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Motor paralysis of the lower extremities in herpes zoster

Review of Medical Writings and Report of a Case

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SINCE antiquity, the cutaneous and sensory manifestations of herpes zoster have been known. It was called zona (girdle) by the ancient Greeks. Herpes zoster and motor paralysis rarely occur simultaneously. Weakness of the lower extremities is particularly unusual in patients who have herpes zoster.

We recently examined a patient whose presenting symptoms were acute sciatica and pain in the left lower extremity, and with a typical herpetic rash on the left buttock. Subsequently, sensory loss and motor paralysis of the left gastrocnemius-soleus group developed, with absence of the reflex of the left ankle. This rare complication of herpes zoster prompted us to review the medical writings, and to present a report of the case.

Review of Medical Writings

Historical aspects. In 1866, Broadbent[1] published an account of motor involvement of the upper extremity, which he ascribed to herpes zoster. In 1876, Hardy[2] reported a case of motor involvement in the lower extremity. Since then there have been many reports of motor involvement of the trunk muscles,[3, 4] including the diaphragm[5] and the trigeminal and facial nerves.[6] Taterka and O'Sullivan,[7] in a review of reports of motor paralysis in association with herpes zoster, found that the upper extremity was affected in 45.5 percent of patients, the trunk in 40.9 percent, and the lower extremity in 13.6 percent. The incidence of motor involvement was three times more common in men than in women, and almost always occurred in middle-aged or older persons. In 1961, in a review of medical writings, Grant and Rowe[8] found that of 42 cases of paralysis of an extremity, which was caused by herpes zoster, in 30 the upper extremity was affected, in 12 the lower extremity. In 1961, Knox, Levy, and Simpson[9] reported three cases of quadriplegia associated with herpes zoster. Weseley and Barenfeld[10] reported two cases of lower extremity motor paresis due to herpes zoster, and noted pretibial pitting edema in both patients.

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Knox, Levy, and Simpson[9] classified herpes zoster with motor involvement into four main groups: (1) flaccid paresis localized to the region affected by the rash, and attributed to involvement of the anterior root as it passes the necrotic posterior root ganglion; (2) spastic weakness and sensory impairment due to involvement of the spinal tracts and possibly related to the demyelinating diseases; (3) spastic weakness of one or more limbs with occasional sensory loss and associated with disorders of consciousness and other signs indicating involvement of the brain substance; (4) widespread flaccid paresis and various degrees of sensory loss developing a short time after the herpetic rash and showing the features of peripheral neuritis or polyradiculitis.

Pathology. There is general agreement that herpes zoster is a disease due to a virus similar to the varicella virus. Herpes zoster can produce cranial or spinal sensory ganglionitis, posterior poliomyelitis, leptomeningitis and/or peripheral mononeuritis. Herpes zoster is characterized by a vesicular skin eruption, radicular neuralgia, and, less frequently, by segmental palsies and sensory loss.

In 1861, according to Head and Campbell,[11] Von Bärensprung's autopsy studies showed that herpes zoster was a disease of the nervous system, and demonstrated that the skin affected correlated with a dorsal root ganglion. In 1900, Head and Campbell[11] described an inflammatory reaction with round cell infiltration and hemorrhages in the posterior root ganglia and secondary changes in the posterior nerve root, spinal cord, peripheral nerves, and skin. They noted a similarity of the histopathologic changes in acute anterior poliomyelitis and in herpes zoster. The autopsy findings of Lhermitte and his co-workers[12, 13] revealed not only involvement of the spinal ganglia, posterior root, and peripheral nerves, but also involvement of the posterior horn, the gray substance of the anterior horn, and the anterolateral columns. Some observers believe that herpes zoster should be renamed acute posterior poliomyelitis or neuroganglioradiculomyelitis.

Adams,[14] and Denny-Brown, Adams, and Fitzgerald[15] have clarified the histologic changes that distinguish herpes zoster from other pathologic conditions. Adams[14] lists these changes:

> "First, a ganglionitis marked by necrosis of all or part of the ganglion, with or without hemorrhage and surrounded by an intense lymphocytic infiltration. This phenomenon is, in all published accounts and in our own cases, associated with the eruption of vesicles so characteristic of the disease in the corresponding cutaneous segment. Secondly, a poliomyelitis which closely resembles anterior poliomyelitis (infantile paralysis) but is readily distinguished by its unilaterality, segmental localization, and greater involvement of posterior horn, root, and spinal ganglion. Thirdly, a relatively mild localized leptomeningitis in which the cellular infiltrate is relatively

slight and limited principally to the involved spinal segments and nerve roots. Fourthly, a true peripheral mononeuritis seen not only in the nerves distal to the ganglion but also in the anterior root both within the meninges and in the portion contiguous to the involved spinal ganglia."

In 1883, Landouzy[16] suggested that herpes zoster might be an infection. Von Bokay[17] reported a case, seen by him in 1887, in which he noted the similarity of herpes zoster and varicella. Brain[18] and Amies[19] demonstrated by complement fixation technics that a cross relationship exists between the two viruses. Amies[19] noted that the agglutinins of varicella and of herpes zoster appeared simultaneously in the sera of patients with these diseases. This observation was confirmed by Weller and Coons.[20] Rake and associates[21] reported that varicella and herpes zoster viruses were indistinguishable from one another by electron microscopy, using the fluorescent antibody technic of Coons for their identification. In 1958, Weller and his associates,[22, 23] using tissue culture and also serologic methods, demonstrated the validity of the monistic theory concerning the herpes zoster and varicella viruses.

Knox, Levy, and Simpson[9] reported an isolated case of a patient with quadriplegia, zoster ophthalmica, and shingles of the ophthalmic division of the trigeminal nerve from which a type nine ECHO virus was recovered from the cerebrospinal fluid.

Clinical manifestations. The vesicular skin eruption of herpes zoster precedes motor paralysis in three quarters of the patients, and follows the paralysis in one quarter of the patients. The time lapse between eruption and onset of paralysis (or vice versa) ranges from one day to two months, but the two conditions never appear simultaneously.[7] Joffroy[24] reported one case in which the interval was seven months. When the eruption has been unilateral and the paralysis has been unilateral, both manifestations have been ipsilateral in all reported cases but one.[25] Electromyographic studies of herpes zoster have been compatible with those of peripheral neuropathy.[7]

Taterka and O'Sullivan[7] reported that 16 percent of 44 patients with motor involvement completely recovered within from 6 to 12 months; 81.7 percent did not fully recover, and one patient died of ascending myelitis. Grant and Rowe,[8] and Weseley and Barenfeld[10] reported a better prognosis, with complete recovery in five of a total of seven patients.

Treatment. Treatment of herpes zoster paresis has been largely supportive and symptomatic. Weseley and Barenfeld[10] used elevation, elastic stockings, and daily passive range of motion exercises to prevent contractures. Knox, Levy, and Simpson[9] suggested that steroids might be of some benefit, especially when used early in the course of the disease, but they believed that their series of patients was too small for general conclusions to be made.

Report of a Case

A 40-year-old Caucasian housewife was brought to the Cleveland Clinic Hospital emergency room on January 16, 1967, because of pain in the left side of the lower part of the back, hip, and leg. Ten days before admission to the hospital, she had experienced spontaneous onset of pain in the left lumbar region, with radiation to the left hip. Three days later, while lifting a heavy object, a radicular pain developed down the posterior aspect of the left thigh, the lateral portion of the left leg, and the ankle. The symptoms were worse with standing, coughing, or sneezing. The pain was described as burning in nature, with frequent waves of severe lancinating pain. There was no history of back trauma and no disturbance of bladder or bowel control.

Seven days before admission to the hospital, the patient noticed a rash on the left buttock. She had been given penicillin orally for acute tonsillitis earlier that same day, and thought that the rash might be an allergic reaction to penicillin.

Twenty-five days before admission to the hospital, the patient spent the day with a young nephew who that day was diagnosed as having chicken pox. The patient had contracted chicken pox in childhood. Except for having undergone appendectomy, rhinoplasty, and salpingectomies for two ectopic pregnancies, her medical history was not remarkable.

Examination revealed a well-developed, well-nourished Caucasian female, who appeared to be in acute pain. Motions, including jarring of the bed, elicited pain in the back and legs. There was a hyperesthetic, vesicular, herpetiform rash (*Fig. 1*) on the superior, posterolateral left gluteal region,

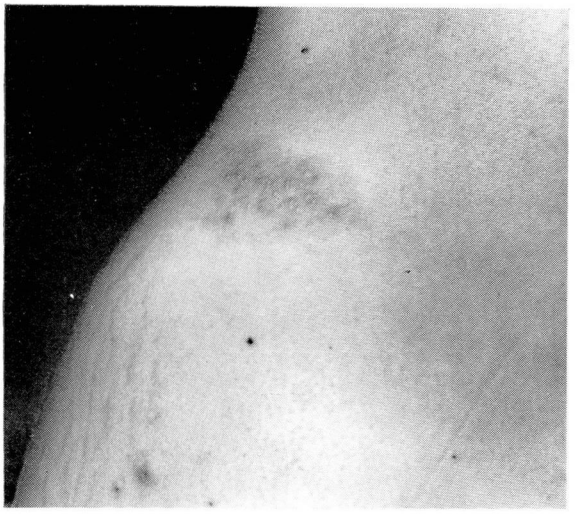

Fig. 1. Photograph of herpes zoster rash on left gluteal region.

10 by 25 cm, and affecting the first and second lumbar dermatomes of cutaneous innervation. There were no motor weakness of the lower extremities and no sensory deficit. The patient could not bear weight on the left foot because of pain. Left straight-leg raising was positive at 45 degrees. There was tenderness over the left sciatic notch, and pressure aggravated the radicular pain. Babinski's sign was absent. Deep tendon reflexes were active and equal.

The patient was treated with analgesics, bed rest, traction, intravenous injections of sodium iodide, and oxytetracycline and hydrocortisone ointment (topical to the rash). The patient could not tolerate either pelvic or bilateral Buck's traction. On the next day the pain was more severe, and there were numbness and hypesthesia of the lateral aspect of the left leg, ankle, and foot, and anesthesia of the lateral aspect of the left heel. The left ankle jerk was absent. There was weakness of plantar flexion of the left foot, with a motor power rating of 4 minus of the gastrocnemius-soleus muscle group.

Electromyography revealed normal motor action potentials. Nerve conduction studies were not performed because of inability of the patient to cooperate. Lumbar puncture revealed normal manometrics and normal cerebrospinal fluid. All laboratory data were within normal limits. Roentgenograms of the chest and spine were normal.

The patient was given intrathecal injections of methylprednisolone acetate on the third and on the sixth hospital days; lessening of pain occurred in less than 24 hr after the injections.

The patient gradually improved and she was discharged from the hospital after 12 days. She continued to have numbness of the lateral aspect of the left foot and ankle. The left ankle deep tendon reflex did not return. The plantar flexor muscles of the left foot remained weak for two months. Four months after admission to the hospital, the patient had a transitory recurrence of some herpetic type of vesicles on the left buttock, with exacerbation of radicular pain. In less than two weeks after intravenous injections of sodium iodide, the symptoms and rash regressed completely.

Nine months after the onset of illness, the patient had mild pain in the left thigh, with some numbness of the posterior part of the left calf and the lateral aspect of the left foot. The deep tendon reflex of the left ankle was absent, and there was hypesthesia of the lateral aspect of the left foot, with anesthesia of the lateral aspect of the left heel. Plantar flexions of both feet were of equal and normal strength.

Comment

Motor paralysis, in the extremities, caused by herpes zoster is rare; it is even rarer in the lower extremities. By our count, 52 cases of motor paralysis of the extremity, related to herpes zoster, have been reported in the last

101 years. In 20 cases, the lower extremities were affected. Eight of the latter cases were reported in the last seven years, indicating that the incidence may be greater than is generally recognized.

In regard to the patient we treated, the skin eruption was correlated with levels of the first and second lumbar dermatomes, whereas the sensory deficit was in the spinal level of the first sacral dermatome, and the motor deficit affected the first and second sacral dermatome levels. This disassociation is similar to the cases reported by Kendall,[25] who noted that in three of five patients the paralysis and rash affected different dermatomes. One of the patients he treated had a rash over the seventh and eighth cervical and first thoracic dermatomes on the left side, and motor loss of the third, fourth, fifth, and sixth cervical dermatomes on the right side, with complete anesthesia of the sixth and seventh cervical dermatomes. Ambercrombie[26] reported a case in which the patient had a herpetic rash of the third sacral dermatome, a sensory loss at the first and second sacral dermatome levels, with motor deficit of the fourth and fifth lumbar dermatomes. Knox, Levy, and Simpson[9] attributed this type of distribution to involvement of the spinal tracts, and postulated that this form of herpes zoster may be related to the demyelinating diseases. Taterka and O'Sullivan[7] noted that in 90 percent of 44 patients, the rash, paralysis, and sensory changes affected the same levels.

In this century, all patients with herpes zoster paralysis have been more than 50 years old, except one patient who was 45 years old. The patient we treated, who was 40 years old, is the youngest patient in reported cases in recent publications. Generally, the incidence of herpes zoster paralysis parallels the incidence of shingles.[8] The relationship of the viruses of herpes zoster and varicella has been discussed. The patient we treated had a definite history of contact with varicella 14 days before onset of symptoms of herpes zoster. This fact adds one additional bit of clinical evidence to the monistic concept of the two viruses. While the route of entry of the virus is a moot point, we noted that the patient had a sore throat in the early stages of the disease.

Recovery of motor function is complete—at least to a fully functional level—in an estimated 75 percent of patients.[7, 8] The patient we treated regained full motor strength within two months. Nine months after her episode she still had minimal pain and hypesthesia with a small area of anesthesia at the level of the first sacral dermatome. The ankle reflex has not returned.

Our use of intrathecal injections of methylprednisolone acetate dramatically relieved the patient's pain, but it did not appreciably improve the motor paralysis. It is interesting to speculate as to whether or not the temporary paralysis might have been prevented with the use of intrathecal

injections of steroids on the day of hospital admission rather than on the day after the onset of paresis.

SUMMARY

A case report of herpes zoster with motor paralysis affecting the left lower extremity of a 40-year-old Caucasian woman is presented. The motor function has returned to normal, but sensory changes and the lack of an ankle jerk have persisted. This is thought to be the twenty-first case of its kind to be reported, and the youngest patient in modern medical writings.

The history, pathology, etiology, clinical course, and treatment of herpes zoster associated with motor paralysis have been discussed. Intrathecal injections of steroids were beneficial in the symptomatic treatment of herpes zoster.

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