

Herpes zoster following *Streptococcus suis* infection: A case report

Putu Shinta Widari Tirka¹, Ni Made Dwi Puspawati¹, Tommy Sarongku², I Putu Eka Widyadharma²

¹ Department of Dermatovenerology, Faculty of Medicine, Udayana University, Sanglah General Hospital, Bali, Indonesia

² Department of Neurology, Faculty of Medicine, Udayana University, Sanglah General Hospital, Bali, Indonesia

ABSTRACT

Background. Herpes zoster is a disease caused by reactivation of endogenous viruses that cause local dermatomal disorders characterized by unilateral radicular pain and vesicular dermatomal eruption. Some of the factors that responsible for reactivation of the herpes zoster virus are older age and low immune status.

Case presentation. A 54-years-old presenting with meningitis after consumed a raw pork. Five days later developed herpes zoster.

Conclusions. Herpes simplex reactivation in *Streptococcus suis* infection might relate to low immune system caused by the poor immune response to antibody and the use of high-dose systemic steroid.

Keywords: herpes zoster, *Streptococcus suis*, meningoencephalitis, immune, infection

BACKGROUND

Herpes zoster is a viral infection that cause a local dermatomal disorders characterized by unilateral radicular pain and vesicular dermatomal eruption. The incidence of herpes zoster is 1.5-3 people per 1000 population per year in Europe and North America and increases with age, especially when over the age of 50 years [1,2]. Some of the factors responsible for herpes zoster reactivation are older age and poor immune status [2].

Bacterial meningitis is a severe infectious disease with high mortality and morbidity. One of the most common pathogens that cause bacterial meningitis is *Streptococcus suis*; this pathogen has natural reserves in pigs and can cause meningitis in humans after contact with infected pigs. *Streptococcus suis* infection is endemic in Southeast Asia, including Bali, Indonesia [3].

Meningitis infection due to *Streptococcus suis* causes a decrease in the immune system, so it has a higher chance of suffering from herpes zoster as

part of an opportunistic infection and as an adverse reaction of high-dose systemic steroid. Here we report a case of herpes zoster in a patient with bacterial meningoencephalitis caused by *Streptococcus suis*. This case presented to add knowledge about herpes zoster and its relation to meningitis *Streptococcus suis*.

CASE PRESENTATION

A Balinese male, 54-years-old, with complaints of vesicles on the cheeks and lip area. Five days before, the patient was complaining of fever, headache, and pain on the left cheek and around the lips. Then reddish spots appeared on the left cheek that spread to the lip area. Then vesicles started to appear on the reddish spots. The vesicles multiplied, some of them broken and causing crusted wounds. The patient complained that the pain felt like prickling and burning heat on the left cheek and lips. The pain was so intense that the patient was difficult to open his mouth and eat.

Corresponding author:

Tommy Sarongku

E-mail: tommysarongku@student.unud.ac.id

Article history:

Received: 15 September 2021

Accepted: 20 December 2021

Previously, the patient was unconscious and admitted to the Emergency Room. Initially, the patient was said to have a fever, headache, general weakness, nausea and vomiting since five days before, the symptoms were worsening the patient was said more restless and unable to communicate then was found unconscious. The patient had a history of raw pork consumption a week before and a history of chickenpox in his childhood.

The patient's vital sign was normal, with a high temperature of 39.5°C and meningeal sign (+). CT scan with contrast showed a leptomeningeal enhancement on the right parietooccipital (Figure 1). Lumbar puncture performed, cerebrospinal fluid was yellowish (Figure 1), with Nonne (+++) and Pandy (++++), on microscopic the total cells were 3,350 cells/mm³ with the dominance of 80% polymorphic cells. Cerebrospinal were sent to microbiology for culture, and *Streptococcus suis* bacteria were isolated. Tzank test was showing multinucleated giant cells found from the vesicles.

Dermatological status on the lip and left mandibular showing multiple form of vesicles on erythematous base, containing serous fluid, round to oval shape with a diameter of \pm 0.3 - 0.5 cm and with healthy skin between the lesions. In some places, there were multiple erosion, clear edges, geographic shapes with a size of \pm 0.2 x 0.3 cm to 0.3 x 0.4 cm,

partly covered with black-brown-yellow crust. The lesions were unilaterally distributed according to the cranial nerve V3 (figure 2).

The patient was diagnosed with meningoencephalitis caused by *Streptococcus suis* infection and herpes zoster. The patient was given 2 grams of Ceftriaxone injection every 12 hours for 21 days, 10 mg of dexamethasone injection every 6 hours for 5 days, 30 mg of lansoprazole every 12 hours and 750 mg paracetamol every 8 hours, and 200 mg gabapentin every 24 hours orally.

For the herpes zoster, the patient was treated by dermatovenereologist with 800 mg acyclovir tablets every 4.5 hours, vitamins B1, B6, B12 tablets every 24 hours, topical gentamicin ointment given every 12 hours in the erosion site, compress saline solution 0.9% every 8 hours for 10-15 minutes in crusted lesions.

In day 16, no new lesions found. The painful sensation on the lip and left cheek diminished. Dermatological status on the lip and the left mandibular showed macular hypopigmentation and multiple hyperpigmentations, firm edges, geographic shapes with a size of 0.5 x 0.7 cm - 0.5 x 1 cm, partially covered by black crust (Figure 3). Patient was allowed to be discharged from the hospital and was asked to do a follow up on the 21st day in polyclinic.

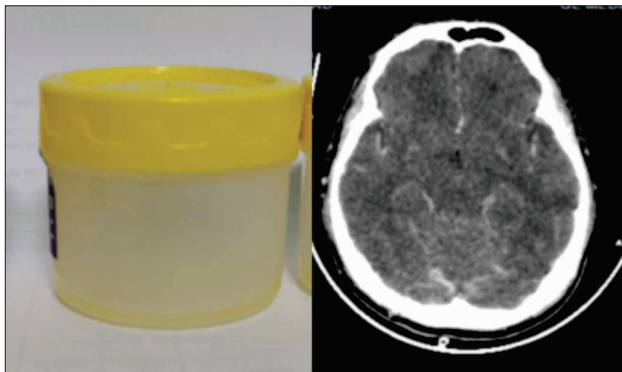


FIGURE 1. A yellowish scanty cerebrospinal liquor collected from the patient before the antibiotics administered (left). Head CT scan with contrast showing a leptomeningeal enhancement on the left parietooccipital (right)



FIGURE 2. Multiple form of vesicles on erythematous base, containing serous fluid, round to oval shape. The lesions were unilaterally distributed according to the cranial nerve V3



FIGURE 3. Follow-up day 16, macular hypopigmentation and multiple hyperpigmentations, firm edges, geographic shapes with a size of 0.5 x 0.7 cm - 0.5 x 1 cm, partially covered by black crust



FIGURE 4. Follow up day 21, no lesion appeared, and the wound had dried

In day 21, no lesion appeared, and the wound had dried. Fever was absent, pain in the lips, and the left cheek were absent. Dermatological status on the lip and left mandibular showed macular hypopigmentation and multiple hyperpigmentation, firm edges, geographic shape with the size of 0.5 x 0.5 cm - 0.5 x 1 cm partially covered by white crust (Figure 4).

DISCUSSION

Bacterial meningitis is a severe infectious disease with high mortality and morbidity [1]. The estimated incidence is 2.6-6 per 100,000 adults per year in developed countries and is several times higher in low-income countries [1]. One of the most common pathogens that cause bacterial meningitis is *Streptococcus suis*, where this pathogen has natural reserves in pigs and can cause meningitis, endocarditis, and sepsis in humans after contact with pigs [4]. As a result of high pig consumption and maintenance of small scale pigs, *Streptococcus suis* infection is endemic in Southeast Asia, where several studies reported, are mostly associated with pig breeding, butchers and history of eating raw pork [1,2]. The most common clinical symptoms in meningitis are fever or a history of fever, meningeal sign, altered mental status, and headache [1]. In *Streptococcus suis* infections, sensorineural hearing loss is a common complication [1,2,3].

Herpes zoster is a local dermatomal disease characterized by unilateral radicular pain and ve-

sicular dermatomal eruption [4]. Herpes zoster is the result of reactivation of the latent endogenous virus in ganglion neurons after previous varicella infection. Clinical manifestations of herpes zoster begin with prodromal symptoms such as pain, itching, hyperesthesia, allodynia, and unilateral paraesthesia in the affected skin dermatome. These symptoms can be accompanied by fever, headache, malaise, and anorexia, which can last for 1-5 days [4,5].

The risk factor that responsible for herpes zoster reactivation are older age and low immune system, induced by high dose of systemic corticosteroid, and also some conditions like bone marrow transplant, hematologic metastasis, solid tumor, and immune-mediated disease such as systemic lupus erythematosus, rheumatoid arthritis [4,6].

The use of corticosteroids in adults and children with acute bacterial meningitis as an adjuvant therapy is favourable to prevent neurologic complications and mortality [7]. But a high dose of systemic steroid would be harmful to some patients with low immune status. The recurrence of herpes simplex were found in patient that were treated with high dose of systemic corticosteroid.

The reactivation process of the varicella-zoster virus (VZV) is related to specific immunity to herpes zoster or VZV-specific CD4 T cells. When VZV-specific CD4 T cell levels fall below the threshold, it can facilitate the reactivation of the varicella-zoster virus, which causes herpes zoster infection [4]. Reac-

tivation of the varicella-zoster virus is also associated with a decreased ability of T cell memory to recognize VZV antigens in the body [4]. Varicella primary infection will form VZV-specific CD4 T cells in different amounts depending on the individual's immunity at that time [4]. After a primary VZV infection, the virus will remain latent in the dorsal nerve ganglion. VZV-specific CD4 T cell levels decrease gradually with age but are also periodically increased by subclinical infections due to VZV exposure [5].

In general, clinical symptoms of *Streptococcus suis* infection and poor antibody responses will in-

terfere with the adaptive immune response [3]. Cell-mediated immunity failed to prevent the herpes zoster reactivation because the reduced CD4 cell count is associated with the development of herpes zoster [2].

CONCLUSION

Herpes simplex reactivation in meningitis infection might relate to low immune system caused by the poor immune response to antibody and the use of high-dose systemic steroid.

REFERENCES

1. Dutkiewicz J, Sroka J, Zając V, Wasiński B, Cisak E, Sawczyn A, Kloc A, Wójcik-Fatla A. *Streptococcus suis*: a re-emerging pathogen associated with occupational exposure to pigs or pork products. Part I – Epidemiology. *Ann Agric Environ Med.* 2017;24(4):683-695.
2. Lecours MP, Letendre C, Clarke D, et al. Immune-responsiveness of CD4+ T cells during *Streptococcus suis* serotype 2 infection. *Sci Rep.* 2016;6:38061.
3. Susilawathi NM, Tarini NMA, Fatmawati NND, Mayura PIB, Suryapraba AAA, Subrata M, Sudewi AAR, Mahardika GN. *Streptococcus suis* associated Meningitis, Bali, Indonesia, 2014-2017. *Emerg Infect Dis.* 2019;25(12):2235-42.
4. Levin MJ, Schmader KE, Oxman MN. Varicella and herpes zoster. In: Fitzpatrick's dermatology in general medicine, 9th ed. New York: McGraw-Hill; 2019:3035-3064.
5. Johnson RW, Alvarez-Pasquin MJ, Bijil M. Herpes zoster epidemiology, management, and disease and economic burden in Europe: a multidisciplinary perspective. *Ther Adv Vaccines.* 2015;15(2):108-124.
6. Smith EB, Powell RF. Corticosteroids in herpes simplex and zoster. *Int J Dermatol.* 1975;14(5):341-344.
7. Brouwer MC, McIntyre P, Prasad K, van de Beek D. Corticosteroids for acute bacterial meningitis. *Cochrane Database Syst Rev.* 2015; (9):CD004405.