Clinical Department.

BULBAR PARALYSIS IN TYPHOID FEVER.

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On Sept. 22, 1910, a patient was admitted to the wards of the East Medical Service in the Massachusetts General Hospital, with the following history:—

CASE 172,090.¹ 40, male, white, married. Born in Italy. Fifteen years in Massachusetts. Stationary engineer.

Family History. Negative for tuberculosis or cancer. Patient has been twice married. He has two healthy children. His wife has had no miscarriages.

Previous History. The patient does not remember having been sick in bed. Three months ago he began to feel weak and to have occasional headaches. Since then he has lost considerable weight. Two months ago he noticed a temporary difficulty in swallowing, and a thickness in speech which passed away quickly. There is no history of venereal disease. The patient has had no fits nor paralysis.

Habits. His appetite and digestion are usually good. His bowels move regularly. There is no history of nocturia, dyspnea nor cough. He drinks and smokes moderately.

Present Illness. Five days ago the patient was suddenly seized with a severe right frontal headache. There was no vomiting nor chill. Two days later he noticed a sudden difficulty in swallowing, accompanied by marked thickness of speech, and weakness of both arms and legs. These symptoms have increased steadily. This morning his neck feels stiff.

Physical Examination. The patient is well developed and powerfully built. The skin is clean, moist, and warm. The mucous membranes are of good color. There is no tenderness over the ears, the mastoid processes nor the frontal sinuses. The tongue has a heavy white coat. The teeth are good, but poorly cared for. The throat is uniformly red-dened. The tonsils are not hypertrophied and do not show an exudate. There is no general superficial glandular enlargement.

The cardiac impulse is diffuse. The left border of relative dullness on percussion is 9.5 cm. from the mid-sternal line in the fifth interspace. No right-sided enlargement is made out. The upper border of relative dullness is in the third interspace. The action is regular and rather rapid. The heart sounds are of good quality. A faint systolic murmur is heard over the precordia, not transmitted to the axilla or back. At the base, the aortic second sound is slightly louder than the pulmonic.

The pulses are equal, regular, of good volume, and synchronous at the wrist. All beats are transmitted. The systolic blood pressure is 150 mm. The peripheral vessels are not thickened nor tortuous.

The chest wall expands well and equally. The percussion note over both lungs is resonant. The breath and voice sounds are normal. No râles are heard.

The abdomen is level, soft, tympanitic throughout. No masses or areas of tenderness are made out. The liver dullness extends from the fifth rib to the costal margin, the edge is not felt. The spleen is not enlarged to percussion, the edge is not felt. The kidneys are not felt. There is no costo-vertebral tenderness.

Examination of the nervous system shows no loss of sensation. The patient is conscious and not in acute pain. There is no stiffness of the neck. Fundus examination is negative. The movements of both legs are weak. The superficial reflexes are present. There is no Kernig's sign nor ankle clonus. On the left Babinski's phenomenon is suggested. There is evidence of partial or complete paralysis of the muscles supplied by the third, sixth, seventh and twelfth cranial nerves, more marked on the right side.

The blood examination shows 95% of hemoglobin; 9000 white cells, of which 58% are neutrophilic, and 42% basophilic. The red cells are normal.

The urine passed during the twenty-four hours following admission has a specific gravity of 1015. There is a slight trace of albumin, no sugar. The sediment contains hyaline and granular casts.

The temperature is 101° F., the pulse-rate 100, and respiration 25 to the minute.

Subsequent Notes. On the day following admission, the patient developed marked dyspnea with breathing of the intercostal type, suggesting partial paralysis of the diaphragm.

Two days after entry the patient was unable to swallow and bilateral deltoid paralysis was noted. The legs were nearly immovable. No leg reflexes were obtained.

The patient died early the next morning, respiration stopping before the heart beat. He was conscious until death.

While under observation the temperature varied between 101-103.5° F. The pulse between 100-110, and respiration between 25-40.

The urine was small in amount but was passed without difficulty. The bowels moved only as the result of an enema. No blood cultures or agglutination tests were made.

SUMMARY.

A well developed man, previously in good health, was seized suddenly with fever accompanied by difficulty in swallowing and weakness of the legs. He died eight days after the onset of his illness with symptoms suggesting bulbar paralysis and an increasing paralysis of areas supplied by spinal, as well as cranial nerves. The course of the paralysis was not typically ascending or descending, but diffuse, suggesting a rapid involvement of one nerve area after another.

An autopsy² was made 56¾ hours after death. The following noteworthy features are recorded.

The meninges, vessels of the circle of Willis, the sinuses and middle ears on section are not remarkable. The brain weighs 1385 gms. Section of tissue from the hemispheres and brain stem shows nothing to record. The upper end of the cervical portion of the cord is not remarkable.

The small intestines to the region of the ileum are negative. The mucosa in this area shows scattered smaller and larger grayish elevated plaque-like masses of roughly round to oval shape and rather firm. Some of these have a flat button-like appearance. There is hyperplasia of the mesenteric lymph glands and of the spleen. Cultures from the spleen show many colonies of Gram decolorizing non-gas producing motile bacilli morphologically like typhoid, and giving a positive agglutination reaction against an anti-typhoid serum diluted 1-100.

The anatomical diagnosis of typhoid fever, therefore, was made, the clinical picture being that of an acute bulbar paralysis.

Various standard text-books^{3, 4, 5, 6, 7} of medicine, pathology and neurology mention that bulbar paralysis is among the rare complications of typhoid fever. The following cases have been reported.

Fritz⁸ writes that on the tenth or twelfth day of a typhoid fever, a female patient was suddenly seized with an incomplete paraplegia. The paralysis extended upwards, first affecting the arms and then respiration, "so that a grave danger threatened the patient's life." After bleeding, however, gradual recovery took place although complete motility was never regained.

Curschmann⁹ describes in detail the following case:—

A male patient 31 years old with unimportant family and previous histories was admitted to the hospital. Six days before entry he had a violent chill followed by weakness and pain in the legs.

General physical examination was negative except for an enlarged spleen. The patient could not move legs, forearms, hands nor fingers, but could move slightly both shoulders. The skin reflexes were diminished. The knee and ankle jerks were absent. On the following morning he was unable to extend his tongue. The left facial nerve area was partially paralyzed. In the evening he died.

The autopsy findings showed an enlarged spleen and a small intestine characteristic of typhoid. The gross appearances of the brain were not especially abnormal. Cultures from the spleen, the cervical cord and medulla showed colonies of typhoid bacilli. Microscopic examination of the brain and cord showed many typhoid organisms either isolated or in groups with but slight tissue changes. Curschmann considered that the symptoms were due to a disturbance in brain function caused by the bacillary invasion.

Eisenlohr¹⁰ reports three interesting cases of typhoid with symptoms of bulbar paralysis. All the patients were young and severe cases. Two lived, one died on the seventeenth day of the disease. All showed involvement of the bulbar nerves, and marked motor weakness of the entire body musculature. Other nerves were affected variously. Two cases recovered entirely. In the fatal case, autopsy showed typical typhoid changes in the intestine and mesenteric

lymph glands. Cultures from the medulla produced no typhoid organisms, but a few colonies of a yellow coccus, not identical with either staphylococcus aureus or citreus. Microscopical examination of the brain was not remarkable except for the presence of organisms. Eisenlohr's conclusions were similar to Curschmann's.

Henneberg's¹¹ case is that of a man 26 years old, of unimportant previous history. Eight days before admission to the hospital he was taken sick with headache and general lassitude. At entry he was slightly delirious. His spleen was palpable, otherwise his general physical examination was not remarkable. He was, however, unable to move his tongue or lips and could not swallow. The Widal reaction was negative. The cerebro-spinal fluid was normal. A week later there developed slight ataxia of the arms and legs. On the tenth day after entry evidence of bulbar involvement became suddenly augmented. The patient collapsed and died. The autopsy showed a typical typhoid intestine and spleen, from which cultures of the typhoid bacillus were grown. The brain, cord, medulla and vagus nerve showed nothing abnormal either by culture, by gross examination or by careful histological study. A toxic origin for the symptom complex was assumed.

Mori¹² saw a patient 25 years old who entered the hospital with typhoid fever, as proven by a positive Widal reaction and typhoid bacilli found in the feces. His family and previous histories were negative, except for beriberi ten years previously. Three weeks after the onset of fever, paralysis of both legs occurred, later involving the abdomen, diaphragm, arms and intercostal muscles. The paralysis progressed for three weeks, and then terminated fatally. No autopsy was made. Mori considered the clinical manifestations to be of toxic origin.

Symptoms of bulbar paralysis in typhoid fever have been described as a late complication developing after defervescence has begun.

Leudet¹³ mentions a case in which, toward the end of the third week, when convalescence seemed beginning, a woman who had been through a typical typhoid fever, suddenly was seized with paralysis in both legs. This extended progressively upwards, ending with paralysis of respiration and asphyxia. There was integrity of intelligence to the moment of death, which occurred seven days after the onset. At autopsy the brain and cord were not abnormal. In the intestine there were typhoid ulcers, some of which were partially scarred.

Kümmell¹⁴ cites the following. A patient 25 years old had been ill for six weeks with lassitude, fever, loss of appetite and weakness of the legs. Two days before admission to the hospital, loss of strength was noticed in the back, followed by weakness of the arms. An acute ascending motor paralysis followed, producing death in four days from vagus involvement.

Autopsy showed typhoid scars in the intestine. Small microscopic hemorrhages were found

scattered through the medulla and brain at various levels. They appeared to be of varying age. Kümmell believed that these changes might be due to bacillary emboli. However, there is no record of any cultures, nor were any organisms seen.

Another case is reported by Pitres¹⁵ and Vaillard. Three days after defervescence in typhoid the patient developed broncho-pneumonia. Nine days after the temperature had become normal, paralysis of the legs occurred. An ascending paralysis rapidly followed, involving the abdomen, arms, face, and finally the region of distribution of the bulbar nerves. The patient died with evidence of respiratory insufficiency.

The autopsy notes are especially interesting. The intestine and spleen showed typhoidal lesions. The gross and microscopical appearances of the medulla oblongata and cord were not remarkable. Various of the peripheral nerves, however, showed atrophied fibres with the myelin sheaths in various stages of fragmentation. The authors believe that the changes were produced by the typhoid toxin. In this case, for some reason, the nerves generally had been affected, causing a clinical picture of acute ascending paralysis.

Geniez¹⁶ describes a case in a man, beginning with difficulty of movements of the feet four days after defervescence from typhoid. By the following day paralysis of the legs was complete; on the third day the arm and trunk were affected. On the fifth day the patient could not speak; there was paralysis of respiration and the patient died asphyxiated. No autopsy was made.

Thus in the literature there are twelve cases of typhoid fever in adults complicated by symptoms of bulbar paralysis. In eight the manifestations occurred at the height of the disease, in four after convalescence apparently had begun. In two the disease started suddenly with a chill and various nervous manifestations. In the others there was nothing to suggest an atypical typhoid fever. Eight of the patients died.

Symptoms of paralysis began acutely or gradually, and lasted from a few days to several weeks. In none of the cases did the time of the onset of the paralytic symptoms after the beginning of the disease bear any relation to its eventual outcome. In the fatal cases, the progress of the paralysis was more rapid than in those recovering.

In one case the paralysis was descending and in one diffuse. All the others were cases of ascending paralysis.

Seven cases were examined pathologically. In all, typical typhoidal changes were found in the intestines, spleen and mesenteric lymph nodes. In all the gross appearances of the brain and cord were normal.

Of the five cases which were submitted to thorough histological and bacteriological study, two showed nothing abnormal in the brain or cord. One, however, showed atrophic changes in various of the peripheral nerves. One case had

vascular lesions consisting of scattered microscopic hemorrhages at different levels of the medulla and brain. Two cases showed positive bacteriological findings. From the brain and cord of one, typhoid bacilli were recovered, from the other, an unidentified coccus not identical with either the staphylococcus aureus or citreus. The organisms were seen histologically in scattered foci through the brain and cord without any marked tissue reaction.

The clinical picture encountered in several of the cases, suggests strongly an acute poliomyelitis. This cannot be definitely excluded. From the facts that in all cases examined, typical typhoidal lesions were found in the intestines and spleen, and that in no cases were pathological changes observed which suggested anterior poliomyelitis, it seems justifiable to believe that the symptom complex was due to the original infection.

On the whole, acute bulbar paralysis must be recognized as being a rare complication of typhoid fever. The symptoms usually suggest at the onset an acute ascending paralysis, developing either at the height of the disease or after convalescence has begun, and ending with evidence of bulbar involvement. Recovery may take place after a prolonged illness, or death may occur within a few hours.

The varying pathological findings described show that such a clinical picture may exist without evident tissue change in the nervous system or be due to peripheral nerve lesions, or to a hemorrhagic encephalitis analogous to that found in other forms of infection, or finally to the presence of organisms in the brain, whether typhoid bacilli or secondary invaders, in sufficient numbers to cause the symptoms encountered.

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