

Case report

Obsessive-compulsive disorder in the elderly

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Four cases of obsessive-compulsive disorder arising in late life in association with a presumed organic aetiology are described. Three of the four had brief episodes of OCD earlier in their lives. Neuropsychological assessment demonstrated impairments in verbal fluency and visuo-spatial tasks. No case exhibited global intellectual impairment. The two patients who complied with appropriate treatment became asymptomatic after 4–6 months.

Keywords: Obsessive-compulsive aged organic depression

1. Introduction

Obsessive-compulsive disorder (OCD) first occurring in later life is relatively uncommon although evidence from the Epidemiological Catchment Area study suggests a 6 month prevalence of 1% [20] and an annual incidence of approximately 0.6% [11] in the over 65s. Systematic case series of OCD in late life have not been published and individual case reports have focused on patients' response to various forms of treatment [2, 3, 6, 8]. Even so, the majority of these cases were of chronic OCD with onset occurring in early or middle age without remission or patients who presented with prominent depressive symptoms.

Recent interest has centred on the neurobiological and neuropsychological manifestations of OCD but again studies are almost exclusively limited to early onset OCD (e.g., [31, 32]). The present paper details the clinical, radiological and neuropsychological features of 4 cases of OCD occurring in late life in the absence of primary depressive or cognitive disorder.

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2. Methods

2.1. Case studies

Case 1 was a 74 year old man. At the age of 71 he experienced the insidious onset of recurrent intrusive blasphemous thoughts and began checking that all the windows and doors in his house were locked each night. His checking was so meticulous that it took up to 3 hours each time. These symptoms led to marital problems and a moderate depression. He was medically fit and had no relevant family history. In his mid fifties he had experienced a brief period of depression and obsessional thoughts *without* rituals when under stress at work but this had resolved spontaneously after a few months.

Brain CT revealed a moderate degree of cerebral atrophy and a much reduced left cerebral hemisphere and hemi-cranium which was thought to be the result of a forgotten childhood injury or infection. The patient was treated successfully with a combination of fluoxetine 20 mg once a day, relaxation training and response prevention, and over a period of 6 months his symptoms receded. He remained well at 12 month follow-up.

Case 2 was an 83 year old woman. At the age of 81 she collapsed at home as a result of a cardiac arrhythmia and subsequently had a pacemaker inserted. Shortly afterwards she developed a number of bizarre rituals involving the compulsion to visualise the faces of TV stars and count when passing through doorways. If she failed to carry out these rituals she had to walk back through the doorway and complete additional counting before she was able to continue. She suffered from arthritis which severely limited her mobility so these procedures took several hours each day and as a result depressive symptoms developed. She had experienced a brief period of similar problems during the 2nd World War, when she was 29 years old, but had recovered spontaneously after a few months. Her daughter also suffered from OCD.

Brain CT showed lacunar infarcts in the left basal ganglia and corona radiata. The patient refused treat-

ment with fluoxetine after coming across adverse publicity about the drug. She was prescribed paroxetine but she complied poorly and was resistant to offers of behavioural therapy. She remained symptomatic a year later.

Case 3 was an 82 year old man who, at the age of 81, suffered 2 transient ischaemic attacks each lasting about 2 hours and characterised by dysphasia and right sided sensory and motor symptoms. A few weeks later he developed a 'phobia' of dog's faeces together with recurrent, intrusive thoughts about contamination, and compulsions to check the house and visitors' shoes for faeces. He washed his hands repeatedly. These rituals took 3 to 4 hours per day to perform. He had a past history of a fractured skull at 29 and minor poliomyelitis at 39 which left him with no residual signs but which was followed by a phobia of dirt and handwashing rituals. These resolved spontaneously after about a year. He was physically fit apart from a mild hand tremor which was worse on the right side.

Brain CT scan showed moderate cortical atrophy and periventricular lucencies. The patient responded to treatment with paroxetine 20 mg once a day alone. His symptoms resolved over a period of 4 months and he remained well after 12 months.

Case 4 was a 66 year old man. He had suffered from lumbar pain since the age of 35 and had become a chronic user of prescribed narcotic analgesics which had led to renal failure in his late fifties. He had been a research chemist but had retired on medical grounds at the age of 45. Thereafter he had adopted a reclusive and disorganised lifestyle. At the age of 60, in an attempt to kill some bothersome house-flies, he used a whole canister of a commercially restricted organophosphate insect spray (2% pirimiphosphomethyl) in a confined space. At the time he became faint but did not lose consciousness. Over the following months he developed an obsessional slowness of thinking and speaking, subjective memory impairment, and orofacial and truncal abnormal involuntary movements. He became depressed and even more socially withdrawn. In addition to the involuntary movements he had a bilateral hand tremor with cogwheel rigidity and impaired co-ordination on the left side.

Renal function tests revealed mild chronic renal failure and brain CT a moderate degree of cerebral and cerebellar atrophy. Treatment with paroxetine was attempted but the patient did not comply and he remained symptomatic at 1 year follow-up.

2.2. Assessments

The severity of obsessive-compulsive symptomatology was assessed using the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS, [13]). Depressive symptoms were evaluated using the Montgomery-Asberg Depression Rating Scale (MADRS, [23]). All cases fulfilled ICD-10 criteria for OCD (WHO, [36]). Premorbid intelligence was predicted using the National Adult Reading Test (NART, [24]). General cognitive performance was assessed using the Mini-Mental State Exam (MMSE, [12]), primary memory function by digit span and secondary memory function the Object Learning test (OLT, [19]). The Very Short Minnesota Aphasia Battery [25] was used as a screen for language impairment and the FAS verbal fluency [15] and animal names category fluency test [35] were used to examine verbal fluency. The NART score was used to derive a predicted verbal fluency score by the method of Crawford et al. [9]. Lastly, psychomotor speed were assessed using the Digit Copying test (DCT, [19]) and Trail-making A [26]. Trail-making B was used as a simple test of executive function. Normative data for the Trail-making test was taken from [29]. Y-BOCS and MADRS were repeated at one year follow-up.

3. Results

Table 1 summarises the clinical data. 3 of the 4 patients had entered higher education and had above average premorbid IQs as estimated by the NART [24]. The MADRS scores were moderately high in cases 1 and 4 but in all cases the history clearly suggested the OC symptoms *preceded* the depressive symptoms. Y-BOCS scores indicated moderately severe disorder. OCD remitted in cases 1 and 3, as indicated by a fall in Y-BOCS scores to 10. Both patients' MADRS scores fell below 6 which is the cut-point for that scale [28].

Table 2 shows results of neuropsychological tests performed. MMSE was within normal limits for the patients age, as were digit span and OLT scores. Case 4 had the lowest score on the OLT test but this was still above the cut-point for dementia at his age. No language deficits were identified using the screening test. Verbal fluency was impaired in Case 4 and, in particular, letter fluency was significantly below the predicted score. Case 4 was also impaired on DCT and Trail-making. Lastly, Case 2 was impaired on Trail-making.

Table 1
Demographic and clinical data at baseline assessment and one year follow-up

Case	1	2	3	4
Age (years)	74	83	82	66
Years of Education	11	9	15	16
Estimated premorbid IQ	122	97	123	121
MADRS score (follow-up score)	27 (4)	11 (12)	8 (5)	23 (23)
Y-BOCS score (follow-up score)	29 (10)	25 (24)	22 (10)	23 (26)

Table 2
Neuropsychological test results

Case	1	2	3	4
MMSE	30	27	29	29
Digit span scaled score	11	10	10	9
Object Learning Test quotient	109	103	117	85
Very Short Minnesota error score	0	3.5	0.5	1
FAS letter fluency (words per min)	14	9.3	14	8.7 ^a
Predicted letter fluency	17.3	9.7	17.7	17
Category fluency (words per min)	26	12	19	9 ^a
Digit Copying Test quotient	109	90	113	79 ^a
Trail-making A (s)	48	100 ^a	67	123 ^a
Trail-making B (s)	118	333 ^a	106	321 ^a

^aScores below the cut-point for dementia (see references in text).

4. Discussion

A relationship between neurological disease and OCD has long been suspected [31] and an early case control study by Grimshaw [14] found that 20% of OCD patients had a history of neurological disease compared to 8% of non-obsessional neurotic 'controls'. Prominent among the list of conditions identified by Grimshaw were Sydenham's chorea, infant convulsions, diphtheria and encephalitis. The association between OCD and Sydenham's chorea has been confirmed more recently [30]. Neuroimaging studies of OCD have demonstrated abnormalities in cerebral structure, especially in the basal ganglia [10, 18, 21, 34]. Berthier et al. [4] compared cases with OCD acquired secondary to brain injury and 'idiopathic' OCD. The former group had a variety of lesions in the frontal, temporal and cingulate cortices or the basal ganglia, and were more likely to have a later onset of symptoms and a negative family history. Case studies have described associations between OCD and infarcts in the right parietal lobe [27] or the basal ganglia [22], and organophosphate poisoning [5].

Known vulnerability factors seem to have been present in three of the four cases in this series; Sydenham's chorea (case 2), poliomyelitis (case 3) and the circumstantial evidence provided by the presence of hemicranium (case 1). Precipitating factors were present in three of the four cases; basal ganglia in-

farction (case 2), transient ischaemic attacks (case 3), and high-level organophosphate exposure (case 4). In cases 3 & 4 the OCD was found in association with motor abnormalities. Tomer et al. [33] reported that the severity of OCD was correlated with the severity of *left* sided motor signs in patients with Parkinson's Disease and others have emphasised the similarities between neuropsychological profiles of the two disorders [17].

In addition, 3 of the cases had experienced brief periods of OC symptoms associated with depression or anxiety earlier in their lives. These episodes had all resolved spontaneously but would indicate an earlier predisposition to OCD. These cases had experienced risk factors prior to the onset of the first episodes. Case 4 may have developed obsessional traits long before the onset of more pervasive obsessional symptoms but the evidence is only circumstantial.

None of the patients had clinical evidence of global cognitive impairment but in two patients (cases 2 & 4) poor verbal fluency, executive function and psychomotor speed were found. Several studies have reported impaired verbal fluency in patients with OCD [1, 7, 16, 21]. The latter authors also found OCD patients had impaired performance on the Trail-making test. These impairments are considered to indicate frontal lobe dysfunction, although evidence of other disruption of executive function is inconsistent [32]. Tallis [32] drew attention to the reported im-

pairments of visuo-spatial memory and any tests with a timed element in OCD. Berthier et al. [4] found that neuropsychological performance was similar in patients with OCD acquired as a result of brain injury and idiopathic OCD patients.

The presumed organic aetiology in some late onset cases of OCD does not preclude the possibility of successful treatment. Cases of elderly patients responding to serotonergic antidepressants and/or behaviour therapy exist [2, 6, 8, 27]. In this series, the patients who complied with treatment had a good outcome.

The cases described here support the view that organic factors may be important in the recrudescence of OCD in late life. We have only found 3 cases in the literature in which late onset OCD arises in the absence of a presumed organic precipitant [2, case 2], [3, 6]. While this may reflect a reporting and publication bias it may also reflect the importance of organic factors.

Given the paucity of information regarding OC symptomatology in late life *per se*, a systematic study of OC syndrome in the broad range of psychiatric disorders affecting the elderly is now indicated.

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