Case Report / *Olgu Sunumu*

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A Serious and Rare Complication Following Varicella Infection; Streptococcal Toxic Shock Syndrome-like Case Report

Su Çiçeği Enfeksiyonu Sonrası Gelişen Ciddi ve Nadir Bir Komplikasyon; Streptokokal Toksik Şok Sendromu Benzeri Olgu Sunumu

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Abstract

Varicella is a primary disease of varicella zoster virus (VZV), which is among the human herpes viruses. Chickenpox is a disease that can be easily transmitted by airway, droplet and direct contact to the infected person. Skin lesions after varicella infection are the most common complication of staphylococcal or streptococcal secondary infections. Occasionally, necrotizing fasciitis and severe skin defects develop afterwards. Pneumonia, carditis, hepatitis, glomerulonephritis, encephalitis are among the criminal commissions. Streptococcal toxic shock syndrome (STSR) is a severe clinical picture of one of the group of streptococcal invasive infections, with sudden and rapid onset of shock and organ failure, with a mortality rate of 30-50%. In this study, we present a case of STSR which is a rare and life-threatening clinical entity that develops rarely after varicella infection.

Keywords: Chickenpox, streptococcal toxic shock syndrome, varicella zoster virus

Özet

Suçiçeği, insan herpes virüsleri arasında yer alan varisella zoster virüsünün (VZV) birincil bir hastalığıdır. Suçiçeği enfekte olan kişiden hava yolu, damlacık ve direkt temas ile kolaylıkla bulaşabilir bir hastalıktır. Suçiçeği enfeksiyonu sonrası deri lezyonlarının stafilokok ya da streptokoklarla ikincil enfeksiyonu hastalığın en sık görülen komplikasyonudur. Bazen nekrotizan fasiit ve sonrasında gelişen ağır cilt defektleri de görülebilir. Pnömoni, kardit, hepatit, glomerülonefrit, ensefalit ise suçiçeğinin diğer komplikasyonları arasındadır. Streptokokal toksik şok sendromu (STŞS), A grubu streptokokların invaziv enfeksiyonlarından birisi olup, ani ve hızlı bir başlangıçla birlikte şok ve organ yetmezliği ile kendini gösteren ve mortalitesi %30-50 olan ağır bir klinik tablodur. Bu çalışmada suçiçeği enfeksiyonu sonrası nadiren gelişen, ağır ve hayatı tehdit eden klinik bir tabloya neden olan STŞS benzeri olgu sunulacaktır.

Anahtar Kelimeler: Suçiçeği, streptokokal toksik şok sendromu, varisella zoster virüsü

Introduction

Chickenpox is a primary disease of varicella zoster virus (VZV), which is one of the human herpes viruses. VZV is a double-helix DNA virus of the alpha herpes virus family. Chickenpox is a disease that can be easily transmitted via air and aerosols from and direct contact with the infected person. The epidemiologic studies have shown that its contagiousness is more at the early stages of the disease (1). After a short incubation time, the lesions start recovery through formation of vesicles, macules, papules, and crusts. Chicken pox recovers within 7-10 days spontaneously, however, it may cause serious morbidity with

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more frequent complications and even mortality with increasing age. About two thirds of the hospitalizations due to chicken pox is seen in children (1). The VZV is specific to species and infects only humans; thus, it has no host other than humans (2).

Skin lesions after varicella infection are the most common complication of staphylococcal or streptococcal secondary infections. Occasionally, necrotizing fasciitis and severe skin defects may develop afterwards. Pneumonia, carditis, hepatitis, glomerulonephritis, encephalitis are other complications of chickenpox (3-6). Streptococcal toxic shock syndrome (STSR) is one of the invasive infections of the A group streptococci and is a severe clinical picture with sudden and rapid onset of shock and organ failure, with a mortality rate of 30-50% (7). It has been reported that the STSR incidence is 5-10/100.000, which is mostly sporadic (8). M protein and streptococcal pyrogenic exotoxins (SPE-A, SPE-B, SPE-C), which are virulence factors of group A streptococci (GAS), are indicated to be responsible in the pathogenesis of the disease. These factors, which act as superantigens as well as their anti- phagocytic and pyrogenic effects, respectively, increase endotoxin sensitivity, suppress IgM synthesis, stimulate cytotoxin release, thus leading to excessive T-cell proliferation and cause STSR formation (9).

In the present study, we present a streptococcal toxic shock syndrome-like case which is rare and a life-threatening clinical picture that develops after varicella infection.

Case Report

A seven-year two-month girl was administered to our hospital with complaints of rash and edema at the right half of the body, fever and weakness. The medical history revealed that the patient contacted a child having active varcella lesions about 2 weeks ago, that the patient had sporadic vesicular and papular itching lesions with slight erytrema at hairy skin, face, body, back and extremities for 5 days, that she was hospitalized in a different health care establishment to which they had gone due to the development of a bullous lesion on the right shoulder, was treated with parenteral ampicilin-sulbactam and teicoplaninfor for 2 days. However, the lesion that had started on the right shoulder firstly had spread rapidly despite the treatment, over the right side of the body and had been accompanied by concurrent fever (Figure 1-3). With the

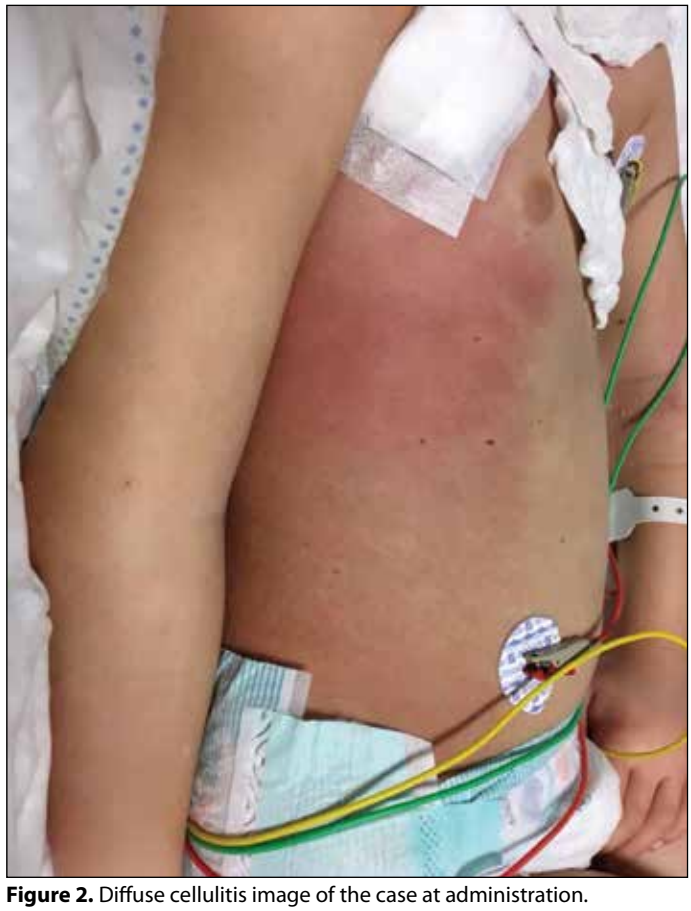

Figure 2. Diffuse cellulitis image of the case at administration.

Figure 1. Diffuse cellulitis image of the case at administration.

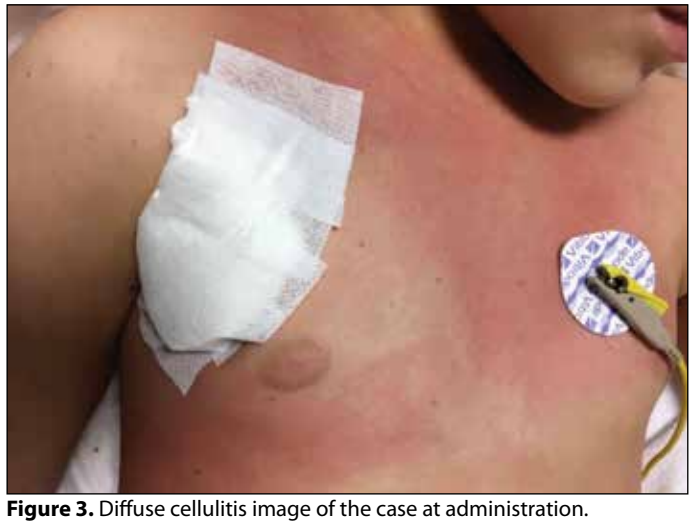

Figure 3. Diffuse cellulitis image of the case at administration.

deteriorating of the condition, low albumin, restriction in diuresis, the patient was referred to our hospital for further examinations and treatment. No specialty was detected in the background and family history. When her vaccination card of the patient was examined, it was seen that all her vaccinations were regularly made according to the national vaccination calendar, however, since varicella vaccination was not routine in Turkey at the time she was born, she was not vaccinated. In the physical examination, it was observed that her general condition and activity was at a medium level, she had a weak appearance. Her body weight was 20.3 kg (10-25 p), height 121 cm (25-50 p), armpit body temperature was 38.9°C. There were pleomorphic lesions, some crusted, some active varicella, on the hairy skin, body, back and extremities; there were 3 bullous lesions on the rght shoulder region, and there were pleomorphic varicells lesions with erythema and edema appearance, compatible with diffuse cellulites on the left half of the body. Cardiac apex beat was 127/min and was rhythmical; there were no additional sounds and no murmur. Arterial blood pressure was measured as 86/50 mmHg. Diuresis was slightly restricted, 0.7 cc/kg/h. other systemic and neurological examinations were assessed as normal.

In the laboratory assays, Hb was 11.8 g/dL, white cell count was 18.600/mm^3, thrombocyte count was 215.000/mm^3; in peripheric spread PNL was 82%, lymphocyte was 10%, monocyte was 8%. Erythrocyte sedimentation rate (ESR) was 90 mm/h and C-reactive protein (CRP) was 227 mg/L. Blood biochemistry values were as follows: Urea: 68 mg/dL, creatinine: 1.87 mg/dL, albumin: 2.3 mg/dL, AST: 38 U/L, ALT: 29 U/L, GGT: 42 U/L, total bilirubin: 0.96 mg/dL, direct bilirubin: 0.27 mg/dL. Routine urine tests, blood sugar, serum electrolytes and coagulation tests were within normal ranges. The serological tests were VZV IgM positive. *Streptococcus pyogenes* growth was observed in both sample cultures taken from the lesions and the case was assessed as a streptococcal toxic shock syndrome-like picture associated with invasive skin infection developed after varicella infection. Parenteral cephazolin (75 mg/kg/day) and clindamycin (40 mg/kg/day) were started as anti-biotherapy. A dramatic amelioration occurred within the first 12-24 hours of the treatment in the erytrema and edema cellulitis in the right half of the patient's body and a rapid decrease was observed in acute phase reactants. Supportive therapy was administered for acute renal failure and low level of albumin. Her therapy was completed to 10 days and after recovery in the lesions and laboratory findings, she was discharged.

Discussion

STSR is one of the serious invasive diseases; it develops associated with streptococci and has bas prognosis. Factors that are involved in STSR development include very small or old age, diabetes, alcoholism, surgical intervention, trauma, varicella infection, pregnancy, direct contact with a patient, high prevalence of invasive strains in the community and non-steroid drug use (1,2). In the present case, the patient was completely healthy previously, with no disease or immune-suppression condition. There was only a varicella infection onset about 2 weeks before. There are cases that in which STSR develops without any preparative factor in children and in healthy adults in the literature; however, the entrance door of the infection agent could not be found in 50% of such cases (7,8,10,11). In the present case, it is believed that the entrance door of the agent was the bullous lesions on the right shoulder. A serious cellulitis had appeared, rapidly spreading downwards from the right shoulder and "*Streptococus pyogenes*" growth was detected in both sample cultures.

The treatment of the toxic shock syndrome includes the detection of the source of the infection, surgery if necessary, appropriate supportive therapy in the fight with shock and immunotherapy as well as appropriate antibiotic use (7,8). Treatments in which anti-cytokines are used are still controversial (8). This patient, who had slightly restricted dieresis and hypoalbuminemia when admitted in the hospital, was administered parenteral cephazolin (75 mg/kg/day) and clindamycin (40 mg/kg/day) as anti-biotherapy, taking into account the kidney and liver functions. The patient had no hypotension after intravenous fluid treatment and no supportive cardiac inotropic treatment was necessary. It has been demonstrated in studies that Clindamycin is more effective because it inhibits SPE-A and SPE-B synthesis more than penicillin and thus, clindamycin is suggested in standard STSR treatment (12).

Toxic shock syndrome caused by streptococci is a severe picture. As in the present case, STSR must be considered in patients with a sudden and rapid shock picture in patients admitted with a diffuse skin and soft tissue infection. In the clinic, there is a bacteriamia picture associated with skin and soft tissue infection, shock, ARDS and kidney failure. Anti-biotherapy started at an appropriate and early stage and other supportive organ treatments must be urgently administered. Studies report that the mortality of STSR is about 30-50% and that this rate can rise up to 80% in the presence of necrotizing fasciitis (8-10). Consequently, streptococcal toxic shock syndrome is a picture which is rarely observed after varicella infections and which progresses rapidly with high mortality.

Therefore, early diagnosis, starting the therapy quickly and appropriately without delay is very important with respect to prognosis.

With respect to preventive medicine, it is suggested that the varicella vaccination, which has been included in the national vaccination program for about four years, is administered as a single dose in postnatal 12th month. The present case is presented accompanied with literature, so that the diagnosis and treatment of this disease is reviewed.

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