A Possible Case of Paraplegia Caused by S. Mansoni

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The details of the following case might be published with advantage, for while the diagnosis lacked proof, the findings are sufficiently suggestive to attribute the lesion of the spinal cord to bilharzial infestation, since the patient showed considerable improvement after antimonial treatment had been given.

There are already several published reports that bilharziasis may be found in a segment of the cord, giving rise to a paraplegia. In Africa there are a few references to this possibility (Gelfand, 1950; Abbott and Spencer, 1953).

CASE ILLUSTRATION

The patient was a Coloured male from Tete, Portuguese East Africa. He was 50 years of age and first became ill about July, 1960. He first developed a sudden pain in the lumbar region whilst he was driving his motor car. The pain lasted a few days. A week after this he noticed that his legs were somewhat weak and numb and he began to experience some difficulty in emptying his bladder, being able to pass only small amounts of urine. He noticed, too, that he was having to pass urine frequently.

A year later he decided to seek medical advice in Salisbury. Here a surgeon removed his prostate, but he was no better. His lower limbs were becoming weaker and he was finding it difficult to move about. He mentioned that his feet felt "hot," but there was no pain in his lower limbs; all that concerned him really was the weakness and the feeling of pins and needles. He was otherwise well. Because of the nature of his work he often had to wade through streams in Mozambique when his car failed to pass through any of them. He was the owner of a number of small stores in the district of Tete and was fairly well off financially.

On examination, a marked weakness was found in both lower limbs, which were stiff. Clonus of the legs could be elicited, but this was not sustained. The knee and ankle jerks were exaggerated on both sides and the plantar responses were extensor. The only sensory change present was an inability to feel light touch on the soles of his feet. His sense of touch and vibratory sensation were normal. He felt pain normally in the limbs. The upper limbs were unaffected.

A lumbar puncture was carried out. The pressure of the fluid was 140 mm. Though the fluid was clear and colourless, there were five lymphocytes per c.mm. The protein was 80 mg. per cent. and the Nonne-Apelt positive.

The Wassermann reaction of the blood was negative and the blood sugar normal. The white and differential counts were normal. A myelogram was done, but no hold up was found nor anything to indicate a localised lesion. X-rays taken of the spinal column revealed nothing of note. The urine was normal and a stool specimen revealed nothing of note.

Mr. Laurence Levy, F.R.C.S., saw the patient in consultation and agreed that he was suffering from a motor type of paraplegia, the site of the lesion being in the region of D. 10, although he was not certain of its exact localisation. Mr. Levy could not explain the paraplegia. A course of deep X-ray therapy was suggested in case the patient had chronic arachnoiditis, but it was decided not to follow this course. Thus, when he was about to be discharged from hospital, it occurred to me that I should do a rectal biopsy in case a bilharzial lesion might account for his
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The rectal biopsy revealed viable ova of *S. mansoni*.

Course.—It was decided to give a full course of S.A.T., which is probably the most effective treatment for a *S. mansoni* infestation. Unfortunately his veins were quite unsuitable for intravenous injection. As I was able to give only one injection of $\frac{1}{2}$ gr. satisfactorily by this route, I decided to change over to Astiban at a dosage of $\frac{1}{2}$ gr. daily for four days by the intramuscular route. He was confined to bed and was not upset by this preparation.

There was little to note at first, but after about six days it was clear that the patient was much improved. Both the hospital sisters and the patient himself were aware that he was able to move about with greater ease and his legs were becoming stronger. At the end of two weeks he walked about without sticks; indeed, he could walk at a good speed, although there was still a spastic element about his gait. The plantar responses were now flexor, but his bladder disorder continued as before. Nevertheless he was delighted with the progress and left hospital a much happier individual. At the end of the third week after treatment it was quite clear that he had benefited from the antimony. I asked Mr. Levy to see him again and he was pleasantly surprised with the possible diagnosis of a bilharzial myelitis.

The patient returned to see me from Tete in early March, 1962, but although he was able to walk without his sticks and he could hold his urine a little better than before, he was not satisfied. I decided to repeat the rectal snip, and this time viable ova of *S. haematobium* were found. He was re-admitted to hospital and the course of Astiban was repeated. A week after the treatment he still clearly had his paraplegia, but the drag of his right leg was less than before. No sensory changes could be detected; both light touch and pinprick were unaffected, as well as sense of position. The reflexes were brisk on both sides of the body. I felt when he left for Mozambique that little more help from antibilharzial treatment could be expected, probably because irreparable damage had been done to the cord.

In June, 1962, he was seen again. He walked unaided and fairly briskly, but the gait was spastic. There were no other finds of note to record.

**Discussion**

The published records on bilharzial myelitis very interestingly stress that the onset of the paraplegia is fairly acute and rapid and rather similar to that in the present case. In Muller and Stender's case (1930) the paraplegia developed within a night, the lesions being due to *S. mansoni* in the cord at the level of D2 and D3 vertebrae. In the instance quoted by Gama and de Sa (1944) the paraplegia was of rapid onset. A myelogram showed an obstruction, and at operation bilharzial granulation tissue due to ova of *S. mansoni* was found at the level of the second lumbar vertebra. A feature of special interest in this case was that the patient responded well to treatment. Abbott and Spencer's case (1953) also showed a rapid onset in that the patient developed pains in the back followed within 24 hours by weakness first in the left leg and shortly after in the opposite one. Within a further 24 hours both legs were totally paralysed, the patient being unable to pass urine. There was complete loss of motor power with flaccid paralysis and sensory loss below the level of the eleventh dorsal segment. The cerebrospinal fluid was normal. The patient died shortly after admission, and at autopsy a mild thickening of the lumbar enlargement of the cord at the affected site was found. The lesion proved to be bilharzial due to ova of *S. mansoni*.

Abbott and Spencer, in discussing the pathogenesis of bilharzial myelitis, suggest that a wandering gravid female settles in the extradural spinal veins or the adjacent vertebral venous plexus. An established venous anastomosis exists between the pelvic vein and the vertebral venous plexuses. There is a further good anastomosis existing between the systemic pelvic veins and the haemorrhoidal vein into the spinal veins. Later, ova deposited in the extradural spinal veins leads to obstruction to the venous return. Small venous infarcts develop in the cord and later granulation tissue forms.

**Summary**

1. A Coloured male suffered from a severe paraplegia which may have been due to bilharzial disease in the spinal cord.

2. It would appear that bilharzial paraplegia has a fairly rapid onset.

**References**


