CASE REPORT

Eastern Journal of Medicine

East J Med 28(3): 541-543, 2023
DOI: 10.5505/ejm.2023.04900

Concurrent Acute Rheumatic Fever and Acute Poststreptococcal Glomerulonephritis: A Case Report

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ABSTRACT

Acute poststreptococcal glomerulonephritis and acute rheumatic fever are both postinfectious non-purulent sequelae of A group β-hemolytic *Streptococcus* infections. In spite of prominent falls in the incidence, Acute Rheumatic Fever remains (ARF) an important cause of morbidity and mortality associated with acquired heart disease in underdeveloped territories. Acute poststreptococcal glomerulonephritis (APSGN) still remains a frequent form of glomerulonephritis in third-world countries, particularly in areas where the disease occurs in epidemics. Both diseases may be seen simultaneously in the same patient, but both diseases have different epidemiological, immunological and bacteriological features, however, this is a rare condition. We present a case of a 6.5 year old child with concurrent APSGN and ARF.

Key words: Child, glomerulonephritis, rheumatic fever.

Introduction

Both acute poststreptococcus glomerulonephritis (APSGN) and acute rheumatic fever (ARF) are non-suppurative sequelae of group A β-hemolytic streptococcal (GABHS) infections. In spite of dramatic falls in the incidence, ARF persists a major cause of mortality and morbidity associated with acquired heart disease in underdeveloped countries (1). The annual incidence of APSGN in the worldwide is about 472000, and approximately 404000 of these cases occur at childhood (2). Although ARF and APSGN have different epidemiology, immunology and bacteriology features; the occurrence of these two diseases in the same patient is rarely described (3,4). We presented a 6.5 year old male with ARF who presented with typical findings of APSGN.

Case Report

A 6.5 year old boy applied with the complaint of macroscopic hematuria, fever, headache, arthritis and epistaxis. His urine was dark-brown during last four days. He had fever and migratory arthritis firstly at left knee and later at left ankle during last four days. He had headache during last two days and had epistaxis two times before admission to hospital. In his history he had tonsillitis three weeks ago. On examination obtained a fever of 37.6°C, respiratory rate 25 / min, heart rate of 132 / min, and blood pressure of 140/100 mmHg. His left knee and left ankle was hot and tenderness with range of motion. He had palpebral edema but no pretibial edema was present. In the cardiovascular system examination, a 3/6 degree pansystolic murmur was heard at the apex. On laboratory evaluation; blood urea nitrogen 29 mg / dL, creatinine 0.56 mg / dL, total protein 6 g / dL, albumin 3.5 g / dL, hemoglobin 10.7 g / dL, WBC 12500 / mm^3, thrombocyte 241000/mm^3, CRP (C reactive protein) 2 mg / dl, ESR (erythrocyte sedimentation rate) 57 mm / h, serum anti-streptolysin O titer was 770 IU/mL. Urinalysis revealed 2+ proteinuria and hematuria (>34 erythrocytes / field with dysmorphic erythrocytes) while urine culture was negative. Spot urine protein/creatinine ratio was 1.36. Serum complement analysis viewed revealed decreased C_3 (13.2 mg / dl) with the other blood tests as serum C_4, antinuclear antibodies and anti-DNA within normal range. With clinical and laboratory evaluations he was diagnosed as APSGN and as he had cardiac murmur and migratory arthritis he was referred to echocardiographic evaluation with the suspicion of ARF. 2-D and Doppler echocardiography revealed

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Received: 03.05.2020, **Accepted:** 25.07.2021

severe mitral regurgitation and minimal aortic insufficiency. The electrocardiography and the chest X-ray film was normal. Diagnosis of ARF, with the merger of 2 major Jones criterium (cardit, arthritis) and two minor criteria (fever plus C-reactive protein and erythrocyte sedimentation rate elevation), in the proof of a prior streptococcal infection was made (5).

He was given furosemide and secondary prophylaxis with intramuscular benzanthine penicillin. After hospitalisation of patient blood pressure and urine output were closely monitored. With furosemide treatment blood pressure was within normal limits and prednisolone at the dose of 2 mg/kg/d was started. Child's general condition improved fastly. Prednisolone was given for five weeks. Acetyl-salicylic acid therapy at 65 mg/kg/day was started in last week of treatment of prednisolone and continued for four weeks. When the patient was discharged, he was asymptomatic, had no hematuria or cardiac murmur, only mild mitral regurgitation on echocardiographic examination. There was no hematuria and proteinuria in the last urine test performed at the end of the first month. The patient is still followed by our pediatric cardiology and pediatric nephrology clinic. Consent was obtained from the patient's family for the case presentation.

Discussion

Both ARF and APSGN are non-infectious, nonuppurative sequelae of GABHS infections: the characteristic of clinical and laboratory properties of the two diseases preceded by a attested GABHS pharyngitis is still an opportunity of great importance for clinicians (6,7). In this report, the patient admitted to hospital with macroscobic hematuria and after laboratory and clinical evaluation he had nephritic syndrome as a presentation of APSGN. He had migratory arthritis, fever, high acute phase reactants (high CRP and ESR) and mitral valve regurgitation at echocardiography. He had diagnosed as ARF according to 2 major Jones criterium (arthrit, cardit) and 2 minor criteria (C-reactive protein and erythrocyte sedimentation rate elevation plus fever).

ARF and APSGN have distinct immunology, epidemiology, and bacteriological attributes, and their simultaneous occur in the same patient is known, but this is a uncommon condition (4). Group A streptococcal serotypes may be divided into nephritogenic and rheumatogenic (8). In particular, the prevalence of APSGN has recently declined, but although the outbreaks and clusters of APSGN continues to emerge, it is difficult to determine the current global burden of the disease (9). Two major antigens are blamed as the potential cause of APSGN. These are nephritis related plasmin receptor and a cationic cysteine proteinaceous, both of which activate the path to the alternate complement system (10).

The nowadays incidence of ARF after an GABHS infection is less than 1% in developed territories, and the disease develops as a result of a type II hypersensitivity, resulting from molecular similarity between mesenchymal structures and streptococcal M-protein antigen, in case of T and B cell stimulation together (11).

The role of genetic factors leading to ARF and APSGN in response to streptococcal infections has not yet been identified, but ARF longtime trouble is interrelated to the significant risk of lasting cardiac valvular damage, which may occur after recurrent GABHS infections in children with a prior unobserved rheumatic carditis (7).

In spite of the different characteristics of the two diseases, a genre of streptococcus can produce both ARF and APSGN in the same child at the same time as in our patient. This togetherness should be kept in mind by physicians for the purpose of recommend that bacteriological analysis be performed more systematically to look for GABHS infections, immediate a primary prophylaxis or a secondary prophylaxis of RF and restrain the risk of permanent heart disease.

There are previously reported cases with concomitant ARF and APSGN in literature. Some cases in literature initial feature was ARF which was followed by glomerulonephritis (3,12-14). There is also some cases presenting with features of APSGN and ARF at the same time (15). In our case the patient has the features of both diseases at the same time also.

Consequently, in patients who have fever, joint pain, headache, nasal bleeding, edema, hematuria complaint must be careful in terms of both APSGN and ARF. Despite the different features of the two diseases, there is a risk of concurrence of APSGN and ARF in the same patient. In order not to miss these entities, doctors should take this into consideration when evaluating the patient, and patients should be detected carefully for this aspect to protect the patient from the risk of permanent heart disease.

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