CASE REPORT

SIMULTANEOUS ONSET OF HERPES ZOSTER IN A FATHER AND SON

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The Varicella Zoster virus persists in sensory nerve ganglion cells after chicken pox and gets reactivated to cause herpes zoster after variable periods of time as a result of waning of specific cellular immunity. Susceptible contacts of herpes zoster can develop chicken pox and very rarely herpes zoster. We report an interesting case of a father and his son who developed herpes zoster simultaneously without any obvious common predisposition and discuss the possible underlying mechanism.

Key Words: Herpes zoster, Simultaneous onset, Incubation period, Family.

INTRODUCTION

Varicella Zoster virus, a ubiquitous neurotropic human herpesvirus, was isolated in 1958.1 It causes chicken pox and then remains latent for decades in cranial nerve, dorsal root and autonomic nervous system ganglia along the entire neural axis. Virus reactivation produces herpes zoster, usually restricted to 1-3 dermatomes.2 Waning of specific cellular immunity is said to be the cause of this reactivation, therefore, incidence of herpes zoster increases with advancing age. However, young adults also suffer from herpes zoster and the overall incidence of herpes zoster is 215 per 100,000 person-years.3

Patients suffering from herpes zoster are infectious and chickenpox or very rarely herpes zoster develops in susceptible contacts of herpes zoster. Under such circumstances the incubation period of herpes zoster is not known precisely.

We describe a 58 years old father suffering from herpes zoster, whose, otherwise healthy, 19 years old son, also developed herpes zoster within 24 hours of his father’s eruption.

CASE REPORT

A 58 years old man reported in dermatology outpatient department of Combined Military Hospital, Abbottabad, along with his 19 years old son with the complaints that both of them almost simultaneously developed a painful eruption over one side of the chest.

CASE 1

A 58 years old male patient told that about three days back he developed pain over left side of chest. Next day, a few lesions appeared over the site of pain, which rapidly increased in extent and involved both anterior as well as posterior aspect of left side of chest. There was no history of any unusual physical or mental stress, trauma, and exposure to any patient suffering from Varicella or Herpes zoster during past 2-3 weeks. However, he was exposed to X-Rays chest for respiratory tract infection a week prior to the onset of herpes zoster. The patient told that he developed chickenpox at the age of about 5 years when he started his school. On examination, there was a grouped vesicular eruption over the distribution of left T7 dermatome. Systemic examination was unremarkable. Blood Complete Picture, Urine Routine Examination, X-Rays Chest, ECG, Blood Sugar, Stool Routine Examination & for occult blood, Ultrasound Abdomen and serology for Human Immunodeficiency Virus, Hepatitis B Virus and Hepatitis C Virus were all within normal range.

He was given acyclovir 800 mg 5 times daily for 7 days, analgesics, amitryptaline 25 mg orally at night, and a topical soothing lotion. He was advised to report back after every 5 days for follow up. His lesions healed in about two weeks time with minimal scarring and no post herpetic neuralgia.

CASE 2

19 years old son of the above reported patient also developed similar eruption within 24 hours after the onset of his father’s eruption. There was no history of any unusual physical or mental stress, trauma, radiotherapy, ill health, and exposure to any patient suffering from Varicella or Herpes zoster other than his father during past 2 weeks. He, according to his father, also suffered from chickenpox at the age of 1 year. On examination, he was found to have a grouped vesicular eruption over the distribution of right T8 dermatome. His eruption was slightly more extensive than that of his father; however, it was restricted to right T8 dermatome only. Blood Complete Picture, Urine Routine Examination, X-Rays Chest, ECG, Blood Sugar, Stool Routine Examination & for occult blood, Ultrasound Abdomen and serology for Human Immunodeficiency Virus, Hepatitis B Virus and Hepatitis C Virus were all within normal range. He was given acyclovir 800 mg 5 times a day for 7 days,
analgesics and a soothing topical application. His lesions healed completely within 15 days with minimal scarring. No post herpetic neuralgia or any complications were noted.

**DISCUSSION**

Herpes zoster is a common disease caused by Varicella Zoster virus. The virus causes varicella as a primary infection after which the virus persists in the cells of sensory ganglia. Reactivation of the latent residual virus occurs after a variable latent phase of between 5 and 40 years in 15% of the patients and results from waning specific cellular immunity. The possible precipitating factors include trivial trauma, physical and mental stress, radiotherapy and surgery. The precise incubation period of herpes zoster is unknown and probably depends on multiple factors like the status of cellular immunity, magnitude of provoking factors and extent of exposure to exogenous Varicella Zoster virus.

Zoster patients are infectious, both from virus in lesions and in some instances, the nasopharynx but the dose of virus is very low. In susceptible contacts of zoster, chicken pox can occur and the incubation period of chickenpox under such circumstances is 9 to 23 days which is more than the usual incubation period of 7-14 days. The possible reason is low viral dose in lesions of herpes zoster. Transmission of herpes zoster from a patient suffering from herpes zoster, although described, is very rare, again possibly due to low amount of virus in herpes zoster. The incubation period of herpes zoster under such circumstances depends upon the specific immune status of the patient for Varicella Zoster virus. If the contact has not previously been exposed to varicella, herpes zoster will result after the usual incubation period of 7-14 days. However, prolonged contact is required in this situation because of low viral contents of the lesions in herpes zoster. In case, the contact has previously been exposed to the virus, exposure to the exogenous Varicella Zoster virus can quickly trigger reactivation of the latent virus. The possible mechanism is blocking of cell mediated defenses by specific antibodies the levels of which rise following sub clinical/clinical re-infection. In this situation herpes zoster will develop after a much shorter interval after exposure. Simultaneous onset of herpes zoster in contacts of chickenpox can also be explained on the same basis.

This later mechanism was responsible for the simultaneous/early development of herpes zoster in our case No.2. Such simultaneous onset of herpes zoster in closely living family members has not been reported before.

**REFERENCES**