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Clinical Case Report

SEVERE DEMENTIA ASSOCIATED WITH NEUROCYSTICERCOSIS

ANDRES ROSSELLI, MONICA ROSSELLI, ALFREDO ARDILA and BEATRIZ PENAGOS

Instituto Colombiano de Neuropsicologia

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A severe case of dementia associated with a parenchymous miliar neurocysticercosis in a 15-year-old girl is described. Although there was general cerebral involvement, EEG, neurological and neuropsychological examinations showed a marked decrease in right hemisphere activity. Treatment with Praziquantel reduced the patient's intellectual deterioration, but hydrocephalus developed.

Keywords: neurocysticercosis, dementia, Praziquantel, neuropsychology

Neurocysticercosis is a systemic infection of the CNS by the Taenia solium or cysticerca (Escobar, Nieto & Aruffo, 1983). Epileptic-like attacks are the main clinical sign, followed by various mental disorders in about 10% of the cases (Cendes & Ferreira, 1986; Grisolia & Wiederholt, 1982). Gomez, Bustamante and Sanchez (1985) mention 8% mental changes in their Colombian studies while Ramirez et al. (1986) estimate a 5% incidence. In Colombia the prevalence of neurocysticercosis is 400/100,000, while in Mexico it is 2,500/100,000 (Escalante, 1977).

The mental changes most frequently reported are: confusion, disorientation, memory loss, hallucinations, psychomotor incoordination, progressive deterioration of language ability, and mental deterioration (Cendes & Ferreira, 1986). Dixon and Lipscomb (1961) report schizophrenic and manic-like episodes as possible initial signs of neurocysticercosis. They found only 5 cases of true dementia in 450 patients (1.1%).

However, precise descriptions of the intellectual deterioration in cysticercosis are seldom reported. It is believed that three forms of the infection many cause such damage: parenchymous cysticercosis, which affects large areas of the CNS; ventricular cysticercosis, which cause hydrocephalus by obstruction, usually at the IV ventricle; and meningeal cysticercosis, which causes a strong inflammatory reaction with associated extensive meningitis (Escobar, Nieto & Aruffo, 1983). McCormick, Chi-Shing-Zee and Heiden (1982) report parenchymous cysticercosis in which more than 200 cerebral cysticerca were observed.

The case described here is of a miliar cysticercosis, affecting both cortical and subcortical structures within the cerebral parenchyma.

Send correspondence to: Dr. Alfredo Ardila, Apartado Aereo 17021, Bogota - Colombia, South America.

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CASE REPORT

A 15-year-old, left-handed girl showed adequate intellectual and physical development up to the time the condition appeared. Her parents own and manage a pig-farm and market the processed product. In early 1981, she complained of headaches of increasing intensity and frequency. By 1984 her schoolwork, which had been outstanding, deteriorated. She became indifferent, apathetic and depressed. She failed her senior high school year. In 1985 she returned to school, but went home each day after two or three hours complaining of headaches and nausea. She dropped out of school in August, 1985 and remained at home, usually in bed. Subcutaneous nodules appeared first on her brow and soon over her entire body. She showed unusual behavior, e.g., speaking strangely, trying to leave her room at night through the window, she wrote down things differently from what she intended, etc. Her headaches increased in intensity. On January 14, 1986, she vomited during the night. The next day she appeared completely indifferent to her surroundings, without spontaneous speech. On January 16, she woke up screaming that she was going to be devoured, that she saw snakes everywhere, that somebody had killed her child (?); she talked to a grandparent who died several months earlier and had short periods (5–10 minutes) of agitation. Indifference set in again and she then began hallucinating. She became delirious, declared that she could see the Virgin Mary, ran around as if trying to escape, and could hear voices calling her. At times she stood rigid, eyes fixed on nothing, not responding to questions; there was occasional micturation. In this state of intermittent agitation, she was taken to the mental ward of a local hospital. The initial diagnosis was of a psychotic state.

Neurological examination showed bilateral papilledema, pathological reflexes (hand and foot pressure, stronger on her left side, snout and bilateral Babinski sign), apraxic gait, discrete lateropulsion and moderate hyperreflexia in the left hemibody. The rest of the examination was normal. The EEG showed multifocal slowing in both hemispheres, but predominantly on the right. Epileptic-like focal discharges in the right temporo-occipital region and independently in the right frontal region were observed. A CT scan showed a very severe miliar neurocysticercosis (Figure 1). 790 cysticerca were counted (as far as we know, the largest number ever reported in human brain) of which about 75% were calcified.

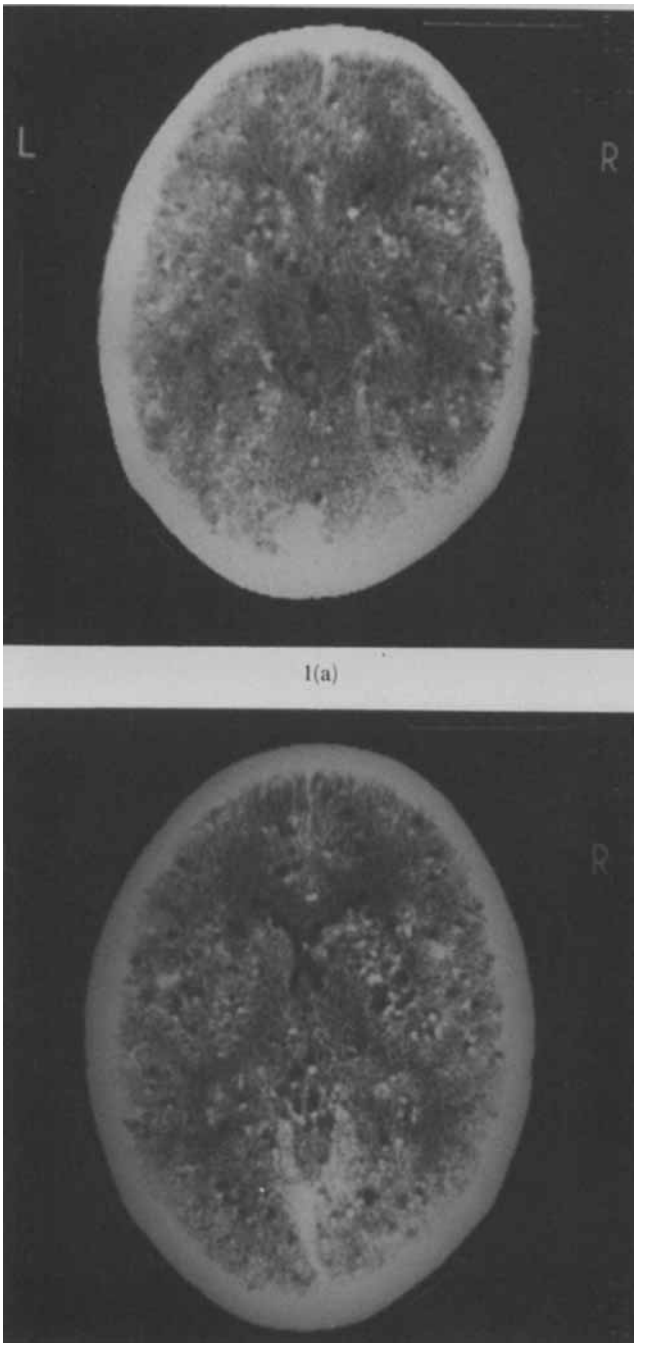
The predominant mental alterations observed were emotional flattening, episodes of unmotivated laughter, tendency to perseverate, and a catatonic-like attitude. She had an orientative permanent response to all stimuli and a definite tendency towards echopraxia.

NEUROPSYCHOLOGICAL ASSESSMENT

An absent-minded patient was observed. She suffered sudden and frequent fits of laughter and crying. She seemed space-oriented but was disoriented in time, with nonspontaneous, slow and hypophonic speech.

1) *WISC-R.* A higher level was attained in her verbal subtests (verbal IQ = 57), but she was unable to do the performance subtests (Table 1).

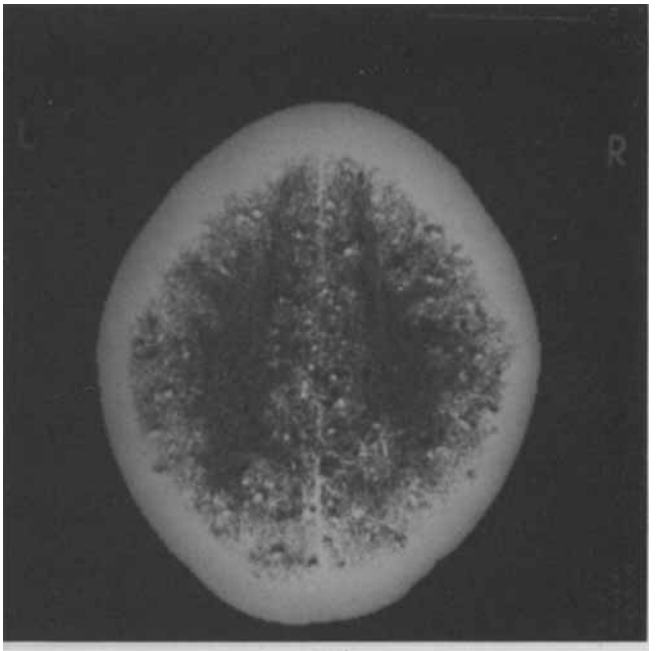
2) *Boston Diagnostic Aphasia Examination* (Goodglass & Kaplan, 1972). Repetition and automatic language were adequate, writing was virtually impossible; fluency, auditory understanding, naming and reading were moderately diminished (Figure 2).



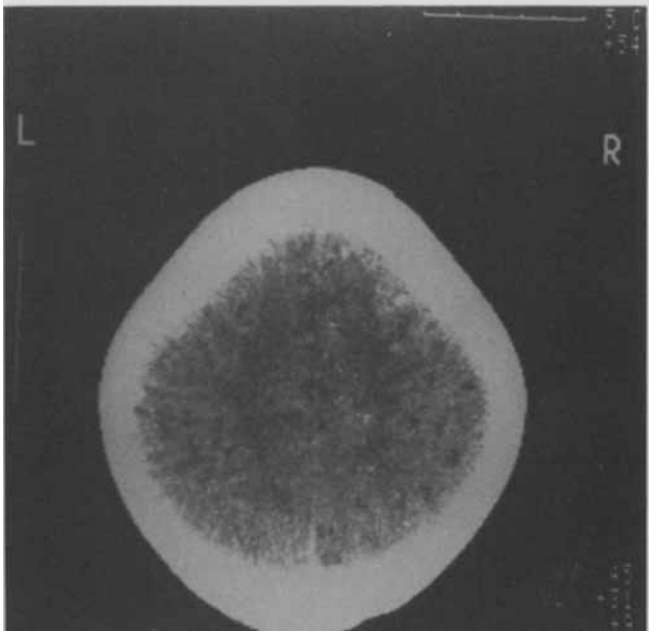
1(a)

1(b)

FIGURE 1 CT scan before treatment. A large number of cysticerca are observed, not only in the cortex, but also in the subcortical structures.



1(c)



1(d)

Figure 1 *continued*

TABLE 1
WISC-R. Scale Scores in Subtests Before and After (in parenthesis) Treatment with Praziquantel. Final Total IQ = 73 (VIQ = 87, PIQ = 63)

Verbal		Performance	
Information	3(9)	Picture completion	3(5)
Comprehension	1(6)	Picture arrangement	1(3)
Arithmetic	0(6)	Block design	0(5)
Similarities	11(11)	Object assembly	0(7)
Vocabulary	0(8)	Digit-symbol	0(1)
Digit-span	2(6)		
Verbal scale score	17(40)	Performance scale score	4(21)

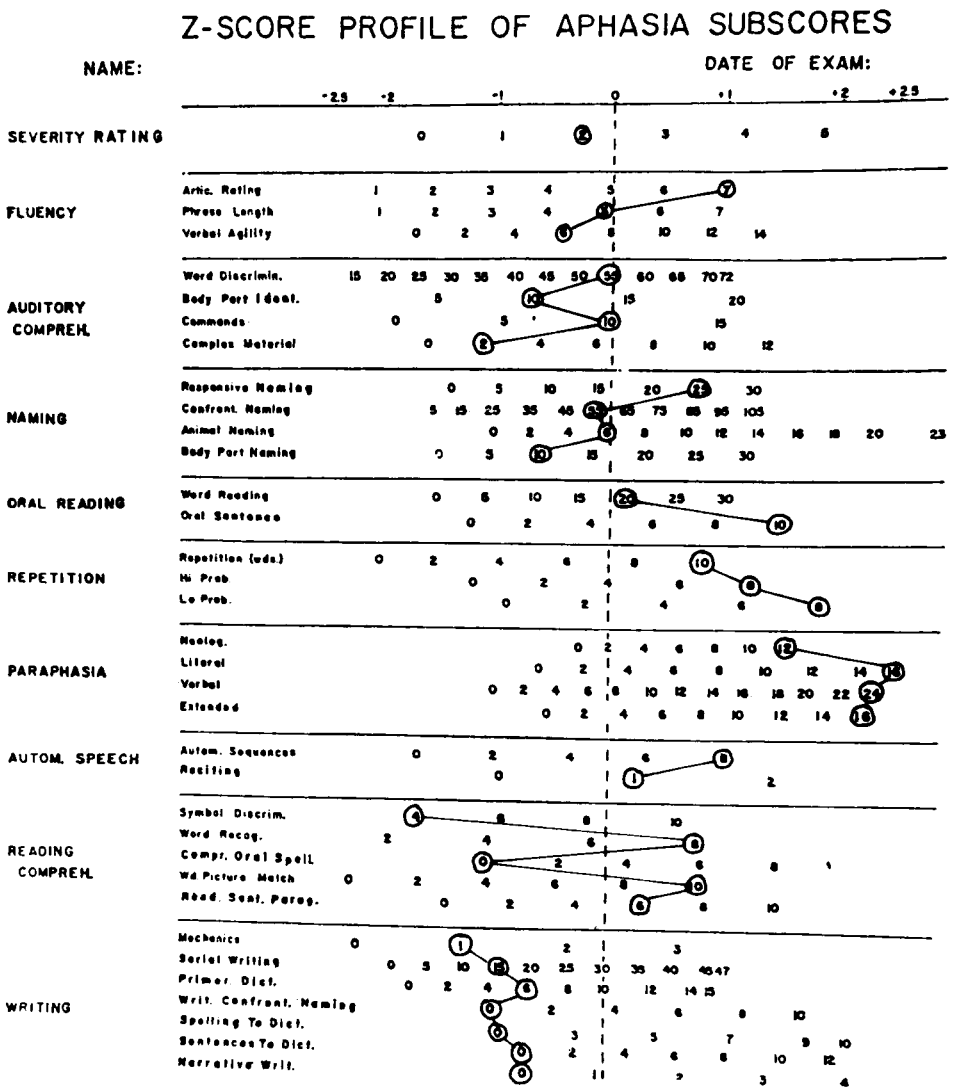


FIGURE 2 Patient's profile on the Boston Diagnostic Aphasia Examination.

3) *Token test—shortened version* (De Renzi & Faglioni, 1978). The patient could only respond to single orders (15/36) and failed all longer and more complex orders.

4) *Rey–Osterrieth complex figure.* Spatial rotations, left-sided neglect, severe spatial disorganization and fragmentation were observed (Figure 3).

5) *Memory.* In the memory curve, she repeated 5 out of 10 bisyllabic common words after four repetitions. In sentence memory she could not remember any of the three sentences given in sequence by the examiner. In logic memory she retained 4 out of 16 ideas from a text read by the examiner.

6) *Praxis.* Bucofacial: she carried out five movements by verbal order. Ideomotor: she carried out two of five orders but she could not imitate with her hands any of the movements made by the examiner.

7) *Verbal generation.* Using semantic categories (animals and fruits) she only produced one element in one minute. She completely failed the phonological categories (searching for words beginning with A and F).

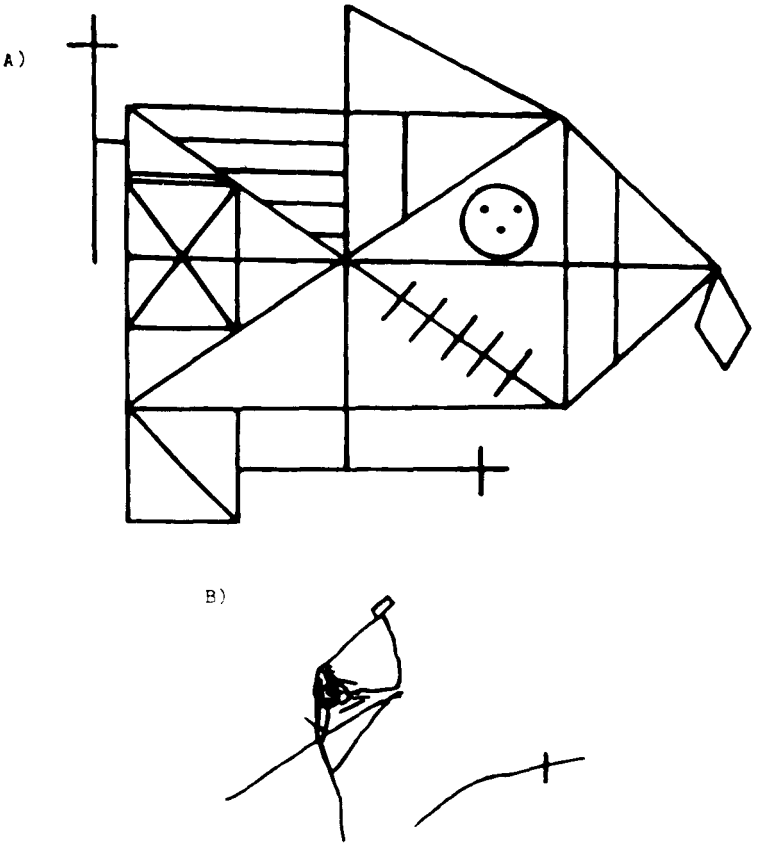


FIGURE 3 Rey–Osterrieth complex figure. (A) Model, (B) copy.

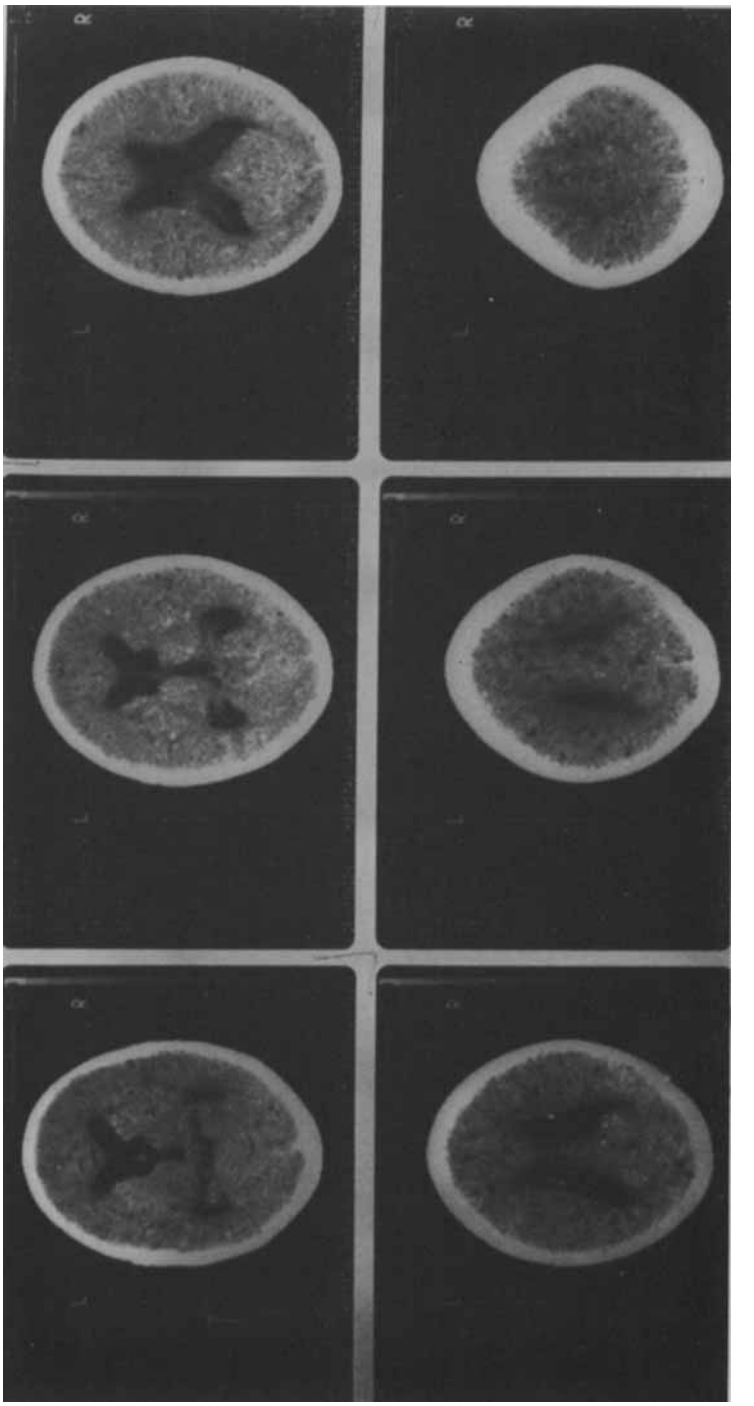


FIGURE 4 CT scan after treatment. Significant increase in the size of the ventricles is observed.

TREATMENT AND EVOLUTION

On May 25, 1986, treatment with Praziquantel (a synthetic derivative of isoquinoleina) at 1800 mg/day, divided in three doses, for 15 days, was begun. The second day she was in a stupor, presented bilateral midriasis, hyperreflexia and vomited. She was put on Decadron (8 mg, three times a day). On the third day a bilateral Babinski sign was observed, bilateral papilledema was present, and she showed stiffness with flexion of her upper extremities. On the fifth day she improved. The papilledema diminished, she opened her eyes spontaneously and there were some spontaneous movements. She was alert and responded orally on day seven; the spasticity and the bilateral Babinski sign disappeared. On day 12 she appeared logorheic, excited and had visual hallucinations. On the last days of treatment, she was alert but indifferent to her surroundings; there was a slight increase in muscle tone, but no other neurological changes. There was occasional urinary incontinence. She cooperated with the exam and answered questions.

The treatment was repeated in August and November, 1986. A new neuropsychological evaluation was done in February, 1987 (see Table 1).

At the end of treatments, another CT scan (Figure 4) showed significant increase in the ventricular size (communicating hydrocephalus). Later neuropsychological evaluation showed only moderate improvement in her intellectual ability compared to the initial evaluation.

DISCUSSION

The clinical picture began with headaches, associated with apathy, poor school work and finally hallucinations, confusion and excitement in apparently paroxysmal episodes. Although she presented extensive parenchymal cysticercosis, there was a larger involvement of right hemisphere activity, as shown by the EEG, neurological and neuropsychological exams. There was a deep deterioration of nonverbal functions, as well as signs of frontal involvement (general behavioral changes, pathological reflexes, lack of attention, tendency to muteness, etc.). Her verbal abilities as shown by the WISC-R verbal scale and the Boston Diagnostic Aphasia Examination, were in better condition.

At the beginning of the treatment there was a clear deterioration of the patient's general state, perhaps due to an inflammatory reaction to Praziquantel to the point of presenting stiffness with flexion of the upper limbs (decerebrate posturing). After the fifth day, the patient's general state retured to the level observed before the Praziquantel treatment. A CT scan showed hydrocephalus, not observed in the initial scan.

Since bradypsychia, emotional alterations, apathy and memory loss deficits predominated, the clinical picture resembled a subcortical dementia. The initial psychiatric examination indicated a psychotic state of the catatonic schizophrenia type. However, there was a correlation found between the epileptic-like focal temporo-occipital and right frontal discharges, and the hallucinogenic behavior.

It is important to note that the patient's intellectual condition improved slightly after treatment. Even though the activity of a large number of cysticerca was eliminated, the patient still showed hundreds of calcified foci, widely extended throughout the brain (besides hydrocephalus) which in some way must be correlated with the cognitive-intellectual deficiency that remained.

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