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# CAPGRAS SYNDROME AND ORGANIC BRAIN DYSFUNCTION

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*Abstract*

*Capgras Syndrome was described in the late nineteenth century but its exact pathogenesis is still a source of controversy. Some believe its origin is due to psychodynamic factors whereas others have found the evidence of a generalized or localized brain lesion. We report three cases of Capgras Syndrome occuring in association with frontal lobe lesion.*

*Key words : Capgras syndrome, organic brain dysfunction*

## INTRODUCTION

The clinical description of the Capgras Syndrome appeared in the literature as early as 1893 by Magnan and in 1903 by Janet. Initially it was referred to 'as illusion des sossies' (the illusion of double), and in 1929, was named as Capgras Syndrome in honour of the authors contribution. In this syndrome, the patient believes that a person, usually a close relative, has been replaced by an exact double or imposter and maintains this false belief despite evidenes to the contrary.

Enoch et al (1979) assert that, since the syndrome occurs in a clear sensorium, it is obvious that there is no organic basis to the condition. but is rather a functional illness and as such is explained on a psychodynamic basis. Other authors (Haslam, 1973; Vogel, 1974) have supported this view. The Capgras syndrome is usually described as a manifestation of schizophrenia, althogh it also has been reported as occuring in association with affective disorders (Singer & Isbister, 1987).

However in recent years, inceasing number of reports have appeared in the literature describing Capgras Syndrome as a manifestation of organic brain disease. MacCallum (1973) presented five cases of the Capgras Syndrome occuring in association with toxicity and pneumonia, right sided hemiplegia secondary to basilar migraine, alcoholic paranoid state, poorly controlled diabetes mellitus and malnutrition. Capgras Syndrome has also been reported with various other organic conditions such as temporal lobe epilepsy (Chawla & Virmani, 1977), myxoedema (Madakasira & Hall 1981), viral encephalitis (Nickolvski & Fernandaz 1978), brain tumour (Todd et al, 1981), neuroleptic toxicity (Waziri, 1978), Vitamin B-12 deficiency (Zucker et al, 1981), hepatic encephalopathy (Cummings, 1985), chloroquine induced state (Bhatia et al, 1988), dementia (Kumar, 1987), sensory inattention (Stuss & Benson, 1984), bilateral subcortical lesions in the occipitotemporal and frontal region (Lewis, 1987).

We present below three case reports of Capgras Syndrome associated with organic brain disease.

## CASE REPORTS

Case 1 : A 48-year-old woman was admitted to the psychiatric ward complaining that her real husband, working in the armed forces, had been killed and an imposter had been sent by the enemy as her husband. She had this delusional idea for five months and asserted that she could differentiate him from her real husband. After having this symptom for two weeks, her husband noticed that the patient developed some abnormality in her gait and that the power to hold objects in both hands had also decreased. There was also some difficulty in understanding her speech. At times, she became violent and abusive against her husband. She also complained that she could hear voices of her two sons, crying for

help, though both of them were living in different cities far from her residence. She said that her husband was trying to kill her children.

On admission, she appeared confused; had neglected her personal hygiene. She was very much guarded at the time of interview and had the delusion that her real husband had been killed and replaced by an imposter, who was trying to kill her children. She had auditory hallucinations. There was no evidence of formal thought disorder. Physical examination revealed slurring of speech, decreased motor power and claspknife rigidity in both upper and lower limbs, although there was no sensory deficit. Both plantars showed the Babinski sign. The skull x-ray, C.S.F. and blood examination did not reveal any abnormality. Computed tomography (CT) of the brain showed an infarction in the right fronto-pariental region.

She was prescribed haloperidol 20-50 mg per day in divided doses. During the 10 weeks in the ward, she showed gradual reduction in her delusion and auditory hallucinations. At the time of discharge, she showed improvement in her neurological deficit and the Capgras Syndrome and hallucinations had disappeared. Haloperidol was given in doses of 15 mg per day for another four weeks and then, gradually reduced and discontinued. During follow up for one year, the patient did not develop these symptoms again, although the power in both lower limbs was still decreased.

## CASE 2

A 24-year-old female was brought to the psychiatric ward accusing her mother, father and brother of being imposters for the last 6 months.

The patient was apparently well upto ten years previously when on returning from school, she had an accident, was unconscious for about 5 minutes. She was admitted to hospital where the investigations, which included blood, CSF, and Xray skull, did not reveal any abnormality. Following this episode, however, the parents noticed that her scholastic performance had declined. Previously, she was very interested in the studies and secured good marks, but after this episode, she twice failed in her examinations. The parents also noticed that she had also gradually lost interest in family affairs. She preferred to remain alone and did not bother much about her appearance. About 6 months prior to her admission, her parents and brother observed that she had become suspicious. She started cooking food for herself and refused to eat any thing given by her mother. She stopped talking to her parents and brother. whereas she had no hesitation in talking to other relatives and friends. She started accusing her parents and brother of being imposters and alleged that they had killed her real parents and brother. She maintained that she could differentiate her real parents and brother from these imposters by the way they talked. She alleged that the person posing as her brother wanted to molest her and that parents were not only helping him, but were also trying to take her property after killing her with poison. At times, the patient also showed abusive and violent behaviour towards the parents.

On admision, she appeard apprehensive and guarded but alert, with blunting of affect. Her speech, though coherent, became irrelevant at times. There were delusions of persecution against her parents and brother. There was no evidence of any formal thought disorder or hallucinations. Her memory and orientation were normal. Physical examination did not reveal any neurological deficit. On the Wechsler Adult Intelligence Scale (WAIS), her performance I.Q. was 80 and verbal I.Q. 96. The routine blood, urine and Xray skull examinations were normal. CT Scan revealed frontal lobe atrophy. There was no past or family history of mental illness. The patient was kept on haloperidol 30 mg per day, which was gradually increased to 60 mg per day. She was hospitalized for 8 weeks during which she showed improvement. When discharged, she had no residual symptoms of the Capgras Syndrome. At 2 year follow up, it was reported that during this period of two years she had no delusions or any other symptom of Capgras Syndrome.

## CASE 3

A 50-year-old widow was brought to the psychiatric clinic by her son who complained that she talked irrelevantly, was forgetful and incapable of managing her own affairs. Her son had difficulty in looking after her because she insisted that he and her daughter-in-law were not real but imposters.

The patient was apparently well one year prior to the admission, when her son and daughter-in-law noticed that she was unable to trace articles kept by herself. She could remember events which had occured in the remote past, but had difficulty in recalling recent ones. For the past six months, she had become unmanageable as she continuously accused her son and daughter-in-law of being imposters. This delusional misidentification persisted for hours and at times for days. During these episodes her son and daughter-in-law tried to persuade her of their true identity by showing her photographs and giving other evidene through their relatives but without success. She complained to the visitors that the imposters of her son and daughter-in-law were living in her house and would kill her. For one month, she had also started talking irrelevantly and showed abusive and violent behaviour.

On being interviewed, she appeared confused and mutered to herself. Her recent memory and immediate recall were impaired but her past memory was intact. She was disoriented for time and place but could identify the relative correctly except her son and daughter-in-law. She insisted that her son and daughter-in-law were imposters, and were trying to kill her to take her money. There was no evidence of any hallucination, depersonalization or formal thought disorder. Her blood investigations and Xray skull were normal but a CT can showed generalized cerebral atrophy with dilatation of the ventricles. She was referred for a neurological opinion and was diagnosed as suffering from dementia of the Alzheimer type.

She stayed in the ward for about two months and was prescribed haloperidol 15 mg per day in divided doses. Her confusion, irrelevant talking and muttering to herself gradually subsided and the Capgras Syndrome also disappeared. During follow up for a period of six months, there was no recurrence of any of these symptoms including the Capgras Syndrome, although her recent memory was found to be still impaired.

## DISCUSSION

These three cases illustrate the organic dysfunction associated with Capgras Syndrome. In a study of ten patients by Christedoulou (1977), nine were having specific impairment of visuospatial perception and memory, signifying the regional deficit in Capgras Syndrome, more specifically in the non-dominant occipitoparietal region. Morrison and Tarter (1964) showed the evidence of frontotemporal impairment, in patients having Capgras Syndrome but it was not replicated by Merrin and Silberfarb (1976). In our three cases there was involvement of the frontal lobe. As reported by Kumar (1967) case 3 in our study had generalized degenerative disorder of brain of Alzheimers' type. Some authors (Hayman & Abrams, 1977; Alamanier et al, 1979; Quinn, 1981) have suggested that the syndrome is much more common in the localized right than the left hemisphere disease, but Weston and Whitlock (1971) described Capgras Syndrome in a young male who sustained head injury with bilateral temperopartietal damage. Some authors, however, reported Capgras Syndrome in left-sided brain pathology. Hay et al (1974) described a case of mental retardation suffering from pseudohypoparathyroidism. She developed Capgras Syndrome after a course of electroconvulsive therapy. Her E.E.G. showed medium voltage dominant theta activity with low voltage delta activity spreading into all the areas, more marked on left side. As the Capgras syndrome subsided, the EEG dysarrythmia also improved. Thompson et al (1980) reported that the CT findings were normal while Hayman & Abrams (1977) Quinn 1981 and Todd et al, (1981) found evidence of

cortical atrophy. Alexander et al (1979) also reported marked frontal and right temporal atrophy in a patient who developed Capgras Syndrome following head injury. On this basis, it was suggested that the frontal lobe impairment was important in the genesis of the syndrome. This suggestion is further strengthened by the findings of our cases. In case 1. there was isolated involvement of the frontal lobe, in case 2 frontal lobe and the adjacent parietal lobe were involved while in case 3 there was generalized atrophy of the cerebral contex.

Lewis (1987) reported a patient developing Capgras Syndrome as a part of an interictal psychosis in which magnetic resonance imaging (MRI), revealed bilateral subcortical lesion in the occipito-temporal and frontal regions. Several authors (Hayman & Abrams, 1977; Sharberg & Weitzel, 1979) have suggested that prosopagnosia (failure to recognise the familiar faces) may be the primary expression of a specific cerebral dysfunction which forms the basis for a delusional elaboration resulting in Capgras Syndrome. But in cases of Capgras Syndrome, no satisfectory evidence of prosopagnosia could be found. Meadows (1974) studied 42 patients of prosopagnosia and found visual field defects in 38 cases. This was, however, not found in our cases. The literature on prosopagnosia (Cohn et al, 1974; Meadows, 1974) indicates that the patients who suffer from prosopagnosia fail to recognise previosly familiar faces and when auditory or other clues induce recognition, they immediately acknowledge the identity of the person with neither paranoid fear nor allegation of imposters. The prosopagnosia is unlikely in our cases as the visual and other cues failed to induce recognition.

Another phenomenon closely linked with Capgras Syndrome is depersonalization-derealization (Todd et al, 1981). In patients with Capgras Syndrome, there is feeling of unfamiliarity which may originate from derealization. However, the phenomenon of depersonalization-derealization itself cannot be the basis of the origin of the Capgras Syndrome because the patients afflicated by depersonalization derealization report an 'as if' feeling and have insight into the illusionary nature of the phenomenon, whereas the patients with Capgras Syndrome have firm delusions without such insight. Depersonalization-derealization phenomenon was not present in our cases.

Chasla et al (1987) have reported intrapsychic stress and difficulties in interpersonal relationship as the precipitating factor in their case, but in our cases, there has no evidence of any such precipitating factors.

Berson (1983) mentioned the preconditions for developing Capgras Syndrome as a psychotic state, a marked paranoid tendency and pathological splitting of internalized object representations. The process of splitting of internalized object representations (good and bad self) has been an explanatory hypothesis capable of illuminating Capgras Syndrome (Cavenar et al. 1977; Dally & Gomez 1979). Mahler (1974) described it as the result of disturbance in the mother child interaction especially in the separation-individuation proess, but it is not clear how the disturbance in mother child interaction, the evidence of which is found only in few cases, resultes in Capgras Syndrome.

Though, there have been numerous reports supporting the psychological mechanism of Capgras Syndrome but the evidence demonstrating the lesions in the frontal lobe, temporal lobe and occipito-temporal lobe have strengthened the conceptual link between organic brain dysfunction and Capgras Syndrome.

Thus, it appears that all the patients presenting with Capgras Syndrome should be investigated thoroughly for an organic pathology, as this syndrome does not appear to be specific to functional psychosis, as was believed in the past.

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