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Original Article

Pathology Section

Comparative Changes Noted in Renal Biopsies on Light Microscopy of ANCA Positive Vs ANCA Negative Serology

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ABSTRACT

Objectives: Pauci-immune glomerulonephritis is the commonest cause of rapidly progressive glomerulonephritis (RPGN) which is associated with increased mortality and morbidity. More than 90% of these patients have serological presence of either antineutrophil cytoplasmic antibodies (ANCA), of cytoplamic (C) or perinuclear (P) type. "Immunofluoresence studies" exhibiting minimal or no fluorescence is diagnostic in all such cases. The present study aims to study the differences between renal biopsies of serologically ANCA negative versus ANCA positive individuals.

Materials and Methods: One hundred and twenty renal biopsies (of clinically suspected cases of systemic vasculitis) were sub-divided sub-divided under the heading of serologically ANCA positive and serologically ANCA negative; and scoring them by means of a semi-quantitative scoring system devised at the beginning of the study to identify statistically significant, specific light microscopic features in the sub-components of renal biopsy.

Results: Fifteen parameters were found to be statistically significantly (p-value < 0.05) in ANCA positive serological cases. These were glomerular capillary loop infiltration by neutrophils, cellular crescents, fibro-cellular crescents, glomerular fibrinoid necrosis, glomerular sclerosis, peri-glomerular infiltration, interstitial oedema, interstitial eosinophils, tubular atrophy, tubular necrosis, tubulitis, arterial hyalinization, arterial necrosis, arterial vessel wall polymorpho nuclear infiltrate and myointimal hypertrophy.

Conclusion: The presence of above parameters in a renal biopsy report of a patient (in absence of facilities of autoimmune serology and immunofluoresence) can alert both nephrologist and nephropathologist to keep a possibility of renal symptoms arising out of systemic vasculitis.

Keywords: Acute renal failure, Pauci-immune glomerulonephritis, Vasculitis

INTRODUCTION

Apart from the clinical history, ancillary investigations, including urine examination and radiological investigations; renal biopsy is considered the gold standard in reaching a diagnosis in renal diseases, especially in acute renal failure (ARF). Rapidly progressive glomerulonephritis (RPGN) is a type of ARF syndrome associated with rapid deterioration of kidney function (within days and weeks). It is conventionally categorized as Type-1 RPGN which is associated with anti-glomerular basement membrane antibody (Anti-GBM), Type-2 RPGN associated with immune-complex deposition leading to complement cascade and a third type (Type-3 RPGN) which is classically called pauci-immune type, defined by the lack of both anti-GBM antibodies or immune complexes by immunofluorescence (IF) and electron microscopy (EM). Although this is a misnomer because the pathogenesis of type-3 RPGN is also immune mediated as most patients (>90%) with this type of RPGN have antineutrophil cytoplasmic antibodies (ANCA), of cytoplasmic (C) or perinuclear (P) patterns, in the serum [1,2]. Hence, it has been proposed that all cases of so-called "pauci-immune" type RPGN also called ANCA associated (positive) glomerulonephritis are manifestations of systemic (small vessel) vasculitis and polyangitis [3] and are usually associated with rapid deterioration of kidney function, poorer prognosis and thus it very essential to diagnose these quickly for rapid therapeutic intervention. All other type of glomerulopathies including Type-1 and 2 RPGN are not associated with ANCA positivity.

The diagnostic modalities in ANCA associated glomerulonephritis which are an exhaustive 7 part serological panel, IF and EM are out of reach of most teaching and even renal conservation institutes/ hospitals of the third world countries with most treating physician dependant on the cynical susupicion and patient symptomatology. Although to diagnose RPGN these patients do undergo an ultra sound guided core needle biopsy.

The present study attempts to study the differences in the histological parameters on light microscopy alone by broadly dividing 120 renal biopsies under the heading of serologically ANCA positive and serologically ANCA negative; and scoring them by means of a semi-quantitative scoring system devised at the beginning of the study. Therefore; by means of statistical analysis endeavour of the study was to identify statistically significant, specific light microscopic features in the sub-component of renal biopsy which could be helpful while examining renal biopsies to segregate patients of pauci-immune RPGN (ANCA positive) without having to undergo any other ancillary investigation (serology/IF/EM).

MATERIALS AND METHODS

Patients who were clinically suspected of systemic vasculitis (comlains of joint pain with or without fever, palpable purpuras, scleritis an other eye manifestations etc) with probable lesions of renal system especially the kidney, were screened out of all the patients who attended the outpatient department (OPD) of medicine, surgery and paediatrics; of a tertiary care centre in north India during the period of January 2006 to September 2007.

One hundred and twenty cases in total who were screened out on basis of clinical suspicion (as well as abnormal urine examination) of systemic vasculitis were selected for renal biopsies and an autoimmune panel was employed in all these cases which included- complement levels (C3/C4), anti nuclear antibody (ANA), double stranded DNA (dsDNA), P-ANCA, C-ANCA, anti glomerular basement memberane (Anti-GBM) and cryoglobulins detection.

Glomerular Changes		Score
Mesangial Proliferation	Present	1
	Absent	0
Increased Mesangial Matrix	Present	1
	Absent	0
Glomerular Capillary Loop Neutroplhils	Present	1
	Absent	0
Glomerular Capillary Loop Mononuclear Cells	Present	1
	Absent	0
Glomerular Basement Membrane Thickening	Present	1
	Absent	0
Cellular Crescents	Present	1
	Absent	0
Fibrocellular Crescents	Present	1
	Absent	0
Fibrous Crescents	Present	1
	Absent	0
Glomerular Fibrinoid Necrosis	Present	1
	Absent	0
Glomerular Sclerosis	Present	1
	Absent	0
Periglomerular Infiltration	Present	1
	Absent	0
Interstitial Changes		
Interstitial Oedema	Present	1
	Absent	0
Focal Interstitial Infiltration	Present	1
	Absent	0
Diffuse Interstitial Infiltration	Present	1
	Absent	0
Interstitial Polymorphonuclear Cells	Present	1
	Absent	0
Interstitial Mononuclear Cells	Present	1
	Absent	0
Interstitial Eosinophils	Present	1
	Absent	0
Interstitial Fibrosis	Present	1
	Absent	0
Interstitial Granuloma	Present	1
	Absent	0
Tubular Changes		
Tubular Casts	Present	1
	Absent	0
Tubular Necrosis	Present	1
	Absent	0
Tubular Atrophy	Present	1
	Absent	0
Tubulitis	Present	1
	Absent	0
Arterial Changes		
Arterial Hyalinization	Present	1
	Absent	0
Arterial Necrosis	Present	1
	Absent	0
Arterial Vessel Wall Inflammation	Present	1
	Absent	0
Arterial Vessel Wall Polymorphonuclear Infiltrate	Present	1
	Absent	0
Arterial Vessel Wall Mononuclear Infiltrate	Present	1
	Absent	0
Arterial Vessel Wall Eosinophilic Infiltrate	Present	1
	Absent	0
Myointimal Hypertrophy	Present	1
	Absent	0

[Table/Fig-1]: The scoring system of the 30 parameters in the study

Subsequently they were then segregated under the two groups, as "ANCA positive" (C-ANCA, P-ANCA or both) and "ANCA negative" biopsies according to the serological positivity and negativity of the patients for ANCA. Biopsies in those individuals who showed positivity for markers other than ANCA (C3/C4, ANA, dsDNA, anti-GBM and cryoglobulins) were kept under ANCA negative group.

The fluorescent studies were taken up separately and the biopsies were evaluated and categorized as pauci-immune or immune complex for therapeutic purposes. However, these findings were not kept in the study design; as the aim of the present study was to pick up histological findings on light microscopy alone.

In the present study the "4 components" of kidney (glomerulus, tubules, interstitium and blood vessels) were further subdivided and a semi quantitative scoring was done by means of previously agreed definitions according to the reviewing pathologists and nephrologists. Overall 30 parameters were taken into account and scored [Table/Fig-1]. One positive (+) was given the score 1 and negative/ absent finding (–) was given a score of 0.

Sections were prepared and stained with haematoxylin and eosin (H&E) stain in all the 120 cases and were given random codes which were scored independently by means of double blinding (ANCA status not being known to the reviewing pathologist) by three pathologists. Final score was recorded when at least two or all experts showed agreement towards the final score.

STATISTICAL ANALYSIS

Statistical analysis (Chi-square test) was employed to enquire if the difference in the histological parameters as scored by the above mentioned scoring system were significantly different in the ANCA positive versus ANCA negative biopsies, in order to pick some inherent features which can help in differentiating ANCA positive renal biopsies from ANCA negative biopsies by means of light microscopy alone.

OBSERVATIONS AND RESULTS

In the present study comprising of 120 renal biopsies of clinically suspected cases of systemic vasculitis, the maximum number of cases were of End Stage Renal Disease (ESRD) - 17.50%, followed by membranous-13.33% and mesangio-proliferative glomerulonephritis-9.16%. Benign Nephrosclerosis, focal necrotizing glomerulonephritis and focal global glomerulosclerosis were the least common of the glomerulopathy recorded with 1 case each [Table/Fig-2]. Serological studies concluded that 15 cases (12.5%) were serologically P-ANCA, C-ANCA or both positive and were categorized as ANCA positive biopsies. P-ANCA positivity accounted for the maximum number of ANCA cases (80.00%) whereas C-ANCA positivity was seen in 13.33% of all ANCA cases. Rest of the cases (20.00%) on serology were showed positivity for both C-ANCA and P-ANCA. The rate of demonstration of ANCA positive cases (12.5%) was significant despite a small sample size of 120 biopsies. Of all the serologically ANCA positive cases; the maximum number of cases were of end stage renal disease (ESRD) (26.66%). Focal and segmental mesangial proliferative and crescentic glomerulonephritis without fibrinoid necrosis were the next

Lesion	Total	% of All Glo	Male	Female	Avg.Age
• **Glomerulopathies**	120		86	40	34.67
• End Stage Renal Disease (ESRD)	21	17.50	15	06	37.91
• Membranous	16	13.33	12	04	34.75
• Mesangial Proliferative	11	09.16	07	04	31.77
• Diffuse Proliferative	10	08.33	08	02	36.66
• Focal Segmental/ Proliferative	10	08.33	07	03	49.33
• Membrano Proliferative (MPGN)	08	06.66	04	04	27.64
• Systemic Lupus Erythematosus (SLE)	08	06.66	01	07	33.56
• Diabetic Nephropathy	07	04.34	04	03	48.20
• Focal Segmental Glomerulosclerosis (FSGS)	07	04.34	06	01	17.57
• Crescentic	05	03.10	05	00	27.00
• Minimal Change Disease	05	03.10	03	02	14.40
• Amyloid Nephropathy	04	02.48	04	00	33.75
• Normal	03	01.86	03	00	37.00
• Renal Cortical Necrosis	02	01.24	00	02	37.00
• Benign Nephrosclerosis	01	00.62	01	00	60.00
• Focal Necrotizing	01	00.62	01	00	60.00
• Focal Global Glomerulosclerosis	01	00.62	00	01	07.00

[Table/Fig-2]: Spectrum of all glomerulonephritis in the present study

S.No	Parameter	ANCA +Ve (%)	ANCA -Ve (%)	X^2	p-value
1	Mesangial proliferation	66.66	45.71	2.307	0.129
2	Increased mesangial matrix	40.00	40.00	0.000	1.000
3	Glomerular capillary loop neutroplhils	80.00	18.09	26.252	0.001
4	Glomerular capillary loop mononuclear cells	46.66	29.52	1.783	0.182
5	Glomerular basement membrane thickening	60.00	48.57	0.686	0.408
6	Cellular crescents	26.66	01.90	16.942	0.001
7	Fibrocellular crescents	40.00	03.80	22.504	0.001
8	Fibrous crescents	26.66	16.66	1.510	0.219
9	Glomerular fibrinoid necrosis	80.00	05.71	56.807	0.001
10	Glomerular sclerosis	73.34	41.90	5.222	0.022
11	Periglomerular infiltration	66.66	16.19	19.177	0.001
12	Interstitial oedema	80.00	36.19	10.364	0.010
13	Focal interstitial infiltration	53.33	44.76	0.388	0.533
14	Diffuse interstitial infiltration	26.66	24.76	0.025	0.873
15	Interstitial polymorphonuclear cells	26.66	10.47	3.146	0.076
16	Interstitial mononuclear cells	86.66	69.52	1.900	0.168
17	Interstitial eosinophils	20.00	02.83	8.120	0.004
18	Interstitial fibrosis	60.00	51.42	0.387	0.534
19	Interstitial granuloma	----	----	-	-
20	Tubular casts	73.33	72.38	0.060	0.938
21	Tubular necrosis	33.33	02.85	19.592	0.001
22	Tubular atrophy	73.33	44.76	4.290	0.038
23	Tubulitis	53.33	20.95	7.340	0.007
24	Arterial hyalinization	80.00	48.57	5.199	0.023
25	Arterial necrosis	13.33	02.85	3.807	0.038
26	Arterial vessel wall inflammation	13.33	04.76	1.755	0.185
27	Arterial vessel wall polymorphonuclear infilterate	13.33	----	14.237	0.001
28	Arterial vessel wall mononuclear infilterate	13.37	04.76	1.755	0.185
29	Arterial vessel wall eosinophilic infilterate	----	----	-	-
30	Myointimal hypertrophy	66.66	30.47	7.536	0.006

[Table/Fig-3]: Results of Chi-Square test with p values along with the percentage histopathological parameters in ANCA positive Vs ANCA negative biopsies

most common category (20.00%) followed by necrotizing crescentic glomerulonephritis (13.33%). Necrotizing glomerulonephritis, focal proliferative and membranous with foci of fibrinoid necrosis were the least commonly seen (6.66%).

The most common histological findings observed in ANCA positive biopsies were- glomerular loop neutrophil infiltration, fibrinoid necrosis, interstitial oedema and arterial hyalinization (all seen in 80% ANCA positive renal biopsies). Tubular changes in the form of

- Glomerular capillary loop infiltration by neutroplhils.
- Cellular crescents
- Fibro-cellular crescents
- Glomerular fibrinoid necrosis
- Glomerular sclerosis
- Peri-glomerular infiltration
- Interstitial oedema
- Interstitial eosinophils
- Tubular atrophy
- Tubular necrosis
- Tubulitis
- Arterial hyalinization
- Arterial necrosis
- Arterial vessel wall polymorphonuclear infilterate
- Myointimal hypertrophy

[Table/Fig-4]: The significant histological parameters (p-value < 0.05)

- Mesangial proliferation
- Increased mesangial matrix
- Glomerular capillary loop mononuclear cells
- Glomerular basement membrane thickening
- Fibrous crescents
- Focal interstitial infiltration
- Diffuse interstitial infiltration
- Interstitial polymorphonuclear cells
- Interstitial mononuclear cells
- Interstitial fibrosis
- Interstitial granuloma
- Tubular casts
- Arterial vessel wall mononuclear infiltrate
- Arterial vessel wall eosinophilic infiltrate

[Table/Fig-5]: The histological parameters found not significant (p-value > 0.05)

atrophy and presence of casts and glomerular sclerosis (73.33%) were the next most common changes. Peri-glomerular infiltrate along with myointimal hyperplasia accounted for 66.66% of the changes. On the other hand in ANCA negative cases, the most common histological parameters were tubular casts (72.38%) followed by interstitial fibrosis (51.42%), glomerular basement membrane thickening and arterial hyalinization (48.57%) and tubular atrophy and focal interstitial infiltration (44.76%) were the other significant histological findings [Table/Fig-3].

The other changes in the subcomponents of the biopsy (glomeruli, tubules, blood vessels and interstitium) individually are noted in [Table/Fig-3] and discussed in details later on.

The essence of the present study was to identify probable histological features which were significantly present in ANCA positive biopsies by means of the scoring system described above. 15 parameters were found to be statistically significantly (p-value < 0.05) in ANCA positive serological cases. These were glomerular capillary loop infiltration by neutrophils, cellular crescents, fibro-cellular crescents, glomerular fibrinoid necrosis, glomerular sclerosis, peri-glomerular infiltration, interstitial oedema, interstitial eosinophils, tubular atrophy, tubular necrosis, tubulitis, arterial hyalinization, arterial necrosis, arterial vessel wall polymorphonuclear infiltrate and myointimal hypertrophy [Table/Fig-4].

In contrast to the above findings 14 histological parameters were found not significant (p-value > 0.05) in the present study which included mesangial proliferation, increased mesangial matrix, glomerular capillary loop mononuclear cell infiltration, glomerular basement membrane thickening, fibrous crescents, focal interstitial infiltration, diffuse interstitial infiltration, interstitial polymorphonuclear cells, interstitial mononuclear cells, interstitial fibrosis, interstitial granuloma, tubular casts, arterial vessel wall mononuclear infiltrate and arterial vessel wall eosinophilic infiltrate [Table/Fig-5].

Parameters of interstitial granuloma and arterial wall eosinophilic infiltrate kept in the scoring system were not noted in either of the subgroups.

DISCUSSION

Patients with ANCA positive glomerulonephritis usually present in emergency with features of rapidly progressive glomerulonephritis with hematuria, proteinuria and rapidly progressing renal biochemical parameters (serum urea and creatinine levels) [3-5].

In an 8 year long similar study done at PGI Chandigarh, India; which is an apex centre having all the facilities of serology, EM and IF- the rate of diagnosed ANCA positive cases in renal biopsies was 2% (48 ANCA positive cases out of total 2150 renal biopsies received) in which comparative analysis was done between the two sub-groups. In contrast the rate of positivity detected in our study was higher (around 12%) for comparative analysis [1].

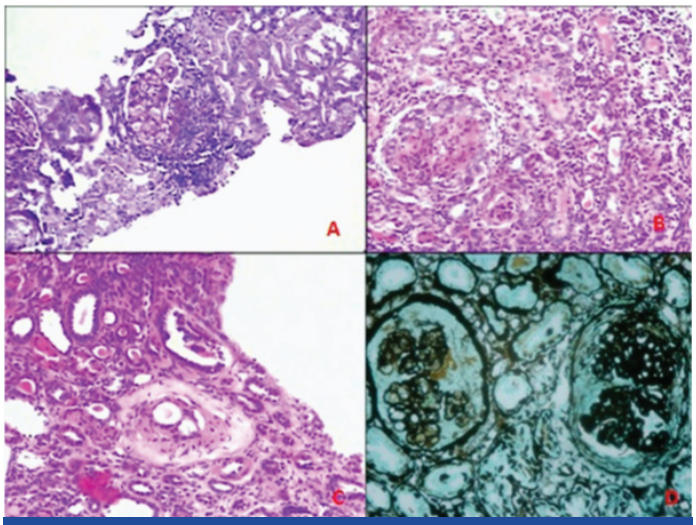
The hallmark histologic lesions in most of the studies of acute pauci-immune ANCA positive as well as ANCA negative glomerulonephritis are documented as crescents and fibrinoid tuft necrosis, which occur at the same frequency irrespective of the presence or absence of associated systemic vasculitis [3,6,7]. In an analysis of 45 ANCA-positive patients with glomerulonephritis and systemic small-vessel vasculitis in a research study, renal biopsies demonstrated crescents in 93% of patients and glomerular necrosis in 98% [3].

Hauer et al., in their European Vasculitis Study Group (EUVAS) of 173 patients with renal disease in microscopic polyangitis and Wegener's granulomatosis observed necrotizing crescentic glomerulonephritis (65%) as the commonest histopathological diagnosis followed by crescentic glomerulonephritis without fibrinoid necrosis (23%), as the next most common histological diagnosis in patients with ANCA positive serology which almost corresponds to the work conducted by researchers in the western hemisphere [8].

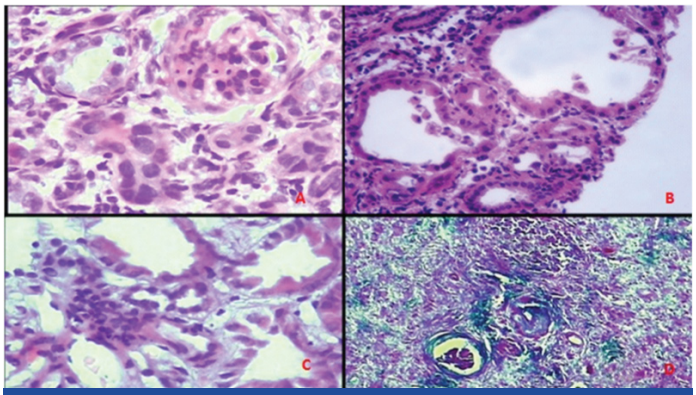
In the present study however, of all the ANCA positive cases on histology, the maximum number of cases were of ESRD accounting for 26.66% of cases. Focal and segmental mesangial proliferative and crescentic glomerulonephritis without fibrinoid necrosis were the next most common category (20.00%) followed by necrotizing crescentic glomerulonephritis (13.33%). Necrotizing glomerulonephritis, focal proliferative and membranous with foci of fibrinoid necrosis were the next most common category having 1 case each (6.66%). Thus in contrast to the documented finding of most common histological findings of cresenteric type glomerulonephritis in cases of systemic vasculitis by various researchers, diffuse global glomerulosclerosis/ESRD was the most common histological finding in our group. The fact this was also the most common histological diagnosis in ANCA negative sub-group also speaks volume about the delayed presentation and patient ignorance as a great challenge to nephrologists in developing countries of Asia and Africa.

Of the 30 parameters under which every renal biopsy (120) in the present study was scored as detailed in materials and methods; the most common parameters observed in ANCA positive biopsies were- fibrinoid necrosis, glomerular loop neutrophil infiltration, interstitial oedema and arterial hyalinization (all seen in 80% ANCA positive renal biopsies). Tubular changes in the form of atrophy and presence of casts and glomerular sclerosis (73.33%) were the next most common changes followed by peri-glomerular infiltrate along with myointimal hyperplasia accounted for 66.66%. On the other hand in ANCA negative non-pauciimmune glomerulonephritis cases the most common histological parameters were tubular casts (72.38%) followed by interstitial fibrosis (51.42%). Glomerular basement membrane thickening and arterial hyalinization (48.57%) and tubular atrophy and focal interstitial infiltration (44.76% in each) were other significant histological findings [Table/Fig-3].

Glomerular Changes: Of the ANCA positive biopsies crescents were noted in all the cases with fibrocellular being the commonest (40 %) followed equally by cellular and fibrous crescents (26.66%). Fibrinoid necrosis of the glomerular tuft was seen in 80% of the glomeruli. In 46.66% of cases there was neutrophilic infiltration of the glomerular capillary loops [Table/Fig-3,6].



[Table/Fig-6]: Microphotographs depicting glomerular changes in ANCA positive biopsies
(a) Peri-glomerular inflammation [H & E 100 X]
(b) Cellular Cresecent [H & E 200 X]
(c) Fibrous Crescent [H & E 200 X]
(d) Crescent with special stain [PMS 400 X]



[Table/Fig-7]: Microphotographs depicting changes noted in tubules, interstitium and blood vessels in ANCA positive biopsies
(a) Tubulitis with lymphocytic infiltration [H&E 400X]
(b) Tubular necrosis and epithelial shedding [H&E 400X]
(c) Vasculitis [H&E 400X]
(d) Interstitial fibrosis, inflammation along with periglomeruar fibrosis and obsolscent glomeruli with increased vessel wall thickness noted in ESRD [MT 200X]

Other studies such as EUVAS study have shown that 45% of glomeruli had (predominantly cellular) crescents and 23% were globally sclerotic. Fibrinoid necrosis of the glomerular tuft was seen in 22% of the glomeruli [8]. In 5% of cases the fibrinoid necrosis was present with out crescents [Table/Fig-6].

In a study of 32 renal biopsies from patients with microscopic polyangiitis, Savage et al., identified glomerular segmental necrosis in 100 % and crescent formation in 88 % [9] but in another study of 20 renal biopsies by D'Agati a year later in patients of microscopic polyangitis only 80% had segmental glomerularnecrosis and 85% had crescents [10].

In comparison to ANCA positive biopsies; crescents noted in ANCA negative biopsies were mainly fibrous (16.6%) followed by fibro-cellular (3.80%) and cellular (1.90%). Other glomerular changes seen in this subset of biopsies were increased glomerular basement membrane thickening (48.57%), glomerulosclerosis (41.90%) and increased mesangial proliferation (45.71%) [Table/Fig-3].

Tubules and Interstitium: Various researchers have noted interstitial edema and focal tubular epithelial flattening as the most common findings seen in tubules of pauci-immune glomerulonephritis. In interstitium; interstitial infiltration by leukocytes is common and is most pronounced adjacent to severely inflamed glomeruli or vessels [10]. In the present study, interstitial oedema was seen in 80% of ANCA positive biopsies and interstitial infiltrate was seen either focally (53.33% cases) or in diffuse (26.66% cases) fashion. In both cases mononuclear infiltrate formed the predominant part (86.66%), of the infiltrate of ANCA positive cases followed by neutrophils and eosinophils in 26.66% and 20.00% cases respectively. Interstitial fibrosis was present in 60% of ANCA positive biopsies in this study. Many studies such as the one conducted by Haeur et al., have also noted the presence of interstitial oedema but only in 34% of biopsies along with interstitial infiltrates (predominantly mononuclear) in 92% of biopsies and fibrosis was present in 83% of biopsies [8,10,11] [Table/Fig-3].

In contrast to above findings; in ANCA negative biopsies the statistically different parameters were interstitial oedema (36.19%) and interstitial eosinophilic infiltrate (2.83%).

Although tubular casts were seen in almost same frequency in both ANCA positive and negative biopsies (73.37% & 72.38%); tubular necrosis, atrophy and tubulitis were noted more in ANCA positive biopsies in comparison to ANCA negative biopsies (33.33% & 2.85%; 73.33% & 44.76%; 53.33% & 20.95%) [Table/Fig-3,7].

According to the EUVAS study tubular casts and necrosis were present in 87% and 66% of biopsies, respectively. Tubular atrophy was present in 86% of biopsies. Tubular intra-epithelial infiltrate (tubulitis) was seen in 64% of biopsies [8].

Blood vessels: Vessels by definition are the compartment most affected by systemic vasculitis. Researchers such as Jennette et al., [2] identified arteritis in 13% of renal biopsies from 45 patients with ANCA glomerulonephritis, Savage et al., [9] identified vasculitis in 19% of 32 renal biopsies from patients with microscopic polyangiitis, and Vizjak et al., [12] identified active vasculitis in 23% of 55 patients with PR3-ANCA glomerulonephritis and 23% of 74 patients with MPO-ANCA glomerulonephritis.

In the present study vessel wall inflammation and polymorphonuclear infiltration of the vessel wall in ANCA positive biopsies were seen in only 13.33% cases. Arteriosclerosis (Hyaline change) was seen in 80.00% renal biopsies whereas; arteriolosclerosis (Hyperplastic myointimal changes) was present in 66.66% of cases. In the EUVAS Study interstitial vasculitis was present in only 12% of biopsies. Arteriosclerosis and arteriolosclerosis were present in 70% and 32% of biopsies, respectively. Vessel wall changes in ANCA negative biopsies were either absent (arterial wall neutrophilic infiltration) or were low in frequency (arterial necrosis-2.83% and arterial vessel wall lymphocytic inflammation-4.76%) [Table/Fig-3].

CONCLUSION

The histological parameters obtained statistically in the present study on light microscopy alone in a biopsy report of any renal patient (in absence of facilities of autoimmune serology and IF/EM) should alert both nephrologist and nephropathologist to keep a possibility of renal symptoms arising out of systemic vasculitis.

The present study does not aim to undermine the conventional protocol of investigation and treatment of patients of systemic vasculitis with kidney change. Rather it is a view point/ commentary from nephropathologists and nephrologists working in one of the developing countries where resources are limited and often the therapy has to be instituted on basis of clinical suspicion and ancillary investigations.

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