A CASE OF JAPANESE B ENCEPHALITIS

BY

J. H. HALE, M.D.,
Captain P. C. FARRANT
Royal Army Medical Corps

and

Captain D. EDWARDS, M.R.C.P.
Royal Army Medical Corps

(From the Department of Bacteriology, the University of Malaya, and the Command Laboratory of the Far East Land Forces.)

The following case is reported as it may be of interest to R.A.M.C. officers who might at some time serve in Malaya.

CLINICAL FINDINGS

A private soldier aged 19 was admitted to the British Military Hospital, Kluang, on 26th January, 1952. He had a two days’ history of headache; there was no vomiting, but on the day prior to admission he had been unconscious for a period of twenty minutes. At the time of admission the temperature was 99°F. and the cerebro-spinal fluid (C.S.F.), which was sterile, contained 22 cells per c.cm., 71 per cent. of which were polymorphs and 29 per cent. lymphocytes.

27.1.52. The patient was drowsy and showed a tendency to reversal of sleep rhythm. The temperature was over 102°F., and at this time there was some neck rigidity: otherwise examination of the central nervous system was negative. At 1600 hours he had an epileptiform convolution. The C.S.F. was slightly turbid; the protein was 60 mg. per cent., and cells 155 per c.cm. Of these 95 per cent. were polymorphs and 5 per cent. lymphocytes.

28.1.52. At this stage he was comatose and incontinent. There was a slight stiffness of the neck and later in the day he had a second epileptiform convolution and developed athetoid movements of the right arm. The C.S.F. contained 114 cells per c.cm., but the cell picture had changed, for now 69 per cent. were lymphocytes and only 31 per cent. polymorphs. In the evening he had passed into a deep coma and the temperature was 104°F. 750 micrograms of chloramphenicol were given intrathecally.

29.1.52. The coma was lighter and the patient showed photophobia. The cell count of the C.S.F. had risen to 970 per c.cm., 91 per cent. of which were lymphocytes and 9 per cent. polymorphs. A further 750 micrograms of chloramphenicol were given intrathecally, but later there was a sudden onset of dyspnoea and respiratory embarrassment and the patient died in the early hours of the following day.
A Case of Japanese B Encephalitis

Pathological Findings

Apart from the brain, the other organs showed congestion and evidence of toxic changes. Both hemispheres of the brain were slightly hyperæmic, while the brain stem and pons showed frank congestion.

Histology.—Sections made from the pre-central, post-central and hippocampal gyri and the thalamus all showed perivascular lymphocytic cuffing and scattered foci of neuronal degeneration. Associated with these areas there were accumulations of neuroglial cells and lymphocytes (Fig. 1), and in some neuronophagia was also present. The cerebellum showed slight inconstant perivascular cuffing and degeneration of the Purkinje cells. The changes in the spinal cord were similar to those found in poliomyelitis. There was cellular infiltration and some neuronal degeneration (Fig. 2).

<table>
<thead>
<tr>
<th>Virus</th>
<th>Mice 3 weeks old</th>
<th>Guinea Pigs 250 gms.</th>
<th>Rabbits</th>
<th>Monkeys</th>
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<tbody>
<tr>
<td>Western Equine</td>
<td>I.C.</td>
<td>I.P.</td>
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<td>Venezuela Equine</td>
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<td>Lymphocytic choriomeningitis</td>
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<td>Theiler's Virus</td>
<td>+</td>
<td>0</td>
<td>++++</td>
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<td>Japanese B</td>
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<td>0</td>
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<td>Virus isolated from Military Case</td>
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I.C. = intracerebral inoculation. I.P. = intraperitoneal inoculation. Table taken from Diagnostic Procedures for Virus and Rickettsial Diseases.

Laboratory Investigations

A post-mortem was conducted within two hours of death and portions of the brain and the spinal cord were placed in a container refrigerated with dry ice for transport to Singapore.

The material was ground up with sterile sand in nutrient broth to give a 20 per cent. w/v suspension. After centrifuging, 0.03 ml. of the clear supernatant fluid was inoculated intracerebrally into three-weeks'-old Swiss mice. Within four to five days infected mice showed convulsions, were ataxic, and some were paralysed in the hind limbs. These were sacrificed and 1 per cent. suspensions of their brains passaged into other mice. By such means the infection could be maintained indefinitely, and a bacteriologically sterile filtrate of such a suspension prepared by the use of 0.69 μ gradacol membrane filter (Elford, 1931) resulted in an infection of the mice and thus the virus etiology was established.
**FIG. 1**

Section of Spinal Cord showing Infiltration and Commencing Degeneration of Nerves

**FIG. 2**

Section of Human Cortex showing Lymphocytic Perivascular Cuffing and Cellular Infiltration
At this stage the species range of infectivity of the virus suspension was tested and the results in Table I show that the strain we had isolated was probably Japanese B Encephalitis virus. The histology of the infected animal brains was identical with that found in the patient.

Dr. Smadel of the Walter Reed Army Medical Centre, Washington, D.C., kindly supplied us with the Nakayama strain of Japanese B virus and also its homologous antiserum. Cross-neutralization tests were carried out by the method described in Diagnostic Procedures for Virus and Rickettsial Diseases. We found that the Nakayama strain antiserum neutralized both the Nakayama strain and our virus and that conversely an antiserum prepared against our virus neutralized both strains.

Complement fixation tests, using antigens prepared from the two strains and antisera to these strains prepared in mice, confirmed the identity of our strain with the Nakayama strain of Japanese B Encephalitis virus.

SUMMARY

The clinical and pathological findings in case of a Japanese type B Encephalitis are described.

The diagnosis was established by the isolation and serological identification of the virus.

ACKNOWLEDGMENTS

We wish to thank Dr. Smadel of the Walter Reed Army Medical Centre for kindly supplying the Nakayama strain of Japanese B Encephalitis virus and a sample of homologous antiserum. One of us (P. C. F.) is also indebted to the Under-Secretary of State for War and the Director of Medical Services, Far East Land Forces, for permission to publish.

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