



COINCIDENTAL OCCURRENCE OF ACUTE POST-STREPTOCOCCAL GLOMERULOEPHRITIS AND ACUTE RHEUMATIC FEVER IN THE SAME CHILD: A CASE REPORT

Pediatrics

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ABSTRACT

Acute Glomerulonephritis and Rheumatic fever are the two serious sequelae in children followed by Group A Streptococcus infection.(GAS) Though these two are the well known non suppurative complications of GAS infection, their occurrence in the same patient is rare. We report a case of a 14-year-old girl, who presented with complaints of generalized swelling of face and feet, hematuria, breathlessness. She was febrile at admission. Examination and laboratory data revealed a coincidental occurrence of two severe complications of GAS infection in the girl. Treatment given with diuretics and penicillin prophylaxis was started. Child condition improved and later discharged in healthy status.

KEYWORDS

Group A Streptococcus, Acute post-streptococcal glomerulonephritis, Acute rheumatic fever.

INTRODUCTION:

Group A streptococcal infection is considered notorious for its association with two serious non-suppurative sequelae: Acute Rheumatic Fever (ARF) and Post-Streptococcal Glomerulonephritis (PSGN). The organ systems involved in both ARF and PSGN are not directly invaded by microorganisms itself but are immunologically mediated disease process. Though their occurrence in the same patient concurrently is exceptional and poorly documented in the literature, coincidental occurrence of these two diseases has also been described earlier.¹

CASE REPORT:

Patient history

A 14-year-old girl presented to the emergency department with complaints of fever, cough and breathlessness for the past four days. There was a history of progressive increase in swelling of the face followed by swelling of both feet from 15 days prior to these complaints. The child noticed passing red colour urine for the last five days. History of fever with upper respiratory tract infection and headache three weeks ago for which medication was used. At admission, her vitals recorded were Pulse rate- 153/min, BP- 160/120mmHg, Respiratory rate of 35/min, with a saturation of 85% in room air.

Clinical findings, diagnostic assessment, and the course of treatment at the hospital

On examination at admission weight recorded was 57kg and height of the child was 167cm. On General examination, the child was conscious and oriented, pale and anasarca present. Systemic cardiac examination revealed apex beat at left 5th intercostal space at the midclavicular line. On auscultation S1S2 soft, S3 gallop present. On Respiratory systemic examination, bilateral basal crepitations were present. Flame shaped haemorrhages were present in both eyes on fundus examination. Other systemic examination was normal. Supportive treatment was given and started further evaluation. Laboratory investigations showed: haemoglobin 9.5 g/dl, WBC 23.870/mm3 (Neutrophils- 88%). C reactive Protein – 48mg/dl. Haematuria was confirmed by the urinalysis – 418 RBCs/hpf, with dysmorphic RBCs present, while urine culture was negative. Blood urea – 31mg/dl and Creatinine value was 1.2mg/dl. Anti – Streptolysin O titre (ASO) was positive with 800IU/L. Complement value C3 – 40mg/dl. Chest X-ray showed pulmonary congestion with cardiomegaly. Other investigations were otherwise normal. With the history of antecedent upper respiratory tract infection and features of edema, hematuria, hypertension post-streptococcal complications were suspected. High ASO titre and low complement values diagnosis of post-streptococcal glomerulonephritis was confirmed. Features of congestive cardiac failure secondary to hypertension were also in favor of acute glomerulonephritis. The child was started on amoxicillin/clavulanate

intravenously (at the dose of 100 mg/kg/day) and Furosemide. Echocardiography done showed global hypokinesia with Left ventricular dysfunction with mitral regurgitation and tricuspid regurgitation. Throat swab culture showed growth of Streptococcus pyogenes. As the patient fulfilled the revised Jones criteria for Acute Rheumatic Fever-ARF (carditis, fever, and raised acute phase reactants) one major and two minor criterion fever, in the evidence of a previous streptococcal infection, diagnosis of ARF was established.² Treatment was initiated Prednisolone and with Aspirin at the dosage of 75 mg/kg/day was and secondary prophylaxis with intramuscular benzathine benzylpenicillin (at the dose of 12,00,000 IU) started. Fever subsided, hypertension under control with B.P – 110/70mm Hg and child's general condition improved with resolution of hematuria (RBC- /hpf) and edema decreased as evidenced by the reduction in weight (53 kg after one week). Features of Congestive cardiac failure resolved. The serum antistreptolysin O titre was negative after treatment. At discharge, the girl was asymptomatic: neither haematuria nor cardiac murmurs could be detected. During follow up echocardiographic findings revealed a nonsignificant mitral regurgitation. Aspirin was continued for an overall period of 6 weeks. On later follow up visits child was asymptomatic, and continued to receive secondary benzathine penicillin prophylaxis.

DISCUSSION:

As discussed earlier, PSGN and ARF are both postinfectious non-suppurative sequelae caused by Group A beta hemolytic Streptococcus infections. The presentation of clinical and laboratory features of these two diseases preceded by a documented streptococcal pharyngitis is still an opportunity of scholastic significance for clinicians and students.³ PSGN and ARF have different epidemiology, immunology, and also different bacteriology features. Their concurrent development in the same patient is known and has been described in the literature, but these are rare presentations.⁴ The serotypes of group A Streptococci are divided into those with rheumatogenic and nephritogenic potential on the basis of the complication sequelae induced by them.

The pathogenesis of ARF is well known. ARF is known to be caused by the mechanism of molecular mimicry. Some streptococcal antigens, like M protein and hyaluronate capsule, have identical antigen epitopes similar to human tissue proteins in the myocardium, brain, and joints. This way, they cause inflammation by inducing cross-reacting autoantibodies against human tissue proteins stimulated by cell-mediated immunity in genetically predisposed individuals.⁵ The immunopathogenesis of PSGN is not yet completely understood. The most extensively studied mechanisms suggest circulating immune complex deposition, in situ immune complex formation, and molecular mimicry between streptococcal and human glomerular proteins, with consequent autoimmune response results in PSGN.⁶

Though both ARF and PSGN are secondary to immunological injury, their coincidental occurrence is not usually seen, and there is no explanation yet for this interesting occurrence. It might be explained that some of the streptococcal strains had both nephritogenic and rheumatogenic features.⁷

In 2003 study and literature review by Kula et al., stated that most of the previously reported cases of this condition were adults. They studied the characteristics of the pattern of occurrence in the previously reported cases. Of the nine cases, they studied five were male, and four were female, aged 3.5-16 years (mean 11.27 ± 1.42 years). All patients were treated with diuretics and salicylate and given secondary prophylaxis with intramuscular benzathine penicillin. In 4 of the reported cases of co-existent APGN and ARF acute rheumatic fever was the first feature which was followed by glomerulonephritis. Acute glomerulonephritis was the first feature in the remaining of the cases. However, some of these cases had a longer intermittent period between both features and even symptoms for both APSGN and acute rheumatic fever can present at the same time.¹

In a study done by Vardi et al., it was found that mitral insufficiency is frequently associated with patients in PSGN.⁸ Much remains to be learned about the complex interrelationships between streptococcus and host that result in the occurrence of rheumatic fever, glomerulonephritis, or, on occasion, both. Progress will undoubtedly be slow in those with this coincidental occurrence.⁴ Consequently, physicians should be careful about this unusual condition so as to initiate adequate prophylaxis in these patients. Early recognition of both the disease process is important to prevent future morbidity.

REFERENCES:

1. Kula et al. Acute Glomerulonephritis and Acute Rheumatic Fever in the same patient: a case report and review of literature.-Anadolu Kardiyol Derg 2003;3: 272-274.
2. Gewitz MH, Baltimore RS, Tani LY, Sable CA, Shulman ST, Carapetis J, et al. Revision of the Jones criteria for the diagnosis of acute rheumatic fever in the era of Doppler echocardiography: A scientific statement from the American Heart Association. *Circulation*. 2015;131:1806-1818. [PubMed].
3. Fioretti M, Napodano S, Patti M, Rigante D. Poststreptococcal glomerulonephritis and rheumatic fever: Two faces of the same coin. *Eur Rev Med Pharmacol Sci*. 2013;17:1139-1140. [PubMed].
4. Bisno et al. The coexistence of acute rheumatic fever and acute glomerulonephritis. *Arthritis Rheum* 1989; 32: 230-232.
5. Martin WJ, Steer AC, Smeesters PR, Keeble J, Inouye M, Carapetis J, et al. Post-infectious group A streptococcal autoimmune syndromes and the heart. *Autoimmun Rev*. 2015;14:710-725. Medline:25891492 doi:10.1016/j.autrev.2015.04.005.
6. Rodriguez-Iturbe B, Batsford S. Pathogenesis of poststreptococcal glomerulonephritis a century after Clemens von Pirquet. *Kidney int*. 2007;71:1094-1104. Medline:17342179 doi:10.1038/sj.ki.5002169.
7. Potter E, Svartman M, Mohammed I. Tropical acute rheumatic fever and associated streptococcal infection compare with concurrent acute glomerulonephritis. *J Pediatr* 1978;92:325-333.
8. Vardi P, Markiewicz W, Levy J, Adler O, Riss E, Benderley A. The heart in acute glomerulonephritis: an echo-cardiographic study. *Pediatrics* 1979, 63:782-787.