An unusual case of reactivation of herpes zoster after Guillain-Barré syndrome in an immunocompetent child

Lahmouad Asma, Mouaffak Youssef, Younous Said

ABSTRACT

Introduction: Herpes zoster has been widely described in the context of different systemic autoimmune diseases, but not in the Guillain-Barré syndrome. The Guillain-Barré syndrome that develops as a result of herpes zoster is rare; and when it does occur, it usually follows a cutaneous herpes zoster eruption. We present here a case of an acute inflammatory demyelinating polyradiculoneuropathy that occurred prior to the cutaneous herpes zoster eruption.

Case Report: A six-year-old healthy immunocompetent child with Guillain-Barré syndrome presenting with: acute tetraplegia, areflexia, and albuminocytologic dissociation in the cerebrospinal fluid. Electrophysiological studies indicated acute inflammatory demyelinating polyneuropathy (AIDP). Two doses of intravenous immunoglobulin (IVIG) were administered. Two months later, the patient had developed a typical herpes zoster eruption. He was treated symptomatically and had complete resolution of the lesions. To our knowledge, this is the first case report about herpes zoster complicating Guillain-Barré syndrome in an immunocompetent child which has not yet been found in literature.

Conclusion: Guillain-Barré syndrome may be an unrecognized risk factor for reactivation of a latent infection such as herpes zoster in children.
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Keywords: Children, Guillain-Barré syndrome, Herpes zoster, Immunocompetent

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INTRODUCTION

Herpes zoster (HZ) is a dermatomal viral infection characterized by painful vesicular lesions clustered along one and rarely two dermatomes. It is caused by reactivation of varicella zoster virus (VZV) that had persisted in latent form within sensory ganglion following an earlier attack of Varicella [1]. However, the pathomechanism is still not well understood. Reactivation of latent infection results from declining specific cell-mediated immunity [2, 3]. Herpes zoster is rare in healthy immunocompetent children of less than 10 years of age except for the infants who were infected in uterus, or in the first year of life [4]. Diminished cellular immunity seems to increase risk of reactivation because incidence increases with age and in immunocompromised states. Among the most frequent complications of HZ, one that stands out is post-herpetic neuralgia, and others not very frequent are: myelitis, diffuse encephalitis, cerebral
vasculitis and Guillain-Barré syndrome (GBS) [5]. Jiunn-Horng Kang et al., found that 0.03% of patients developed GBS in the two months following a herpes zoster attack [6]. Guillain-Barré syndrome is a rare and chronic disease manifesting as severe, generalized, flaccid paralysis and areflexia is typically caused by an autoimmune attack of peripheral nerve. The incidence of GBS is 1 to 2 per 100,000 annually worldwide [7]. However, Herpes zoster following Guillain-Barré syndrome is a rare and unusual association. This study reports a case of HZ associated with the Guillain-Barré syndrome (GBS), whose importance is based on its infrequent association and atypical, where peripheral neuropathy preceded the appearance of skin lesions.

CASE REPORT

We report the case of a six-year-old child who had no known co-morbidities and was up-to-date with immunizations but he was not vaccinated against varicella in which a cutaneous varicella zoster eruption developed two months after an acute inflammatory demyelinating polyradiculoneuropathy, fulfilling all the clinical and laboratory diagnostic criteria for Guillain–Barré syndrome developed. Past history of the child revealed that he had suffered from varicella at the age of one year. On admission, the patient had a rapid progression of muscle weakness and dyspnea beginning four days after a flu disease. Upon examination, the patient was hemodynamically stable but unresponsive, with flaccid quadriplegia and areflexia. He was admitted to the intensive care unit and required intubation and mechanical ventilation. Other physical examinations were normal. The brain magnetic resonance imaging was normal, and the electromyography (EMG) showed acute inflammatory demyelinating polyneuropathy (AIDP). Lumbar puncture indicated albuminocytologic dissociation and negative results of culture. Intravenous immunoglobulin (IVIG) was given for two days at a dosage of 1 g/kg/day. Our patient received an additional 1 g/kg of intravenous immunoglobulin a few days later, for duration of two days, in an attempt to ameliorate the final clinical outcome. His respiratory function slowly improved. After two weeks, he was able to breathe spontaneously during daytime, but during nighttime artificial ventilation had to be continued for two months. Three weeks after admission, oral intubation was changed to tracheotomy, which remained for eight months. Two months later, the patient had developed a typical herpes zoster eruption on the right side of trunk (Figure 1), the child was investigated: the complete blood tests were normal. Serologic study demonstrated anti-VZV IgG to be positive and HIV serology was negative. He was managed with analgesics and topical antibiotics and recovered completely. At that time, the patient was able to sit up in bed and on a chair.

DISCUSSION

Herpes zoster affects 10–30% of the population during their lifetime and its incidence and severity increase substantially with age and the presence of some chronic diseases [8–10]. The incidence of HZ per 1,000 person-years ranges between 1 and 5 [11, 12]. In pediatric population, the incidence is the lowest in the group 0–5 years of age (20 per 100,000 person-years) compared with adolescents (63 per 100,000 person-years) [11]. Herpes zoster is very rare in healthy children <10 years of age. Our case presented at an early age of six years and had a history of household exposure to varicella at an age of one year.

Reactivation of latent varicella zoster virus (herpes zoster) is a painful, cutaneous eruption, dermatomal in distribution, and is associated with the risk of dissemination. The VZV can interfere with adaptive immunity becomes latent in the dorsal root ganglia and reemerges when there is a weakening of the immune system [13, 14]. The cause and mechanism of reactivation of herpes zoster are not well-understood. Reactivation of HZ occurs with decreased cell-mediated immunity. This decline in immunity can result from increasing age, medical conditions and medications that suppress the immune system.

The incidence of herpes zoster is markedly increased in patients with malignancies such as lymphoma, with human immunodeficiency virus infection, or those taking drugs to suppress the immune system due to autoimmune diseases or organ transplants. Autoimmune disease, systemic lupus erythematosus (SLE) [15, 16],

![Figure 1: Herpes zoster eruption on the right side of trunk after healing: Vesicles turned to dry crusts on fifth day.](image-url)
rheumatoid arthritis (RA) [17, 18], psychological disease [19], and major depression [20] are also reported as risk factors for HZ. Other not less important risk factors have been identified recently: Diabetes mellitus (DM) [21] and patients with hypertension and congestive heart failure [22]. However, it is still unknown if the Guillain-Barré syndrome that might alter immune functions increases the risk of developing HZ. Guillain-Barré syndrome (GBS) is an acute immune-mediated disorder characterized by acute polyneuropathy; it is caused by an autoimmune reaction against peripheral nerve components [23]. It has been suggested that the GBS is associated with a perturbation of the circulating lymphoid cell population [24]. A perturbation of CD4 and/or CD8 lymphocytes in the circulation during the course of herpes zoster occurrence has been observed [25]. It therefore seems reasonable to suggest that both herpes zoster and GBS may be associated with an underlying common condition. Viral infections, such as herpes zoster (HZ), have been increasingly reported in individuals with systemic autoimmune diseases but not in the Guillain–Barré syndrome. Older age, Disease activity [26, 27], immunobiological therapies [28] and immune system dysregulation [29] are among the possible risk factors for HZ in the patients with systemic autoimmune diseases. Our case presented with herpes zoster that involved rash at thoracic dermatome, two months after the Guillain–Barré syndrome without significant well-known risk factors for HZ such as older age, use of corticosteroids and immunosuppressive agents, so we suggest that this disease activity and immune system dysregulation may have played a pivotal role for the development of HZ after GBS, although the pathomechanism is not clearly defined. Our patient was hospitalized for more than three months and he was received 80 g of intravenous immunoglobulin. we assume that the patients with Guillain-Barré syndrome may be at a greater risk of varicella zoster infection due to their impaired cellular immunity and depressed nutritional status or that IVIG might suppress the immune response against VZV and promote the reactivation of herpes zoster or because both herpes zoster and Guillain-Barré syndrome are reasonably common chronic diseases. However, the association between the two conditions may have been fortuitous. An episode of HZ in a person with Guillain-Barré syndrome could theoretically be a cause of decompensation or complication of this disease. Treatment of uncomplicated herpes zoster in the child with an antiviral agent may not always be necessary, although some experts treat it with oral acyclovir (20 mg/kg/dose) for 5 days to shorten the duration of the illness [4, 30]. Our case was also managed symptomatically with analgesics and topical antibiotics.

CONCLUSION

In this report, we describe an unusual case of reactivation of herpes zoster after Guillain-Barré syndrome in an immunocompetent child. Herpes zoster has not, to our knowledge been previously described as a complication of Guillain-Barré syndrome. This prompted us to report this unusual clinical entity.

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