While corticosteroid therapy was maintained in both our cases, methandienone was introduced, and improvement occurred. In the first patient muscle-weakness regressed in a slow but sustained fashion over a period of months, whereas in the second patient the improvement was more dramatic, and was evident in the first 10 days of treatment. While we may have observed the natural remission of the disease in these patients, its direct relationship to the start of anabolic hormone suggests that it was drug-induced. We consider that anabolic hormones were of value in the cases described and are worthy of further trial in dermatomyositis.

Summary

Two cases of dermatomyositis are described. Although the results of corticosteroid therapy were disappointing, improvement occurred in both cases after the use of methandienone, an anabolic hormone.

We are grateful to Dr. J. Laurie for his helpful criticism. We thank Dr. A. L. Scott for the reports on the sections, and Mr. A. A. Jenkins, who prepared the photomicrographs. We are indebted to Dr. C. D. Falconer, of Ciba Laboratories Limited, for a generous supply of dianabol.

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SCHISTOSOMIASIS OF THE SPINAL CORD

REPORT OF A CASE

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Schistosomiasis of the cord has been infrequently reported. It is therefore considered worth while reporting a further case.

Kane and Most (1948) reviewed the literature up to 1944. They recorded 24 cases of cerebral complications of schistosomiasis, of which 14 were due to *S. japonicum*. Two authenticated cases of myelitis due to *S. haematobium* and two due to *S. mansoni* had been described during this period. They comment on the tendency of *S. japonicum* to affect the brain and of *S. mansoni* and *S. haematobium* to affect the cord. Since their paper further cases of spinal-cord involvement have been reported sporadically.

Gama and de Sá (1946) record a case operated on for suspected spinal tumour. A cystic swelling at the second lumbar segment was removed from which *S. mansoni* eggs were isolated. The neurological signs were not affected. Raper (1948) records the finding, at necropsy on an African soldier, of a schistosome

in a dense scar compressing the thoracic cord. Two years previously the patient had been offered but had refused surgery for a suspected spinal-cord tumour. Gelfand (1950) reported a personal case in which *S. haematobium* eggs were found in the cord. He comments on the frequency in which schistosome ova of both species are found on digestion of the brains of cases of schistosomiasis, but found ova only in one of the 25 cords examined. Ross *et al.* (1952) report a case of *S. mansoni* of the cord discovered at operation for suspected tumour. The patient improved after operation and antimonial therapy. Horrax *et al.* (1958) describe a case of *S. mansoni* granuloma of the cord which was operated on for suspected spinal tumour.

There are at least four further cases of cord involvement with schistosomiasis in the South American literature not available to us.

Case Report

A European man aged 73 had been a pilot in the first world war. After a flying accident in 1918 he had had an arthrodesis of his right hip and an ankylosis of the right knee-joint. Despite this he had been very active and had actually been playing tennis not very long before the present illness. In July, 1958, he fell and sustained severe bruising of his back and left hip. After this his wife thought he did not walk so well and that his left foot turned in more. Before his arrival in Uganda he had spent some time in Southern Rhodesia and then some months in Tanganyika.

Ten days before admission he had motored up 1,400 miles (2,250 km.) from Tanganyika, driving himself all the way. He complained of stiffness and cramps in his legs. He attended the out-patient department. No abnormalities were noted. A white count at this time was normal. Five days before admission he had another fall; he was unable to get up and was helped into bed. He had some difficulty in moving his legs and had pain in his legs and lower abdomen, but attributed this to the fall.

After this he found difficulty in passing urine and had to strain. The same day he had an involuntary movement of the bowels. Two days before admission he developed retention with overflow. The day before admission he became completely unable to move his legs.

On admission he was an elderly alert man. He had a blood-pressure of 180/100. C.N.S.: The cranial nerves and upper limbs were normal. There was a band of hyperaesthesia to pin-prick from D9 to D12. He stated that he felt as though he had an iron band round his abdomen. There was a motor paresis of the lower extremities, more marked on the right than on the left, although fixation of the right knee- and hip-joints made testing difficult. There was a loss of all forms of sensation below D12, except for some sparing on the inner side of the right thigh and left calf, and some diminution of sensation to light touch in D11 and D12.

The abdominal reflexes were brisk. The knee- and ankle-jerks were not elicited, nor were the plantar responses. The bladder was distended, with a dribbling overflow, and the rectal sphincter was toneless. The prostate was normal. There was a suggestion of a kyphus at L3 to 4, and there was tenderness over the lumbar spines. A white count showed 10,000 cells, of which 78% were neutrophils. There were no eosinophils at this time. The C.S.F. showed a normal pressure with a free rise and fall on jugular compression. The fluid was clear, contained 90 mg./100 ml. of protein, and 55 lymphocytes per c.mm.; the sugar was 46 mg./100 ml., and the Khan reaction was negative.

A further C.S.F. three weeks later showed a protein of 10 mg./100 ml., 15 lymphocytes per c.mm., and a sugar of 14 mg./100 ml. This low figure, which was checked, was not due to delay in examining the fluid.

Urine showed no protein, sugar, or abnormal cellular deposit. The test for Bence Jones proteose was negative.

X-ray examination of the spine showed marked calcification of the thoracic and abdominal aorta. There was an old crush fracture of the body of L2 and an old anterior displacement of L5 on the sacrum. No secondary deposits were seen.

The paraesthesiae round the abdomen gradually disappeared. The sensory level seemed to fluctuate a little, but there was virtually no alteration of sensation in the legs below L2. The patient began to complain bitterly of shooting pains going down the legs to the knees and later to the ankles.

There appeared to be some recovery of power in the lower limbs. Six weeks after admission a muscle chart showed power ranging from 3 in the hip extensors and quadriceps to 0 in the peronei and tibialis anticus on the right side, and 0 in the flexor longus digitorum and hallucis on the left.

The illness was complicated first by a bladder infection, which was overcome, and then six weeks after admission he developed a cellulitis in the perineum followed by sloughing, and had a large ulcer in the sacral region. This was followed by spreading pressure-sores over both trochanters.

The patient's general condition deteriorated after this and his memory became poor. He slept a good deal of the day, though when questioned he complained bitterly about pains in the legs.

Attempts were made to cover the pressure-sores with pedicle grafts. The first was successful, but much bleeding occurred on the second occasion. Difficulties were experienced in the grouping of blood to transfuse him. He became very anaemic, and the grafts broke down despite eventual transfusion. His general condition, which had greatly deteriorated, did not improve; he became progressively more comatose, and died 10 months after admission.

A white count a month before death showed an eosinophilia of 21% of 13,000 cells.

Necropsy Findings

External Examination.—The body was that of an emaciated elderly male. There were extensive pressure-sores over sacrum and bony prominences of lower limbs. There was an old operation scar over the right hip and the right knee-joint was fixed and enlarged.

Internal Examination.—There was no evidence of recent injury to the vertebrae. Examination of the brain and spinal cord showed no macroscopic abnormality, and specimens were taken for histological examination. Atheroma of the cardiovascular system was moderate except in the coronary vessels, where there were some very thickened plaques, and these vessels were unduly dilated. Consolidation of the lower lobe of the left lung was present and there were some adhesions. The bladder was inflamed. All other organs appeared normal.

Histological examination of sections of the lumbar and lower thoracic cord showed giant cells and infiltration with lymphocytes and some neutrophils and many schistosome ova. The variety of schistosome was not positively identified, but one terminal-spined ovum was seen in section. Some ova appeared to have been recently deposited. There was also some degeneration of the tracts. No abnormality of the brain was seen on histological examination.

Discussion

Various diagnoses were entertained at different times. Initially the possibility of secondary deposits or myeloma of the vertebral bodies appeared possible, but the negative x-ray findings and lack of evidence of cord compression on lumbar puncture seemed to exclude this. The diffuse flaccid paralysis with sensory disturbance, with a suggestion in the history that the fall was a result of weakness rather than the cause of it, suggested the possibility of an ascending myeloradiculitis.

The actual diagnosis was never entertained; nothing in the history, apart from possibly his complaint of

burning micturition, suggested that this patient had suffered from vesical schistosomiasis. Original routine urinary findings were normal, and the subsequent bladder infection obscured, even at biopsy, the possibility of detecting vesical schistosomiasis. The only pointer towards the possibility of bilharzia was a subsequent white count showing a fair degree of eosinophilia.

The findings at necropsy showed a rather diffuse granulomatosis of the lower dorsal and lumbar cord. In some of these granuloma schistosome eggs were visible. In Fig. 1 there would appear to be a terminal-spined ovum, and in Fig. 2 there is an ovum which is

FIG. 1.—A schistosome egg showing a terminal spine lying in a granuloma in the cord.

FIG. 2.—High-power view of three schistosome eggs in the cord. The spine of one is well shown.

either an *S. haematobium* broken open and the spine displaced or an *S. mansoni* egg. It was, however, not possible to identify positively whether this was an egg of *S. haematobium* or *S. mansoni*, though the appearance is perhaps a little in favour of the former. Further attempts to digest the fixed cord to identify other ova were not successful. The lesions appeared to be of very different ages, some of long standing and some comparatively recent. In addition to the granulomata there seemed to be considerable degeneration of tracts.

The ova appear to have been deposited diffusely over quite a large section of the cord. The factor determining the onset of the acute symptoms is not clear. Possibly at some point there was a critical disturbance of the cord blood supply in this region, or possibly there was a reaction of an allergic nature consequent upon the deposition of ova in the cord itself. Elaborate neurohistology was not possible, and the pathological findings do not shed further light on this.

It is not difficult to conceive how bilharzia eggs can reach the spinal canal from an infection with *S. haematobium* or *S. mansoni*.

Batson (1940) and Franks (1953) have shown that there are communications with the common iliac and inferior vena cava from the vertebral venous plexus veins. They have shown that raising the intra-abdominal pressure will cause blood from the iliac veins to pass into this system, and that this is the probable route taken by secondaries to the cord in carcinoma of the prostate.

Coman and deLong (1951) showed experimentally that they could induce tumour growth in this situation by suitable injection of a rat tumour into the iliac vein with the vena cava compressed. One of these tumours actually grew in the spinal cord.

That adult schistosomes themselves may enter the spinal canal, presumably by this route, has been demonstrated by Raper (1948).

Cases described in the literature suggest either a localized lesion with cord compression or a rather diffuse lesion as in the case described.

The one might be thought to be due to a pair of adult schistosomes ovipositing in veins in close proximity to the spinal canal, and the other as due to a rather diffuse and irregular bombardment of the cord via the vertebral venous plexus, a state that would more easily arise in *S. haematobium* infections. In fact, in view of its situation it might give rise to this complication of cord involvement more often than *S. mansoni*. That it does not do so more frequently is perhaps a little surprising. Gelfand (1950), looking for eggs, found that only one out of 25 cords of bilharzia cases showed ova, whereas they were common in the brain of these cases.

From reports in the literature there is a distinct suggestion that *S. mansoni* tends to produce a localized granuloma whereas *S. haematobium* produces a more diffuse lesion.

Diagnosis during life is not easy; schistosomiasis is widespread in many areas, yet this complication appears to be a rarity. The only cases where diagnosis has been established during life are those operated on for a suspected spinal tumour. Obrador (1948), discussing the diagnosis of cerebral cysticercosis, has mentioned that the two most helpful findings were a low sugar in the C.S.F. and an increase of eosinophils in the C.S.F. Conceivably the same condition might obtain in schistosomiasis. An increase of cells was noted in the C.S.F. of this case, but eosinophils were not specifically looked for. It was also noted that the sugar content in one specimen was unaccountably diminished.

Clearly schistosomiasis must be borne in mind when considering obscure spinal-cord lesions in those who may have been exposed to a schistosome infection at some time.

We are grateful to the Director of Medical Services, Uganda, for permission to publish this paper.

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RAPID ESTIMATION OF FIBRINOGEN IN OBSTETRIC CASES

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Patients who have had a concealed accidental haemorrhage, a death *in utero*, amniotic fluid embolism, or premature separation of the placenta are prone to severe haemorrhage associated with a defect of blood coagulation (Weiner *et al.*, 1950; Reid *et al.*, 1953). This is thought to be due to a reduction in the fibrinogen level of the blood (Dieckmann, 1936), and while theories about the causation of the defect are various (Weiner *et al.*, 1950; Albrechtsen *et al.*, 1955; Ratnoff *et al.*, 1955), the response to fibrinogen therapy has now been proved by experience (Weiner *et al.*, 1950; Reid *et al.*, 1953; Barnett and Cussen, 1954; Scott, 1955).

The clinician faced with a patient in one of these categories wishes to know as quickly as possible whether she has a deficiency of fibrinogen or whether the haemorrhage has a purely obstetric origin. A simple coagulation-time will give some information, but does not indicate anything short of a complete absence of fibrinogen and does not really enable any quantitative control of treatment to be maintained. For the most satisfactory handling of such a case a rapid estimation of the fibrinogen level is required in order that the progress of the patient may be followed and the treatment planned to meet the patient's needs. In practice it is important to decide when it is necessary to give fibrinogen and at what point in labour or the puerperium it can be used with most advantage, since it should not be administered unless really required. Of the simple methods available a straightforward coagulation-time will not always reveal the true condition of these patients, and tests which use an excess of thrombin to clot samples of whole blood (Scott, 1955) will do no more than indicate the presence of a severe defect and give no help in deciding when to intervene in the less serious cases. It is, however, of use in circumstances where it is not possible to carry out other tests.

The methods at present available for estimating fibrinogen are as follows: (1) Kjeldahl, (2) turbidimetric estimation of fibrinogen after precipitation by ammonium or other salts, (3) colorimetric estimations of fibrinogen after coagulation by thrombin or calcium salts, (4) electrophoretic methods, and (5) methods based on the addition of thrombin to varying dilutions of plasma to form a fibrin clot—for example, Rosenberg's (1956) technique.

The first four methods, though variable in their accuracy, are chemically satisfactory, but take far too long for practical use in these types of cases. The precipitation methods reviewed by Podmore (1959) can give variable results, and are dependent on such extraneous factors as the correct amount of anti-coagulant being used. Methods based on clot formation by thrombin with varying dilutions of plasma—for example, Rosenberg's technique—are quick and can be calibrated to give a useful and known range of accuracy.