Computed tomography in neurocysticercosis

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SUMMARY Neurocysticercosis is a major public health problem in developing countries. Before computed tomography became available its diagnosis was very restricted and the conventional diagnostic methods were unreliable. It also was frequently necessary to submit patients to costly and dangerous surgical procedures to confirm the precise nature of the disease. One hundred and seventy-one patients with neurocysticercosis were evaluated by computed tomography. The diagnostic findings of the different types of lesions produced by the larva of the parasite (*Taenia solium*) in the central nervous system, and the advantages of CT in the diagnosis of this clinical entity are described, as well as the main signs and symptoms of the patients referred for examination. The effect of corticosteroids in the acute stages of the disease and the changes they provoke in the CT images are described.

Teniasis is a parasitosis of worldwide distribution, its incidence being considerably higher in developing countries. In Brazil, it is endemic in several areas, particularly in the south and middle west. There are two types of taenia: *Taenia solium* (pork tapeworm) and *Taenia saginata* (beef tapeworm). In the adult form, both live in the human small intestine. However, *Taenia solium* can also occur in man in its larval stage, causing cysticercosis. This larva (*Cysticercus cellulose*) may be found in numerous sites such as subcutaneous tissues, muscle, eyes, heart, liver, lung and peritonium.¹ In the central nervous system it causes one of the most grave human health problems. When this occurs, the clinical picture is called neurocysticercosis, and a great variety of signs and symptoms related to it have been described, such as convulsions, headache, motor and sensorial disturbances, intracranial hypertension, psychotic disturbances and ocular manifestations. Before the era of the CT scan the diagnosis of neurocysticercosis was difficult, mainly because there is no defined clinical picture in this disease. Traditional clinical investigations were based mainly on: (1) radiographic

studies of the skull, which occasionally revealed small calcifications (calcified cysticerci) in the brain parenchyma; (2) CSF studies, which sometimes showed an increased number of lymphocytes and eosinophils; and, (3) a positive complement-fixation test. The EEG, although frequently abnormal, does not show any specific findings. Even when carotid or vertebral angiograms, or ventriculography were performed the correct diagnosis was not always achieved, and craniectomy was indicated to obtain an accurate diagnosis.

In the authors' experience, computed tomography has proved to be an excellent method for the precise diagnosis of neurocysticercosis, as well as considerably decreasing the previous high incidence of morbidity and mortality associated with the traditional diagnostic methods. Unfortunately, the high cost of CT scanners and the maintenance of this type of equipment makes it inaccessible for many of the developing countries where the incidence of neurocysticercosis is high.

Subjects and methods

During the period March 1978, to July 1982, 7567 patients were scanned in the Center of Computerized Tomography, 171 of whom had neurocysticercosis. Ninety were males and 81 females. The patients were examined on a "Delta Scan" (Ohio Nuclear) brain scanner using 130 Kv, 30 mA and matrix 256 × 256. For middle or anterior fossa studies, slices of 8 or 13 mm thickness were obtained in planes parallel to the orbito-meatal line. The posterior fossa was studied using

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8 mm slices in planes of -20° in relation to the orbito-meatal line. The search for orbital or sela lesions was done using 8 or 5 mm thick slices in planes of $+10^\circ$ to the orbito-meatal line.

These patients were scanned both before and after the injection of iodinated contrast medium which was applied in a dose of 30.0 to 31.5 g of total iodine for adults (75 ml of Vascoray or 80 ml of Hypaque M respectively). Children received 2 ml/kg of body weight of either one of the media above. The contrast was injected as a bolus and the scanning was restarted 3 to 5 minutes later. The nature of the disease in the 171 patients was confirmed by: (1) the classic CSF findings as described by Lange² (33 cases); (2) the positive complement-fixation test³⁻⁵ (46 cases); (3) close follow-up using repeated scans (87 cases); or, (4) in a few cases by surgical explorations (5 cases).

Results

The great difficulty when using CT in the diagnosis of neurocysticercosis is the large variety of lesions caused by the larva of the parasite in the CNS and the similarity that some of these lesions may have with other space-occupying lesions. Table 1 summarises the main types of CT image found in the 171 patients selected for the present study. As can be seen from table 1, simple small areas of calcification were the

most common findings, consisting of rounded areas of high attenuation values varying from 80 to 360 Hounsfield Units (H). In the post-contrast studies these lesions did not show any enhancement and there was no perifocal oedema (figs 1 and 2), unless the calcification was associated with convulsions in the last 24–72 hours. The number of areas of calcification was quite varied and they occurred in any part of the brain, but very rarely were found in the cerebellum—only three cases in the present series—or in the brain stem—one case. When brain calcification was present, the differential diagnosis included toxoplasmosis and tuberous sclerosis.

The calcification sometimes was associated with cysts (figs 3 and 5) and sometimes occurred together with hydrocephalus. These cysts were lesions of varying number and size and had low attenuation values (10–20 H), which may or may not have presented slight enhancement at the periphery (figs 3 and 5). In general, perifocal oedema was not visualised, but big cysts may have produced a mass effect requiring surgery and small calcifications frequently were seen in the wall of the cysts (fig 3). When in the posterior fossa, they were commonly in the mid-line producing hydrocephalus of varying degree (fig 4). The differential diagnosis of neurocysticercus cysts included arachnoid cysts and porencephaly, as well as cystic astrocytomas.

The most striking type of neurocysticercosis diagnosed by CT was the acute or oedematous type (table 1). When acute lesions were diffuse, pre-contrast studies showed a great deal of oedema: the cisterns were hardly seen, the ventricles were compressed and frequently shifted, and cortical sulci were not visualised. Scanning after contrast injection revealed enhancement of numerous small ring-like areas (figs 6 and 9). Frequently, bulging occurred in the wall of

Table 1 CT images in neurocysticercosis

Types of lesions	No of cases
Calcification only	82
Calcification plus cysts	31
Calcification plus hydrocephaly	12
Brain cysts	10
Posterior fossa cysts	4
Difuse acute lesions	19
Single acute lesions	6
"Tumoral" lesions	7

Fig 1 A 40-year-old woman complaining of headache and generalised seizures. A, B and C: Pre-contrast—several small rounded areas of high attenuation values (90–180 H) are seen diffusely located in the brain parenchyma. Lesions are not seen in the posterior fossa.

Fig 2 A 10-year-old boy complaining of headache, convulsions, weakness in the lower limbs and unsteadiness when walking since the age of four. A, B and C: Pre-contrast —numerous small calcifications and cysts are seen throughout the brain parenchyma. Inside the cysts there may be seen smaller calcifications. There are no cysts nor calcification in the posterior fossa.

these small rings which were better seen when the image is magnified. Patients with this type of neurocysticercosis were referred for examination in a very poor condition, with complaints of severe headache, nausea and vomiting, and papilloedema was present. Sometimes they were admitted in a stupor or coma

with a suspicion of a space-occupying lesion. When only one of such lesions was identified in the brain (table 1), sudden attacks of focal seizures were the main complaint. In these cases, perifocal oedema always occurred (fig 8). The differential diagnosis included a slowly growing astrocytoma or a granuloma. Multiple lesions mainly have to be differentiated from multiple granulomas or acute multiple sclerosis. In relation to five of the patients examined, the CT images were similar to those found in other space-occupying lesions (table 1). In these cases, the association of the lesions with small areas of calcification in the brain parenchyma was an important clue in the diagnosis of neurocysticercosis. Such lesions were large, have considerable perifocal oedema, produced mass effect and presented contrast enhancement in a similar fashion to gliomas, metastases, abscesses or granulomas.

Finally, table 2 summarises the main signs and symptoms of the patients referred for scanning, and table 3 shows the incidence of the disease in different age groups.

Discussion

Neurocysticercosis is one of the most serious problems of public health in the developing nations. Although much has been described about teniasis itself, the way that the larva of the parasite reaches the central nervous system is obscure and is still subject to speculation. Owing to the great variety of clinical presentations of this entity,^{7,8} computed tomography has shown itself to be helpful not only in determining the aetiology but also number, localisation and extent of the lesions. As mentioned before, the most common CT findings in the present series were small rounded areas of calcification dispersed throughout the brain which are only occasionally detected by radiography of the skull. The differential diagnosis then included toxoplasmosis and tuberculous sclerosis. In the latter condition, a typical history of mental retardation, convulsions, and cutaneous lesions on the face (adenoma sebaceum), were useful in making the diagnosis. As is well known, calcification in tuberculous sclerosis is more frequently located around the ventricles in the sub-ependymal tissues. Calcification in toxoplasmosis is often linear and is not very abundant and associated with a marked dilatation of the cella media of the lateral ventricles, while the frontal horn shows an almost normal size.⁶

The cysts associated with neurocysticercosis have a fluid component (10–20 H) slightly denser than the CSF (0–10 H). These cysts may or may not show enhancement at the periphery, and the reason for that remains obscure. On the contrary, lesions found in the acute stages or even those called “tumoral”

Fig 3 A 43-year-old man complaining of headache and left hemiparesis. A, B and C: Post-contrast scans—two small areas of calcification (arrowed) are seen in the left frontal lobe. A large cyst occupies most of the frontal and part of the parietal lobe of the right hemisphere. A small calcification (arrowhead) is seen on the wall of the cyst. Thin arrows indicate small cysts in the brain parenchyma. No contrast enhancement occurs. Surgery confirmed the diagnosis of neurocysticercosis.

Fig 4 A 38-year-old woman with history of long duration headache. A: Pre-contrast—the image shows a large cyst in the medial line of the posterior fossa which did not enhance contrast. Posterior fossa surgery confirmed neurocysticercosis.

Fig 5 A 45-year-old man complaining of headache, nausea, vomiting and right focal seizures. A: Pre-contrast—the image reveals several cysts of varying sizes in the brain parenchyma. Calcification is seen in this slice (arrowed). B: After contrast injection—the cysts in the left fronto-temporal region are enhanced with contrast. One of the cysts was removed surgically confirming neurocysticercosis.

Fig 6 A 7-year-old girl with intracranial hypertension of acute onset. Faecal examination showed *Taenia Sp* ++. A: Pre-contrast—the image reveals intense oedema. B and C: after contrast injection—small ring-like enhancing areas are seen.

Fig 7 The same patient as in fig 6, 23 months after the first examination. The small ring-like structures which enhanced with contrast are no longer visualised, but, in their places, there appeared small areas of calcification. (A, B, and C are pre-contrast scans.)

Fig 8 A 4-year-old girl with history of sudden attacks of right focal seizures. A: Pre-contrast—the image shows an area of decreased attenuation in the left parietal lobe. B: After contrast injection—a small ring-like enhancing area is visualised in the centre of the area of decreased attenuation seen in the left parietal lobe. An operation was performed elsewhere, and microscopic studies revealed a larva of *Taenia solium*.

forms always show contrast enhancement and this may be due to a rupture of the blood-brain barrier in this region of the lesion as it occurs in gliomas, metastases, and abscesses, and not by neovascularisation as in the case of meningiomas. From the clinical point of view the acute stages of the disease deserve special medical attention since they produce a severe picture of intracranial hypertension due to the intense oedema created by the numerous acutely developing lesions, and by the fact that this stage is typically found in young patients. It was also interesting to note that all patients but one with acute development of neurocysticercosis had *Taenia Sp* in the faeces, in contrast to the chronic patient group in which the faecal examination was only occasionally positive for the parasite.

In the authors' experience, it is possible to obtain a dramatic reduction of the brain oedema in the acute stage with corticosteroids, the use of which completely changes the CT images. For example, fig 9 shows CT scans of a 20-year-old female admitted in a state of stupor. The first clinical diagnosis was abscess and a carotid angiogram gave evidence of space-occupying lesion in the left temporal lobe. Following the angiographic studies, plain CT scanning showed a great deal of brain oedema and after contrast injection several small ring-like areas showing contrast enhancement appeared. Twenty-four hours after the beginning of intravenous injections of dexamethazone (32 mg, daily), the patient was alert and co-operative. The full treatment was kept up for 8 days and the drug was then gradually discontinued over a 7 day period after which a second CT study was carried out which showed no abnormality. The cisterns are large and well visualised, the ventricles were normal in size and position and the brain sulci were visible as were the Sylvian fissures. The small ring-like lesions, which appeared after injection of contrast, were no longer identified, but signs of their presence could be found, if looked for carefully. The patient left the hospital completely normal but was brought back 28 days afterwards with return of headache, nausea, vomiting and papilledema. The CT scan obtained at that time was similar to the first scan. A new course of corticosteroid therapy was started and again a dramatic improvement occurred. Upon her subsequent dismissal the patient was kept on physiological doses of corticosteroids to avoid new relapses. The follow-up of one patient has shown that acute or oedematous lesions may calcify within a 23 month period after the invasion of the parasite in the CNS (figs 7 and 8). Unfortunately in the other cases of this study it was not possible to establish the elapsed time between the acute phase and the first signs of calcification.

Fig 9 A 20-year-old woman with headache, nausea, and vomiting followed by stupor. **A:** Pre-contrast—the image reveals intense brain oedema. **B and C:** after contrast injection—there appeared small ring-like lesions diffusely located in the brain parenchyma which enhanced with contrast. Faecal examination showed *Taenia Sp* ++. **D, E and F:** post-contrast scans of the same patient after corticosteroid treatment (see text). The ventricles are not shifted, and their lumen is clear. The cisterns are well visualised and marked brain sulci are identified. The lesions previously described are no longer seen. **G, H and I:** post-contrast scans of the patient 28 days after ceasing corticotherapy; the oedema has returned and the small ring-like enhancing areas are again visualised in the brain parenchyma.

Table 2 *The main complaints of patients referred for examination*

Clinical features	No of cases
Convulsions (focal and generalised)	83
Headache	31
Intracranial hypertension	24
Involvement of long tracts	10
Involvement of cranial nerves	5
Mental deterioration	5
Vertigo	2
Occasional finding	11

Table 3 *Incidence of the disease in different age groups*

Age in years	No of cases
0-10	20
11-20	33
21-30	40
31-40	25
41-50	29
51-60	14
61-70	8
71-80	2

Perhaps the follow-up of those patients in the acute stages may help in clarifying this point, but the authors think that study with animals would be the appropriate model for answering the question. There are other unanswered questions. Why do the larvae develop either to a calcified or cystic or even tumoral type of lesion? Why do the cysts behave differently with contrast injection? The study of

neurocysticercosis in animals by computed tomography may help us provide answers to these questions.

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