



Improvement of persistent cenesthopathy and depersonalization after treatment with brexpiprazole in schizophrenia

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Received May 18, 2020 / Accepted May 18, 2020 / Published June 12, 2020.

Letter to the Editor

Somatic hallucination is defined as the false sensation of an occurrence in the body. When the hallucination is grotesque and visceral, it is classified as a cenesthopathy [1]. Cenesthopathy presents comorbidly to various other psychiatric diseases including schizophrenia, major depression, and somatoform disorders [2]. It is interesting to note that cenesthopathy is likely influenced by culture, with one study reporting that, of the seven unique countries from which patients were recruited, those from Ghana and those with chronic schizophrenia were significantly more likely to report cenesthesia [3]. Herein, we present a case of persistent cenesthopathy associated with depersonalization, which improved drastically when treated with brexpiprazole. Written informed consent was obtained from the patient for publication of this case report.

Case report: The patient was a 41-year-old Japanese male diagnosed with schizophrenia, based on DSM-5 criteria 15 years ago. He demonstrated persistent active commentary auditory hallucinations, persecutory delusions, disorganized behavior, cenesthopathy, depersonalization, and agitation. His development and past history of physical illness were unremarkable. The initial presentation of his psychosis consisted of commentary auditory hallucinations, delusions of persecution, psychomotor excitement, disorganized behavior, cenesthopathy associated with depersonalization, and restlessness. The patient's cenesthopathy was consist of perceiving or

feeling his skull was melting, and the cenesthopathy was often accompanied with depersonalization. He was first administered risperidone at 4mg/day. Auditory hallucinations, persecutory delusions, psychomotor excitement, disorganized behavior, and agitation gradually ameliorated with treatment. However, his cenesthopathy associated with depersonalization persisted. Treatment with risperidone was changed to olanzapine at 20mg/day, and he complained of hyperphagia without improvement of his cenesthopathy or depersonalization. Olanzapine was tapered and ceased and he was started on blonanserin, which was gradually increased to 16mg/day. His cenesthopathy and depersonalization did not respond to blonanserin. Thus, brexpiprazole was prescribed after blonanserin was tapered. On the day 14 after starting brexpiprazole and increased to 2mg/day, he reported his cenesthopathy was slightly relieved, stating that the melting feeling in his scalp was weaker than before. On day 28 of treatment, his cenesthopathy was completely resolved. The depersonalization also improved. He reported he could feel the world more clearly than before. He has been well without relapse at 2 mg/day of brexpiprazole.

To our knowledge, this is the first report of persistent cenesthopathy associated with depersonalization responding to brexpiprazole in a patient with chronic schizophrenia. It has been reported that cenesthopathy may also occur in patients with Parkinson's disease during dopamine agonist and anti-parkinsonian drug treatment [4]. A case report

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exists in which quetiapine improved strange tactile hallucinations, similar to cenesthopathy, in a patient with Parkinson's disease [5]. The mechanism by which brexiprazole, but not risperidone, olanzapine, or blonanserin improve cenesthopathy and depersonalization in chronic schizophrenia remains unknown. The differences between pharmacological profiles among brexiprazole, which possesses partial agonistic effects on D_2 and $5-HT_{1A}$, and also potent antagonistic activity on $5-HT_{2A}$, and the other antipsychotics may contribute its efficacy. Further research in similar clinical situations should be conducted to examine the effectiveness of brexiprazole for the management of cenesthopathy.

CONFLICT OF INTEREST

none

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