

# MANAGEMENT OF CHARLES BONNET SYNDROME IN THE ELDERLY

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## ABSTRACT

*The case of an 82 year old lady who suffered from vivid episodic complex visual pseudohallucination is presented. The alleviation of symptoms with carbamazepine monotherapy is highlighted. Correction of visual defect as another aspect of the management of Charles Bonnet syndrome in elderly patients is recommended.*

*Key Words : Charles Bonnet syndrome ; Carbamazepine ; visual defect ; pseudohallucination.*

## INTRODUCTION

Charles Bonnet Syndrome is the eponym given to vivid complex, monomodal visual pseudohallucination occurring usually in elderly individuals in the absence of psychosis and cognitive impairment. Usually the visual experiences are vivid, episodic and picturesque with the sufferer having full insight into its unreality (Adams & Victor, 1903). Victor and Adams (1993) described the syndrome of Bonnet as ophthalmopathic hallucination occurring in patients with partial or full visual sensory defect or deprivation.

## CASE REPORT

An 82 year old housewife, mother of five children, from Jakarta, who did not have any history of mental illness, complained of episodic visual hallucination of vivid scenes for a period of one year. She was having bilateral cataract with reasonably good vision in right eye (visual acuity left 6/60, right 6/24) and had got admitted in JIPMER hospital, Pondicherry for cataract surgery. These visual experiences which contained scenes in and around her house in Jakarta, like the kitchen, the garden, the panoramic landscape around, people entering and leaving the house, etc, were transient. She could narrate and list the things she saw. From her description the patient's daughter could make out similarity between her

visual experiences and the facilities available in her home in Jakarta. Patient was aware and amused that she was in JIPMER hospital, while having these unreal colourful and panoramic visual experiences. She added that whenever she wanted to have a closer look at the scenes she saw, it disappeared and she could not bring back these scenes voluntarily. There was no alteration of consciousness preceding, during or following the hallucinatory experience. There was no history suggestive of complex partial phenomena, other temporal lobe phenomena or episodic abnormal behaviour. Her psychomotor status was normal. Her primary mental functions were normal even during the episodes. She was cordial to the attending doctors. There were no neurological soft or hard signs. There were no frontal lobe release signs. Pulse was 70/mt; blood pressure was 170/100 mmHg with no postural fall. Other systems were clinically normal. Her haemogram, blood biochemistry including liver and thyroid function tests were normal. Her blood Venereal Disease Research Laboratory test and ELISA for HIV antibodies were reported negative. Her electroencephalogram and computerised tomography of the brain were normal. Neuropsychological tests for organicity were within normal limits. Her ability to narrate her history and different

topics was normal with regard to chronological sequence, logic and association. Her electrocardiogram was normal. Doppler imaging revealed mild aortic stenosis, mild mitral regurgitation and both mitral leaflets were found to be prolapsing mildly to the left atrium; all the three cusps of the aortic valve showed mild thickening.

With a diagnosis of Charles Bonnet Syndrome she was treated with carbamazepine 400 mgs daily in divided doses (100-100-200). She became completely asymptomatic within two weeks. She underwent successful left sided cataract extraction and was discharged after two weeks on the same dose of carbamazepine. She was given aphakic spectacles for the left eye and advised to undergo cataract surgery for the right eye in due course. She was followed up for a period of four months till she went back to Jakarta. She stopped medicines after another four months, and for a period of one year she was asymptomatic and functioning well. Then she fell down and sustained a fracture neck of femur and a 3 cm cut injury to the left side of the forehead. Her relatives brought her back to JIPMER hospital where she was admitted for Austin-Moore hemi-arthroplasty for Garden Type II fracture neck of femur. Detailed clinical, laboratory, and radiological investigations repeated in the same way as before were normal. Vision in right eye had deteriorated to 5/60. She withstood the surgical procedure well. During the post operative period she had the same episodic visual experiences. Her mental status and repeat psychodiagnostic evaluation were normal. She was put back on carbamazepine 400mg daily with which she became asymptomatic within two weeks. Her orthopedic status was satisfactory at the time of discharge. She was advised to continue carbamazepine and regular physiotherapy.

## DISCUSSION

Ribeiro et al (1989) reported a case of Charles Bonnet Syndrome associated with blindness due to bilateral eye disease and a posterior parasagittal meningioma. These authors were of the view that

apart from the visual defect, a 'cerebral factor' is operative if hallucination is to occur. They highlighted the fact that one should not put the blame on obvious eye or visual pathways affection when facing cases of Bonnet Syndrome. These areas will not explain the complex array of images perceived by any given patients. These authors advise that one should intensively look for and rule out covert intracranial disease by appropriate investigation.

McNamara et al (1982) reported the case of a 64 year old lady who had 14 years of complete blindness following which she developed Charles Bonnet Syndrome; neurosurgical removal of a suprasellar meningioma made the hallucination disappear. Suzuki et al (1992) reported a 65 year old male who suffered from vivid colourful complex visual hallucination. He had a small infarction in the left tegmentum on magnetic resonance imaging. Nakajima (1991) reported a 50 year and a 70 year old patient having computerised tomography proved frontomedial cerebral infarction. Both of them suffered from complex, vivid, colourful visual hallucinations. One of Unni's (1994) patients who was only 41 year old, had a self limiting Charles Bonnet Syndrome whereas the other, a 53 year old patient refused carbamazepine therapy and continued to be symptomatic.

When one takes up a neuropathological approach to the diseases affecting perception, one or the other subtle changes may be demonstrable in the brain but it's relevance to the disorder will be difficult or impossible to establish (Corsellis and Janota, 1985). The present patient did not have any computerised tomography proved intracranial disease. Even then the "cerebral factor" as proposed by Ribeiro et al (1989) can be speculated and conceptualised in the following manner: Hosty (1990) had reported that Charles Bonnet syndrome is more common in the elderly. The present patient is an 82 year old lady who may have age related neuronal loss. Corsellis and Janota (1985) are quoting Monagle and Brody (1955) and Henderson, Tomlinson and Gibson

(1980) to say that neuronal loss in inferior olivary areas of hind brain will be much lesser than the superior temporal gyrus. The same authors are quoted by them to point out that in the ninth decade of life there is approximately a 50% neuronal loss. The aging brain is susceptible to faulty circulation (Corsellis and Janota, 1985). Doppler imaging studies have demonstrated atherosclerotic changes in different areas of the patient's heart. The present patient has got an aging brain with probably a faulty circulation, subtle changes not demonstrated by computed tomography scan. The effect of general anaesthesia and water and electrolyte imbalance while on IV fluids superadded to the aging brain and deteriorating vision in her right eye might have triggered the visual pseudo hallucination during the second postoperative period. The period of normalcy without medication prior to her second hospital stay could be explained by the fact that she had good vision in her left eye after the lens extraction. Based on the literature on Charles Bonnet syndrome and the present patient, the authors would like to highlight that a "cerebral factor" and an "ophthalmic factor" may be working together in an interactive way for the clinical production of Charles Bonnet syndrome especially in elderly patients.

Nakajima (1991) highlighted the possibility of occipital or temporal lobe involvement in clinical cases of complex visual hallucination. It is striking that in the majority of cases reported in elderly Charles Bonnet syndrome patients, temporal lobe or cerebral peduncle are involved. Bhatia et al (1992) had successfully treated their young patients suffering from Charles Bonnet syndrome with carbamazepine. Their report goes in favour of attributing temporal lobe affection as a causative factor for Charles Bonnet syndrome. The present patient also responded well to carbamazepine monotherapy. The authors would like to have the view that probably the causative "cerebral factor" in Charles Bonnet syndrome in the present case could be subtle affection of the temporal lobe. Review of literature shows that

tegmental infarction, frontomedial infarction, suprasellar meningioma, or posterior parasagittal tumours have also produced Charles Bonnet syndrome. So whatever be the logical arguments regarding localisation of lesion in the present case, a generalisation cannot be done based on the present knowledge of the clinical entity.

It is felt that the original concept of Charles Bonnet syndrome is to be broadened to accommodate the "ophthalmic factor" and the "cerebral factor". The correction of the visual defect side by side with the correction of the cerebral factor is recommended in the management of Charles Bonnet syndrome in the elderly. Berrios and Brook (1982) have already challenged the original concept, as the syndrome, as per their review of literature, has become progressively enlarged.

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