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Paroxysmal Perceptual Alteration: Drug-Induced Phenomenon or Schizophrenic Psychopathology?

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ABSTRACT *~ Brief and repetitive episodes of perceptual changes, termed paroxysmal perceptual alteration (PPA), have been described in association with antipsychotic treatment. We report a case of paranoid schizophrenia who had such perceptual changes akin to PPA for 15 years, that was not related to antipsychotic treatment. There was a rapid resolution of PPA following treatment with low dose clonazepam.* Psychopharmacology Bulletin. *2012;45(1):44–47.*

INTRODUCTION

Brief and repetitive episodes of perceptual changes, termed paroxysmal perceptual alteration (PPA), have been described in association with antipsychotic treatment in patients with schizophrenia[1–3] and other non-psychotic disorders such as generalized anxiety disorder,[4,5] major depression[6] and bipolar disorder.[7] Uchida et al.[1] in 338 antipsychotic-treated schizophrenia patients, found the prevalence of PPA to be 3.25%. PPA has been reported to occur more frequently with oculogyric crisis (OGC) (36.4%).[1] We report a case of paranoid schizophrenia who had such perceptual changes akin to PPA for 15 years, that was not related to antipsychotic treatment.

CASE STUDY

Mr. A, 48-years-old unmarried male, diagnosed with paranoid schizophrenia for 31 years, presented with exacerbation of psychotic symptoms. He was treated with both typical and atypical antipsychotics in the past with poor response.

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Considering treatment resistance, he was started on clozapine and there was significant decrease in positive symptoms. He has been on maintenance dose of clozapine 600 mg per day for past seven years. There were two exacerbations of psychosis and he had received a course of electroconvulsive therapy and pimozide augmentation with reduction in symptoms. There was no history suggestive of any organic disorder or substance abuse. There was family history of bipolar disorder and schizoaffective disorder in first degree relatives. Mental status examination showed somatic passivity in which he had experiences of energy being pushed inside his body by some external force. He would feel distressed by these and get agitated.

For the past 15 years, he had been experiencing episodes during which objects appeared distorted. These episodes occurred frequently (occurred almost daily) and would last for two to three hours. During these episodes, he perceived an increase in the brightness as well as contrast of the colored objects and surface of the walls appeared irregular and rough. The patterns on the wall or table appeared more prominent and elevated from the surface, the circular patterns on wall and objects appeared as hollow tubes. Also, the rice grains appeared reddish in color, and the shapes of leaves appear distorted. While looking at other people during these episodes, their arms and forearms appeared folded several times and eye brows and hair appeared darker with furrows within them with enhanced contrast making it frightening. These changes were not under his control and were not related temporally to psychotic symptoms. He had full insight into these experiences and did not attribute to any external agency. These symptoms were intrusive and evoked significant anxiety and distress. Furthermore, these phenomena persisted all these years in spite of changes in antipsychotics several times. Considering a possibility of PPA, he was started on clonazepam 0.5 mg twice daily, with which there was significant decrease in frequency and associated distress. After two days of medications he did not experience such episodes though somatic passivity persisted.

DISCUSSION

The phenomenology of perceptual alteration in our patient is similar to that of PPA described in literature;[1–5] there was hypersensitivity of perception in the visual modality, it was ego dystonic and lasted for two to three hours. Our patient also had the "wall sign",[2] which is commonly observed in PPA, in which patterns and spots on the wall are perceived vividly with sharp contrast. The perceptual alterations involve several sensory distortions including size, shape, color, depth, brightness and contrast

of the objects. The distortions of limbs and face in our case are suggestive of "somatic schema disorder" described previously.[8] The distress associated with these symptoms is marked and these phenomena have some "obsessoid quality" in them. Based on the observation almost all reported cases of PPA have schizophrenia, it has been suggested as a schizophrenic symptom.[9] It is interesting to note that these perceptual phenomena, although akin to PPA, was unrelated to changes in the antipsychotic medication (both typical and atypical) and has been present continuously for 15 years. Also, these perceptual phenomena were not associated with somatic passivity experiences and appeared separate from them. This raises the possibility that in some patients with schizophrenia, PPA may be an independent psychopathology in patients with schizophrenia.

PPA reportedly increases with escalating dose of antipsychotics and improves with anticholinergics and benzodiazepines.[1,5,10] In our patient, PPA of such prolonged duration decreased rapidly within few days following administration of clonazepam. For antipsychotic-induced PPA, discontinuation or dose reduction of antipsychotics should be attempted if feasible, which may not benefit if PPA is part of schizophrenic psychopathology as seen in our case. Changing to atypical antipsychotics may help some patients,[11] though PPA has also been observed with atypical agents such as risperidone and clozapine.[3,8]

The mechanism underlying this phenomenon is poorly understood. Based on their association with OGC, the basal ganglia structures have been presumed to be involved in their genesis.[9] Also, stereotypic paroxysmal psychiatric symptoms have been described in patients with schizophrenia, which bear a striking resemblance to PPA.[12] It is possible that the frontal-subcortical circuits[13] may underlie the pathophysiology of both the symptom groups. Imbalance of several neurotransmitter systems including dopamine, noradrenaline, and serotonin have been implicated in the pathophysiology of PPA.[2,8] Furthermore, improvement with benzodiazepines treatment points towards possible involvement of GABA in the pathophysiology of PPA. ✤

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