Obsessive compulsive disorder in a woman with left basal ganglia infarct: A case report

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This report presents a syndrome resembling obsessive compulsive disorder (OCD) secondary to a stroke in the left basal ganglia. The patient's syndrome is virtually identical to those that have been described in bilateral damage of the basal ganglia. However, the stroke described in this case report is located unilaterally in the left basal ganglia. In addition, experience in treating a patient with OCD induced by structural damage of basal ganglia is presented.

Keywords: Obsessive compulsive disorder – Stroke

INTRODUCTION

In recent years numerous studies have suggested that functional abnormalities of the basal ganglia are involved in the pathogenesis of OCD (for review, see Insel, 1992). In addition, there are a number of cases in which basal ganglia structural damage has been found in patients with obsessive compulsive (OC) symptoms (Croisile *et al.*, 1989; Laplane *et al.*, 1989; Maraganore *et al.*, 1991; Weilburg *et al.*, 1989), accompanied at times by a frontal lobe syndrome (Croisile *et al.*, 1989; Laplane *et al.*, 1989).

This report presents a syndrome resembling OCD secondary to a stroke. The patient's syndrome is virtually identical to those that have been described in bilateral damage of the basal ganglia (Laplane *et al.*, 1989). However, the stroke described in this case report is located unilaterally in the left basal ganglia. In addition, experience in treating a patient with OCD induced by structural damage of basal ganglia is presented.

CASE REPORT

Mrs J is a 73-year-old right handed white female who, at the time of presentation to a geriatric psychiatry ward, displayed a marked loss of drive, apathy, emotional blunting and indifference to social interaction without a dysphoric mood. Neurological examination was remarkable only for the presence of frontal release signs as well as difficulties with alternating motor sequences (fist-blade-palm). In addition, she had com-

pulsions to clean her mouth with tissue paper or other items, depending upon availability, and compulsions to drink sips of water continuously throughout the day. With questioning, she revealed that these behaviours were done to clean her mouth of "phlegm" which, although not readily seen, she experienced as distressing and contaminating and needing to be neutralised. If the tissue papers were removed from her side, she would get anxious and try to obtain other materials with which she could continue cleaning her mouth. Her obsessions and compulsions were not ego-dystonic as she lacked the insight that her obsession was bizarre and that her behaviour was ritualistic and abnormal. She did not try to resist these behaviours which occupied most of her time. Due to her apathy, she did not engage in a variety of activities, including the routine activities of daily living.

The past psychiatric history revealed a remote history of two post-partum depressive episodes, at least one of which was successfully treated with ECT. The patient did well until two years prior to admission, at which point she had a stroke which is not clearly defined symptomatically by the patient or her family other than as "equilibrium problems", mainly seen as an unsteady gait. A CT scan performed at that time showed a lacunar infarct in the left basal ganglia. Six months later, the patient began exhibiting the compulsive rituals described above and she became depressed. The depression failed to respond to treatment with nortriptyline and paroxetine. Two months prior to the

current hospitalisation, the patient received 10 ECT treatments with resolution of the depression but she continued to manifest OC symptoms. After ECT, the patient was discharged on fluoxetine. At the time of readmission, the patient had been on fluoxetine 40 mg for two months with no relapse of depression but without any amelioration of obsessions or compulsions.

MRI scan performed seven months after the stroke showed a lacunar infarct located in the lateral portion of the left putamen, extending to the external capsula. Additional findings in the MRI scan included generalised cortical atrophy and periventricular deep white matter ischemic disease not greater than expected for her age. A repeat MRI scan 17 months after the stroke did not show any significant change compared with the previous MRI. Conventional EEG studies depicted normal electrocerebral activities in awake and drowsy states. However, a functional quantitative EEG with brain mapping showed abnormal excess energy in slow wave bands in the left fronto-temporal areas, consistent with frontal dysfunction.

Neuropsychological testing revealed intact attention, word naming, basic language abilities and memory with impairment of fine motor coordination, figural and verbal fluency and executive functions. Other findings were behavioural disinhibition, poor initiation and difficulty sustaining effort.

Treatment goals during the hospital stay were to diminish the frequency of rituals, decrease apathy, and increase social interaction. Behaviour therapy was attempted and consultation sought from a cognitive-behavioural psychologist; however, the patient was a poor candidate due to her lack of insight and motivation. As the patient had not shown any response to two months of treatment with fluoxetine, risperidone 1 mg every four hours was added without any effect after six days. Extrapyramidal symptoms did not appear after the neuroleptic was added. At this point, lithium carbonate 300 mg twice daily was added and a dramatic decrease in the compulsive behaviour was seen four days later. However, there was no improvement in the frontal symptoms. Therefore, amantadine 100 mg twice daily was added and an increase in initiative and decrease in apathy were noted. The patient was seen seven and 28 days post-hospitalisation by one of the team physicians and continued to maintain the improvement described above.

DISCUSSION

The fact that the patient did not have any psychiatric problems prior to the stroke indicates that the stroke may be responsible for the appearance of the OCD-

like symptoms. The location of the stroke in the left basal ganglia is in agreement with current theories that posit basal ganglia dysfunction in the pathogenesis of OCD (for review, see Insel, 1992) and with reports of OCD appearance after bilateral structural damage of basal ganglia (Croisile *et al.*, 1989; Laplane *et al.*, 1989).

Another coincidence between the case reported in the present paper and classical OCD is the comorbidity of OCD with frontal syndromes. This comorbidity has been demonstrated by clinical criteria and neuropsychological testing (Head *et al.*, 1989; Khanna, 1988; Laplane *et al.*, 1989; Martinot *et al.*, 1990) and confirmed by decreased power in the nondominant frontomedial and posterior temporal regions in computerised EEG (Gloor *et al.*, 1977) and dysfunction of cortical metabolism (Laplane *et al.*, 1989; Martinot *et al.*, 1990; Rubin *et al.*, 1992; Baxter *et al.*, 1987, 1988; Nordhal *et al.*, 1989; Swedo *et al.*, 1989). The most plausible explanation for the appearance of frontal syndrome and EEG slowing after a stroke in the basal ganglia is a functional abnormality of the cortex as a consequence of deafferentation from subcortical structures. This hypothesis is consistent with animal studies in which white matter lesions made beneath the cortical surface result in EEG slowing (Luria, 1966) and with findings in humans of reduced glucose metabolism in cortical regions ipsilateral to caudate and internal capsula lacunar lesions (Metter *et al.*, 1981). It is also possible that the absence of ego dystonic components could correlate with a decrease in frontal activity. In fact, Laplane *et al.* (1989) presented several cases of OCD with low levels of anxiety that resulted from bilateral basal ganglia necrosis, in which there was a decrease in frontal metabolic rate.

First line treatment for OCD includes cognitive behavioural (Dar and Greist, 1992) and/or pharmacological therapy (Jenike, 1992). The specific cognitive behavioural therapy for OCD is exposure and response prevention (Dar and Greist, 1992). The patient was deemed inappropriate for reasons that have been reported to be predictors of treatment failure (Jenike, 1990); these are poor compliance, cognitive impairment, inability to tolerate the demands of the treatment and fixed belief in the necessity of the ritual.

First line pharmacological treatment for OCD are the serotonergic antidepressants, fluoxetine, fluvoxamine, clomipramine, paroxetine and sertraline (Jenike, 1992). An eight week trial of fluoxetine at 40 mg a day following her course of ECT failed to ameliorate her OC symptoms. Guidelines in adults call for increasing fluoxetine doses (Jenike, 1992) in the face of refractory response. However, the patient presented in this paper

is a geriatric patient, and it was considered that she was already on a relatively high dose because of her age. Therefore, augmentation strategies were applied. The patient began to demonstrate decreased frequency of her OC symptoms after risperidone and lithium were added to her drug therapy. It is unclear which of these two augmenting medications was responsible for the improvement but it is unlikely that the improvement was a consequence of neuroleptic-induced bradykinesia because the patient did not show signs of extrapyramidal symptoms. Despite a decrease in her OC symptoms, the patient continued to manifest profound apathy. Amantadine, an indirect dopaminergic agonist, was added to her therapy because it has been reportedly successful for the treatment of apathy (Marin *et al.*, 1995).

In summary, this report presents, possibly for the first time, a case of OCD that resulted from an unilateral insult to the basal ganglia. In addition, experience in treating a patient with OCD symptoms secondary to known structural damage is presented. Although this patient failed to improve with monotherapy, a satisfactory treatment outcome was achieved with polypharmacy.

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(Received 21 March 1997; accepted as revised 25 May 1997)