South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

Cape Town, 17 December 1960 Volume 34 No. 51 Deel 34 Kaapstad, 17 Desember 1960

A PARASITIC INFESTATION INDISTINGUISHABLE FROM TRICHINOSIS — A NEW DISEASE ?

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As far as we have been able to ascertain, infestation by trichinosis has not previously been reported within the Union of South Africa. We present here what we believe to be the first instance of a trichinosis-like disease, or a disease which is clinically indistinguishable from trichinosis, contracted in South Africa. We make this statement because the final laboratory diagnosis of trichinosis is lacking, and there is no other way of proving the presence of the disease.

CASE REPORT

History

Mr. D.N., aged 24 years, a medical student, said he was well until 12 October 1959, when his face became extremely itchy. He did not notice any swelling. Very soon after this, while writing examinations, he noticed that he was unable to concentrate, nor could he crystallize his thoughts in a coherent manner. He then consulted Dr. S. Dubb in Cape Town, who later very kindly supplied us with some notes on his condition. Dr. Dubb saw him on 9 November 1959, at which time he complained of not having felt well for the previous 2 weeks. He also lacked energy and was persistently tired. A mild cough had been present for the previous 2 - 3 weeks. He had also noticed slight epigastric pain unrelated to meals for 1 week. The bowels had not acted for 5 days. He complained of painful fingernails with 'pigmentation' of 2 days' duration. The fingers had been swollen for 1 day. He had felt febrile for the first few days of the illness and considered that he had a recurrence of malaria which he had had previously. There was no complaint of muscle pain. On examination, Dr. Dubb noted the following features: The patient did not look ill and did not have a raised temperature. His fingers were swollen and the swelling was of a fusiform type affecting the proximal interphalangeal joints. They were not red, sore, or tender. There was an unusual rash on the buttocks, fairly profuse, with small scattered patches on the trunk. It was described as resembling a papular urticaria. The papules were 3 - 4 mm. in diameter, raised and not itchy. The patient was unaware of the rash. Splinter haemorrhages were present in the fingernails. The rest of the physical examination was non-contributory.

The patient was referred to the medical out-patient department at Groote Schuur Hospital on 10 November 1959. There is no certainty about what happened at the hospital. It would appear that in a state of mental confusion the patient went home to his lodgings without apparently having been seen at the out-patient department. There he remained for approximately 1 week. He was unable to give an account of what took place during that period. He had vague memories of profuse sweating and of changing his pyjamas and bedclothes. On one or two occasions he thinks he staggered from his room in order to get food, but nothing is certain.

On 17 November he drove himself from Cape Town to Johannesburg and saw one of us (L.L.) on 18 November. He claimed that he had no complaints but that his mother had referred him for examination because of the haemorrhages under the fingernails (Fig. 1). His speech was slurred, and apart from haemorrhages under the finger- and toenails, the only other abnormal physical signs found were in the central nervous system. A blood count and urinalysis were done and he was referred for further opinion. He was examined by one of us (R.M.) on 19 November. He denied having any physical complaints apart from some lethargy, and insisted that mentally he was his usual self. His memory for events leading up to the time he left for Johannesburg was imperfect and without the information we obtained from Dr. Dubb we would have been unable to fill in the gaps in his previous history. Direct questioning revealed the fact that a few days previously he had noticed blurring of vision and difficulty in focusing. There had also been flashes of light in the visual fields. This phenomenon persisted for a few days after his admission to a nursing home on 20 November.

Past History

As a child he had measles, mumps, whooping cough, chicken pox and boils. An appendicectomy was done in 1954 and in May 1958 he had contracted malaria in the Livingstone area. This had recurred in England in August 1958.

Physical Examination

The blood pressure was 110/70 mm.Hg. The only abnormal physical signs were those of the nails and the central nervous system. There were profuse splinter haemorrhages under all 10 fingernails as shown in Fig. 1. To a lesser degree they were present under the toenails.

Fig. 1. Showing the extensive splinter haemorrhages under the fingernails. (Note — the curved 'black-brush' borders under the distal ends of the nails are the haemorrhages.)

The following physical signs were found in the central nervous system: all the reflexes, including the jaw jerk, were hyperactive, the abdominal reflexes were absent, the right plantar response was flexor and the left, equivocal; there was a marked tremor of the right hand; fine movements were poor on both sides; coordination, as shown by the finger-nose test, and dysdiadokokinesis were poor on both sides; the left hand wandered with the arms outstretched; there was pseudo-clonus of both ankles, the left being more marked than the right; there was questionable hyperaesthesia to pin-prick on the right hand; the palate failed to move properly; Romberg's sign was strongly positive and, when it was elicited, the patient fell

over backwards on each occasion; and the visual fields were full to rough testing.

The urine contained neither albumin nor sugar.

Electrocardiography

The initial tracing (Fig. 2A) run on 19 November 1959 showed inverted T-waves in the left ventricular surface leads and gross depression of the ST-segments. The standard limb leads suggested a developing posterior myocardial infarction. A tracing done 2 days later showed similar changes which were slightly more marked (Fig. 2 B). By 30 November 1959 the ECG suggested a well-established posterior myocardial infarction (Fig. 2 C). Subsequent tracings done on 31 December 1959 and 28 January 1960 showed resolution of the pathological process. This was almost complete by 18 April 1960 (Fig. 2 D).

LABORATORY INVESTIGATIONS

18 November 1959

Venous blood. Haemoglobin 14·3 g. per 100 ml.; packed cell volume 39%; leucocytes 16,600 per c.mm.; polymorphonuclears — neutrophil 22·5%, eosinophil 58·5% (9,700 absolute count), basophil 0·0%; monocytes 3·5%; lymphocytes 15·5%; platelets — numerous; erythrocyte sedimentation rate (Wintrobe) 48 mm. in 1 hour; 'corrected' sedimentation rate 33 mm. in 1 hour; malaria parasites were not seen in thick or thin films. There was a marked eosinophilia. The stained red and white cells showed no abnormality.

Urinalysis. Microscopic examination of the centrifuged deposit showed amorphous material only. Casts, crystals, erythrocytes, inflammatory cells, and schistosoma ova were not seen.

The *bilharzia complement fixation test* was negative.

20 November 1959

Radiology. X-rays of the skull and chest, a soft tissue exposure, and an anteroposterior view of the thighs were all negative.

Cerebrospinal fluid. The fluid was clear and colourless, cells 1 lymphocyte per c.mm., pressure 130 mm. water, no block, total protein 46 mg. per 100 ml., glucose 49 mg. per 100 ml., chlorides (as sodium chloride) 710 mg. per 100 ml., Lange's colloidal gold test gave 0000000000 precipitation, the Kolmer cardiolipin Wassermann test was negative, neither trypanosomes nor torula bodies were observed in preparations from the cerebrospinal fluid.

Blood serum. Thymol turbidity 2 units, thymol flocculation negative.

Urine. Urobilin present (not in excess), bilirubin absent.

21 November 1959

Venous blood. Haemoglobin 14·5 g. per 100 ml.; packed cell volume 43%; mean corpuscular haemoglobin concentration 34%; leucocytes 9,900 per c.mm.; polymorphonuclears — neutrophil 29·5%, eosinophil 47·5% (4,700 absolute count), basophil 0·0%; monocytes 4·0%; lymphocytes 19·0%; platelets—numerous; erythrocyte sedimentation rate (Wintrobe) 44 mm. in 1 hour; 'corrected' sedimentation rate 40 mm. in 1 hour. A marked eosinophilia was present. Neither atypical nor immature cells were found in either series. Prothrombin index 98%.

23 November 1959

Stool. Brown and formed; microscopic examination of direct and concentrated preparations showed no amoebae, protozoa, cysts or helminth ova. Inflammatory exudate was not seen.

25 November 1959

Bilharzia complement fixation test. Negative.

Further Venous Blood Tests

On *18 December 1959* the total white-cell count was 8,300 per c.mm. and the eosinophils were 29·0% (2,400 absolute count). The erythrocyte sedimentation rate (Wintrobe) was 11 mm. in 1 hour. There was no other particular change from previous findings.

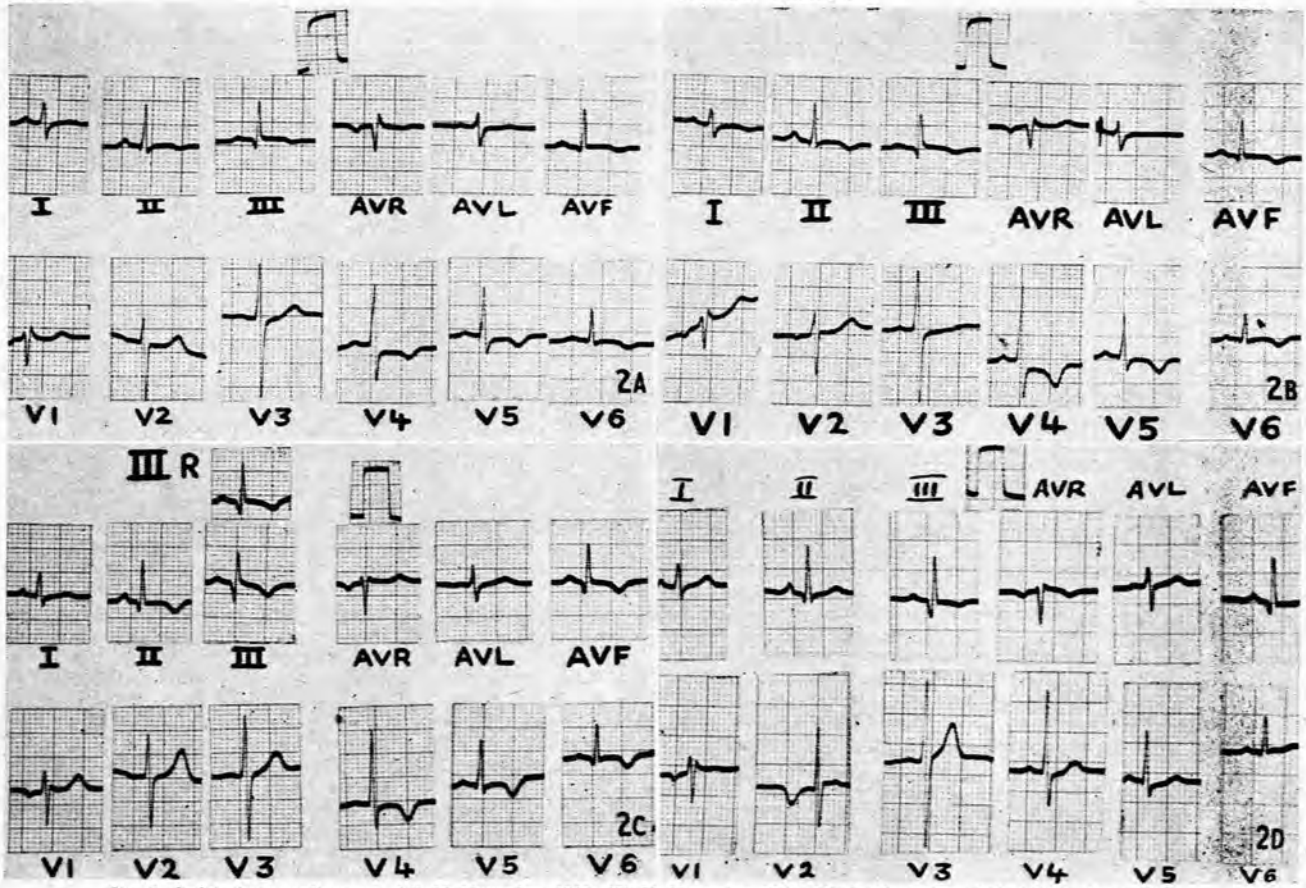

Fig. 2. Serial electrocardiograms. (*A*) 19 November 1959. (*B*) 21 November 1959. (*C*) 30 November 1959. (*D*) 18 April 1960.

On *29 January 1960* the total white-cell count was 10,100 per c.mm. and the eosinophils were 22·0% (2,220 absolute count). The erythrocyte sedimentation rate (Wintrobe) was now down to 6 mm. in 1 hour. There was no other particular change from previous findings.

Blood serum was sent to the USA, and on 27 January 1960 Dr. Irving G. Kagan, Ph.D., chief of the Helminthology Unit, Microbiology Section, Communicable Disease Centre, Chamblee, Georgia, USA, reported that he had tested the specimens of sera with the bentonite flocculation test, which they performed as a routine in their laboratory for the diagnosis of trichinosis infestation. Both samples were negative.

On *2 March 1960* the total white-cell count was 8,100 per c.mm. with 15·5% eosinophils (1,260 absolute count). Thus there was still an eosinophilia present. There was no other change of note in the blood count.

TREATMENT AND SUBSEQUENT PROGRESS

In view of the probable diagnosis of trichinosis and the favourable effects reported in this infestation from steroid therapy, it was decided to adopt this form of treatment. The patient was given 'meticortelone', starting with a dosage of 30 mg. daily, which was gradually tapered off to zero over a period of 2 weeks. After this he received injections of ACTH to prevent any rebound phenomena.

He was re-examined on 31 December 1959. He had gained 8 lb. weight, which he had previously lost. The reflexes were still very brisk, especially the jaw jerk. Dysdiadokokinesia was still present, and so was the tremor of the right hand. He said that he felt perfectly well. He was seen again on 28 January 1960 when he stated that his right hand was shaking more than previously but he thought that he had had this tremor from birth. He said that his thought processes were perfectly clear. He was asked to subtract 7 serially from 100 and accomplished this feat without error but in twice the time taken by the examiner to do so. He had some exertional dyspnoea but no complaint of chest pain. He was re-examined by one of us (G. McL.) who concurred in the diagnosis of trichinosis. The patient was last seen on 18 April 1960. At that stage he denied having any physical complaints. Re-examination revealed that the tremor of the right hand was still present and that the abdominal reflexes could be elicited only with difficulty. The jaw jerk was absent and again it was noted that the palate failed to move. It is likely that this represents an old defect. The electrocardiogram was repeated before and after exercise and no ischaemic changes resulted.

DISCUSSION

From the outset it was apparent that our patient was suffering from a parasitic infestation and as trichinosis had not been reported in the Union of South Africa it took some time to realize that this was the possible diagnosis. Two main factors weighed in the determination of which parasite was the cause of his illness. Firstly, we knew that for about 8 months, in 1957 and 1958, he had been in the Livingstone area of Rhodesia as a surveyor. Here, undoubtedly, he had been exposed to such tropical parasites as malaria, bilharzia, ankylostomiasis, and possibly others. Secondly, after repeated questioning, he gave a history of having had some pork a short while before taking ill on 12 October 1959. In view of the persistently negative bilharzia complement fixation tests, the fact that he had remained well since leaving Livingstone militated against the probability of his disease having been contracted at that time. As he had become ill in Cape Town it was felt that trichinosis was certainly a possibility, in that infected meat, contraband or otherwise, may have reached the restaurant at which he had his meals. Certain other diagnoses, such as periarteritis nodosa, were considered, but subsequently tests, clinical findings, and the patient's clinical improvement made them highly unlikely.

As we are describing a disease clinically indistinguishable from trichinosis we feel it is justifiable to summarize the pathogenesis of this disease. The parasite infesting our patient clearly follows a similar life cycle in the human host.

TRICHINOSIS

Trichinosis is caused by *Trichinella spiralis*. The parasites gain entry to the human body when raw or inadequately cooked pork containing the encysted larvae is eaten, most commonly in the form of sausage. Encystation of the larvae takes place in the stomach or duodenum and the larvae immediately enter the superficial layers of the mucous membrane of the duodenum or jejunum. In 3 - 4 days the worms mature and mate, and by the fifth day the female begins to discharge living young. At first some larvae escape into the lumen of the bowel and are evacuated in the faeces, but as the females migrate nearer to the muscularis mucosae, the larvae gain entrance to the mesenteric lymphatics and venules in increasing numbers and are carried throughout the body, finally settling down in the skeletal muscle, where they soon become encysted. In the human body this ends their life cycle, although they may remain viable for many years. In the reservoir host, such as the hog, the encysted larvae constitute the source of infestation for the next host. Thus, in nature, a susceptible host harbours all the stages in the life cycle of the worm, but the infestation cannot be propagated unless the infested flesh of that host is eaten raw by the next host.[1]

Clinical Course

The usual clinical course of a case of trichinosis may be divided into 3 stages:

1. *The stage of invasion.* This occurs during the first few days after infestation and there are clinical manifestations simulating acute food poisoning, such as nausea, vomiting and diarrhoea. There may be a bright macular or papular eruption on the skin.

2. *The stage of infiltration* is characterized by excruciatingly painful myositis resulting from inflammatory reaction wherever the larvae pass through the muscles. In this stage, spastic paralysis of the muscles of the extremities is not unusual.[3] There is also oedema, especially around the eyes, nose and temples. Thrombi in the vessels of the viscera or extremities may occur, and there may be adenitis, encephalitis and meningitis, ocular disturbances, deafness, and many other functional disturbances indicating the protean character of the lesions.

3. *The stage of encystation.* Cecil mentions cachexia, toxic oedema, nervous disorders of almost any type, and a toxic myocarditis resulting from the damage produced by trichina larvae migrating through the myocardium.[1] There may also be pneumonia, peritonitis, pleurisy and acute nephritis.

Note: Naturally most clinical cases are milder and the majority of patients have infestations below the clinical grade.

Diagnosis

The diagnosis is usually suspected from the clinical features of the disease plus the high blood eosinophilia. However, additional proof may be obtained by doing a muscle biopsy. The muscle fibres which are obtained are digested for a few hours in artificial gastric juice with the liberation of motile larvae. An intradermal test is also described and there are precipitin and complement fixation tests which are quite specific.[2]

Differential Diagnosis

Roberts[2] mentions the fact that trichinosis has masqueraded as rheumatoid arthritis, eosinophilic leukaemia, typhoid fever, meningitis, rheumatic fever, dermatomyositis, and periarteritis nodosa. He states that the search for conjunctival petechiae and splinter haemorrhages under the nails can prove to be most rewarding and goes as far as to say that they are almost pathognomonic of trichinosis. The only other condition likely to be confused in this respect is sub-acute bacterial endocarditis.

In this particular case we also considered trypanosomiasis,

bilharzia, filaria, malaria, lues, onchocercosis, ascariasis, leptospirosis, coxsackie virus, ECHO virus, lympho-choriomeningitis, toxoplasmosis, and diffuse demyelinizing disease.

CONCLUSIONS

Taking into consideration all the positive features shown by this patient, we feel that the diagnosis of trichinosis is justified except for the negative trichina antigen test. Our patient ate pork and some time later took ill with itching of the face, swelling of the fingers, and splinter haemorrhages under the nails which were more profuse than any ever encountered in sub-acute bacterial endocarditis. He had what appears to have been the typical rash of trichinosis and there followed a period of mental symptoms succeeded by evidence of central-nervous-system disease and acute myocarditis. It is true that there was never any evidence of muscular pains or myositis but we feel that the lack of this symptom is probably due to the lack of infestation of somatic muscle. In his case the brain and heart bore the brunt of the infestation. We do not know if he would have recovered equally well without the use of corticosteroids, but we feel that in such cases they should not be withheld as they may be life-saving.[2] In any event his clinical recovery has been complete.

SUMMARY

1. A case is described with clinical and laboratory features indistinguishable from those of trichinosis with the exception of the specific antigen test.

2. The disease process was manifested by a diffuse infestation involving mainly the extremities, brain and heart, and producing in the heart the picture of symptomless myocardial infarction.

3. The result of adrenocorticoid therapy appeared to be beneficial.

4. The patient has made a complete clinical recovery, but the eosinophilia persists.

5. The exact nature of the parasite has not been determined.

We thank Dr. J. H. S. Gear, Director of the South African Institute for Medical Research, for his cooperation in attempting to elucidate the diagnosis, and for sending the patient's serum to the USA.

Our thanks are also due to Mr. A. M. Shevitz of the Photographic Unit, Department of Medicine, University of the Witwatersrand, for the pictures.

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PROGNOSIS OF 'NUTRITIONAL' HEART DISEASE IN THE BANTU

REPORT OF A CASE OF CHRONIC REVERSIBLE HEART FAILURE

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The course of 'nutritional' heart disease described by Gillanders[1] is characterized by frequent relapses leading ultimately to death. That this apparently inevitable progression may not necessarily be the fate of all these patients, can be suspected from the discrepancy between the incidence in the wards and the necropsy room. Clinically, I recorded[2,3] 'nutritional' heart disease ('idiopathic cardiac hypertrophy', 'cryptogenic heart disease,' or 'African myocardopathy') as being the commonest cause of heart disease in the Johannesburg Bantu population; it was responsible for 37·5% of 275 cases of heart disease admitted to my unit at Baragwanath Hospital in 1957. Surprisingly, Higginson *et al.*[4] found it ranked only fourth in the causes of death from heart failure, forming 14·9% of 537 cases. It might be submitted that the diagnosis was inaccurate; however clinico-pathological correlation in individual cases does not support this contention. A more likely explanation is that a number of patients recovered and naturally enough ceased to attend the out-patient follow-up clinic. A chance encounter with an ex-patient afforded me the first proof that this form of heart failure was reversible. Seftel[5] has recorded reversal of a similar condition associated with pregnancy, but these cases had suffered only a single attack of congestive failure; those that relapsed remained in failure or died.

CASE REPORT

First Admission

D.W., a Xhosa male aged 43, was admitted to Baragwanath Hospital in October 1953 in congestive heart failure. He had been born in Queenstown, Cape Province, but had lived in Johannesburg since 1930. A month previously, troublesome palpitations on exertion and a cough productive of a small amount of colourless sputum had started. He had never noticed blood-staining of the sputum. For 2 weeks before admission he had become breathless on walking a few yards and was aware of swelling of the ankles and pain in the right hypochondrium. He slept propped up on 3 pillows but had not suffered from paroxysmal nocturnal dyspnoea. Systematic enquiry revealed that his diet was adequate and regularly included meat. He had been in hospital previously for a minor injury only. There was no history of joint pains.

On examination the jugular venous pressure was elevated to the angle of the jaw, when the patient sat up at 45°. The heart size was clinically indeterminate, the second sound at the pulmonary area was louder than that at the aortic, a rough systolic murmur was audible at the left border of the sternum, and a gallop rhythm was heard towards the (presumed) apex of the heart. There was no right-ventricular lift. Crepitations were heard at the base of each lung and there were signs of bilateral pleural effusion; ascites and oedema were demonstrable. The blood pressure in the right arm was 116/100 mm.Hg and in the left, 120/96 mm.Hg. The liver was tender and the edge palpable 4 fingerbreadths below the costal margin in the nipple line. The pulse was regular and of small amplitude, and the rate was 110 per minute. Fluoroscopic examination on the sixth hospital day showed that the cardiac pulsations were not active.

The patient was treated with bed-rest, low-sodium diet, mersalyl injections and digitalis. The gallop rhythm persisted for 4 days and the patient rapidly lost the signs of congestive failure and was discharged to the out-patient clinic. The urine had contained a trace of albumin, but no sugar was found in it on admission. The standard Eagle test for syphilis was positive, serum albumin was 2·9 g. and the globulin 4·5 g. per 100 ml.

Second Admission

The patient was readmitted in August 1954, again in con-