CLINICAL RECORDS.

A CASE OF TRANSVERSE MYELITIS, SHOWING AN ABNORMALITY OF THE SPINAL CORD.

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Abnormalities of the spinal cord, of developmental origin, are very rare, and especially so since Van Gieson1 has shown many of the appearances described as such to be nothing more than artefacts. And so I do not claim that the condition of the cord in the following case is due to congenital malformation, for I consider it also an artefact. But the appearances of the cord are striking, and as the case otherwise presents some points of interest, it would seem worthy to be put on record.

The case was sent to the post-mortem room as one of Landry’s paralysis, and the following is a brief summary of the illness:—

J. C., 45, a puddler, was admitted to the wards of the Glasgow Royal Infirmary on 17th August 1899, with paralysis in both legs and in both arms of about three days’ duration. On 10th August patient had a specially hard night’s work, and sweated a great deal. On rising from bed the next morning, he experienced a feeling of “needles and pins” with numbness in his toes. He went to his work, but on the way home he noted that there was some weakness about the ankles, and that the prickling sensation had extended up into the thighs. The next day, the 12th inst., patient could still walk, but on the 13th he couldn’t, and on the 14th he was quite unable to move any muscle of the lower limbs. On the 13th and 14th the arms began to feel numb, and this was quickly followed by loss of power, so that on the 15th he could not move the arms at all. The paralysis was experienced first in the hands and wrists, then in the elbows, and lastly at the shoulders. Patient had made no observations as to a feeling of numbness in the trunk, and the first time he experienced any difficulty in breathing was on the day of admission.

He was admitted on the 17th inst., when there were observed to be paralysis in the arms and legs, and in the trunk up to the level of the clavicles. The paralysis in the arms was not complete, for both thumbs could still be adducted and abducted. The breathing was entirely diaphragmatic, except that the chest seemed to be moved to a slight

1 "A Study of the Artefacts of the Nervous System," New York, 1892.
extent by the sterno-mastoids. There was no paralysis in the muscles of the neck, face, eyeballs, or tongue.

The pupil reflexes were normal. The voice was hoarse, and gradually became more so towards the end of life, when patient could neither cough nor swallow. Sensation was not markedly impaired, unless in the legs, and there only below the level of the knees. Both deep and superficial reflexes (those below the level of the clavicles) are noted to have been absent, and it is said that there was no loss of control over bladder or rectum. Patient died some forty hours after admission. The temperature at first remained normal, rising to 102° a few hours before death. The respirations ranged from 22 to 28.

At the post-mortem examination the body was noted to be that of a well-developed and well-nourished man, and, so far as external appearances went, quite free from any congenital deformity. It was permitted to examine the brain and spinal cord only. The brain and its membranes were quite normal in their naked-eye appearances. The cord was removed along with its membranes. On laying open the spinal dura, a portion of the lower dorsal cord, of about 1/2 in. in length, was seen to be considerably swollen, and it was apparently in an extremely disorganised and softened condition, seeming to have the consistence of thick cream. A smaller area of the cord in the upper cervical region was also swollen, and it looked slightly softened. These softenings were very white in colour, much more so than the rest of the cord, and they were recognised by all those present at the examination as being areas of transverse myelitis. Cultures and cover-glass preparations were taken from the softened areas, and Fränkel's diplococcus was found in a practically pure state.

The cord along with its membranes was placed without further manipulation, and with every care to prevent deformity, in 10 per cent. formol. After being twenty-four hours in this solution, the cord was again examined. It was now found that at the lower end of the dorsal softening a considerable amount of the peripheral tissue had disappeared, giving the cord at this point a constricted appearance, but the upper part of the softening remained the same as before. The constriction was doubtless the result of the very soft tissue being washed away by the fixing fluid.

Transverse incisions were now made throughout the whole length of the cord. On reaching the upper part of the dorsal softening (now quite hard), there was found what seemed to be an inflammatory exudation lying to the left and slightly in front of the cord, and occupying about a half of its circumference (Fig. 8), otherwise the cord at this level looked practically normal, quite different from what one seeing it before hardening would have expected. On making serial section throughout the softened part, it was found that this so-called exudation was really an offshoot from a portion of the softening a little lower down. Fig. 1 is meant to represent this softened part in longitudinal section, and its transverse lines, a, b, c, d, e, f, g, indicate the levels of the transverse sections shown in Figs. 2, 3, 4, 5, 6, 7, 8. These sections were made from portions of the cord embedded in celloidin, and they were stained with (a) hematoxylin and eosin, (b) thionin, and (c) alum-carmine.

The first section (Fig. 2), taken from immediately above the
narrowest part of the constriction, shows considerable loss of tissue, with
distortion of the parts remaining. The right anterior horn seems more
or less normal, but the left is elongated and displaced inwards, while
the posterior horns seem to have become fused together. Fig. 3 is but
a slight modification of Fig. 2. But in Fig. 4 the posterior horns have
become separate from each other, the left posterior horn seeming to have
joined the anterior horn of its own side. In Fig. 5 and the succeeding
figures the distortion is but slight, and the disposition of the horns
gradually assumes the normal arrangement. In Fig. 6 we have the first
appearance of the offshoot or accessory portion of the cord represented
in Fig. 1. Fig. 7 shows the process of separation, and Fig. 8 its
complete separation. The accessory portion, it is to be noted, contains
a proportion of grey matter, in shape very like an anterior horn, with
attached to it a more elongated part, which might correspond to a
posterior horn. But these points are sufficiently indicated in the
illustrations, and require no further description. The figures were
drawn by means of the camera lucida, and they may be taken to
represent accurately the outlines of the sections and the disposition of
the grey matter. As to the white matter, in all these sections it
presented a strangely normal arrangement, there being none of the
masses of displaced or distorted fibres so often seen in artefacts of the
cord.
The sections through the cervical softening showed no distortion of white or grey matter and no loss of tissue.

*Histological examination.*—No examination was made of the cord in the fresh state; but numerous sections, fixed and stained as above-mentioned, taken from all levels of the cord, were carefully examined. And the most outstanding feature of these sections, whether from the softened portions or from the rest of the cord, seemed to be the absence of abnormal histological appearances. In none of the sections was there evidence of any inflammatory process. Even in the softened portions the vessels were nowhere found congested, nor were there haemorrhages, dilated perivascular spaces, nor exudation of leucocytes. There was no proliferation of connective tissue elements, and no increase of Deiter's cells. Any changes present were rather of the nature of degeneration and softening. Throughout the whole cord, medulla, and pons, the vessels showed well-marked colloid degeneration, and colloid bodies were likewise present, being especially abundant in the two softened areas. In neither of these areas was there evidence of definite breaking down of tissue, though in the sections through the lower part of the dorsal softening the left anterior horn (except for its ganglion cells) showed little definite structure. In the rest of the dorsal softening and in the cervical softening the axis cylinders and their myeline were in places ill-defined, and though it was difficult to make out that they were structurally altered, their staining conveyed the impression that they were at least altered in chemical composition.
No degeneration was to be made out either in ascending or descending tracts, but it must be noted that none of the sections were stained by Marchi's method.

The ganglion cells, even in the softened areas, were little altered. In the left horn of the sections represented in Figs. 2 and 3 the cells were pale, and their Nissl bodies were somewhat broken up, approaching the appearance of the so-called "ghost cells," but the ganglion cells in the right horns in these sections showed no appearance of chromatolysis. It is to be noted that the accessory portions of grey matter, seen in the sections represented in Figs. 6, 7, and 8, also contain well-stained ganglion cells. The ganglion cells throughout the cord, medulla, pons, and cortex contained quite an unusual amount of yellow pigment. Indeed, scarce a ganglion cell was to be found without some pigment, and many were entirely filled with it. This pigment was quite as abundant in the cells of the healthy cord as in those of the softened portions.

That, then, briefly is the record of the case. And it would seem to be of interest, not only as showing an abnormality in the dorsal cord, but also from the point of view of the pathology of transverse myelitis. That the softenings in the cord were ante-mortem, seems undoubted. The symptoms of the case, which were typically those of transverse myelitis, seem to prove this, and the presence of Fränkel's diplococcus is further evidence. That the symptoms were due to the softenings also seems certain, for careful examination of the peripheral nerves showed very few of their fibres to have undergone degeneration—thus excluding the idea of peripheral neuritis. The softenings were evidently not the result of an inflammatory reaction, and they must have been due either to the direct action of a virulent toxine, or else to the local occlusion of blood vessels. The action of a toxine would perhaps explain the apparent disproportion between the severity of the symptoms in the arms and the slight change to be found in the cervical cord. Certainly to the naked eye there was some swelling and evident softening in the cervical region, but microscopic examination showed but little structural alteration in this softened tissue.

It is difficult to be certain of what exactly is the nature of the accessory portion in the dorsal region of the cord. But in view of the softening and general distortion of that level, and of the loss of tissue lower down, I am disposed to regard it as an artefact. The subject of post-mortem changes in the cord is one of great interest, but one which I cannot discuss in the meantime. It has been fully dealt with by Van Gieson in the paper already referred to, and those interested in the subject should make its acquaintance.

I wish, in conclusion, to acknowledge my indebtedness to Dr. Workman, pathologist to the Glasgow Royal Infirmary, who so kindly placed this case at my disposal, and by whose permission it now appears in print.