ACUTE POSTSTREPTOCOCCAL GLOMERULONEPHRITIS. MODERN MEDICAL APPROACH ON AN EXAMPLE OF A CLINICAL CASE.

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INTRODUCTION

• Acute poststreptococcal glomerulonephritis (APSGN) strongly correlates with high mortality level and disability caused by organic failure.

• Of the estimated 470,000 new annual cases of PSGN worldwide, 97 percent occur in regions of the world with poor socioeconomic status, with an annual incidence that ranges from 9.5 to 28.5 per 100,000 individuals.

• Streptococcal infection (acute and chronic tonsillitis, pharyngitis) is a very common healthcare issue all over the world with morbidity of about 11 million people per year only in USA.

• The role of in time diagnostic and treatment of acute and chronic strep throat along with sanitation of other chronic infection sources in prophylaxis of streptococcus associated diseases (including APSGN) is significant.
DEFINITION

• Acute glomerulonephritis (AGN) - an immune–mediated inflammatory disease of the capillary loops in the renal glomeruli. The antigen–antibody complex deposition within the glomeruli results in glomerular injury. AGN is a representative disease of acute nephritic syndrome characterized by the sudden appearance of edema, hematuria, proteinuria, and hypertension.

• Acute poststreptococcal glomerulonephritis (APSGN) is the prototype of post-infectious glomerulonephritis and is associated with a previous skin or throat infection by group A streptococcus.

• Possible complications include acute kidney injury and chronisation with development of chronic kidney disease.
CLASSIFICATION, ETIOLOGY

Glomerulonephritis

Non-Proliferative

- Minimal Change Glomerulonephritis
  - Abnormal Podocytes
  - Seen on Electron Microscopy
  - Treat with Supportive care + Prednisolone
  - Most respond well

- Focal Segmental Glomerulosclerosis
  - Segments of Glomeruli Develop Sclerosis
  - Presents with Nephrotic Syndrome
  - Genetic causes identified
  - Steroids often ineffective
  - 50% Progress to Renal Failure

Proliferative

- Membranous Glomerulonephritis (MGN)
  - Thickened Glomerular Basement Membrane
  - Usually idiopathic
  - 1/3 have chronic MGN
  - 1/3 go into remission
  - 1/3 progress to renal failure

- IgA Nephropathy
  - Most common type of GN in adults
  - Microscopic haematuria
  - Appears 24-48hrs post URTI/GI infection
  - IgA deposits seen in the matrix

- Membranoproliferative Glomerulonephritis
  - Primary (immune mediated)
  - Secondary (SLE, Hep)
  - Usually progresses to End Stage Renal Failure

Rapidly Progressive Glomerulonephritis (Crescentic)

- Vasculitic Disorders
  - Wegener's Granulomatosis
    - Vasculitis
    - Lungs, Kidney & other organs
    - c-ANCA +ve
    - Treat with Steroids + Cyclophosphamide

- Microscopic Polyangiitis
  - Small vessel vasculitis
  - p-ANCA +ve
  - Treat with long term steroids +/− cytotoxic agents

- Goodpastures Syndrome
  - Autoimmune anti-GBM antibody
  - Glomerular & Lung affected
  - Haematuria & Haemoptysis
  - Treat with steroids +/− steroid sparing agents

Post Infectious Glomerulonephritis

- Occurs weeks after URTI
- Usually Strept Pyogenes
- Supportive treatment
- Resolves over 2-4 weeks
PATHOGENESIS

- Infection of streptococci
- Immune complexes, antigens
- Activation of Compliments Recruitment of leukocytes
- Hematuria, Proteinuria, RBC Casts
- GBM damage, Blood ingredients leakage
- Inflammation mediates, Cytokines, proliferative F.
- Edema hypertension heart failure encephalopathy renal failure
- Oliguria, sodium and water retention, hypervolemia
- Blockage of renal capillaries and decreased GFR
- Proliferation of MC and EC
COMPLICATIONS

- Acute/chronic renal failure
- Hyperkalemia
- Nephrotic syndrome
- Chronic glomerulonephritis
- Hypertension
- Congestive heart failure or pulmonary edema
PATIENT’S DETAILS

Patient Name: V. Y.K
Gender: Male
Age: 34 years old
Occupation: Taxi driver
Date of curation: 21.10.17
COMPLAINS

• Chief complaint of facial edema, mostly on the periorbital area mostly seen in the morning and bloody urine

• Flank pain in the lumbar region (lower back pain), this pain was localized and not radiating.

• Headache

• Weakness
HISTORY OF PRESENTING COMPLAINS

• One week prior to the presentation the patient had throat infection and he thought it was a usual cold, he used NSAIDs to relief the pain.

• On the 6th day of disease, the symptoms were not relieved, and he started noticing decreased urine output which was brownish in colour.

• He also started having pale skin and flank lower back pain, edema around his eyes and headache.

• He visited the general practitioner when he noticed that the symptoms were not subsiding.

• The patient was referred to Kharkiv Emergency Hospital #4, on 10.10.17 with tonsillitis and signs of glomerulonephritis.

• He got development of AKI on 12.10.17, was transferred to ICU and underwent hemodialysis.

• The patient was transferred to the therapeutic department on 15.10.17 with improvement.
FAMILY HISTORY
• The patient’s father had MI and the rest of the relatives have no illnesses

SOCIAL HISTORY
• Not married
• Works as a taxi driver

LIFESTYLE
• Does not drink alcohol and does not smoke

MEDICAL HISTORY
• The patient denies malaria, tuberculosis, diabetes mellitus, dermatovenerologic diseases, HIV-infection and viral hepatitis.
• No previous surgery/operations
• No allergies
OBJECTIVE EXAMINATION

- Condition is satisfactory, clear consciousness, active and emotionally stable.
- Normosthenic type of body constitution (BMI = 25.9 kg/m²)
- Body **temperature** is normal (36.7º C)
- Skin and visible mucous membranes are **pale** and clean.
- Periorbital edema is revealed
- Musculoskeletal system examination is unremarkable.
OBJECTIVE EXAMINATION

- **Respiratory system**: Percussion – resonant sound over the lung fields; auscultation – vesicular breathing. BR = 18/min.

- **Cardiovascular system**: Heart borders are in normal range, heart sounds are clear and rhythmic. BP = 145/95 mm Hg. Ps = HR = 109 bpm.

- **Gastrointestinal system**: Abdomen is symmetric, soft and painless in palpation. Liver is at the rib cage edge.

- **Urinary system**: Kidneys are not palpable. CVAT sign is slightly positive on both sides.
**CBC (18.10.17)**

<table>
<thead>
<tr>
<th>Blood test</th>
<th>Normal ranges for adults</th>
<th>Patient’s results</th>
</tr>
</thead>
<tbody>
<tr>
<td>ESR</td>
<td>0-22mm/h</td>
<td>34mm/h</td>
</tr>
<tr>
<td>RBC</td>
<td>3.9- 5.0*10^12/L</td>
<td>4.5*10^12/L</td>
</tr>
<tr>
<td>HEMOGLOBIN</td>
<td>135-175 grams/L</td>
<td>148 g/l</td>
</tr>
<tr>
<td>WBCs</td>
<td>3.5-10.5*10^9/L</td>
<td>10.5*10^9/L</td>
</tr>
<tr>
<td>LYMPHOCYTES</td>
<td>19.0-37.0%</td>
<td>20%</td>
</tr>
<tr>
<td>EOSINOPHILS</td>
<td>0.5-5.0%</td>
<td>2%</td>
</tr>
<tr>
<td>MONOCYTES</td>
<td>2.0-11.0%</td>
<td>2%</td>
</tr>
<tr>
<td>NEUTROPHILS</td>
<td>47-72%</td>
<td>76%</td>
</tr>
</tbody>
</table>

*SIGNS OF INFLAMMATION: INCREASED ESR, LEUCOCYTOSIS, NEUTROPHILIA*
### URINE ANALYSIS (18.10.17)

<table>
<thead>
<tr>
<th>URINE TEST</th>
<th>NORMAL RANGES FOR ADULTS</th>
<th>PATIENT'S RESULTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colour</td>
<td>Light yellow</td>
<td>brownish</td>
</tr>
<tr>
<td>Specific gravity</td>
<td>1.001-1.040</td>
<td>1.015</td>
</tr>
<tr>
<td>Colour Index</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>0-0.2g/l</td>
<td>1.1g/l</td>
</tr>
<tr>
<td>pH</td>
<td>5.0-7.0</td>
<td>6.0</td>
</tr>
<tr>
<td>Glucose</td>
<td>0-0.8mmol/l</td>
<td>-</td>
</tr>
<tr>
<td>RBCs</td>
<td>0-4 RBCs</td>
<td>10-12, changed</td>
</tr>
<tr>
<td>Casts</td>
<td>0-2 hyaline</td>
<td>4-6, hyaline</td>
</tr>
<tr>
<td>Crystals</td>
<td>absent</td>
<td>absent</td>
</tr>
</tbody>
</table>

- THE PROTEINURIA, RBCs IN URINE, CASTS AND BROWNISH COLOUR SUGGESTS NEPHRITIC SYNDROME
# BIOCHEMICAL BLOOD TEST (18.10.17)

<table>
<thead>
<tr>
<th>BIOCHEMICAL BLOOD TEST</th>
<th>NORMAL RANGES FOR ADULTS</th>
<th>PATIENT'S RESULTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALT</td>
<td>7 - 55 U/L</td>
<td>24.4 U/L</td>
</tr>
<tr>
<td>AST</td>
<td>8 - 48 U/L</td>
<td>37.7 U/L</td>
</tr>
<tr>
<td>UREA</td>
<td>2.5-7.1 mmol/l</td>
<td>5.47 mmol/l</td>
</tr>
<tr>
<td>CHOLESTEROL</td>
<td>&lt;200 mg/dL</td>
<td>62.4</td>
</tr>
<tr>
<td>GLUCOSE</td>
<td>4.4-7.8 mmol/l</td>
<td>4.9 mmol/l</td>
</tr>
<tr>
<td>CREATININNE</td>
<td>62-115 mcmol/L</td>
<td>130 mcmol/L</td>
</tr>
<tr>
<td>INDIRECT BILIRUBIN</td>
<td>Less than 19 mkmol/L</td>
<td>4.9 mkmol/L</td>
</tr>
<tr>
<td>DIRECT BILIRUBIN</td>
<td>0-7.9 mkmol/L</td>
<td>4.0 mkmol/L</td>
</tr>
<tr>
<td>TOTAL BILIRUBIN</td>
<td>17-21 mkmol/L</td>
<td>19.3 mkmol/L</td>
</tr>
</tbody>
</table>

❖ INCREASED CREATININE LEVELS REFERS TO RESIDUAL SIGNS OF PREVIOUS AKI
## OTHER LABORATORY INVESTIGATIONS

<table>
<thead>
<tr>
<th></th>
<th>NORMAL RANGES FOR ADULTS</th>
<th>PATIENT’S RESULTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rheumatoid factor</td>
<td>Less than 8 mg/L</td>
<td>6.0 mg/L</td>
</tr>
<tr>
<td>C-reactive protein</td>
<td>Less than 6 IU/mL</td>
<td>6.5 IU/mL</td>
</tr>
<tr>
<td>Anti-streptolysin O (ASO)</td>
<td>Less than 200 IU/mL</td>
<td>220 IU/mL</td>
</tr>
</tbody>
</table>

- **Throat culture**: Positive for streptococcus bacteria
- **GFR**: 68.3 ml/min

- **Increased titres of ASO (Anti-Streptolysin O) suggest streptococcal infection**
- **Culture from throat is positive for streptococcal infection**
INSTRUMENTAL EXAMINATION

• **ECG**: Sinus rhythm, **tachycardia**. No signs of acute pathology revealed.
• **Echo-CG**: no significant changes.
• **Abdominal USI**: Thickening of renal parenchyma, both kidneys are enlarged.
DIAGNOSIS

❖ Acute post-streptococcal glomerulonephritis
❖ Acute kidney injury (12.10.17), recovery phase
❖ Symptomatic arterial hypertension
❖ Acute lacunar tonsillitis (reconvalescent)
MANAGEMENT OF THE PATIENT

• **Penicillin** 250mg, 4 times a day for 10 days
• **Furosemide** 40 mg, 2 times per day for 7 days
• **Amlodipine** 2.5mg, once a day for 4 weeks
• **Prednisone** 60 mg, per day for 4 weeks
• **Heparin** subcutaneously 1000 u, 3 times per day for 4 weeks
MANAGEMENT OF THE PATIENT

DIET AND ACTIVITY

• Hospitalization

• **Sodium and fluid restriction** should be advised, a maximum of 10g of sodium per day and 1 litre of fluids per day: for treatment of signs and symptoms of fluid retention (eg, edema, pulmonary edema).

• **Diet number 7** which consists of eating cereals (wheat, buckwheat, millet, wholegrain rice, oats and barley) should be followed.

• **Bed rest** is recommended until signs of glomerular inflammation and circulatory congestion subside. Prolonged inactivity is of no benefit in the patient recovery process.
PROGNOSIS

• For recovery – favorable
• For life – favorable
CONCLUSION

- APSGN makes about 10% in total nephrological morbidity statistics and strongly correlates with increased mortality and quality of life worsening.
- The correct and timely treatment leads to significant decrease in the complications frequency and improves patient’s prognosis.
- Such prophylaxis measures as sanitation of chronic sources of inflammation and in time and appropriate treatment of strep throat noticeably decrease the risk of APSGN development.
THANK YOU