Acute poststreptococcal glomerulonephritis and acute rheumatic fever: An uncommon coincidence

Sir,

We report a coincidence of two well-known complications of Group A β-hemolytic *Streptococcus* (GAS) infection: Acute rheumatic fever (ARF) and acute poststreptococcal glomerulonephritis (APGN).

A 5-year-old female child was admitted to the hospital because of fever, unproductive cough, dyspnea, and polyarthralgia. History revealed two febrile episodes of upper respiratory tract infections (URTIs) more than 2 weeks ago (the last one treated with oral co-amoxiclav). Tachycardia, tachypnea, dyspnea, edema of the legs, enlarged tonsils, and Grade 2/6 systolic murmur were found on admission. Signs of cardiomegaly and pulmonary congestion were noted in the chest X-ray [Figure 1]. Echocardiography (ECG) revealed sinus tachycardia with deep negative T-waves [Figure 2]. ECG showed dilation of the left ventricle (LV) (LV end-diastolic diameter 47 mm) and left atrium (40 mm × 36 mm), with mildly decreased LV systolic function (ejection fraction 51.8%) and normal diastolic function. Irregular thickening of mitral and aortic valve leaflets, severe mitral regurgitation, and mild aortic regurgitation were found together with mild pericardial effusion (4 mm). Laboratory tests showed normocytic anemia (hemoglobin 9.4 g/dL) and hematuria (200/μl), with traces of proteinuria. Increased levels of antistreptolysin O (ASO) (3843.52 IU/ml), brain natriuretic peptide (BNP) (1249.8 ng/L), and decreased complement C3 (0.79 g/L) were found. Serum creatinine, electrolytes, C-reactive protein, and troponin I were all normal, and throat culture was negative.

High ASO titer with a recent history of febrile URTI suggested that the patient might be suffering from poststreptococcal complications. As the patient fulfilled the revised Jones criteria for ARF (carditis, fever, and polyarthralgia) and had typical signs of APGN (edema, hematuria, high ASO titer, and decreased C3 levels), diagnosis of both complications was made.[1] Treatment with prednisone, furosemide, and captopril for 3 weeks was initiated along with penicillin. At 2 months follow-up, the patient was asymptomatic, with normal ECG and complement C3, BNP levels. ECG did not reveal significant changes, except for minimal mitral regurgitation. The ASO titer had decreased to 427.73 IU/ml with persistence of hematuria (200/μl). At 6 months follow-up, the child remained asymptomatic with resolution of hematuria.

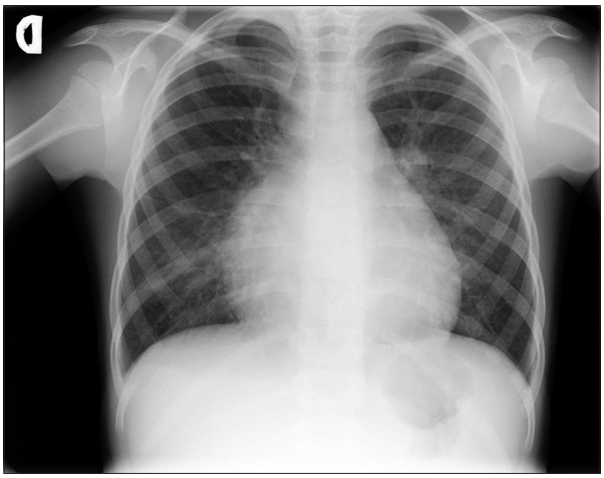

Figure 1: Anteroposterior chest X-ray showing signs of pulmonary congestion and enlarged heart

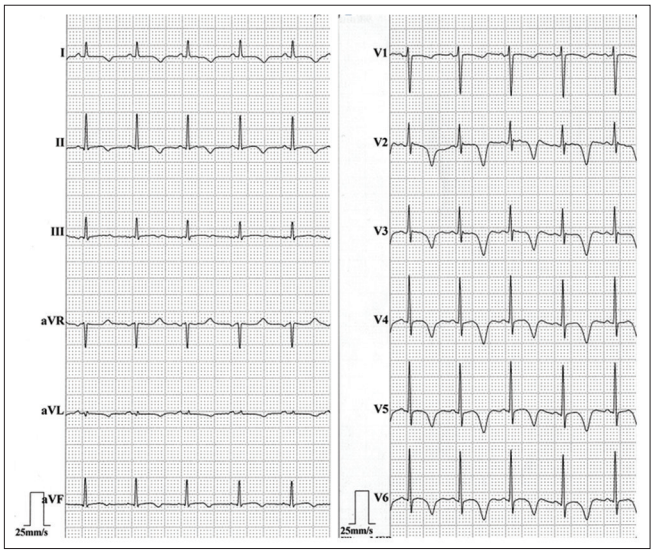

Figure 2: Echocardiography showing sinus tachycardia of 105 beats per minute with deep negative T-waves in I, aVL, V1-V6, and prolonged QTc interval

Both diseases seen in our patient share the same site of injury – specific endothelial cells in target organ. However, the concomitant presence of both is very rare because of different causal pathophysiologic mechanisms. The complications are believed to be caused by different GAS strains explaining the differences in recurrence patterns.[2,3] Unfortunately, antibiotic therapy prescribed earlier precluded us from checking whether previously mentioned episodes of URTI could be caused by different GAS strains.

Early recognition of ARF is of high importance because of the possibility of developing rheumatic heart disease in case of no treatment. Although both complications were rather promptly diagnosed in our hospital by the multidisciplinary team of pediatric cardiologists and nephrologists, the concurrence of both complications might become a diagnostic dilemma because APGN itself is associated with acute echocardiographic changes as shown previously by our group.[4]

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Conflicts of interest

There are no conflicts of interest.

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