Herpes Zoster with severe neurological complication— A report of two cases

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HERPES ZOSTER is a common condition whose skin manifestation is well known since antiquity. Among the Malaysian and Singaporean community of Chinese origin, it is nicknamed the "serpentine" illness. Knowledge of associated neurological paralysis is widespread and much feared. It is the general belief that neurological complications result only when there is bilateral herpetic eruptions on similar dermatomes.

Broadbent (1866) originally described motor paralysis of an arm which was preceeded by herpes zoster. Since then, many cases have been reported of motor involvement, by Waller (1885), Taylor (1896), Barham-Carter and Dunlop (1941), Taterka and O'Sullivan (1943), affecting muscles of the extremities and the trunk, including the diaphragm and those of the cranial nerves. Upper motor neuron lesions were described by Bruce (1907).

Von Barensprung (1861) quoted by Head and Campbell (1900), first established the affliction of dorsal root ganglion and related parts of the spinal nerves. The latter two, in post-mortem examinations of 21 patients, showed that the primary lesions consisted of round cell infiltration and haemorrhages in the posterior root ganglia with secondary changes in the posterior root, the spinal cord, peripheral nerves and skin. Subsequently Lhermite and Nicolas (1928) showed that the nervous system could be more widely affected.

According to Gupta, Helal and Kiely (1969) only one per cent of all cases of herpes zoster are referred to hospital. In reviewing 274 patients, they found 69 (25%) had cranial nerve palsies, 15 (5%) limb paralysis, two bladder and rectal paresis and one trunk paralysis. Neurological complications are therefore not uncommon. Fortunately severe ones are relatively rare and always arouse great interest. Described below are two such patients with extensive involvement of the nervous system.

F.A.J. U.H. R.N. 000838, a 71-year-old Chinese male, was admitted to the University Hospital on 9th August 1967, with a history of pain on the right side of the chest, eight weeks earlier. Three days after the onset of pain, vesicles appeared on the right lateral chest wall at the level of the nipple. Within two to three weeks, the vesicles gradually healed, leaving superficial scars. Four weeks after the onset of symptoms, he developed a weakness of the right leg, followed soon after by a weakness of the left leg. Two weeks later, he felt a mild weakness and numbness of the right hand and fingers. He was constipated for a week or so, which difficulty he relieved with laxatives. He had no disturbance of micturition.

Examination revealed a well-built man. The pupils were equal and reactive to light and accommodation. The fundi were normal. Other cranial nerves were intact. The jaw jerk was brisk. Power in the upper limbs was good and equal. The right upper limb was more clumsy than the left. No sensory impairment was demonstrated. The deep reflexes were intact. In the right lower limb, there was global weakness with an impaired motor power of grade 3. It was also spastic with increased reflexes. An extensor plantar response was present (Babinski's Sign positive). Petallar clonus was demonstrated. Vibration and position sense were impaired below the level of the fifth dorsal dermatome. Pain, temperature and light touch were preserved. On the left side, the power of the lower limb was mildly affected (Grade 4). It was less spastic than the right. Deep reflexes were brisk, but the plantar response was equivocal (Babinski's negative). Proprioceptive sensation was normal. Below the left dorsal seventh dermatome, pain and temperature sensations were impaired.

Over the right fifth dorsal dermatome, there were scars of healed herpetic lesions. This dermatome was hyperesthetic and paraesthetic too.

General examination revealed no other significant findings.

Investigations

The blood contained 12.8 gm% of haemoglobin, a total leucocyte count of 5000 per ul, with a normal differential count. The erythrocyte sedimentation rate was 12 mm in the first hour. The blood Kahn test was negative. A chest X-ray and radiological skeletal survey were normal. No lumbar puncture was done.

Progress

For a while, he experienced neuralgic pains over the healed herpetic lesions. This was easily relieved by mild analgesic. It completely disappeared on the 9th September, 1967. His constipation was relieved by senokot or ducolox. No disturbance of micturition was seen.

He was given physiotherapy. The power of the lower limbs gradually improved. By 28th August 1967, he was able to walk on his own without support, though he still had a slight weakness of the right leg on discharge from hospital. The reflexes were still brisk on the right and the extensor plantar response persisted. Proprioceptive sensations were normal, though superficial modalities of sensation were noticed to be impaired only up to the level of the left knee. He was last seen on 21st November 1967 with almost full recovery, walking well, with no evidence of sensory loss. The right extensor plantar response still persisted.

K.L. U.H. R.N. 127859, a 58-year-old married Indian male, was seen on 26.10.70, with a history of illness which started four weeks prior to admission. Fever and malaise were the initial symptoms. On the fifth day, vesicles were seen on the left side of the neck; the next day, painful vesicles also appeared on the right upper arm which rapidly spread to involve the whole forearm. A week later, he noticed small vesicles on the trunk, face and lower limbs. Three days prior to admission, he fell while going to the toilet. He was helped to bed. Subsequently he was unable to get up because of weakness on both lower limbs and the right upper limb. There was no disturbance of sphincters. He was not a known hypertensive or diabetic. He was a known asthmatic for 18 years.

On examination, he was obese, and mentally alert. Vesicles and pustules on the right upper extremity along the sixth and seventh cervical dermatomes were seen. Scattered over the rest of the body, a few small vesicles and pustules were seen on the trunk and lower limbs. His cranial nerves were intact. Both fundi were normal. The right upper extremity was very weak with wrist drop. No power was found in the small muscles of the hand. On the forearm, upper arm and shoulder girdle muscles, the power was grade 2 to 3. On the left upper extremity, the power was generally grade 4. On the lower limbs, the proximal muscles were more affected with a power of grade 2, whereas distally it was grade 4. Sensory impairment to all modalities of sensation was found below both knees. On the right upper limb, hypalgesia was confined to the sixth and seventh cervical dermatomes. Reflexes were absent in all the limbs. Plantar response was going down on both sides. No significant finding was found in the other systems. Investigations showed a haemoglobin of 16 gm/100 ml with a total white cell count of 9500 cells/ul. The differential count was normal. E.S.R. was 39 mm/hr. He had a total serum protein of 6.2 gm/100 ml, albumin of 3.3 gm/100ml, a globulin of 2.9 gm/100 ml, and an A:G ratio 1.14. Fasting blood sugar was 103 mg/100 ml. Blood urea was 124 mg./ml. Blood and C.S.F. Kahn test were negative. In the C.S.F. 220 lymphocytes/ul, sugar of 110 gm/100 ml, protein of 340 mg/100 ml and chloride of 115 meq/L were obtained. Smears for organisms and cryptococus were negative. Cultures from C.S.F. were negative. E.E.G. was mildly abnormal with delta and theta waves in both frontal and temporal regions. Chest, skull and pelvic X-rays were normal.

Electromyography and nerve conduction studies were performed, three weeks after onset of paralysis. Motor nerve conduction studies showed prolonged velocities affecting the right median and lateral popliteal nerves, and the left ulnar and median nerves. The electromyography showed evidence of fibrillation potentials affecting the muscles of the right limb. No spontaneous activity was seen in other muscles examined, which had reduced interference patterns on maximal contraction. These findings suggested a peripheral neuropathy.

After a week in hospital, he suddenly developed a lower motor neurone type of left facial palsy. Horner's syndrome was also noticed. Wasting of the small muscles of the right upper limbs was becoming noticeable. Gradually the patient improved, and with physiotherapy he began to walk. Movements returned to the fingers of his right hand. When last seen, almost 11 months later, he had returned to his job as a night watchman with little residual neurological deficit.

While in the ward, he was given tetracycline for the superimposed bacterial infections of the herpetic vesicles. Mild analgesics were prescribed for his pain.

Discussion

The latent period for paralysis following herpetic eruptions varies from one day to two months (Taterka and O'Sullivan, 1943). Rarely paralysis preceeds the rash. The latency period is stated to be related to the severity of the neurological complications, a short latent period of less than two weeks was followed by a severe illness while a long one from two weeks onwards leads to less serious complications (Knox, Levy and Simpson, 1961). In case one, the latency was four weeks whereas in case two onset of symptoms appeared less than two weeks.

Both patients were more than 55 years old. It has been noted that severe neurological complications tend to occur more in the elderly. Of the 45 cases reviewed by Gupta et al, all were over 40. The incidence is highest in those in the sixties and seventies.

In the second patient, herpes zoster preceeded a generalised eruption typical of chicken pox. In 1887, Von Bokay, cited by Fee and Evarts (1968) in which he noted the similarity of herpes zoster and varicella. Brain (1933) showed by complement-fixation test that the two viruses are closely related serologically. These two viruses cannot be distinguished from each other by electromicroscopy, (Rake and associates 1948) and by tissue cultures, (Weller and his associates, 1958).

Knox et al, in an analysis of 13 cases, three of which were their own, and the rest collected from the literature, broadly classified the paralytic complications into four main groups. Group 1 consist of those in which flaccid paralysis is localised to the region of the rash. Group 2 consist of those in which there is spastic weakness and sensory impairment with a definite upper level. Group 3 are those with spastic weakness of one or more limbs, associated with sensory involvement and symptoms of brain involvement. Finally the last group are those with widespread flaccid paresis and varying degrees of sensory disturbances following shortly after the eruptions and showing features of peripheral neuritis and polyradiculitis.

In the first patient, the clinical findings exhibited features similar to those of the second group. His neurological symptoms began on the side of the herpetic lesions. With the findings of ipsilateral weakness and impairment of proprioceptic sensation, plus a contralateral loss of superficial sensation, Brown-Sequard syndrome was diagnosed. K. Wilson (1940) reported one of these cases as having an incomplete Brown-Sequard syndrome, after shingles of the twelfth dorsal root, with a paresis of the left leg, a left extensor response and reduction of sensibility on the right leg. Kendal (1957) reported a similar case.

In the second patient, evidence of peripheral neuropathy of the Landry-Guillain-Barre syndrome was confirmed by nerve conduction studies and C.S.F. findings. In addition, the right upper limb muscles, where the site of the initial zoster was situated, were more severely affected as indicated by electromyography, suggesting involvement of the cervical plexus or the anterior horn cells of the cervical spinal cord over several segments. These findings fit in the final group of classification of Knox et al.

The motor paralysis of the second patient involved many more segments than the zoster on the right upper limb. According to the cases reported by Kendall and Ambercrombie (1941), motor and sensory disturbances and the rash may occur in different dermatomes, sometimes even on opposite sites of the rash. Four of the 42 cases reviewed by Taterka and associates were in different dermatomes.

The generally accepted explanation for these neurological complications is that the herpetic virus initiate an "allergic" process leading to demyelination such as is seen in the demyelination following chicken-pox and other exanthema (Miller and

Stanton, 1954). The maximal site of the lesion is at the level of the herpetic lesion.

McAlpine and associates (1959), Knox et al attribute similar varied neurological involvement to a form of demyelination of the long spinal tracts or the peripheral nerves.

Both the patients recovered with minimal residual signs. Gupta et al, in order to settle the controversy between the views held by Doucet (1906) who claimed that improvement was generally rapid and by Brain (1962) who stated that palsies were usually permanent, found that of 45 patients, 30 recovered satisfactorily and eight patients were left with permanent paralysis. Taterka et al found that 16 per cent of their patients recovered with no residual abnormalities. The process usually requires six months to a year. Both these two cases recovered almost completely over a period of one year.

Summary

Two cases of herpes zoster, with severe neurological complications, are described. A 71-year-old man with Brown-Sequard syndrome and the other, 58 years old, with Landry-Guillian-Barre syndrome and cervical motor paralysis, recovered almost completely. The pathogenesis and history of the neurological manifestations are briefly discussed.

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