THE SYNDROME OF CAPGRAS — A CASE REPORT

Tan Chue Tin

SYNOPSIS

A case of Capgras Syndrome is described and the psychopathology discussed. This is the first reported case in Singapore.

INTRODUCTION

The Syndrome of Capgras is a rare psychopathological manifestation characterised by the delusional negation of the identity of a familiar person. The patient believes that a close relative has been replaced by a double, and although he does not dispute the misindentified person's extreme resemblance to the familiar person, he nevertheless believes they are in fact different. The following report describes a case seen recently at Woodbridge Hospital and discusses the presenting clinical, neuro-psychiatric and psychopathological features with reference to the recent literature.

CASE REPORT:

This is a 63 years old "mentally deranged" housewife who lived in seclusion for the past 10 years. She was formally admitted on 14th July 1978 to Woodbridge Hospital because she "consituted a danger to life and property". This is because her neighbours saw smoke coming out of her house frequently and lodged complaints to the local police.

She was borned in China in 1915 and migrated to Singapore at age 25, eight years after her marriage to a farm hand. Both her parents died when she was 3 years old and she was subsequently brought up by her paternal uncle until her marriage at age 17. Coming from a well to do family background, she had led a sheltered and 'pampered' way of life since young and never had to do any housework until she migrated to Singapore with her husband.

Her marriage was a failure right from the start, with frequent stormy quarrels and husband often showing outbursts of temper and violent behaviour. Being brought up in a traditional and conservative manner, she has always longed for a son — but instead gave birth to 4 daughters in succession, 2 of whom were given away at birth for adoption. Thus she felt guilty that she had failed in her chief role as a 'good wife' to produce 'many sons!' This guilt was reinforced and made worse when, after the birth of her youngest daughter, her husband found a mistress who bore him a son on her first pregnancy. This feeling was later projected on to a delusional idea that she had 2 sons.

Woodbridge Hospital
Singapore

Tan Chue Tin, MBBS, DPM, MRC Psych., MRANZCP.
Senior Registrar

Her premorbid personality was describ d as a gentle, soft spoken, introverted woman who does not like to mix or socialise with people, not even relatives. She was submissive and dependent on her husband and during the quarrels would remain passive and quietly tolerated all her husband's verbal and occasionally physical assaults. She was generally unhappy although she did not express her inner feeling easily to anyone. There was however no history of a significant depressive or hypomanic episode for any length of time, and there was no disturbance of appetite, sleep, weight or other physiological parameters suggestive of depression.

The event that appeared to precipitate the present illness was the decision by her husband to leave her and stay with his mistress in 1959. She was then 44 years of age. Over the past 19 years her mental state progressively worsened. Initially she was suspicious and fearful of people wanting to harm her. She complained of being charmed by Malay 'black magic' and believed her relatives were collaborating with the Malays to do harm to her and her daughters. She became homebound because of this fear.

Sometimes in 1966, her delusions became worse and she was also noted to talk and laugh to herself and complained of hearing voices. In 1968, when her youngest daughter returned from Australia after a nursing course, she claimed that she (daughter) was not the real daughter but was a replacement by an exact double. Later her other daughter and the President of Singapore were also noted by her to be replaced by imposters with exactly similar faces.

From then on, she gradually isolated herself from the outside world. Even her "daughters" were not allowed into the house as they were "imposters" but only permitted to pass food daily through the front gate. The house was poorly kept and patient's personal hygiene and habits deteriorated. The total seclusion and withdrawal rendered her inaccessible to early medical intervention. It was only when the neighbours made several complaints to the local police about smoke coming out of her house, and about her deranged mental state and isolation that the local authority decided she posed a danger to life and property and, with the consent of her daughters, entered her house by force and brought her to Woodbridge Hospital to be formally admitted for psychiatric observation and treatment.

Physical examination revealed a thin, unkempt but satisfactorily nourished woman wearing several layers of thick dirty and soiled clothings. The left elbow was heavily bandaged to cover an imaginary 'growth'. There were no abnormality on general systems examination. Her blood pressure was normal, and the peripheral vessels were not thickened. Carotid bruits were absent. There was no physical evidence of vitamin deficiency states.

Mental state examination revealed formal thought disorder, inappropriateness of affect, and systematised paranoid delusions and auditory hallucinations in the absence of a depressive or hypomanic mood state. There were symptoms of depersonalisation in that she claimed her left elbow had a large growth (despite my showing her the unbandaged normal elbow), that her eyes were gradually shrinking in size to that of a rat's eyes, and that her face was different from her original face because it had undergone three transformations. There was no derealisation symptoms or the deja-vu phenomenon. The features of the syndrome of Capgras have been noted, she believing that her real daughters and the President of Singapore were replaced by exact doubles.

There were also evidence of cognitive deficits in that her attention, concentration, temporal orientation, memory and intellectual performance were all impaired. Further examination for organic cerebral disease was generally negative, except to confirm the presence of the cognitive deficits already noted. Symptoms of anxiety, depression, hypomania, phobic and obessional states, hypochondriasis, visual hallucination, and derealisation were absent.

In investigations, the basic routine urinalysis, haemotology and syphilis serology were normal. The ESR was not raised. Blood urea and sugar were normal. X-rays of the chest and skull were normal. The EEG showed bilateral abnormal slow waves and 'epileptic' discharges bitemporally. The computerised axial tomography of the brain indicated cortical atrophy, but with no ventricular enlargement, shift of intracranial structures or change of density. Examination of the CSF was normal.

Results of psychological assessment were suggestive of organic cerebral dysfunction. The WAIS Verbal IQ was 85, while Performance IQ was 72, a discrepancy of 13. The Benton Visual Retention Test revealed impairment of short term memory and cerebral function.

She was diagnosed as a case of Chronic Paranoid Schizophrenia associated with a) the later development of the Syndrome of Capgras and b) an underlying organic cerebral dysfunction.

Once diagnosis was established patient was started on Sparine 150mg per day in divided doses. Sparine was chosen because of patient's age and her small body frame. Four weeks later, when asked about her sons, she replied that she had no son, but had four or maybe six daughters. There was noticeable improvement in the irrelevent, irrational thinking and she was observed to talk more 'within the points'. There was no more auditory hallucination. She bathed daily and took care of her personal hygiene and continued to do this after discharge. She gained partial insight and accepted the inevitable demolition of her terrace house, and her having eventually to live with her daughters. By the eighth week of treatment, there were no systematised delusions of persecution.

The features of Capgras however persisted and despite the improvement shown, patient felt obligated to the 'impersonating doubles' who had so kindly provided her with a place to lodge. On the twelveth weeks of Sparine treatment she gained full insight into her condition, including her Capgras delusions. The cognitive deficits however persisted.

DISCUSSION

The syndrome of Capgras, a rare psychopathology manifestation, was originally described in 1923 by Capgras and Reboul — Lechaud who termed it 'l'illusion des sosies' (the illusion of doubles). It was in 1929 that Levy-Valensi called it 'syndrome of Capgras'. The essence of the syndrome is the delusional negation of identity of a familiar person. The patient believes that a person closely related to him has been replaced by a

double, and although he does not dispute the misidentified person's extreme resemblance to the familiar person, he nevertheless believes they are in fact different.

This is the presentation of this patient. Owing to the rarity of the Capgras syndrome, most publications deal only with case descriptions except for Christodoulou (1977) who attempted a systematic study of this syndrome.

Most investigators have commented on the psychotic background (mostly Schizophrenia), and irrespective of diagnosis, the clinical picture is usually of a marked paranoid type. This is clearly illustrated by this patient.

Most patients in Christodoulou's series and other case reports (Nilsson and Perris 1971; Vogel, 1974; Lansky, 1974 and others) developed depersonalisation feelings. Ackner (1954) held the view that depersonalisation experiences tend to be included within the delusional system if there is a delusional development, with accompanying reduction of the feelings of strangeness and unreality. This inverse relationship means that the syndrome of Capgras may represent a delusional evolution of the phenomena of depersonalisation. This case, have both the depersonalisation and Capgras features, but I was unable to verify the inverse relationship noted by others mainly because the patient was living alone for the past 10 years with the Capgras syndrome, and the depersonalisation symptoms were only noted during her stay in the ward. It is not known if she had depersonalisation features before.

In recent years there is an increasing number of reports describing the Capgras syndrome as a manifestation of organic brain disease (Gluckman, 1968; Weston and Whitlock, 1971; MacCallum, 1973; Faber, 1975 and Christodeulou, 1977).

The investigations findings in this case support this view on the grounds of an abnormal EEG with 'epileptic' discharges, a marked difference (13 points) between the verbal and performance IQ, a deficient performance in the Benton Visual Retention Test, and a computerised tomographic brain picture of cerebral atrophy. Such abnormal laboratory findings are frequent in the reported cases just mentioned and the general concensus is that a primary cerebral dysfunction probably contributes to the pathogenesis of the syndrome of Capgras. My view is that the number of cases so far reported is too small to make any meaningful conclusion on this interesting tripartite relationship between the basic functional psychotic illness, the cerebral dysfunction and the evolution of Capgras syndrome.

In this patient, the disappearance of the Capgras features by the twelveth week of Sparine treatment suggests that the syndrome is more related to the psychosis than the cerebral dysfunction. All the evidence in the literature so far only suggests that the appearance of Capgras syndrome in the setting of a functional psychosis calls for the careful investigation (and possibly treatment) of organic contributing factors, especially cerebral dysfunction.

REFERENCES

1. Ackner, B: Depersonalisation. II Clinical Syndromes. Journal of Mental Science, 100: 854-72, 1954.
2. Christodoulou, G.N.: The Syndrome of Capgras. British Journal of Psychiatry, 130: 556-63, 1977.
3. Gluckman, J.K.: A case of Capgras Syndrome. Australian and New Zealand Journal of Psychiatry, 2: 39-43, 1963.
4. Faber, R and Abrains, R: Schizophrenia in a 47, XYY male. British Journal of Psychiatry, 127: 401-3, 1975.
5. Lansky, M.R.: Delusions in a patient with Capgras Syndrome. Bulletin of the Menninger Clinic, 38: 360-4, 1974.
6. MacCallum, W.A.G: Capgras symptoms with an organic basis. British Journal of Psychiatry, 123: 639-42, 1973.
7. Nilsson, R. and Perris, C: The Capgras Syndrome: a case report. Acta Psychiatrica Scandinavica, 222: 53-8, 1971.
8. Vogel, F.B.: The Capgras syndrome and its psychopathology. American Journal of Psychiatry, 131: 922-4, 1974.
9. Weston, M.J. and Whitlock, F.A: The Capgras Syndrome following head injury. British Journal of Psychiatry, 119: 25-31, 1971.