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CASE RECORDS
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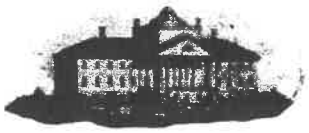

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 40-1977

PRESENTATION OF CASE

A 62-year-old right-handed man was admitted to the hospital because of progressive dementia.

The patient was a native of the Cape Verde Islands, from which he emigrated to this country at the age of 52 years. There was a history of severe head injury in his twenties that resulted in coma for eight days. Convalescence was prolonged, but he finally improved and was able to return to work as a civil servant fluent in Portuguese and English. Ten years before admission he had a seizure that involved the right extremities, followed by a brief period of speech difficulty and confusion. Five years before entry he was admitted to another hospital, where he was found to have mild hypertension. Neurologic examination was reported to be negative. Laboratory studies were negative except for mild hyperglycemia. X-ray films of the skull, an electroencephalogram and a radioactive brain scan were reported to be normal. A lumbar puncture disclosed no abnormality except that the cerebrospinal-fluid protein content was 56 mg per 100 ml. Four years before admission another right-sided seizure occurred, and there was recurrence at intervals of six to 12 months thereafter. During the year before entry the patient's English deteriorated, and his son observed that his Portuguese was imperfect.

Six months before admission there was the onset of progressive loss of memory and worsening of speech, with impairment of use of the right extremities. Five weeks before entry he returned to the same hospital because of persistent symptoms. Repeated examination disclosed no abnormality. An automated scan of a blood specimen gave normal values except that the glucose ranged from 186 to 266 mg per 100 ml. An electrocardiogram showed incomplete right-bundle-branch block. An x-ray film of the chest and films of the skull were reported to be normal. An intravenous pyelographic examination, a barium-enema examination and an upper gastrointestinal study were negative. A computed tomographic study (CT-scan) of the brain disclosed multiple areas of decreased density surrounded by haloes of increased density; the left lateral ventricle was considerably enlarged and displaced toward the left. A lumbar puncture revealed an initial pressure of 240 mm and a final pressure of 175 mm of water; the cerebrospinal fluid was clear, colorless and acellular; the protein was 90 mg per 100 ml; microscopical examination of a cell-block specimen showed no tumor cells. The patient was referred to this hospital.

There was no history of a prolonged febrile illness, abdominal disease or myalgia.

The temperature was 36.7°C, the pulse 80, and the respirations 16. The blood pressure was 120/75 mm Hg.

On examination he appeared well. The head was normal; the carotid pulses were ++, and no bruit was heard. The lungs, heart, abdomen and extremities were normal. Neurologic examination disclosed that he was alert and walked carefully with a cane in the right hand. He had difficulty understanding and speaking English and Portuguese; there were occasional brief pauses in his speech, but no seizure activity was observed. The cranial nerves were intact. There was slight drift of the outstretched right arm. Muscle strength was mildly to moderately diminished in the right upper extremity and diminished to a lesser extent in the right lower extremity. There was less spontaneous use of the right side than of the left; the patient stood well on the left leg but not on the right. Pinprick, light-touch and vibratory sensation were less well perceived on the right side than the left. Attempts at testing graphesthesia and stereognosis were unsuccessful. The finger-to-nose test was well

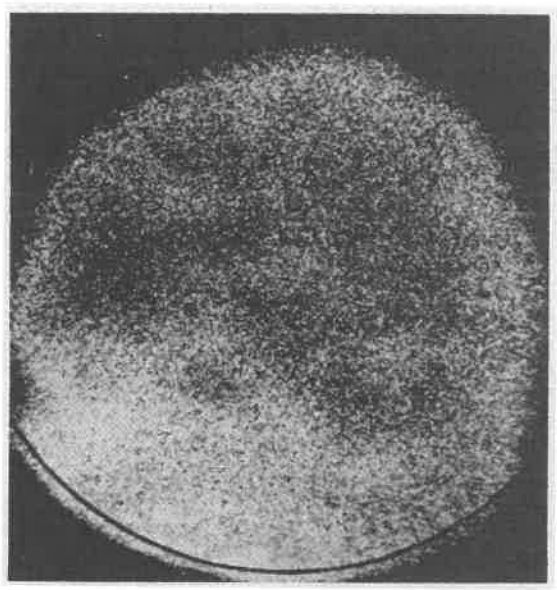

Figure 1. Radionuclide Brain Scan, Left Lateral View, Showing Multiple Ill Defined Areas of Abnormally Increased Activity.

performed bilaterally, with a slight tremor on the right side; the Romberg test was negative. The tendon reflexes were ++, and the plantar responses flexor.

The urine gave a ++++ test for glucose; the sediment contained rare white cells per high-power field. The hematocrit was 41 per cent; the white-cell count was 8100, with 53 per cent neutrophils, 38 per cent lymphocytes, 8 per cent monocytes and 1 per cent metamyelocytes. The platelet count was 270,000. The urea nitrogen was 14 mg, the creatinine 0.9 mg, the fasting glucose 175 mg, the bilirubin 0.2 mg, the calcium 9.5 mg, the phosphorus 3.3 mg, and the protein 7.4 g (the albumin 4.2 g, and the globulin 3.2 g) per 100 ml. A serologic test for syphilis was negative. An electrocardiogram demonstrated incomplete right-bundle-branch block, old inferior myocardial infarction and left atrial enlargement. An x-ray film of the chest revealed minor pleural thickening along the right posterior chest wall; the lungs, heart and mediastinum appeared normal. On films of the skull the pineal gland was not calcified, and the bony calvarium appeared intact; a prominent density was observed in the region of the right maxillary sinus; the mastoid air cells were not well aerated, and the remaining paranasal sinuses appeared normal. A radionuclide brain scan (Fig. 1) showed multiple areas of focally increased activity throughout the head, believed to be intracranial. A CT-scan (Fig. 2) disclosed enlargement of the left lateral ventricle, with slight shift of the midline to the left; scattered throughout the brain were multiple areas of increased attenuation surrounded by ringlike areas of increased density; the lesions were most concentrated

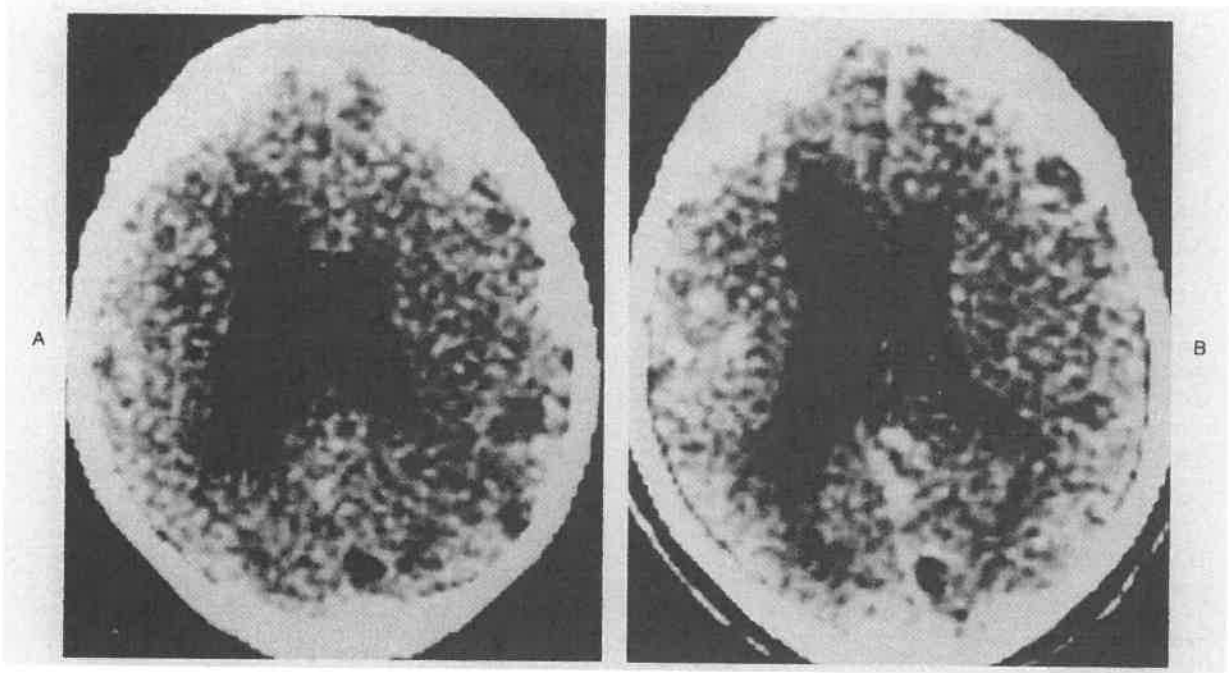

Figure 2. CT-Scan, without Contrast Material (A), at the Level of the Lateral Ventricles, Demonstrating Numerous Widely Distributed Ovoid Areas of Low-Absorption Value within the Cerebrum.
Slight right-to-left midline shift is evident. The CT-scan with contrast material (B) shows peripheral enhancement of the lesions.

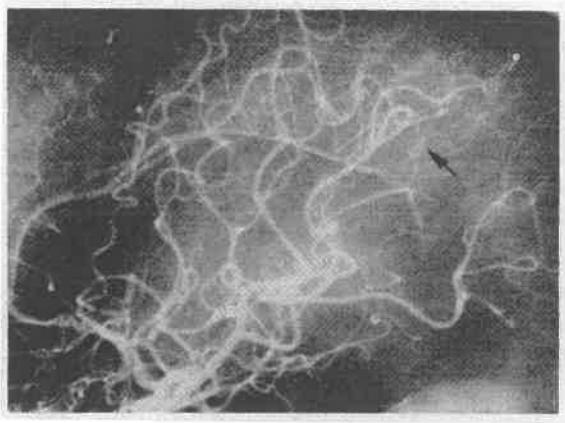

Figure 3. Right Carotid Angiogram, Lateral View, Taken at Three Seconds, Showing Slow Flow and a Cluster of Abnormal Vessels in the Parietal Region (Arrow).

in the right parietal, temporal and occipital areas, with a single lesion in the left parietal region and another in the left occipital pole. A ^{99m}Tc diphosphonate bone scan demonstrated increased activity over the right ethmoid and maxillary sinuses; tracer material was otherwise normally distributed throughout the bony skeleton. A bilateral carotid angiographic study (Fig. 3) revealed slow filling and emptying of the superior division of the right middle cerebral artery, with diminished perfusion in the anterior segment of the middle cerebral artery; the anterior cerebral artery was slightly shifted to the left. There was delay in venous drainage in the right frontal region, with a slight shift of the middle cerebral vein from right to left. On the left side there was evidence of slow filling and emptying of the anterior segment of the middle cerebral artery; the anterior cerebral artery was shifted to the left, along with the internal cerebral vein. Both lateral ventricles appeared slightly enlarged. There was no evidence of endarteritis, neovascularity or tumor. A ^{99m}Tc sulfur-colloid liver-spleen scan disclosed that the liver and spleen were normal in size, configuration and uptake. Xeroradiographic examination of the limbs showed no soft-tissue lesions. An electroencephalogram revealed occasional left-sided slow activity. A microcytotoxicity assay of the patient's serum gave a negative response for astrocytic tumor. A tuberculin skin test (intermediate-strength PPD) was positive. Microscopical examination of a stool specimen disclosed no ova or parasites.

An operation was performed on the 19th hospital day.

Differential Diagnosis

Dr. James A. Schnur*: It is tempting to begin by suggesting that the diagnosis in this case must be a chronic and doubtless exotic disease in a middle-aged man who became ill after leaving his tropical homeland for the United States. The case also affords us an opportunity to consider certain disorders of the central nervous system that we do not ordinarily encounter and appraise the role of imaging studies as they supplement clinical data in the diagnosis. The neurologic abnormality was clearly a slowly progressive process involving the cerebrum. More particularly, the right hemiparesis and dysphasia strongly implicate the left hemisphere. The dementia could well reflect bihemispheric disease. The seizures suggest a disorder of gray matter. The long-time residence in the Cape Verde Islands, the remote head trauma and the diabetes mellitus may all be relevant.

At this point it is appropriate to turn to the radiographic and nuclear-medicine studies. A lateral view of the skull reveals neither intracranial calcifications nor evidence of long standing increased intracranial pressure. The radionuclide brain scan (Fig. 1) demonstrates numerous areas of ill defined increased activity indicative of multifocal cerebral-hemisphere involvement and suggestive of an active rather than a quiescent process.

The CT-scan (Fig. 2) is germane. It shows evidence of a diffuse right hemispheric mass, as reflected by slight compression of the right lateral ventricle and shift of the ventricles and midline structures toward the left side. In addition, there is expansion of the third ventricle and of the left lateral ventricle due to either loss of cerebral substance or, more likely, obstruction. Of particular interest is the finding of multiple round to ovoid areas of abnormally reduced absorption values (slightly higher than that of cerebrospinal fluid), measuring no more than 15 mm in diameter. The distribution is largely but not entirely peripheral. The individual lesions are surrounded by rims of higher absorption value (slightly greater than that of gray matter), which are minimally enhanced after the administration of contrast material.

The left carotid angiograms demonstrate slowed blood flow. The opercular branches of the left middle cerebral artery appear stretched, largely as a result of expansion of the left lateral ventricle. The right carotid angiograms (Fig. 3) demonstrate slow flow and a cluster of small, tortuous abnormal vessels in the midparietal region. The frontal projection confirms the CT-scan impression of a subtle midline shift from right to left.

At this point we must resist the temptation to draw too precise a pathological inference from these findings. The lesions of low absorption value demonstrated on the CT-scan do indeed have the appearance of cerebrospinal-fluid-containing cysts surrounded by capsules of higher absorption value, but this is a histopathologic inference and not a fact that can be deduced from the scan. Moreover, the nature of the apparent capsules, whether gliotic, granulomatous or microcalcific, is not evident from this study. The pathological correlate of contrast-produced enhancement on CT-scans remains an enigma. Whether this

*Radiologist, Peter Bent Brigham Hospital; assistant professor of radiology, Harvard Medical School.

phenomenon reflects abnormal vascularity, an active inflammatory response, uptake by disordered tissues or some other process awaits resolution. Finally, unless lesions are of sufficient size and have disparate absorption values they may easily be missed on a CT-scan. Thus, in this case we can only approximate the number of separate lesions within a broad range. With respect to the category of this patient's disease, it seems safe to exclude cerebrovascular, demyelinating, metabolic, toxic and degenerative disorders. A biologically benign, diffuse cystic primary neoplasm is exceedingly unlikely. Old trauma is of interest only insofar as resultant malacic change might have rendered this diabetic patient more susceptible to a chronic infectious process, fungal or parasitic, of the brain.

Before turning to specific disorders, I might point out that we have little evidence of extraneural systemic disease on either clinical or radiologic grounds. In addition, the results of the lumbar puncture five weeks before admission to this hospital are not particularly helpful. The opening pressure of 240 mm was only minimally elevated and could in part have been related to faulty technic. The protein value of 90 mg is intriguing, but in a diabetic patient a protein of 50 to 100 mg per 100 ml is not unusual. The reported absence of cerebrospinal-fluid pleocytosis is puzzling.

With respect to specific disorders, the diagnosis of tuberculosis with caseating granulomas of the meninges or tuberculomas of the brain is not substantiated on pathological, clinical or radiologic grounds. The possibility of gummas of tertiary syphilis is similarly unsupported. Actinomycosis is a systemic mycosis of worldwide distribution caused by an obligate parasite of human beings and animals.[1,2] Meningitis is reasonably common, and multilocular brain abscesses surrounded by granulation tissue may be present. However, the patient's long survival, the lack of pulmonary or other characteristic extraneural involvement and the absence of purulent spinal fluid are strongly against that diagnosis. Cryptococcosis is of particular interest because of its predilection for the central nervous system, of which it is the most common mycotic disease.[1,3] Human infection has been reported from tropical and temperate zones of virtually every major continent. Involvement of the nervous system may assume the form of granulomatous meningitis, small granulomas or cysts within the cerebral cortex or deeply placed solid or cystic nodules, especially within the basal ganglions. The cystic lesions appear gelatinous, whereas the granulomas may have areas of necrosis. There is remarkably little inflammatory response. The clinical picture mirrors the type, severity and duration of the underlying pathologic process. The usual course is one of a progressive decline with increasing emaciation and debility, leading to death within a year in the majority of the untreated patients. There are occasional remissions, but survival for more than three years without treatment is unusual. The spinal fluid may be acellular but most often is not.[4] Although cryptococcosis is a possibility in this case, the 10-year course without treatment argues strongly against it. Coccidioidomycosis is occasionally associated with meningitis or intracerebral abscesses, presumably as a result of metastatic spread from a primary pulmonary infection.[1,5] On clinical grounds alone, this diagnosis seems unlikely. Histoplasmosis may involve the central nervous system, but it is usually manifested as a primary pulmonary infection that heals by calcification or as a progressive systemic disorder largely involving organs of the reticuloendothelial system.[1,3] It therefore seems reasonable to dismiss this type of infection.

Among the parasitic infections, we can exclude both amebiasis and toxoplasmosis. Cysticercosis, which is usually produced by the pork tapeworm, *Taenia solium*, has a worldwide distribution, with special prevalence in countries where raw or undercooked pork is eaten.[6,7] Human infection occurs when ingested or activated endogenous ova of this parasite release larvae, which bore into the intestinal wall and enter the bloodstream, spreading particularly to the subcutaneous tissue, muscles, viscera and brain. The inflammatory response to dead larvae is more marked than that to living forms. The extraneural symptoms are typically minor and include myalgia, weakness and sometimes slight fever. Several forms of neurocysticercosis have been described.[8,9] In the racemose variety irregular vesicles fill the subarachnoid space of the brain and on occasion invade the brain substance. Hydrocephalus may occur as a consequence of obstruction. Cystic cysticercosis is characterized by numerous cortical and subcortical vesicles, 5 to 20 mm in diameter, that contain a liquid. A glial reaction tends to localize and encyst the parasite. Radiograpically visible calcification is probably less common than has been generally assumed.[9] The neurologic damage and the resulting clinical picture are related to the location and type of lesion, the associated mass effect, the complication of hydrocephalus and possibly the effects of toxic metabolites produced by the parasite. Seizures are frequent with the cystic form. Conspicuous focal neurologic signs are less common. The spinal-fluid examination, the presence of eosinophilia or the identification of intestinal worms is not of diagnostic value in most cases. Palpable subcutaneous nodules as well as radiographically demonstrable calcified cysticerci are too infrequent to be of much help in the diagnosis. Some investigators[10] have stated that certain serologic studies, notably hemagglutination and complement-fixation tests, are both sensitive and specific in most of the cases, but recent work appears to refute this conclusion.[11]

In favor of a diagnosis of neurocysticercosis in this case are the abnormalities demonstrated on the CT-scans and radionuclide studies, which are in keeping with the cystic form of the disease. Additional support for the diagnosis is lent by the long clinical course marked by both seizures and focal signs. Against this diagnosis is the absence of most of the nonspecific or

infrequent physical, radiographic and laboratory findings that have been described.[12] It should be noted that the absence of ova in the stool is more common than their presence since the worm usually sheds proglottids or egg sacs rather than free ova.

Finally, I come to the possibility of echinococcosis, or hydatid cysts, caused by the larval stage of *Echinococcus granulosus*. Initially microscopic in size, the fluid-filled cysts may eventually assume huge dimensions. In one series 60 per cent of the lesions were found in the liver, 20 per cent in the lungs, and only 1 per cent in the brain, principally in the subcortical cerebrum.[13] Echinococcal cysts in the brain usually evoke an extensive glial reaction. Daughter cysts are characteristic, but multiple cysts are rare. As expected, the neurologic symptoms and signs are by no means specific. Seizures or signs of an expanding intracranial mass may dominate the clinical picture. The diagnosis of echinococcosis depends heavily on laboratory and other special studies. Although this possibility cannot be excluded in the case under discussion, the absence of either hepatic or pulmonary involvement and the evidence of multifocal cerebral involvement make it unlikely.

I suspect that the physicians involved in this case shared the diagnostic dilemma that I now face. The operation was probably a brain biopsy. If this assumption is correct it supports my impression that the available data are not sufficient for a precise pathological diagnosis. Accordingly, I shall suggest the diagnosis of a parasitic infection, with cysticercosis far more likely than echinococcosis. Alternatively, a systemic mycosis such as cryptococcosis might be considered.

Clinical Diagnosis

Cerebral cysticercosis.

Dr. James A. Schnur's Diagnoses

Cerebral cysticercosis (*Taenia solium*).
? Echinococcosis.
? Cryptococcosis.

Pathological Discussion

Dr. Edward P. Richardson, Jr.: As Dr. Schnur predicted, the operation was a brain biopsy. Will you tell us your findings, Dr. Chapman?

Dr. Paul H. Chapman: The operation was an excisional biopsy of the right frontal lesion, which proved to be a thin-walled cyst approximately 1.5 cm in diameter surrounded by a zone of firm, yellowish-white tissue (Fig. 4). It was located within the cortex but was also adherent to the undersurface of the dura mater.

Dr. Richardson: Dr. Hopkins followed the patient from the standpoint of a possible infectious disease. Are there any comments that you wish to make, Dr. Hopkins?

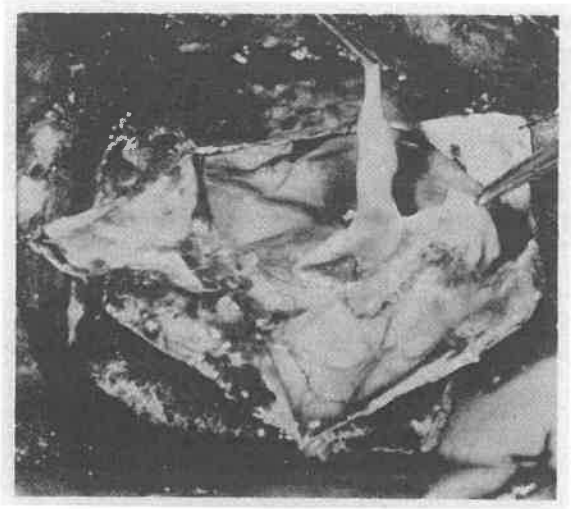

Figure 4. Photograph Taken at the Operation, Showing Removal of the Cyst from the Brain.

Dr. Cyrus C. Hopkins: I should point out that tissue from an operation of this kind should be examined by several laboratories. Even though the gross appearance strongly suggested the final diagnosis tissue was submitted to the routine bacteriology, fungus and mycobacteria laboratories as well as the pathology department to exclude the possible, though less likely, diagnoses that Dr. Schnur mentioned.

Dr. Richardson: The tissue that we received consisted in part of a membranous structure that was thrown into many folds. It was lined by a simple epithelium on the surface of which was a somewhat refractile layer. In addition, there was a rounded structure with a central cavity showing multiple branchings and containing the scolex of a tapeworm (Fig. 5). In addition to the characteristic suckers the scolex had a ring of hooklets that when viewed laterally could be seen to have a curved-sword or scimitar-like configuration (Fig. 6), which is characteristic of the cysticercus larva of the pork tapeworm, *T. solium*. The beef tapeworm, *T. saginata*, does not have a similar ring of hooklets. Moreover, as far as is known, it does not give rise to cerebral infestation.[14] The scolices of other forms of tapeworm, such as those of the echinococcus or *Multiceps multiceps*, may have hooklets, but infections caused by them are characterized not by a cysticercus, a single parasitic body, but by many scolices within a cyst. Thus, a positive identification of *T. solium* can be made on morphologic grounds. In the brain tissue surrounding the parasite there was a considerable degree of inflammatory reaction (Fig. 7) characterized by a wall of dense, hyalinized connective tissue, outside of which was a ring of lymphocytes, histiocytes and a few plasma cells, without eosinophils. The inflammatory layer was surrounded in turn by a band of fibrillary gliosis.

The patient was, and still is, under the care of Dr.

Wolpow, who has looked into a number of the questions raised by this disease.

DR. EDWARD R. WOLPOW: The Cape Verde Islands, of which this patient was a native, is a tropical country off the northeast coast of Africa at the latitude of the Sahara Desert and a convenient stepping stone for the colonialization of Brazil, Guinea and Angola. It had been a Portuguese colony for almost 500 years but recently became independent. The population is 270,000. According to information from the World Health Organization[15] the pigs in these islands are heavily infested with *T. solium* as well as the echinococcus.

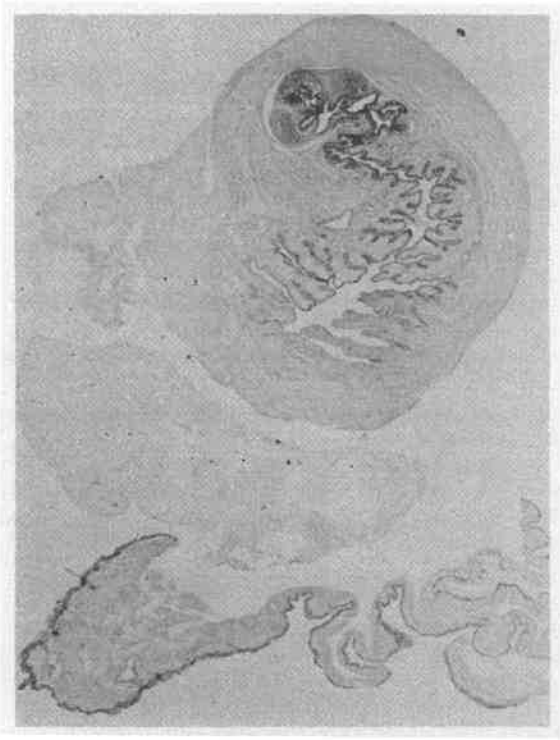

Figure 5. Low-Power Photomicrograph of the Biopsy Specimen. (× 21)

The rounded structure (above) is the encysted larva (cysticercus) of *Taenia solium*. The multiply folded membrane with a hyaline lining (below) is a portion of the cyst wall.

Progressive dementia was this patient's main problem. In view of his age the possibility of Alzheimer's disease was considered. In addition, it seemed possible that the head injury that produced at least eight days of coma had resulted in the small size of the left hemisphere and the large left lateral ventricle. We then considered the possible mechanisms for the development of progressive dementia in a patient with cysticercosis, including the complication of hydrocephalus. Cysts lying freely within the ventricular system may plug the aqueduct, producing noncommunicating hydrocephalus, and an inflammatory reaction in the meninges may result in communicating hydrocephalus.[16] There has been a recent report of cases of cysticercosis in which all the criteria for the di-

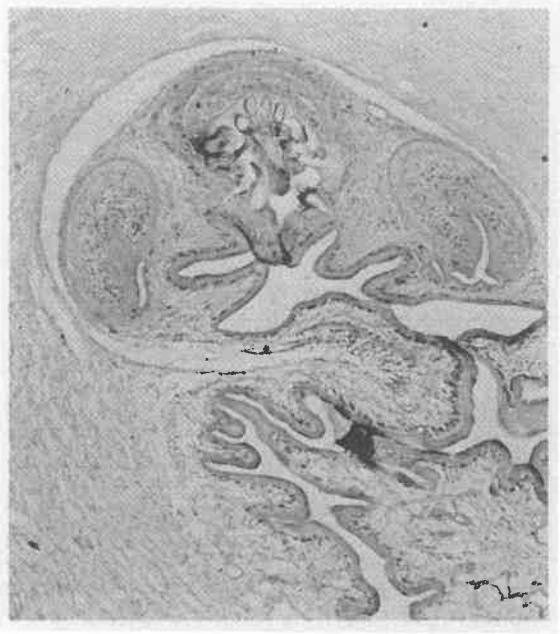

Figure 6. Scolex of the Cysticercus, with Suckers and Hooklets. (× 90)

The lowermost hooklet has a scimitar shape.

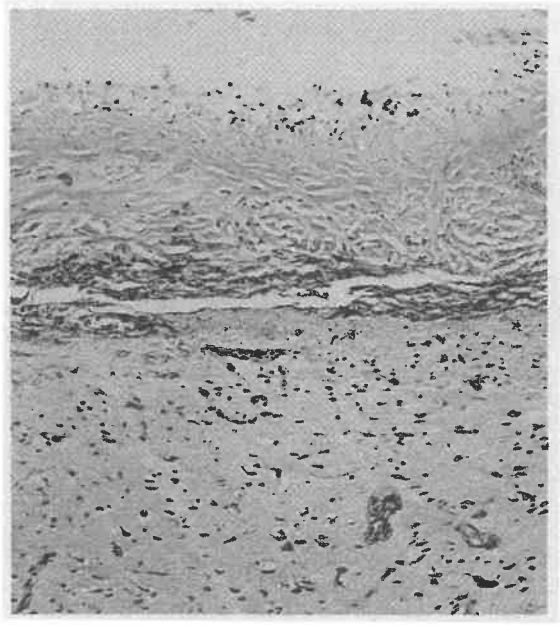

Figure 7. Tissue Reaction Adjacent to the Intracerebral Cysticercus. (× 90)

Note the successive layers, from above downward, of dense connective tissue, inflammatory cells and gliosis.

agnosis of normal-pressure hydrocephalus were fulfilled, and great benefit was obtained by shunting.[17] This man, however, did not have hydrocephalus according to either the clinical examination or the CT-scans.

Cerebrospinal cysticercosis produces a wide variety of neurologic signs and symptoms.[18-23] In one group of patients who do not have hydrocephalus or increased intracranial pressure progressive dementia occurs, presumably as a result of the large number of lesions in the brain and the inflammatory reaction around them. After seeing the inflammatory reaction around the cyst in the surgical specimen we concluded that the patient under discussion probably belongs in this category. The inflammatory reaction around the cyst presumably corresponds to the mottled pattern on the radionuclide scan and the enhancement of the region around the cysts in CT-scanning.

The question that was immediately raised by the inflammatory reaction was whether this man could be benefited with corticosteroids, particularly since it was not possible to remove all the lesions. The value of such therapy has not been well studied, although it clearly warrants careful investigation. A recently published overview of research needs in this disease reflects this issue,[24] and in the small number of case reports in this country[25,26] (about one a year) this option has not been mentioned. There is a recent report from India,[27] however, of a case in which cysticerci in muscle produced severe pain and "hypertrophic" muscular weakness. That patient also had seizures and presumably had cerebral cysticerci. After treatment with prednisone, not only did the size and tenderness of the muscles recede, but the seizures ceased.

Armed with this information and in co-operation with Dr. Hopkins, we started a regimen of 40 mg of prednisone a day, watching the patient's diabetes very carefully and administering antituberculous medication prophylactically. He was seen one month later, when he was no longer using a cane and was much more able to use his right limbs. For several years he had not written, but he had begun to write notes and letters spontaneously, indicating improvement in the function of his right hand as well as his mind. Also, he no longer had a problem in communication either in English or in Portuguese. Because of a misunderstanding he had not been given anticonvulsants on discharge from the hospital. Therefore, the improvement can be ascribed to the steroids alone. It is now our aim to taper the steroid therapy gradually to the lowest level that is consistent with improvement.

DR. RAYMOND D. ADAMS: With worldwide travel increasing cases of this type are being seen at hospitals like ours, not frequently, to be sure, but regularly. In our experience this disease has usually presented in the form of one of three syndromes; the most common is epilepsy, often with cerebral calcification, the next most frequent is hydrocephalus with its characteristic effects, and the third is a mixture of dementia, hemiparesis and focal seizures, sometimes in combination with hydrocephalus. In South America cysticercus encephalitis is considered the leading diagnosis in any patient in the adolescent-adult age period who has focal epilepsy with calcification of the brain. In this case, however, there were no calcifications in the brain or thigh muscles or other muscles, which are also thought to be diagnostic.

DR. HOPKINS: An additional point about *T. solium* infestation is that this organism illustrates an interesting facet of the problem of species specificity in parasitism. Although the larval form seems poorly adapted to the human host and often undergoes degenerative changes, the adult form thrives in the human intestine. It is the only major tapeworm that exists in both larval and adult forms in human beings.

DR. PETER G. BERNAD: At the Fourth Pan American Congress of Neurology in 1975 the question of the cerebrospinal-fluid immunoglobulins in cases of cysticercosis of the central nervous system was discussed, with reports on 30 patients with this disorder.[28] Semiquantitative evaluation of IgG, IgA and IgM was carried out on concentrated samples of cerebrospinal fluid. Low-concentration immunoplate diffusion based on Mancini's technic was used to calculate the concentration of these globulins. IgG was found to be the major immunoglobulin that was elevated in patients with cysticercosis of the central nervous system.[29]

In regard to management and treatment, Dr. A. Spina-França has pointed out to me that on the basis of the South American experience with this disease steroids are indicated when intracranial hypertension is present as a result of brain swelling.[30] Dexamethasone has been administered with a gradual reduction in the dosage as the clinical manifestations of the intracranial hypertension have receded. Trimethoprim sulfamethoxole, sulfadiazine, trivalent antimonials and hycanthone have not been effective. The patients in São Paulo are followed by repeated spinal-fluid analysis, cytologic examination, determinations of the total protein and protein electrophoresis and complement-fixation tests for cysticercosis. The CT-scan should also be useful in following these patients.

ANATOMICAL DIAGNOSIS

Cysticercosis cerebri (Taenia solium).

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