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Case Report

Serum Sickness and Uveitis

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A B S T R A C T

Serum sickness is immune response to a foreign antigen, usually a heterologous protein. Incidence rate is less than 0.5%. Antigens and responding antibodies form circulating immunocomplex that is characteristic for serum sickness. The condition occurs 7–15 days after exposure to the antigen, usually with clinical picture of glomerulonephritis. The immunocomplex circulates to other tissues where it sediments and causes inflammation, such as arteritis, neuritis, synovitis. The aim of this research is to present the break out of serum sickness in form of anterior uveitis due to azithromycin therapy administered by mouth. Identifying of anterior uveitis may help in early diagnostics and treatment of serum sickness.

Key words: *serum sickness, uveitis*

Introduction

Serum sickness is a clinical syndrome that occurs after the injection of a foreign or heterologous serum (such as heterologous antitoxin for rabies, diphteria, Clostridia) or after taking medications like penicillin, sulphonamide etc. Immunocomplex composed of antigens and antibodies is responsible for tissue damage and creation of inflammatory response in patients with serum sickness. Antigen concentration influences the amount of formed immunocomplex as well as the clinical picture and the course of disease.. If there is certain antigen surplus, the amount of immunocomplex is small and remains permanently present in the circulation. If there is antibody surplus, immunocomplex is eliminated mononuclearly by phagocytes[1–3]. In the elimination process the immunocomplex sediments in various tissues, most frequently in kidneys, causing inflammation such as glomerulonephritis, arteritis, synovitis, uveitis. Symptoms commonly occur 7–15 days following the exposure to the antigen. The most frequent symptoms are fever, myalgia, urticaria, lymphadenopathy, splenomegaly. Laboratory results identify leucocytosis and increased erythrocyte sedimentation rate, sometimes also eosinophilia, proteinuria, haematuria and hypergamaglobulinemia. Most patients with serum sickness get well spontaneously a few days or weeks after the elimination of the antigen that caused the reaction.

Patients and methods

A ten-year-old boy who was suffering from a respiratory infection and therefore receiving azithromycin therapy was referred to ophthalmologist for conjunctivitis by his pediatrician. At the examination hyperplasia of the papilla and conjunctival – cilliary injection were found. Conjunctival swab was taken to be tested for bacteria and for Chlamydia (the result was negative). Tobramycin (drops and ointment) was ordered locally. Ten days following the beginning of azithromycin therapy the boy's condition deteriorated and he lost sight to both eyes. *Patient's condition at admission:*visual acuity, both eyes: waving a hand in front of the eye; slit-lamp biomicroscopy, both eyes: retinal limbus injection, profuse adipose precipitates in the endothelium. Underlying layers could not be analysed. The boy was admitted to Department of Pediatrics where extensive laboratory tests were done (CBC, coagulogram, proteinogram, urine, stool-findings were normal; ASTA, ANCA, ENA, Waaler-Rose, Latex-rheumatoid factor, ANF – were negative) in order to diagnose systemic connective tissue disease which was not confirmed. Hypergamaglobulinemia, as well as increased sedimentation rate and mild leucocytosis. Corticosteroids mydriatic and cycloplegic (drops and ointments) were ordered locally.

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Results

Laboratory tests did not identify systemic connective tissue disease and conjuctival swab analysis for bacteria and Chlamydia did not discover any infection. Hypergamaglobulinemia with increased erythrocyte sdimentation rate and leucocytosis led to conclusion that the boy was suffering from serum sickness. Local corticosteroid therapy applied during two weeks resulted in complete uveitis regression (visual acuity, both eyes: 1.0; slit-lamp biomicroscopy: no signs of uvea inflammation). The antigen that trigggered the immune response and caused forming of circulating immunocomplex which resulted in serum sickness in this case was azithromycin.

Discussion

Serum sickness occurs rarely, the incidence rate is less than 0.5% and therefore it is not frequently described in literature. The outbreak of the serum sickness is often dramatic, but early identification helps choosing the suitable treatment. Patients often get well spontaneously within a few days or weeks after the elimination of the antigen and spare themselves unnecessary tests. Serum sickness often breaks out as glomerulonephritis and signs of inflammation occur in other tissues where immunocomplex sediments, so that also neuritis, vasculitis, synovitis and/or uveitis may occur.

The connection between anterior uveitis and serum sickness is described in literature in just a few cases[5,6], probably because uveitis is frequently asymptomatic or characterized by mild symptoms[7] and therefore often remains unrecognized. Serum sickness induced by streptokinase is well known and most commonly described in literature[3]. Seibert et al.have found out that about 6% of all patients treated by streptokinase develope serum sickness[4] but it often remains unrecognized. It is also known that serum sickness can be induced by sulphonamide and penicillin. The presentation of this case shows that azithromycin can also be the antigen that causes forming of circulating immunocomplex responsible for development of serum sickness. Uvea inflammation developed ten days after azithromycin therapy by mouth was applied and that is the period necessary for forming the circulating immunocomplex as response to a foreign antigen. Eleven days after the azithromycin intake into the organism its concentration decreases below the treatment level and after twenty one day it is completely excreted from the organism which coincides with the regression of the choroid inflammation.

Identification of the anterior uveitis may help in early diagnostics and treatment of serum sickness. Furthermore, anterior uveitis combined with serum sickness may be treated only by local application of corticosteroids. Spontaneous recovery follows after the elimination of the antigen that caused the reaction.

REFERENCES

1. EMANCIPATOR, S. N., G. R. GALLO, M. E. LAMM, Clin. Nephrol., 24 (1985) 161. — 2. SCHIFFERLI, J. A., R. P. TAYLOR, Kidney Int., 35 (1989) 993. — 3. CHAPEL, H. M., M. HAENY: Essentials of clinical immunology. (Blackwell Scientific Publications, Oxford, 1984). — 4. DAVIDSON, J. R., R. K. BUSH, E. W. GROGAN, L. A. BOH, F. M. GRAZIANO, 6 (1988) 381. — 5. WONG, V. G., R. R. ANDERSON, P. R. MCMASTER, Arch. Ophthalmol., 85 (1971) 93. — 6. THEODORE, F. H., Arch. Ophthalmol., 21 (1939) 828. — 7. PROCTOR, B. D., P. G. MURRAY, B. C. JOONDEPH, N. Engl. J. Med., 330 (1994) 576.

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SERUMSKA BOLEST I UVEITIS

SAŽETAK

Serumska bolest je imunološka reakcija na strani antigen, obično heterologni protein. Incidencija je manja od 0,5%. Bolest je karakterizirana stvaranjem cirkulirajućih imunokompleksa sastavljenih od antigena i protutjela. Manifestira se 7–15 dana nakon izlaganja antigenu; najčešće kliničkom slikom glomerulonefritisa. Imunokompleksi se odlažu i u drugim tkivima, stvarajući sliku upale kao npr. arteritis, neuritis, sinovitis. Želimo prikazati pojavu serumske bolesti u obliku prednjeg uveitisa koja se razvila nakon peroralne terapije azitromicinom. Prepoznavanje prednjeg uveitisa može pomoći u ranoj dijagnozi i liječenju serumske bolesti.