DOI: 10.14260/jemds/2015/1082

ORIGINAL ARTICLE

CLINICAL PROFILE OF ACUTE GLOMERULONEPHRITIS IN NORTH INDIA: A SINGLE CENTRE STUDY

Shazia Hamid[1], Vinod Kumar[2], Amit Badgal[3], Ritu Bhagat[4]

HOW TO CITE THIS ARTICLE:
Shazia Hamid, Vinod Kumar, Amit Badgal, Ritu Bhagat. "Clinical Profile of Acute Glomerulonephritis in North India: A Single Centre Study". Journal of Evolution of Medical and Dental Sciences 2015; Vol. 4, Issue 43, May 28; Page: 7458-7460, DOI: 10.14260/jemds/2015/1082

ABSTRACT: Acute glomerulonephritis is a set of diseases in which an immunologic mechanism triggers inflammation and proliferation of glomerular tissue leading to damage to the capillary endothelium, mesangium or basement membrane. The exact triggers for inflammation are unknown. The present study was conducted to describe the clinical profile of patients with acute glomerulonephritis in North India.
KEYWORDS: Acute glomerulonephritis, clinical profile, renal biopsy.

INTRODUCTION: Acute glomerulonephritis (GMN) results from either the primary renal disease or secondary to the systemic illnesses. It is characterized by the presence of impaired renal function, hypertension and edema. Acute glomerulonephritis may occur sporadically or in cyclic epidemic form in people with lower socioeconomic status. Subclinical disease is more prevalent than the clinical disease during epidemics.[1] Tpically, a younger patient presents with swelling around eyelids, puffiness of face, reduced urine output, teac coloured urine, high blood pressure and hematuria. Edema, hematuria, oliguria and hypertension are the cardinal features of acute nephritic syndrome.[2] Acute GMN following throat infections occurs after a latent period of 1-2 weeks; whereas it follows skin infections after 3-6 weeks. When impetigo and pharyngitis are coexistent, the infection in the throat is usually due to contamination from the skin.[1] A correlation between serum complement levels and post streptococcal GMN has been found in many clinic-pathological studies.[3] The nephritogenic strains include M types 1, 2, 4 and 12 in patients with sore throat and M types 25, 45, 47, 55, 57 and 60 in patients with impetigo.[4] Acute GMN is more common in males than females in younger age groups, the predispositions being tonsillitis, nasopharyngitis and otitis media.[5] The basic alteration in acute GMN occur in intercapillary mesangium resulting in ischemia. Later on, fibrosis develops leading to subacute AGN.[6] The acute GMN in older patients is more serious and carries a higher mortality rate. The most common cause of GMN in the world is IgA nephropathy characterized by the presence of IgA mesangial deposits. However, the most common cause of nephrotic syndrome in Indian children is Minimal Change Disease and membrano proliferative GMN in adults.[7] Subepithelial deposits are the characteristic all found in acute PSGN and may occur in glomerulonephritis due to bacterial endocarditis, systemic lupus erythematosus and cryoglobulinaemia.[8]

METHODOLOGY: The present study was conducted in the department of Medicine in Government Medical College, Jammu over a period of one year. The 65 patients suspected of acute GMN due to peculiar clinical presentation underwent all the routine investigations. Out of 65 patients, 9 responded to the medical treatment and 56 patients underwent renal biopsy by the Nephrologist. The patients with Diabetes mellitus, chronic failure, heart failure and chronic liver disease were excluded from the study.

DOI: 10.14260/jemds/2015/1082

ORIGINAL ARTICLE

RESULTS AND DISCUSSION: The clinical data obtained during the study has been presented in the tabulated form below:

Children: Adults	8(12.3%): 57(87.7%)
Male: Female	40(61.53%): 25(38.46%)
Duration of illness < 1 week	12(18.46%)
History of sore throat	42(64.60%)
Hypertension	53(81.53%)
Edema Periorbital: Pedal: Generalised	6(9.2%): 4(6.2%): 55(84.6%)
Gross hematuria: microscopic hematuria	18(27.7%): 45(69.2%)
Proteinuria >3g/ 24 hours <3g/ 24 hours	 49(75.38%) 16(24.61%)
Oliguria	60(92.3%)
Reduced creatinine clearance	62(95.4%)
Cholesterol level >200mg/dl	24(36.9%)
Raised ASO titre	17(26.2%)
Response to medical treatment	9(9%)

Table 1

In our study, 61% of the patients were males and the rest were females. Murphy et al (1938) had similarly found that 53% were males and the rest were females.[9] 64% of the patients reported previous sore throat, a finding similarly reported by Burke et al (1947). Hematuria, gross and microscopic were observed in 27% and 69% of the patients.[10] Murphy et al (1934) in their study had reported gross hematuria in 34% of the patients.[9] Oliguria was observed in 92% of the patients. Goldman et al. (1962) showed the percentage of oliguria as 42% in acute nephritis.[10] Hyperchlosterolemia was observed in 36% of the patients.

Lesions found on renal biopsy:

Diffuse Proliferative GMN	4(7.1%)
Focal Segmental Glomerulosclerosis(FSGS)	2(3.6%)
Membranoproliferative GMN	13(20%)
Membranous GMN	8(14.3%)

Table 3

In our study, FSGS was observed in 3.6%, MGN in 14.3%, Diffuse proliferative GMN in 7.1% and MPGN in 20% of the patients. Richard et al (1984) had FSGS in 31% of the patients.

DOI: 10.14260/jemds/2015/1082

ORIGINAL ARTICLE

CONCLUSION: The patients presenting with edema, reduced urine output, proteinuria, hypertension and hematuria are more likely to reveal histopathologic finding of acute glomerulonephritis. PSGN should be treated aggressively for better outcome.

REFERENCES:

1. Anthony et al. Attack rates of acute nephritis after type 49.Streptococcal infections of the skin and of the respiratory tract. J Clin Invest. 1969; 48: 1697-704.
2. Rodriguez-Iturbe et al. Epidemic post-streptococcal glomerulonephritis (nephrology forum). Kidney Inter.1984; 25: 129-136.
3. Hisano S et al. Activation of the lectin complement pathway in PSGN. Pathol Inter.1997; 57: 351-357.
4. Francis et al. Investigation of milk-borne Streptococcus zooepidemicus infection associated with glomerulonephritis in Australia. J Infection. 1993; 27: 317-323.
5. Borok et al. Clinicopathological features of Zimbabwean patients with sustained proteinuria. Central Afr J Med. 1997; 43: 152-8.
6. Cavallo T et al. Glomerular permeability: ultrastructural quantitative studies relating proteinuria to pathologic features in murine lupus nephritis. Am J Pathol. 1980; 101: 93-100.
7. Shao YN et al. Serum immunoglobin E can predict minimal change disease before renal biopsy. Am J Med Sci. 2009; 338: 264-267.
8. Oda et al. Glomerular proliferating cell kinetics in acute post-streptococcal glomerulonephritis. J Pathol. 1997; 183: 359-368.
9. Murphy et al. Acute glomerulonephritis with special reference to the course and prognosis: a study of 150 cases. JAMA. 1938; 111 (8): 668-74.
10. Burke JB. Prophylactic sulphadimide in children subject to recurrent infections of upper respiratoty tract. 1956. BMJ. 538-541.
11. Goldman R et al. Sporadic acute glomerulonephritis in adults. California Med. 1962; 96 ed.
12. Richard et al. Dense intramembranous deposit disease. New pathological features. Kidney Inter. 1984; 25: 660-70.

AUTHORS:

1. Shazia Hamid
2. Vinod Kumar
3. Amit Badgal
4. Ritu Bhagat

PARTICULARS OF CONTRIBUTORS:

1. Lecturer, Department of Medicine, GMC, Jammu.
2. Senior Resident, Department of Medicine, GMC, Jammu.
3. Senior Resident, Department of Medicine, M.M. Medical College, Solan.
4. Post Graduate, Department of Pathology, GMC, Jammu.

FINANCIAL OR OTHER COMPETING INTERESTS: None

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Shazia Hamid,
Department of Medicine,
GMC, Jammu-180001.
E-mail: drshaziahamid@gmail.com

Date of Submission: 19/05/2015.
Date of Peer Review: 20/05/2015.
Date of Acceptance: 21/05/2015.
Date of Publishing: 26/05/2015.