

Charles Bonnet Syndrome Occurs Due to the Occipital Lobe Infarction

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Abstract

CBS can be defined as a complex and recurrent syndrome which is characterized by the presence of shaped and vivid hallucinations, the complete protection inside as well as the absence of delusions and cognitive deteriorations. The most frequent reason for CBS is the senile macular degeneration. Other reasons can be enucleation, optic neuritis, retinitis pigmentosa, diabetic retinopathy, macular laser photocoagulation, Leber's hereditary optic neuropathy and macular translocation. In this study, we reported a patient who was admitted to the hospital due to complex hallucinations and who had isolated ischemic occipitoparietal lesion.

Keywords: Charles Bonnet Syndrome; Hallucinations; Infarct

Introduction

Charles Bonnet Syndrome (CBS) was defined for the first time in the 1760s by Charles Bonnet. Bonnet realized that his grandfather had visual hallucinations upon being blind after the cataract operation although he had normal cognitive skills [1]. Bonnet had the same problems in the last period of his life and Morsier defined this clinical issue as 'Charles Bonnet Syndrome' in 1967 [2]. CBS is a clinical syndrome characterized by visual hallucinations even though cognitive skills are conserved. It can be observed in the damage of central visual pathways and the occipital lobe lesions together with ophthalmologic diseases. Occipital lobe lesions are an important reason for the visual field defects as well as simple and elementary hallucinations. Complex hallucinations which contain humans, animals and landscape images are observed more in occipitotemporal and occipitoparietal visual cortex lesions [3]. In this study, we reported a patient who was admitted to the hospital due to complex hallucinations and who had isolated ischemic occipitoparietal lesion.

Case

The 63 years old male patient was admitted to our clinic for sudden onset of visual hallucinations three days ago. Patient stated that he observed various bright animals and people who were sitting. He specified that he was observing these images continuously during the day even though they sometimes disappeared. Patient was aware of that these images were not real but he specified that he was admitted to the hospital because these images scared him. The patient did not have an important health problem according to his anamnesis. As a result of the neurological examination, there was no pathological finding except left homonymous hemianopia. His mini mental score was 28/30 and there was an infarct area in the right parieto-occipital region according to the tomography. The medical treatment was applied after he was examined in terms of the ischemic stroke. Carbamazepine was prescribed for hallucinations and he was asked to come for

medical follow-up one month later. After one month, he still had complaints, and therefore; 50mg/day quetiapine was administered. One week later, his complaints were partially improved and hallucinations disappeared entirely one month later.

Discussion

CBS can be defined as a complex and recurrent syndrome which is characterized by the presence of shaped and vivid hallucinations, the complete protection inside as well as the absence of delusions and cognitive deteriorations [1]. The most frequent reason for CBS is the senile macular degeneration. Other reasons can be enucleation, optic neuritis, retinitis pigmentosa, diabetic retinopathy, macular laser photocoagulation, Leber's hereditary optic neuropathy and macular translocation [4]. It can also be seen in the damage of central visual pathways and the occipital lobe lesions together with ophthalmologic diseases [3]. In our case, CBS was developed upon right occipital infarct. According to the neurological examination of our patient, we observed left homonymous hemianopia. The pathophysiology of this syndrome has not yet been clarified even though some studies have tried to explain it with various mechanisms. According to the 'deafferentation status', decreased sensory and/or visual skills stimulate intracerebral perceptions as happens in phantom pain syndrome [5]. On the other hand, according to another theory named 'perceptual release', increased perception leads to the decrease in the high cortical center suppressions and it generally causes the liberalization of the inhibited perceptual ways. Rosenbaum specified that there was a spontaneous cell discharge from the visual association cortex because of the decreased normal afferent stimulus due to the blindness and he defined the 'irritable cortex' concept in 1987. This theory 'sensory deprivation' was also accepted by Schultz and Melzack in 1991 [6]. Pharmacological agents such as anticonvulsants and antipsychotics have been tried in CBS. These were carbamazepine, valproate, gabapentin, melperone, ondansetron, mirtazapine, donepezil, cisapride and risperidone [4]. Ffytche, et al. performed functional magnetic resonance imaging study in patients who had visual hallucinations and they showed that there was an increment in the cerebral activity in the ventral extrastriate visual cortex. They observed that the neural activity increased particularly in these areas when they administered carbamazepine to these patients who had visual hallucinations [7]. In our case, there was no positive response when our patient primarily received carbamazepine but then, his complaints entirely disappeared in the end of one month treatment with the low dose quetiapine. CBS is not a clinically clarified syndrome. Its high prevalence was shown as %11 in one study and it was emphasized that this syndrome can be misjudged as a mental disorder [8]. Our patient was also admitted to the hospital since he worried about his situation.

Conclusion

Elder patients who are under the risk should be questioned in terms of these kinds of complaints and they should be treated appropriately. There should be further large scale studies in order to clarify the pathophysiology and the treatment of this syndrome.

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