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Virus-Associated Hemophagocytic Syndrome Caused by Influenza B and Varicella-Zoster Virus Co-Infection

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Abstract

Background: Varicella (chicken pox), which caused by the Varicella-Zoster Virus (VZV), is usually self-limiting and benign. However, VZV can lead to significant and serious complications, especially in immunocompromised patients or accompanied by other pathogens infection. HPS caused by varicella-zoster and influ B virus co-infection is rare.

Case presentation: A 15-years-old boy was admitted to our hospital because of general rash, severe back and low back pain. CT scan revealed pneumonia in left upper lobe. Initial blood tests showed normal blood WBC and PLT, mild liver dysfunction, enhanced D-dimer and myocardial enzyme. However, after 4-days treatment of acyclovir, antibiotics, and analgesic therapy, his pain did not relieved and fever developed. At the same time, hyperferritinemia, abrupt reduction on blood WBC and PLT count were observed. Virus-Associated Hemophagocytic Syndrome (VHAS) was confirmed. Then, intravenous drip dexamethasone (10 mg/day) and gamma globulin (10 g/day) were administrated. He recovered completely at last.

Conclusions: Disseminated Varicella which was accompanied by influenza B virus and bacteria infection is infrequent. Disseminated varicella may cause significant morbidity and even mortality in immunocompromised patients. Hemophilic syndrome induced by duel-virus and bacteria infection is limited and usually fetal. Anti-bacterial therapy, early identification of this syndrome and timely administration of glucocorticoids and gamma globulin are the key links of treatments.

Keywords: Coinfection; Virus; Varicella pneumonia; Varicella hepatitis; Varicella myocarditis

Abbreviations: VZV: Varicella-Zoster Virus; WBC: White Blood Cells; HPS: Hemophagocytic Syndrome; VHAS: Virus-Associated Hemophagocytic Syndrome; AIHA: Autoimmune Haemolytic Anaemia; VZP: Varicella-Zoster Pneumonia; ALF: Acute Liver Failure; CNS: Central Nervous System; PLT: Platelet; PCT: Procalcitonin; ECG: Electrocardiogram

Introduction

Varicella, which caused by the Varicella-Zoster Virus (VZV), is usually self-limiting and benign. However, VZV can lead to significant and serious complications, especially in immunocompromised patients or in those who is accompanied by other pathogens infection. HPS caused by varicella-zoster and influ B virus co-infection is rare. In this study, we report a HPS case which is correlated with influ B and varicella-zoster virus co-infection in a boy who is immunocompromised because of oral prednisolone.

Case presentation

A 15-year-old boy went to the hospital complaining of 2 days of rash (Figure 1), back pain and headache, 1 day of aggravation. Two days ago, he felt headache along with fixed, unbearable and continuous pain in his back. At the same time, distributed red papules were discovered in his chest, back, and both upper limbs. Then he was transferred to hospital and admitted to Department of Pain Management treatment of our hospital.

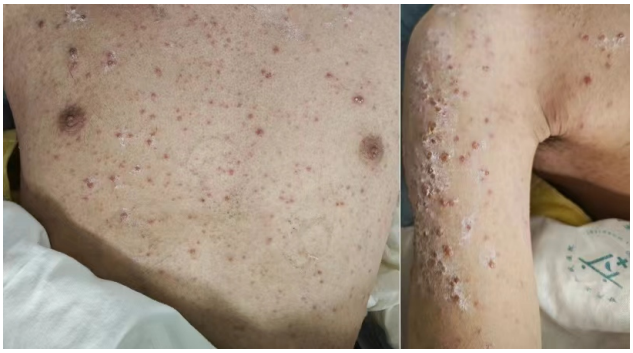

Figure 1: Appearance of rash on admission.

The patient suffered Mycoplasma pneumonia two weeks ago. He was treated with 10 days anti-Mycoplasma antibiotics and had a complete recovery on both symptoms and imageological examination. Oral 20 mg prednisolone per day was administrated as discharge medications.

At admission, the patient's temperature was 36.7°C, heart rate was 61 bpm, respiratory rate was 13 per minute, blood pressure was 119/66 mmHg, and oxygen saturation in room air was 97%.

The young man was diagnosed as varicella, varicella pneumonia, varicella hepatitis, varicella myocarditis and influ B infection. However, after 4-days treatment of acyclovir, antibiotics, and analgesic therapy, his pain did not relieved and fever developed. At the same time, hyperferritinemia, abrupt reduction on blood WBC and PLT count were observed. Virus-Associated Hemophagocytic Syndrome (VHAS) was confirmed. Then, intravenous drip dexamethasone (10 mg/day) and gamma globulin (10 g/day) were administrated. Other adjuvant therapy, including liver protection and down-enzyme measurements, were also used. His temperature dropped to normal rapidly. The papules gradually dried up and crusted (Figure 2).

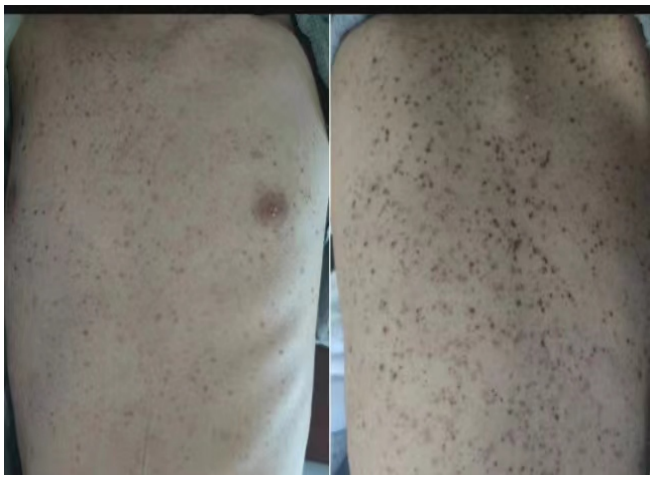

Figure 2: Rash changes after treatment.

Further laboratory examination showed blood WBC of 9.9 × 10^9, lymphocyte of 2.27 × 10^9, PLT of 247 × 10^9, PCT of 0.358 ug/ml, IL-6 of 9.04 ug/ml, SAA of 31.65 mg/L, D-dimer of 1.65 ug/ml and FDP of 5.8 ug/ml. Liver function revealed TP of 60.4 g/L, ALB of 39.7 g/L, globulin of 20.7 g/L, AST of 52 U/L and ALT of 49 U/L. Myocardial enzymes showed AST of 318 U/L, CK of 578 U/L, CK-MB of 52 U/L and LDH 1405 of U/L. ECG displayed nodal tachycardia. Cerebrospinal fluid tests showed no abnormalities. Triglyceride and FDP were normal. IgM antibody of Mycoplasma pneumoniae was positive. PCR of oropharyngeal swab was positive on Influ B. Both oropharyngeal and rash swab results on VZV were positive. T cell subsets revealed $CD3^+$ of 1069/ul, $CD4^+$ of 133/ul and $CD8^+$ of 934/ul. Serum soluble IL-2 receptor was 1197 U/mL and ferritin is greater than 2000 ng/mL. CT-scanning displayed infiltration in multi-lobes (Figure 3a).

The pain eased until it disappeared. Reduced blood PLT slowly rising to normal. Rising serum PCT, ferritinemia, myocardial and liver enzyme lowered to normal gradually. Exudative lesion in left superior lobe was absorbed completely (Figure 3b). He recovered completely. After 4 weeks of follow-up, the rash basically subsided.

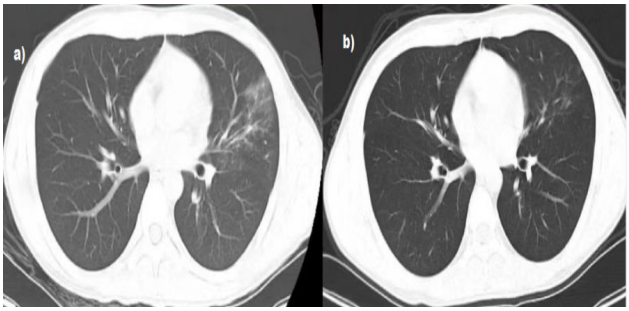

Figure 3: a) Pulmonary exudation changes after treatment; b) Rash changes after four weeks later.

Discussion

Varicella zoster virus infection can occur in any population, but is common in immunocompromised individuals. The boy oraled prednisone treatment for other reason before admission, which suppressed his immune system and result in his lowed $CD4^+$ count of 133/ul. Abnormal immunity increased risk of opportunistic VCV infection.

VZV infection causes varicella or herpes zoster (shingles). Mild varicella is a self-limiting disease. Disseminated varicella involves varicella pneumonia, varicella hepatitis, varicella myocarditis, varicella encephalitis and other complications. Varicella-Zoster Virus (VZV) may cause significant morbidity and even mortality in immunocompromised patients. Varicella has more serious consequences than herpes zoster, although zoster is more common [1].

In central nervous system, progressive encephalomyelitis with rigidity and myoclonuss, progressive lower cranial and upper cervical polyneuropathy or Encephalitis can be triggered by VZV infection or other virus coinfection [2-6]. Rare myelitis cases were presented in the literature [7]. A case of multifocal intracranial stenosis which was ultimately discovered to be led by vasculitis due to VZV infection was reported [8]. A Korea retrospective analysis showed meningitis incidence of 17.8% and meningoencephalitis incidence of 1.5% in adult patients with clinical manifestations of CNS disease [9].

In digestive system, VZV infection may lead to gastritis [10], acute pancreatitis [11], or Acute Liver Failure (ALF) [12]. VZV infection also play a role in severe autoimmune hepatitis because of immune cross reaction and autoimmune disorder [13,14].

In blood system, Autoimmune Haemolytic Anaemia (AIHA) associated with varicella infection was reported [15]. Yeager AM et al., hypothesize the existence of at least infectious and post-infectious pathogenetic mechanisms in varicella-associated thrombocytopenia [16]. In cardiovascular system, acute myopericarditis can be sequelae of varicella [17,18].

VZV infection have a causal role in triggering autoimmunity which can induce polymyositis [19]. Varicella zoster also have a rare association with erythema multiforme [20]. Few cases of acute calvarial osteomyelitis or rhabdomyolysis associated with VZV infection or reactivation were reported [21,22]. VZV is also considered

to be a common causative virus in younger acute retinal necrosis patients [23].

According to literature, incidence of Varicella-Zoster Pneumonia (VZP) is less than 5% in healthy individuals and 5%-10% in immunocompromised hosts respectively. Patients with VZP have the rash for a few weeks. A neonatal case of bronchopneumonia and hemorrhagic pulmonary edema caused by VZV infection was reported in literature [24].

Conclusion

Disseminated Varicella which was accompanied by influenza B virus and bacteria infection is infrequent. Clinicians need to recognize this infection which caused by more than one pathogens. Hemophilic syndrome induced by duel-virus and other pathogen infection is limited and usually fetal. Anti-pathogen therapy, early identification of haemophilus syndrome and timely administration of glucocorticoids and gamma globulin are the key links of treatments.

Authors' contributions

Wang Yuyu and Yan Dongning took care of the patient, wrote the draft. Wang Yuyu prepared Figures 1-4. Chen Xiaojun took care of the patient and critically revised the manuscript. All authors read and approved the final manuscript.

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Availability of data and materials

All data generated or analysed during this study are included in this published article.

Consent for publication

Verbal and written consent for publication was obtained from patient and his parents.

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