J. Neurol. Neurosurg. Psychiat., 1961, **24**, 167.

HERPES ZOSTER AND THE LANDRY-GUILLAIN-BARRE SYNDROME

BY

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In 1900 Head and Campbell described the pathological features in 20 cases of herpes zoster. They showed that the main sites affected in this disease are the posterior root ganglia and the ganglia of certain cranial nerves. Associated paresis was originally described by Broadbent (1866) and Waller (1885) and Lhermitte emphasized that the nervous system may become more extensively involved (Lhermitte and Nicolas, 1924; Faure-Bealieu and Lhermitte, 1929; Lhermitte and Vermès, 1930). Cases associated with limb weakness may be broadly classified into four main groups: (1) Those in which there is flaccid paresis localized to the region affected by the rash, and attributed to involvement of the anterior root in its course past the necrotic posterior root ganglion (Denny-Brown, Adams, and Fitzgerald, 1944). They are well recognized and have recently been reviewed by Kendall (1957). (2) Those in which there is spastic weakness and sensory impairment with a definite upper level. These are due to involvement of the spinal tracts and may be related to the demyelinating diseases (Alajouanine and Bernard-Griffith, 1931; Gordon and Tucker, 1945; Kendall, 1957; McAlpine, Kuroiwa, Toyokura, and Araki, 1959). (3) Those in which there is spastic weakness of one or more limbs, occasionally with sensory disturbance and associated with disorders of consciousness and other signs pointing to involvement of the brain substance (Thalhimer, 1924; Gundersen, 1925; Faure-Beaulieu and Lhermitte, 1929; Lhermitte and Vermès, 1930; Schiff and Brain, 1930; Biggart and Fisher, 1938; and Gordon and Tucker, 1945). (4) Those with widespread flaccid paresis and varying degrees of sensory disturbance developing a short time after the rash and showing the features of peripheral neuritis or polyradiculitis.

Since only a few cases belonging to this last group have been described, we think it worth while reporting three which we have recently encountered in Edinburgh.

Case Reports

Case 1.—In June, 1959, J.N. a 69-year-old engineer-mechanic developed a crop of blisters over the medial aspects of the left forearm. He complained of constant gnawing pains in the affected region and, although the blisters crusted and disappeared over two weeks, this pain persisted. A month later, his left hand became numb, and he began to have difficulty in gripping, which progressed until he had to give up work one week later. In August he had a sudden attack of deafness in the right ear associated with tinnitus and vertigo. The whole episode lasted only about an hour. He was admitted for investigation on August 20, 1959.

On examination, the cranial nerves were intact and the fundi were normal. He had slight bilateral middle ear deafness, more marked on the right side. There was scarring in the distribution of the left first thoracic nerve. The skin of both hands was thick, shiny, and atrophic. There was wasting and fasciculation of the triceps and of the first dorsal interosseous muscles on both sides. On the left, the shoulder girdle muscles, the deltoid, and the long extensors of the wrist and of the fingers all showed wasting. The tendons of the long extensors of the fingers of the right hand were shortened to such an extent that passive closure of the fist was impossible.

Widespread weakness was noted in the upper limbs and although there was no wasting in the legs, both tibialis anterior muscles were weak. All tendon reflexes were greatly diminished; abdominal reflexes were present and the plantar responses were flexor. There was an area of diminution of sensation to pin prick and light touch over the scarred area and along the medial border of the foot. Proprioception was intact. The muscles were not unduly tender.

Investigations.—The blood contained 14.0 g. haemoglobin per 100 ml. (Sahli), 6,800 leucocytes per c.mm. and had a sedimentation rate of 5 mm. in the first hour (Westergren). The urine contained no protein or sugar. A chest radiograph was normal and the cervical spine radiograph showed minor degenerative changes. On August 21 the cerebrospinal fluid (C.S.F.) pressure was 150 mm. of water with free rise and fall on jugular compression with the neck in extension or flexion. The fluid was clear and colourless. It contained 45 mg.

TABLE I

ELECTROMYOGRAPHIC FINDINGS IN CASES 1, 2, AND 3

Case	Date	Muscle	Fasciculation	Fibrillation	Polyphasic Units	Maximum Voluntary Activity*
1	26.8.59	L. first dorsal interosseous	—	+	—	+
		R. tibialis anterior	—	+	—	++
	14.10.59	R. first dorsal interosseous	—	+	—	+
		R. tibialis anterior	—	+	—	+
2	13.11.59	L. first dorsal interosseous	++	++	+	++
		L. tibialis anterior	+	—	+	++
	19.1.60	L. first dorsal interosseous	+	—	+	+++
		L. tibialis anterior	+	—	+	+++
3	27.1.60	R. triceps	—	—	—	++

* +++ Normal interference pattern ++ Reduced interference pattern + Discrete units only — No voluntary activity

protein per 100 ml., 3 cells per c.mm., and gave a negative Lange curve.

Electromyography on August 26 showed changes suggestive of neuropathy (Table I).

Progress.—A week later, the weakness in the anterior tibial muscles on both sides had increased considerably and the left hamstrings were now affected. The weakness of the upper limbs was unchanged. The tendon reflexes were now absent. During the following week his condition remained static and he was discharged home at the end of August.

He was admitted for further assessment on October 6. Two weeks previously he had had another brief attack of vertigo and tinnitus, severe enough to force him to bed for the day. In addition, he complained of difficulty in walking and said that his feet 'came down with a bang'.

Examination revealed that weakness of all limbs had become slightly more marked. There was now wasting and fasciculation in the left tibialis anterior, and he had developed a 'high-stepping' gait. The tendon reflexes were still absent and the plantar responses were flexor.

Lumbar puncture was repeated on October 12. The cerebrospinal fluid contained no cells and 50 mg. protein per 100 ml. Lange and Wassermann reactions were negative. Electromyography on October 14 showed little change (Table I) and nerve conduction studies, using the method of Simpson (1956, 1958), confirmed the diagnosis of peripheral neuritis (Table II).

On October 16, Prednisolone was started. The initial dose of 60 mg. daily was reduced to 40 mg. daily on the 19th, and the patient was discharged to a convalescent home on October 22 on this maintenance dose. On November 1, the dose of steroid was further reduced to 30 mg. daily. Although he felt better, there was no objective change. He died suddenly while running for a bus on November 21, and no necropsy was performed.

Case 2.—A.B., aged 54, developed severe right-sided herpes zoster ophthalmicus about May 24, 1959. Because of corneal ulceration, he was admitted to the Ophthalmic Department, but was discharged after six days when his condition appeared to be settling. About six weeks later he developed gradual loss of power in both legs, and tingling in both feet. Within a few days the tingling spread to both hands which also became weak. His condition slowly deteriorated, so that by the time of admission on July 30 he was unable to sit up unaided.

On admission he was apyrexial. There was scarring and residual crusting of the skin over the distribution of the ophthalmic division of the fifth nerve and a small corneal opacity on the right side. Coarse, unsustained nystagmus was present when he looked to the left. There was marked generalized weakness of the limbs and trunk other than the muscles of respiration. The tendon and abdominal reflexes were absent. The plantar responses were flexor. Vibration sense was absent distal to the knees and elbows, but all other modalities of sensation

TABLE II

NERVE CONDUCTION STUDIES

Case	Date	Nerve	Stimulation at	Muscle*	Length of Nerve (cm.)	Latency of Response (in msec.)†
1	17.10.59	R. ulnar	Elbow	Abd. dig. min.	32	21
		R. ulnar	Wrist	Abd. dig. min.	8	10·5
2	13.11.59	L. ulnar	Elbow	First dors. interos.	35	14
		L. ulnar	Wrist	First dors. interos.	8	8·5
	19.1.60	L. ulnar	Elbow	First dors. interos.	37	15
		L. ulnar	Wrist	First dors. interos.	10	6·5
		R. ulnar	Elbow	First dors. interos.	35	15
		R. ulnar	Wrist	First dors. interos.	9	7·5
		L. median	Elbow	Abd. poll. brev.	30	12
		L. median	Wrist	Abd. poll. brev.	6	8·5
3	25.1.60	R. ulnar	Elbow	First dors. interos.	33	9·5
		R. ulnar	Wrist	First dors. interos.	8	5

*Belly-tendon surface electrodes.

†Normal latency for each nerve and muscle tabulated does not exceed 5·0 msec. and 12 msec., stimulating at wrist and elbow respectively.

were normal. The muscles were not unduly tender. Both hands and feet perspired profusely.

Investigations.—On July 31 the blood contained 13.3 g. haemoglobin per 100 ml. (oxyhaemoglobin method), 8,200 leucocytes per c.mm., and had a sedimentation rate of 10 mm. in the first hour (Westergren). There was no protein or sugar in the urine. Radiographs of the skull and chest were normal. Lumbar puncture showed that there was no block to C.S.F. flow with the neck flexed or extended. The C.S.F. was clear and colourless, and was sterile on culture. It contained 3 lymphocytes per c.mm., 97 mg. protein, and 67 mg. sugar per 100 ml. The Wassermann reaction was negative and the Lange curve was 122220000 in the cerebrospinal fluid. Serial changes in C.S.F. constitution are plotted in Fig. 1. The fluid

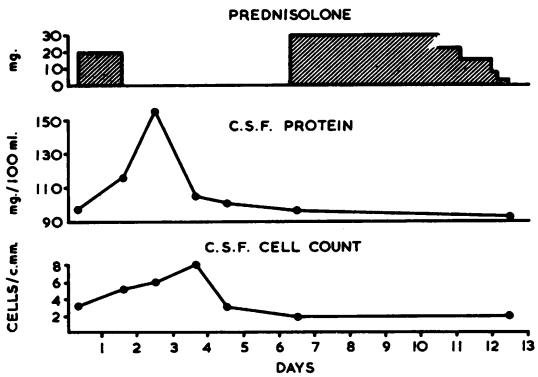

FIG. 1.—Cerebrospinal fluid protein and cell count and associated steroid dosage in Case 2.

was cultured on monkey kidney tissue. A virus was grown which was finally identified as being of the ECHO group, type 9. No virus was isolated from his stools.

Electromyography and nerve conduction studies on November 13 showed changes which were compatible with a diagnosis of polyneuritis (Tables I and II).

Treatment and Progress.—Despite Prednisolone (20 mg. daily starting on July 3), his condition continued to deteriorate slowly and muscular weakness increased. The steriod was, therefore, stopped after five days and high potency Parentrovite was given intramuscularly, but this also had no appreciable effect. He then developed a right pulmonary embolus from phlebothrombosis of leg veins and this was treated with anticoagulants and penicillin. On September 19 his neurological condition was unchanged. There was generalized wasting of muscles and fasciculation in the forearms, the hands, and the calves. A second course of Prednisolone (30 mg. daily) was started. Thereafter his condition improved, so that within four weeks he was able to walk unaided. He was finally discharged home at the end of October. When he was seen again on January 19, 1960, there was only slight residual weakness in the extensors of the wrists on both sides. The ankle jerks were absent and the other tendon reflexes were elicited with difficulty.

Electromyography and nerve conduction studies were repeated. These showed considerable improvement (Tables I and II).

Case 3.—Mrs. E. McC., a widow aged 65, had rheumatic mitral incompetence with associated atrial fibrillation. On January 27, 1958, she developed herpes zoster affecting the eleventh and twelfth thoracic segments on the left. This condition settled gradually, but on March 12 she noticed constant pain in the region of the left hip. The pain persisted, being worse at night, and a week later she suddenly developed weakness in the left leg. Next day she felt pins and needles in both hands, and the following day in both feet. By this time she was unable to walk because of weakness in both legs. A few days later she became incontinent of urine.

On examination on March 25 she was apyrexial. The cranial nerves were intact. There was widespread muscular weakness involving all limbs with pain on movement. The trunk and respiratory muscles were not affected. The tendon and abdominal reflexes were absent. The right plantar response was extensor. Exteroceptive sensation was impaired over the dorsum of the left foot and the finger tips of both hands. Vibration sense was absent in both legs. The muscles were not unduly tender.

Investigations.—The blood contained 13.7 g. haemoglobin per 100 ml., 7,600 leucocytes per c.mm., and had a sedimentation rate of 9 mm. in the first hour (Westergren). The urine contained a few pus cells and a trace of protein but no sugar. Radiography showed slight cardiac enlargement. Cerebrospinal fluid obtained by lumbar puncture (with normal manometric response) was sterile on culture. It contained 4 lymphocytes per c.mm., 162 mg. protein, and 69 mg. sugar per 100 ml. The Wassermann reaction was negative.

Treatment and Progress.—Prednisolone was given in a dose of 30 mg. daily starting on March 27, but the condition progressed and two days after the drug was started, she developed a partial paralysis of the right third cranial nerve with ptosis and slight diplopia. Thereafter she made a slow but almost complete recovery, and was discharged home on June 5.

A repeat lumbar puncture on October 21 gave normal C.S.F. containing 47 mg. protein per 100 ml.

Seventeen months later she was re-admitted with left hemiplegia, which was attributed to cerebral embolism. Neurological examination at this time revealed no sign of her previous illness apart from residual weakness of the right triceps.

She came to our notice at this time and electromyography and nerve conduction studies were carried out on January 25, 1960. The findings were considered to be within normal limits, but compatible with recent polyneuritis (Tables I and II).

Review of Cases Reported

Polyradiculopathy following herpes zoster was first described by Wohlwill (1924). This case, however, was by no means clear-cut as the patient had a positive Wassermann reaction and post-mortem

TABLE III

CLINICAL FEATURES AND CEREBROSPINAL FLUID FINDINGS IN THE CASES COLLECTED FROM THE LITERATURE TOGETHER THOSE IN OUR OWN CASES

Author	Sex	Age	Site of Eruption	Latent Period	Site of Paresis	Sensory* Disturbance	Cranial Nerve Involvement	Other Features	C.S.F.		Onset	Cou Ou
									Cells (per c.mm.)	Protein (mg./100 ml.)		
Wohlwill (1924)	F	44	R. Th.5	Two weeks	Four limbs	Yes	No	Wassermann reaction +ve	'Normal'†	'Raised'†	Acute	Death i
Schuback (1930)	F	62	R. Th.3	Seven days	Four limbs	?	No	—	?†	?†	Acute	Death i
Riser and Sol (1933)	M	30	L. Th.3-5	Two months	Four limbs	Yes	No	Swelling and redness of fingers, urinary symptoms	'Normal'†	1,750	Insidious	Recove areflex
Gilpin *et al.* (1936)	M	52	R. Trig. (first d.)	'Few days'	Bilaterally face and four limbs	Yes	Bilaterally seventh	Dysarthria	'Normal'†	'Raised'†	Subacute	Death month
Maggi *et al.* (1956)	M	68	L. Th.4	One month	Four limbs	Yes	R. eighth	—	'Normal'†	'Normal'†	Insidious	Partial four n.
Friart and Jeanty (1956)	F	72	L. L.5	?	Both legs	Yes	No	Incoordination and stupor, bowel and bladder symptoms, postural hypotension	25 max.	3,300 max.	Subacute	Recove month
Stammler and Struck (1958)	F	66	L.3	Three days	Four limbs	Yes	Bulbar	—	430	5,000	Acute	Death tive he
Pálffy and Balázs (1958)	F	63	R. Th.6-7	Two months	Four limbs	Yes	No	Unsteady legs	9	146	Insidious	Recove month
Pálffy and Balázs (1958)	F	53	L. Th.11-12	Seven days	Four limbs	Yes	Bulbar	—	14	24	Acute	Death i
Duperrat and Pringuet (1958)	M	53	R. Th.2-3	Two days	Four limbs	Normal	R. seventh	—	3·8	480	Acute	Death i
Present Series												
J.N.	M	69	L. Th.1	Two weeks	Four limbs	Yes	No	Atrophic skin and contractures	3	50	Insidious	Death, unrelat months
A.B.	M	54	R. V. first div.	Seven weeks	Four limbs	Yes	No	Profuse sweating of feet, ECHO 9 virus in C.S.F.	8	89-150	Subacute	Recover months
E.McC.	F	65	L. Th.11-12	Seven weeks	Four limbs	Yes	R. Third	Urinary incontinence, extensor L. plantar response	4	162	Acute	Partial r in three

*Other than those localized to site of eruption. †No values published.

evidence of syphilitic cardiovascular disease. Since that time nine further cases have been reported. The main features of these and of our own are shown in Table III. It will be noted that most cases occurred after the age of 50 years. This is probably merely an expression of the higher incidence of herpes zoster in the middle-aged and elderly. Both sexes were equally affected.

After the appearance of the eruption, there was a latent period varying from a few days to two months. This was followed by the neurological complications which progressed over a matter of hours (Wohlwill, 1924; Schuback, 1930), or, more gradually over a few days. The length of the latent period appears to be a good index of the severity of the disease: a short latent period (up to two weeks) was followed by a severe and fatal illness, while a long latent period (two weeks to two months) was followed by a mild illness. With one exception (Friart and Jeanty, 1956), all patients had paresis of the four limbs. Taterka and O'Sullivan (1943) and Halpern and Covner (1949) have drawn attention to a similar latent period for paralysis in the more common type where this is of limited distribution and usually anatomically related to the sensory disturbance.

For many of the cases, the nature and extent of the sensory involvement have not been recorded in detail so that it is impossible to give a comprehensive analysis. However, it would appear that they were very variable. In some cases they were purely subjective, consisting of paraesthesiae in the affected area; in others, objective disturbances were demonstrated ranging from mild hypoaesthesia to severe anaesthesia.

Cranial nerve involvement was seen in five cases. Gilpin, Moersch, and Kernohan (1936) and Duperrat and Pringuet (1958) reported bilateral facial palsy, Maggi, Meeroff, Cosen, and Hirschman (1956) unilateral nerve deafness, and Pálffy and Balázs (1958) severe bulbar symptoms. Our own Case 3 had a partial right third nerve palsy. In some instances there were changes suggestive of autonomic involvement. Thus, swelling and redness of the fingers was observed by Riser and Sol (1933), Friart and Jeanty (1956) recorded severe postural hypotension, and profuse sweating of the hands

and feet was noted in our second case. Case 1 developed atrophic skin changes and shortening of the extensor tendons of the fingers, an appearance rather similar to that seen in scleroderma.

The C.S.F. changes were variable. Usually, but not invariably, the protein was raised with a normal or slightly elevated cell count. The fluctuation in the C.S.F. protein in our second case is shown in Fig. 1.

Of the 13 patients reviewed, seven died, six within two weeks of the onset of the neurological complication. It seems likely that in our Case 1, death, which occurred five months after the onset of the condition, was due to cardiac failure. Those patients who recovered, did so after a period of months and were left with diminished or absent tendon reflexes and minimal weakness. With our limited experience we are not able to draw any conclusions about the efficacy of steroid therapy.

Discussion

The clinical picture presented by these cases can only be adequately accounted for by widespread disease of peripheral nerves or of spinal nerve roots, either alone or in combination. The electromyographic findings, including the demonstration of slowed conduction velocity of motor nerves, supports this view (Simpson, 1958).

The term "Landry-Guillain-Barré syndrome" has limitations which we fully appreciate, but it is a convenient one for the present purpose. Haymaker and Kernohan (1949) reported 50 cases of the Landry-Guillain-Barré syndrome confirmed by necropsy. They showed that many of the classical features of the condition may not appear and pointed out the extreme variability of the C.S.F. changes and the fact that the protein level may rise only very late or not at all.

The appearance of those neurological complications following herpes zoster might be accounted for in different ways. The two conditions could have been entirely unrelated. Secondly, the herpes zoster could have been "symptomatic" in a polyradiculopathy of unknown origin as suggested by Friart and Jeanty (1956) and by Pálffy and Balázs (1958). The sequence of events, the zoster always occurring first, makes both of these possibilities unlikely. The zoster virus could have "activated" a second hypothetical virus. This suggestion was made by Friart and Jeanty (1956) only to be dismissed. "Activation" of one virus by another is an unproven concept, indeed, the reverse, virus "interference", is biologically more probable. The isolation of the virus ECHO 9 from the C.S.F. from one of our cases and of an ECHO 6 virus from the C.S.F. and stools of a patient with the Guillain-Barré syndrome by Parker, Wilt, Dawson, and Stackiw (1960) may be relevant to this point. There is, however, no evidence that these viruses play a significant role in the motor complications of herpes zoster or in the Landry-Guillain-Barré syndrome.

The most likely explanation is that the causal agent in this condition is the zoster virus and that it produces the picture we have described either by spreading within the nervous system or, more likely, by initiating an "allergic" process such as that which is believed to occur in the Landry-Guillain-Barré syndrome and in the demyelinating diseases complicating chickenpox and other exanthemata (Miller and Stanton, 1954). This hypothesis would harmonize best with the long latent period to which we have drawn attention and which also characterizes the rare herpes zoster encephalitis.

Whatever the pathogenesis, it is possible that the cases reviewed here form part of a spectrum at one end of which there are the cases of polyradiculoneuropathy with no cord involvement, at the other cases with severe encephalomyelopathy (McAlpine *et al.*, 1959), our own patient described as Case 3, who had an extensor plantar response, falling into an intermediate group. If our hypothesis is correct the use of steroid drugs in treatment of the motor complications of herpes zoster is rational but it is probable that therapy must be started earlier if it is to be valuable.

Summary

Three cases are presented in which herpes zoster was followed, after a varying interval, by widespread lower motor neurone paresis associated with sensory disturbances and, in two of the three cases, albumino-cytological dissociation in the cerebrospinal fluid. In one case, ECHO virus type 9 was isolated from the C.S.F. Electromyography and nerve conductivity studies indicated peripheral neuropathy.

Ten similar cases have been traced in the literature, and the findings in all 13 cases are reviewed.

The pathogenesis is discussed.

We wish to thank Dr. Murray-Lyon and Dr. Chalmers Davidson for allowing us to study cases under their care.

Expenses were met by grants from the Secretary of State for Scotland on the recommendation of the Advisory Committee on Medical Research and the Scottish Hospitals Endowment Research Trust.

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THE FEBRUARY (1961) ISSUE

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The Effect of Ageing upon Physiological Tremor. John Marshall

Quadriceps Myopathy Occurring in Middle Age. J. W. Aldren Turner and K. W. G. Heathfield

Origin of Echoes in the Echoencephalogram. Brita Lithander

Cerebral Trauma in Infancy and Intellectual Defect. R. C. Oldfield and Moyra Williams

Observations on Selective Leucotomy. Walpole Lewin

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The Electroencephalogram in Congenital Hypothyroidism: A Study of 10 Cases. Eric A. Nieman

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