Literature Review

Segmental Zoster Paresis: A Literature Review

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Disclaimer: There was no external funding in the preparation of this manuscript.

Conflict of interest: Each author certifies that he or she, or a member of his or her immediate family, has no commercial association (i.e., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted manuscript.

Manuscript received: 08-14-2020
Revised manuscript received: 10-21-2020
Accepted for publication: 10-29-2020

Free full manuscript: www.painphysicianjournal.com

Background: Herpes zoster is an acute infectious skin disease that is induced by the re-activation of the virus incubated in nerve ganglions following initial infection with varicella-zoster virus in childhood. Herpes zoster mainly affects sensory nerves, resulting in severe acute pain, which is also the most common reason for medical intervention in this patient group. The concurrent involvement of motor nerves could induce the symptoms of segmental zoster paresis, which is manifested by localized asymmetric myasthenia, whose range generally follows the distribution of myomere with skin rashes. Due to the low incidence and unspecific clinical manifestations, segmental zoster paresis has not been sufficiently recognized by clinicians, and can easily be misdiagnosed.

Objective: To summarize the previous studies on segmental zoster paresis and analyze the pathogeneses, diagnosis, and treatment of this disease, as well as stress the challenges in current treatment, which could provide useful evidence for the clinical diagnosis and better the treatment of patients with segmental zoster paresis in the future.

Study Design: We conducted a narrative review.

Setting: Hospitals, neurology departments, pain departments, and private practices.

Methods: We searched PubMed and Chinese CNKI libraries using the terms "herpes zoster," "muscle paresis," "segmental zoster paresis," and "motor nerve." Clinical trials, reviews, and case reports were collected and reviewed.

Results: As a rare complication following varicella-zoster virus infection, segmental zoster paresis has not been sufficiently recognized by clinicians, and there are still no guidelines available to guide the clinical treatments. The exact mechanism of segmental zoster paresis is still unclear. Electromyographic and magnetic resonance imaging examinations could be used as auxiliary diagnostic methods for segmental zoster paresis. Early regular anti-viral therapy could substantially decrease the risk of herpes zoster related complications. Combined application of glucocorticoids and some other physical therapy may also be useful in certain patients. The general prognosis of segmental zoster paresis is relatively good, with 67% patients achieving complete or almost complete recovery of the muscle function.

Limitations: More clinical trials are needed to clarify the exact mechanisms and best treating methods.

Conclusion: As the symptom in most segmental zoster paresis patients is self-limited, and the patients' prognosis is generally good, early diagnosis of the disease is especially important, due to the avoidance of unnecessary diagnostic procedures and incorrect treatments. Standard treatment guidelines regarding the functional rehabilitation are still needed for patients with refractory segmental zoster paresis.

Key words: Herpes zoster, postherpetic neuralgia, segmental zoster paresis, muscle strength, motor nerve, spinal dorsal ganglion, zoster infection, muscle paresis

Pain Physician 2021: 24:253-261

Herpes zoster is the specific symptom of skin lesion that is induced by the reactivation of varicella-zoster virus (VZV), which initially occurred in childhood. It tends to be accompanied by severe pain that is generally the major cause of hospital admission (1). Clinical recovery is achieved in most patients after the skin lesions of herpes zoster disappear; however, herpes related complications including postherpetic neuralgia (PHN), herpes zoster myelitis, meningitis, and segmental zoster paresis (SZP) can occur in some patients (2). SZP is the complication of the motor nerve following the viral infection, which is relatively rare in clinical practice. The manifestation of SZP mainly includes localized asymmetric myasthenia, with a range that is generally in agreement with the distribution of myomere with skin rashes.

Previous studies have reported that the infection of VZV accounts for 0.5% – 5% of all the motor impairments (3). Nevertheless, the exact odds related to this complication still remain unclear, which is mainly due to the following 2 factors: 1) the difficulties in clinical diagnosis (the differentiation from myasthenia induced by other causes, or the limb weakness masked by drastic pain); and 2) atypical clinical symptoms of segmental intercostal paresis and abdominal muscle paresis. However, electromyographic (EMG) studies have shown denervation potential in 71.4% of thoracic zoster patients with no clinical evidence of weakness (4). Therefore, the incidence of SZP may be underestimated (5,6). The low incidence and lack of specific clinical manifestations have led to difficulties in the clinical diagnosis of patients with SZP. Although the incidence of SZP is very low, the influences of this disease on patients are substantial and could increase the burden of medical service seeking. In A previous study reported on 2 patients with foot drop secondary to an infection of varicella-zoster who were incorrectly referred to an orthopedic clinic by their general practitioners. In addition, the influence of extremity muscle strength could also restrict the daily working and living activities of the patients. As the prognoses of patients with SZP are generally good, the definite early diagnosis and consequent appropriate treatments are necessary. Therefore, this review summarized the previous findings on herpes zoster and complicated SZP, which could help the identification and diagnosis for such patients in future clinical practices, thus eliminating unnecessary diagnostic processes and medical interventions (7), and promoting the patients' recovery.

Pathogeneses of SZP

VZV Infection and Complications

VZV is one of the 8 herpes viruses that are pathogenetic to human beings, while the most common transmission routes are the airborne transmission between humans and direct contact with infectious lesions (8). VZV can sit in dorsal root ganglion neurons after initial infection or vaccination. The initial infection of this virus could induce the generation of VZV-specific memory T cells, whose immunity gradually decreases with time (9). When the immunity decreases to the theocratical "threshold of herpes zoster," the risk of VZV infection correspondingly increases (8). Exogenous and endogenous enhancement could increase the immunological memory of VZV. The average time of persistent immunological period is about 20 years after the chickenpox infection. To date, the exact underlying mechanisms of VZV activation remain unclear. Still, any of the factors influencing the cell-mediated immunity could be relevant. For instance, previous studies have demonstrated that VZV could be reactivated by several factors including ageing (10), stress, low immunity (11), and application of immunosuppressors.

After reactivation, the incubated VZV could travel along sensory nerve fibers and induce damage to sensory nerves, then reversely travel to the corresponding innervated skin areas, leading to herpes zoster-specific cutaneous herpes, which mainly manifests as painful vesicular exanthema with involved unilateral side. If motor nerves are also concurrently involved, symptoms of SZP, including localized, asymmetric myasthenia, may occur, with the myasthenia generally ranging in agreement with the distribution of myomere with skin rashes. In clinical practice, the most commonly affected area is the face (namely, Ramsay-Hunt symptom), followed by limbs (12). The involvement of motor nerves generally occurs within 1 – 8 weeks after the appearance of skin lesions. However, symptoms of motor nerve involvement can also rarely appear before the occurrence of skin lesions in limbs (13). The mechanisms of clinical manifestation related to herpes zoster are illustrated in Figs. 1 and 2.

Pathogenesis of SZP

As a neurotropic virus, varicella zoster mainly invades and incubates in nerve cells of dorsal root ganglions or cerebral ganglions. Polymerase chain reaction (PCR) has shown that in patients with Ramsay-Hunt syndrome, VZV can be found in the facial nerve sheath,

geniculate ganglion, trigeminal ganglion, vestibular ganglion, spiral ganglion, stapes muscle, and even middle ear mucosa or cerebrospinal fluid (CSF). When VZV invades the geniculate ganglion, inflammation may occur in the trunk of the facial nerve, leading to edema of the surrounding tissues. Because the osseous facial nerve canal is narrow, the inflammatory edema of the facial nerve may lead to compression of the facial nerve and vessels, thus causing facial paralysis (14). In SZP patient with an affected dorsal ganglion, etiological studies have suggested that the anterior root might be the most common site affected by inflammation and degeneration. In addition, anterior horn cells, brachial plexus, lumbar plexus, and peripheral nerves could also be involved (15,16). Considering the relationship between the ranges of impaired motor functions and distribution of skin lesions related to herpes, it is a predominant opinion that the development of SZP is caused by the direct spread or indirect spread of VZV through CSF from dorsal root ganglion to spinal anterior horn cells, anterior root, and distal nerves, which consequently induce the motor nerve damage and functional changes (17).

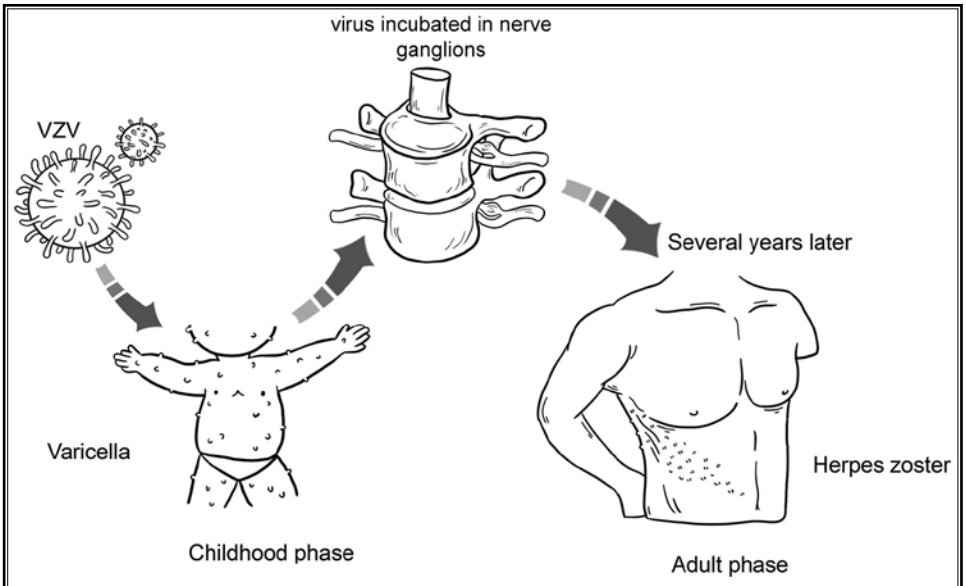

Fig. 1. *VZV infection and herpes zoster.*

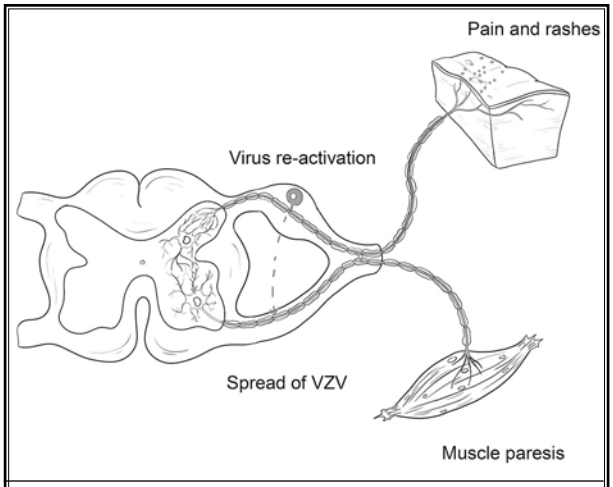

Fig. 2. *Possible mechanism of spinal motor nerve neuropathy.*

By using selective magnetic resonance imaging (MRI) for nerve roots, Yoshioka et al (18) have investigated a case of pain in the right C5 nerve innervated area accompanied by progressive myasthenia in the right shoulder following herpes zoster. The results showed enhancement in both the posterior and anterior roots of spinal nerves in the C5 segment, which was below the C4 vertebral body. Nevertheless, no enhancement was found in other segments. These findings suggested that inflammatory responses in motor nerves could be caused by the direct viral invasion of motor root spinal nerves or the indirect spread of the virus that invades dorsal root ganglion to anterior roots of spinal nerves. In a different study, MRI examination of a patient with residual motor weakness revealed glial scars in the spinal cord after the remission of multiple radiculitis that could possibly explain the segmental motor paresis in patients with incomplete remission of VZV infection (19).

In addition to the evidence provided by MRI scanning, electrophysiological examinations from previous studies have also provided evidence on motor nerve lesions induced by the virus. Liu et al (20). conducted electrophysiological examinations in 8 SZP patients with limbs involved, revealing the reduced amplitude of compound muscle action potential (CMAP) or sensory nerve action potential (SNAP) in all 8 patients. These

findings suggested that all these patients had damaged axons of both motor and sensory nerves. The findings were in agreement with the results reported by Sachs (21), who used EMG in following up an SZP patient with lower limbs involved for 22 months, revealing the complete denervation and delayed reinnervation of the muscles. In detail, the CMAP and motor unit action potential (MUAP) were both absent in the early stage of SZP. The time of the first record of CMAP was the tenth month after disease occurrence. Besides, the amplitude of CMAP gradually increased over time, which reflected the repairment following axon damages. In contrast, the time from the first record of MUAP was 8 months after disease occurrence, and the number of motor units gradually increased with time. According to the reparation speed following axon damages of motor nerves, the authors suggested that the site of the motor nerve damage in this patient with lower limb paresis could be the lumbar plexus or anterior roots of spinal nerves.

Although the spread of the virus from dorsal root ganglion to the cellular bodies or axons of motor neurons could explain the condition in most patients, there were cases with no herpes in the area of muscular paresis. Cioni et al (22) suggested that such cases could be caused by the direct invasion of VZV to anterior roots of spinal nerves or cellular bodies, while the virus might not be transported along the axons of sensory nerves. Therefore, there were no corresponding skin lesions in such patients.

Risk Factors of SZP

Advanced age is a definite influencing factor of SZP. The aging has a significant positive association with the incidence of SZP and the severity of electrophysiological abnormalities (23). Previous studies have shown that the average age of SZP occurrence is about 70 years. The other influencing factors, including gender (24), diabetes, rheumatoid arthritis (RA), and immunosuppression have also been suggested by several previous studies, but not confirmed (7,25).

Clinical Manifestations and Diagnosis of SZP

Clinical Characteristics of SZP

SZP is most commonly found in the head and face. The invasion of VZV to the geniculate ganglion of the facial nerve could lead to Ramsay-Hunt syndrome, which is manifested by drastic pain in the unilateral ear, as well as ear herpes accompanied with ipsilateral peripheral facial paresis (26), which accounts for about 80% of all the SZP induced by VZV. Meanwhile, Ramsay-Hunt syndrome is one of the most common causes of atraumatic facial paralysis (27). Peripheral facial paresis is manifested as the weakness of facial muscles, including the decreased ability to wrinkle the forehead, difficulty in closing the eye, and a drooping corner of the mouth. Due to the anastomoses between cranial nerves, a multitude of neurological disturbances such as hearing loss, vertigo, etc., might follow if the herpetic lesions progress (28).

Although the thoracic segment is the most frequently involved segment of the spinal nerve by herpes zoster (29,30), once the nerve roots of the upper thoracic segment get affected, the symptoms of segmental intercostal paresis are generally imperceptible. It has been reported that only 1% of such patients manifest relatively evident clinical manifestations (31). Yet, clinical manifestations, including abdominal muscle paresis and abdominal wall protrusion, can occur when the spinal nerves of the lower thoracic segment are affected. In a review of zoster abdominal muscle paresis, Chernev and Dado (7) retrieved 35 related studies, reporting on 36 cases of segmental zoster abdominal muscle paresis and found that the most frequently affected skin area was innervated by the T11 nerve, followed by the areas innervated by T12 and T10 nerves. The manifestation in some patients mimicked abdominal hernia (32,33), while asymmetric abdominal walls and disappearance of segmental abdominal wall reflexes were found in physical examinations. Yoo et al (34) reported on a case of abdominal muscle bulge following VZV infection, which was initially misdiagnosed as a hernia. Fortunately, no discontinuity of the abdominal wall was found in the consequent abdominal ultrasound examination. Some patients with abdominal SZP could also experience complications with other clinical manifestations of visceral nerve involvement, such as gastrointestinal dysfunction (such as constipation and intestinal pseudo-obstruction) (29,35).

Limb involvement by SZP is relatively rare. Liu et al (20) have conducted a retrospective study of zoster paresis of limbs. Their findings showed that among the 1,393 patients with herpes zoster, 8 had motor disturbance of limbs (incidence of 0.57%), where the upper limbs were affected in 6 patients, and the most frequently affected segments were C5-C7 nerves. The involvement of upper limbs is more common compared to lower limbs among SZP patients with motor disturbance of the limbs, while the L1-L4 nerves are the most

commonly affected spinal nerve segment in patients with motor disturbance of lower limbs (36), where the severity of limb weakness could range from slight to severe. The clinical manifestations may vary in relation to the muscles affected by SZP. Paresis of the ipsilateral shoulder or arm, which is usually underdiagnosed, may manifest as difficulty in elevating the affected arm or bending the forearm at the elbow joint (37). Lower limb neuropathy may manifest as leg and foot weakness; however, in some cases, it has been reported as foot drop caused by peroneal nerve mononeuropathy (38,39), which is most commonly seen in trauma. The motor disturbance of limbs could also be misdiagnosed as lumbar disc disease of corresponding segments (40,41). The involvement of C3-C5 motor nerves could lead to diaphragmatic paresis and induce the clinical manifestations, including dyspnea and pulmonary dysfunction (42,43). The affected ganglion segments and possible clinical manifestation are summarized in Table 1 (3,7,28,34,44-63).

Diagnosis and Auxiliary Examinations of SZP

The primary etiology of SZP is the infection of VZV, and herpes zoster is relatively easy to be diagnosed in the presence of the typical zoster skin lesions and other clinical manifestations. For patients without typical skin lesions but who are highly suspected of having VZV infection, several methods including polymerase chain reaction (PCR) (64), direct immunofluorescence assay (DFA), skin biopsy, and viral culture could help with the diagnosis. The specimens for PCR include blister fluid at the lesions, blood, plasma, CSF, and bronchoalveolar lavage fluid (BALF) (65). DFA could be used as a replacement for PCR since it has higher sensitivity but lower price than viral culture. For patients with SZP, the nerve segments innervating the herpes zoster related skin lesion, and the myasthenia could help the clinical diagnosis. Yet, the segment of muscle paresis and skin lesions could be inconsistent, which may increase the difficulties with clinical diagnosis (41,66).

EMG could help with accurate identification of the motor impairments induced by the infection of VZV, which has relatively high sensitivity (6,67), and thus could be used as an auxiliary examination method in clinical practice. When combined with electroneurography and fine-needle EMG, EMG examination could help to infer the types and sites of the nerve damages. Mondelli et al (68) conducted EMG in 158 patients with herpes zoster and found that one-third of the patients had motor nerve damages, half of whom with evident clinical manifestations, namely SZP. However, no evident clinical manifestations were found in the other half of the patients, who only showed electromyogram abnormalities (68). These findings demonstrated that EMG has relatively high sensitivity in diagnosing SZP. In addition to EMG, MRI examination also has relatively high sensitivity in detecting the involved nerves and muscles. Various previous studies have described the widening of the affected nerve and T2-weighed high-signals (63,69). The enhanced scanning through gadolinium (Gd)-contrast agents could also help to detect the inflammation levels of the involved nerve roots

Table 1. *Affected ganglion segment and possible clinical manifestation.*

Affected ganglion segment	Possible clinical manifestation	Reference
Geniculate ganglion of the facial nerve	Acute ear pain Herpes of external auditory canal Peripheral facial paralysis Hearing loss Vertigo Tinnitus	(28,44,45)
Cervical ganglion	Pain and rashes (neck, upper chest or upper limbs) Breathlessness (hemidiaphragmatic paralysis) Weakness of shoulder and/or arm muscles	(46-512)
Thoracic ganglion	Pain and rashes (thoracic and abdominal region) Abdominal pseudohernia Abdominal distention constipation Intestinal pseudo-obstruction	(7,33,52-58)
Lumbosacral ganglion	Pain and rashes (abdomen, low back, vulva and buttock, lower limbs) Leg and foot weakness foot drop urinary retention	(3,59-63)

(9). Also, MRI examination could also help with sensitive detection of muscle denervation, thus facilitating the speculation of involved nerves. The extracellular water contents of the denervated muscle cells increase in the acute phase; therefore, high bright signals can be found in the water-sensitive MRI sequences. Gupta et al (70) suggested that although the images of such muscles could also appear in some non-neurogenic diseases, such as inflammation and drug-induced rhabdomyolysis, only the muscles innervated by the affected nerves could be found in patients. In contrast, the muscle images in other disease conditions did not show the characteristics restricted in areas innervated by specific nerves. The MRI examination could also help rule out the nerve root compression induced by the protrusion of intervertebral disc.

The Treatment Principle of SZP

Treatment Target

For the complications induced by the infection of VZV, the treatment principles were based on the regular, appropriate anti-viral therapy, where symptomatic and supportive treatment should be conducted according to the corresponding clinical manifestations to improve the symptoms of the patients. The treatment methods include early anti-viral therapy, application of analgesic agents, protection of damaged muscles, and progressive rehabilitation exercises. Such evolutionary treatment could help to avoid muscle atrophy, edema, and contracture. Myasthenia and muscle atrophy can further induce the shift of affected joints, which should be closely monitored by clinicians during the treatment. Nevertheless, various guidelines for the treatment of herpes zoster fail to clearly recommend the standard treatment protocol for SZP.

Current States of SZP Treatment

A prospective clinical trial conducted by Mondelli et al (71) suggested that oral intake of acyclovir in the acute phase of herpes zoster could reduce the incidence of consequent motor nerve involvement. Various consequent clinical studies have confirmed the evident efficacies of acyclovir in patients with SZP (72). Acyclovir inhibits viral DNA replication and reduces nerve damage. Therefore, early application of acyclovir is of great significance in improving the prognosis of patients. The usual oral dose for adults with herpes zoster is 800 mg per time, 5 times a day for 7 days. Nevertheless, nowadays, acyclovir is least favored because it requires more frequent dosing because of lower bioavailability (71,72); thus, the use of other antivirals, such as valaciclovir and famciclovir may be more preferred (73,74). Currently, the predominant opinion is that regular anti-viral therapy within 72 hours after the appearance of skin rash could substantially shorten the pain time, as well as the risk of other related complications (75). Early anti-viral treatment after the appearance of skin rash is essential in improving the prognoses of herpes zoster patients (76). Early application of glucocorticoids can reduce edema and demyelination of the affected nerve, as well as prevent axonal degeneration. Application of glucocorticoids can also promote recovery of muscle paralysis. Prednisone tablets are commonly used and the oral dosage is 30 mg/day, taken in the morning for 7 days, and gradually reducing the dosage after 7 days. Kinishi et al (77) reported that early combination of acyclovir and prednisone may reduce damage to the facial nerve, enhance nerve excitability, and restore hearing. Some other researchers have also suggested that local, epidural, and sympathetic nerve blocks within the first 2 weeks after disease occurrence could reduce the pain severity and decrease the risk of complications (78). Combined application of glucocorticoids during nerve block could help alleviate the pain and accelerate the functional recovery of the patients. However, the application of glucocorticoids in patients with immunosuppression should be done very careful, and combined anti-viral agents should be administered. The drug therapy in some subgroups of patients, such as the ones with hypertension and diabetes, should also be very carefully administered. Researchers had also proved that acupuncture, which has a long history as a treatment of Traditional Chinese medicine, may also serve as an effective means for the treatment of Hunt syndrome, with few adverse events and low cost (79). Protection of the eyes is especially important in herpes zoster patients with Ramsay-Hunt syndrome, as peripheral facial palsy can impair the patient's normal ability to blink. As a result, the cornea can become dry, risking various injuries, including corneal ulceration. Therefore, all patients with peripheral facial palsy should be instructed to use artificial tears every hour when awake to keep the cornea moist, thus preventing any possible injuries. If patients cannot close one of their eyes, they should be instructed to manually close it at regular intervals to simulate blinking and to wear a patch at night after applying lubricant to keep the eye shut while sleeping (80).

For SZP patients with affected limbs, early func-

tional rehabilitation exercises should also be applied in addition to the above-mentioned drug therapies. Zhang (81) has reported the experience in treating 4 herpes zoster patients with motor nerve damage by early electrotherapy and rehabilitation massage, who fully recovered during follow-up. A pilot study assessed transcutaneous electrical stimulation in 10 patients with unresolved facial nerve paralysis, where below sensory threshold transcutaneous electrical stimulation was applied to all patients, and the duration of electrical stimulation was gradually increased to 6 hours/per day. The results showed a positive effect, and the authors suggested that transcutaneous electrical stimulation might serve as an effective treatment option for patients with chronic facial paresis with no other expectations of recovery (82). If the involvement of visceral nerves is suspected in SZP patients, nerve blocking through the intervertebral foramen should be conducted by using the mixture of glucocorticoids and local anesthetics, which could also help alleviate the symptoms (29).

Challenges in Current Treatments

The prognosis for SZP patients is generally good if the disease is correctly diagnosed and appropriately treated (83). Gupta et al (84) have reported that the percentage of complete or almost complete recovery of the muscle function is experienced by some 67% patients and partial recovery by 9%. The time of recovery varies among different individuals, while the average recovery time is about 1 – 2 years. Although the overall efficacy is good, the symptoms in some patients could persist over a longer period.

The number of patients with herpes zoster has been gradually increasing on a yearly basis with the aging of the population. The estimated number of Asians aged > 60 in 2015 was 489,397,421, which by 2035 will nearly double to 924,520,454. Assuming an annual incidence of herpes zoster in unvaccinated individuals of approximately 12/1000 person-years, this should equate to 30,395 new herpes zoster cases per day in 2035 (85). To date, various studies have investigated the susceptible population and risk factors related to herpes zoster; yet, the risk factors related to herpes zoster accompanied by SZP still remain unclear. Further identification of the susceptible population could favor the application of prophylactic treatments in clinical practice, thus reducing the incidence of SZP following the infection of VZV as much as possible. Guidelines and expert consensus on the recovery of limb motor function following herpes zoster are also needed to better guide clinical practices and promote the early functional rehabilitation in patients.

Summary

SZP is a rare complication that occurs following the infection of VZV that can lead to misdiagnosis and over-treatment in clinical practices. Increasing the clinician's understandings of SZP could help patients to undergo specific examinations and treatments, and therefore promote early functional rehabilitation. As a rare complication following VZV infection, SZP has not been sufficiently recognized by clinicians, and there are still no guidelines available to guide the clinical treatments. EMG and MRI examinations could be used as auxiliary diagnostic methods for SZP. As the disease in most SZP patients is self-limited, and the patients' prognosis is generally good, early diagnosis of the disease is especially important, due to the avoidance of unnecessary diagnostic procedures and incorrect treatments. In addition, SZP should be considered in the differential diagnosis of the acute painful motor weakness of limbs. To our knowledge, no standard treatment guidelines regarding the functional rehabilitation are available for patients with refractory SZP, who should be given more attention.

Acknowledgments

The authors thank Wenchen Jiang and Baosen Zheng at Tianjin First Center Hospital for the revision of the study and figure design. Lei Zhuang participated in revising the manuscript. Buwei Yu contributed to the design and revision of this article and made the final approval of the version to be submitted. All the authors have read and approved the final manuscript and declared no conflict.

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