BILHARZIAL PARAPLEGIA
Report of a Case

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Very few proven cases of paraplegia from bilharziasis have been reported: Barnett (1965) could quote only fifteen cases confirmed histologically, and a smaller number of cases in which the diagnosis was presumed from a successful response to anti-bilharzial drugs.

CASE REPORT

An English girl aged fifteen years was admitted to hospital in November 1959. Three months previously she had returned from a holiday in Kenya. She complained of low back pain of increasing intensity for five days without root pain, but with paraesthesiae in both feet and weakness of both lower limbs for two days before admission. This weakness and numbness had spread upwards until it reached waist level by the time of admission. There had also been retention of urine for twenty-four hours. There was no headache, dizziness or stiffness of the neck, and no history of injury.

On examination there was no pyrexia and little disturbance of her general condition. There were no abnormal neurological signs above the tenth thoracic level. There was loss of power, tone and deep reflexes in both lower limbs. The plantar and abdominal reflexes were absent. Sensibility was impaired up to the umbilicus and was completely lost during the succeeding three days. Radiographs of the spine, including myelography, were normal.

Investigations—Haemoglobin was 13.7 grammes per 100 millilitres. The white blood cell count was 18,000 per cubic millimetre (polymorphonuclear cells 63 per cent, eosinophils 14 per cent, lymphocytes 16 per cent and monocytes 7 per cent). The erythrocyte sedimentation rate was 7 millimetres in the first hour (Westergren). Lumbar puncture showed no evidence of block. Pressure was 160 millimetres of fluid. Examination of the cerebrospinal fluid showed: proteins 190 milligrams per 100 millilitres (globulin + + ); no red blood cells; white blood cells 180 per cubic millimetre (lymphocytes 70 per cent and eosinophils 30 per cent); sugar 52 milligrams per 100 millilitres; chlorides 660 milligrams per 100 millilitres. The Kahn test and Wassermann reactions were negative.

The urine and stools did not show any parasitic ova. Because of the high level of the eosinophils in the blood and the presence of eosinophils and a high protein level with raised globulin in the cerebrospinal fluid, parasitic myelitis was suspected.

A bilharzial complement fixation test was found to be strongly positive.

Treatment and progress—Treatment was started four days after admission. Anthiomaline 2 millilitres intramuscularly each day (total 776 milligrams in seventeen days) and prednisolone 5 milligrams three times a day for six weeks were given.

The patient continued to have retention of urine and needed frequent catheterisation. Urinary infection followed, but was successfully controlled with antibiotics. Later she developed spasticity of the lower limbs, and this was followed by incontinence of urine and faeces. The following week gradual improvement of the paraplegia began, and two weeks after the start of treatment the patient was able to move her toes. By the end of five months she was walking with crutches, and had recovered sensibility almost completely. At this
stage muscle power below the knees was M.R.C. Grade 3 to 4, and above the knees Grade 2 to 3. The ankle reflex had returned but the knee jerks were absent. The plantar reflex on the left was extensor, and that on the right was equivocal. There was bilateral impairment of proprioception. The patient was fully continent.

The muscle power improved gradually with rehabilitation, so that three years after the original illness she was walking without support, but with a wide base and rolling gait. She had full power in the thigh and leg muscles on both sides, but the glutei and psoas muscles were weak. Sensibility was normal and the knee and ankle reflexes were present. Plantar reflexes remained extensor on the left and equivocal on the right.

Eight years after the original illness the only abnormal findings are slight bilateral weakness of the glutei and psoas muscles. The patient is now leading a normal life, she works as an occupational therapist, enjoys dancing and finds difficulty only in running.

**DISCUSSION**

Bilharziasis is a trivial illness if discovered and treated early but its complications, mainly of the genito-urinary system, are disastrous when the disease becomes chronic.

Involvement of the spinal cord is very uncommon. Kane and Most (1948) in a review of bilharziasis affecting the central nervous system reported up to 1944, found only four cases with spinal cord involvement. Since then cases have been reported occasionally in the literature (Gama and de Sa 1946, Raper 1948, Gelfand 1950, Pepler and Lombaard 1958).

Maciel, Coelho and Abath (1954) reported a case and in a review of the literature between 1890 and 1952 found twelve cases with cord involvement. Six of these cases were caused by Schistosoma haematobium, five by Schistosoma Mansoni and one by Schistosoma japonicum. Barnett (1965) found only fifteen histologically proven cases of paraplegia due to bilharziasis. Bird (1965) reported eight cases which he treated in two years; three of these were proved histologically at operation.

Three types of spinal complication are described. Firstly, transverse myelitis as in the cases reported by Faust (1948), Gelfand (1950), Abbott and Spencer (1953), Maciel and colleagues (1954) and Hutton and Holland (1960). Some of these were proved at necropsy. Secondly, a bilharzial granuloma with compression of the cord. This type was described by Gama and de Sa (1946), Faust (1948), Ross, Norcross and Horrax (1952) and Pepler and Lombaard (1958). Some of these cases were proved by biopsy. The third possible type is radiculitis affecting chiefly the cauda equina, as suggested by Bird (1964, 1965). Bird proved the disease histologically at operation: bilharzial ova were found in the centre of necrotic and cellular areas in the cord, roots and meninges. Schistosoma haematobium tends to produce diffuse lesions, while Schistosoma Mansoni tends to produce a localised granuloma (Hutton and Holland 1960). The latter shows as an obstructive lesion on myelography.

The route of infection is not certain. The adult worm is believed to gain access to the spinal canal through branches of pelvic veins, which anastomose with the vertebral venous plexus (Barnett 1965). Raper demonstrated experimentally that adult schistosomes can enter the spinal canal by this route. The possibility of the ova being disseminated as arterial emboli from a gravid worm after reaching the pulmonary blood stream is a mere speculation (Bird 1964, 1965).

In all cases in which the diagnosis was confirmed by histological examination the paraplegia affected the lower thoracic or upper lumbar segments. Eight out of ten of the patients of whom details were given had abnormalities in the cerebrospinal fluid. The commonest abnormalities were: 1) slight pleocytosis; 2) a small rise in protein content; 3) diminished sugar content; 4) presence of eosinophils; 5) Froin's syndrome (Raper 1948, Maciel and colleagues 1954, Pepler and Lombaard 1958). The first four abnormalities were found in our case.
The complement fixation test is one of the most reliable diagnostic tests for bilharziasis (Dunston and Pepler 1965). In our case the test gave a triple+ response, although there was no evidence of bilharzial infection of the urinary or alimentary tracts.

**Differential diagnosis**—Bilharziasis has to be considered as a possible diagnosis in cases of paraplegia of rapid onset with severe sensory and sphincteric disturbance affecting the lower thoracic and lumbar levels. This is of course especially so in areas where the disease is endemic.

Acute compressive lesions of the cauda equina usually give severe root pains which are not common in bilharziasis. Epidural spinal abscess can simulate the picture of bilharziasis, but pyrexia and toxaemia are prominent features in the former. In polyneuritis, severe sphincteric disturbance is not seen. Neurosyphilis must of course be excluded as well.

**Treatment**—Antimony preparations are the standard treatment for bilharziasis. However, it is important to remember that in its larval stage the schistosoma is not affected by these. Treatment should therefore begin four to six weeks after infestation is suspected as indicated by "swimmer's itch." In proven cases of bilharziasis of the genito-urinary or intestinal system treatment has to commence immediately. Although antimony does not affect the deposited ova, it will prevent their further production, so treatment should be started before irreversible changes occur.

Spontaneous clinical improvement may occur as the inflammatory process subsides. Steroids are always advisable in cases with neurological symptoms to counteract the inflammatory reaction and to minimise the chance of side reactions to antimony compounds (Bird 1964, 1965).

**SUMMARY**

A case of paraplegia presumed on clinical grounds to be due to bilharziasis is reported. The patient was treated with antibilharzial drugs and steroids. She has been followed up for eight years and has recovered almost completely.

The literature is reviewed, and the incidence and types of spinal lesion, the possible routes of infection and the diagnosis and treatment are discussed.

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**REFERENCES**


