SPINAL CORD COMPLICATIONS OF ACUTE SCHISTOSOMIASIS
MANSONI

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Introduction

Most textbooks of tropical medicine and parasitology describe the acute or toxaemic form of mansoni schistosomiasis as an entity with particular signs and symptoms which are distinctive and easily identified. Despite this fact, acute schistosomiasis is frequently overlooked. We have insisted, however, that it actually presents with such a diversity of symptoms and with such an astonishing mimicry that we have been able to record several mistaken diagnoses in people who were suffering from acute schistosomiasis (NEVES, 1970; 1971; NEVES et al., 1966a, b). In fact, we have demonstrated this diversity in clinical presentation, in which schistosomiasis has simulated cholera, typhoid fever, gastroenteritis, acute enterocolitis, viral hepatitis, nephrotic syndrome, acute glomerulonephritis, leptospirosis, the acute abdomen, malaria, pulmonary tuberculosis, broncho-pneumonia enterovirus infections, etc. (NEVES, 1970; 1971). This protean aspect of acute schistosomiasis reflects the difficulty in diagnosis and recalls those cases well described by HALL (1937), who recorded 50 mistaken diagnoses in patients suffering from trichinosis. The validity of this comparison is substantiated by the varied clinical picture of trichinosis documented by GOULD (1945) and described in Liverpool (KERSHAW et al., 1956) and in Kenya (FORRESTER et al., 1961).

Although several factors are undoubtedly responsible for the variability seen in the clinical course of acute schistosomiasis, the purpose of the present communication is to describe particular severe infections involving the spinal cord. These differ from those hitherto described in chronic forms of the infection in that, besides the usual signs and symptoms of the acute phase, the neurological deficit became the most prominent finding. The clinical pictures will be described in detail in order to demonstrate some peculiarities of acute schistosomiasis as it has been observed in our endemic area.

Case reports

F.B.N., a 10-year-old boy, was examined on March 15, 1972, along with 3 siblings. The 3 latter developed high fever, slight stupor, severe digestive manifestations (nausea, vomiting and relatively intense, watery diarrhoea), signs of dehydration, abdominal and muscular pains, cough, weight loss, and tender hepatosplenomegaly. In contrast to the picture observed in his brothers, F.B.N. presented with a relatively asymptomatic clinical course of an apparently identical infection. It is worthy of mention that the infection ran a different course in all members of the family. Prior to this examination, however, eggs of S. mansoni had been found in the stool of F.B.N.

On physical examination, the patient appeared well-nourished and asymptomatic. The temperature was 36.8°C.; pulse rate 80 and blood pressure 100/60 mm. Hg.; slight general lymphadeaopathy, dry tongue, distended abdomen and enlarged, tender liver...
extending 2 cm. below the costal margin. At first no abnormal neurological signs were apparent.

The patient was maintained under observation, while his brothers were receiving treatment for acute enterocolitis and allergic bronchitis related to acute schistosomiasis. However, 7 days later, intense fever, numbness, muscular pain and rapidly progressive difficulty in walking appeared. Paraparesis and transient muscular pains occurred and within 4 days there was complete flaccid paraplegia below T.11 with corresponding exteroceptive and proprioceptive deficits. Superficial and deep reflexes were absent. There were no contractures. The patient was incontinent of faeces and urine.

The history revealed that the patient and his 3 brothers had bathed in streams suspected to be contaminated by *S. mansoni* about 50–60 days before the onset of the acute disease. Immediately after bathing a slight skin reaction was present which soon vanished spontaneously. Surveys conducted in the streams in which the boys swam revealed a high percentage of snails liberating cercariae of *S. mansoni*.

Laboratory studies disclosed the following: R.B.C. 4,680,000/c. mm.; haemoglobin 13.9 g./l.; haematocrit 41%; W.B.C. 14,000/c. mm. of which 4,760/c. mm. were eosinophils; E.S.R. 49/1 hour. At the time of lumbar puncture there was no evidence of block. The C.S.F. contained 218 cells of which 60% were lymphocytes. The total protein was 50 mg./100 ml.; sugar 50 mg./100 ml.; chlorides 804 mg./100 ml. The Pandy, Ross-Jones and None-Apelt tests were positive 1+. Culture of the C.S.F. was sterile. Liver function tests revealed total serum protein of 6.0 g./%, serum albumin 3.2 g./%, globulins 2.8 g./%; cephalin-floculation 2 + ; thymol-turbidity 3.2 units; total bilirubin 0.60 mg.%; direct bilirubin 0.4 mg.%; S.G.P.T. 20 units and S.G.O.T. 10 units. Viable eggs of *S. mansoni* were found in the stool. X-rays of the chest appeared normal.

The acute illness was treated both symptomatically and specifically. When the neurological symptoms developed, a steroid (dexamethasone) was given initially. The decision was made to use the new antischistosomal agent, hycanthone. The drug was given as a single intramuscular injection of 2 mg./kg. on the 10th day after the onset of the neurological symptoms. Subsequently, steroid treatment was resumed for 30 days and the patient remained in hospital for physiotherapy. Because hycanthone failed to achieve parasitological cure (viable immature and mature eggs of *S. mansoni* in oögram about 30 days after specific treatment), the drug was repeated in the same dosage. A few days later the patient was admitted to the Institute of Rehabilitation Medicine of New York University Medical Centre. At present, the patient is having intensive physiotherapy in order to prevent deformities and to strengthen the muscles. Although parasitological cure has been obtained after the second course of hycanthone, it is still too soon to say whether any clinical improvement has been achieved.

N.M.H., a 20-year-old white woman, was admitted on December 13, 1969, with high fever, stupor, severe nausea, vomiting and watery diarrhoea, bleeding from the intestines and gums, purpura, severe dehydration, disturbance of electrolyte balance, and cachexia. Before admission, the disease was characterized by a sudden onset of high fever and shivering followed by abdominal pain, nausea, vomiting, watery diarrhoea (about 20 evacuations daily), numbness and muscular pains in the lower limbs, and productive cough. The amount of macroscopic blood in the faeces was considerable, there was progressive deterioration in the general appearance, and increase in the dehydration.

The patient and 3 companions had bathed in streams suspected of contamination with *S. mansoni* during the last days of September, 1969. Immediately after bathing, the patient's entire body was covered with punctate urticaria that vanished spontaneously.
after 48 hours. The patient’s companions were said to be receiving treatment for acute enterocolitis. Surveys revealed that the streams contained a high percentage of snails infected with cercariae of *S. mansoni*.

On physical examination, the patient appeared acutely ill and cachectic (36.5 kg.) with sunken eyes, hollow cheeks, cold and clammy skin. Temperature 38°C.; pulse rate 116; blood pressure 100/60. Purpuric and petechial eruption on the lower limbs and thorax, general lymphadenopathy, a pan-systolic mitral murmur, spontaneous gingival bleeding, dry and furred tongue, tender and distended abdomen with an extremely tender mass palpable in the right iliac fossa and tender liver extending 5 cm. below the costal margin. Neurological examination revealed a semicomatose patient with flaccid paraplegia, corresponding proprioceptive and exteroceptive losses, and incontinence.

Laboratory studies revealed the following: R.B.C. 3,340,000/c. mm.; haemoglobin 7.9 g.%; haematocrit 30%; W.B.C. 27,600/c. mm. of which 24,654/c. mm. were polymorphs (myelocytes 2%; metamyelocytes 5%; rod stab neutrophils 37%; segmented neutrophils 45%) and 3,036/c. mm. were lymphocytes. Liver function tests disclosed total protein of 5.6 g.%; serum albumin 2.4 g.%; globulins 3.2 g.%; cephalin-floculation 3+; thymol-turbidity 5.3 units; total bilirubin 3.2 mg.%; direct bilirubin 2.0 mg.%; alkaline phosphatase 11.4 units (Bodansky); S.G.P.T. 20.5 units and S.G.O.T. 17 units. Viable eggs of *S. mansoni* were found in the stool. Urinalysis showed albumin 1+, sugar 1+, pus cells 20/field, red cells 15/field, hyaline casts 2+. Blood chemistry revealed the following: K+ 4.4 mEq/L; Na+ 117.6 mEq/L; blood urea nitrogen 39.76 mg.%; creatinine 0.95 mg.%; blood sugar 113 mg.%; prothrombin content 100%; and inorganic phosphorus 2.45 mg.%. Blood cultures were negative. E.C.G. showed significant alteration of the ventricular repolarization. Endoscopy and rectal biopsy demonstrated a hyperaemic, granulomatous, friable, injected and diffusely oedematous mucosa with multiple punctate haemorrhages and shallow ulcerations. An oögram performed (85 days after the infection and 15 days after specific therapy), was negative for *S. mansoni* eggs. At the same time, histology of the rectal mucosa demonstrated the presence of shells and viable eggs in the *lamina propria*. Instead of the typical granulomatous reaction, there was a moderate infiltration with histiocytes, plasma cells, lymphocytes and scanty eosinophils. Needle biopsy of the liver performed about 150 days after the onset of the infection and 80 days after specific treatment revealed several granulomata. Endoscopy, scraping and rectal biopsy (80 and 140 days after specific therapy) on both occasions revealed granulomatous mucosa without eggs of *S. mansoni*. The remaining laboratory examinations returned to normal.

The patient initially had been admitted to the Intensive Care Centre (I.C.C.) with the diagnosis of a toxaemic form of schistosomiasis, including circumscribed peritonitis probably due to intestinal perforation, and flaccid paraplegia. Surgical intervention was judged contraindicated. Besides blood transfusion and fluid and electrolyte replacement, antibiotic therapy was maintained (chloromycetin: 2.0 g./24 hours; streptomycin: 2.0 g./24 hours) in conjunction with prednisolone 40 mg. thrice daily. Anti-schistosomal treatment was then discussed as a possible means of interrupting the vicious circle of antigen liberation and hypersensitivity of an organism not yet suppressed by hormonal therapy. The decision was made to use the new antischistosomal product hycanthone. The drug was administered in a single intramuscular injection of 2 mg./kg.

On the 2nd day after hycanthone treatment, the patient answered questions. On the 8th day she was discharged from I.C.C. Her general appearance had improved (Figure). She was no longer incontinent and the stools became free of blood. Under these circumstances, food by mouth was resumed, antibiotic treatment was stopped,
and corticoid administration was tapered off. An oögram performed on the 15th day after specific treatment revealed interruption of oviposition.

After 60 days in hospital, general improvement was slow but steady. On discharge the patient looked well and weighed 45·5 kg. (an increase of 9 kg.). When she first became ambulatory, great difficulty in walking was still present. Neurological examination demonstrated persistence of sensory disturbances in the left lower limb. Abdominal examination revealed no abnormalities, except that the liver was still palpable below the costal margin and the mass in the right iliac fossa remained palpable. The laboratory data showed a tendency toward normality. The oögram demonstrated the presence only of dead eggs of *S. mansoni*. During the out-patient examination on March 16, 1970 (100th day after specific treatment), the patient looked well but complained of numbness in the left lower limb, where sensory impairment and absence of deep reflexes were observed. 10 months after hyancanthone treatment the neurological features of the infection showed slight improvement.

W.M.M., a white boy of 11 years, was admitted on March 31, 1967, complaining of intermittent high fever, loss of weight, watery and bloody diarrhoea, and unproductive cough. In December, 1966, he had developed a generalized itch on the body after swimming 4 consecutive days in a reservoir located in an endemic bilharzia region. Despite these characteristic epidemiological data the patient was treated for several infectious diseases without improvement. On physical examination the following were observed: T. 37·8°C.; P.R. 110; B.P. 110/70; tender and slightly distended abdomen with liver and spleen extending respectively 4 and 3 cm. below the costal margin, and slight lymphadenopathy. Neurological examination revealed no abnormalities.

Laboratory studies disclosed: R.B.C. 5,300,000/c. mm.; haemoglobin 16 g./l.; haematocrit 48%; W.B.C. 22,000/c. mm. with 3,080/c. mm. eosinophils, 10,000/c. mm. monocytes and 2,200/c. mm. plasma cells; E.S.R. 25/1 hour. Endoscopy and rectal biopsy showed a hyperaemic, friable and granulomatous rectal mucosa; the oögram was positive for viable immature eggs of *S. mansoni*. Liver biopsy revealed several granulomata of *S. mansoni* in the exudative phase and degeneration of the hepatic cells. Liver function tests revealed total serum proteins of 6·5 g./l.; serum albumin 3·5 g./l.; globulins 3·0 g./l.; cephalin-floculation 2--; thymol turbidity 5-0 units; total bilirubin 0·50 mg./l.; and direct bilirubin 0.15 mg./l. X-rays of the chest showed increased density of the hilar shadow, micronodules scattered throughout both pulmonary fields, and a prominent pulmonary artery shadow. Urinalysis was within normal limits. ECG revealed no abnormalities.

During the next 7 days the patient received only symptomatic treatment. Then he was treated with niridazole, 25 mg./kg./day for 7 days. On the 4th day of this treatment he complained of urinary retention and numbness, weakness and pains in the lower limbs. At the end of antischistosomal treatment, neurological examination disclosed flaccid paraparesis with sensory loss below L.3. The deep tendon reflexes were present. Walking became impossible. The neck exhibited soreness and stiffness of the myalgic type, and Laségue's sign was positive. Lumbar puncture showed there was no evidence of block, and the initial and terminal pressures were 85 and 80 mm. respectively. The C.S.F. contained 4 monocytes/c. mm. The total protein was 97 mg./100 ml.; sugar 80 mg./100 ml.; and chlorides 760 mg./ml. The Pandy, Ross-Jones and None-Apelt tests were positive 2+. Blood examination revealed leucocytosis of 18,000/c. mm. of which 4,320 c. mm. were eosinophils and 6,840/c. mm. were lymphocytes. Liver function tests disclosed total serum protein of 8·3 g./l.; albumin 4·4 g./l.; globulins 3·9 g./l.; cephalin-floculation 4++; thymol-turbidity 4-6 units; total serum bilirubin 0·50 mg./l. with 0·17 mg./l. reacting directly.
30 days after antischistosomal treatment and the beginning of rehabilitation the patient showed marked improvement in his neurological condition. The schistosome ova disappeared from the stool. C.S.F. taken at this time contained 14 monocytes/c. mm.; total protein 53 mg./100 ml.; sugar 85 mg./100 ml.; and chlorides 643 mg./100 ml. Pandy and Ross-Jones tests were 1+. 4 months after specific treatment the patient was almost asymptomatic. Rectal biopsy and oögram showed rectal mucosa with normal appearance and no eggs of *S. mansoni*. X-rays of the chest were unchanged.

W.C.R., a 24-year-old white man was admitted on April 17, 1972, complaining of high fever, profuse sweating, severe watery diarrhoea, diffuse abdominal soreness and intense muscular pains in the lower limbs. The disease started suddenly about 40 days before admission and was treated as enteric fever with chloramphenicol, antipyretics and analgesics. After 10 days of this treatment he returned to normal activities, but 20 days later all the symptoms reappeared along with productive cough, weight loss, low back pain, and numbness, weakness and pains in the lower limbs with difficulty in walking. 50 days before the beginning of the illness, the patient was exposed to schistosome-infested streams together with 8 members of the family, all of whom were admitted to the same hospital with signs and symptoms of an apparently identical infection.

On physical examination the patient appeared well-nourished but pale with temperature of 38.2°C., a pulse rate of 100 and blood pressure of 100/60 mm. Hg. Examination disclosed these findings: distended and tender abdomen; slight hepatomegaly with tenderness; and no lymphadenopathy. The rest of the physical examination was within normal limits except for the following pertinent neurological signs: stiff neck, positive Kernig and Lasègue's signs, flaccid paraparesis with sensory loss below L.2 and superficial and deep reflexes virtually absent in the lower limbs. There was no incontinence of bowel or bladder. The patient and his family did not permit lumbar puncture.

Blood examination revealed these values: R.B.C. 4,800,000/c. mm.; haemoglobin 15.0 g.%; haematocrit 43%; W.B.C. 13,200/c. mm. with 2,640/c. mm. eosinophils and 2,640/c. mm. lymphocytes. Liver function tests showed total serum protein of 7.2 g.%; cephalin-floculation 3+; thymol-turbidity 8-8 units; thymol-floculation 3+; alkaline phosphatase 64 mg./ml.; S.G.P.T. 20 units, and S.G.O.T. 14 units. Viable and immature eggs of *S. mansoni* were found on examination of the stool. Endoscopy and rectal biopsy demonstrated a hyperaemic and friable rectal mucosa with slight haemorrhages; the oögram was negative for mature, immature and dead eggs of *S. mansoni*. Liver biopsy revealed several foci of hepatitis with intense eosinophilic exudation, and focal necrosis. A few schistosome granulomata were seen in the fragment. X-rays of the chest were normal. The patient appeared well and was treated both symptomatically and specifically. Hycanthone was used in a single dosage of 2 mg./kg. on the 10th day after admission. Within 10 days the patient appeared well, still complaining of severe muscular pain and numbness in the lower limbs with consequent difficulty in walking. Other signs and symptoms had disappeared. The leucocyte and eosinophil counts returned to normal (7,000 c. mm. and 420 c. mm.). 30 days after specific treatment the patient was asymptomatic. At examination on the 60th and 120th days after antischistosomal treatment, the patient was clinically cured, and no eggs of *S. mansoni* were found in the oögrams.

M.T.A., a 15-year-old girl, was admitted on February 2, 1969, complaining of fever and progressive difficulty in walking. The disease started 10 days previously with a general and transient erythematous dermatitis followed by high fever and pain in the lower limbs. A few days later, the slightest passive movement or pressure applied to lower limb muscles caused acute discomfort. On the day before admission, when she arose from bed her legs collapsed beneath her. Other symptoms included slight swelling of the lower limbs, abdominal pain and watery and bloody diarrhoea.
Although the epidemiological data pointed initially towards poliomyelitis or the Guillain-Barré syndrome, it was later discovered that the patient and 7 brothers had bathed in streams about 50 days before the onset of the disease. In addition, all the companions were admitted to hospital with different clinical features of acute schistosomiasis.

On physical examination the patient appeared well-nourished with a temperature of 38.2°C, a pulse rate of 108, and a blood pressure of 100/70 mm. Hg. The abdomen was slightly tender with the liver extending 5 cm. below the costal margin and the spleen barely palpable on deep inspiration. The neurological examination revealed paraparesis, sensory loss below L3., virtual absence of superficial and deep reflexes in the lower limbs, absent plantar responses, and a positive Lasègue’s sign.

Laboratory examination disclosed: R.B.C. 4,500,000/c. mm.; haemoglobin 13·3 g.%; haematocrit 42%. W.B.C. 8,000/c. mm. of which 4,480/c. mm. were eosinophils and E.S.R. 70 mm. 1 hour. Endoscopy and rectal biopsy showed a friable, oedematous and hyperaemic rectal mucosa with a marked granulomatous appearance. In the òogram were found immature, mature and dead eggs of *S. mansoni*. Liver function tests demonstrated total serum protein of 7·7 g.%; serum albumin 3·9 g.%; serum globulins 3·8 g.%; cephalin-floculation 3--; thymol-turbidity 4·5 units; thymol-floculation 3--; total bilirubin 0·29 mg.%; and direct bilirubin 0·18 mg.%. On liver biopsy schistosoma granulomata in the necrotic exudative phase were present. Blood culture was negative. X-rays of the chest were normal.

The acute illness was treated symptomatically. The decision was to use neither steroid nor antischistosomal therapy. However, physiotherapy was promptly begun, and on discharge, 20 days after admission, the patient appeared well and complained only of slight difficulty in walking. 4 months later the patient was asymptomatic despite moderately tender hepato-splenomegaly. Specific treatment with niridazole was performed 3 months later with success.

Discussion

Although the clinical course and pathology of acute schistosomiasis are subject to a considerable amount of controversy, some aspects of the protean nature of its clinical picture may be found in the literature. It was from Tel-el-Kebir that LAWTON (1918) first described the early “toxic” features associated with *S. mansoni* infection in Egypt. Symptoms included fever, urticaria, abdominal pain and diarrhoea. Clinically, some cases resembled typhoid fever, while others resembled bronchopneumonia. Later, in one way or another, many authors referred to the peculiarities of the early clinical syndrome of schistosomiasis (FAIRLEY, 1916; HOUGHTON, 1923; GIRGES, 1934; HERNANDEZ-RODRIGUES, 1935; PIFANO and MAYER, 1942; MAGALHAES and ROCHA, 1947; PEREIRA and SIMON, 1954; RUIZ-RODRIGES, 1956; DIAZ-RIVERA et al., 1956; NEVES, 1965, 1970, 1971; NEVES and RASO, 1963, 1965; NEVES and TONELLI, 1970; NEVES et al., 1965, 1966a).

Based on our personal experience to date, among more than 200 cases of acute schistosomiasis we have recorded several varieties in which the disease presents many features in common with other infectious conditions commonly observed in tropical regions. In these circumstances we have stressed the importance of fully investigating the possibility of exposure to schistosomiasis before considering cholera, typhoid fever, acute abdomen, pulmonary tuberculosis, bronchopneumonia, leptospirosis, viral hepatitis, bacillary dysentery, amoebiasis, malaria, acute glomerulo-nephritis, nephrotic syndrome, enterovirus infections etc. Another feature in which our records differ from those referred to in the literature is the extreme severity of many infections which have
ultimately caused death of some patients (NEVES and RASO, 1963, 1965; NEVES et al., 1965).

In the present cases, however, one must acknowledge the difficulty in making the diagnosis of acute schistosomiasis if the patients were considered alone. It is reasonable to question the probability of schistosomiasis being responsible for such dramatic manifestations.

The neurological manifestations of chronic schistosomiasis are well known, both clinically and pathologically. In this respect, a relatively long list of references indicates the prevalence of nervous system involvement by ectopic schistosomiasis. Epilepsy (TAVARES, 1935; PINTO and ALMEIDA, 1945, 1948; REZENDE BARROS, 1946); delirium (PINTO and ALMEIDA, 1945); spastic paraplegia (ABBOT and SPENCER, 1953; CASTAIGNE et al., 1959); headache, dizziness and vertigo (MONTEIRO DE BARROS et al., 1956); sensory and vasomotor disturbances (MACIEL, 1930); Korsakoff's psychosis (TAVARES, 1935); meningitis (PONDE, 1942); myelitis (RAPER, 1948; DOMINGOS and BORGES, 1964); hemiplegia (LYRA, 1945); paraplegia (COITO and COSTA, 1949; CANELAS et al., 1951); cord compression (GAMA and MARQUES DE SA, 1944, 1945; ROCHA and ROEDEL, 1952; ROSS et al., 1952; GAMA, 1953; MARTINEZ-NOCHET and POTENZA, 1956; WILLEMIN-CLOG et al., 1957; BUDZILOVICH et al., 1964; BIRD, 1965); and transverse myelitis (MULLER and STENDER, 1930; MACIEL et al., 1954; ABBOT and SPENCER, 1953; MARIOL-ROJAS and FIOL, 1963; SCAFF et al., 1971; OLIVEIRA and ALENCAR, 1964). A more dramatic case was reported by one of us (RASO et al., 1964) describing both clinically and pathologically a massive cerebral haemorrhage due to ectopic depositions of eggs. On the other hand, ectopic localization of schistosomiasis in the nervous system apparently may produce no symptoms. We detected the presence of several ova and granulomata of *S. mansoni* in the denticulate nucleus of the cerebellum and in the basal nucleus of a patient who presented with no neurological manifestations.

With regard to the present cases, some facts must be stressed again to insure a firm understanding of the neurological changes described.

(a) One of the patients exhibited a clinical picture very similar to that of the Guillain-Barré syndrome; the neurological manifestations were transient and cure was not dependent upon the use of corticoid hormone or antischistosomal therapy.

(b) In 2 other cases, the neurological lesions improved after specific treatment (hycanthone). One patient had exhibited paraparesis, the other, paraplegia, semicoma, and signs and symptoms of intestinal perforation.

(c) The sudden appearance of a picture suggesting nerve-root involvement was seen in a patient during niridazole treatment, the symptoms being transient and cure apparently resulting from the physiotherapy.

(d) Another patient developed signs of sudden transverse myelitis with flaccid paraplegia below T.11; the antischistosomal treatment apparently did not influence the course of the neurological lesion.

Concerning our present knowledge, relatively little detailed information is available on nervous system involvement in the early stages of schistosomiasis. The fact is that the pathological basis of acute schistosomiasis in man remains quite unknown. Our present ideas have been based upon the experimental data of animal research. Under these circumstances, it must be recognized that in our cases the appearance of spinal cord involvement, by whatever mechanism, was contemporaneous with involvement of other organs and systems, and upon the organism as a whole. In other words, among the various clinical manifestations of specific tissue damage and total organism response, the
spinal cord involvement became the most important aspect in some cases and was less important in others.

Noteworthy in the present cases is the absence of any correlation between the intensity of constitutional symptoms and the severity of neurological involvement. Severe general reactions of the organism such as the toxacemic state, intestinal perforation, coma and cachexia were followed by a mild and reversible spinal cord injury; a relatively benign general reaction did not preclude the sudden appearance of transverse myelitis. Corticosteroid hormone and antischistosomal treatment, which appeared to be beneficial in some patients, were unable to prevent or minimize paraplegia in another patient, while in yet another patient, clinical features of neurological involvement were detected during the course of specific therapy with niridazole.

**Figure.** (a) The patient 30 days after discharge from the Intensive Care Centre; there were still signs of cachexia, paraparesis and incontinence of bowel. (b) Same patient 140 days after the completion of specific treatment; note the improvement in general appearance and absence of hepato-splenomegaly.

Based on our experience (Neves, 1970, 1971; Neves and Raso, 1963, 1965; Neves et al., 1965; Neves and Tonelli, 1970; Santiago et al., 1965), we must emphasize that various organs and systems other than the central nervous system may be predominantly involved in the course of acute schistosomiasis. As a matter of fact, when an inexperienced physician makes an incorrect diagnosis it is generally because one of these dominant focal reactions has made the clinical picture atypical.
It is not relevant to speculate on the different routes by which the schistosome eggs may reach the nervous system. Although several possibilities have been raised (Greenfield and Pritchard, 1937; Batson, 1940; Raper, 1948; Potenza, 1955), the fact is that the ova are carried and deposited in the nervous system, as well as in other tissues, and may cause either different kinds of reactions (Bogliolo, 1958; Neves, 1965; Neves and Raso, 1963, 1965; Neves and Tonelli, 1970) or little or no immediate reaction (Budzilovich et al., 1964). With regard to acute schistosomiasis, one of its most striking anatomical features is the miliary dissemination of ova throughout the body particularly in the liver, lungs, intestines, peritoneum and pancreas. On the other hand, study of autopsied patients (Bogliolo, 1958; Neves and Raso, 1963; Neves et al., 1965) has shown the disproportion between the intensity of the miliary distribution of granulomata in tissues and the number of worms. With this in mind, it is not possible to explain the disease as resulting exclusively from the action of the eggs.

It is logical to assume that the diversity of the immune response of the host generally accounts for the multiplicity of forms and types with which the disease presents in clinical practice. Fairley (1916, 1920, 1951) apparently accepted this view, for he demonstrated the mechanical action of worms and their eggs in tissues, their secretion or excretion whilst alive of a "toxic" substance and their release of products of disintegration after death. It was Fairley's belief that the "anaphylactoid reaction in the sensitized tissues of the host plays an important part in the production of both pathological lesions and the clinical picture".

To sum up, the nervous system involvement observed in these cases must not be considered to result exclusively from the mechanical action of eggs and worms and the resultant granulomata formation. One may assume that an anomalous response of the nervous system to the immuno-allergic products derived from dead worms and/or their eggs may have brought about distinct clinical manifestations. At any rate in the patients who recovered, either spontaneously or by means of corticosteroid and antischistosomal treatment, the mechanisms of spinal involvement, because it was reversible, must have been less severe, and the direct effect of eggs and granuloma therefore were probably not the most important factors.

Bird (1965) stated that corticosteroids should be administered to every patient with schistosomiasis and neurological symptoms, to counteract the allergic response and to minimize the damage caused by side-reactions to the specific compounds. He also suggests that for treatment to be effective it must be instituted before irreversible changes have occurred within the nervous system. Based on the present cases, however, it is difficult to say whether specific treatment was beneficial or whether the products derived from the massive death of worms might have aggravated the tissue reaction. From the cases reported herein no conclusion can be drawn.

Summary

5 cases of clinically unsuspected involvement of the spinal cord by S. mansoni are reported. In contrast to the cases usually described in the literature, the nervous system involvement was observed during the acute phase of the infection. One of the patients exhibited a clinical picture closely resembling that of the Guillain-Barré syndrome, and cure was not dependent upon the use of corticoid hormone or antischistosomal therapy. 2 other patients improved after neurological involvement (paraparesis and paraplegia) following the completion of specific treatment (hyacanthone). The sudden appearance of a polyradiculoneuritis syndrome was observed in 1 patient during treatment with niridazole. In another patient who developed a sudden transverse myelitis at T.11
with flaccid paraplegia, antischistosomal therapy apparently did not influence the course of the neurological process.

It is suggested that the nervous system involvement in the reported cases cannot be explained entirely by the mechanical action of eggs and worms and the resultant granuloma formation. In the authors’ opinion an anomalous response of the nervous system to the immuno-allergic products derived from dead worms and/or their eggs probably was responsible for the clinical manifestations of spinal cord involvement.

REFERENCES


