

Letter to the Editor

Poststreptococcal glomerulonephritis and rheumatic fever: two faces of the same coin

Dear Editor,

We report the case of a 4.5-year-old boy who came to our observation because of fever, productive cough, and haematuria. He had presented a sore throat without fever 2 weeks prior to hospital admission, which subsided spontaneously within 3 days. No antibiotics were administered. On admission, physical examination revealed a body temperature of 40°C, pulse rate of 110/min, and blood pressure of 100/60 mmHg. Laboratory investigations showed: haemoglobin 10.5 g/dl, WBC 24.470/mm³ (N 78%), erythrocytation rate (ESR) 99 mm/h, C-reactive protein (CRP) 231 mg/l, with the other blood tests within normal range. Haematuria (> 50 RBCs/hpf, 40% dysmorphic RBCs, and 5.3% acanthocytes) was confirmed by the urinalysis, while urine culture was negative. Left basal and right paracardiac opacities were found at the chest X-ray film: amoxicillin/clavulanate was started intravenously (at the dose of 100 mg/kg/day). In addition, the patient had a positive throat culture for group A *Streptococcus*, while serum complement assay revealed decreased C3 (17 mg/dl, n.v. 90-180) and normal C4. The serum antistreptolysin O titre was 878 IU/ml. The combination of haematuria and hypocomplementaemia, in a scenery revealing a streptococcal infection, supported the diagnosis of poststreptococcal glomerulonephritis (PSGN). Fever subsided on day 4 and child's general condition improved fastly, with complete pneumonia resolution, while the serum antistreptolysin O titre increased to 1300 IU/ml 6 days after the first test. On day 7 the cardiac examination showed a grade 2/6 systolic murmur at the heart apex, and echocardiography revealed both mitral and aortic valve regurgitations. Diagnosis of rheumatic fever (RF), upon the combination of one major Jones criterium (carditis) and two minor criteria (fever plus ESR and PCR elevation), in the evidence of a previous streptococcal infection, was established. Aspirin at the dosage of 85 mg/kg/day was prescribed, and secondary prophylaxis with intramuscular benzathine benzyl penicillin (at the dose of 600,000 IU) started. Aspirin was continued for an overall period of 6 weeks. At discharge the boy was asymptomatic: neither haematuria, nor cardiac murmurs could be detected, and echocardiographic follow up revealed a nonsignificant mitral regurgitation. The last urinalysis on day 30 showed only minimal hematuria (3-5 RBCs/hpf).

PSGN and RF are both postinfectious non suppurative sequelae of group A beta hemolytic *Streptococcus* infections: the presentation of clinical and laboratory features of the two diseases preceded by a documented streptococcal pharyngitis is still an opportunity of scholastic significance for clinicians and students. PSGN and RF have different epidemiology, immunology, and bacteriology features, and their concurrent development in the same patient is known, but rare¹. The serotypes of group A *Streptococci* can be divided into those with rheumatogenic and nephritogenic potential². In particular, the incidence of PSGN has decreased in the past three decades, but the present-day global burden of the disease is difficult to establish, being disappeared in Central Europe, though epidemics and clusters of cases of PSGN continue to appear³. Two antigens are actively investigated as the potential cause(s) of PSGN: the nephritis-associated plasmin receptor, identified as glyceraldehyde-3-phosphate dehydrogenase, and a cationic cysteine proteinase, known as streptococcal pyrogenic exotoxin B, both activating the alternate pathway of the complement system⁴. The current incidence of RF after a streptococcal infection is now thought to have decreased to less than 1% in the industrialized countries, and the disease derives from a type II hypersensitivity reaction, emerging from molecular mimicry between the Streptococcal M protein antigen and mesenchymal structures, in the presence of B and T cell co-stimulation⁵. The role of genetic factors in the response to streptococcal infections leading to PSGN and RF is not yet defined, but RF major long-term concern is related to the substantial risk of permanent heart valvular injury, which might take place after repeated group A *Streptococcal* infections in children with a previous overlooked rheumatic carditis.

Despite the distinct properties of the two diseases, a *Streptococcus* strain might have the chance of giving simultaneously both PSGN and RF in a same child. This co-occurrence should be kept in mind by general practioners in order to recommend that bacteriological analysis be performed more systematical-

ly to search for group A *Streptococcus* infections, prompt a primary prophylaxis (to treat an acute infection and prevent a first attack of RF) or a secondary prophylaxis of RF (with regular benzathine penicillin G every 21 days to prevent relapses) and call off the risk of permanent heart disease.

Conflict of Interest

The Authors have no conflict of interest to disclose.

Financial Disclosure

The Authors have no financial relationships relevant to this article to disclose.

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