Proteinuria: usually <3 g/day Edema
Hypertension
Abrupt onset, usually self-limiting

**5-Rapidly progressive glomerulonephritis**

Renal failure over days/weeks Proteinuria: usually <3 g/day
Hematuria: red cell casts.

Kidney biopsy is generally required to establish the type of glomerular disease and to guide treatment decisions. In some patients, however, kidney biopsy is not performed. If nephrotic children (ages 2–12) have no unusual clinical features, the probability of MCD is so high that corticosteroids can be initiated without biopsy. In patients with acute nephritic syndrome, if all features point to poststreptococcal, especially in an epidemic, biopsy can be reserved for those without early spontaneous improvement. In Goodpasture disease, the presence of lung hemorrhage and rapidly progressive kidney failure with urinary red cell casts and high levels of circulating anti-GBM antibody establishes the diagnosis without the need for a biopsy. In patients with systemic features of vasculitis, a positive ANCA titer, negative blood cultures, and a biopsy specimen from another site showing vasculitis are sufficient to secure a diagnosis of renal vasculitis. However, even when kidney biopsy is not needed for diagnosis, it may provide important clues to disease activity and chronicity. Biopsy is also not generally performed in patients with long-standing diabetes with characteristic findings suggestive of diabetic nephropathy and other evidence of microvascular complications of diabetes. Biopsy may not be indicated in many patients with glomerular disease presenting with minor, asymptomatic urine abnormalities and well-preserved kidney function because the prognosis is excellent and histologic findings will not alter management.[14] Kidney biopsy is a useful diagnostic tool in both the acute and chronic setting, especially in the presence of active urinary sediment. However, active urinary sediment does not confirm intrinsic kidney disease and biopsy poses unnecessary risk in cases of pre-renal and post-renal causes of kidney impairment. Furthermore, an active urine sediment is not a prerequisite for additional investigation; kidney biopsy may also be appropriate in cases of bland urinary sediment, for instance, non-recovering AKI and suspected tubulointerstitial nephritis.[15]

Therefore should know the indications and contraindications of kidney biopsy as showen in box 1and 2 below.

**BOX 1 Indications for Kidney Biopsy[4]**

| | |
|---|---|
| Nephrotic Syndrome | • Routinely indicated in adults • In prepubertal children, indicated only if clinical features atypical of minimal change disease are present. |
| Acute Kidney Injury | Indicated if obstruction, reduced kidney perfusion, and acute tubular necrosis have been ruled out |
| Systemic Disease With Kidney Dysfunction | • Indicated in patients with small-vessel vasculitis, anti–glomerular basement membrane disease, and systemic lupus<br>• Indicated in patients with diabetes only if atypical features present |
| Nonnephrotic Proteinuria | May be indicated if proteinuria is >1 g/24 h |
| Isolated Microscopic Hematuria | • Indicated only in unusual circumstances |
| Unexplained Chronic Kidney Disease | •May be diagnostic (e.g., identify immunoglobulin A nephropathy even in "end-stage kidney") |
| Familial Kidney Disease | • Biopsy of one affected member may give diagnosis and minimize further investigation of family members |
| Kidney Transplant Dysfunction | • Indicated if ureteral obstruction, urinary sepsis, renal artery stenosis, and toxic calcineurin inhibitor levels are not present |

**BOX 2 Contraindications to Kidney Biopsy**

| | |
|---|---|
| Kidney Status | .Multiple cysts<br>.Solitary kidney<br>.Acute pyelonephritis. Perinephric abscess<br>. Kidney neoplasm |
| Patient Status | .Uncontrolled bleeding diathesis<br>.Uncontrolled blood pressure. Uremia Obesity<br>.Uncooperative patient |

**So about the finding of biopsy about glomerular diseases**

**1-acute renal failure (ARF):** describes the clinical syndrome in which an abrupt (hours to days) decrease in kidney function leads to the accumulation of nitrogenous waste products and, commonly, a reduction in urine output. Acute kidney injury (AKI) has become the consensus term for ARF. This change in terminology served to standardize a definition for the syndrome, as well as to incorporate knowledge that increases in serum creatinine as small as 0.3 mg/dL (27 μmol/L) are associated with increased morbidity and mortality. AKI has since been defined as an increase in serum creatinine of 0.3 mg/dL or greater within 48 hours of observation or 1.5 or more times baseline, which is known or presumed to have occurred within 7 days, or a reduction in urine volume below 0.5 mL/kg/h for 6 hours. Sub classification of AKI based on disease severity as indicated by the level of increase in serum creatinine and reduction in urine output has been adopted as a three-stage classification by KDIGO. Increasing severity of AKI based on creatinine and urine output associates with increased risk for death. There are many different causes of AKI, and most are identified through clinical investigation. Globally, hypotension and volume contraction or dehydration accounted for 40% of AKI cases in both the hospital and nonhospital setting. In higher-income countries, hypotension and shock were the most common causes of AKI, whereas volume contraction or dehydration predominated in lower-income countries. Nephrotoxic agents were implicated in up to a quarter of AKI events across all countries. However, nephrotoxin administration may account for a larger proportion of hospital-acquired AKIs in older patients. Kidney biopsy is rarely performed to establish the cause of AKI.[14]

**2-Chronic kidney disease CKD:** is defined as abnormalities of kidney structure or function, present for at least 3 months, with implications for health The relevant Kidney Disease: Improving Global Outcomes (KDIGO) guideline recommends classification of CKD based on cause, category of glomerular filtration rate (GFR), and albuminuria. Because of the impracticalities of using radioisotopes and 24hour urine collections, the KDIGO classification system recommends that kidney function be assessed by estimating GFR (eGFR) from the serum creatinine concentration using an appropriate equation, except in circumstances in which eGFR estimations are known to be less accurate, such as when there is significant muscle wasting. Initially, the Modification of Diet in Renal Disease (MDRD) equation was used, but this has predominantly been replaced by the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation, which more accurately categorizes the risk for mortality and progression to ESKD (Recently, this formula has been updated in order to eliminate race from the equation KDIGO-GFR_calculator/formula), and although the formula is less precise than the earlier CKD-EPI equation, it is sufficiently accurate for clinical practice. Although staging systems for CKD based on eGFR have limitations, they have proved useful in many clinical settings and are now deeply embedded in KDIGO and other guidelines developed for CKD management and in research.[14]

**3- Minimal change disease (MCD):** is the most common cause of nephrotic syndrome in children (approximately 80%) and accounts for 10% to 20%of cases in adults. MCD is so called because on kidney biopsy, glomeruli are normal or near normal on light microscopy, with podocyte foot process fusion or effacement found on electron microscopy. Compared to other causes of nephrotic syndrome, MCD has a higher rate of remission after steroid treatment, with better long-term kidney outcomes. However, MCD still causes significant morbidity, especially in older patients, due to complications such as acute kidney injury (AKI) and thrombosis. A high proportion of patients with MCD relapse, resulting in the need for multiple courses of steroids or alternative immunosuppressive therapy and significant associated side effects.[14]

**4- Focal segmental glomerulosclerosis (FSGS):** is a histologic pattern of glomerular injury that represents the final common pathway of podocyte injury and depletion. FSGS may be primary (idiopathic) or secondary to diverse. Early in the disease process, the pattern of glomerulosclerosis is focal, involving a minority of glomeruli, and segmental, involving a portion of the glomerular tuft. As FSGS progresses, more diffuse and global glomerulosclerosis evolves. Although it accounts for only a small proportion of nephrotic syndrome in young children, FSGS represents as many as 35% of cases of primary nephrotic syndrome in adults and is a major cause of end stage kidney disease (ESKD) in the United States. FSGS can be caused by a diverse set of pathogenetic mechanisms, some of which manifest as particular histologic subtypes of disease. Although primary (idiopathic) FSGS is potentially treatable and curable in many patients, the optimal type and duration of immunosuppressive, as well as adjunctive therapy, remain controversial. For secondary FSGS, effective therapies exist to slow or modify the disease course.[14]

**5-Membranous nephropathy (MN)**: is an immune complex disease in which deposits of immunoglobulin (Ig)G and complement components develop predominantly or exclusively beneath podocytes on the subepithelial surface of the glomerular capillary wall. Podocyte injury resulting from the immune deposits increases glomerular permeability, which results in proteinuria and potentially in nephrotic syndrome. Primary (formerly called idiopathic) MN is an organ-specific autoimmune disease in which autoantibodies target an intrinsic podocyte antigen. It accounts for about 75% to 80% of patients with MN and typically occurs in the absence of any identifiable initiating event. It is the most common cause of primary nephrotic syndrome in older (>60 years) White adults, but the age range is broad and patients may present for the first time as teenagers. Various conditions have been identified in association with MN, some of which are likely to be causal, and are known as secondary MN. The term "membranous" refers to thickening of the glomerular capillary wall on light microscopy of a kidney biopsy. Currently MN is more often defined by immunofluorescence and electron microscopy showing the pathognomonic diffuse, finely granular or electron-dense immune deposits in the subepithelial space. Consequently, MN is a pathologic diagnosis made in patients with proteinuria whose glomeruli exhibit these immune deposits without associated hypercellularity or inflammatory changes.[14]

**6-Immunoglobulin A (IgA) nephropathy (IgAN):** is a mesangial proliferative glomerulonephritis (GN) characterized by diffuse mesangial deposition of IgA. IgAN was first recognized in 1968 by Jean Berger when immunofluorescence (IF) techniques were introduced for the study of kidney biopsy specimens. IgAN is unique among glomerular diseases in being defined by the presence of an immune reactant rather than by any other morphologic feature on kidney biopsy, and the light microscopy changes are variable. IgAN is the most prevalent pattern of glomerular disease seen in most Western and Asian countries where kidney biopsy is widely practiced. It is likely that IgAN is not a single entity but rather a common response to various injurious mechanisms.[14]

**7- Lupus nephritis (LN):** the prototypical immune complex glomerulonephritis (GN), occurs commonly in patients with systemic lupus erythematosus (SLE) and can be very severe. SLE is defined by specific clinical and laboratory features. To be considered as having SLE, a patient must demonstrate antinuclear antibody (ANA) positivity of 1:80 or more using a Hep2 cell or equivalent assay. Other clinical and serologic parameters are assigned points and, in addition to a positive ANA, a patient must have sufficient clinical and laboratory findings to achieve a score of at least 10. Importantly, proteinuria and kidney biopsy findings are heavily weighted. A kidney biopsy showing an immune complex GN consistent with an International Society of Nephrology/Renal Pathology Society (ISN/RPS) class III or IV LN will receive a score of 10, so this plus a positive ANA are together sufficient to establish a diagnosis of SLE and LN.[14]

**8-Diabetic kidney disease:** Thirty percent to 40% of people with type 1 and type 2 diabetes mellitus (DM) in the UK develop CKD and their risk of requiring renal replacement therapy is over three times the general population.[16,17] 'Diabetic kidney disease' refers to the structural and functional changes caused by DM, while 'diabetic nephropathy' refers to specific histological findings on biopsy. Alternative or superadded diagnoses may co-exist (eg hypertensive disease, unresolved AKI or glomerulonephritis).[18] The distinction is important for treatment, prognosis and future transplant decisions. The natural history of diabetic kidney disease in type 1 and type 2 DM is well defined and the development of albuminuria is a consistent predictor for progression to ESKD.[19,20] In most patients, clinical history, course of disease and non-invasive investigations can identify where CKD is likely a consequence of DM. National

Kidney Foundation guidelines advise that CKD can be attributed to DM in the presence of macroalbuminuria or microalbuminuria with retinopathy, or the presence of microalbuminuria in patients with type 1 DM for >10 years.[21] In these instances, the risks of biopsy are thought to outweigh the benefits of a confirmatory diagnosis.

**OBJECTIVES OF OUR STUDY**

1. To asses types of glomerular diseases and their frequency in adult patients.
2. To identify role of renal biopsy in patients with glomerular disease in treatment, prognosis and fate of diseases.
3. To asses the percentage of interstitial fibrosis and tubular atrophy and its association with each glomerular diseases according to result of renal biopsy.
4. To know distribution of glomerular diseases according to age, sex, present of crescent, and present of previous hypertension or not all of these previous four objectives in adults patients in single nephrology center in medical city in Baghdad \Iraq.

## 2-SUBJECTS AND METHODS

### 2-1-Study Design

Retrospective study design.

### 2-2-Study Population

Includes the native and transplanted kidney biopsies performed at the Renal Transplant Centre and the Renal Unit at gazi al Hariri hospital in medical city during the 2024. A total of 100 cases of different age groups were biopsied and included in this study. The indications for renal biopsy included patients with, nephritic syndrome, renal insufficiency (failure) due to an unknown etiology, nephrotic syndrome and asymptomatic urinary abnormality. The biopsy procedure followed an established operative protocol.

### 2-3-Ethical consideration

-Approvals from Ethical Committee in Ministry of health, Arab board and administration from each patient(written consent) after explaining the steps of procedure and the expected complications.

### 2-4- Inclusion criteria

Patients with elevated renal indices plus minus any other indications for renal biopsy.

### 2-5-Exclusion criteria

patients with normal renal indices who need renal biopsy.

### 2-6-Study Procedures

A detailed history and physical examination was undertaken to all patients, serological tests and various investigations, according to hospital policy, were sent; to identify secondary causes of glomerular diseases and other possible risk factors for glomerulonephritis, And then in our center, the kidney biopsy is performed by nephrologist using disposable automated biopsy needle. We use 18-guage needle to minimize bleeding complications as our patient included in our thesis had elevated renal index, although we use 16–guage sometimes when there is no 18-guage. Premedication or sedation was not required. The patient is laid pron, on special modifiable surgical couch that permit us to put the patient in apposition with maximum access to the kidney then we check patients kidneys with ultra sound [right and left] and an indelible pen mark is used to indicate the point of entry of the biopsy needle (usually the left kidney). The skin is sterilized with Butadine 10%solution. Local anesthetic agent (2% Lidocaine [5cc] is infiltrated in the skin at the point previously marked.. with ultrasonic probe on the skin above his left kidney we insert our needle biopsy slowly. sometimes the patient is instructed to take a breath until the kidney is moved to a position such that the lower pole rests just under the biopsy needle and then to stop breathing. The trigger mechanism is released, firing the needle into the kidney. The needle is immediately withdrawn, and probe removed, then the patient asked to resume breathing if he stopped it and the contents of the needle examined. Once sufficient renal tissue has been obtained is placed in formaline for conventional light microscopy and the process above repeated again but now the renal tissue inserted in Michel,s solution for immunofluorescences microscopy, the skin incision is dressed and the patient is rolled directly into bed for observation. All patients who had renal biopsy left the hospital after up to six hours with stable hemodynamic status, no pain or frank hematuria. All patients were advised to avoid any strenuous effort for the next few days.

### 2-7-Data collection & Analysis

a convenient sample of100 patients with elevated renal index and need renal biopsy at the Renal disease and kidney Transplant center in medical city during the 2024. Descriptive analysis performed in summarizing all demographics and clinical characteristics of the patients. The graphical presentation and statistical analysis were conducted via the Statistical Package for the Social Sciences (IBM SPSS version 28).

## 3-RESULTS

**Table 1: Sociodemographic characteristics of patients.**

**A-According to gender**

| Gender | Frequency | Percentage |
|---|---|---|
| Male | 60 | 60% |
| Female | 40 | 40% |
| Total | 100 | 100% |

**B-According to age**

| Age | Frequency | Percentage |
|---|---|---|
| 15-45 | 70 | 70% |
| 45-65 | 25 | 25% |
| Above 65 | 5 | 5% |
| Total | 100 | 100% |

The sociodemographic characteristics show the male predominance and the average age of 15-45 was with highest percent.

**Table 2: Percentage of patients presentation (indication) for renal biopsy (native and Transplanted kidney biopsies**).

| Indications for biopsy | Frequency | Percentage |
|---|---|---|
| Elevated renal indices | 26 | 26% |
| Elevated renal indices and proteinuria | 20 | 20% |
| Elevated renal indices and edema | 17 | 17% |
| Elevated renal indices andSLE | 11 | 11% |
| Elevated renal indices and hematuria | 6 | 6% |
| Elevated renal indices and graft dysfunction | 20 | 20% |
| Total | 100 | 100% |

Elevated renal indices was the highest percent indicator for renal biopsy followed by elevated renal indices and proteinuria and graft dysfunction. while the elevated renal indices and hematuria was the lowest percent of indication for renal biopsy.

**Table 3: Distribution of diagnosis of patients according to histological finding (native and Transplanted kidney biopsies.**

| Renal pathology \native kidney | Frequency | Percentage |
|---|---|---|
| Focal segmental glumeruloseclorosis (FSGS) | 18 | 18% |
| Immunoglobulin A glomerulonephritis (IGA GN) | 14 | 14% |
| Lupus nephritis (LN) | 12 | 12% |
| Membranous glomerulonephritis (MGN) | 12 | 12% |
| diabetic nephropathy (DN) | 3 | 3% |
| Arteriosclerosis | 2 | 2% |
| minimal change disease (MCD) | 2 | 2% |
| Light change deposition disease (LCDD) | 2 | 2% |
| Arterionephroseclrosis | 2 | 2% |
| Pausi immune | 2 | 2% |
| Amyloidosis | 2 | 2% |
| CNI toxicity | 2 | 2% |
| Acute tubular injury (ATI) | 2 | 2% |
| Normal | 2 | 2% |
| Collagen fibrotic glumerularpathy | 1 | 1% |
| Crystalline nephropathy | 1 | 1% |
| C3 glumerulopathy (C3GN) | 1 | 1% |
| Total | 80 | 80% |
| **Renal pathology \transplanted kidney** | **Frequency** | **Percentage** |
| Chronic active antibody mediated rejection | 5 | 5% |
| Acute T-cell mediated cellular rejection ATMR | 4 | 4% |
| Focal segmental glumeruloseclorosis (FSGS) | 3 | 3% |
| Acute tubular necrosis (ATN) | 2 | 2% |
| Immunoglobulin A glomerulonephritis (IGA GN) | 2 | 2% |
| CNI toxicity | 1 | 1% |
| B.K nephropathy | 1 | 1% |
| Acute tubular injury (ATI) | 1 | 1% |
| Acute T-cell mediated cellular rejection ATMR+ Immunoglobulin A glomerulonephritis (IGA GN) | 1 | 1% |
| Total | 20 | 20% |
| Total native and Transplanted kidney biopsies | 100 | 100% |

The Focal segmental glumeruloseclorosis (FSGS) was the highest percent diagnosis 18% in native kidney biopsies. while in transplanted kidney biopsies the Chronic active antibody mediated rejection was the highest percent 5%.

**Table 4: Distribution of patients according to presence or absence of crescent (native and Transplanted kidney biopsies).**

| Crescent \native kidney biopsies | Frequency | Percentage |
|---|---|---|
| Presence of crescent | 15 | 15% |
| Absence of crescent | 65 | 65% |
| Total | 80 | 80% |
| Crescent\transplanted kidney biopsies | Frequency | Percentage |
| Presence of crescent | 2 | 2% |
| Absence of crescent | 18 | 18% |
| Total | 20 | 20% |
| Total native and Transplanted kidney biopsies | 100 | 100% |

The highest percent of patients presented with absence of crescent: in native kidney biopsies (65%) and in transplanted kidney biopsies (18%).

**Table 5: Distribution of patients according to the percent of IFTA (native and Transplanted kidney biopsies).**

| IFTA\ native kidney biopsies | Frequency | Percentage |
|---|---|---|
| Below 30% | 43 | 43% |
| 30-50% | 24 | 24% |
| Above 50% | 13 | 13% |
| Total | 80 | 80% |
| IFTA\ transplanted kidney biopsies | Frequency | Percentage |
| Below 30% | 12 | 12% |
| 30-50% | 6 | 6% |
| Above 50% | 2 | 2% |
| Total | 20 | 20% |
| Total native and Transplanted kidney biopsies | 100 | 100% |

The highest percent of native and transplanted biopsies were with IFTA below 30% (43), (12%) respectively.

**Table 6: Distribution of patients according to the fate of only 92% of patients (native and Transplanted kidney biopsies).**

| Fate \ native kidney biopsies | Frequency | Percentage |
|---|---|---|
| same | 26 | 26% |
| Improved | 24 | 24% |
| CKD | 15 | 15% |
| Hemodialysis | 7 | 7% |
| Total | 72 | 72% |
| Fate \ transplanted kidney biopsies | | |
| Improved | 7 | 7% |
| CKD | 13 | 13% |
| Total | 20 | 20% |
| Total native and Transplanted kidney biobsies | 92 | 92% |

In our study we got the fate only 92% of patients:the same fate were 26% in native kidney biopsies and in transplanted kidney biopsies 13% of patients their fate were chronic kidney disease.

## 4-DISCUSSION

Our study shows a comprehensive evaluation between the demographic parameters and clinical presentation, the patients had an average age of 38.63years and they were older in comparison with patients from other study done in Erbil kidney center: 30.9 years.[22] As showed in table 1(A)there was a slight male predominance in our study as comparable to the other study in Oman where the male-to female ratio was different.[23] Indication for renal biopsy: as showed in Table(2) the higher indication was elevated renal indices only(26%) then elevated renal indices and proteinuria and elevated renal indices with graft dysfunction(20%), this our result when comprise with other study was(19.04 %) and (7.28%) respectively[22] so it was higher. The other indications

were elevated renal indices and hematuria (6%), elevated renal indices and edema (17%) and elevated renal indices and SLE (11%). while the distribution of diagnosis of patients according to histological finding as showed in table (3), FSGS was the highest pathological lesion (18%) encountered in our study which was lower than result in study in single kidney center in Iraq was (33.3%).[24] Immunoglobulin A glomerulonephritis (IGA GN) was found to be the second frequent pathology in the our study 14%, IgAN is the most frequent glomerular disease in Japan and Korea.[25,26] Lupus nephritis (LN) (12%) and Membranous glomerulonephritis (MGN)(12%) were represents the third most frequent diagnosis among patients with primary glomerular diseases in our study. In Iran, MGN was the most frequent renal pathology encountered among patients with primary glomerular disease.[27] This difference in the first, second and third pathological causes of renal diseases between places and countries may be due to genetic, environmental or nutritional reasons. The other pathological causes of primary renal disease in our study were: diabetic nephropathy (DN)(3%), Arteriosclerosis (2%), minimal change disease (MCD)(2%), Light change deposition disease (LCDD), (2%)Arterionephroseclrosis (2%), Pausi immune (2%), Amyloidosis (2%),CNI toxicity(2%), Acute tubular injury (ATI)(2%), Normal(2%), Collagen fibrotic glumerularpathy (1%), Crystalline nephropathy (1%), C3 glumerulopathy (C3GN)(1%). While in transplanted kidney patients the first pathological cause was chronic active antibody mediated rejection (5%) then the second cause was acute T-cell mediated cellular rejection ATMR(4%), The reason for rejection of a transplanted kidney due to antibodies was the highest may be due to the patient's lack of confidence in taking immunosuppressants after the transplant, perhaps severe infection after the transplant., or may be the HLA matching was low. while the Focal segmental glumeruloseclorosis (FSGS) was the third cause (3%), and the other pathological causes were: Acute tubular necrosis (ATN)(2%), Immunoglobulin A glomerulonephritis (IGA GN)(2%), CNI toxicity (1%), B.K nephropathy (1%), Acute tubular injury (ATI)(1%), Acute T-cell mediated cellular rejection ATMR+ Immunoglobulin A glomerulonephritis (IGA GN)(1%). The definition of glomerular crescent as given by the WHO is, two or more layers of cells partially or completely filling the glomerular space, Diffuse crescents when present usually imply a poorer prognosis, however, focal crescents may be reversed if the disease is detected early and could be adequately treated.[28] in our study as showed in table (4)15 out of 80(15%) patients with native kidney disease and 2 out of 20 (2%) of transplanted kidney disease show crescent in biopsy, by comparison with other study[29] 17 out of 40 cases of renal biopsy show crescent in biopsy. IFTA is a strong predictor of ESRD and death, even in proliferative nephritis, and a risk factor for poor outcomes independent of class. Vascular injury is a strong predictor of prognosis, but not independent of serum creatinine and class. The prognostic value of these lesions calls for consideration when determining treatment for lupus nephritis.[30] In our study as showed in table (5): IFTA\ native kidney biopsies below 30% (43%), 30-50% (24%), above 50% (13%), while IFTA\ transplanted kidney biopsies was; below 30% (10%), 30-50% (6%), above 50% (2%).In other study Of those with evidence of IFTA, mild was most common, observed (65%) and moderate or severe IFTA was observed in (35%).[30] We obtained the fate of 92 out of 100 patients as showed in table (6): **Fate \ native kidney biopsies**: Improved (24%), CKD(15%), Hemodialysis(7%), same(26%) and **the fate \ transplanted kidney biopsies**: Improved (7%), CKD(13%).

## 5-CONCLUSION

Renal biopsy still a gold stander in the diagnosis and management of different renal diseases. It has an essential role in taking histopathological information that cannot be obtained from clinical examination or imaging alone, by renal biopsy we know classification of glomerular, tubular, interstitial, and vascular pathologies, also can follow appropriate treatment strategies and guess prognosis. In spite of renal biopsy is invasive procedure with l complications, but its diagnostic role outweighs the risks in many patients. So, renal biopsy is a key tool in the correct diagnosis, management planning, and prognosis of renal diseases.

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