Histological features of Glomerulonephritis in adult with Nephritic Syndrome

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Abstract
Background: Nephritic syndrome refers as kidney disorder that causes the body to excrete an excessive amount of protein in the urine. It's usually results from damage to the clusters of small blood vessels in the kidneys that filter waste and extra water from the blood. The condition causes swelling, particularly in the feet and ankles, and increases the risk of other health problems. One form of glomerulonephritis of special interest is membranous nephropathy. In nondiabetic adults like some cases in the current study, membranous nephropathy is the leading cause of nephrotic syndrome. Methods: Renal biopsy is still the compulsory method to establish diagnosis and to offer the information about the severity of renal damage. Histological examination of 30 adults male and female cases of kidney specimen of 22-50 years old from Al Kut city, Wasit, Iraq. Routine tissue sections prepared and stained in different stains included hematoxylin and eosin (HE), periodic acid-Schiff (PAS), silver stain and Masson's trichrome stain. All biopsies were subjected to light microscope. The histopathological spectrum was analyzed according to the various clinical parameters as hypertension history of four years ago on irregular treatment, complaining of bilateral leg swelling for the last four years and no diabetic. Results: Examination of kidney sections revealed that at least one glomerulus from 11 glomeruli showed segmental sclerotic changes, no crescents formation. Focal adhesion to Bowman's capsule seen in two glomeruli, eight of eleven glomeruli are increased in size with no significant thickening of glomerular basement membrane, mild lobular accentuation and generally patent capillary lumens. Mesangial expansion by PAS and Trichome red positive matrix with only few intra glomerular lymphocytic infiltrations seen in few glomeruli. Microscopical findings of interitem showed no fibrosis or tubular atrophic changes with focal mononuclear inflammatory cell infiltration.
Keywords: Histopathological, Nephritis syndrome, glomerulonephritis

1. Introduction

One of the worldwide disease affecting populations in developing countries is chronic kidney disease. In many cases, glomerulonephritis is the underlying disease leading to kidney failure. One hallmark of glomerulonephritis is proteinuria, which may in its most severe form lead to nephrotic syndrome (NS) (Cameron and Hicks, 2002). Clinically, the syndrome presents with hypoalbuminemia, edema and hyperlipidemia. In children idiopathic nephrotic syndrome is the commonest manifestation of glomerular disease (Karnath and Keddis, 2007). Although the genetics of congenital forms are well known to the pediatrician, secondary form of nephrotic syndrome caused by different types of glomerulonephritis are encountered. The etiopathogenesis of nephrotic syndrome has evolved over several years and theories (Charleswort et al., 2008).

But also in adults, nephrotic syndrome caused by different kinds of glomerulonephritis occurs. One form of glomerulonephritis of special interest is membranous nephropathy (Karnath and Keddis, 2007). In nondiabetic adults, membranous nephropathy is the leading cause of nephrotic syndrome. There, its most often primary (idiopathic) and the remaining are secondary to systemic disease or exposure to infection or drugs (Jayawardene et al., 2002).

This syndrome is one of the best-known presentations of adult or pediatric kidney disease. It is a relatively rare but important manifestation of kidney disease, caused by a range of different disorder that damage the glomeruli that filter fluids, electrolytes and wastes from the blood, preventing the loss of protein, higher molecular weight solutes and cellular compartment. So glomeruli damaging leads to proteinuria, hypoalbuminaemia and also peripheral edema and several associated complications (Bellomo and Atkins, 1993)

The classical presentation of a primary or secondary disease is a triad of microscopic hematuria, proteinuria and impaired renal function, and when the etiology of this syndrome is unclear, renal biopsy used to confirm the diagnosis, and the classification of NS is based on the pattern of injury that seen on light microscopy and analyzed using immunofluorescence and electron microscopy to determine any changes in podocytes, glomerular basement membrane and capillary endothelial cells (Palmer et al., 2008).

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Membranous nephropathy MN is a glomerular disease that accounts for 20-30% of causes of nephrotic syndrome in none diabetic adults, that affect patients in both sexes and all ages (Debiec and Ronco, 2016). Despite the availability of anti-PLA2R antibody, kidney biopsy remains the standard of care in diagnosing MN (Bellomo and Atkins, 1993).

2. Material and Method

30 cases of renal biopsies obtained from adult's male and female, ages about 22-50 years old suffer with kidney disease in different area of Kut, wasit province, Iraq. The clinical records of all patients were reviewed and nephritic syndrome was diagnosed according to the accepted definition.

Histopathological preparation of cross-sectional renal biopsy was conducted at the Histopathology Department of Al Karama Hospital, Wasit, Iraq. Two core biopsy each 1*1.5 cm taken immediately in formalin and prepared to histopathological work.

All renal biopsies specimens were prepared as per the standard histological protocol and examined by the hospital pathologists. Analysis included light microscopy (LM), routine tissue processing was performed from formalin-fixed, dehydrated, cleared and wax paraffin-embedded tissue. Tissue sections were cut at 4-5µm thickness. Staining included hematoxylin and eosin (HE), periodic acid-Schiff (PAS) (Resch et al., 2002), silver stain and Masson's trichrome stain (Farris and Alpers, 2011).

3. Results and Discussion

History case results showed hypertension history of four years ago on irregular treatment, complaining of bilateral leg swelling edema (fig. 1) for the last four years, edema is the major clinical manifestation of NS, that caused by a reduction in plasma oncotic pressure from the urinary loss of albumin, which promotes the movement of fluid from the vascular space into the interstitial, the same result recorded by Adedoyin et al., 2016 from cases of acute glomerulonephritis.

Normal renal biopsy sections reveled that, the nephron was comprised of a glomerulus with complex tubular system. Glomerulus forms by a tuft of capillaries surrounded by an impervious capsule denominated Bowman's capsule; glomerular capillaries are flanked afferent and efferent arterioles. Glomerulus and the first portion of the tubular system, known as the proximal convoluted tubule located in the renal cortex. Following by loop of Henle, a hairpin-like structure penetrates the medulla and returns to the cortex to connect with the distal convoluted tubule, the nephron drains into the collecting duct via connecting tubules (figs. 2, 3, 4).

Biopsy material composed of renal cortex and medulla, consist of 11 glomeruli, none are globally sclerosed, at least single glomerulus from eleven glomeruli showed segmental sclerotic changes, no crescents formation. Focal adhesion to Bowman's capsule seen in two glomeruli, 8 of ele11 glomeruli are increased in size with no significant thickening of glomerular basement membrane, mild lobular accentuation and generally patent capillary lumens. Mesangial expansion by PAS stain and Trichome red positive matrix with only few intra glomerular lymphocytic infiltrations seen in few glomeruli (figs. 5, 6,7) which agreed with finding of Adedoyin et al., 2016 that involved endocapillary proliferation with infiltration of neutrophils and mesangial cellular proliferation consistent with diffuse proliferative lesion, also agreed with the findings of John et al., 2022

Microscopical findings of interterm showed no fibrosis or tubular atrophic changes with focal mononuclear inflammatory cell infiltration (fig. 8) which resemble the results of John et al., 2022 which described glomerulus with mild mesangial expansion in studied the diagnosis of non-diabetic kidney disease (NDKD) in a diabetic patient

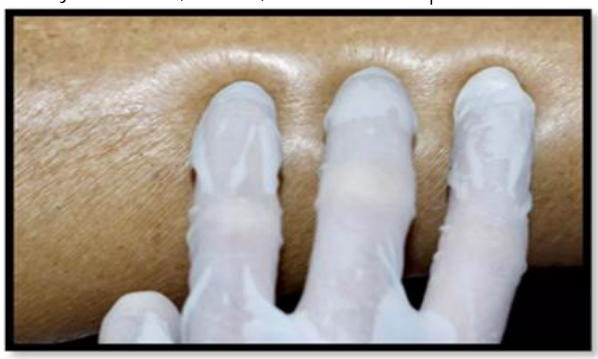

Figure 1: testing pitting edema in the leg of a patient with kidney injury

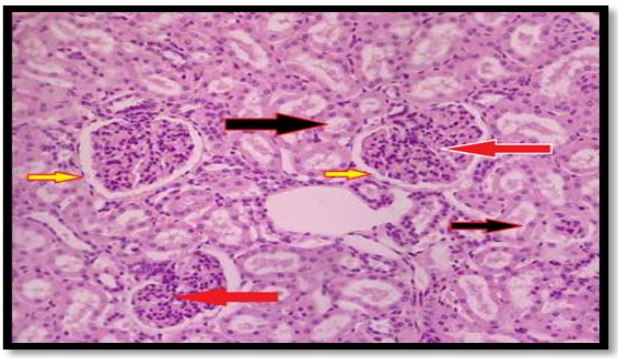

Figure 2: normal renal cross section shows glomerulus (red arrow), Bowman's capsule (yellow arrow), and convoluted tubules (black arrow). H&E stain, 400X

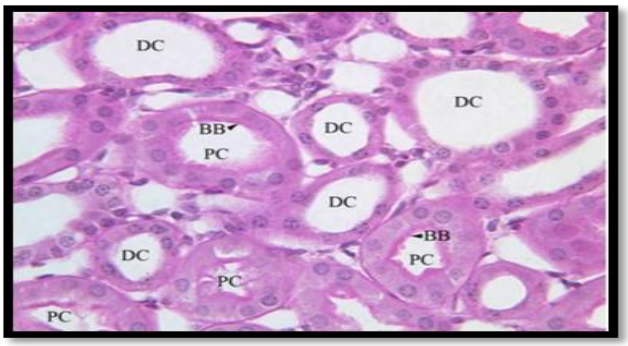

Figure 3: Cross section of normal kidney shows distal convoluted tubule (DC), proximal convoluted tubule (PC). H&E stain, 400X

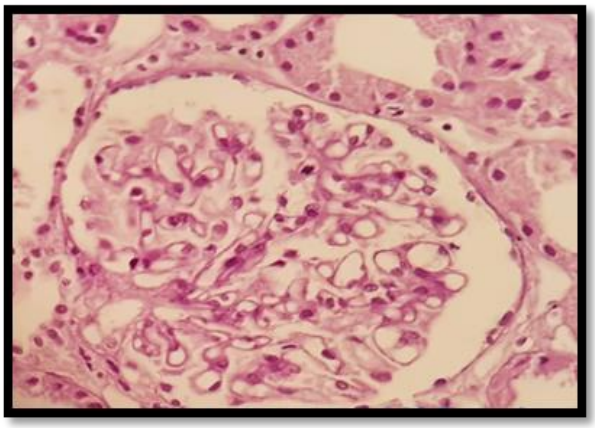

Figure 4: cross renal glomerulus with mild mesangial expansion, mild mesangial hypercellularity, and global uniform capillary wall 40x H&E

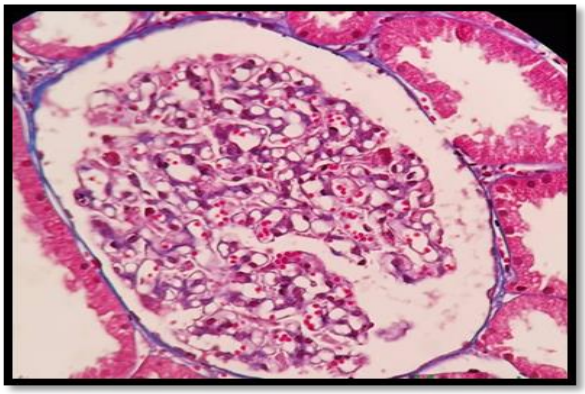

Figure 5: cross section shows glomeruli of kidney with glomerulonephritis. Masson's trichrome stain, 400x

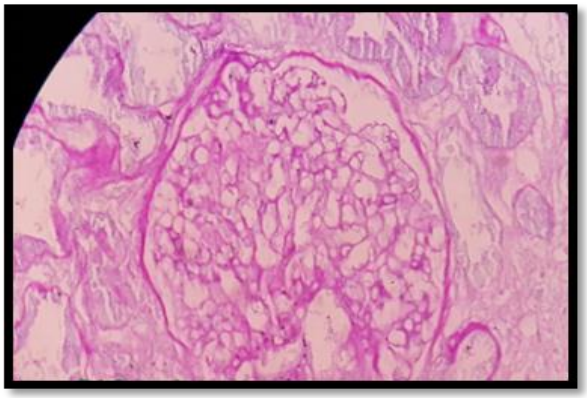

Figure 6: cross renal section shows glomeruli of kidney with glomerulonephritis. PAS stain, 400x

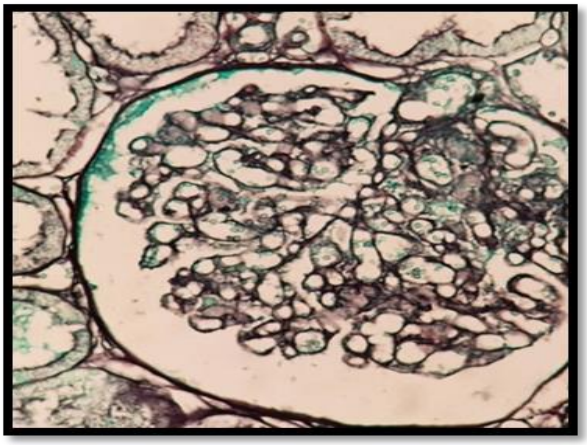

Figure 7: cross renal section shows glomeruli of kidney with glomerulonephritis. Silver green stain, 400x

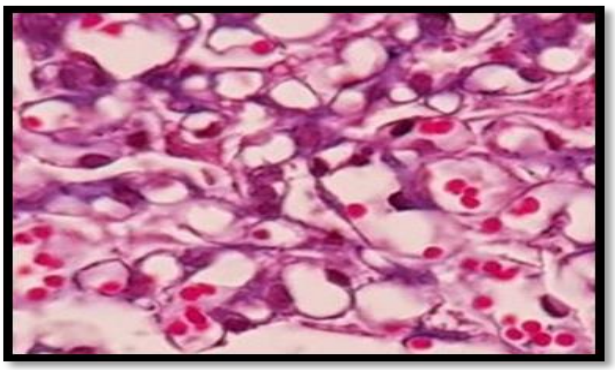

Figure 8: cross renal section shows endocapillary proliferation with infiltration of neutrophils and mesangial cellular proliferation. Masson's trichrome stain, 400x

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