

Research Article

Observation on Clinicopathological Profile of Post-Infectious Glomerulonephritis

Mangal Charan Murmu*, Arakhita Swain and Saroj Kumar Satpathy

Department of Paediatrics, SCB Medical College & Hospital, Cuttack, Odisha, India

Abstract

Introduction: Post-infectious glomerulonephritis is one of the most important and intriguing conditions in the discipline of paediatric nephrology. Although the eventual outcome is excellent in most cases, Post Streptococcal Glomerulonephritis (PSGN) remains an important cause of acute renal failure and hospitalization for children in both developed and underdeveloped areas.

Methods: It's a direct prospective study done in the Paediatric Department of SCB Medical college, Cuttack, Odisha, India.

Results: Most common predisposing factor was scabies along with other infected skin lesion. Most patients had diminished complement level.

Conclusion: Post streptococcal glomerulonephritis is the most common type of glomerulonephritis in childhood. Decreased Serum C3 level is the one of predictor of PSGN.

Keywords: Acute glomerulonephritis; Antistreptococcal; Complement C3

Abbreviations

AGN: Acute Glomerulonephritis

ASO: Antistreptococcal

CRP: C-Reactive Protein

C3: Complement 3

ESR: Erythrocyte Sedimentation Rate

HSP: Henoch-Schonlein Purpura

PSGN: Post Streptococcal Glomerulonephritis

RBC: Red Blood Cell

SLE: Systemic Lupus Erythematosus

WBC: White Blood Cell

*Corresponding author: Mangal Charan Murmu, Department of Paediatrics, SCB Medical College & Hospital, Cuttack, Odisha, India, Tel: +91 9437445041; E-mail: mangal74murmu@yahoo.co.in

Citation: Murmu MC, Swain A, Satpathy SK (2017) Observation on Clinicopathological Profile of Post-Infectious Glomerulonephritis. J Nephrol Renal Ther 3: 014.

Received: June 07, 2017; **Accepted:** August 19, 2017; **Published:** September 04, 2017

Introduction

Presence of oliguria, oedema, hematuria, proteinuria and hypertension are the essential criteria of acute glomerulonephritis [1,2]. Acute glomerulonephritis is an immune complex disease where there is inflammation of glomerulus occurs by proliferations of cellular elements i.e., endocapillary proliferation with mesangial proliferation and neutrophil cell in the capillary of the glomerular tuft through an immunological response as seen in optic microscopy [2,3]. There have been stages of development of our knowledge with regards to etiology and clinical course of acute glomerulonephritis since Bright's description [4]. Post-infectious glomerulonephritis is the most common cause constituting 80% cases of post streptococcal glomerulonephritis. Postinfectious glomerulonephritis is results from an antecedent infection of the skin (impetigo) or throat (pharyngitis) caused by nephritogenic strains of group A beta-hemolytic *streptococci* and is coined as Post Streptococcal Glomerulonephritis (PSGN) [5-7]. In 1941, Seegal and Earl described the concept of nephritogenic strain causing PSGN [8]. The M and T proteins in the bacterial wall have been used for characterizing *streptococci*. Nephritogenicity is mainly restricted to certain M protein serotypes (i.e., 1, 2, 4, 12, 18, 25, 49, 55, 57 and 60) that have shown nephritogenic potential. These may cause skin or throat infections. The specific M types 49, 55, 57 and 60 are most commonly associated with skin infections. Some strain of Group C *streptococci* like *Streptococcus zooepidemicus* have been seen responsible for recent epidemics of APSGN. Hence it can be stated that nephritogenic antigens are present and possibly shared by *streptococci* from several groups [9]. The Incidence of acute post streptococcal glomerulonephritis is more common with antecedent history of pharyngitis or pyoderma [10-12].

Objectives

The objectives of the study is to know the clinicopathological profile of PSGN and its association with sore throat and pyoderma, clinical presentations, various laboratory parameters including Serum C3 level and course of illnesses.

Method

This study was done in the Department of Paediatrics, SCB medical college, Cuttack, a tertiary care referral hospital from October 2014 to September 2016 after obtaining clearance from institutional ethical committee. Total number of case study were 56, who were admitted to indoor of Paediatrics ward of SCB Medical college hospital, Cuttack with the features of PSGN.

Inclusion criteria

The child who satisfy the criteria of PSGN were taken into study. The criteria taken in our study were sudden onset of oliguria, hematuria and proteinuria with presence of hypertension, edema and impaired renal function [2,13].

Exclusion criteria

The child having similar presentation with other systemic disease like secondary nephrotic syndrome, Ascitis of different origin, Congestive Cardiac Failure were excluded with allied investigations.

Methods

It was a direct prospective study. After the children were admitted, a detailed history of present illness, past history and through clinical examination were done in a prescribed performa to rule out any bias. All the investigation are done in the central laboratory, pathology department and biochemistry department of SCB Medical College Hospital, Cuttack. The following examination was being done. 1) Urine: routine and microscopic (including Ph, specific gravity, Benedict's Qualitative Glucose Test, heat coagulation test & culture) 2) Blood: Hb, DC, TLC, ESR, blood urea, serum creatinine, serum electrolyte, serum cholesterol, serum complement-3 (nephelometry method) and ASO Titre (ortho ASO slide test). 3) Throat swab/skin swab for grams stain and culture-sensitivity. 4) X-ray chest, Ultrasound of abdomen (KUB) and 5) Kidney biopsy (in selected case of renal failure).

Observation

The observations were recorded and tabulated (Tables 1-7, Figures 1-3).

Age in years	Male	Female	Total	Percentage
Less than 3 years	2	1	3	5.35
3 to 8 years	24	10	34	60.71
8-14 years	10	9	19	33.92
Total	36(64.28)	20(35.71)	56	100

Table 1: Age and Sex distribution in PSGN.

AGN is most common in between 3-8 years age group, male were more affected than female. Average ratio is M:F =1.8:1.

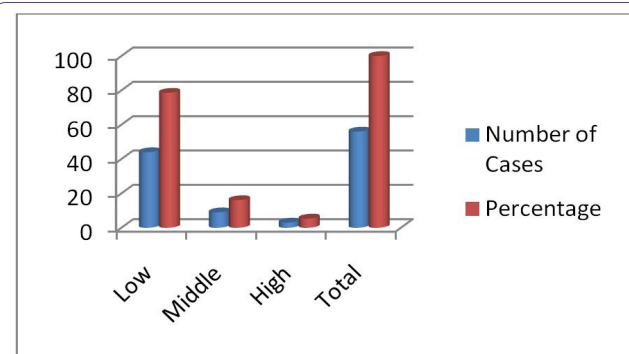


Figure 1: Socioeconomic status of parents of patients of PSGN.

Most cases belong to low socioeconomic group (78.57%). The middle income groups were 16.07% and high income group's were 5.35 %.

Latent period	Sore throat		Pyoderma		Total	
	No	%	No	%	No	%
Less than 7 days	-	-	-	-	-	-
8-14 days	13	27.08	5	10.41	18	37.50
15-21 days	1	2.08	22	45.83	23	47.91
More than 21 days	-	-	8	14.58	7	14.58

Table 2: Latent period of infection and onset of PSGN.

The majority of patients have latent period between 8 to 21 days (85.41%). Patient with sore throat have latent period of 8 to 14 days in majority cases (27.08%). The patient with pyoderma has latent period of 15 to 21 days in majority of cases (45.83%)

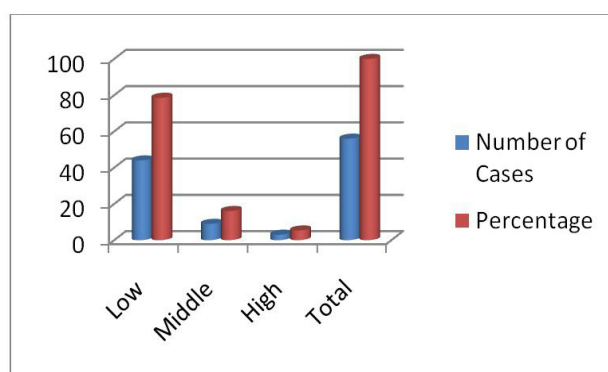


Figure 2: History of preceding infection in PSGN.

In 85.71% cases of PSGN there was history suggestive of infection, out of which history of Pyoderma was 60.71%, Sore throat was 25.00%.

Symptoms	Number of cases	Percentage
Oliguria	53	94.64
Puffiness of face	53	94.64
Pedal edema	34	60.71
Hematuria	32	57.14
Fever	24	42.05
Breathlessness	16	28.57
Headache and vomiting	14	25.00
Palpitation	8	14.28
Convulsion	5	8.92
Altered sensorium	4	7.14

Table 3: Presenting symptoms of PSGN.

The most common presentation was oliguria, and puffiness of face in 94.64 % cases, pedal oedema in 60.71% cases, hematuria in 57.14% cases, fever in 42.05% cases and convulsion in 8.92% of cases.

Presenting signs	Number of cases	Percentage
Hypertension	42	75.00
Oedema	34	60.71
Congestive cardiac failure	7	12.50
Ascitis	7	12.50
Pulmonary oedema	5	8.92
Acute renal failure	3	5.35
Papilloedema	2	3.57

Table 4: Presenting sign of PSGN.

The most common sign in PSGN in our study was hypertension (75%) followed by oedema (60.71%), congestive cardiac failure was seen in 12.50% of cases.

Discussion

In our study commonest presentation of PSGN is between 3-8 years age group (60.71%), male (64.28%) were more affected than female (35.71%), ratio was 1.8:1. Sarkissian A, et al., [14] found similar type of study. We have found in 60.97% cases of PSGN there was history suggestive of infection, most common being the scabies, out of which pyoderma was 60.71%, sore throat was 25%. Popovic- Rolovic

Urine examination finding	Number of cases	Percentage
Albuminuria		
Trace	4	7.14
+	24	42.85
++	15	26.78
+++	10	17.85
++++	3	5.35
Haematuria		
Gross	32	57.14
Microscopic	56	100
Pus cell(more than 5/HPF)	18	32.14
Cast		
RBC Cast	18	32.14
Granular cast	12	21.42
Hyaline cast	9	16.07
Urine Culture		
Positive	9	16.07
<i>Klebsilla</i>	5	8.92
<i>E. Coli</i>	3	5.35
<i>Acenetobacter</i>	1	1.78

Table 5: Urinary finding in PSGN.

All cases had albuminuria and microscopic hematuria (100%) in our study. Gross hematuria was seen in 54.14% cases. Pyoderma was found in 32.14% of cases. RBC cast was present in 32.14% of cases. Urine culture was positive in 16.07% of cases and most of which was infection with *Klebsilla* in 12.85% of cases.

Examination finding	Number of cases	Percentage
Raised ESR	49	87.50
Raised CRP	8	14.28
ASO Titer		
Positive (Total)	26	46.42
Pharyngitis	13	50.00
Pyoderma	7	26.92
No infection	6	23.07
Low Complement 3 level		
Positive	49	87.50
Negative	7	12.50

Table 6: Acute phase reactant level: ESR, CRP, ASO & C3 finding in PSGN.

The ESR was raised in 87.50% of cases; CRP was raised in 14.28% of cases. The ASO titer was positive in 26 (40.42%) patients out of which 13 patients (50%) had pharyngitis, 7 patients (26.92%) had pyoderma. Out of 14 patients with pharyngitis, 13 patients had ASO Positive and out of 34 patients with pyoderma only 7 (26.92%) patients had ASO Positive. Low Complement 3 level was positive in 49 patients (87.50%) and negative in 12.50% patients. Only 5 patients could be followed up after 8 weeks and out of which 4 patients (80%) had complement level came back to normal level.

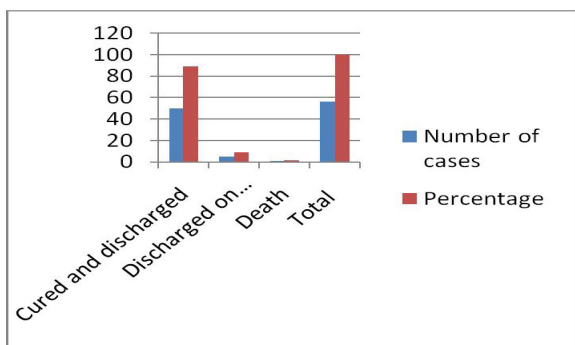


Figure 3: Treatment course and outcome of PSGN.

Complete recovery was seen in 89.28% of cases of PSGN, 5 patients (8.92%) were discharged on request (latter on followed up), one patient died (1.78%) in hospital (after all investigation were done) due to associated sepsis.

Organism isolated	Number of cases	Percentage
Throat Swab		
No growth	6	42.85
Group A Beta haemolytic <i>Streptococci</i>	5	35.71
<i>Klebsilla</i>	3	21.42
Total	14	100
Skin Swab		
No growth	15	44.11
Group A Beta haemolytic <i>Streptococci</i>	11	32.35
<i>Staphylococcus aureus</i>	8	23.52
Total	34	100

Table 7: Throat swab and skin swab culture finding in PSGN.

Throat swab was taken from 14 patients with preceding history of sore throat shows no growth in 42.85% of cases. Group-A Beta haemolytic *streptococci* were seen in 35.71% of cases. *Klebsilla* was found in 21.42% cases. Skin swab was taken from 34 patients with preceding history of pyoderma shows no growth in 44.11% of cases. Group-A Beta haemolytic *streptococci* were seen in 32.35% of cases. *Staphylococcus aureus* was in 23.52% cases

et al., [15] found preceding infection of respiratory tract in 82%, skin infection in 10 % and no infection in 8% of cases. Puri RK, et al., [16] found 67.4% having history of skin infection and 8.9% with respiratory tract infection. Manhas et al., [17] found 60 % cases with history of pyoderma and 20% with sore throat. Dillon et al., [18] have reported pyoderma as the leading cause of PSGN in 66% cases. The finding is consistent with our results. Out of 56 patient have 41 patients latent period between 8 to 21 days (85.41%). Patient with sore throat have latent period of 8 to 14 days in majority cases (27.08%). The patient with pyoderma has latent period of 15 to 21 days in majority of cases (45.83%). Rajajee et al., [19] had reported that the latent period in AGN after streptococcal pharyngitis is 7 to 14 days and that after skin infection is 14 to 21 days. Similar finding about the latent period were reported by Hingalis et al., [20] and Lewy et al., [21]. The most common presentation was oliguria, and puffiness of face in 94.64 % cases, followed by pedal edema (60.71%) hematuria in 57.14% cases and fever in 42.71% cases. This finding was consistent with study done by Puri et al., [16] (85.7%) and Manhas et al., [17] (90%) respectively. Lewy et al., [21] reported edema in 61.1%. Hingalis et al., [20] observed gross hematuria in 77.8% cases. Puri et al., [16] reported 55.7 % cases PSGN with hematuria. Berry et al., observed association of hypertension in 69.1% cases. Puri et al., [16] found hypertension in 74.6% of cases. Our finding is consistent with them. Hypertension was found in 49% of cases by Meharban Singh et al., [22]. Fever, vomiting & headache are encountered in 55.71% & 24.28% respectively. Puri et al., [16] and Manhas et al., [17] have found similar type of presentation.

Atypical presentation like hypertensive encephalopathy was seen in 2.85% of cases where as Puri et al., [16] have encountered 8.8% of cases having hypertensive encephalopathy. We have seen 7.14% of patient having convulsion where as Strauss and Welt et al., [23] reported 5% of PSGN cases having convulsion. Acute renal failure was observed in 5.35% of cases with PSGN in our study. Our finding is consistent with the finding of Earle DP et al., [24] and Sharma BK et al., [25], who found prolonged oliguria in 5-13% of cases but differ from the study done by Manhas et al., [17], who have reported 1.4% cases with anuria. Puri et al., [16] have reported 14.3% cases with ARF. Chugh et al., [26] reported 9.8% incidence of ARF due to PSGN. Papiloedema was seen in 2.85% of cases of PSGN. These cases were associated with convulsion and hypertensive encephalopathy. Similar finding was observed by Manhas et al., [17].

In our study ESR was raised in 87.50% of cases. Manhas et al., [17] reported raised ESR in 56.6% of cases of PSGN. Puri et al., [16] and Berry et al., [27] found raised ESR in about 95% of cases of PSGN. This is at par with our finding. CRP was raised in 17.14% of cases. Valyasevi A et al., [28] reported raised CRP in 15.38% of PSGN. Puri et al., [16] reported it to be raised in much higher percentages of cases.

In our study 46.42% of cases of PSGN had ASO positive. Out of which 50% had pharyngitis and 26.92% had pyoderma. Leung DT et al., [29] in Hong Kong had shown that 78% of PSGN in children had elevated ASO titer more than 200IU/L. Puri et al., [16] have found a raised titer in 75% cases. Rajajee et al., [19] have not found significant raised in ASO titer like our study with pyoderma. Wannamaker LW et al., [30] have found feeble ASO response in patients with pyoderma induced nephritis.

Complement 3 level was positive in 49 patients (87.50%). Popovic-Rolovic M et al., [15] had found low complement C3 level. Shroff KJ et al., [31] found low complement C3 level in 88% of patients. Federic strife et al., [32] and Vijaykumar M et al., [33] found hypocomplementemia in about 90% of patient with PSGN. Complete recovery was seen in 89.28% of cases of PSGN, 5 patients (8.92%) were discharged on request (latter on followed up), one patient died (1.78%) in hospital (after all investigation were done) due to associated sepsis with hypertensive encephalopathy. Puri et al., [16] found mortality rate of AGN to be 1.4%. Devid Charles et al., [34] and Berry S et al., [27] found mortality to be 1%. Our finding is consistent with their findings. Renal biopsy was done in all 3 cases who developed renal failure, it showed the futures of PSGN.

Summary

This work comprised of prospective study of post infectious glomerulonephritis from October 2014 to September 2016. In this study an attempt was made to know the various predisposing factors, presentations, course and outcome of post infectious glomerulonephritis. The numbers of patient taken in our study were 56. PSGN in children was presented more in males than females. Most common age group was 3 to 8 years. Most common preceding infection was infective scabies along with other infected skin lesions. Preceding history of Pharyngitis was found in fewer cases. Latent period between preceding infection and onset of disease varies from 8 to 30 days. The patients with sore throat had shorter latent period than that with skin lesions. oliguria, facial puffiness, pedal edema, hematuria and hypertension were the most common mode of presentation of the disease, observed in this study. Microscopic hematuria and proteinuria were invariably present in all cases. RBC cast in urine was found in about 1/3rd of cases. Most of the patients with PSGN had diminished complement 3 levels, majority of which return to normal on follow-up after 8 weeks. Beta-hemolytic *streptococci* were isolated from either skin or throat swab in 1/3rd of patient studied. All most all cases recovered completely within two to three weeks of admission. Mortality from the disease was very low.

Conclusion

Post streptococcal acute glomerulonephritis is the most common type of acute glomerulonephritis in childhood. In this part of India infected scabies (pyoderma) is the major preceding infection. Serum C3 level can be a useful tool in diagnosing PSGN. Serum C3 level is more sensitive than ASO estimation for diagnosing PSGN. Early and appropriate treatment of scabies and pharyngitis, improvement of socio-economic status and general hygiene can prevent development

of PSGN. Early diagnosis of PSGN by clinical and laboratory investigations, timely and appropriate treatment can result in complete cure.

References

- Eison TM, Ault BH, Jones DP, Chesney RW, Wyatt RJ (2011) Post-streptococcal acute glomerulonephritis in children: clinical features and pathogenesis. *Pediatr Nephrol* 26: 165-180.
- Rodríguez-Iturbe B, Batsford S (2007) Pathogenesis of poststreptococcal glomerulonephritis a century after Clemens von Pirquet. *Kidney Int* 71: 1094-1104.
- Avner ED, Davis ID (2004) Acute post-streptococcal glomerulonephritis. In: Behrman RE, Kliegman RM, Jenson HB (eds.). *Nelson Textbook of Pediatrics* (17th edn). Elsevier Science, Amsterdam, Netherlands.
- Bright R (1836) Cases and observations illustrative of renal disease accompanied with the secretion of albuminous urine. In: Johnson HJ (ed.). *The Medico-chirurgical Review*, S Highley Publishers, University of Michigan, USA. PG no: 338-379.
- Blyth CC, Robertson PW, Rosenberg AR (2007) Post-streptococcal glomerulonephritis in Sydney: a 16-year retrospective review. *J Paediatr Child Health* 43: 446-450.
- Sanjad S, Tolaymat A, Whitworth J, Levin S (1997) Acute glomerulonephritis in children: a review of 153 cases. *South Med J* 70: 1202-1206.
- Sagel I, Treser G, Ty A, Yoshizawa N, Kleinberger H, et al., (1973) Occurrence and nature of glomerular lesions after group A streptococci infections in children. *Ann Intern Med* 79: 492-499.
- Kimmelstiel P (1965) The hump-a lesion of glomerulonephritis. *Bull Pathol* 6: 187.
- Blue Star (2012) *Acute Glomerulonephritis (AGN)*. Pocket Atlas of Human Anatomy (4th Edn). Blue star.
- Kleinman H (1954) Epidemic AGN at salt Lake. *Minn Med* 37: 479
- Dillon HC (1967) Pyoderma and Nephritis. *Annual Review of Medicine* 18: 207-218.
- García-Fuentes M, Arias M (1981) Poststreptococcal glomerulonephritis. *An Esp Pediatr* 15: 166-181.
- Parmar MS (2016) *Acute Glomerulonephritis Updated*. Medscape, New York, USA.
- Sarkissian A, Papazian M, Azatian G, Arikiant N, Babloyan A, et al., (1997) An epidemic of acute postinfectious glomerulonephritis in Armenia. *Arch Dis Child* 77: 342-344.
- Popovic-Rolovic M (1973) Serum C3 levels in acute glomerulonephritis and postnephritic children. *Arch Dis Child* 48: 622-626.
- Puri RK, Khanna KK, Raghu MB (1976) Acute glomerulonephritis in children. *Indian Pediatr* 13: 707.
- Manhas RS, Patwari A, Raina C, Singh A (1979) Acute nephritis in Kashmiri children-A clinical and epidemiological profile (A study of 350 cases). *Indian Pediatr* 16: 1015-1021.
- Dillon HC, Dillon MS (1974) New Streptococcal Serotypes Causing Pyoderma and Acute Glomerulonephritis Types 59, 60, and 61. *Infect Immun* 9: 1070-1078.
- Rajajee S (1990) Post-streptococcal acute glomerulonephritis: A clinical, bacteriological and serological study. *Indian J Pediatr* 57: 775-780.
- Hingalis N (1974) Long term prognosis of AGN. *Am J Med* 56: 52-60.
- Lewy JE, Salinas-Madrigal L, Herdson PB, Pirani CL, Metcalf J (1971) Clinico-pathologic correlations in acute poststreptococcal glomerulonephritis. A correlation between renal functions, morphologic damage and clinical course of 46 children with acute poststreptococcal glomerulonephritis. *Medicine (Baltimore)* 50: 453-501.
- Singh M, Azizi E, Qureshi MA, Arya LS (1984) Clinical profile of acute glomerulonephritis in children. *Indian J Pediatr* 51: 553-557.
- Strauss MB, Welt LG (1971) *Diseases of the Kidney* (2nd edn). Little Brown Publishers, Boston, USA.
- Earle DP, Farber SJ, Alexander JD, Pellegrino ED (1951) Renal Function and Electrolyte Metabolism in Acute Glomerulonephritis. *J Clin Invest* 30: 421-433.
- Sharma BK, Mahakur AC, Datta BN, Mathew MT, Bansal VK (1974) Acute oliguric glomerulonephritis. *J Assoc Physicians India* 22: 581-588.

26. Chugh KS, Narang A, Kumar L, Sakhuja V, Unni VN, et al. (1987) Acute renal failure amongst children in a tropical environment. *Int J Artif Organs* 10: 97-101.
27. Berry S, Prakash K, Srivastava G, Gupta S (1971) Acute glomerulonephritis in children. *Indian Pediatr* 8: 198.
28. Valyasevi A, Sloan JM, Barness LA (1960) C-reactive protein in the serum in nephrosis and acute glomerulonephritis. *Pediatrics* 25: 106-111.
29. Leung DT, Tseng RY, Go SH, French GL, Lam CW (1987) Poststreptococcal glomerulonephritis in Hong Kong. *Arch Dis Child* 62: 1075-1076.
30. Wannamaker LW (1976) Differences between Streptococcal Infections of the Throat and of the Skin. *N Engl J Med* 282: 23-31.
31. Shroff KJ, Ravichandran R, Acharya VN (1984) ASO titre and serum complement (C3) in post-streptococcal glomerulonephritis. *J Postgrad Med* 30: 27-32.
32. Strife CF, Forristal TJ, Forristal J (1994) Serum complement levels before and after the onset of acute post-streptococcal glomerulonephritis. A case report. *Pediatric Nephrology* 8: 214-215.
33. Vijayakumar M (2002) Acute and crescentic glomerulonephritis. *Indian J Pediatr* 69: 1071-1075.
34. Florey DV, Kessner DM, Kashgarian M, Senter MG (1971) Mortality trends for chronic nephritis and infections of the kidney: A clinical and statistical comparison between mortality in New Haven, Connecticut and the United States, 1950-1960. *J Chronic Dis* 24: 71-77.

Supplementary Files

Cast	Culture	Hb(gm/dl)	TLC/cmm of blood	ESR mm fall in 1st hour	ASO	CRP	Blood urea (mg/dl)	Serum creatinine (mg/dl)	Serum cholesterol (mg/dl)	Serum Sodium (meq/l)	Serum Potassium (meq/l)	Serum C3 level (mg/dl)	Throat swab/skin swab culture	CXR/Xray KUB	Outcome
24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39
-	-	8	10000	96	+	-	59	1.3	149	142	5.7	15	-	N	R
-	-	14.43	15800	32	+	+	53	1.4	187	133	3.3	41.6	GAS	A	R
R	KLEB	7.6	8600	80	+	-	58	1.9	160	153	4.8	14.12	GAS	N	R
GR	KLEB	9.9	10800	45	-	-	160	1.8	211	130	5.8	26.8	GAS	N	R
GR	-	8	8600	22	-	-	190	4.8	130	130	4.9	118.8	SA	N	R
HY	-	11	16000	26	+	-	104	4.2	171	140	4.2	45	GAS	N	R
R	-	9.9	8800	8	-	-	62	2.2	146	136	5.6	45	GAS	N	R
GR	-	10.12	10400	24	+	-	29	0.8	136	136	3.6	17	KLEB	N	R
-	-	13	7800	35	-	-	40	1	166	139	3.9	30	GAS	A	LA
R	E.C.	4.8	10000	48	+	-	37	1.2	152	132	5.4	17	-	N	R
-	-	9.62	15000	28	-	-	72	1.4	212	132	5.4	15	GAS	N	R
GR	-	10	9800	67	-	-	30	0.8	236	130	5.3	103.6	-	N	R
R	-	9.2	10800	26	+	+	40	0.9	232	138	5.2	11	GAS	N	R
-	-	10.2	6200	50	-	-	46	1.3	152	136	5.6	79.5	-	N	R
R	CAN	9	8100	50	-	-	84	2.5	178	141	3.9	25	GAS	A	R
R	KLEB	7.16	12000	90	+	-	63	1.5	215	143	3.9	132	-	N	R
-	-	14.9	12450	10	+	-	31	0.4	492	140	5.6	67	GAS	N	R
HY	-	11	16500	46	-	-	40	1.4	188	141	4.8	14.4	GAS	N	R
HY	-	11	9800	90	+	+	21	0.7	116	134	4.6	36.6	GAS	N	LA
R	-	11	14000	26	-	-	26	0.7	154	134	3.3	38.4	SA	N	R
GR	-	13.69	15000	48	-	-	54	1.7	156	137	3.5	15.6	GAS	A	D
R	EC	8.2	12400	48	-	-	78	1.7	178	132	5.8	17.82	-	N	R
-	-	9.2	10800	82	-	-	23	0.7	132	154	3.8	74.32	SA	A	R
HY	-	9.24	12400	29	+	-	47	1.5	176	142	4.6	108.6	-	N	LA
R	-	7.4	8400	7	+	+	66	1.8	136	136	3.6	84.6	GAS	N	LA
GR	-	9.6	10000	32	+	-	172	3	239	132	3.8	98.4	-	N	R
GR	-	8	7000	140	-	-	22	0.9	160	126	4.2	10.62	SA	N	R
R	-	8.2	8200	42	-	-	40	1.1	310	138	1.6	17.42	-	N	R
R	-	7.8	7800	46	-	-	48	1.3	210	138	3.5	22.24	-	N	R
R	-	10.4	5600	52	+	-	36	0.8	184	142	4.2	22.84	-	N	R
GR	KLEB	10.2	10200	45	+	+	46	1.3	186	141	4.6	24.8	-	N	R
HY	-	10.8	6800	9	+	-	68	1.6	130	136	5.2	66.4	GAS	N	R
GR	-	9.6	9200	56	-	-	102	1.8	212	134	4.8	76.42	SA	A	R
HY	-	9.8	8600	33	+	-	96	1.8	186	128	3.8	28.42	-	N	R
-	-	10.6	14200	110	-	-	82	1.6	126	132	5.6	82	-	N	R
HY	-	12.6	11200	26	-	-	48	1.4	148	142	5.2	28.42	GAS	N	R
-	-	9.6	6500	19	-	-	52	1.3	184	141	4.4	82	-	N	R
R	-	7.6	7200	18	-	-	32	0.6	180	140	3.8	46.44	-	N	LA
R	-	9.4	8600	76	-	-	18	0.4	202	138	3.7	32.1	-	N	R
-	-	10.2	10400	74	+	-	56	1.4	154	138	4.2	24.06	GAS	N	R
-	-	9.4	9300	56	-	-	12	0.4	148	136	4.9	36.8	-	N	R
R	-	7.4	7800	10	-	-	52	1.4	148	134	4	24.06	SA	A	R
-	-	8	8000	54	+	-	54	1.8	256	156	5.6	14.14	GAS	N	R
GR	EC	12	6500	48	+	+	106	2.2	146	136	4.2	15	-	N	R
R	-	9.6	5600	47	+	-	108	2.1	180	138	4.9	110	GAS	A	R
HY	-	7.6	4800	46	+	-	112	2	310	142	3.4	16	SA	N	R
R	-	9.6	10000	6	-	-	20	0.8	200	140	5.2	28.6	-	N	R
R	KLEB	10.4	9200	54	-	-	30	1.3	210	128	5.8	140	-	N	R

GR	-	10.6	9000	33	+	+	32	0.4	206	138	4.8	42.4	KLEB	N	R
GR	-	9.4	4800	31	-	-	132	2.4	206	146	3.8	56.8	SA	N	R
R	-	13	6200	29	-	-	84	1.6	201	145	3.6	17.42	-	N	R
-	-	9.4	7200	24	+	+	34	0.6	184	138	5.9	22.22	GAS	N	R
HY	-	9.6	8400	31	-	-	24	1	186	140	3.9	38.4	-	N	R
-	-	10.4	6400	92	-	-	84	1.6	174	146	5.2	15	-	N	R
-	-	10.6	10000	42	-	-	20	0.8	210	144	4.8	17	-	N	R
-	-	11	7200	28	-	-	36	0.6	176	142	3.9	38.6	SA	N	R