Before the Vaccines: Medical Treatments of Acute Paralysis in the 1916 New York Epidemic of Poliomyelitis

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Abstract: Hospitals in New York were overwhelmed by the epidemic. With nothing known about the virus, most cases were treated with traditional or symptomatic remedies. New treatments were made though unsuccessful. Serum from various sources was given although it was many years before this was found to be ineffectual. Lumbar puncture was made, sometimes with additions. Although this became standard treatment, there were some who thought it was harmful. Many histories of polio ignore treatment.

Keywords: Lumber puncture, New York, poliomyelitis, serum therapy, treatment.

INTRODUCTION

The 1916 epidemic was the most devastating epidemic in the history of poliomyelitis and is widely quoted in books about polio. Nowadays we are aware of the dangers of the escape of disease agents from laboratories, but in 1916, this was not considered [1] although impressive studies of the epidemiology were made [2]. The treatments used in the epidemic have been discussed by Rogers [3] and few others although the case-fatality rate was very high.

TREATMENT

Very little was known about poliomyelitis except that some believed it was caused by a virus, others by bacteria. However, ideas about viruses were almost lacking. There was no recognised treatment, either during the illness or after paralysis. Laboratory knowledge was rudimentary, but the epidemic of 1916 which started in New York City, was so overwhelming that no studies or even meaningful statistics could be organised.

The nearly nine thousand cases in Greater New York were treated in the four New York City hospitals and in 27 others [4]. Almost half, 4516 (of which 3083 were given symptomatic treatment and 956 had a lumbar puncture) were treated in the City hospitals [5]. The figures did not distinguish cases which were non-paralytic from those which were paralytic, minor from severe, or those with one treatment or several. Particular treatments seem to have been made to those most affected, but without controls or comparisons and no indication of severity of paralysis. Naomi Rogers in her chapter ‘the promise of science: polio and the laboratory’ explored the extent of the treatments without considering their impact on patient survival [3]. In 18 fascinating pages, Rogers discussed various non-medical treatments which had been suggested by well-wishers [3].

But it was clear that by the time children had arrived at hospitals, the virus had reached the motor neurons and the damage had already begun.

CONVENTIONAL TREATMENTS

Some treatment was based on traditional medicine such as blisters, and mild anti-irritants including ergot. The disinfectant hexamethylamine (urotropin) was used not only externally, but also as an internal disinfection: after intravenous injection, it appeared in the spinal fluid. It was given to 51 cases by intraspinal injection every six hours [5]. Hydrogen peroxide was also used externally and as a nasal spray. Symptomatic remedies were very common to reduce fever, pain etc. Rogers reproduced the table of treatments [5] which showed that the majority of treatments were symptomatic. Doctors also gave tonics in an effort to do something. The rationale of a number of treatments was not specified or was based on false reasoning. The rationale for giving tetanus antitoxin was that tetanus, rabies and poliomyelitis all attacked nerve cells, so perhaps giving the antitoxin would block access to absorption sites on the cells [6, 7]. But the giving of diphtheria antitoxin to five cases with three deaths was ‘a most unwarrantable procedure [with] no good results’ [5].

Adrenaline was injected intraspinally, but there were 100 cases with 45 deaths although Meltzer claimed that it aided recovery [8]. It was also given intramuscularly, although of 35 cases, 23 died. Quinine and urea hydrochloride were given intraspinally: three of six children given many doses by mouth and intramuscularly died.

Fischer at the Willard Hospital reported on several hundred patients, saying that lumbar punctures should be done and blood transfusions given if the patients were emaciated. Five cases were given intravenous salvarsan ‘with excellent results’. Deep intramuscular injections of strychnine (given as a tonic) were given three times a day on alternate days. He gave 10-15 cc of convalescent serum if
possible [9]. Fischer noted that ‘a severe febrile reaction frequently follows the intraspinal injection of human serum. In some case nausea and vomiting followed’. He also gave intraspinal irrigations with normal saline after withdrawing 30-100 cc of CSF [10].

Barber in a lecture in 1915 had advocated heat treatment and massage, but was doubtful about treatment with electricity. He suggested giving strychnine internally or subcutaneously, but said that hexamethlenamine (urotropin) with benzoic acid was of no value after infection [11].

TREATMENT WITH INJECTED SERUM

The use of antiserum in cases of diphtheria and tetanus suggested that serum might be used in cases of polio. Unfortunately, polio is a much more complicated disease. It was 35 years before it was confirmed that there were three antigenic types of the poliovirus. It was even longer before it was shown that although there was a viraemic stage, the virus then began a long passage along the motor neurones to the spinal cord during which time it was protected from antibodies in the blood. Even when the virus was in the cells in the spinal cord, the most damage was caused by lymphocytes and other immune cells entering from blood vessels, not by poliovirus infected cells. The use of normal citrated blood in two cases and Special Jobling serum in five cases was useless. It was thought that normal (adult) human serum neutralized the poliovirus and was employed in 114 cases, intraspinally or intramuscularly. In one instance, 23 cases were given the serum, but 15 died. Convalescent serum was tried with nine cases injected into the spine and six into the muscles.

In the Monograph about 11 pages were devoted to treatment with different sources of immune or normal serum [5]. We now know that some of the ‘immune’ sera might have had antibodies to other types of poliovirus, but that most of the ‘normal’ sera would have had antibodies to the same type of virus as caused the epidemic. The treatment was to give 15cc after removal of slightly more spinal fluid. This dose was repeated up to four times every 22-24 hr, but in more severe cases was repeated every 12 hr. Some cases were treated by physicians outside Department Hospitals.

Meltzer criticised auto inoculation of serum as there was no evidence that it did any good and that it might actually be harmful [8]. Emerson et al were clearly worried about the safety of serum therapy, but their experiment was limited by the number of monkeys they possessed. Four of six control monkeys died (66%) compared with five of six treated with spinal injection of serum (83%). The numbers were small, but they commented that ‘it would seem, therefore, that from this point of view the action of the serum may even be distinctly harmful’. Their conclusion about the treatment for children was that ‘intraspinal injections of serum are not only of no value, but also that there may be in them an element of harm’ [5, p. 286]. No date was given for the experiments on the effects of serum treatments on the monkeys, so we cannot know whether the shortage of monkeys was due to the use of at least 17 for passage of virus to produce a more virulent strain – a curious priority in the middle of an epidemic and as their experiments showed that serum treatments were probably harmful [5]. No wonder the case-fatality rate was not discussed. In later trials, the use of immune serum was found to be useless [12].

Horses did not react to poliovirus, but normal horse serum was injected intraspinally in 98 cases (with 35 deaths) and ‘its use was not justified’. Horse serum, given to 159 cases in Chicago, was prepared by immunising horses with a gram positive coccus derived from the CSF of patients [13].

LUMBAR PUNCTURE – A SUSPECT PROCEDURE

Lumbar puncture (LP) is used to obtain cerebral spinal fluid (CSF) for examination by the pathologist or microbiologist and LP is a recognised procedure when an enterovirus infection of the central nervous system (CNS) is suspected. The removal of some CSF relieves the pressure on the spinal nerve and microscopic inspection will show the presence of lymphocytes – a key finding for poliomyelitis diagnosis. It is so routine that the need for a LP is not questioned, the patient not monitored for possible side effects and case-histories are not scrutinised.

LP was commonly used and by itself in 956 cases in the City hospitals. At one hospital there were 209 single punctures to relieve severe meningitis or hydrocephalic symptoms and 119 with multiple punctures: 121 died. In another hospital 43 were treated by spinal puncture of whom 17 died. At Riverside Hospital however, 156 cases received lumbar puncture and only 21 died. Adrenaline was also given with LP in 100 cases: 19 cases were given intraspinally and 10 died. Meltzer of the Rockefeller Institute, after withdrawing fluid by LP, injected two cc of adrenaline every six hours for four days [8]. Removal of fluid calmed the patient and perhaps took away some poisons. However, in some hospitals, spinal fluid was given back by intramuscular auto-inoculation to 27 cases, of which 15 died.

The use of LP as a diagnostic tool for bacterial meningitis was questioned by two army doctors in 1919 [14]. They found that patients who were punctured before intravenous serum therapy, developed meningitis the following day, whereas those who were given serum first did not. They also gave a six page review of the literature. Poliovirus has already reached the spinal cord when a LP is made, so that comparison with bacterial entry from the bloodstream is difficult.

The taking of CSF by lumbar puncture from suspected cases of poliomyelitis was, however, a controversial technique in the early 1950’s. Two French experts, Debre and Thieffry, advocated routine LP and examination of the CSF for the diagnosis of acute and non-paralytic polio and to differentiate polio from Guillain-Barre syndrome [14]. Mollaret, however, compared the progress of patients who received lumbar punctures after the onset of paralysis with those without paralysis [15]. Of 17 who received lumbar punctures on the day of paralysis or the next, all had ‘aggravation’ of paralysis within 12 to 24 hr and 15 developed respiratory problems at the same time or a little later. Ten patients with clinical signs, but no paralysis, were given lumbar punctures and all developed paralysis 12 to 48 hr after and seven developed respiratory problems. One patient in each group died. Mollaret quoted other cases and showed that if virus was inoculated into monkeys and
paralysis did not occur, then a lumbar puncture made subsequent paralysis more likely. In a 1954 review of provoking and localising factors in polio, Trueta and Hode, from Oxford, England, wrote of the potential damaging influences commonly used in medicine, among the most potent are spinal puncture...[17]: they did not elaborate. However, in a book published two years later, Trueta did not mention LP as a factor precipitating frank disease [18].

By definition, provocation occurs when infection and injection overlap, with paralysis following 7 to 28 days later. Intramuscular injections (IM), exercise and child-birth in the 24-48 hours before paralysis occurs, result in more severe paralysis of the regions concerned and are called aggravation [19]. The enhancement of paralysis 12 to 48 hours after LP [2] suggests that because the virus must be already in the spinal cord, this may be aggravation and cannot be provocation.

Although LP may be a normal treatment, it may not be suitable for polio. When I showed pictures of sections of polio post-mortem slides (from Wickman's monograph) to immunologists, they identified these as a host versus graft reaction. In polio there is a sudden invasion of the privileged CSF site by activated lymphocytes with the main areas of reaction.

For this, spinal fluid would be drained through a hollow needle, to be replaced by large quantities of transfused salt solution. Fortunately the National Foundation asked Dr. Kramer to test the treatment in control, as well as infected monkeys. The results were unambiguous, as even many of the controls died [21].

**CASE-FATALITY RATES**

In the US during the first two decades of the 20th Century, polio was largely a disease of very young children and so was known as infantile paralysis. Fischer reported that in the 1907 epidemic in New York the case-fatality rate was about 5% of less than 1000 cases but that in the 1916 epidemic of more than 9000 cases it was 20 - 25% [10]. However, polio was not a notifiable disease and some cases of paralysis were unreported. Children with minor paralysis were concealed at home, as parents were scared of the subsequent paralysis more likely. In a 1916 epidemic, doctors used many treatments, with others used by lay persons. The earliest examination of the CSF by LP was advocated.

**HISTORIES OF THE EPIDEMIC**

Many books on polio give some details of the 1916 epidemic, but with scarcely any mention of the treatments given: they concentrate chiefly on the vaccines of Salk and Sabin. Paul, in his excellent history of polio, devoted a chapter to the epidemic, but only discussed quarantine, research projects and the career of Haven Emerson [12]. Offit also concentrated on Emerson’s application of quarantine and sanitary measures with a brief mention of remedies with spinal fluids [22]. Oshinsky [23] as well as Bookchin and Schumacher [24] briefly mentioned the epidemic, but without the effects of treatments. Gould discussed the use of sera and a few other treatments including those of Meltzer [25].

**CONCLUSION**

The 1916 epidemic coincided with the war in Europe and was overtaken by the entry of the US against Germany in early 1917. It was then overshadowed by the great influenza pandemic of 1918. Doctors were overwhelmed by the epidemic with only two treatments which were favoured. The use of immune serum was later shown to be ineffective. The use of lumbar puncture for poliomyelitis has been questioned, but it is difficult to question a tool used routinely in many other conditions. Fortunately, the success of the eradication programme with the Sabin vaccine has made the use of lumbar puncture for poliomyelitis a rare procedure.

**CONFLICT OF INTEREST**

The author confirms that this article content has no conflict of interest.

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