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Clinical Note

TEMPORAL LOBE INVOLVEMENT IN CAPGRAS SYNDROME

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A Capgras syndrome case is presented. The clinical profile appeared at the onset of treatment with Praziquantel for neurocysticerosis. A left-temporal cysticercum was shown by means of a CT scan. Delusion, accompanied by some neurological symptoms diminished but did not disappear after treatment. The hypotheses for Capgras syndrome are reviewed and it is concluded that it constitutes a cognitive-dysmnesic phenomenon (feeling of unreality, impossibility to integrate recently acquired information with old memory traces) and, as such, its origin is likely temporal.

Keywords: Capgras syndrome, temporal lobe, psychosis

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The Capgras syndrome consists of the belief that one or more people (usually close relatives) have been substituted by imposters or doubles (Capgras & Rebou-Lachaux, 1923). Some researchers (Alexander *et al.*, 1979) consider it to be a form of reduplicative paramnesia (Pick, 1903); reduplicative paramnesias may include the feeling of reduplication of people, places, time, events, parts of the body and even of one's self (Weinstein, 1969; Weinstein & Kahn, 1955). With the exception of a few cases, the Capgras syndrome has been reported almost exclusively in the psychiatric literature (Berson, 1983).

We present a rather unusual case, analyzing its implications regarding the possible underlying mechanisms.

CASE REPORT

A 31-year-old male, final year law student, consulted a physician in 1980 because of depression, headaches and adynamia. On occasions he reported nausea. The EEG showed high theta activity level in the temporal areas at the end of hyperventilation, but without a true paroxystic character. A diagnosis of depression and headaches caused by stress was made. During the following years, the patient consulted different psychiatrists and psychologists because of these symptoms. He followed short therapy sessions and took antidepressants.

In May 1986, he presented partial elementary motor seizures of the right upper limb and face. At times, there was sudden anacusia and sucking without loss of consciousness. Rivotril was prescribed. On June 11, the EEG showed minimal, nonspecific anomalies in the left hemisphere and occasional bursts at the same frequency but without a true epileptic character during hyperventilation. Tegretol (600 mg/day) was prescribed. On July 1, a generalized tonic-clonic seizure set in. On July 24, a new EEG was taken, this time under sleep deprivation. It appeared quite abnormal, showing theta activity bursts intermixed with acute elements in the left temporal regions. At times, there was continual theta activity which became marked under hyperventi-

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lation. On July 11, a CT scan was taken. A low density cysticercum was observed; it was approximately 6 × 8 mm in size, localized at the left external capsule which remained unmodified through contrast injection and did not produce a mass effect. The ELISA test was positive for cysticercosis and neurocysticercosis was diagnosed.

The patient was hospitalized on August 5 for treatment. The incoming neurological examination was normal. Epamin (300 mg/day) and Decadron (8 mg/day) were administered. On August 7, an event which is important to the understanding of his later delusion, occurred. The patient, bothered by another patient's complaints, asked the nurse to give his neighbour a tranquilizer and argued with the nurse. The next morning, treatment with Praziquantel (50 mg/kg/day) was begun. Two days later, while the patient's mother was visiting him, he told her that his neighbour in the next bed had died because of an overdose of tranquilizers administered at his request. He also said that the person who was in that bed was double of the one who had died; that it was not the actual patient. A delusion set in and the patient was handled throughout his stay at the hospital with Haloperidol (10 mg/day) and Akineton (2 mg/day).

On August 1, a neuropsychological evaluation was carried out. The patient presented a central right facial, was adequately oriented; his speech was slow, and there was a decrease in his articulatory ability and syllabic reiterations were observed. There were changes in his handwriting which he performed with noticeable slowness. His reading was slow; there was difficulty in understanding complex texts; his conversational understanding was adequate; he followed verbal orders without any difficulty. He pointed at the named objects, parts of the body and colors. There was slight right hemispatial neglect when copying drawings. He retained seven bisyllabic common words after four repititions and was able to recall them at the end of the examination.

During testing, the patient reported that the nurses were wearing masks to confuse him; he insisted that they were not the same people but were only pretending to be. Moreover, he claimed that at times a tall, blonde nurse came in, and later a short, dark one. To him, they were one and the same person; he realized this because they had the same voice. Everyone was trying to discover who was responsible for the other patient's death. When another patient underwent a blood transfusion, he understood it to be a message telling him that he was going to die. Everyone had been trying to confuse him and that was why they changed faces. A hospital doorman was passing as a doctor and the doctors as visitors.

We should point out that at no time were any signs of prosopagnosia present. The patient recognized photos of well-known personalities, matched faces, remembered and recognized people he had recently met, and was able to describe familiar faces.

He left the hospital on August 25 having completed his treatment. Upon returning home, he reported feeling it to be different; "the rugs seem thicker and brighter in color." When he went to the dentist, he found that the dentist was not the same person as the one who used to see him, but looked like him. He realized this because the original dentist had been more talkative.

On September 9 and 20, he returned for follow-ups. He mentioned how much things had changed, little was the same as before. At the hospital he had felt reborn, like a different person, as in a dream. He has seen the world as a different place, and himself as a different person since being at the hospital. He continued blaming himself for the other patient's death and reported the existence of a plot against him for the same reason. He also said that people had changed places or that the same person had taken on a different appearance. He mentioned that everything was absurd but true at the same time because he knew how frequently people got killed in hospitals. At this time, around September 20, the patient visited the hospital daily and went back to the ward he had been in, trying to clarify what had really happened. He took on

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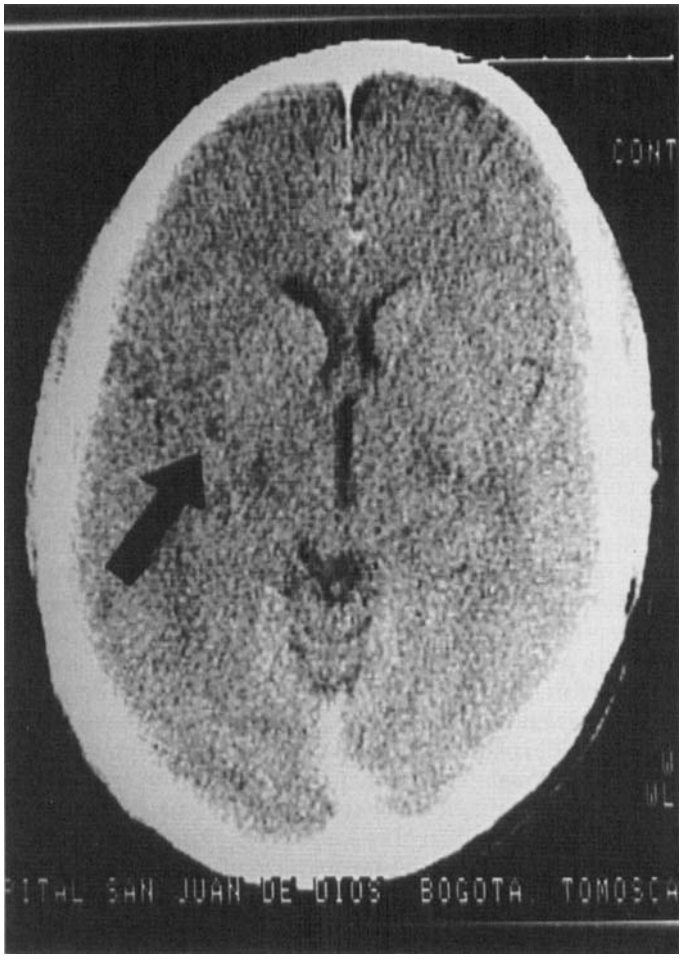

FIGURE 1. CT scan showing a small residual lesion after the treatment with Praziquantel

the role of a messenger, taking other patient's requests to and from their families. The central right facial was not observed; neither were defects in language production. A new CT scan was taken which showed a small residual lesion (Figure 1).

DISCUSSION

Berson (1983) reviewed all the cases described in the literature to date. Of the 133 reported cases, 31 presented some type of brain damage associated with a psychiatric diagnosis; seventeen were reported as depressive and in four there was prosopagnosia. The most frequent diagnosis was one of schizophrenia in its paranoid form (74 cases). Our pateint showed a clearly defined profile of depression over the past years, but did not show prosopagnosia. There was definite brain damage. His delusion began at the same time treatment for neurocysticercosis with Praziquantel was initiated. It is known that an inflammatory process follows the death of the parasite (Escobar *et al.*, 1983). Paranoid elements are clearly maintained for at least a month and a half longer. During treatment, previously unreported neurological symptomatology was observed,

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consisting of the presence of right central facial, a decrease in articulatory ability and syllabic reiterations. This symptomatology accompanied the delusion.

At the time of discharge, the neurological symptomatology had almost disappeared and the delusion had decreased. There were indications of brain inflammation at the time of treatment, which would have caused enhancement of the symptoms and possibly some permanent brain damage due to the destruction of the parasite.

Several hypotheses of the causes of Capgras syndrome have been proposed.

1. Some researchers (Hayman & Abrams, 1977; Shranberg & Weitsel, 1979) have suggested that prosopagnosia is the most likely cause of the syndrome. However, it is only present in 4 of the 133 patients mentioned by Berson (1983). This condition was not exhibited by our patient, and the site of damage was totally unexpected for a case of prosopagnosia.

2. At times, a memory defect, such as the one proposed by Pick (1903) in reduplicative paramnesia, has been considered to be the most central aspect of the syndrome. This appears to arise from the impairment of newly acquired memories by old ones. Staton *et al.* (1982) mentioned a patient who described all his current experiences as being like bad copies of previous, similar experiences. Our patient, although emphasizing that the world seemed unreal, did not report having had experiences of a similar nature. Statton *et al.* reported atrophy of the right posterior hippocampus, which we suppose to be linked with the patient's déja-vu experience (Ardila *et al.*, 1986) and to the visual memory defects observed. Both conditions were absent in out patient. Their case and ours shared the feeling of unreality and the difficulty in integrating current experiences with past memories ("It is like having been reborn, with no memories of what happened before." our patient stated). Staton *et al.* suggest an occipital-hippocampus disruption that causes the disconnection between present and past experiences. It is certainly a plausible explanation. In our case, the topography of the lesion does not coincide with theirs, although the experiences reported by the patients are similar.

3. The presence of spatial defects, with bilateral frontal pathology, has also been suggested (Alexander *et al.*, 1976; Benson *et al.*, 1976). It is proposed that right hemisphere damage would produce visuospatial malfunction and the frontal malfunction would cause conflict between past and present memories. The cases reported by these researchers (as well as Staton *et al.*) have a traumatic etiology which would permit the patients to distinguish two groups of memory traces: pre- and post-traumatic. Furthermore, the Capgras syndrome patient reported by Alexander *et al.* (1979) had apparently developed psychosis before the trauma. The topography of the damage in our patient does not coincide with the proposal.

4. Although the syndrome can occur in the presence of a wide variety of organic conditions, it is not likely that organic factors alone can be held causally accountable for the content of the delusion (Berson, 1983). A condition and a precipitant must exist before the delusion will appear. The preconditions would be: a) psychotic state, b) a marked paranoid tendency, and c) a pathological splitting of the internalized object. Although a review of the literature shows some type of depression as the second cause, after paranoid schizophrenia, no special mention of it has been made. Our patient showed a clearly depressive history at least six years long.

Some peculiarities in which our case differs from other reported cases are: a) the functional background is of a long evolving depression; b) the patient showed partial motor seizures, perhaps even complex one, associated with an EEG left-temporal focus; c) the structural damage was definite and clear from the CT scan: a left-temporal cystericercum was present; d) there was no prosopagnosia or spatial defect; minimal memory defects were manifest; and e) delusion began once treatment for cysticercosis was initiated and was accompanied by some neurological symptoms.

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There is a component of the Capgras syndrome which some researchers have mentioned (Staton *et al.*, 1982) but others have considered perhaps of little importance. The feeling of unreality is the basis of the syndrome from our point of view. As epileptic phenomena, cognitive changes including the paroxysmal feeling of unreality have been associated with temporal foci in either hemisphere (Ardila *et al.*, 1986; 1988). The world has been replaced and/or substituted. It is not the same as before; people are not the same either, they seem unreal as does everything else. In this sense, it would be a purely cognitive phenomenon. It would not be either a spatial or memory defect, nor one of failure of frontal logic.

Studies using electrical stimulation of the brain have shown that phenomena of the cognitive-experiential and dysmnesic type (like feeling strange, déja-vu, etc.) are associated with either right or left limbic temporal lobe stimulation (Gloor *et al.*, 1982; Halgren *et al.*, 1978). This is also true in the case of partial cognitive-dysmnesic seizures (Ardila *et al.*, 1988). In the case of electrical brain stimulation, the real content of the patient's experience depends on his/her ongoing activity and variables such as personality (Halgren *et al.*, 1978). At the beginning of the treatment and concurrent appearance of delusion and neurological symptoms, there was a great deal of importance given by the patient to the argument he had with the nurse and with the other patient. This incident became the topic of daily conversation. Furthermore, the underlying feeling was one of guilt and depression which agrees with his previous personality.

For our patient, not only could real people be substituted but a single person could take on a different appearance (the tall, blonde nurse was the same person as the short, dark one). In the outside world, objects now are different from what they had been before. The patient associated the initiation of this period of unreality with the time he was hospitalized and with the incident of the other patient's "death."

A month and a half later, depersonalization continued, but to a lesser degree. Delusion continued, also to a lesser degree. There is no indication of previous paranoid ideas in the patient's history.

We believe this experience of unreality to constitute the most important factor in the Capgras syndrome. It is linked to determining factors which give content to the experience. Antecedents of depression may play some role in our patient's delusion.

In conclusion, we consider that Capgras syndrome arises from the feeling of unreality for present experiences. This might help to explain it as a cognitive or dysmnesic type of phenomenon, the limits between the two conditions being subtle and difficult to establish. As it is, the origin would be either right or left temporal, according to the reading taken during epileptic seizures and during electrical brain stimulation. We believe, as Berson (1983) a does, that the real content of the delusion lies in situational factors, personal charcteristics and the learning history of the patient.

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