EPIDEMIC ENCEPHALITIS WITH MYELITIS *

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The spinal cord has been little implicated in epidemic encephalitis and I know of no case except the one I now report in which intense inflammation was found as low as the lumbar region. The case reported in this paper is interesting in its symptoms suggesting brain tumor; in its pathology presenting a myelo-encephalitis; and in the resemblance of its lesions to those of multiple sclerosis, affording support for the theory of the inflammatory origin of the latter disease.

Nuclear stains in this case yielded different findings from those of multiple sclerosis in the intense perivascular infiltration and the presence of many fatty granular or gitter cells in the tissues, and yet it seems reasonable to suppose that had death been delayed a year or more the structure of these sclerotic areas would have resembled that of the areas in multiple sclerosis. The Weigert hematoxylin stain gives findings closely resembling the sclerotic patches of multiple sclerosis, as shown by the illustrations. If we accept the view that fatty granular cells are derived from glia cells and cells in the walls of the blood vessels, as Schröder¹ maintains, we may expect these cells to assume again more the appearance of glia cells in lesions of long duration.

The spinal cord has been found to be implicated in epidemic encephalitis by Harbitz and by Winkelman and Weisenburg.² In the case reported by the latter the lesions in the cord were not intense, they were similar in character to those in the brain, and the spinal cord below the middle or lower thoracic region was entirely normal. There were no sclerotic patches.

REPORT OF CASE

History.—I. R. B., a girl, aged 15 years, was admitted to the University Hospital, April 20, 1921. About Feb. 1, 1921, the father noticed that she walked with her head farther forward than usual. She vomited a few times about the first of March and complained of dizziness and headache. The vomiting usually occurred a short time after eating. She complained of the dizziness and headache all the time she was standing. She began to stagger a little in walking, early in March, and appeared as though she might be intoxicated. Later

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1. Schröder: Berl. klin. Wchnschr. **54**:714, 809, 1917.

2. Winkelman, N. W., and Weisenburg, T. H.: A Case of Myoclonic Lethargic Encephalitis with Unusual Pathologic Manifestations, Phila. Neurol. Soc. Trans., Arch. Neurol. & Psychiat. **5**:224 (Feb.) 1921.

in March it became necessary for her to hold on to objects in walking. Her father noticed that she was becoming dull during the latter part of February, 1921. She did not notice things as she had formerly, and she did not play. She did not seem to understand immediately when spoken to. She did her work as usual, but when she had nothing to do she sat listless, and this languor persisted.

She was taken to St. Luke's Hospital, Bethlehem, and it is not surprising that a diagnosis of cerebellar tumor was made. She began to have a little difficulty in talking in April; she knew what she wanted to say but had difficulty in forming the words. This difficulty rapidly increased. She began to complain of numbness of the right upper and lower limbs early in April, also of numbness of the tongue and some difficulty in swallowing. Her father thought her whole right side had become weak, but this paralysis is uncertain. She had had no convulsions. She still had slight headache when she came to the

Fig. 1.—Weigert medullary sheath stain of a section of the pons. Sclerotic patches are sharply defined on one side of the raphé, while on the other side the degeneration is more diffuse.

University Hospital. Constipation had become marked. She had no dyspnea or palpitation. She had difficulty in passing the urine, and at times had to be catheterized. Menstruation was regular up to the time she came to the hospital. A severe attack of scarlet fever had left her deaf in the left ear.

Examination.—When she entered the University Hospital she was lethargic; she answered when spoken to but became tired after answering a few questions, and did not notice what was going on about her. Her articulation was poor. Respiration was normal. The tongue was protruded poorly and deviated to the left. There was little movement of the eyes laterally, but some move-

ment upward with nystagmus, and better downward movement. There probably was bilateral ptosis.

Biceps tendon reflexes were not obtained and the triceps tendon reflexes were slight. The left patellar reflex was moderately exaggerated, and the right was somewhat diminished. Both Achilles reflexes were exaggerated, and bilateral ankle clonus was obtained. In the Babinski reflex there was upward movement of the toes on each side.

The handgrasp was weak on each side. The patient was unable to straighten her fingers, and they were held slightly flexed.

Dr. B. F. Baer found the optic disks sharply marked and the eye-grounds negative.

Notes obtained from Dr. Francis J. Dever, Bethlehem, stated that she had been admitted to St. Luke's Hospital in April, 1921, with nausea, vomiting and headache. The blood Wassermann reaction was negative. An examination of

Fig. 2.—Section through the medulla oblongata.

the spinal fluid revealed 3 cells per cubic millimeter. The Wassermann test of the fluid was negative, and Fehling's solution was reduced. The right internal rectus muscle was paralyzed and the external rectus was weak. Similar muscle changes were obtained in the left eye. The only fundus change was slight pallor of the temporal side of the disks.

Dr. A. B. Hamilton had found partial ptosis of each upper lid, slight nystagmus on looking to the left, marked nystagmus on looking to the right, and marked nystagmus on looking upward. The left ear was completely dead. Biceps and triceps reflexes were slightly exaggerated, and marked dysmetria was present on each side in the finger to nose test. Diadokokinesia was normal. The upper and lower limbs were not believed to be paralyzed. The gait was of the cerebellar type, and Romberg's sign was marked. Tactile and pain sensations were normal.

On April 24 her mentality was too feeble to permit careful examination, but it was possible to determine that the left lower facial nerve supply was completely paralyzed, and that the upper supply was weak, though she could close the left eyelids. She understood a simple command, such as to show the teeth, and performed this movement several times. She gradually became worse and died May 5, 1921.

The important symptoms in this case were: headache, vertigo, vomiting, mental impairment and lethargy, numbness and possibly weakness of the right limbs, nystagmus, dysmetria, incoordination of gait and station, exaggeration of the tendon reflexes of the lower limbs, the Babinski sign, paralysis of the left seventh nerve and ocular palsies.

A diagnosis of cerebellar tumor was not unreasonable, and certain symptoms suggested multiple sclerosis; but the case appeared to me to be one of epidemic encephalitis, and microscopic study of the tissues proved that this diagnosis was correct.

Fig. 3.—Section about the first thoracic segment.

Microscopic Examination.—Sections from the frontal and occipital lobes and the paracentral lobule showed nothing abnormal. The inflammation was of moderate intensity in the basal ganglions, but it was intense in the cerebral peduncles. Here some sclerotic patches were found by the Weigert hematoxylin stain, but they were not so numerous as in the pons and medulla oblongata. The cerebral peduncles were the highest level at which these patches were seen. The characteristic lesions of epidemic encephalitis were pronounced in the cerebral peduncles—the perivascular infiltration of lymphocytes and plasma cells and the diffusion of these cells throughout the tissues, especially in the gray matter below the aqueduct of Sylvius. Occasionally an area was found containing many spaces from which the nerve fibers had disappeared, resembling the areas so common in severe anemia of the spinal cord; in some of these the cellular infiltration was only of moderate intensity.

The pons showed numerous sclerotic patches by the Weigert hematoxylin stain (Fig. 1), resembling closely those of multiple sclerosis as presented by this stain. I have sections from cases of multiple sclerosis stained by this method which would be hard to distinguish from these of epidemic encephalitis. These areas were in places sharply defined like those considered so characteristic of multiple sclerosis. I have found nothing like them in any other case of epidemic encephalitis except a single area in the cerebral peduncles in one case, nor have I seen such areas described in the literature. With thionin or a nuclear stain like Delafield's hematoxylin they showed intense cellular infiltration, consisting of lymphocytes, plasma cells, glia cells and large globular cells with a small nucleus (fatty granular cells or gutter cells), differing from the areas of acute or subacute multiple sclerosis but resembling them. The characteristic lesions of epidemic encephalitis were intense in the pons and medulla oblongata. I have not seen more intense perivascular cellular infiltration in any case of this disease. The medulla oblongata was affected as severely and in the same way as the pons (Fig. 2). The cerebellum showed nothing striking.

Fig. 4.—Section from the lumbar region showing sharply defined sclerotic areas.

The lower cervical and upper thoracic region (Fig. 3) presented marked sclerotic patches, and they consisted largely of proliferated neuroglia. There was moderate cellular infiltration in these areas but not so intense as in the lumbar region. The degeneration was not confined to any system of fibers, as would be the case in secondary degeneration.

The midthoracic region appeared to be normal, except that in the lateral columns there were one or two areas of very loose neuroglia tissue. This was interesting in view of the great implication of the cervical and lumbar regions, but axis cylinders escaped degeneration, at least partly. It is possible that the spinal inflammation was not of sufficient duration for the development of secondary degeneration.

The midlumbar region (Fig. 4) showed a condition similar to that of the cervical region, but here there was a pronounced cellular infiltration of the gray matter of much the same intensity as in the pons and medulla oblongata, remarkable in its occurrence so low in the cord. I have not seen so grave an implication of the lumbar region in epidemic encephalitis.