ORIGINAL ARTICLE

SPECTRUM OF ACUTE GLOMERULO NEPHRITIS IN CHILDREN AT GOVERNMENT GENERAL HOSPITAL, ANANTAPURAMU

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ABSTRACT: AIM: Aim of the study is to study the spectrum of AGN in children and to assess the age, sex and seasonal incidence and prognostic factors. Acute glomerulonephritis is one of the most common condition seen in children. The study group included 50 children. In most of the children presenting complaints were puffiness of face, haematuria and oliguria. METHODS AND MATERIALS: Fifty children who were admitted in the government hospital during the period of September 2013 to January 2015 were included in the study. RESULTS: The maximum admissions were seen from the months of September to December. Common age group was between 3 and 8 years. Rare age group was below 2 years. Hypertension was noticed in 32 out of 50 children. Albuminuria and hematuria were commonest urinary abnormalities. CONCLUSION: acute glomerulonephritis is less common below 2 years. Hypertension was of varying degree. Cardiomegaly by x-ray was an added feature. KEYWORDS: Acute glomerulonephritis, Hypertension, Hematuria.

INTRODUCTION: Acute glomerulo nephritis also known as acute nephritic syndrome. It is the most common form of glomerulonephritis in childhood. It is the best understood disease. A specific cause, streptococcal infection can be established in majority of the cases. Clinical course of the disease is well defined and prognosis is generally good. AGN consists of sudden onset of edema, hematuria, and cases. Hypertension, oligoanuria and varying degree of proteinuria¹. It may follow infection with a variety of bacteria, viruses and parasites. Or be a part of systemic disease. AGN is regarded as primary disease of the kidney. The main cardiovascular change is cardiac failure. It is often seen in infants and children. In AGN the myocardial function is not commonly depressed. Myocardial failure may occur secondary to arterial hypertension, severe electrolyte disturbances, anaemia. In some, cardiac failure has been present in the absence of significant hypertension. Congestion in these cases being secondary to myocarditis. Since histological changes in cardiac muscle have been reported in some patients with acute nephritis. The histological findings are probably due to oedema with no significant cellular destruction or inflammatory changes in the myocardium. Cardiac failure whether due to hypertension or carditis, the heart is deprived of its contractility activities with reduction of cardiac output. Causes of acute nephritic syndrome are post infective, primary glomerulonephritis, secondary glomerulonephritis and others.

Immune reaction within the glomerulus is responsible for majority of nephritis. The role of immunological mechanisms in acute glomerulonephritis was suggested in 1908 by schick. Evidence in support of this concept is the depression of serum complement during early stages of the disease.² Although the causes of glomerulonephritis is still at a basic level inflammation is
thought to be autoimmune mediated and involve both cellular and humoral immune systems. In acute glomerulonephritis antibodies are formed not to an endogenous antigen but to an exogenous streptococcal antigen but to an exogenous streptococcal antigen planted in the glomerulus at the time of the infection. Whatever is the initial events, common inflammatory pathways follow with activation of the coagulation and complement cascades and production of pro inflammatory cytokines. Four possible basic mechanisms initiating the injury are fixation of antibody to glomerular antigen, deposition of preformed immune complex, formation on immune complex to neo-antigen and modification of glomerular basement membrane. The inflammatory damage to the filtration surface due to acute glomerulonephritis causes shunting of blood, which causes reduction in glomerular filtration rate manifesting as uraemia clinically. The inflammation of glomerulus causes haematuria. To compensate for the reduction in GFR, the ultrafiltration pressure within glomeruli increases. The reduction in GFR causes a reduction in absolute proximal tubular reabsorption. The resultant reduced distal delivery, in order to maintain positive sodium balance is associated with critical Sodium intake by constant or increased distal reabsorption. The maintained positive Sodium balance and resultant extracellular expansion leads to oedema, increased cardiac output, arterial hypertension and congestive cardiac failure. The extra cellular expansion is compensated by reduction in plasma Renin and Aldosterone.

The streptococcal infection of the upper respiratory tract and skin is known to lead to post streptococcal glomerulonephritis. There is always a latent period of 8-14 days for pharyngitis and 14-21 days for skin infection. History of streptococcal infection may precede clinical disease onset by 1-2 weeks for throat infections and 3-4 weeks for skin infections. Strains of streptococci capable of producing AGN are called nephritogenic strains. These include several protein M types, 4, 12, 25 and 49 being the commonest. Strains of pharyngitis include 1, 3, 4, 6, 12, 25, 49. Strains of skin infection are 2, 49, 56, and 51. A.G.N. is a disease affecting both kidneys. Recovery from acute stage is complete. In overall over 80% of these patients completely recover without residue. The remaining go in for complications. The complications are mainly cardiac failure, hypertensive encephalopathy, multiple recurrences leading to renal failure and pulmonary oedema.

The importance of supportive therapies in acute glomerulonephritis cannot be overemphasised. Tight blood pressure control, appropriate use of diuretics and control of hyperkalaemia, uraemia and fluid overload, if necessary by dialysis are quite literally lifesaving. Management mainly consists of bed rest, salt and fluid restriction. Daily recommended allowance of proteins is given. If renal failure is present protein is restricted. In the presence of pulmonary oedema early use of oxygen, IV Frusemide, intermittent positive pressure ventilation is very useful. If pulmonary oedema is refractory peritoneal dialysis is indicated. Anti-hypertensive to control hypertension is indicated. Dialysis is indicated if the child is presenting with anuria of more than 24 hours duration and pulmonary oedema. Antibiotics are given for a period of 10 days. Long term follow up is required.

METHODS AND MATERIALS: Fifty children with acute glomerulo nephritis who were admitted at Government General Hospital between September 2013 to January 2015 were included in this study.
INCLUSION CRITERIA: All children with clinically diagnosed A G N.

EXCLUSION CRITERIA: Those children admitted as A G N initially and later found to have chronic glomerulo nephritis with features of C R F were excluded. Those children with history antecedent renal disease were also excluded from the study.

The age of the children ranged from 1 to 12 years. After admission informant consent was taken. Initial evaluation included a complete medical history and thorough physical examination. Laboratory tests included Haemoglobin, total differential count, detailed urine examination which included analysis for protein and deposits, 24 hours urinary proteins. Before treatment, blood sample was sent for electrolytes urea, creatinine, as cholesterol, serological tests such as ASO titre, C R P, X- Ray chest, ultrasound abdomen and ECG as indicated were also taken. The blood pressure of each patient was recorded at least twice a day. When diastolic pressure exceeded 95th percentile for age the child was taken as hypertensive. All the patients were started on no added salt. Those who were hypertensive on admission received frusemide and anti-hypertensive. Dosage was increased based on level of blood pressure and depending on initial response. Those children with diastolic blood pressure exceeding 100 mm of Hg are subsequently were put on sublingual Nifedipine in the dose of 0.25 mg/kg/ dose. All patients received antibiotics penicillin/ampicillin for 7 – 10 days.

Systematic analysis of data 2015, fifty cases of acute nephritic syndrome were admitted in Government general hospital and considered as the study group.

Seasonal incidence maximum admissions were made during the month of September to December as shown in the table no. 1.

RESULTS AND DISCUSSION: Incidence: Out of the total incidence from September 2013 to January 2015, fifty cases of acute nephritic syndrome were admitted in Government General Hospital and considered as the study group.

Seasonal incidence Maximum admissions were made during the month of September to December as shown in Table no. 1.

SEASONAL INCIDENCE:

| Month    | Year | No. of cases | % |
|----------|------|--------------|---|
| September| 2013 | 3            | 6 |
| October  | 2013 | 3            | 6 |
| November | 2013 | 4            | 7 |
| December | 2013 | 4            | 8 |
| January  | 2014 | 2            | 4 |
| February | 2014 | 4            | 8 |
| March    | 2014 | 2            | 4 |
| April    | 2014 | 2            | 4 |
| May      | 2014 | 2            | 4 |
| June     | 2014 | 2            | 4 |
AGE BY SEX:

| Age   | Male | Female |
|-------|------|--------|
| 2 yrs | 01   | 1      |
| 2-5 yrs | 14  | 10     |
| 5-10 yrs | 12  | 9      |
| >10 yrs | 2   | 1      |
| Total  | 29  | 21     |

Male: Female Ratio = 1.4:1.

BLOOD PRESSURE:

| Hyper tension | < 3 years | 3-6 years | 6-9 years | 9-12 years |
|---------------|-----------|-----------|-----------|------------|
|               | Male | Female |
| Normal        | 4    | 2       | 2         | 4          |
| Mild          | 2    | 2       | 4         | 2          |
| Moderate      | 1    | 1       | 2         | 1          |
| Severe        | 0    | 1       | 1         | 1          |

LAB INVESTIGATIONS:

| Investigations | No. of cases | Percentage |
|----------------|--------------|------------|
| 1. Urine       |              |            |
| Albuminuria    |              |            |
| Mild +         | 45           | 90         |
| Mod 2+         | 28           | 58         |
| Heavy 3+       | 14           | 28         |
| Heamaturia     | 2            | 4          |
| (microscopic)  | 48           | 96         |
### BLOOD UREA:

| Value        | No. of Cases | Percentage |
|--------------|--------------|------------|
| < 20 Mg      | 5            | 10         |
| 20-40 Mg     | 28           | 56         |
| 40-100 Mg    | 15           | 30         |
| More than 100 Mg | 2     | 4          |

Table 5

### SERUM CREATININE:

| Value        | No. of Cases | Percentage |
|--------------|--------------|------------|
| < 1 mg.      | 34           | 68         |
| 1-2 mg.      | 15           | 30         |
| 2-4 mg.      | 1            | 2          |

Table 6

### SERUM ELECTROLYTES:

| Value       | No. of Cases | Percentage |
|-------------|--------------|------------|
| Sodium      |              |            |
| <130 meg/L  | 2            | 4          |
| 130-155 meg/L | 48          | 96         |
| Potassium   |              |            |
| <3 meg/L    | 1            | 2          |
| 3.55 meg/L  | 47           | 94         |
| >5.5 meg/L  | 2            | 4          |
| Bicarbonate |              |            |
| >15 meg/L   | 48           | 96         |
| <15 meg/L   | 2            | 4          |

Table 7

### SERUM CHOLESTEROL:

| Value     | No. of Cases | Percentage |
|-----------|--------------|------------|
| < 200 mg  | 45           | 90         |
| >200 mg   | 5            | 10         |

Table 8
AGE AND SEX DISTRIBUTION: Post streptococcal glomerulonephritis is common in 3–8 years and rare below 2 years as shown in Table number 2.

CLINICAL PROFILE: Analysis of the symptoms in 50 cases of A G N, majority of them presented with puffiness of face. Besides oliguria, fever, breathlessness, head ache, vomitings, abdominal distension, convulsions, hematuria and altered sensorium were other presenting symptoms. Other common findings include anaemia, hepatomegaly, signs of pulmonary oedema and congestive cardiac failure. Hypertension was recorded in 32 children. The hypertension is classified into mild, moderate and severe as depicted in table. Albuminuria (89%), and hematuria (96%) were commonest urinary abnormalities. the increase in A S O titre begins 1 -3 weeks after infection.

CONCLUSION: in this study from September 2013 to January 2015 out of 50 cases of AGN, the peak incidence was from September to December (59%). Manhas et al study of 250 children has shown peak incidence from August to November. In our study, out of 50 cases only 2 cases (4%) are below the age of 2 years. 45 cases (90%) are between the ages of 2 and 10 years. Only 6% are above the age of 10 years. So the maximum incidence is between 2 and 10 years. In this study the most common presenting symptom is puffiness of face 79%. The other presenting symptoms are oliguria 63%, haematuria 26% and oedema feet 39%. Blood pressure recordings are taken many times in a day and classified as normal, mild and severe. Hypertension was present in 32 children out of 50. Mild hypertension in 18 children (26%). Moderate hypertension in 10 children (20%). And severe hypertension in 4 children (8%) was noted. Roentgenogram analysis of 50 children with AGN in this study showed 60% normal study, cardiomegaly 34% and pneumonic changes in 6%.

SUMMARY: AGN is less common below 2 years. Common between 3 and 8 years. Pyoderma is the common associated feature. Typical features are not present in all the cases. Hypertension is absent in 36.3% children. Haematuria in 4%. Azotemia and renal failure need not be present in every child with AGN. Hypertension will be of varying degree. Cardiomegaly by x–ray is an added feature.

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