DDC AVAILABILITY NOTICE

This document has been approved for public release and sale; its distribution is unlimited.

DEPARTMENT OF THE ARMY
Fort Detrick
Frederick, Maryland

Reproduced by the CLEARINGHOUSE for Federal Scientific & Technical Information Springfield Va. 22151
Aperyanov, I. A.

Clinical Characteristics of a Japanese Encephalitis Outbreak.

The end of July 1946 was the time of an outbreak of Japanese encephalitis in the Far East, appearing as a result of a very hot summer with a small quantity of precipitation. According to Japanese and Soviet authors, the meteorologic factor plays an important role in the appearance of the epidemic outbreaks.

The first case was registered 24 July, the last—29 September.

Hospitalization of the patients in the first 2 days took place in 55% of the cases, which evidently facilitated some decrease in the mortality rate.

According to the data of Prof. Smorodintsev, the cycle of infection in each center lasts from 4-7 weeks, 40 days average. This can be fully applied to this outbreak, which significantly decreased by the first 10 days of September, and by the 20th and 30th of the month there was one case.

Death rate, according to our data, constituted 40%. According to Japanese data, it fluctuates from year to year, depending on the severity of the epidemic, from 49.6 to 77.8%; the average mortality for 10 years (1924-33) was 64.8%. The low mortality rate among our patients can be explained in the first place by the younger age of the patients: with us the prevalent age was from 19 to 22 years, according to Nako, Kingo and Kuroiwa, 77% of the patients were older than 40 yrs.

According to data from the Okayama Prefecture for 1933, the mortality among oldsters and children was 77% over a 5 year period. The period of death, relative to the day of infection, fluctuates in short levels, the average maximum rate on the 6th day of infection.
Due to the odd clinical traits of Japanese encephalitis and its neurological form, only about 40% of the patients entered with correct diagnoses. In 14 patients there was no diagnosis established, relapsing chronic malaria was decided in 10 patients, clinical dysentery in one, pneumonia in 4 and, finally, grippe in 3.

The infection began acutely, the epidemic curve was well expressed in the initial period. Infections, which would start with subfebril temperatures and gradual appearances of symptoms, were not noted by us. A less rough beginning, with a temperature of 38-39, was noted by us in the terminating period of the epidemic curve.

Almost 30% of the lethal cases entered with hazy consciousness or complete loss of it. Disruption of consciousness appeared, as a rule, on the 2-3rd day of illness; all the patients who died were in a deep coma. Generally, the loss of consciousness was noted in 61.3% of all cases. In 17% there was an acutely expressed motive disturbance (resembling infectious delirium).

In acute and extra severe courses with lethal ends, the lethal temperature reached 41.5°. In the first days of illness there were symptoms which are characteristic of severe infectious illnesses; high temperature, quick appearance of oppression of the nervous system. Such patients usually are position on their spine, legs extended, hands near the body, fingers in fist form, head cocked, eyes half open.

In the first days of illness there are appearances of meningeal symptoms—signs of affection of the brain membranes, rigidity of the occiput, positive Kernig symptom; the latter is noted in almost all cases, but appeared somewhat later than the rigidity of the occipital muscle. The Brudzinski sign was noted in 22%. Very often severe
headaches were noted, dizziness and nausea, vomiting was seldom. Pain in the extremities was noted in 70% of the cases.

Hypertonia of the muscles was noted in 80% of the cases, in certain more severe cases the pain of increased muscle tone attained a degree of decerebrational rigidity. Regarding motive spheres, there was an acute tremor, and in some of the cases there was a coarse trembling. Often there were uniform, monotone movements of some of the extremities. In 5 cases there were symptoms of catalepsy, in 8-10% cramps of the extremities.

During the acute stage there were appearances of pathologic reflexes, which were not constant and were detected on the right, left; most often there was the Babinski symptom, Gordon, and less often, the Oppenheim. Tendon reflexes in a majority of the patients were increased and only in some severe cases did they disappear.

Bulbar disruptions, characterized by disruption of swallowing action, phonation and articulation, were noted in 10 patients, a majority of the time in those patients with lethal courses.

A white dermographism was noted in almost 30% of the cases, usually in the first days of illness. In 43% the Horner syndrome was well established.

The pulse in the first half of the illness, and often in the convalescent period, was too slow, in cases of unfavorable terminations did it reach 120 or more.

With infections of medium severity, it was not clinically possible to detect variations of the valve apparatus of the heart or its muscles, with severe courses there was toxic miocardia.

Blood pressure, systematically checked in 24 patients, was basically normal, lowered only in the pregonal stage. The arte-ri-venous sizes were normal.
The organs of breathing in 36% had mild bronchitis, and in 3 cases there was pneumonia. Edema in the agonal period does not attain significant measures, very likely because of quick death. Only once was there massive edema of the lungs (called "drowning on land" by Americans). Urine output was increased and often involuntary; as a rule, constipation was present.

No pathology was noted in the kidneys or urine passages. Literary data, indicating presence of albumen in the urine of 40% of the patients, were not confirmed by our examinations.

Blood chart: erythrocytes 4 000 000 - 5 000 000, hemoglobin 65-85%, color index 0.5-0.9. Degenerative forms were not detected. Data of Japanese and Russian authors, indicating presence of leukocytosis, were not supported by us. Thus, according to Glazunov, Panov and Ionin, the quantity of leukocytes fluctuated in the levels of 10 000-18 000, sometimes more.

Our data are:

<table>
<thead>
<tr>
<th>Quantity of leukocytes</th>
<th>% of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>4000-6000</td>
<td>18.2</td>
</tr>
<tr>
<td>6000-8000</td>
<td>27.3</td>
</tr>
<tr>
<td>8000-10000</td>
<td>21.8</td>
</tr>
<tr>
<td>10000-12000</td>
<td>10.9</td>
</tr>
<tr>
<td>12000-15000</td>
<td>14.5</td>
</tr>
<tr>
<td>16000-20000</td>
<td>7.3</td>
</tr>
</tbody>
</table>

Thus, the number of leukocytes did not exceed 10 000 in 67.3% of the cases. In the formula there was an increase of neutrophils, to 80 on an average. The quantity of lymphocytes is decreased and, as a rule, there is an eosinophilia. The ESR fluctuated in wide levels—from 3-60 mm per hour, according to Panchenkov. Thus, the hematological data indicate the disruption of the hemopoiesis apparatus, characteristic of any disease, and does not appear specific for Japanese encephalitis.