

CAPGRAS SYNDROME IN CHLOROQUINE INDUCED PSYCHOSIS

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Introduction

Capgras syndrome is considered a rare syndrome in which the patient believes that a person usually a close relative, has been replaced by an exact double or imposter and maintains this false belief despite evidence to the contrary (Capgras and Reboul-Lachaux 1923). Capgras' syndrome has usually been described as a manifestation of schizophrenia. Some others (Vogel 1974, Hayman and Abrams 1977) believe that, since this syndrome occurs in clear sensorium, there is no organic basis for this. However in recent years an increasing number of reports have appeared describing the Capgras' syndrome as a manifestation of many organic conditions (Alexander 1979, Madakasira and Hall 1981, MacCallum 1973).

Though, literature till date has reported only few cases (Kimura et al. 1981, Sullivan et al. 1978, Waziri 1978) of Capgras' syndrome as being drug induced, no such report is available in children.

We report a case, of Capgras' syndrome in a child secondary to toxic psychosis due to Chloroquine therapy.

Case Report

A 9-year-old girl was admitted with fever with chills and rigors, headache and

bodyache. Blood investigations showed mild leucocytosis and malarial parasite. The patient was put on chloroquine sulphate 0.5 gm followed six hours later by 250 mg chloroquine. On second day 125 mg chloroquine was given. On third day morning, she became restless, followed by outburst of abusive, violent behaviour. She continuously maintained that her parents were dead and the actual persons accompanying her, though look-alikes of her parents, were imposters. The detailed physical and neurological examination did not reveal any abnormality. The urine, blood and CSF studies, X-ray chest and skull, fundus examination, EEG, were within normal limits. Mental status examination revealed irrelevant talks (in relation to imposters), poor judgement, and lack of insight.

There was disorientation to time and mild impairment of recent memory. She continuously believed that her father and mother were dead and the actual persons, though look-alikes of her parents, were imposters. Prior to admission, she had no any past or family history of mental illness. The diagnosis of toxic psychosis was made and chloroquine was immediately stopped. She was put on chlorpromazine 300 mg daily in divided doses.

Within four days, her mental status

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improved and so the drug was stopped. The child became completely normal within 10 days and accepted her parents. The patient was followed up for six months and she did not show any abnormal behaviour.

Discussion

The psychosis due to chloroquine has been reported in adults (Reagan 1985) and recently, in children also (Bhatia et al. 1988) but Capgras' syndrome resulting from chloroquine intake has not been reported in literature. A diagnosis of chloroquine psychosis, in the present case, was made on the basis of the similarity of the patient's symptoms to the reported cases (Reagan 1985, Bhatia et al. 1988), the absence of previous psychiatric history, neurological and related investigations being within normal limits and rapid recovery on stoppage of chloroquine.

The various hypotheses put forward to explain the mechanism by which chloroquine can induce psychosis are through cholinergic imbalance (David and Berger 1978), prostaglandin-E-antagonism (Malik-Ahmadi 1981), a predisposing factor of G-6-P-D-deficiency (Malik-Ahmadi 1981), or due to idiosyncratic or antigen-antibody reaction triggered by chloroquine in previously sensitized people (Bhatia et al. 1988).

Although Capgras' syndrome has been reported in many functional and organic conditions, the exact mechanism by which it is induced, is still not fully understood. The psychoanalytic school believes that it results due to disturbances in mother-child interaction, especially in the separation-individuation process (Mahler 1974). The process of splitting of internalized object representations (into 'good' and 'bad' self or object representation) has

also been an explanatory hypothesis for Capgras' syndrome.

Bodamer (1947) suggested that inability to recognize familiar faces is a quite specific variant of visual agnosia which he called prosopagnosia (i.e. 'face non-recognition') but classical prosopagnosia, in the sense of an isolated loss of the ability to recognise previously familiar faces has never been satisfactorily demonstrated in patients with Capgras' syndrome (Lewis 1987). The idea of a possible relationship between prosopagnosia and Capgras' syndrome has been rejected for several reasons. Firstly, they have not been shown to coexist; patients with prosopagnosia do not show psychiatric symptoms and conversely, classical isolated prosopagnosia is not displayed in Capgras' syndrome to the identity of those most closely familiar to the patients is an observation often used to discount organic explanations in favour of psychodynamic ones (Berson 1983), yet prosopagnosia sometimes show exactly the same pattern. Lastly, critics point to the lack of an established neuropathology common to both but recently, a case of Capgras' syndrome as part of an interictal psychosis on Magnetic Resonance Imaging (MRI) was found to have bilateral subcortical lesions in occipitotemporal and frontal regions (Lewis 1987). This case strengthens the conceptual link between the two disorders. They remain separate clinically, but in at least some cases of Capgras' syndrome, a neuropsychological mechanism analogous to that postulated for prosopagnosia might be at work. Pincus and Tucker (1974) suggest that this phenomenon originates in a single retrolandic parietal lobe lesion and occurs three times more frequently with lesions of the right cerebral hemisphere than with lesions in the left but in our case, there was no demonstrable brain lesion.

This case report adds to the list of causes resulting in Capgras' syndrome.

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