Cysticercosis of the dorsal cord

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The pig tapeworm has a cosmopolitan distribution and is seen in places where improperly prepared pork and pig by-products are eaten. It is found in some European countries but mostly has been eradicated by public-health control; it is still seen commonly in Asia and South America.

The mature parasite lives in the human jejunum and obtains its nourishment from the intestinal contents of the host. It is attached to the gut wall by a scolex or head, which is fastened in the submucosa by a halo of hooklets. Distally, there are a large number of developing and ripe segments which are cast in the stools, liberating large numbers of eggs.

The larval stage of the parasite is passed usually in the pig but in areas where human food becomes contaminated by excreta, eggs may be swallowed. In Asia where 'night soil' is a common addition to the soil for growing vegetables this is an ever-present risk, unless strict preventive measures are taken to avoid infestation. Hatching in the gut, the larvae pass into the circulation and to the tissues as emboli. Where they come to rest they excite an irritative tissue response and are usually walled off by a dense layer of fibrous tissue. Sometimes they calcify and this becomes an aid to their location.

The symptoms they cause depend on their position and number. In the nervous system multiple or single foci are not uncommon in the brain itself where they are known to cause focal epilepsy or mimic cerebral neoplasms. Accounts of disease of the spinal cord are very rare indeed. The last published account was from Lima, Peru, in which two cases of intramedullary disease are described (Cabieses, Vallenas, and Landa, 1959). In 1936, López and Feijóo reported a case which they said was the third case diagnosed during life in Spain. Lima Costa, Figueiredo, and Puig Serra (1957) reported one case from Brazil together with two cases of cerebral disease.

The diagnosis may be made in the known presence of infestation elsewhere, haemo-eosinophilia and neurological deficit. The presence of eosinophils in the cerebrospinal fluid may be taken as strong evidence of parasite disease. In the present case none of these factors was present and the diagnosis was not made before operation.

It is thought reasonable to report it as the relatively large tumour which was found had produced no interference with cord conduction.

CASE REPORT

The patient was a 22-year-old Nepalese who was admitted to hospital elsewhere in the middle of October 1963. He complained of vague malaise of one week's duration accompanied by an ill-defined pain in the interscapular region centred at the spinous process of the sixth dorsal segment. There was no fever or systemic illness. The erythrocyte sedimentation rate was 5 mm./hr.; total white cell count 8,600 (polymorphs 77%, lymphocytes 21%, eosinophils 2%); haemoglobin 107%. Serological tests for syphilis were negative. The urine was normal.

A week later he developed meningism but remained afebrile. X-ray films of the skull were normal. Lumbar spinal puncture produced slightly xanthochromic cerebrospinal fluid at normal pressure with no spinal block demonstrated by Queckenstedt's manoeuvre. Analysis of the fluid showed a moderate number of red cells, protein 1,200 mg., chlorides 765 mg., and sugar 74 mg. per 100 ml., and an excess of globulin. Wassermann and Lange tests were negative. Subsequent culture grew no tubercle or other organisms. At this time there was a short-lived retention of urine which passed off with the other symptoms in the next two days. Signs of meningeal irritation recurred after a week and a further spinal puncture produced clear fluid at normal pressure: protein 240 mg. per 100 ml., four red cells, 320 white cells (90% lymphocytes), and excess globulin. Further radiographs taken of the spine and pelvis did not contribute any further information.

He continued to show intermittent signs of meningeal irritation with severe headaches and neck stiffness. Repeated examinations of the cerebrospinal fluid showed persistently high protein values. Other investigations continued to be normal. The last of these acute episodes was recorded at the end of March 1964.

He was transferred to Singapore in the middle of May 1964 where he was admitted to the Orthopaedic Unit with persistent pain and tenderness at the mid-dorsal level. There were no neurological signs and repetition of his previous investigations did not help. A presumptive diagnosis of intraspinal tumour was made and ascending myelography undertaken. A specimen of cerebrospinal fluid at that time contained 135 mg. of protein per 100 ml. but analysis was otherwise unremarkable. The column of opaque dye ran freely up to the level of the fourth dorsal vertebra but from there to the

FIG. 1. The pre-operative myelogram showing the filling defect. This picture was taken with considerable cephalic tilt and the reluctance of the contrast medium to pass the obstruction is seen.

dorso-cervical junction it would only trickle along the right-hand border of an apparently long filling defect with an upper limit at the level of the last cervical vertebra (Fig. 1).

On 15 June 1964 laminectomy was performed. The extradural space was normal and the dura pulsating. Opposite the third dorsal vertebra an intradural tumour could be felt and on opening the dura this was seen to lie in the cord. The cord was incised vertically to reveal a white, nodular, firm tumour which was dissected free from cord tissue without great difficulty and removed intact. No attempt was made to aspirate the tumour before removal.

The mass was lobulated and was at least half the diameter of the dorsal cord in each direction. On microscopy it was found to consist of a mass of granulation tissue infiltrated by plasma cells, collections of foamy

macrophages, and masses of polymorphs which were forming microabscesses. This was surrounded by a dense fibrous layer. In the middle of the tumour were cystic spaces lined with reticulum cells and one of these contained a larva of *Cysticercosis cellulosae*. Within this larva were a number of small, oval structures resembling ova but which were calcareous corpuscles. Serial sections of the larva demonstrated an organized structure forming part of the scolex (Figs. 2, 3, and 4).

Following operation both legs were markedly spastic and urine was retained but bladder control returned within a few days. The spasticity progressively reduced and the patient was encouraged to walk. There was at first gross ataxia but this also improved more rapidly than was anticipated. Six months later he was discharged from care with minimal unsteadiness of gait. No infestation was ever demonstrated in the stools or elsewhere in the body.

DISCUSSION

If it had been possible to establish a firm diagnosis in this case it might have been justifiable to delay operation in the hope that the lesion would become walled-off by fibrous tissue, with spontaneous resolution of symptoms. This would have avoided operative risks to nervous conduction. However, this was hypothetical and without a diagnosis, operation was necessary. That there was no pre-operative neurological deficit is interesting. It is recognized that the lesion had not invaded nervous tissue but the amount of displacement might reasonably be expected to have compressed at least the most sensitive elements in the confined limits of the upper dorsal canal.

SUMMARY

A case of cysticercosis of the upper dorsal cord is described. This is noted to be a very rare occurrence

FIG. 2. A general view of the parasite. $\times 35$.

FIG. 3. *An enlarged view from Figure 2. $\times 64$.*

FIG. 4. *Further enlargement showing the scolex. $\times 160$.*

and particularly remarkable in this instance because of the lack of neurological signs. The patient recovered after operative excision of the lesion and cord function was only minimally impaired.

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