Study of Urinary Microalbumin and Allied Renal Function Tests in Children with Acute Glomerulonephritis

SHAHID A. MUJAWAR, JAYASHREE V. GANU AND VINAYAK W. PATIL

ABSTRACT

Renal function assessment is important for clinical management of patients and for intervention studies. It has specific issue in children because most parameters are influenced by age, by body size, and by the level of renal function itself. In addition, technical problem might occur owing to vascular accesses, bladder control attainment, urine collection, and other factors. The renal function tests of 30 children with acute glomerulonephritis (AGN) were assessed. The alterations of serum urea, creatinine, uric acid, sodium, potassium and also urinary urea, creatinine, total proteins, microalbumin levels in AGN children as compared to that of control group. Alteration in these tests may be because of the strong inflammatory response in the glomerular basement membrane due to streptococcal infection, other infection or other underlying diseases.

Key words: Acute glomerulonephritis, Microalbumin, Renal function tests

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Acute glomerulonephritis (AGN) is the most common glomerulopathy among children. Typical features of the disease are acute onset with appearance of hematuria, edema and hypertension. In typical cases the diagnosis is easily established upon the history of antecedent streptococcal infection, acute onset of nephritic signs, transitory depression of C3 complement level, isolation of group A beta hemolytic streptococci from throat swab or pyoderma, and significant titer of streptococcal antibodies.

Infectious agents remain the most common cause inciting antigen associated with acute immune complex-mediated glomerulonephritis (AICGN) in which IgG and C3 are usually deposited in granular fashion along the glomerular basement membrane and in the mesangium. Most deposits disappear after a few weeks, but mesangial deposits may persist for prolonged periods. Antigens involved in the formation of nephritogenic immune complexes are thought to be the products of infectious agents (for example, nephritogenic streptococci). Glomerular immune complex deposition results in complement activation, release of vasoactive amines and activation of kinin or coagulation systems. Release of chemotactic factors results in polymorphonuclear leukocyte infiltration and acute glomerular injury.

This study was planned to determine concentration urea, creatinine, uric acid, sodium, potassium in serum and urea, creatinine, total proteins, microalbumin in urine in patients with AGN. The information of this marker of kidney among AGN in Western Maharashtra would allow medical practitioners to better manage their patients to prevent complications, improve life expectancy, and quality of life.

MATERIALS AND METHODS

In this prospective study, 30 children (mean age = 8.14 ± 2.08 years) with acute glomerulonephritis (AGN) (twenty boys, ten girls) were admitted in pediatric ward, Padmbhushan Dr. Vasantdada Patil General Hospital, Sangli and General Hospital, Miraj and composed the study group. The control group consisted of 30 children matching in age and sex without a history of acute illness or renal disease.

Inclusion criteria:

Patients with symptoms and signs suggestive of AGN supported by routine investigations like Hb, CBC, ESR, blood pressure, blood sugar and urine report.

Exclusion criteria:

Patients with cardiac disease, hepatic disease, diabetes mellitus, septicemia, and
human immunodeficiency virus (HIV) infection were excluded from the study.

The study was conducted as per approval of institutional ethical committee.

Control group:
In this group, 30 control of identical age and sex without any disease were enrolled.

Sample collection and analysis:
Blood collection:
Venous blood samples were collected in test tube with aseptic precautions. After 2 hours of collections sample was centrifuged at 3000rpm for 5 minutes. Serum was separated and collected in polythene tube with cork. The sera with no sign of hemolysis was used for the analysis of urea, creatinine, uric acid and electrolytes.

Urine collection:
After collection of blood sample early morning urine sample were collected on the next day. To avoid contamination, morning urine sample were collected in sterile 10ml polythene wide mouth container. The fresh urine sample was collected and part of which diluted for urea and creatinine estimation. Remaining urine was taken into plane polythene tube with cork as well as sodium azide as a preservative, for estimation of total protein and microalbumin. Also urine centrifuged and clear supernatant was used for estimation of microalbumin.

Serum urea was estimated by DAM method. The concentration of creatinine was measured by Jaffe’s colorimetric method. Serum uric acid was estimated by the method of Uricase - PAP method. Serum electrolytes concentrations were measured by flame photometer. Urinary total proteins and microalbumin were estimated by pyrogallol red method and immunoturbidimetric method, respectively. The concentration of total proteins and microalbumin in urine were expressed as µg/mg urinary creatinine.

Statistical analysis:
All observations were tabulated and analyzed Numerical variables were reported in terms of mean and standard deviation. Statistical analysis of results was done by normal ‘z’ test. In this analysis, variables showing p value less than 0.001 was considered to be statistically highly significant respectively.

RESULTS AND DISCUSSION
Alterations in urine profile when 30 controls were compared with an equal number of acute glomerulonephritis (AGN) children is shown in Table 1. Urinary urea and creatinine excretion were decreased (p < 0.001) significantly whereas excretion of total proteins, and microalbumin levels showed highly significant increase (p < 0.001).

Table 1 : Urine profile: renal function tests in controls and children with AGN

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Acute glomerulonephritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microalbumin (µg/mg creatinine)</td>
<td>24.57 ± 3.99</td>
<td>195 ± 53**</td>
</tr>
<tr>
<td>Total protein (µg/mg creatinine)</td>
<td>78.82 ± 45.33</td>
<td>1298 ± 448**</td>
</tr>
<tr>
<td>Creatinine (mg/dL)</td>
<td>88.11 ± 10.39</td>
<td>62.4 ± 5.15**</td>
</tr>
<tr>
<td>Urea (Gm/dL)</td>
<td>14.07 ± 1.83</td>
<td>8.28 ± 1.28**</td>
</tr>
</tbody>
</table>

** indicates significant of value at P = 0.001

Table 2 depicts changes in serum profile when 30 controls were compared with an equal number of AGN children. As can be observed, highly significant increases (p< 0.001) was observed in urea, creatinine, uric acid, and potassium levels. Concentration of serum sodium which showed a highly significant decrease (p < 0.001).

The kidneys play a central role in the homeostatic mechanism of the human body, and reduced renal function strongly correlates with increasing morbidity and mortality. Biochemical investigations, both routine and specialized, are an important part of the clinician’s diagnostic armamentarium and investigations from the nephrology wards. All admitted patients with AGN were given diuretic lasix (furosemide) as an antihypertensive drug.

Table 2 : Serum profile: renal function tests in controls and children with AGN

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Acute glomerulonephritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea (mg/dL)</td>
<td>24.2 ± 1.44</td>
<td>48.2 ± 11.4**</td>
</tr>
<tr>
<td>Creatinine (mg/dL)</td>
<td>0.68 ± 0.081</td>
<td>1.98 ± 1.02**</td>
</tr>
<tr>
<td>Uric Acid (mg/dL)</td>
<td>4.78 ± 0.28</td>
<td>7.53 ± 1.80**</td>
</tr>
<tr>
<td>Na⁺ (mmol/L)</td>
<td>141 ± 2.27</td>
<td>132 ± 6.12**</td>
</tr>
<tr>
<td>K⁺ (mmol/L)</td>
<td>4.01 ± 0.22</td>
<td>4.8 ± 0.72**</td>
</tr>
</tbody>
</table>

** indicates significant of values at P = 0.001

Urinary microalbumin:
Urinary microalbumin was found to be elevated in patients with AGN as compared to that of control. The elevation (p<0.001) observed was highly significant. The mean urinary microalbumin level in patients with AGN was 195 ± 53 µg/mg creatinine and that of control was
24.57 ± 3.99 µg/mg creatinine (Table 1). All of the patients with AGN were suffering from hypertension. Microalbuminuria appears to be a predictor of future development of clinical renal disease in patients with hypertension. Similar to present findings Sesso et al. (2005) showed that patients with AGN have hypertension, reduced renal function and increased microalbuminuria\(^\text{12}\).

**Urinary total proteins:**

In the present study, the urinary total proteins excretion in AGN patients was 1298 ± 448 µg/mg creatinine and that of control was 78.82 ± 45.33 µg/mg creatinine (Table 1). In AGN; granular deposits containing immunoglobulins and complement component have been demonstrated on the glomerular membrane. In such progressive renal diseases, filtration of relatively small proteins, including albumin is lost first; thus, albumin levels in urine increased\(^\text{3}\).

**Urinary urea and creatinine:**

Result shows highly significant fall (p<0.001) in urinary urea and creatinine levels in patients with AGN as compared to control group. The mean urinary urea and creatinine level in patients with AGN were 8.28 ± 1.28 mg/dL and 62.4 ± 5.15 mg/dL respectively and that of control group were 14.07 ± 1.83 mg/dL and 88.11 ± 10.39 mg/dL, respectively (Table 1). In AGN patients decreased excretion of nitrogenous compounds was observed as a result of reduced GFR and decreased tubular function. This may lead to retention of urea and creatinine.

**Serum urea and creatinine:**

In the study, serum urea level in AGN patients showed highly significant increase (p<0.001) when compared with those of control group. The mean serum urea level of AGN patient was 48.2 ± 11.4 mg/dL and that of control group was 24.2 ± 1.44 mg/dL. Serum creatinine concentration was significantly increased (p<0.001) in AGN patients when compared to that of controls. The mean serum creatinine concentration levels of AGN patient was 1.98 ± 1.02 mg/dL and that of control was 0.68 ± 0.081 mg/dL (Table 2).

The significantly elevated mean levels of serum urea and creatinine indicate impaired kidney function in children with AGN. Sarkissian et al. (1997) also observed increased serum creatinine concentration in children with AGN\(^\text{11}\). In the study, in approximately 1/3\(^\text{rd}\) children with AGN, serum urea and creatinine levels were raised above the normal range. This showed more reduction in GFR, in these children. In the remaining 2/3\(^\text{rd}\) patients the reduction in GFR was not sufficient to raise serum urea and creatinine levels above the normal range.

**Serum uric acid:**

Result of the study indicated highly significant increase (p<0.001) in serum uric acid level in patients with AGN as compared to that of control group. The mean serum uric acid concentration in APSGN was 7.53 ± 1.80 mg/dL and that of control group was 4.78 ± 0.28 mg/dL (Table 2). Renal retention of uric acid may occur in the acute renal diseases like AGN or as a consequence of administration of drugs: diuretics in particular\(^\text{8}\). All the 30 patients with AGN were given diuretic frusemide.

**Serum sodium:**

In the present study, the patients with AGN showed highly significant (p<0.001) reduction in the mean serum sodium levels when compared to control group. The mean serum sodium level of AGN patients was 132 ± 6.12 mmol/L and that of control was 141 ± 2.27 mmol/L (Table 2). Out of the 30 patient with AGN, 12 patients were hyponatremic (serum Na\(^+\) = 136 mmol/L). Hyponatremia presented by these patients may be secondary to dilution\(^\text{5}\).

**Serum potassium:**

Serum potassium in AGN patients showed highly significant increase (p<0.001) as compared to that of control group. The mean serum potassium in patients with AGN was 4.80 ± 0.72 mmol/L and that of control was 4.01 ± 0.22 mmol/L (Table 2). Out of 30 children with AGN 10 patients were hyperkalemic (serum K\(^+\) = 5.0 mmol/L). Hyperkalemia of varying severity was observed.

In this preliminary study, it was observed that the renal function tests and some allied test altered in acute illness of childhood; particularly in AGN. Changes in these biochemical parameters may affect the physiological processes like acid-base balance, maintenance of plasma volume, and functions of kidney. In the management of hospitalized sick child, if the pediatrician gets information about abnormalities of these biochemical parameters; it will not only help him for rapid diagnosis and immediate treatment but also for preventing the life threatening complications. It was concluded that biochemical tests are of paramount importance in treatment of AGN. Further study with more number of patients and follow up experiments will confirm this. Hence, it is necessary to perform these tests routinely to avoid progression of end stage renal disease of children.
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