Available online at www.sciencedirect.com
SCIENCE DIRECT®
European Psychiatry 21 (2006) 343–344
EUROPEAN PSYCHIATRY
http://france.elsevier.com/direct/EURPSY/

Letter to the Editor

Antipsychotics-induced hypersensitivity of visual perception

Hiroyuki Uchida, M.D. *, Takefumi Suzuki, M.D., Koichiro Watanabe, Ph.D., Haruo Kashima, Ph.D.

Department of Neuro-Psychiatry, School of Medicine, Keio University, 35 Shinanomachi Shinjuku-ku, Tokyo 160-8582, Japan

Received 8 October 2004; accepted 25 January 2005

Available online 13 April 2005

Keywords: Antipsychotic; Paroxysmal perceptual alteration; Oculogyric crisis; Dystonia; Side-effect

1. Introduction

Antipsychotic agents have been reported to sometimes induce recurrent brief episodes of hypersensitivity of visual perception, known as paroxysmal perceptual alteration (PPA) [3–5]. Yamaguchi and Nakai [6] reported PPA for the first time and regarded it as a schizophrenic symptom because the patients who experienced PPA had been diagnosed with schizophrenia.

We recently reported a mean overall prevalence of PPA among 338 patients treated with antipsychotic agents of 3.25%, and a higher rate among patients treated with high-potency antipsychotic agents (3.91%) than in those treated with medium- or low-potency agents (1.16%) [5]. We also found that PPA and oculogyric crises (OGC) occurred simultaneously in 36.4% of our subjects.

Here we report the case of a non-schizophrenic patient who experienced PPA that was worsened by taking more of the antipsychotic agent and ameliorated by reducing the dose of the antipsychotic agent during the first occasion, and ameliorated by taking an antiparkinsonian agent in the second series of episodes.

2. Case report

The patient was a single 24-year-old woman with a 6-year history of generalized anxiety disorder (GAD) and complained of anxiety and severe insomnia had been treated at our clinic for 2 years. A neurological examination that included computed tomography and an electroencephalogram showed no abnormal findings, and she had good vision and no history of use of illicit drugs. None of various antidepressants and anxiolytics ameliorated her symptoms, however, propericyazine 45 mg/day diminished the symptoms. Two months after introducing propericyazine, she became terrified by recurrent unpleasant episodes, and OGC had started and ended simultaneously with each episode. She described the episodes as follows:

"Tiny objects seem to be prominent and approach me. The light became brighter during each episode, and the fine pattern of the wall appeared more vivid than usual. The symptoms start suddenly in the evening, last about 30 min, and occur once a week. They terrify me. My eyes roll upward at the same time."

The symptoms were temporarily relieved by injecting biperiden 2 mg i.m., and reducing the dose of propericyazine to 5 mg/day was followed by resolution of both the perceptual alteration and OGC. Although the anxiety disorder remitted for a year, she relapsed. We then increased the dose of propericyazine to 45 mg and added biperiden 3 mg to prevent OGC, which occurred previously. Her anxiety symptoms improved, but 2 weeks later the same bizarre symptoms recurred.

"I am having the same symptoms. Light seems much brighter than usual, and tiny objects seem to be emphasized for about 30 min in the evening once a week. Colors seem more vivid, and the contrast between light and shadow seems more prominent. But my eyes have not been rolling upward this time."

She took an extra tablet of propericyazine (5 mg) to try to relieve the symptoms, but they deteriorated. We diagnosed the symptoms as PPA and increased the dose of biperiden to 5 mg/day, and PPA disappeared successfully in 2 weeks. The

* Corresponding author. Tel.: +81 3 5363 3829; fax: +81 3 5379 0187.
E-mail address: mcn41320@biglobe.ne.jp (H. Uchida).

0924-9338/$ - see front matter © 2005 Elsevier SAS. All rights reserved.
doi:10.1016/j.eurpsy.2005.01.007

https://doi.org/10.1016/j.eurpsy.2005.01.007 Published online by Cambridge University Press

patient's anxiety disorder has been well controlled on the same regimen ever since.

3. Discussion

To our knowledge this is the first report of an antipsychotic agent worsening already existing PPA and an antiparkinsonian agent diminishing PPA in a non-schizophrenic patient. The PPA was diminished by reducing the dose of propericyazine and taking biperiden, and it was worsened by taking additional propericyazine. OGC occurred and disappeared simultaneously with the PPA during the first series of episodes. The fact that reducing the dose of antipsychotic agents or injecting biperiden ameliorated both the PPA and OGC suggests a relationship between them. The findings in this case indicate that PPA represents an adverse effect of antipsychotic agents.

PPA is often regarded as a sign of exacerbation of the primary illness, especially schizophrenia. However, our patient was non–schizophrenic and her primary illness, GAD, was well controlled when she experienced PPA. Therefore, the recurrent episodes of perceptual alteration are thought to be unrelated to GAD.

What is the mechanism of PPA? We can speculate about the mechanism based on the similarities between PPA and OGC. Although no definitive mechanism for the dystonias, including OGC, has been established, the basal ganglia are thought to play an important role in their genesis [2]. The basal ganglia have a wide variety of functions and are known to affect not only motor function but emotions, such as anxiety and arousal, by themselves and their network [1]. The change in the arousal level caused by dopaminergic imbalance may induce the visual hypersensitivity and dysphoric sensations, and thus the dopaminergic imbalance induced by antipsychotic agents in the basal ganglia may lead to PPA as a sensory manifestation and/or OGC as a motor counterpart.

Because PPA is frequently misdiagnosed as a sign of exacerbation of the primary illness, patients and even physicians tend to add antipsychotic agents to the current regimen, which often worsens PPA, as it did in our case. Although the ideal treatment strategy to PPA is dose reduction of antipsychotics, there exist patients whose symptoms require antipsychotics. Therefore, an alternative way for those patients might be to titrate the dose and to add minimum antiparkinsonian agents on the ongoing regimen.

References

[1] Afifi AK. The basal ganglia: a neural network with more than motor function. Semin Pediatr Neurol 2003;10:3–10.
[2] Borison RL, Diamond BI. Neuropharmacology of the extrapyramidal system. J Clin Psychiatry 1987;48:7–12.
[3] Higuchi H, Shimizu T, Hishikawa Y. Recurrent paroxysmal episodes characterized by perceptual alteration in three schizophrenic patients on neuroleptic medication. Psychiatr Clin Neurosci 1997;51:99–101.
[4] Uchida H, Suzuki T, Watanabe K, Yagi G, Kashima H. Antipsychotic-induced paroxysmal perceptual alteration. Am J Psychiatry 2003;160: 2243–4.
[5] Uchida H, Suzuki T, Tanaka KF, Watanabe K, Yagi G, Kashima H. Recurrent episodes of perceptual alteration in patients treated with antipsychotic agents. J Clin Psychopharmacol 2003;23:496–9.
[6] Yamaguchi N, Nakai H. In: Attacks of altered perception in schizophrenics. In: Uchiyama Y, (Ed.), Psychopathology of Schizophrenia. Tokyo: Tokyo University Press; 1985. p. 295–314 [in Japanese].

https://doi.org/10.1016/j.eurpsy.2005.01.007 Published online by Cambridge University Press