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# Case Study of Monosymptomatic Delusion of Unpleasant Body Odor with Structural Frontal Abnormality

### **Key Words**

Delusion Paranoid disorder Monosymptomatic hypochondriacal psychosis Organic delusional syndrome

#### Abstract

We report a 63-year-old man with a monosymptomatic delusion of unpleasant body odor contracted at the age of 61 years, who believed that his body reeked of some type of paint. He had symmetrical atrophy in the bilateral frontal lobes of unknown etiology on MRI. Further, quantitative rCBF measurement with SPECT brain scan using <sup>99m</sup>Tc-HMPAO showed relative hypoperfusion localized in the bilateral frontal lobes. EEG, neurological and neuropsychological tests were all normal, including the WAIS-R and Wechsler Memory Scale tests. However, he showed poor results on the Wisconsin Card Sorting test, presumably on account of frontal dysfunction, attaining only two categories with frequent perseverative errors. Although it is undetermined whether the illness in this case can be classified as organic delusional syndrome or paranoid disorder according to DSM-III-R criteria, these results imply that delusions are associated with frontal lobe abnormality.

The pathogenesis of delusion, which is a representative psychotic symptom, is an important and still unresolved problem in the field of neuropsychiatry. Recently, Benson and Stuss [1] postulated an association between delusion and prefrontal malfunction based on descriptions of a number of organic delusion syndromes associated with frontal damage on computed tomographic (CT) scans, e.g., a case with Capgras syndrome. The present report provides supportive data for this hypothesis from neuroimaging and neuropsychological examinations in a specific case with monosymptomatic delusion of unpleasant body odor contracted in late life.

## **Case Report**

The patient was a 63-year-old right-handed man with an introverted and earnest personality. After completing an 8-year schooling he served for many years as an artisan in a factory making household Buddhist altars. He brought up 2 children, a son and a daughter, single-handedly after he divorced at the age of 41 years. He had no occupation after retiring from the factory at the age of 60 years. Since the marriage of his daugther 10 years previously he had been living with his 37-year-old unmarried son. There was no evidence of any significant medical illness including psychosis in either his own or his family history. Further, he had no history of drug addiction, and did not consume either alcohol or cigarettes.

Itsuki Jibiki Department of Neuropsychiatry Kanazawa University School of Medicine 13-1 Takaramachi Kanazawa, Ishikawa Prefecture, 920 (Japan) © 1994 S. Karger AG, Basel 0302-282X/94/0301-0007 \$5.00/0



Fig. 1. MRI findings. a-c  $T_1$  intensity-weighted axial images indicating frontal atrophy. d Intermediate intensityweighted axial image showing periventricular high intensity lesion. e  $T_2$  intensity-weighted axial image with multiple small lacunes (arrow marks). R, right; L, left.

One day in October, 1991, at the age of 61 years, he perceived an intense smell of a type of paint he applied on a pillar in his house to suppress white ants. Subsequently, he developed a delusional belief of so-called 'unpleasant body odor' in which his body recked of this paint which he could not get rid of. Further, at the same time he developed a hypochondriacal delusion in which his tongue and the mucous membrane in his nose and mouth were affected, because of the smell, with a feeling of physical disorder. As he felt the smell most strongly when he was in his home, he often left his home. On December 15, 1992, he visited our clinic with his son.

In the first psychiatric examination, his facial expression, gestures and behavior appeared natural. His understanding, contact and attitude during the interview were also normal. His general mental state showed neither intellectual nor memory deficits with both his orientation and consciousness level normal. He revealed no symptoms except for the above-mentioned delusions and feeling of physical disorder. Personality change, too, was unlikely to be present in view of the history outlined by the son. Routine physical and neurological tests showed no abnormalities nor did routine neuropsychological tests reveal any signs of aphasia, apraxia or agnosia. For the diagnosis

Jibiki/Kagara/Kishizawa/Kurokawa/ Fukushima/Furuta/Yamaguchi of his illness, a number of further examinations were carried out in his neuroleptic-naive condition in our outpatient clinic over the subsequent 2 weeks, as described below.

As shown in figure 1, MRI revealed symmetrical atrophy localized in the bilateral frontal lobes with obvious dilatation of the anterior portion of the longitudinal cerebral fissure (fig. 1a-c). Further, slight periventricular cerebral arteriosclerotic change (fig. 1d) and multiple small lacunes (fig. 1e) were detected in the deep white matter on MRI. Quantitative rCBF measurement with SPECT brain scan using 99mTc-HMPAO showed relative hypoperfusion localized in the bilateral frontal lobes, with 46 and 47 ml/100 g/min in the right and left frontal lobes, respectively, whereas the absolute values of rCBF ranged from 50 to 59 ml/100 g/min in the other regions in the cerebral cortex. Routine scalp EEG showed normal findings, consisting of occipital-dominant background activities of 9-10 Hz mixed with frontal-dominant fast activities of 20-25 Hz with no slow waves or paroxysmal discharges. In recordings of event-related potentials, P300 revealed relatively low amplitudes, with heights of 3.07 and 4.82  $\mu$ V in Cz and Pz recordings, respectively, these latencies both showing the normal value of 358 ms. Further, contingent negative variations (CNVs) showed relatively low amplitudes with the heights of the late CNV component in Fz and Cz recordings indicating 3.80 and 4.64 µV, respectively. In neuropsychologial tests, the WAIS-R demonstrated average intelligence, with scores of 98, 87 and 93 for verbal, performance and full IQ scores, respectively, although scores on comprehension and combination tasks were relatively low. Further, the Wechsler Memory Scale test demonstrated almost normal memory function, which was however somewhat low as compared with his intelligence level, with an MQ score of 90. In contrast, he showed poor results on the Wisconsin Card Sorting test (WCST), which is known as a test of basic abstract thinking and the ability to shift simple mental sets [2], attaining only two categories (color and figure) with frequent perseverative errors (13 in color and 57 in figure). In addition, laboratory examinations, including hematology, liver function tests, electrolytes, urinalysis, ECG and blood pressure, showed no abnormalities. Further, he showed no abnormalities in otolaryngological examinations, including routine olfactory tests.

After these examinations, he was treated as an outpatient with a representative antipsychotic, haloperidol 2.25 mg/day, as well as biperiden 3 mg/day to prevent the haloperidol-induced extrapyramidal symptoms. The haloperidol exerted a slight but beneficial effect on his delusions and the feeling of physical disorder in his tongue, mouth and nose, because he appeared to be less nervous about them after than before the administration. However, these symptoms persisted even with a subsequent increase in the haloperidol dose. Pimozide 3–6 mg/day was added but led to only slight improvement of these symptoms.

#### Discussion

It is clear from the MRI findings that the present case had an atrophied structural or organic abnormality in the bilateral frontal lobes. The SPECT findings support this. Further, his poor results on the WCST imply a frontal abnormality, because it is known that this test is closely associated with dorsolateral prefrontal function [2]. His illness characterized by the delusion of unpleasant body odor and related symptoms contracted late in life would have been diagnosed as 'monosymptomatic hypochondriacal psychosis' as proposed by Munro [3] or paranoid disorder on DSM-III-R criteria, had it not been for the MRI findings. However, the present frontal abnormality suggests that his illness should be classified as organic delusional syndrome by DSM-III-R criteria, which means that the delusion resulted from the organic frontal abnormality. Benson and Stuss [1] stated that the delusion of unpleasant body odor is not uncommon in organic delusions, although they illustrated no cases with this delusion. Further, Miller and Lesser [4] described that neuroimaging was useful for the diagnosis or pathogenetic understanding in late-life psychosis. However, in the present case, the origin of the atrophied frontal lobes is unknown, since his illness is not symptomatically compatible with any of the established organic cerebral diseases, including degenerative diseases such as Alzheimer's disease, Pick's disease and dementia of frontal lobe type reported by Gustafson [5] and Neary et al. [6]. It is most probable that the present frontal atrophy resulted from relatively severe aging changes consisting of cerebral degenerative and arteriosclerotic changes. Further, in the present case, it is unknown when the frontal abnormality developed, in particular with regard to the onset of the delusion. Moreover, there is no direct evidence for demonstrating a causal relation between the delusion and frontal abnormality, although the disability in mental set shifting on the WCST may be related to the disturbance of reality testing underlying the delusion [1]. Therefore, a definite diagnosis of organic delusional syndrome in the present case may be difficult.

It has been reported that a number of schizophrenic patients have organic cerebral anomalies on CT or MRI, e.g., small or atrophied frontal and temporal lobes, and dilated lateral ventricle [7–9]. Although these findings imply that organic anomalies may partly contribute to the origin of schizophrenia, they do not mean that schizophrenia is an organic mental disorder beause many schizophrenics have no organic anomalies on CT or MRI. One may argue that the existence of an organic frontal abnormality in the present case is compatible with that of such organic anomalies in schizophrenics, not necessarily leading to the diagnosis of organic delusional syndrome but suggesting that there may be a number of paranoid disorders having organic cerebral anomalies. Further, it has been opined that the organic anomalies on MRI in schizophrenics may be due to cerebral developmental disorders [8]. But, in the present case, the frontal abnormali-

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(arolinska Institutet, University Library 130.237.122.245 - 1/12/2019 1:43:31. ty is more likely to be due to atrophy rather than a developmental disorder in view of the patient's advanced age and MRI findings.

In conclusion, it is undetermined whether the illness in the present case should be classified as organic delusional syndrome or paranoid disorder on DSM-III-R. However, in either case the present clear coexistence of the monosymptomatic delusion and organic frontal abnormality implies that delusions are associated with frontal lobe abnormality.

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