

Case Report

Capgras Syndrome in a Patient with Severe Hyponatraemia

Vijay Delaffon, Arshya Vahabzadeh, Srinivas Naik, Daniel Kinnair and Janet Bruce

The Brandon Mental Health Unit, Leicester General Hospital
Leicestershire Partnership NHS Trust, Gwendolen Road, Leicester, UK LE5 4PW

Corresponding author: Vijay Delaffon MRCPsych, Brandon Mental Health Unit, Leicester General Hospital, Gwendolen Road, Leicester, UK LE5 4PW. vijay.delaffon@yahoo.com

Abstract

This case report describes Capgras delusion occurring in a previously well 63 year old woman following correction of severe hyponatraemia. It raises interesting points of discussion around etiological models to explain Capgras delusion and the neuropsychiatric complications associated with the correction of severe hyponatraemia (German J Psychiatry 2010; 13 (4): 175-177).

Keywords: Delusional misidentification syndromes, capgras syndrome, severe hyponatraemia, osmotic demyelination syndrome

Received: 4.8.2010

Published: 31.12.2010

Introduction

Capgras syndrome is an uncommon syndrome in which the patient has a delusional belief that a person, usually a relative, has been replaced by an impostor or double. The syndrome was first described in 1923 (Capgras & Reboul-Lachaux), and it is now classified as one of the delusional misidentification syndromes. The second case was reported in 1924 and there have been various case reports and variations of the originally described phenomenon since. Whereas it was previously thought to be more often associated with purely 'functional' illnesses, a long list of organic conditions with which it is associated is emerging. These include case reports of Capgras syndrome occurring as a postictal phenomena, (Drake 1987), in Alzheimer's disease (Ballard et al., 1995), Parkinson's disease (Roane et al., 1998), and right hemisphere white matter pathology (Edelstyn et al., 2001). Here, we report a case of a 63 year old lady who developed Capgras on the background of severe hyponatraemia. Hyponatraemia is a common electrolyte abnormality associated with neurological complications and mortality. To our knowledge this is the first case report

associating hyponatraemia with the uncommon Capgras syndrome.

Case Report

A 63 year old married lady was apparently well until she developed a cough, fever, diarrhoea and vomiting of approximately one week's duration. She was witnessed by her husband as having a single generalised tonic clonic seizure at home and was taken to hospital where she was investigated. Her investigations revealed severe hyponatraemia (sodium 108 mEq/L, Urinary sodium 11 mmol/l, urine osmolality 442 mosmol/kg, serum osmolality 226 mosmol/kg). Routine urea and electrolytes done two months prior to this episode were within normal limits. Other investigations including full blood count, C-reactive protein, autoimmune screen, liver function tests, lumbar puncture, and EEG done at the time of admission were unremarkable. An MRI of the brain revealed a mild degree of small vessel cerebrovascular disease with no evidence of recent infarction, haemorrhage, or mass lesion.

She was diagnosed to have had a seizure secondary to hypovolemic hyponatraemia and bendroflumethazide, which she was on for hypertension was discontinued. Her electrolyte imbalance was corrected with isotonic saline over a 48 hour period and discharge was being planned when she was noticed to be withdrawn and preoccupied. A referral was made to the liaison psychiatric team whose assessment did not identify any immediate risks and discharge with follow up in psychiatric outpatients was recommended.

At home over the next 3–4 weeks she continued to be withdrawn and became mute. She neglected her self-care and had poor food and fluid intake and disturbed sleep. She occasionally described the feeling that objects within the home were being moved and ‘cameras and microphones’ were monitoring her actions. She would not interact with friends and family who became increasingly worried for her health. This was her first presentation to psychiatric services, with no significant past psychiatric history or family history.

Apart from hypertension she was physically well; she did not have any sensory impairment and had no past history of a seizure disorder. Her memory and social functioning had been good, and until this episode she had enjoyed an active social life. The patient was a non-smoker and did not drink alcohol regularly.

At admission to a general psychiatric ward (about 7 weeks after the seizure) a complete physical examination, including a thorough neurological examination was unremarkable. Further investigations including a dementia screen, EEG, and a repeat of previous blood tests were done and found to be within normal limits. In hospital she refused antipsychotic medication and physical health medications, and personal care and oral intake of food and fluid declined further. She became further withdrawn with psychomotor retardation.

A diagnosis of catatonia was made and she was treated with twice weekly electroconvulsive therapy (ECT). A dramatic improvement in her mental state was seen after the third session of ECT and she was described by herself and by the family as being ‘back to normal’. No further ECT was given but she was observed in hospital over the next week and then had graduated periods of leave from hospital which were uneventful. She was eventually discharged from the hospital.

Post recovery she described how she had been convinced that her husband had been replaced by a ‘double’. She could see that ‘the scar on his forehead, and his eyes were different’. She confirmed this by comparing his face to a photograph that hung on their wall and based on these subtle differences she concluded that there were two different doubles of her husband. The patient has had two readmissions since and responded again to ECT and has maintained remission on long term antipsychotic medication.

Discussion

The case presented here has two aspects to it, the psychiatric sequelae following a severe hyponatraemic episode and the

Capgras delusion itself. The investigations and history suggest extra renal loss of sodium (mainly gastrointestinal), possibly exacerbated by diuretic medication. Homeostatic mechanisms in the brain do not react rapidly to acute falls in sodium potentially leading to cerebral oedema, seizures and irreversible neurological damage. In a sample looking at 184 patients treated for severe hyponatraemia, it was found that 0.5% suffered hallucinations, 3.3% seizures and 0.5% from acute psychosis (Ellis 1995). Further, the correction of hyponatraemia is in itself associated with complications like central pontine and extrapontine myelinolysis (CPM/EPM) – the osmotic demyelination syndromes (ODS). EPM in particular has been associated with neuropsychiatric presentations including behavioural changes, mutism (Price & Mesulam 1987), Parkinsonism (Tomita et al., 1997), and catatonia (Chalela & Kattah 1999). The ODS can present with behavioural symptoms including akinetic mutism and catatonia with normal MRI and transient corticospinal tract signs (Chalela & Kattah 1999).

In the case described here, no neurological signs were picked up at psychiatric admission; however, there was evidence of mutism, behavioural changes and catatonia. We argue that the possibility of ODS cannot be ruled in light of the clinical presentation on the background of correction of severe hyponatraemia of which is a well known risk factor.

Christodoulou et al. (2009) have reviewed the pathogenic hypotheses that have been proposed to explain the Delusional Misidentification Syndromes including Capgras syndrome. One of the hypotheses proposed by Ellis and Young (1990) attributes the phenomenon to defects in information processing in relation to face recognition. Accordingly, they have used Bauer’s (1984) dual recognition model for facial recognition which suggests that there is a ventral route from the visual cortex to the temporal lobes through the inferior longitudinal fasciculus responsible for overt or conscious recognition and a dorsal route from the visual cortex to the limbic system via the inferior parietal lobule which is responsible for covert recognition or recognition at an unconscious level. They proposed that when there was a disconnection of the dorsal route the person perceives the facial image (of say a family member) through the intact ventral route which stimulates stored appropriate semantic information but due to the dorsal disconnection there is a lack of a set of information which carries affective confirmation associated with the person. The delusion of a double or imposter is explained thus as a rationalisation strategy when the patient is faced with a conflict of recognition of the face with no emotional familiarity.

In the case of our patient the disconnection hypothesis as posited above seems to be suitably explanatory. Functional disconnection as a result of cerebral oedema may explain the emergence of the DMS with subsequent resolution of symptoms over time. To the best of our knowledge this is the first report of delusional misidentification evolving on the background of hyponatraemia and its correction.

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