LETTERS TO THE EDITOR

Obsessive-compulsive behaviour and cognitive impairment in a parkinsonian patient after left putaminal lesion

Various studies have reported the occurrence of obsessive-compulsive behaviour in patients with different neurological disorders mostly affecting the basal ganglia. The association between obsessive-compulsive behaviour and structural lesions of the basal ganglia has been well documented.

We describe here a parkinsonian patient who, after an ischaemic lesion confined to the left putamen, developed polymorphic obsessive-compulsive behaviour and dementia with clinical features of frontal lobe dysfunction (apathy, grasp reflex, utilisation, and obsessive-compulsive behaviour).

The patient was a 63 year old, right handed housewife with a four year education. At the age of 37 she developed bilateral upper limb resting tremor and rigidity, followed by progressive slowing of movement and akinesia. She was treated for several years with levodopa and benserazide, which improved her motor performance. The patient presented the typical clinical picture of idiopathic Parkinson's disease.

Regular neurological and neuropsychological follow up did not disclose any sign of cognitive impairment or any behavioural abnormality until July 1991, when she was aged 70. During a hypertensive episode, the patient had a left hemispheric ischaemic accident, which acutely brought about a temporary severe reduction of spontaneous speech. Until then, insight, judgement, and effectiveness in performing customary daily activities had been entirely preserved. On admission, she had mutism and a severe parkinsonian syndrome with rigidity and pronounced flexion of the trunk, and mild inconstant resting tremor, more evident in her upper left limb. No other neurological signs were seen. Muscle strength and reflexes were unaltered. Some days after the ischaemic episode, the patient recovered from mutism and started to report obsessive thoughts (fear that her daughter would divorce, that her son would go to jail, etc.).

In the subsequent weeks, changes in social conduct and personality and increasing apathy, together with cognitive decline, gradually appeared. In September 1991, the patient began to exhibit different compulsions, such as repetition of sentences and of single words, going up and down the stairs, and putting on and taking off her shoes. At that time, she presented a left sided palmo-mental reflex, bilateral grasp reflex, and utilisation behaviour. The unified Parkinson's disease rating scale motor score in defined "off" conditions was 75 (maximum score 100), indicating severe impairment. The compulsive verbal alterations gradually became more and more persistent and other compulsive behaviours (coprolalia and aggressive outbursts) also emerged. Since November 1991, the compulsive verbal alterations had occurred daily for about 18-19 hours and disappear only during sleep, making family life exasperating.

Brain MRI in February 1992 disclosed a small area of increased signal intensity in the anterior portion of the left putamen on T2 weighted and proton density images (figure, A). A slight bilateral reduction in size of the substantia nigra pars compacta was also seen. No other structural brain abnormalities were detectable. These findings were unchanged on a follow up MRI one year later. SPECT was performed in April 1992, while the patient was at rest but producing her customary verbal iterations. Statistical analysis was carried out by computing regional cerebral blood flow (rCBF) right to left ratios in 14 regions of interest. SPECT showed a considerable reduction of rCBF in the left frontal cortical regions, and in the basal ganglia and the thalamus of the left hemisphere (figure, B). These findings are in keeping with earlier clinical and SPECT findings showing that a dysfunction of the frontal cortex may result from selective damage to the basal ganglia.

In April 1992, the patient was given a battery of neuropsychological tests, including tasks of verbal and spatial memory (digit span, Corsi's span forward and backward), Rey's auditory verbal learning test, tasks sensitive to frontal lobe dysfunction (Wisconsin card sorting test, verbal fluency), Raven's progressive matrices, and tasks of constructional apraxia (copy of drawings).

The table shows the results of neuropsychological assessment and the mean scores obtained by 30 healthy controls matched for age and education. The patient was severely impaired on all the cognitive tasks. During the neuropsychological testing, she was uncooperative and unconcerned, showing a remarkable difficulty in paying attention to any task. The patient was continuously and compulsively producing verbal iterations, mainly consisting of repetition of her daughters' names. Perseverative behaviour occurred across several tests, including tasks sensitive to frontal lobe dysfunction. For instance, she produced very many perseverative errors not only on the Wisconsin card sorting test, but also on Raven's progressive matrices. On the second test, she repeatedly pointed to responses in the same spatial position as the previous one.

To quantify the severity of her compulsive symptoms, the patient was also rated with the compulsive items (items 6 through 10) of the Yale-Brown obsessive compulsive scale. On this scale, the compulsive subtotal score was 18, consistent with a very severe compulsive behaviour (maximum score = 20).

Until now, periodic follow up clinical and neuropsychological examinations have shown no further cognitive decline and no significant change in her obsessive-compulsive behaviour.

To our knowledge, this is the first report on the occurrence of obsessive-compulsive behaviour after a unilateral lesion restricted to the left putamen. The association between neurological disorders and obsessive-compulsive behaviour has been mostly reported in patients with bilateral basal ganglia lesions. Interestingly, Marangone et al described an adolescent boy who, after a small infarction in the right putamen, developed obsessional thoughts and complex motor stereotypes which, however, were not defined as compulsions.

It seems likely that in our patient the putaminal lesion might have played a crucial part in the determinism of obsessive-compulsive behaviour. It has already been shown that focal lesions of the basal ganglia thm-

**Performance on neuropsychological testing**

<table>
<thead>
<tr>
<th>Task</th>
<th>Patient scores</th>
<th>Control scores mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digit span</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Forward</td>
<td>3</td>
<td>5.1 ± 0.8</td>
</tr>
<tr>
<td>Backward</td>
<td>2</td>
<td>3.3 ± 0.9</td>
</tr>
<tr>
<td>Corni's span</td>
<td>2</td>
<td>4.9 ± 0.8</td>
</tr>
<tr>
<td>Backward</td>
<td>1</td>
<td>3.7 ± 1.0</td>
</tr>
<tr>
<td>Rey's auditory verbal learning test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Immediate recall (max = 75)</td>
<td>35.0 ± 6.6</td>
<td></td>
</tr>
<tr>
<td>Delayed recall (max = 15)</td>
<td>6.7 ± 2.4</td>
<td></td>
</tr>
<tr>
<td>Wisconsin card sorting test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of correct responses (max = 6)</td>
<td>4.7 ± 1.9</td>
<td></td>
</tr>
<tr>
<td>Total errors (max = 128)</td>
<td>33.7 ± 26.3</td>
<td></td>
</tr>
<tr>
<td>Ratio perseverative/total errors</td>
<td>93%</td>
<td></td>
</tr>
<tr>
<td>Verbal fluency (F, A, S)</td>
<td>21.4 ± 9.8</td>
<td></td>
</tr>
<tr>
<td>Raven's progressive matrices</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correct responses</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(max = 10)</td>
<td>21.7 ± 4.7</td>
<td></td>
</tr>
<tr>
<td>Copying of drawings</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correct responses</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(max = 10)</td>
<td>8.7 ± 1.9</td>
<td></td>
</tr>
</tbody>
</table>

Figure 1 (A) Axial T2 weighted MRI (February 1992): small area of increased signal intensity in the anterior portion of the left putamen. (B) **Ts HM-PAO SPECT (April 1992): reduction in regional cerebral blood flow in the frontal regions, the basal ganglia (thin arrow), and the thalamus (thick arrow) of the left hemisphere on axial images.**
selves may be sufficient to give rise to obsess-
ive-compulsive behaviour.1 The behavioural
symptoms appeared in our patient after an interval
of 3 months (obsessions to weeks of com-
pulsions, apathy) after the putaminal stroke and
progressed over subsequent months. Likewise, pa-
ients have been described who presented obsessive-compulsive behav-
ior or “psychic akinnesia” (apathy, lack of moti-
vation) after a period varying from days to
years after basal ganglia lesions, often with a
progressive worsening over time in the initial
phase.1 The behavioural changes in our patient occurred about 23 years after
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