Obsessive Compulsive Disorder Following Left Middle Cerebral Artery Infarct

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ABSTRACT

A case of a 37-year-old male who developed compulsive washing after a massive infarct involving the left middle cerebral artery, is described. Although an obsessive compulsive disorder can occur following neurological disorders, there have been no previous reports of it occurring following a middle cerebral artery infarct.

Keywords: Obsessive Compulsive Disorder, Middle Cerebral Artery Infarct.

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INTRODUCTION

Obsessive compulsive disorder (OCD) is a neuro-psychiatric illness[1]. Although pathophysiologically heterogenous, anatomical sites such as the orbital frontal lobe structures as well as the basal ganglia region have been implicated[2]. Generally however, OCD patients do not have gross cerebral lesions, although there have been reports of an obsessive compulsive disorder occurring following head injury or temporal lobe epilepsy[3]. Soft neurological signs are more common[4].

We report a case in whom an obsessive compulsive disorder developed following a left middle cerebral artery infarct. Our literature search revealed no previous reports of such cases. While left middle cerebral artery infarcts can cause severe neurologic deficit, such as hemiplegia and hemisensory loss in the face, arm and leg, hemianopia, global aphasia and reduced consciousness and more commonly than other types of infarct, reports of psychiatric complications are rare[5]. There have been reports of an agitated confusional state characterized by hyperactivity, restlessness and easy distractibility and depression[6,7].

CASE REPORT

Patient P, a 37-year-old Chinese male, had a cerebral infarct in January 1996 involving the left middle cerebral artery territory. This resulted in expressive aphasia and a dense right hemiplegia. He also developed epileptic seizures and the family reported that his temper worsened. The CT scan showed a large infarct involving the left frontal, temporal and parietal lobes. P was found to be hypertensive at that time. His cholesterol, urea and electrolyte levels were normal. Investigations for a hypercoagulable state were negative. His autoimmune markers were negative. He was started on aspirin and rehabilitation in the ward. On discharge in February 1996 he still needed some assistance with Activities of Daily Living.

Two years later he was admitted to the psychiatric hospital as he was bad-tempered and violent at home particularly when his demands were not met. He was found to be irritable and hostile and tended to be easily frustrated and agitated when he was unable to answer questions. He was not depressed, his sleep and appetite were good. There was little improvement in the expressive aphasia and although he kept to himself, he was willing to participate in ward activities and could watch television.

While in the psychiatric ward, the staff noted that he had compulsive symptoms and rituals during bath times and before he sat down anywhere. At bath time he would wash the shower head, taps and pipes in the bathroom before he bathed. This would follow a particular pattern which he repeated many times before actually starting to bathe. Bathing involved soaping and scrubbing in a repetitive manner and in a fixed sequence. Bathing time was prolonged and he would put up a struggle if staff tried to get him out of the bathroom. He also washed his hands repeatedly whenever he could and would only stop when he felt satisfied. His family confirmed he did not have this behaviour or other obsessive compulsive traits prior to the cerebral infarct. On the Yale-Brown Obsessive Compulsive Symptom Checklist, patient's principal symptoms were cleaning/washing compulsions, checking compulsions and repeating rituals. Obsessional symptoms were not elicited at that time. On the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), the score was 15[8]. The Clinical Global Impression Scale severity score was 3[9]. The symptoms were treated with Clomipramine up to a total of 150 mg a day. The symptoms resolved after four weeks. The Clinical Global Impression Scale severity

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score fell to l and the improvement score was 5. His other medication included Carbamazepine 800mg per day, Haloperidol l0 mg per day for control for agitation, Benzhexol 6 mg per day, Aspirin 100 mg in the morning Nifedipine 30 mg per day and Atenolol 50 mg in the morning.

A repeat CT scan in April 1998 showed the old infarct, evidence of atrophy with dilatation of the frontal part of the left lateral ventricle and widening of the Sylvian fissure. There was no significant enhancement after intravenous contrast and no evidence of any mass lesions.

Neuropsychological testing was done. Non-verbal tests were administered because of his expressive aphasia. Effort and concentration was good. Based on the performance subtests from the WAIS-R, his Performance IQ was estimated to be in the borderline range of intelligence (PIQ 76). His awareness of essential details, visuo-constructional and perceptual organisational skills were below average. His immediate recall for visual material was also below average and he was unable to reproduce anything after a time lapse of thirty minutes. His performance on the Trail Making Test was slow indicating he had difficulty responding to a visual array of some complexity. In general the neuropsychological testing showed a decline in intellectual functioning compare to his estimated premorbid functioning of about average to low average intelligence. He also showed deficits in his visual memory and in his mental flexibility.

DISCUSSION

This case is interesting as there have been no previous reports of an OCD following left middle cerebral artery infarct. This despite the fact that these infarcts are the most common of all cerebrovascular infarcts[10]. The entire territory was involved here indicating occlusion of the middle cerebral artery and the perforating branches. Given the regions supplied by the middle cerebral artery, such as the orbital surfaces and its anterolateral (thalamostriate) group of cerebral branches which penetrate the caudate and lentiform nucleus, the areas damaged are those associated with obsessive compulsive phenomena. Interestingly a PET study found that OCD patients as a group showed positive correlations between symptoms intensity and blood flow to the right inferior frontal gyrus, caudate nucleus, putamen, globus pallidus and thalamus[11].

An abnormal serotonergic mechanism is believed to contribute to the disorder. Interestingly animal studies have shown that norepinephrine - epinephrine content (NE-E) of ischemic cerebral tissues was low twenty-four hours and seven days after middle cerebral artery occlusions[12]. Given these factors then, it is surprising that there have been no previous reports of OCD with middle cerebral artery infarcts. While neurological disorders could predispose to OCD, developmental, psychodynamic and learnt behaviors still have an etiological role as well[13].

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