Results of Kidney Biopsies among Adult Iraqi Patients in a Single Center

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Abstract

Background: Patients requiring renal biopsies have various glomerular diseases according to their demographic characteristics.

Objective: To study types of glomerular disease among adult Iraqi patients in a single center in Baghdad/Iraq

Material and Methods: A total of 120 native kidney biopsies were studied. All biopsies were adequate and were processed for Light Microscopy.

The age range of the study patients was 17-67 years, with a mean of 38.5 years. The mean follow up period was 28 weeks (4-52 weeks)

Indication for biopsy included: Nephrotic syndrome (N=72; 60%), Asymptomatic proteinuria (N=21; 17.5%), acute nephritic presentation (N=17; 14.16%), asymptomatic haematuria (N=10; 8.33%).

Results: Primary glomerulonephritis (GN) was seen in 102 of 120 patients (85%), focal segmental glomerulosclerosis, and membranous nephropathy were the most common histological diagnosis (33.3% and 21.5% respectively).

Conclusion: The study further emphasize the need for national GN registry and long term follow up, to recognize the common pattern of GN, their natural history, the appropriate line of management, and to try to halt their progression to end stage renal disease (ESRD).

Key Words: Iraq, Glomerulonephritis, Renal Biopsy.

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Introduction:

Numerous inflammatory and non-inflammatory diseases affect the glomerulus and lead to alteration in glomerular permeability, structure, and function[1]. Many glomerular diseases come under the generic title glomerulnephritis (GN) which implies that there is an immune pathogenesis. [2]

GN may be primary restricted in clinical manifestation to the kidney, or it may be part of multisystem disease, most frequently Systemic Lupus or vasculitis[3]. While the likelihood of a patient having GN can be estimated with varying degree of confidence from the clinical setting and laboratory tests, it cannot be diagnosed without histological examination of cortical renal tissue[4].

GN is classified by the different patterns of histological injury seen on renal biopsy sample examined by light microscopy, immunofluorescence and electron microscopy (EM)[5].

It is more helpful to regard the renal biopsy appearance as a pattern rather than a disease[6]: a pattern that may frequently have a number clinical correlates and a number of clinical putative etiologic agents and that may eventually will prove to have more than one immune mechanism[7].

The classification of GN remains largely based on renal pathology[8]. In GN the dominant but not the only histological lesion are in the glomeruli[9].

Percutaneous renal biopsy was first described in the early 1950s by Iversen and Brun[10] and Alwall[11] . In these reports, the biopsies were performed with the patient in the sitting position using a suction needle. In 1954, Kark and Muehrcke[12] reported a modified technique that used the Franklin-Modified Vim-Silver needle and the patient in prone position[13,14].

Renal biopsy is now able to provide tissue diagnosis in more than 95% of cases with a life threatening complication rate of less than 0.1%[15].

Method:

A total of 120 patients were included in this study. Those patients were referred to Al-Kindy Teaching Hospital-Renal Unit from January to the end of December 2009, from different parts of Baghdad, Kut, Ammara and Najaf; for diagnosis, evaluation, treatment and follow up.

The age range of the patients was 17-67 years with a mean of 39.5 years. The mean follow up period was 28 weeks (4-52 weeks). Indications for biopsy included, nephritic syndrome, asymptomatic proteinuria, acute nephritic presentation, and asymptomatic haematuria. (table 1).

Table (1): Percentage of patients presented for renal biopsy

Presentation	Number of patients	Percentage
Nephrotic syndrome	**72**	**60%**
Asymptomatic proteinuria	**21**	**17.5%**
Acute nephritic syndrome	**17**	**14.16%**
Asymptomatic haematuria	**10**	**8.33%**
Total	**120**	**100%**

In our center, the kidney biopsy is performed by nephrologist using disposable automated biopsy needle. We use 16-guage needle as a compromise between the greater tissue yield of the larger needle and the trend of the fewer bleeding complications[16]. Premedication or sedation was not required.

The patient is laid prone, and a pillow is placed under the. 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 either Butadine or Chlorhexidine solution.

Local anesthetic agent (2% Lidocaine [lignocaine]) is infiltrated in the skin at the point previously marked. A stab incision is made through the dermis to ease the passage of the biopsy needle.

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, the patient asked to resume breathing and the contents of the needle examined.

Once sufficient renal tissue has been obtained, the skin incision is dressed and the patient is rolled directly into bed for observation.

The renal tissue is placed in formaline for conventional light microscopy.

Yet and since the embargo applied on Iraq in 1990, Immunofluorescence (IF) and Electron Microscopy (EM) are not available.

Almost all patients who had renal biopsy left the hospital after few hours with stable haemodynamic status, no pain or frank haematuria. All patients were advised to avoid any strenuous effort for at least the next few days.

Histological classification: The histological classification suggested by the World Health Organization was used in the labeling of L.M. findings, i.e., mild glomerular abnormalities (Mild) FSGS, focal mesangial proliferative glomerulonephritis (FMGN), diffuse mesangial proliferative GN (DMPGN), mesangiocapillary GN (MCGN), membranous nephropathy, and crescentic GN[17].

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.

Results:

The principle indication for renal biopsy was the nephrotic syndrome (NS), which was noted in 72 of 120 study patients (60%). The other indications were asymptomatic, non nephrotic range proteinuria in 21 (17.5%), acute nephritic presentation in 17 (14.16%) and asymptomatic haematuria in 10 (8.33%).

Primary GN was seen in 102 of 120 patients (85%); FSGS was the most common histological diagnosis on LM (34/102), followed by membranous nephropathy (22/102), membranoproliferative GN in 10/102, mesangial proliferative GN 17/102, minimal change disease 19/102.(table 2)

Table (2): Results of renal biopsies of 120 patients.
FSGS: Focal Segmental Glomerulosclerosis, MN: Membranous Nephropathy, MPGN: Membranoproliferative GN, MesPGN: Mesangioproliferative GN, and MCD: Minimal Change Disease.

Primary Glomerulonephritis (GN)	Number(102)	Percentage (85%)
FSGS	**34**	**33.3%**
MN	**22**	**21.5%**
MPGN	**10**	**9.8%**
MesPGN	**17**	**16.6%**
MCD	**19**	**18.6%**
Other Renal pathologies	**Number(18)**	**Percentage (15%)**
Lupus nephritis	**12**	**66.7%**
Amyloidosis	**4**	**22.2%**
Interstitial Nephritis	**2**	**11.1%**

Discussion:

The most frequently diagnosed lesion in patients with primary glomerular disease was FSGS and this represented (33.3%) of the cases. This is similar to the results in Saudi Arabia where FSGS represented (34.9%)[18].

A higher incidence of FSGS had been reported from HongKong (23.6% in adults) and Ghana (36% in adults)[19].

Since Renal Unit in Alkindy Teaching Hospital tends to receive more patients with steroid resistant nephrotic syndrome and probably there is referral bias of biopsying steroid resistant minimal change disease, this could accounts for the high frequency of FSGS.

We believe that patient's selection inherent in the pattern of the referral is probably a significant factor.

Additional epidemiological studies are needed to understand the various factors responsible for the high frequency of FSGS in the country.

The second most common diagnosis in our series was MN which represented (21.5%) of the cases.

This observational study recommends and emphasizes the importance to have a GN registry. This will definitely help in identifying the problem of GN better, and its aetiopathogenesis, and with this knowledge therapeutic and preventive strategies can be outlined so that the progression to ESRD can be stopped or at least slowed down.

Conclusion:

The study further emphasize the need for national GN registry and long term follow up, to recognize the common pattern of GN, their natural history, the appropriate line of management, and to try to halt their progression to end stage renal disease (ESRD).

References:

1. Couser WG. etal. Pathogenesis of glomerular damage in glomerulonephritis. Nephrol Dial Transplant 1998,13(Supplement 1):10-15
2. Mayers BD. etal. Mechanisms of proeinuria in nephritic humans. Pediatr Nephrol 1994;8:107-12.
3. Cameron IS. Systemic Lupus Erythematosus In: Nielson EG. Couser WG. Eds. Immunologic Renal Disease. Philadelphia. Lippincot-Raven 1997: 1055-94
4. Hamburger, Crosmer. Nephrology Hereditary Chronic Nephritis & Variants. 1979; 977-82.
5. Couser WH. Glomerular Disorder. In:Wyngaarden Smith, LH Jr(eds). Cecil Textbook of Medicine JB. 19th ed. Philadelphia. WD Saunders 1992;P.551-68.
6. Couser WG. Glomerular &Vascular Disease. In:Jacobson HR. Striker GE. Klahr S (eds). The Principle and Practice of Nephrology. 2nd ed. St. Louis. Mosby-Yearbook Inc. 1995;P.102-2000.
7. Glassock RJ. Cohen AH. Adier SG. Primary Glomerular Diseases. In: Brenner BM (ed). The Kidney 5th ed. Philadephia. WB Saunders 1996;P.1392-479.
8. Glassock RJ. Cohen AH. Adier SG. Secondary Glomerular Disease. In: Brenner BM(ed). The Kidney 5th ed. Philadephia. WB Saunders 1996;P.1498-596.
9. Shah SV (ed). etal. Mechanisms of Glomerular Injury. Semin Nephrol 1991;11:253-372.
10. Iveson P, Brun C, etal. Aspiration biopsy of the kidney. J.Am.Soc.Nephrol 1997;8:1778-81.
11. Madaio MP. etal. Renal Biopsy. Kidney Int 1990;38:529-43.
12. Brenner DM. (ed.): Renal Pathology with Clinical and Functional Correlations. 2nd ed. Philadelphia,PA,Lippincott 1994;P85-115.
13. Renal Biopsy in the Elderly 544 AJKD. 2000;35(3).
14. Tophan P. Renal Biopsy. In: Feehally J, Flaege J, Johnson R. Comprehensive Clinical Nephrology. Mosby, Elsevier, 3rd ed.,2007; Chapter 6, pp 69-76.
15. The Principles and Practice of Nephrology. 2nd ed. Jacobson HR. Striker GE. Klahr S. (eds). St. Louis, Mosby Yearbook 1995, P.544-94.
16. Pirani CL. etal. Evaluation of Kidney Biopsy Specimens. In:Tisher CG. Bopsy Specimens. AJKDs 2000.
17. Churg J, Sabin LH. Classification of glomerular disease. Curg J, Sobin LH (eds.): Renal disease classification and Atlas of glomerular disease. Tokyo-Igaku Shoin 1982; pp3-19
18. Bed Side Renal Biopsy (Kaled Nass and William Charles O Nieff 955 AJKD 1999:5)
19. Saedi etal, Journal Family Medicine, Baghdad 2004 (Volume 46; No 1,2)

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