PARESIS FOLLOWING HERPES ZOSTER

A REPORT OF TWO CASES

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Herpes zoster is usually a benign type of infection mainly affecting the sensory part of the nervous system. Motor symptoms occur infrequently, although both upper and lower motor neurone palsies following typical zoster have been recorded. Upper motor neurone lesions were described by Besta and Vercelli (quoted by Rocchi, 1938), Bruce (1907), and Worster-Drought and McMenemey (1933). In each of these cases there seemed no other explanation for the pyramidal lesions, although Alajouanine and Griffith (1931) recorded an enlightening case in which herpes zoster followed by upper motor neurone paresis of the right leg occurred in a girl aged 17 who six years later developed typical disseminated sclerosis. Among the lower motor neurone palsies the one most often encountered is facial paralysis and occasional deafness complicating geniculate zoster. Examples of this syndrome were described by Ramsay Hunt (1907), and it is now known by his name. The next most frequently reported palsy appears to be oculomotor paresis complicating trigeminal zoster. Cases of this type have been described by Essen-Möller (1936), Federici (1937), Carmody (1937), and others.

Brachial and abdominal palsies are more uncommon: Kinnier Wilson (1940) describes a zoster of C4 segment with weakness of the deltoid and biceps (C5). He also mentioned that in any given segment concerned "either all or a few or only one muscle may be affected." Taylor (1895) recorded a case of right abdominal zoster with paralysis of the recti and obliques, and Rocchi (1938) a case of zoster affecting segments D12 and L1 on the left side with weakness of the corresponding obliques, which showed a partial reaction of degeneration. Similar cases have been described by Bloedorn and Roberts, Worster-Drought, Taylor and Chairon (quoted by Kinnier Wilson, 1940).

Lower motor neurone paralysis complicating lumbar or sacral zoster seems a rare condition. Hardy (1876) and Cornil (1930) each described one case—the former zoster of the sciatic nerve with paresis of the calf muscles, the latter zoster of the thigh with atrophy of the quadriceps and loss of the knee-jerk. Davidsohn and Oppenheim (quoted by Rocchi, 1938) observed in a sacral zoster transitory motor disturbances of the bladder and rectum.

Two personal cases of lumbar zoster, very similar in distribution and course, are described here, both complicated by lower motor neurone paresis involving the affected segments.

Case I

A widow aged 73 was admitted to the Central Middlesex Hospital on January 15, 1940. For several years she had had epigastric pain of a typical peptic ulcer pattern. Apart from this she had been well until an attack of shingles in the right leg two months previously. Since then she had been shaky, and complained of neuralgia in and weakness of this leg, which tended to give way on walking.

On examination the patient was found to be, for her age, a very well preserved woman with a slight tremor of the lips, tongue, and arms. The scars of a recent herpes zoster were present on the inner side of the right thigh and on the front

of the leg in the distribution of the third and fourth lumbar roots. Cutaneous sensation to pin-prick and cotton-wool was slightly diminished over this area; elsewhere it was normal. The right quadriceps was slightly wasted and its power and that of the abductors of the thigh diminished. All the tendon reflexes were brisk, with the exception of the right knee-jerk, which was absent. The plantar reflexes were flexor and the abdominals were absent. The arms, the cranial nerves, and the fundi oculi were normal. The peripheral arteries were hardened, but the heart and lungs were normal; blood pressure was 150/90, and the urine was normal. The occult blood reactions in the faeces were positive on two out of three examinations, and a barium meal showed evidence of a simple pyloric ulcer. A fractional test meal gave a normal result.

The cerebrospinal fluid on January 20 was under normal pressure and was clear and colourless. There were 21 cells per c.mm. present—1 polymorphonuclear leucocyte and 20 lymphocytes. The total protein was 0.035% and the Lange curve 0001221000. The Wassermann reaction was negative. On February 1 the fluid contained only 14 cells per c.mm.—1 polymorph and 13 lymphocytes. A radiograph of the spine showed osteo-arthritis and some osteoporosis of the thoracic and lumbar vertebrae.

The patient was given a course of faradism and exercises to the affected muscles. A gradual improvement in power took place, and before her discharge on February 21 the right knee-jerk was noted as just present; the neuralgia was less severe than on admission. In addition she had been put on a peptic ulcer regime with marked benefit.

She has been seen at monthly intervals since, and the cerebrospinal fluid was re-examined on April 22 and was found to contain only 1 lymphocyte per c.mm. The protein was 0.035% and the Lange curve negative. The slow improvement in power continued, and the knee-jerk was gradually increasing in briskness. When last seen on July 20, nine months after the onset of the eruption, she was looking extremely well. She had lost her dyspeptic symptoms and had gained 10 lb. The neuralgia was occurring occasionally at night only and had diminished in intensity. The right leg now appeared to her to be as strong as the left, but on examination slight weakness of the quadriceps remained and the right knee-jerk was not quite as brisk as the left.

Case II

A married woman aged 53 was admitted to Shenley Hospital on April 3, 1938, with a severe depressive reaction state and suicidal tendencies. She slowly improved mentally and was occupying herself fairly well when on September 17 she complained of severe stabbing pain in the front of the right thigh. Next day she had a temperature of 99.8° and developed a typical herpetic eruption on the front of the thigh, which spread to the inner side of the right leg the following day. The lesions approximated to the distribution of L3 and L4 spinal segments. On this day—September 19—lumbar puncture was performed, and clear fluid under normal pressure was obtained. This contained 38 lymphocytes per c.mm. and 0.05% of protein. Four days later her temperature had settled, and she complained that she could not lift her right leg from the bed. The herpetic eruption had begun to heal, although pain was persistent; some analgesia was found between the drying vesicles. Wasting of the quadriceps was observed (half an inch), with almost complete loss of power; the adductors were normal; no fibrillation was present. Tendon reflexes were normal except that the right knee-jerk could not be obtained. On September 26, eight days after the eruption, the cerebrospinal fluid showed 28 lymphocytes per c.mm., with 0.045% protein. There was slight weakness of the adductors and complete flaccid paralysis of the quadriceps; the knee-jerk was still absent, and wasting had increased to 1 inch in circumference of the thigh. There was no improvement in the paralysis until October 1, when she could just extend the knee and flex the thigh, and the cerebrospinal fluid now showed 16 lymphocytes per c.mm., with 0.03% protein. The vesicles had disappeared, but nocturnal neuralgia still troubled her, although it was controlled by analgesics. Movement gradually improved with massage and exercise, and she had regained about half her power by October 14, although the knee-jerk was still absent.

On October 20, nearly five weeks after the beginning of the illness, the cerebrospinal fluid contained 11 lymphocytes, with 0.03% protein. Gradual improvement followed, and on November 17 it was noted that the right knee-jerk was just present. On December 19 the last lumbar puncture was done and 8 lymphocytes with 0.032% protein were found. Wasting was persistent and she still complained of occasional twinges of pain in the thigh and leg. The knee-jerk had returned almost fully five months after the onset, and power was fair, although she still used a stick.

When last seen about a year after her illness she was symptomless, except that the right leg tired easily. Examination revealed half an inch of wasting in the right quadriceps, persistent scars of the eruption, and great improvement in her mental condition. The right knee-jerk seemed as brisk as the left.

Commentary

These two cases demonstrate an unusual complication of herpes zoster. Although no complement-fixation test was done there was no reason to doubt the diagnosis, as the clinical features and the course were typical. It was considered important to exclude any underlying condition which might have been responsible for the paralysis, since it is well recognized that zoster may occur in other diseases of the spinal cord, particularly in disseminated sclerosis, tabes dorsalis, and cases of cord compression. There were no clinical signs to suggest the presence of any such lesion in our cases, although in Case I the history of dyspepsia and a positive occult blood reaction in the faeces led us to exclude by radiograph spinal metastases from possible carcinoma of the stomach.

The pathology of herpes zoster is still somewhat obscure, as so few cases come to necropsy. Head and Campbell in 1900 in an exhaustive paper mentioned that Bärensprung in 1861 first showed that zoster was a disease of the nervous system associated with changes in the posterior root ganglia. They described their post-mortem findings in 21 cases in which death occurred at all stages of the disease from three days to over two years after the outbreak of the eruption, and concluded that the primary lesion was an acute round-cell infiltration with areas of haemorrhage in the affected posterior root ganglia, and that changes found in the posterior nerve roots and spinal cord were secondary degenerative processes. The fact that Price (1937) gives a similar account of the pathology of zoster without mentioning the existence of any spinal cord lesions suggests that Head and Campbell's ideas are still in the main accepted in this country.

This view has been challenged by the French school, notably by Jean Lhermitte and his collaborators, who in three communications reported acute inflammatory changes not only in the ganglia but also in the posterior roots, the meninges, and the cord itself. In the first of these papers Lhermitte and Nicolas (1924) described the post-mortem appearances of a patient dying of bronchopneumonia seven weeks after the onset of the herpetic eruption. They found perivascular infiltration of leucocytes and mononuclear cells with vascular dilatation and demyelination in the posterior horns of the affected segments. The anterior horns showed atrophic changes in the cells with demyelination of the nerve roots and some lymphocytic infiltration: in spite of this no paralysis was observed clinically. There were no changes in the white matter except for one area in the pyramidal tract in which perivascular "cuffing" was observed. It is of interest to note that the cerebrospinal fluid of this case showed a pleocytosis of 88 lymphocytes per c.mm. on the second day of the eruption. Similar observations were recorded in two subsequent papers (Faure-Beaulieu and Lhermitte, 1929; Lhermitte and Vermès, 1930). In the former, lesions spreading to neighbouring segments were found in the posterior horns.

Kinnier Wilson (1940) records similar findings by Nicolesco, Thomas and Lamière, and Schlesinger.

The view that the infection involves the meninges and the cord as well as the posterior root and its ganglion is supported by the frequent occurrence, according to Kinnier Wilson, of a pleocytosis in the cerebrospinal fluid. Both our cases were typical in this respect.

It would appear probable, therefore, that zoster is essentially an acute posterior poliomyelitis involving the whole of what we suggest may be called the "segmental sensory neurone." Although anatomically the root ganglia are placed outside the spinal cord, they consist of cells which have migrated out from the posterior horn, and thus their constant involvement is understandable. The finding of inflammatory changes in the anterior horn by Lhermitte and others is presumably due to the spread of the virus from the posterior horn, and would explain the occasional occurrence of motor lesions in a disease which primarily involves the sensory system.

Since so few cases have been described it is impossible to determine accurately the course and prognosis of paralysis complicating zoster. Our cases, in which identical segments were attacked, ran similar courses. The pleocytosis took many weeks to subside: Case I showed 14 lymphocytes three months after the onset, and Case II 8 cells nine weeks afterwards. When last seen, nine and twelve months respectively after the eruption, neither of these patients had fully returned to normal: Case I was complaining of nocturnal neuralgia in the leg and had a slightly diminished knee-jerk, and Case II showed slight wasting of the affected muscles. The crural paralyses in both Hardy's and Cornil's cases appear to have gradually improved, and in Rocchi's case the paralysis of the obliques had almost disappeared within a year. These cases show that though recovery is slow, satisfactory function can return within a year. The observation of further cases for longer periods would be necessary to determine whether this is the rule and whether complete recovery may not be possible.

Summary

Two cases of lower motor neurone paralysis complicating herpes zoster are described, and the pathology is discussed.

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A. Christie (*J. Pediat.*, 1940, **17**, 502) reports that a follow-up study of children to test the duration of Schick immunity was made with various antigenic materials with the following results. According to the type of material, doses given, and the period between injections and the Schick test, 77 to 95% of the group showed negative Schick reactions in four to twelve weeks. After a four to nine year follow-up period 46 to 88% of the known Schick-negative reactions were found to be still negative and 12 to 54% had become positive. Christie concludes that all children immunized in the first year of life should be Schick-tested again on admission to school, when those who are positive should be given a stimulating dose of toxoid.