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Case of Recurrent Botulism Poisoning

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It has generally been claimed that *Cl. botulinum* produces its toxin exclusively in food products outside an organism. It has also been supposed that *Cl. botulinum* is a toxic saprophyte and is incapable to multiply inside an organism. As the toxin enters the organism with ingested food, it causes a serious poisoning. Hence, the inference that, in the case of botulism, we are dealing exclusively with a poisoning.

But, investigations of MATVEZV, KORYCKI et al. shed in recent years a new light on pathogenesis of the disease; thus, a relative position is assumed that the mode of development of botulism reveals a toxoinfectious pathogenesis. The authors based their opinion on clinical observations and on ample experimental material.

For example, a condition in which dissimilar incubation period is sometimes short and lasts few hours, or, at times, it lasts even up to 14 days, depends on initial dose of the toxin that entered the organism; the larger the dose, the shorter the incubation period.

MATVEEV explained, in some cases, a prolonged incubation period by this that a toxic dose entering the organism with ingested food was too small to develop in a short time the symptoms of poisoning. A full clinical picture develops only after the toxin had been produced in sufficient dose in the organism by Cl. botulinum.

MATVEEV also explained the increased body temperature by activity of Cl. botulinum after poisoning with botulism. A slow course of recuperation from poisoning with botulism may indicate that botulinal bacilli multiply and thus produce a toxin, therefore the nature of the disease is toxinfectious.

In the material obtained from patients stricken with botulism and treated with serum we found (in late stages of their treatment) botulinal bacilli and their toxins in the blood, urine and feces. The followers of the toxinfectious pathogenesis of the disease explain that the appearance of the toxin in the blood and urine of patients treated with serum is due to a condition that the toxin is produced in the organism by Cl. botulinum.

The ample bibliography about botulism invariably implies that in such cases a relapse is very rare; thus, because of this, we described the following case as we observed it.

Z.S., 38 years old, a RR-gang crew member, came in September 1952 to the 1st Clinic for Internal Diseases, Medical Academy, Poznan, with symptoms of dimmed vision, dryness in the mouth, hoarse throat, difficulties in urination and a general weakness. The beginning of the disease occurred suddenly, 5 days before his arrival

to the clinic; it produced vomiting, diarrhea and abdominal pains. The patient attributed his ailments to the ingestion of boiled chicken. But, the whole family ate the same food and nobody else became ill. The indisposition of the alimentary canal disappeared within two days after adaptation of strict diet. However, the weakness became intensified and it was followed by dryness in the mouth and the dimness of vision. The patient went to a doctor, who suspected botulinal infection and directed him to the clinic.

The patient experienced speech difficulties due to hoarse throat. He complained having a double vision and dimmed vision in trying to see distant objects, also a lack of vision in looking at close objects. His appetite was considerably diminished; he also had difficulties in swallowing due to the dryness in the mouth and this was followed by constipation. He complained having difficulties in urination in spite of the tenesmus feeling.

Past diseases. He spent two weeks in bed about three months ago at the City Hospital, Poznan, suffering from botulism, which was a group infection. Eight persons became ill having eaten the same type of food. The patient stated that he experienced at that time similar symptoms, but of a far lesser severity. He did not receive antitoxin serum treatment. After his ailment declined two weeks later, he was discharged from the hospital and had a feeling of being healthy up to the present time. He was never sick before.

While considering his deviations from a normal state, we determined that the patient was apathetic and his facial expressions showed considerable weakness. His skin was pale and dry. The pupils

wide and round, failed to react on light, to converge and to re-adjust. The mucosa of the mouth and nasopharynx were dry. The tongue was dry and coated with a white-gray fur. The patient's conversation was quiet, the voice was hoarse and muffled.

A supplementary examination revealed a decrease of blood corpuscles to $1\frac{1}{2}$ 28. The blood and urine pictures were normal.

The electrocardiography revealed no deviations from a normal condition.

Considering the described clinical picture, we recognized botulinal poisoning after we excluded the atropine and mushroom poisonings on account of the lack of typical symptoms and as a result of anamnesis.

The general condition of the patient was grave during the first 5 days, although he had no fever, but was apathetic and very weak.

He was administered: 200 ml of serum, strychnine injections, intravenous and subcutaneous injections of physiological solution of salt, laxatives and instillation of pilocarpine into the conjunctival sac.

His course of recuperation was slow. The first subsiding symptom was that of difficult urination and then, dryness in the mouth and in the nasopharynx mucosa began to disappear gradually. The longest lasting of all symptoms were the optical disturbances. The patient left the clinic after 3 weeks of hospitalization; his state of health was generally improved and no obvious symptoms remained.

We were dealing in this case with a rare incident of recurrent botulinal poisoning that followed after a complete recovery.

The interpretation of this phenomenon is difficult. As a partial explanation one may accept the toxin-infectious background of the disease.

Experiments prove that spores of botulinal bacillus can remain in animal organs 100 to 120 days without losing their abilities to multiply. A reduced resistance in an organism, caused by additional infection or trauma, favors a development of larger quantities of toxin, it also supports reproduction of bacillus, consequently outbreak of the disease follows.

It should be assumed that botulinal bacillus and spores survived in the organism of the patient after his first illness, and that, due to reduced resistance for unknown reason, the toxin began to develop and caused the recurrent illness.

The described case may serve as a contribution to the postulate of toxin-infectious pathogenesis of botulism.

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