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Varicella Zoster Infection Followed by Steroid Use – A Case Report

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ABSTRACT

Varicella zoster virus (VZV) is a human virus that belongs to the herpes virus family. VZV is present worldwide and is highly infectious. We discussed a case repot of 11- year -old female who is reported to have severe pain along the spine with abdominal pain and skin rashes. Her medical history reveals that she was discharged from the hospital with prednisolone. She returned back on the fifth day after discharge with the presenting complaints. It was observed that her hemoglobin level and platelet counts were decreasing. Even after starting treatment for her condition, she died at the fourth day. The case report underlines the development of immunosuppression in patients taking corticosteroids.

INTRODUCTION

Varicella-zoster virus (VZV) is a human alpha herpesvirus that infects >90% of people. Primary VZV infection, commonly seen during early childhood, leads to varicella (chickenpox) and establishment of a lifelong latent infection in neurons of trigeminal ganglia (TG) and dorsal root ganglia (DRG). Later in life, VZV reactivates in approximately one third of infected individuals to cause herpes zoster ([HZ] shingles) [1]. Whereas primary VZV infection is generally benign, HZ is frequently accompanied by postherpetic neuralgia (PHN), and it is an important cause of uveitis and vasculitic stroke [2-3].VZV is highly communicable and spreads by the airborne route, with an extraordinarily high transmission rate in temperate countries [4]. Varicella-zoster virus (VZV) causes primary, latent, and recurrent infections. The primary infection is manifested as varicella (chicken pox) and results in establishment of a lifelong latent infection of sensory ganglion neurons. Prior to the introduction of vaccine in 1995, varicella was an almost universal communicable infection of childhood in the United States. Varicella is a more serious disease with higher rates of complications and deaths among infants, adults, and immunocompromised persons. Hospitalization rates for varicella are about 3 - 6 per 1,000 cases, and the complication rate ranges between 2% and 4%. Mortality rates are between 0 and 0.05 deaths/100,000 population per year in Europe [2]. Varicella-zoster virus infection reconfigures the T cells to become activated memory T cells with enhanced skin-homing capacity and reduced immune functions [5]. Consequently; VZV-infected T cells transport the virus to skin and possibly ganglia during primary infection [6]. Detection of VZV-infected T cells in blood during acute infection and observations in the SVV model support the role of T cells for interhost dissemination of the virus [7-8]. Although HZ patients may develop a low viremia [9], the analogous role of T cells in VZV reactivation has not been described. We report a case of previously healthy 11-year-old female child with varicella who developed hemorrhagic varicella zoster encephalitis associated with severe sepsis, pneumonia, severe hypoalbuminemia, and thrombocytopenia.

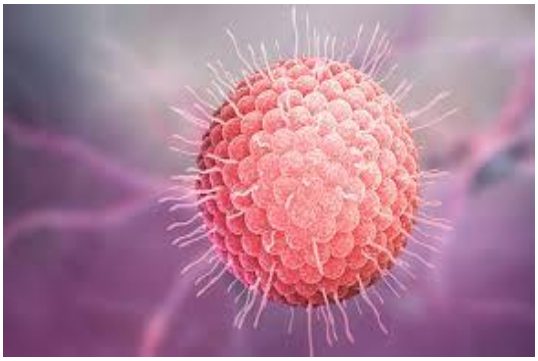

Figure No. 1: varicella zoster virus

CASE PRESENTATION

A 11- year -old South Indian female child weighing 30 kilograms was admitted to the hospital with severe pain along the spine for the last 8 hours, one episode of generalized tonic-clonic seizures, abdominal pain and skin rashes over the trunk.

Past medication history

Her past medication history revealed 30 days back she was hospitalized with complaints of gradual and progressive joints swelling in the lower limbs, fever with rashes involving thigh, abdomen and face. She was diagnosed to have infection associated vasculitis with reactive arthritis and pneumonia. She was treated with acetaminophen p.o. 500mg q.i.d. for 14 days, gentamycin i.v. 75mg b.i.d. for 3 days, amikacin i.v. 220mg b.i.d. for 11 days, ibuprofen p.o. 200mg for 5 days, pethidine i.v. 15 mg t.i.d. for 6 days, cefotaxime i.v. 2gm t.i.d. for 7 days, cloxacillin i.v. 1gm q.i.d. for 10 days, naproxen p.o. 125mg b.i.d. for 19 days, cloxacillin p.o. 750mg q.i.d. for 5 days, ranitidine p.o. 75mg b.i.d. for 12 days, prednisolone p.o. 60mg in three divided doses for 8 days followed by 50mg in three divided doses for 5 days. At the time of discharge, she was prescribed prednisolone p.o. 20mg b.i.d. for 3 days, followed by 10mg b.i.d for 3 days and 10 mg o.d. for next 3 days, ranitidine p.o. 75mg b.i.d. for 10 days and naproxen p.o. 125mg b.i.d for 7 days. She was on these medications for 4 days. On day 5 after discharge, she was readmitted to the hospital with the present complaints.

Physical examination

On examination pustular lesions filled with fluid were observed on trunk and abdomen. On abdominal examination tender hepatomegaly was present. Laboratory investigations at admission revealed positive - Tzank smear of pustular lesions, platelet counts - 2.6

lakhs/mm^3, Hemoglobin – 13.1 gm/dl, total count – 32,500 cells/ mm^3, INR – 1.7, PT – 20.1sec(Control – 12 sec), APTT – 38.1sec (Control – 30 sec), D-dimer – 10,000 ,ESR- 18 mm/hr, serum creatinine 0.7 mg/dl. She was diagnosed to have hemorrhagic varicella zoster encephalitis.

Treatment

She was treated with acyclovir i.v. 300mg q.i.d., pethidine i.v. 15mg SOS, tramadol i.v. 25mg q.i.d., naproxen p.o. 125mg b.i.d., cefotaxime i.v. 125mg q.i.d., gentamycin i.v. 75mg b.i.d., phenytoin i.v. 150mg b.i.d., mannitol i.v. 100ml t.i.d., ranitidine i.v. 50mg b.i.d., vitamin k i.v. 10mg o.d. These medications were administered for 3 days. She was also transfused with 5 pints of platelets, 2 pints of packed cells and 40ml of fresh frozen plasma. Laboratory investigations repeated on day 3 of admissions showed decrease in hemoglobin (5.5 g/dl), platelet count (30,000 cells/ mm^3), and increase in total count (68,100 cells/ mm^3), INR (4.4), PT - 50.1 sec (control- 12 sec), APTT - 48.8 sec (control – 30 secs), serum creatinine (1.6 mg/dl). She died on fourth day of hospitalization.

DISCUSSION

The VZV vaccine was approved by the US Food and Drug Administration in 1995 [10]. An association between steroid use and severe varicella has been recognized for decades, although most reports have come from relatively small retrospective series and case reports [11-14].

Glucocorticoids inhibit the formation of antibodies. The consequence of this interference with immune responses can be multiplication of bacteria and an increased risk of bacterial intoxication when infection does occur; hence, the frequency and severity of clinical infections tend to increase during glucocorticoid therapy. Infections such as chickenpox can have serious consequences, including death, in patients taking systemic glucocorticoids. It has been suggested that Varicella zoster immunoglobulin should be given to patients in contact with chickenpox if they have taken glucocorticoids in dosages over0.5 mg/kg/day during the preceding 3 months, in the context of near-fatal chickenpox in a child receiving prednisolone. A similar incident was reported that a 12-year-old boy received a total of prednisone 4 mg and methylprednisolone 500 mg while in the hospital for treatment of an acute asthma attack. The patient was discharged on prednisone 10 mg/day and 3 days later developed a varicella rash. The immunosuppressive action of prednisone is believed to have

allowed the infection to disseminate which eventually lead to the child's demise. In immunocompromised patients, such as in our case, acyclovir is recommended at the onset of varicella exanthem (Kasper & Howe, 1990). Here, the patient is having back pain as the initial symptom. Use of corticosteroid increases the risk of severe or fatal varicella. Our patient was administrated with high dose of steroid for 4 days. High dose of steroids (>2mg/kg/day) seems to be dangerous than low doses. The glucocorticoid effect of hydrocortisone 20mg/kg/day is equivalent to prednisone 4mg/kg/day. If corticosteroids were given at the incubation period of chicken pox the risk factors are high. It was reported that varicella is seen in patients taking intermittent course of high dose of corticosteroids.

CONCLUSION

The case report emphasizes the fact that immunosuppression occurs in a patient who is been administered with corticosteroids and it also highlights on the importance of starting acyclovir or similar antiviral on such group of patients at the onset of varicella infection. Health care professionals (HCPs) need to focus on these aspects while receiving patients who are on long-term or intermittent corticosteroids and need to provide adequate counselling to the patients who are on systemic corticosteroids.

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