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G. D. CHISHOLM, F.R.C.S.,*
Department of Surgery,
Postgraduate Medical School, Hammersmith
Hospital.

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Medical Memoranda

Sensory Loss in Poliomyelitis

The clinical picture of poliomyelitis is one of meningeal irritation followed by flaccid paresis due to injury of the lower motor neurone. Pains in the back and leg muscles are the rule, and loss or even alteration of sensation is extremely rare. It is only recently that confirmation of infection with the poliovirus has been easily obtainable. Formerly the presence of sensory change would throw doubt on the diagnosis. Plum (1956) reports the only case with full virological confirmation which could be found in the literature. He refers to 10 previous case reports, the earliest in 1909. He throws doubt on the diagnosis of many of these cases, although the diagnosis of the case described by Sandifer (1950) is well substantiated on clinical grounds.

In the past 10 years 800 cases of poliomyelitis have been admitted to this hospital. In only one of them, the case described, was there sensory change.

CASE REPORT

On September 8, 1959, a man aged 21 developed pain in the back and had little sleep that night. Next day he complained of headache and pins-and-needles in the legs, and found he could not walk. He was admitted to another hospital on that day and transferred to the neurorespiratory unit on September 11 because respiration was failing.

On admission his temperature was 103° F. (39.4° C.). He had marked stiffness of the neck and back. There was slight weakness in both arms, the tendon reflexes being reduced; his vital capacity was 1,000 c.cm.; spinal and abdominal muscles were very weak, and the lower limbs were completely paralysed. There was some retention of urine. He had complete loss of all forms of sensation up

Complement-fixation Test for Poliovirus

	Type I	Type II	Type III
September 14	<5	<5	<5
October 12	20	<5	<5
.. 17	20		

to the level of L2 dermatome, both sides. The cerebrospinal fluid contained 21 cells and 262 mg. of protein per 100 ml. No spinal block was found. Blood was taken for complement-fixation test for poliovirus on September 14 and on October 12 and 17 (see Table). A specimen of faeces was obtained on September 29, and from this type I poliovirus was grown.

*Temporary address: Brady Urological Institute, Johns Hopkins Hospital, Baltimore, Md.

The patient was placed in a tank respirator soon after admission. In the next few days he developed abdominal distension and vomiting. He lost the power of swallowing, and his vital capacity fell to 450 c.cm. A tracheostomy was performed under local anaesthesia, and positive-pressure respiration was started through a cuffed tracheostomy tube. By September 15 his acute illness had come to an end and he began to improve. The power of swallowing returned on September 21. By September 24 his vital capacity had increased to 800 c.cm. and artificial respiration was no longer required. By this date sensation was beginning to improve, but retention of urine persisted until November 23. Sensation continued to improve and the upper limit slowly descended. By January 2, 1960, the only sensory abnormality was sensation of cold in the legs. By then the vital capacity was 2,500 c.cm. The power in the upper limbs had much improved, although the recovery in the lower limbs was slight. He was soon afterwards transferred to another hospital for long-term orthopaedic treatment.

COMMENT

The complete loss of sensation within a well-defined upper border and a prolonged retention of urine were striking features in this case, which otherwise presented the picture of poliomyelitis, confirmed by the finding of virus in the stool and a rising complement-fixation titre in the blood. The cases described by Plum and Sandifer, three in all, present a very similar picture with complete loss of sensation up to the same level, retention of urine, complete flaccid paralysis in the legs, as well as scattered weakness in the upper limbs. Their patients, however, failed to recover any power in the legs and their sensation only partly returned. Plum (1956) believed that the changes in the legs in his cases were due to the destruction of the sensory nerve cells in the posterior horn of the spinal cord, which has been described in many cases coming to necropsy, although he recognized that clinically the picture was one of transverse myelitis.

Destruction of the nerve cells of the posterior horn is common in cases which come to necropsy and has been seen regularly in patients dying at this hospital, but in none of these cases has there been any sensory loss of life. As Bodian (1949) makes clear, in these severe cases the destruction of the posterior horn cells is patchy. Sharrard (1955) has shown that 40% or more of the motor cells have been destroyed in every case with the slightest degree of paralysis. It is probable that sensory-cell destruction never reaches this extent. If loss of sensation was due to destruction of the posterior horn cells one would expect it to occur often in severe cases, to be slight in degree, and to be scattered. In contrast the loss of sensation in this case and in the three mentioned was complete and had a definite upper limit. It is probable, therefore, that some process different from that commonly occurring in poliomyelitis is taking place. The diagnosis of transverse myelitis, possibly due to a vascular lesion, as suggested by Sandifer (1950), provides a more satisfactory explanation for the clinical picture during the acute stage and the subsequent progress of this case and the others under discussion.

R. V. WALLEY, M.D.,
Deputy Physician,
Ham Green Hospital, Bristol.

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