Journal of the Postgraduate Institute of Medicine 2023; 10(1): E 211 1-6

https://doi.org/10.4038/jpgim.8419

Case Report

A rare case of disseminated varicella zoster infection in a healthy young adult

Deemantha Lalana Rathnayaka, Inoka Kumuduni Jayasinghe, Chiranjaya Bandara Ekanayaka

National Hospital, Kandy, Sri Lanka.

Key words: **haemorrhagic varicella, thrombocytopenia, varicella encephalitis, immunocompetent adult**

Corresponding Author:, Deemantha Lalana Rathnayaka E-mail:< deemanthalalana@gmail.com > https://orcid.org/0000-0003-1293-6968

Received: 28 Feb 2023, accepted revised version: 29 Mar 2023 Published: 30 Jun 2023

Competing Interests: Authors have declared that no competing interests exist

Introduction

Varicella-zoster virus (VZV) is one of the herpes viruses (herpes virus 3) known to cause human infection and is distributed worldwide. It causes two clinically distinct forms of the disease. Primary infection with VZV causes a diffuse vesicular rash which is called chicken pox. Endogenous reactivation of latent VZV is called herpes zoster or shingles. Primary varicella infection is generally a self-limiting mild disease in the paediatric age group but can cause more severe disease in adults or immunocompromised patients of any age.

Here, we present a case of disseminated varicella zoster infection in a 29-year-old immunocompetent young patient who presented to the National Hospital, Kandy (NHK). It is a rare incident and only a few case reports of disseminated varicella zoster in immunocompetent patients are available, in which the majority are aged above 65 years.

Case report

A 29-year-old man was admitted to NHK with a history of fever of 5 days duration and a generalized skin rash of 3 days duration which had progressed over time. He was a diagnosed patient with epilepsy and had an episode of generalized tonic-clonic convulsions the day before his hospital admission. He did not have any previous history of VZV infection and had not been vaccinated against VZV.

On admission, the patient was febrile and had typical polymorphic macules, papules, vesicles and pustules, together with multiple haemorrhagic blisters. These lesions were predominantly on his face, chest and abdomen (Figures 1&2). There were features of secondary bacterial infection in the facial lesions. Bilateral red eyes with features of scleritis were also noted (Figure 3).

http://doi.org/10.4038/jpgim.8419

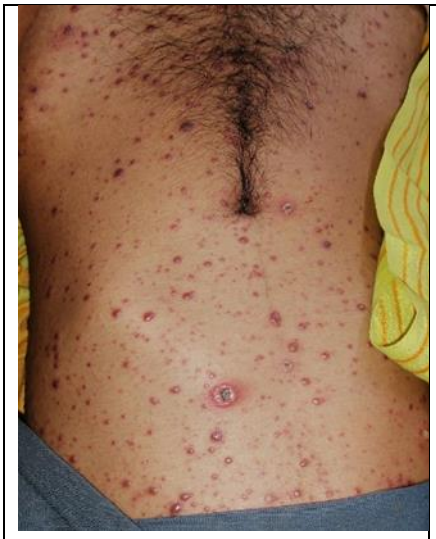

Figure 1: Generalized skin lesions

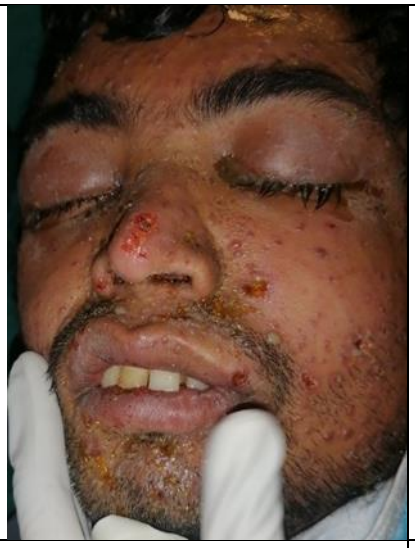

Figure 2:Haemorrhagic blisters on face

Figure 3: VZV scleritis

The Glasgow coma scale (GCS) was 15/15 and there were no signs of meningism. His blood pressure was 110/60 mmHg and he was tachycardic with a pulse rate of 110 bpm. The respiratory examination was normal and he had mild hepatic tenderness on abdominal examination. His basic investigations were as follows (Table 1)

Table 1: Basic investigations

Test	Patient's value	Reference range
Full blood count		
WBC	5.54 x 10^3/µL	4.0 – 10.0
Neutrophils	78%	50 – 70 %
Lymphocytes	12.5%	20-40 %
Eosinophils	0.1%	0.5 – 5.0 %
Hb	14.2	12.1 -16.6
Platelet count	42 x 10^3/µL	150 – 400
C-reactive protein	144	< 8
Liver function tests		
AST	506 U/L	≤ 35
ALT	364 U/L	≤ 45
Total bilirubin	5.1 µmol/L	5 -19
Direct bilirubin	2.7 µmol/L	1.7 – 6.8
Total protein	6.5 g/dL	6.6 – 8.3
PT/INR	1.78	1.0-1.3
Renal function tests		
Serum creatinine	65 µmol/L	65 -120
Sodium	136 mmo/L	137 -148
Potassium	3.8 mmol/L	3.9 – 5.2
Fasting blood sugar	5.4 mmol/L	3.9 -5.4

WBC-white blood cell count, Hb-Haemoglobin, AST – Aspartate aminotransferase, ALT- Alanine transaminase

http://doi.org/10.4038/jpgim.8419

The blood picture revealed a reactive film with moderate thrombocytopaenia and no evidence of microangiopathic haemolytic anemia. Electroencephalogram (EEG) showed features of generalized encephalopathy. Lumbar puncture was not done as the patient had severe thrombocytopenia and active skin infection in the lumbar region.

Blood culture was positive for group B streptococci. Human immunodeficiency virus (HIV) 1 & 2 antibodies were negative. Hepatitis B surface antigen and hepatitis C antibody were negative. VZV IgM antibody was positive and VZV IgG antibody was negative. The patient was managed as disseminated VZV infection complicated by febrile haemorrhagic purpura, VZV encephalitis, VZV induced thrombocytopenia, VZV hepatitis, VZV scleritis and sepsis.

The patient was started on intravenous acyclovir 500 mg 8 hourly for 14 days with broad spectrum antibiotics to treat sepsis. His skin rash improved dramatically (Figure 4) and liver enzymes and thrombocytopenia also resolved with treatment (on discharge, AST – 296 U/L, ALT- 185 U/L, platelet count - 237 10^3/µL).

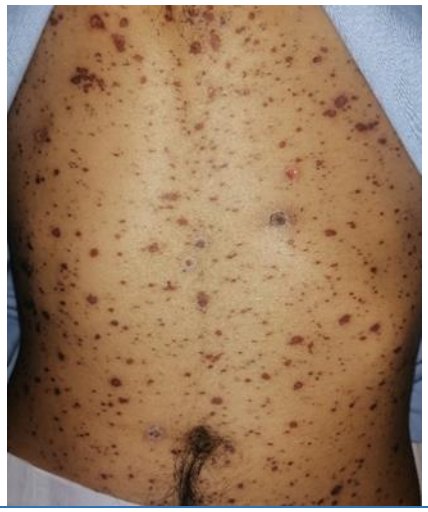

Figure 4 – Healed lesions after treatment

Discussion

Primary VZV infection is generally transmitted by infectious respiratory secretions. Viral replication takes place in the lymphatic tissue. Primary viraemia is followed by a second viral replication within the liver and spleen. A secondary viraemia allows access to the epidermis, whereupon the classic clinical presentation of varicella manifests. The average incubation period for varicella infection is 14 to 16 days [1]. As the cutaneous infection subsides, usually over 1-2 weeks, the virus enters into quiescence within sensory neurons. The virus is kept in this state of quiescence by a competent cell-mediated immune system. Any condition that causes immunity to fade may allow the virus to

http://doi.org/10.4038/jpgim.8419

reactivate, travel down axons, and manifest as the cutaneous infection known as herpes zoster.

Primary varicella infection typically causes a vesicular rash, which is usually pruritic and appears in successive crops over several days. The lesions begin as macules that rapidly become papules followed by characteristic vesicles; these lesions can then develop a pustular component followed by the formation of crusted papules [1]. The patient with varicella typically has lesions in different stages of development on the face, trunk and extremities. New vesicle formation generally stops within four days, and most lesions have fully crusted by day six in normal hosts [2]. Crusts tend to fall off within about one to two weeks and leave a temporary area of hypopigmentation in the skin [1]. The period of infectivity is generally considered to last from 48 hours prior to the onset of rash until skin lesions have fully crusted.

Primary varicella infection is generally self-limiting. Dissemination occurs more commonly in primary infection and almost exclusively in immunocompromised patients resulting in generalized rash, pneumonitis, hepatitis, and encephalitis [3]. In our literature survey, we found only a few cases reports of disseminated VZV infection and, among them, the majority occurred in elderly populations over the age of 65 years with relatively weaker immune systems [4-10].

Our patient, with primary varicella infection complicated by generalized haemorrhagic purpura, encephalitis, hepatitis, thrombocytopenia and scleritis, did not have any apparent underlying immunosuppression, hence we believe this case highlights the rare occurrence of disseminated VZV infection in an immunocompetent young adult.

Disseminated VZV infection can cause many complications. Haemorrhagic varicella is a rare complication of chicken pox which causes cutaneous bleeding and usually occurs in immunocompromised persons and those on immunosuppressive therapy. It has a mortality rate between 7-10% [11]. Diffuse encephalitis is considered to be the most serious complication and causes convulsions and altered level of consciousness. Varicella pneumonia accounts for the majority of adult morbidity and mortality [12-14]. Thrombocytopaenia is also a common complication of chickenpox, especially in adults where it is observed at four times the frequency compared with children [15]. Varicella-associated thrombocytopaenia usually develops early during the viremia phase or in the post-infectious period, weeks and months afterwards. The majority of patients are asymptomatic, although bleeding from the skin or mucus membranes can be seen in some patients. Severe haemorrhage, such as intracranial haemorrhage (ICH), overt gastrointestinal bleeding, and hematuria, is uncommon [16,17]. Hepatitis associated with the varicella infection is uncommon but can be progressed to fulminant hepatic failure if untreated [18].

Conclusion

Although primary VZV infection is a self-limiting, mild disease in paediatric populations it can cause devastating complications in the adult population. These complications are

common in immune deficit patients but can rarely occur in immunocompetent patients as well. Therefore, active management of primary VZV infection in adults is important.

Acknowledgements

Dr. Sawandika Rupasinghe for guidance in writing this case report.

References

1. Heininger U, Seward JF. Varicella. The Lancet. 2006;368(9544):1365-76. https://doi.org/10.1016/S0140-6736(06)69561-5
2. STRAUS STEPHENE. Varicella-zoster virus infections. Annals of Internal Medicine. 1988;108(2):221. https://doi.org/10.7326/0003-4819-108-2-221
3. Arvin AM. Varicella-zoster virus. Clinical Microbiology Reviews. 1996;9(3):361-81. https://doi.org/10.1128/CMR.9.3.361
4. Moriuchi H, Moriuchi M, Sun C-CJ, Trucksis M. Disseminated cutaneous zoster and aseptic meningitis in a previously healthy patient. Journal of Infection. 1997;35(2):183-5. https://doi.org/10.1016/S0163-4453(97)91842-9
5. Burdett C, Mendoza N, Arora A, Bartlett B, Gewirtzman A, Tremaine AM, et al. A rare case of disseminated shingles in an immunocompetent patient following a 7-day treatment with oral valacyclovir. Journal of Clinical Virology. 2008;43(2):233-5. https://doi.org/10.1016/j.jcv.2008.06.018
6. O'Toole EA, Mooney EE, Walsh JB, Sweeney EC, Barnes L. Disseminated herpes zoster in the elderly. Irish Journal of Medical Science. 1997;166(3):141-2. https://doi.org/10.1007/BF02943592
7. Gupta S, Jain A, Gardiner C, Tyring SK. A rare case of disseminated cutaneous zoster in an immunocompetent patient. BMC Family Practice. 2005;6(1). https://doi.org/10.1186/1471-2296-6-50
8. KJ, Kim SH, Lee EH, Choi JH. Disseminated herpes zoster in an immunocompetent elderly patient. The Korean Journal of Pain. 2013;26(2):195-8. https://doi.org/10.3344/kjp.2013.26.2.195
9. Castillo C, Muruaga A, Carbonell A. Disseminated herpes zoster in an immunocompetent host. Journal of the American Geriatrics Society. 2012;60(6):1170-. https://doi.org/10.1111/j.1532-5415.2012.03973.x
10. Capron J, Steichen O. Disseminated zoster in an elderly patient. Infection. 2009;37(2). https://doi.org/10.1007/s15010-008-8287-5
11. Sterling JC. Virus infections. Rook's Textbook of Dermatology, 7th ed. 2004;:1095-178. https://doi.org/10.1002/9780470750520.ch25
12. Heininger U, Seward JF. Varicella. The Lancet. 2006 Oct 14;368(9544):1365-76. https://doi.org/10.1016/S0140-6736(06)69561-5
13. Fleisher G. Life-threatening complications of varicella. Archives of Pediatrics & Adolescent Medicine. 1981;135(10):896. https://doi.org/10.1001/archpedi.1981.02130340008004
14. Anne G. Varicella and Herpes zoster: Clinical disease and complications. Herpes 2006;13:2-7A

15. Marin M, Watson TL, Chaves SS, Civen R, Watson BM, Zhang JX, et al. Varicella among adults: Data from an active surveillance project, 1995-2005. The Journal of Infectious Diseases. 2008;197(s2). https://doi.org/10.1086/522155
16. PF; TPLTFP. [the behaviour of platelets in some viral infectious diseases in childhood (author's transl)]. Annali Sclavo; rivista di microbiologia e di immunologia. U.S. National Library of Medicine; [cited 2023Feb23]. Available from: https://pubmed.ncbi.nlm.nih.gov/7247494/
17. Wright JF, Blanchette VS, Wang H, Arya N, Petric M, Semple JW, et al. Characterization of platelet-reactive antibodies in children with varicella-associated acute immune thrombocytopenic purpura (ITP). British Journal of Haematology. 1996;95(1):145-52. https://doi.org/10.1046/j.1365-2141.1996.d01-1872.x
18. Ajenifuja, Adeniyi A. A rare case of VZV-induced hepatitis. American Journal of Gastroenterology; 2018. https://doi.org/10.14309/00000434-201810001-02446