Bilharziasis of the Spinal Cord

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Following agricultural development and increasing water conservation, bilharziasis is becoming an increasing problem in Southern Africa.

In the absence of adequate control measures, the problem is likely to escalate, bringing with it larger numbers of patients with severe complications.

Bilharziasis of the central nervous system has been well documented in the literature, though it has been regarded as a rare condition.

Gelfand[1] in 1950 quotes four cases from the literature and one of his own. In 1963 Marcial-Rojas and Fiol[2] analysed ninety-five cases of bilharziasis of the C.N.S. Of these, twenty-seven affected the cord.

In 1965 Bird[3] quotes nine cases of his own, dividing the lesions seen into three pathological types: myelitis, granulomatous tumour of the cord, and radiculitis, affecting chiefly the nerve roots of the cauda equina.

He also speculates briefly on the possible routes by which infestation of the C.N.S. may occur, and in addition he summarises the features of examination and special investigations which may lead to diagnosis.

A striking feature in most of the case histories is the disastrous consequences to the patient. In many, the diagnosis was made at autopsy. In others, extensive surgery has been performed under the mistaken impression that the symptoms arose from some other cause such as a prolapsed intervertebral disc. In yet others, the lengthy investigation culminating in a surprised diagnosis of bilharziasis, had allowed the pathological process to progress so far that despite eventual eradication of the disease, paraplegia with all its attendant ills remained unaltered.

It would seem, therefore, that in an endemic area such as ours, a high suspicion index for bilharziasis is the most important means of combating the disastrous results of delayed treatment.

The following two cases of possible spinal bilharziasis are therefore thought to be worth presenting.

Case 1.

J.G. is a diminutive white schoolboy aged 10 years, weighing 24 kg. He is known to be suffering from a congenitally enlarged left pulmonary artery with narrowing of the left main bronchus; thought to be the probable cause of a chronic cough since birth.

During investigations for the cough in May, 1965, he was found to have a 7% eosinophilia; but urine and stool specimens and rectal biopsy revealed no parasites. He was admitted to Gatooma Hospital on 14th June, 1965, complaining of lower abdominal pain, urinary frequency, and pain and weakness of the legs. He walked with difficulty, with a high-stepping widespread gait. Muscle power was diminished in both legs. There was a loss of sensation on the inner aspect of the thighs and in the saddle area. The bladder was distended and overflowing, and the rectum was loaded. The urine was sterile, but the stool contained numerous ova of *Schistosoma mansoni.*

After 325 mg., intravenous sodium antimonyl-gluconate over four days the signs and symptoms cleared rapidly and he was discharged, apparently well—a fortnight after admission. A week later, he was readmitted in much the same condition as before, but the bladder was filled to umbilical level and he had to be catheterised.

A lumbar puncture was performed: The fluid was microscopically clear and colourless. The pressure rose slowly to 60mm. H_2O, above which level it would not rise despite jugular pressure and straining; and there was no subsequent fall. The protein content was increased to 80 mg.%. Sugar and chlorides were normal. There were 10 red cells and one or two lymphocytes per cubic mm. Viable ova were still present in the stool.

On this occasion he was given sodium antimonylgluconate 480 mg. over six days, and in addition sulphonamides to control the catheter induced bladder infection. This resulted in improvement as on the first occasion—but further investigations were carried out after he had recovered.

Intravenous pyelogram showed normal function and anatomy of the urinary tract. Cystoscopy revealed nothing of interest apart from a recent infection and portions of calcareous casts of the catheter. There was no evidence of bladder neck obstruction or of bilharziasis.

Neurological examination was essentially unrevealing. A myelogram showed no obstruction. The cerebro spinal fluid had returned to normal—protein 50 mg.% (Pandy negative) and one lymphocyte per cubic millimetre.

From the middle of July, 1965, the patient made a rapid recovery. In March, 1966, he showed no signs of leg weakness, denied any bladder difficulty, and proudly boasted of his prowess on horseback.

In May, 1966, he felt that his legs were becoming weaker, but there was no clinical evidence of this. However, he was given a further course of antimony, to his own immediate satisfaction.

When last seen in July, 1966, he appeared perfectly fit.

CASE 2.

Mrs. A.T.M.—aged 44 years has been a keen fisherwoman in bilharzial waters for most of her life. She has had treatment with antimony for bilharziasis nine and fifteen years previously.

In August, 1966 she presented with a four-day story of rapidly increasing parasthesia and weakness of the legs, and said she was unable to feel the passage of urine and faeces, though there was no incontinence or difficulty.

Clinical examination showed a patchy loss of sensation to pin prick and light touch below the level of L.2. This was most marked below the knees and in the saddle area. Tendon reflexes were present.

Urine, stool and rectal biopsy were all free from any evidence of infection or parasites.

On lumbar puncture there was no evidence of block and the pressure was 100 mm. The fluid was slightly xanthochromic, there were four lymphocytes per c.c., and the total protein was 40 mg.%.

In the blood 8% of 9,400 white cells were eosinophils. The ESR was 14 mm. after one hour and the haemoglobin 11.8 g. The Wassermann reaction was negative.

On the day of first consultation she was admitted to hospital and treatment begun with Ambilhar. For the first two days the weakness and sensory loss progressed and for one day she was incontinent of urine. After this she improved slowly in this respect, though she suffered from headaches and vomiting which was attributed to the Ambilhar, despite concomitant treatment with anti-histamines.

On the tenth day she was well enough for discharge, though she still complained of paraesthesia below the right knee and in the saddle area.

Before discharge it was ascertained that her fluorescent antibody test was positive and that cystoscopic examining of the bladder showed no evidence of recent bilharziasis.

Frequent follow up checks since discharge have revealed no objective evidence of cord damage, such as wasting, etc., but the loss of sensation has persisted.

At the end of September she felt that the sensory loss was again spreading, so she has been given a course of Astiban which appears to have halted and partially reversed this process.

DISCUSSION:

In neither of these two cases was there incontrovertible proof of bilharziasis of the cord, though subsequent events appear to substantiate the diagnosis.

Had there been any delay in instituting treatment, there are several cases in the literature to indicate that the patients would have been either paraplegic or dead.

Treatment of the condition in the early stages is comparatively simple—for there is a response to any of the commoner methods of treating bilharziasis.

Bird[3] advocates the use of corticosteroids to minimise side reactions and to counteract allergic

reactions, though the absence of steroids in these two cases appear to have had no deleterious affects.

He also emphasises the necessity for treatment before irreversible changes have been brought about in the nervous system.

It must be emphasised therefore, that it may often be necessary to begin treatment before investigations are completed if there is the slightest reasonable suspicion. The absence of ova in bladder or bowel certainly does not rule out the possibility of spinal bilharziasis, just as it need not rule out other ectopic foci.

Findings which may, if present, add to the confidence of diagnosis include eosinophilia, xanthochromia, spinal pleocytosis of low degree, raised CSF protein, with normal sugar; and positive bilharzial blood tests.

The treatment with antibilharzial agents of other conditions giving a similar picture is not likely to obscure the ultimate diagnosis or to adversely affect the patient to any extent. On the other hand, the bilharzial pathology is so rapidly progressive and irreversible that the treatment in itself is a useful factor in differential diagnosis.

It is possible that an occasional viral infection may be confusing, in that spontaneous recovery may occur during treatment with anti-bilharzials —but this confusion may be a low price to pay for the relative certainty of preventing an irreversible paraplegia.

SUMMARY:

Two cases of probable bilharziasis of the cord are presented. As this rare condition is likely to become commoner with the increasing water conservation in Southern Africa, and as the results in the late treated cases are so tragic, a plea is made for an increased awareness of the possibility of such cases occurring and for the institution of early treatment on suspicion only if necessary.

REFERENCES

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Acknowledgments

My grateful thanks to Mr. R. M. Honey and Mr. L. F. Levy, who carried out the later urological and neurological examinations in Case 1.