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RUBEOLA ENCEPHALITIS WITH HEMIPLEGIA; REPORT OF TWO CASES TREATED WITH CORTISONE

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Based on experimental studies by Kabat showing that the demyelinating process in animal encephalitis can be suppressed with Cortisone, steroid therapy has been used by clinicians in the treatment of measles encephalitis. Prior to this, it had been shown that gamma globulin was ineffective in treatment. Allen, using steroids had complete recovery without sequelae in all 10 of his cases. Appelbaum and Abler treated 17 consecutive cases with ACTH with no deaths; there was one case with neurological sequelae and one case of mental retardation and speech impediment. Most of their cases were treated one week or less with a total of 100 to 300 mg. of ACTH. Unfortunately there was no control in either series, but both authors were impressed with the results in the light of their previous experience.

In neither of the above series was there an instance of hemiplegia. Tyler, in reviewing the literature on the neurological complications of rubeola, found 40 cases of hemiplegia including 10 of his own. Instead of a demyelinating process, many authors including Ford feel that the hemiplegias are probably the result of a non-specific effect on the vascular channels, such as one finds in many febrile illnesses. This being the case, the part that steroids would play is difficult to ascertain since the basic pathology is considered more likely to be infarction than perivascular demyelination. However, the exact mechanism of the hemiplegia is still open to question.

The most striking data found in Tyler's series were the persistence and the permanent residual of the hemiplegia, the abnormal electroencephalogram and high incidence of seizures. Of the 40 cases of hemiplegia he reported, 33 had persistent paralysis without recovery. None of the hemiplegia cases was treated with steroids.

Two cases of acute hemiplegia associated with rubeola are presented. Both of these cases had complete clinical recovery with no apparent residual. The remarkable reversal of symptoms in the cases reported would lead one away from the thought that the hemiplegia was due to arterial thrombosis or infarction and would imply some other pathologic mechanism. Further investigation along this line is needed.

CASE 1.

A 26 month old colored female developed measles 7 days prior to admission and 1 hour before admission had a sudden onset of ataxia and irritability. She had no other symptoms. Her past history was complicated by a coarctation of the aorta repaired at the age of 5½ months and an intraventricular septal defect repaired at

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the age of 16 months, both with excellent results. The rest of the history was non-contributory.

On admission, her temperature was 101° and pertinent physical findings were limited to the central nervous system, with the exception of a grade II systolic murmur heard over the entire precardium with a loud second pulmonic sound. Examination of the nervous system showed that she had bilateral Babinski's. She was generally flaccid and completely ataxic being unable to sit or stand and a hemiparesis on the left side was noted. The child was extremely irritable and very difficult to manage at the time of admission. Shortly after admission, she was examined by members of the Department of Neurology who concurred with our findings.

A blood count revealed a hemoglobin of 10 grams, 11,200 leucocytes with 23% polynuclear cells, 68% lymphocytes and 5% monocytes. The urinalysis was within normal limits. Spinal fluid on admission had no cells and the protein was 16 mgms. percent; the culture showed no growth. The spinal tap was repeated 2 days after admission and again no cells were present. The total protein was 16 mgms. percent and culture was negative. An electroencephalogram was done and the impression of the neurologist was that this was a tracing compatible with that of a normal child of this age. Skull x-rays were reported as a negative study.

At the time of admission, the impression was that this infant had either encephalitis due to measles virus or a drug intoxication of unknown etiology despite the negative history for the latter. However, it was felt both by the Pediatric and the Neurology Departments that because of the asymmetric neurological findings a drug intoxication or depression was ruled out. The child was observed for approximately 18 hours and then because there was no change in the neurological findings, she was started on a course of 200 mgms. of Cortisone a day for approximately a day and a half. The Cortisone dosage was then tapered slowly over a period of 10 days. There was marked improvement after 36 hours and the child was able to walk without any ataxia. Subjectively, she became very cheerful and cooperative. The hemiplegia had disappeared on examination. She was observed for a period of 48 hours after the steroids had been discontinued and there was no recurrence of any neurological symptoms. She had no other treatment during this time except supportive and prophylactic antibiotics.

The child was seen in the out-patient department one week after discharge and at that time, neurological examination was normal. The family moved away preventing further follow up.

CASE 2.

A 6 year old female was admitted to the Henry Ford Hospital on May 1, 1958 with a diagnosis of measles encephalitis. She had developed the rubeola rash 3 days prior to admission and at the time of the eruption, the child was put on prophylactic Penicillin 200,000 units twice a day. She was doing well until the night of admission when suddenly she complained to her mother that there were “flies” over the left side of her body; she became disoriented and had a headache. These complaints persisted for about an hour, the child then vomited and fell into a sleep. She was
seen by one of us 2 hours after the onset of the symptoms at which time she could be
awakened. After she was awake from 5 to 10 minutes, it was obvious that the child
was not oriented; she was grossly ataxic and quite irritable. She had a 3+ nuchal
rigidity. In walking she had a definite weakness of the left leg and arm. Her
temperature was 103°. She had a typical rubeola rash with severe conjunctivitis and
bronchitis. She was then admitted to the hospital.

On admission the physical findings were as described above. In addition, her
neurological examination showed that she had a hyperreflexia of both knee jerks
which appeared to be equal. She had a clonus of both ankles. There was a bilateral
Babinski. Her eyegrounds were viewed with some difficulty although it was thought
that she had a slight amount of papilledema. The pupils were unequal although
they both responded to light. Soon after admission, she had a lumbar puncture with
an opening pressure of 360 millimeters of water. The child was quiet during the course
of the lumbar puncture and the pressure readings were thought to be good. The
closing pressure was 180. Five minutes after the lumbar puncture was completed,
the child began to have a left sided convulsion which over the next few minutes
came generalized; this responded over a 15 to 20 minute period to Phenobarbital
and Paraldehyde injections. Spinal fluid revealed 180 cells, all were mononuclear.
Her initial white blood count was 21,300 with a left shift. Her hemoglobin was
12.9 grams. The spinal fluid sugar was 70, the chlorides 120, the protein was 38.
The child was immediately given 100 milligrams Hydrocortisone IV and then started
on Cortisone 300 milligrams a day. This was supplemented by a low salt diet and
Potassium Triplex, 2 drams 3 times a day, Maalox and prophylactic Penicillin. Twelve
hours after the institution of steroid therapy, there was much improvement. Her
sensorium was clearing, she was oriented as to time and place. There was possible
left facial weakness and very mild ataxia. The fundi appeared quite normal. Some
weakness remained in the left arm and leg.

A day and a half after admission, her neurologic examination was entirely un-
remarkable. She had a slight speech impediment but the parents stated that she had
had this before the onset of the encephalitis. From that point she was completely
normal in her physical examination. She was kept on 300 mgms. of Cortisone for
6 days. The dosage was then tapered over a 3 day period, and the patient discharged
10 days after her admission.

Five days after admission and after the onset of the encephalitis, she had an
electroencephalogram. Dr. Churchill of the Department of Neurology stated: “The
EEG revealed distinct abnormality consisting of difference in activity of the 2 hemi-
pheres, the right being the abnormal one. This is the type of abnormality that is
frequently associated with hemiplegias following infection and convulsion in children.
Specifically, the right hemisphere showed high amplitude 4/sec. waves that were not
seen on the left side. Occasional waves that were quite sharp in outline were observed
from the right temporal region.” She had a psychometric evaluation for a baseline
of future evaluation. The child showed a normal I.Q. of 98 and no manifestations
of any behavior disturbance. Her EEG 3½ months later was completely normal, as
was her neurological examination.
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Figure 1. Electroencephalograms of Case 2. The left curve, A, shows distorted waves before treatment, the right curve B, normal waves after treatment.

BIBLIOGRAPHY