(1969) also studied alopecia areata and thyroid disease and confirmed Muller and Winkelmann's findings but were unable to show an increased prevalence of thyroglobulin antibody in comparison to Dingle *et al.* (1966) in a survey of a general practice population in northern England.

On the other hand, Kern *et al.* (1973) looked for additional evidence of autoimmunity in 40 cases of alopecia areata and showed a significantly raised prevalence of antibodies against thyroglobulin, parietal cells, adrenal cells, and thyroid cells. They could not, however, find antibodies against cells of hair follicles. Munro and Chanarin (1974) have confirmed an increased prevalence of intrinsic factor and parietal cell antibodies in female patients with alopecia areata compared with a control group.

Benda (1949) noted a history of thyroid disease in 38·5% of mothers of mongols. Other workers (Kurland *et al.*, 1957; Coppen and Cowie, 1960; Hayles *et al.*, 1965; Fialkow, 1966) have confirmed this and shown mongols themselves to be particularly susceptible to thyroid disease. Recently thyroid antibodies have been shown to be unusually common in mongols and their relatives (Mellon *et al.*, 1963; Fialkow *et al.*, 1965; Saxena and Pryles, 1965; Burgio *et al.*, 1966; Dallaire and Flynn, 1967; Vanhaelst *et al.*, 1970). Similar findings occur in two other chromosomal abnormalities—namely, Klinefelter and Turner's syndromes (Sparkes and Motulsky, 1963; Williams *et al.*, 1964).

In our series 8 (35%) of the female mongols with alopecia areata had thyroid antibodies compared with only 2 (9%) of a group of age-matched female mongols without alopecia areata. Fialkow (1970) found that 34% of 106 mongols in an institution had thyroid antibodies. He did not specify whether any of them had alopecia areata. Jacobs *et al.* (1969) measured antibodies to thyroid cytoplasm, parietal cells, and intrinsic factor in a randomly selected normal population in Wales. They found thyroid antibodies in 12·2% of the female subjects under the age of 55 and in 17·8% of those over this age. Doniach *et al.* (1963), in a survey of 100 normal female subjects selected at random, found antibodies in 13%. Our findings in female mongols with alopecia areata differ significantly ($P < 0.01$) from both those series, whereas the results in the female mongols without alopecia areata do not.

Mongolism is a chromosomal disorder and yet thyroid autoantibodies are often found in the condition. Alopecia areata occurs commonly in autoimmune disorders. We have shown that alopecia areata occurs more frequently in mongols than would be expected by chance, which suggests that there is a link between autoimmunity and chromosomal abnormalities.

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MEDICAL MEMORANDA

Herpes Simplex Virus and Guillain-Barré Polyradiculitis

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Herpes simplex virus (H.S.V.) is a well known aetiological agent in various disorders of neurological interest—for example, acute necrotizing encephalitis, which is often fatal (Overgaard *et al.*, 1971; Johnson *et al.*, 1972), and encephalitis with a more benign course (Buhl, 1974). Mild aseptic meningitis may in some cases be caused by an infection with H.S.V. type 2, in which case it is related to genital herpes infections (Terni *et al.*, 1971; Nahmias and Roizman, 1973).

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In Guillain-Barré polyradiculitis no single aetiological agent has been found though the disorder has been shown to be associated with several herpesvirus infections—namely, herpes zoster (Dayan *et al.*, 1972), Epstein-Barr virus infection (Grose and Feorino, 1972), and cytomegalovirus infection (Klemola *et al.*, 1967).

H.S.V., however, is rarely associated with radicular disorders of the nervous system and we have been able to find only one verified case of polyradiculitis in the literature in which H.S.V. infection preceded the acute neurological symptoms (Melnick and Flewett, 1964). We have had the opportunity of observing a further case in which such a correlation seems to have occurred.

Case Report

The patient, a 62-year-old woman, had no family history of neurological or other diseases. As a child and at the age of 40 she was treated in hospital for respiratory difficulties and bronchitis. She had never had shingles or ulcers in or near the mouth, nose, or genitals.

The present illness started on 6 November 1973 with exhaustion, universal dedolations of the muscles, and slight headache, and for two days she had a fever reaching 39°C. A few days previously she had been in close contact with a grandchild who had "coldsores" around the nose. All her symptoms disappeared on 8 November.

On 9 November she fell ill again, this time with severe, sharp, twingeing pains in the trunk and extremities, and was treated with tetracycline. Next day she experienced numbness of the soles and palms and weakness of the legs. The pains disappeared on 12 November but the symmetrical weakness of the extremities gradually progressed and on the 13th she noticed drooping of the left upper eyelid and complained of double vision. She was admitted to a local hospital and immediately transferred to our department.

On admission she was found to be afebrile but tired though mentally alert. There was left-sided ptosis, slight bilateral facial weakness affecting all branches, and a moderate, hypotonic symmetrical weakness of the extremities with absent tendon reflexes but normal plantar responses. Distal hypalgesia was present in the upper and lower extremities in a glove and stocking distribution. Respiration was normal and there was no cyanosis.

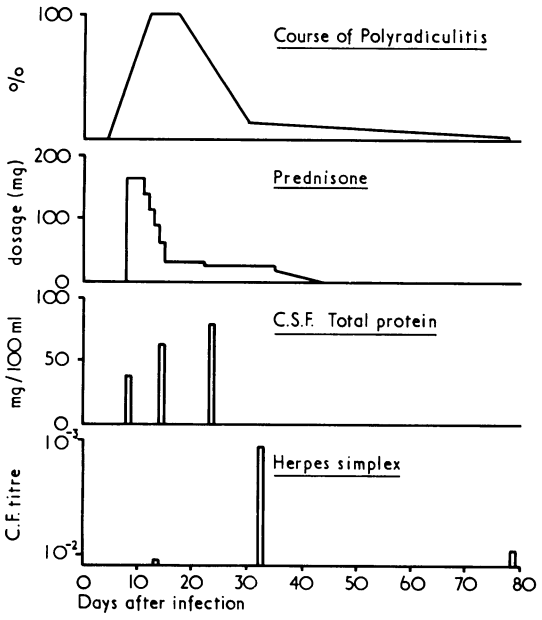
From 14 November steroid treatment was given, initially with dexamethasone and then with prednisone in gradually decreasing doses. She also received physiotherapy.

The weakness of the extremities gradually increased, reaching a maximum within a week. She then developed severe pareses over the hips, knees, and ankle joints and could not lift her extended legs from the bed; furthermore, there was slight weakness of shoulder abduction and extension of the elbows and wrists and there was universal areflexia. The sensory disturbances had become limited to vague hypalgesia of the fingertips. The facial weakness had disappeared and there was only slight left-sided ptosis. Lumbar puncture on 14, 20, and 28 November showed a rising total protein in the C.S.F. of 30, 60, and 72 mg/100 ml, the cell count being consistently normal.

After the neurological state had been stationary for five or six days a gradual improvement occurred, and when transferred to the local hospital for further physiotherapy she was able to walk with the support of one person. When discharged from that hospital on 21 December she had only slight diffuse weakness of the upper extremities and a moderate weakness of dorsiflection over the ankle joints.

When last seen in the outpatient department on 23 January the patient had no subjective complaints. There was a very slight weakness of flexion of the fingers of the right hand and slight trophic changes of the fingers. Power in the lower extremities was normal and she was able to walk on heels and toes. There were no sensory disturbances but the tendon reflexes were still absent.

In the course of this patient's illness, as in all cases of suspected neurological infections, serological tests were done for influenza, mumps, adenovirus, and herpes simplex virus. Widal and Paul-Bunnell reactions and a test for Weil's disease were carried out. The only positive result was in the herpes simplex complement fixation (C.F.) test with the following titres: 19 November, 30; 7 December, 960; and 23 January, 120. The chart shows the pertinent clinical values.



Course of clinical state (expressed as percentage of maximum paresis value) and laboratory findings.

Comment

The diagnosis of Guillain-Barré polyradiculitis was based on the progressive symmetrical sensorimotor disturbances and the characteristic C.S.F. finding of an increase in total protein without an increase in cells. The possibility of the concomitant occurrence of an encephalitis and a polyradiculitis was excluded as there were at no time any symptoms or signs of parenchymal cerebral involvement, and the peripheral cranial nerve involvement is a well known associated feature in some cases of polyradiculitis. The polyradiculitis could not have been caused by the steroid therapy because the peripheral nerve disorder occurred five or six days before steroid treatment was initiated.

Though the full pathogenesis of polyradiculitis is not known an allergic/immunological reaction seems probable, and this is widely accepted (Melnick and Flewett, 1964; Ravn, 1967). It is well known that the immunological reaction is frequently preceded by infections with widely different aetiological agents. Melnick and Flewett (1964) found a single case with H.S.V. infection preceding polyradiculitis and believed it was a reactivation of a latent infection provoked by a common cold.

H.S.V. infection as a cause of radiculitis has been reported in a few cases (Gayral, 1953; Nahmias and Roizman, 1973). They were all lumbosacral root affections with severe sphincter disturbances and pareses of the lower extremities and all occurred in association with genital herpes eruptions. Furthermore, they presented not only with an increase in C.S.F. protein but also with raised leucocyte counts. Thus it is likely that the pathogenesis is different from that of the Guillain-Barre syndrome, possibly involving a direct extension to the lumbosacral roots from the genital herpes infection.

We find it most likely that in our case a primary H.S.V. infection had been present, the strongest argument for this being the great rise in H.S.V. C.F. titre. The symptoms of infectious disease could easily be interpreted as a primary H.S.V. infection (Juel-Jensen and MacCallum, 1972; Nahmias and Roizman, 1973).

Cross-reactions against varicella-zoster virus, which is known to occur (Ross *et al.*, 1965), were not likely in our case as the patient had never suffered from herpes zoster.

Though there may be reservations about the presumed connexion between the polyradiculitis and the herpes virus infection in our patient we find the probability so great that in cases of polyradiculitis we advise repeated determination of H.S.V. C.F. titres during the course of the disease, examination for mucocutaneous manifestations of H.S.V. infection, and inquiry about possible previous infections.

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