II. PATHOLOGIC AND RADIOLOGIC FINDINGS

JUAN CÁRDENAS Y CÁRDENAS, M.D.
Mexico City, Mexico

A. PATHOLOGIC FINDINGS

Cysticercosis of the brain and its envelopes manifests itself clinically as a kaleidoscopic disease with such a variety of symptoms and signs comparable only to the clinical manifestations of multiple sclerosis. This extreme variability is understandable when one becomes familiar with the biology and pathology of this parasitic disorder of the nervous system.

In 2767 cases of unselected autopsy material reviewed from January 1954 up to December 1959 at the Unit of Pathology, Hospital General of Mexico City, U.N.A.M., 97 cases of cysticercosis were found. This is an incidence of 3.5 per cent in the material reviewed.

Macroscopic Findings. The type of lesions encountered in cerebral cysticercosis depends on several factors: the time elapsed following the infection; the number of parasites that reach the nervous system and the possibility of immune allergic reaction.

Escobar and Nieto described four essential forms: a) meningeal, b) ventricular, c) parenchymatous, and d) mixed forms.

The meningeal and ventricular forms are predominant, a fact in accordance with our own studies and those of others (Guccione, Sato, Stepień, Choróbski, Henneberg, and others). López Albo, in his excellent monograph, stated that “since the blood stream is the path through which the nervous system is invaded, and the oncospheres are conveyed by the circulation of the cerebrospinal fluid, it is easy to explain the frequency of this location.” Though the vesicles lodge in any part of the anterior, middle or posterior fossa, inside or outside the brain, they also can be present in the spinal canal, but this rarely happens. In a brain with recent invasion, the parasites are small and solid but soon develop into the vesicular form. If a large number reach the parenchyma, the brain may appear swollen with signs of interstitial edema (flattened convolutions), and small or normal ventricles. The dura mater is not invaded and is intact. The pia arachnoidal may or may not be thickened as a local reaction to neighboring vesicles; this meningeal thickening quite often is seen at the base, subsequent to the racemose forms of cysticercosis, the so-called “basal Cysticercus meningitis.” This adhesive arachnoiditis (Fig. 1) commonly blocks the circulation of the spinal fluid in the subarachnoid space, leading to a communicating hydrocephalus or, if the foramina of Luschka and Magendie are obstructed, it produces an obstructive hydrocephalus. The degree of ventricular enlargement therefore will depend on the time of establishment of this blockade; sometimes these ventricular dilatations reach tremendous degrees.

When a brain is examined one may find the Cysticerci in the convexity or in the base. Those in the convexity usually burrow the cortex of the brain (Fig. 2), being partially hidden, but sometimes they are free (Fig. 3). A single parasite or as many as several hundred may be found. The parenchymatous
I have the impression from our own experience that when the parasites invade the parenchyma solely, and there are only a few, say less than twenty, the disease may cure spontaneously. Many times shadows of intracranial calcifications, compatible with *Cysticerci*, are seen in patients suffering seizures which may or may not be controlled with appropriate medication. Those patients suffered and withstood the acute stage of the disease. Rarely a mass of *Cysticerci* is seen deep in the cortex; usually it is unique but there may be scattered masses. Costero has described the “miliary” form, as if a shotgun had hit the brain. This form is seen mostly in children, and the parasites are small and have not reached their final or adult size.

The parasites in the ventricles may be single or multiple, small or fully developed, free in the cerebrospinal fluid or attached to the walls of the cavities. In these instances they may act as a ball-valve, or encroach calcify.

Vesicles are found more commonly in the grey matter than in the white matter, a fact readily understandable on the basis of vascular supply.

Another important fact is the size of the parasites. In most cases the adult or mature forms (Fig. 4) are found, while in others small, or immature forms (Fig. 5), reaching only 3 or 4 mm. in diameter, are concomitant with intermediate and large sizes. Hence we can assume that there have been several invasions, which account for the severeness and relapses of the clinical picture that often is seen. Occasionally, the cysts adhere together, forming clusters or a single but rather large vesicle. Inside this vesicle the scolex can be seen through the translucent wall. If the vesicle is large enough, the clinical picture may resemble that of a space-occupying lesion.

Quite often in the parenchymatous form, if the number of parasites is not great, and the patient survives, the parasites die and

Figs. 2. and 3. (Upper) *Cysticercus* burrowed in the cortex of the convexity. (Lower) Free *Cysticercus* vesicle in the convexity.

Figs. 4 and 5. (Upper) Large fully developed *Cysticercus* in the left lateral ventricle. (Lower) Multiple small oncospheres not yet developed into cysts in the parenchyma of the brain.
into the ventricular foramina.

Microscopic Alterations of Nervous Tissue, Leptomeninges and Ependyma. a) Microscopic lesions in the nervous tissue show the following characteristics: 1) they are well localized in the neighborhood of the parasite; 2) there are large fibroblasts which form a capsule around the cyst; 3) there is an inflammatory exudate of mononuclear cells, chiefly lymphocytes, plasma cells and eosinophils, and sometimes giant cells of the foreign-body type (Fig. 6), seen more commonly in the 4th ventricle; and 4) one may find gliosis, more or less marked, variable from case to case in accordance with the age of the lesion.

b) Almost all vessels in the vicinity of the parasites show a distinctive process of endarteritis (proliferation of the intima and lymphocytic infiltration). Sometimes there is a complete obstruction of the lumen of the vessel caused by the development of a true thrombosis. These arterial changes are prominent in the acute stage, and they are not caused by the death of the parasite, as has been stated erroneously.

c) Leptomeningitis. The microscopic appearance of this condition is: thickening of both the arachnoid and the pia mater which show an active proliferation of collagenous and precollagenous fibers; and considerable infiltration of mononuclear cells (Fig. 7) with predominance of lymphocytes, plasma cells and eosinophils (proliferative inflammatory reaction). In the final stage of this inflammatory reaction one can find only fibrosis. We have not seen the purulent variety that several authors described. There are cases in which, in spite of a severe invasion, the pia-arachnoid reaction never occurs. Robles and others attribute the phenomenon of pial thickening to an allergic reaction brought forth when the contents of ruptured or degenerated vesicles are mixed with the cerebrospinal fluid, though experimental proof for such is still wanting.

d) Ependyma. When the ventricular lining is affected, macroscopically the wall of the ventricles appears full of “pimples” (Fig. 8). These granulations are distributed regularly, forming patches with the appearance of small droplets of water, each well delimited and translucent. The vessels of the vicinity are engorged slightly. Microscopically there are zones in which the ependymal layer appears well preserved, showing its normal aspect: a continuous row of cuboidal

Fig. 8. Granulomatous appearance of the ventricular ependyma.
cells. In some places, corresponding to the granulations (Fig. 9), the ependymal lining appears interrupted and replaced by a small hillock pointing toward the ventricular cavity; the base of these small nodules may show edema, lymphocytic infiltrate and vascular proliferation; in some only the glial proliferation is apparent. The underlying tissue is loose from edema and may show glial and vascular proliferation. At higher magnification the histological changes are identified easily.

**Summary.** It may be stated that in any case of cysticercosis of the central nervous system a great variety of gross and microscopic lesions may be identified. These lesions depend on the number of parasites, their age, their location, and the particular reaction of each individual to the parasitic invasion. The main alterations in the cerebrospinal fluid are: pleocytosis with eosinophilia, increase in the protein content, decrease in sugar content and a positive complement-fixation test performed with an antigen prepared with *Cysticercus* powder. The latter reaction has been studied thoroughly by Nieto in this country and has proved its usefulness in establishing a correct diagnosis. The author also has postulated the fact that the pathological changes in the cerebrospinal fluid in one patient may show variations in samples obtained from the ventricular, cisternal or lumbar subarachnoid spaces. A patient with basal *Cysticercus* meningitis may show great alterations in the lumbar fluid, while the ventricular fluid remains unaltered. The usefulness of these data for the neurosurgeon is beyond discussion.

**B. RADILOGICAL FINDINGS**

The radiological manifestations of cysticercosis of the nervous system are understood better with the study of the correlations between the clinical, surgical and post-mortem findings. A series of 163 patients seen in 5 years at the Neurological Service of the I.M.S.S. has taught us to look for conclusive radiological data in cases in which cysticercosis was suspected. This information may be obtained by plain roentgen-ray films of the skull, or those utilizing contrast media. We classify the radiological findings as follows: (1) Primary, or those that correspond to the detection of the parasite itself. (2) Secondary, or those caused by (a) the signs of stagnation of cerebrospinal fluid; (b) subsequent increase of the intracranial pressure; and (c) modification in the form or position of the ventricular system visualized by air or Pantopaque (Fig. 10).

In the first group we find intracranial calcifications, single or multiple, unilateral or bilateral. The shadows of the calcified parasites are not always characteristic. This occurs only when the capsule and the scolex have calcium deposits. They usually are supratentorial and intracerebral, and in 5 per cent of the cases are seen as a circular streak of calcium surrounding an eccentric spot of the same material of major density. The size of these shadows varies from 2 to 4 mm. In many other instances the calcium shadows are irregular and can be interpreted as possible calcified *Cysticerci*. In our series we have not seen calcified *Cysticerci* in the subarachnoid space.

When air studies or intraventricular Pantopaque are used the parasites can be delineated. They may be free in the ventricles, and change their position with the movements of the head, or they may be fixed to the walls of any of the ventricular cavities, playing the role of a ball-valve. Dr. Jaime Dorfsman, myself and others have shown the advantages in diagnosing cysticercosis with iodoventriculography. In these in-
stances sudden occlusions of any of the narrow passages where the cerebrospinal fluid circulates may occur, and this causes an acute elevation of the pressure within the ventricles. This is produced by the displacement of the head, and was first described by Bruns for the 4th ventricle.

In the secondary group the radiographic changes consist of separation of the cranial sutures and enlargement of the sella turcica. This latter alteration resembles closely the ballooning of this structure in presence of a pituitary tumor. In cases of tumor this ballooning usually is not accompanied by edema of the optic disk, but by primary optic atrophy. Nevertheless papilledema has been observed in cases of very large hypophyseal tumors or of craniopharyngiomas in their final stages, as we have found in one instance. We have seen a patient with an enlargement of the sella and a large Cysticercus was found in it.

In the encephalitic form the ventricles appear normal, but in chronic cases in which a long-standing obstruction of the spinal

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Fig. 10. Modification in the ventricular shadow by a large Cysticercus vesicle confirmed by operation. (Courtesy of Dr. R. Steimle)

Fig. 11. Displaced anterior cerebral artery in same case as shown in Fig. 10.
fluid has been present the ventricles appear enlarged. This increase in the size of the brain cavities may be symmetrical or asymmetrical, and usually comprises both lateral ventricles and the 3rd and 4th ventricles. Guillermo Santín considers that in about 0.5 per cent of the cases there is lateral displacement of the shadows of the ventricles or of the anterior, middle and Sylvian arteries (Fig. 11). This deviation is produced by accumulation of vesicles, which often form a large cyst with identifiable hooks of the scolex in its contents. The presence of air in the ventricular cavities may help to visualize the Cysticerci. Stereoscopic or laminographic films may be of great help to localize and visualize the parasites in the ventricular cavities.