Neuropsychiatric Background of Alcohol Hallucinosis: A SPECT Study

SIR: Alcohol hallucinosis is a psychiatric condition characterized by auditory hallucinations, without consciousness disturbance, following drinking or cessation of alcohol. Although it is an important clinical issue in the management of alcohol dependence, the neuropsychiatric background of alcohol hallucinosis remains unknown. We report a case of alcohol hallucinosis in which we examined the neuropsychiatric background of the condition using *N*-isopropyl-p-^{123}I iodoamphetamine (^{123}I-IMP) single photon emission computed tomography (SPECT).

Case Report

The patient was a 56-year-old Japanese junior high school graduate. He had a history of alcohol abuse of more than 30 years, with an average alcohol consumption of 200g/day (about 1.31 Japanese sake/day). He had experienced subacute auditory hallucinations and persecutional delusions for a month, during which time he intruded into his neighbor's house and tried to set fire to the neighbor's car. Finally, he was arrested by the police and admitted to our hospital.

On admission, he was alert, and physical and neurological examinations revealed no abnormalities except for a mild finger tremor. Laboratory tests demonstrated only mild elevation of liver enzymes and the result of a magnetic resonance imaging (MRI) scan was intact. However, ^{123}I-IMP SPECT revealed a decreased regional cerebral blood flow (rCBF) in the frontal lobe, left basal ganglia, and left thalamus. A regimen of diazepam, 40mg/day, and haloperidol, 6mg/day, was initiated, and the hallucinations and delusions almost disappeared within a month. At this point, diazepam was tapered off and haloperidol was tapered to 3mg/day. After the disappearance of the hallucinations, the rCBF in the basal ganglia and thalamus normalized, but the rCBF decrease in the frontal lobe remained.

In this case, rCBF decrease was observed in the frontal lobe, left basal ganglia, and left thalamus during the period of hallucinations, but the rCBF then normalized after recovery from the hallucinations, except in the frontal lobe.

Comment

Previous studies have demonstrated a toxic effect of alcohol on the frontal lobe, and hypofrontality is one of the characteristic findings of functional neuroimaging techniques in alcohol dependence.[1] On the other hand, functional abnormality of the frontal-subcortical circuit is well established in schizophrenia, the symptoms of which resemble alcohol hallucinosis. Sabri et al.[2] demonstrated a strong correlation between hallucinations and rCBF decrease in the cingulate, left thalamus, left frontal lobe, and left temporal lobe, and Soyka et al.[3,4] showed frontal and thalamic dysfunction in a case of alcohol hallucinosis but did not report the functional changes following treatment, which we assessed in this study.

Collectively, the above findings suggest that the hypofrontality observed in this case probably reflects the effect of long-term alcohol dependence. Dysfunction of the left basal ganglia and left thalamus or coexistence of these abnormalities with frontal dysfunction could contribute to the development of alcohol hallucinosis, suggesting that alcohol hallucinosis may partially have a mutual neuropsychiatric background to that of schizophrenia.

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Use of Clozapine in Oneroid State

SIR: Oneroid state is an uncommon psychiatric disorder, which has been given little attention in recent classificatory systems. Older textbooks of psychiatry have described it as schizophrenia with a clouding of consciousness and as occurring mostly in the acute stage of schizophrenia.[1] It is characterized by a kaleidoscopic quality of psychopathological experiences wherein reality, illusions, and hallucinations are merged into one.[2] In such a state, a patient may be deeply

perplexed and not fully oriented to time and place. I wish to report an adolescent girl who exhibited oneroid state with no basis for organicity, and who responded well to clozapine after failure of trials with haloperidol and risperidone.

Case Report

"Ms. A" is a 19-year-old unmarried woman from a socioeconomically lower class urban family. In the past, she developed an illness suggestive of paranoid schizophrenia (ICD 10 Criteria), characterized by withdrawn, abusive and violent behavior, delusions of persecution, and inappropriate affect of 5-month duration. She recovered spontaneously and was symptom-free for the next 2 months. Subsequently, she presented to us with confusional behavior without any apparent psychological or physical precipitating factor. Her confusional behavior lasted for 15 days and was almost continuous. During this time, she misidentified her family members and had urinary incontinence. She muttered and talked incoherently. Mental status examination revealed increased psychomotor activity, verbal stereotypy, and perseveration with loosening of association and perplexed mood. She exhibited double orientation and double book-keeping phenomena.[3,4] At times, she would misidentify the doctor as her brother, but on other occasions, she behaved like a patient with the same doctor. She would misidentify the hospital as her home and name a few patients as her relatives. She labeled half of the hospital as her house and half as the hospital. While in the hospital, she would follow regular hospital routine. Her look was perplexed. She had fleeting delusions of persecution, rapidly shifting visual hallucinations, and illusionary distortion of perceptual processes on several occasions.

A detailed, meaningful cognitive assessment was not possible in view of her persistent perplexity. Medical and neurological referrals were taken and no deficit was detected upon a detailed physical examination. Her computed tomography (CT) head scan and electroencephalogram (EEG) were normal. A routine hematological and biochemical investigation did not reveal any abnormal findings. She was put on a regimen of haloperidol, 20mg/day for 3 weeks, and, subsequently, risperidone, 6mg/day, with no improvement. Ultimately, I started her on a regimen of clozapine, and gradually increased the dosage to 200mg over a period of 3 weeks, with proper monitoring of leukocyte count. Ms. A showed marked improvement with clozapine not only in thinking but in her confusional behavior. Hence, she was stabilized on the same dose for 3 months without any relapse.

Comment

Oneroid state is a neglected condition probably because it deals more with clinical descriptions than with a phenomenological approach. This case did not fit into any category of the current psychiatric classificatory system. In contrast to our report where clozapine has been used successfully, there have been few reports of clozapine-related delirium or confusion.[3] In such cases, clozapine's anticholinergic properties or concomitant anticholinergic or CNS depressant medications may have been responsible for the delirium.

In conclusion, clozapine can be used safely in patients with functional confusional psychoses, such as oneroid state; hence, it may be considered as the first line of treatment. Further systematic collection of data in this regard may add to the confidence in prescribing clozapine in this state.

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Clozapine-Induced Tardive Dystonia (Blepharospasm)

SIR: Tardive dystonia is a type of "tardive" movement disorder induced by antipsychotics and is characterized by involuntary muscle contraction, which may be tonic, spasmodic, patterned, or repetitive.[1] Blepharospasm is a type of focal tardive dystonia characterized by chronic intermittent or persistent closure of the eyelids. Although there are rare reports of tardive dystonia induced by atypical antipsychotics, such as olanzapine,[2] clozapine, in contrast, has been reported to ameliorate tardive dystonia.[3] Contrary to existing literature, we report a case of a patient developing blepharospasm with clozapine.

Case Report

"Mrs. F," a 46-year-old woman, presented with one month of suspiciousness, mumbling to herself,