Study of Laboratory Profile of Acute Post Streptococcal Glomerulonephritis at the Time of Presentation in Children

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ABSTRACT

Introduction: Acute glomerulonephritis (AGN) is heralded by acute onset of edema, hematuria, and hypertension, which is usually associated with oliguria and azotemia. Acute poststreptococcal glomerulonephritis (APSGN) is the most common type of in-patient glomerulonephritis (GN) in childhood. Most of the electrolyte disturbances occur particularly in the presence of anuria or oliguria. In 60-85% of children, RBC casts may be seen if sediment from fresh urine specimen is examined in an acid PH. The immunologic response to streptococcal pharyngitis is different from that to streptococcus induced pyoderma. Eighty percent of the untreated cases will have a fourfold rise in antistreptolysin O (ASO) titer.

Materials and methods: The present study was conducted on 50 cases of APSGN admitted to the pediatric wards over a period of one year. All children of 3-12 years of age group presenting with acute onset of edema, oliguria and hematuria with or without hypertension and proteinuria, along with or without any evidence of antecedent streptococcal infection are selected. Complete lab investigations were done with special reference to serum electrolytes, ie sodium and potassium.

Results: Serum sodium and potassium are negatively correlated as potassium increases the sodium decreases in most of the cases. Most of the patient’s potassium level is between 3.5 to 5 meq/lt, and the serum sodium level is between 135-145 meq/lt in 50% of the cases and less than 135 meq/lt in another 50% of the cases. Seventy percent of the cases had normal haemoglobin and only 30% of the cases were anemic (<10gm %). It is found that when the blood urea increases there is moderate increase in serum creatinine. But most of the cases have blood urea between 25-45mg%. The co-variation between blood urea and serum creatinine is 5.328 . The majority of the cases have blood urea < 45mg% and creatinine < 1mg% and it accounts to 60% of the cases. In the cases where ASO is more than 200IU the ratio of occurrence of the site of infection of skin, sore throat and no infection is 5:4:1 and in the ASO < 200IU all the 3 groups were identical.

Conclusion: Acute glomerulonephritis is one of the commonest renal disorders in children. Although majority of cases manifest typically with edema, oliguria and hematuria, atypical manifestations are not uncommon. Gross alterations of serum electrolytes do not occur in APSGN, though occasionally hyperkalemia is serious enough to cause death.

Key words: Acute glomerulonephritis, Electrolyte abnormalities, ASO titer.
INTRODUCTION

Acute glomerulonephritis (AGN) is heralded by acute onset of edema, hematuria, and hypertension, which is usually associated with oliguria and azotemia. (1,2) Acute poststreptococcal glomerulonephritis (APSGN) is the most common type of in-patient glomerulonephritis (GN) in childhood. (3,4) It typically develops 1-3 weeks after an episode of acute bacterial infection commonly by group A beta hemolytic streptococci. Recently other organisms like staphylococcus and other gram negative organisms also emerging as major causative agents. (5) Many studies have been conducted to study the changes in the serum electrolyte profile. Most of the electrolyte disturbances occur particularly in the presence of anuria or oliguria. A number of factors influence sodium and chloride concentration in blood. Impaired renal function and ingestion of salts by the patient tend to elevate sodium and chloride concentration, while hyponatremia may result due to continued intake or administration of hypotonic fluids during severe oliguria or anuria. The potassium level in APSGN may be normal or elevated due to retention. Hyperkalemia is rare in APSGN and may occur only when the patient has developed anuria and decreased renal excretion of potassium and accentuated by metabolic acidosis. Rarely vomiting and diarrhea may produce hypokalemia. Burl R. Donn conducted a study and came to the conclusion that hyperkalemia in APSGN is the manifestation of hyporeninemic-hypoaldosteronism. (6)

Hematuria is present in almost all cases and may be gross or microscopic; urine colour may be light brown to reddish brown. In 60-85% of children, RBC casts may be seen if a sediment from fresh urine specimen is examined in an acid PH. WBC cast, hyaline cast, granular cast and renal tubular epithelial casts may also be seen. The majority of RBCs are small and dysmorphic with irregular cell membrane. A mild normochromic anemia is seen due to hemodilution and low grade hemolysis. A mild leukocytosis with polymorphonuclear neutrophils is usually present. Thrombocytopenia has also been reported. (7) Glomerular filtration rate (GFR) and renal blood flow are probably always decreased. If clinically evident, this usually leads to elevated serum urea nitrogen and creatinine. The immunologic response to streptococcal pharyngitis is different from that to streptococcus induced pyoderma. Eighty percent of the untreated cases will have a fourfold rise in antistreptolysin O (ASO) titre. (8) In pyoderma anti DNAase B titre is a better reflection of streptococcal infection. (9) Measurement of serum complement, particularly C3 and C4 is useful as it is usually decreased. This low C3 levels usually return to normal within 6-12 weeks. In a study conducted by Milan Popovic, shows that during the first 4 weeks of the disease, 90% of the patients had low C3 and 10% had normal values. (10)

MATERIALS AND METHODS

The present study was conducted in the department of paediatrics, Sri Siddhartha Medical college hospital, Tumkur, Karnataka, India. The study includes a clinical and detailed systematic evaluation of 50 cases of APSGN admitted to the pediatric wards over a period of one year. All children of 3-12 years of age group presenting with acute onset of edema, oliguria and hematuria with or without hypertension and proteinuria, along with or without any evidence of antecedent streptococcal infection are selected. Children having history suggestive of renal and cardiac disease in the past were excluded from the study. A detailed clinical
examination was done. Complete lab investigations were done with special reference to serum electrolytes, i.e. sodium and potassium. The results were tabulated and analysed by using the SPSS software for Windows (version 16.0).

**RESULTS**

Serum sodium and potassium are negatively correlated as potassium increases the sodium decreases in most of the cases. Only in few cases the order is reversed. The correlation coefficient between sodium and potassium is -0.4509. Most of the patient’s potassium level is between 3.5 to 5 meq/lt, and the serum sodium level is between 135-145 meq/lt in 50% of the cases and less than 135 meq/lt in another 50% of the cases.

<table>
<thead>
<tr>
<th>Serum sodium (meq/lt)</th>
<th>Serum potassium (meq/lt)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;135</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>135-145</td>
<td>20</td>
<td>2</td>
</tr>
<tr>
<td>&gt;145</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>2</td>
<td>41</td>
</tr>
</tbody>
</table>

In the total cases the ratio of severe hypertension: significant hypertension: normotensive is 3:1:1. It is accepted using $X^2 = 0.5333$, $P=0.7659 > 0.05$. The same test was applied to the hyponatremia < 135meq/lt, and found that the test was satisfied with $X^2 =0.4102$ $P=0.8145 >0.05$. The same test also holds good in normal sodium values with $X^2 = 1.9999$ $P=0.3678 > 0.05$. Only one case of hypernatremia was reported.

<table>
<thead>
<tr>
<th>Blood pressure</th>
<th>Hyponatremia &lt;135meq/lt</th>
<th>Normal sodium levels 135-145meq/lt</th>
<th>Hypernatremia &gt;145meq/lt</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normotensive 9</td>
<td>6</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Significant hypertension 12</td>
<td>6</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Severe hypertension 29</td>
<td>14</td>
<td>15</td>
<td>0</td>
</tr>
</tbody>
</table>

Fifty percent of the study cases come under the normal potassium levels and the ratio of blood pressure –severe hypertensive: significant hypertensive: normotensive is 5:3:2. It is accepted $X^2 =1.3252$, $P=0.5155 > 0.05$.

Seventy percent of the cases had normal haemoglobin and only 30% of the cases were anemic (<10gm %). $X^2= 0.2619$, $P= 0.60880 > 0.05$. Only 10% of the cases had normal ESR and 90% of the cases had rise in ESR. $X^2= 0.2222$, $P= 0.6373> 0.05$. Thirty percent of the cases had a higher WBC count more than 11,000/ mm$^3$.

Macroscopic hematuria is present in 76% of the case and microscopic hematuria is present in 100% of the cases. The urine albumin level 1+, 2+ and 3+ is present in the ratio 6:3:1. $X^2=0.4999$ $P=0.7788 > 0.05$.
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<table>
<thead>
<tr>
<th>Urine examination</th>
<th>No of patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macroscopic hematuria</td>
<td>38</td>
<td>76</td>
</tr>
<tr>
<td>Microscopic hematuria</td>
<td>50</td>
<td>100</td>
</tr>
<tr>
<td>Albumin +</td>
<td>29</td>
<td>58</td>
</tr>
<tr>
<td>Albumin ++</td>
<td>17</td>
<td>34</td>
</tr>
<tr>
<td>Albumin +++</td>
<td>4</td>
<td>8</td>
</tr>
</tbody>
</table>

In the cases where ASO is more than 200IU the ratio of occurrence of the site of infection of skin, sore throat and no infection is 5:4:1. $X^2 = 1.1250$. $P = 0.5697 > 0.05$ and in the ASO < 200IU all the 3 groups are identical. $X^2 = 0.3333$. $P = 0.8464 > 0.05$.

**DISCUSSION**

Both proteinuria and hematuria were the most consistent urinary findings found in all the cases in the present study. In various studies APSGN with absent albuminuria range from 0-5%. The absence of proteinuria has been explained by Burman to be due to the absorption of proteins as they pass through the normal tubules. Microscopic hematuria was found in 100% of the cases in the present study, which is similar to many other studies. According to Puri R K et al, the degree of hematuria does not indicate the severity or prognosis of the disease. $^{(11)}$ In the present study there was no difference in clinical presentation and progress of the disease in those with gross hematuria and those with microscopic hematuria.

In the present study the haemoglobin was significantly low (< 10gm %) in 34% of the cases. According to Nissenson et al slight decrease in haemoglobin and hematocrit are usually present and this correlates with the degree of expansion of plasma volume. $^{(12)}$ According to nelson mild anemia occurring in APSGN is due to hemodilution and low grade hemolysis $^{(9)}$. In the present study erythrocyte sedimentation rate was significantly elevated in 46 cases (92%). Srivasthava $^{(13)}$ noted raised ESR in 97.5% cases while Manhas et al found it raised in only 20% of the cases. $^{(14)}$ Blood urea was significantly high in 20% of the cases and one patient had blood urea more than 100mg% in the present study. Serum creatinine values were elevated in 26% of the cases and one patient had serum creatinine more than 3mg%.

In the present study 46% of the patients had normal serum sodium levels and about 42% of the cases had only mild hyponatremia. Hyperkalemia occurs due to decreased excretion of potassium by renal tubules and it is accentuated by metabolic acidosis. Puri R K $^{(11)}$ mentions an incidence of 15% and Sajad $^{(15)}$ found it to be 10%. ASO titre was raised in 64% of the cases in the present study. Puri R K et al reported elevated ASO in 75% of the cases and Meharban Singh et al reported elevated ASO in 52% of the cases. $^{(16)}$

**CONCLUSION**

Acute glomerulonephritis is one of the commonest renal disorders in children. Although majority of cases manifest typically with edema, oliguria and hematuria, atypical manifestations are not uncommon. In the present study macroscopic hematuria was present in 92% of the cases. Absence of hematuria or proteinuria does not exclude the disease. ASO titre was elevated in 70.5% of the cases with history of respiratory infections and 71.4% cases with history of skin infections. Gross alterations of serum electrolytes do not occur in APSGN, though occasionally hyperkalemia is serious enough to cause death.
REFERENCES


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