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Obsessive-Compulsive Disorder and Traumatic Brain Injury: Behavioral, Cognitive, and Neuroimaging Findings

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Objective: The goal of this study was to evaluate behavior and cognition in a consecutive series of patients who developed obsessive-compulsive disorder (OCD) after suffering a traumatic brain injury (TBI). **Background:** Because OCD is a rare sequelae of TBI, the phenomenology of obsessions and compulsions, the comorbid psychiatric disorders, the performance on cognitive tests, and the neural correlates have not been well characterized. **Methods:** Ten adult patients who met DSM-IV diagnostic criteria for OCD after suffering either mild (6 cases), moderate (2 cases), or severe (2 cases) TBI were studied using structured psychiatric rating scales (i.e., Yale-Brown Obsessive Compulsive Scale), cognitive tests, and magnetic resonance imaging (MRI). **Results:** Global severity of OCD ranged from moderate to severe, and all patients had multiple obsessions and compulsions. There was a high frequency of aggressive, contamination, need for symmetry/exactness, somatic, and sexual obsessions as well as cleaning/washing, checking, and repeating compulsions. Unusual features such as obsessional slowness (3 cases) and compulsive exercising (3 cases) were also documented. Comorbid psychiatric diagnoses were common and included posttraumatic stress disorder, anxiety with panic attacks, depression, and intermittent explosive disorder. Compared with 10 age-matched normal controls, the OCD group had poor performance on tests of general intelligence, attention, learning, memory, word-retrieval, and executive functions; these cognitive deficits were more pervasive among patients displaying obsessional slowness. All OCD patients with mild TBI had normal MRI scans, whereas focal contusions in the frontotemporal cortices, subcortical structures (caudate nucleus), or both were found in OCD patients with moderate and severe TBI. **Conclusions:** Posttraumatic OCD has a relatively specific pattern of symptoms even in patients with mild TBI and is associated with a variety of other psychiatric disorders, particularly non-OCD anxiety. The patterns of cognitive deficits and MRI findings suggest dysfunction of frontal-subcortical circuits. (NNBN 2001;14:23–31)

Impulsivity, affective instability, and disinhibition are the most frequent neuropsychiatric symptoms associated with traumatic brain injury (TBI) (1), whereas depression (2), mania (3,4), schizophrenia-like psychosis (5,6), and

personality disorders (7) are less frequent. Anxiety disorders (generalized anxiety disorder, panic, phobias, and stress disorders) are quite common after TBI to the extent that approximately 30% of injured patients have clinically significant anxiety (8,9). Despite the fact that early studies described an incidence of obsessive-compulsive disorder (OCD) in the TBI population ranging from 0.5% to 7.8% (incidence rates are not mentioned in subsequent reports) (8), a full-blown OCD has rarely been described after TBI (8,10–18).

The emergence of OCD complicating the course of TBI has been reported in individual patients (10–13) or in small series lacking control groups (14–16). This

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could explain the paucity of information available in the literature on demographic variables such as family and personal history of psychiatric disorders, age of onset, interval between TBI and OCD, and risk factors for the development of obsessive and compulsive (OC) symptoms. In addition, there are still some discrepancies about whether this syndrome can fully develop after TBI, and it is unclear whether OC symptoms predated the TBI incident in some cases. Because of the rarity of OCD after TBI, information on its clinical phenomenology is scanty, and an analysis of the literature reveals that there is a difference in the frequency of OC symptoms based on how patients were evaluated. Multiple OC symptoms were mostly observed in patients who were formally evaluated using symptom inventories or checklists (11,16), whereas more restricted patterns of OC behaviors were found in others who were only clinically examined (standardized psychiatric scales were not used), thus rendering the identification of the full spectrum of OC symptoms difficult (10,12,14–16). In the few cases of posttraumatic OCD that have been examined with cognitive testing, the results were disappointing, because mild deficits in visual-spatial memory, verbal learning, and attentional and executive functions were found in three patients with TBI ranging from mild to moderate (16), whereas average performance on these cognitive tasks was found in another patient with severe TBI (11). Anxiety and affective disorders were the most common psychiatric concomitants of OCD, although the characterization of these disorders was vaguely described (11–16).

During the past years, we have studied 10 adult patients who developed an OCD after TBI. These patients were studied with psychiatric rating scales and a battery of cognitive tests tapping major aspects of cognition. Based on previous reports of acquired OCD (10–13,15–17), we hypothesize that OCD occurring after TBI may be associated with other anxiety disorders, impaired performance on cognitive tests sensitive to frontal and temporal dysfunction, and focal lesions in regions belonging to frontal-subcortical circuits.

METHODS

Patients

The sample consisted of 10 patients who were consecutively referred to the Behavioral Neurology Unit of the Clinic University Hospital (Malaga, Spain) during the period between July 1991 and July 1996 with a diagnosis of OCD after TBI. Of these, 6 patients were briefly described in previous reports (17,18) and 4 were new patients. The severity of TBI was based on the lowest score on the Glasgow Coma Scale (GCS) (19) that the patients had at the moment of hospital admission. The GCS measures motor, eye opening, and verbal responsiveness, and scores

range from 3 (unresponsive) to 15 (normal). Thus, the patients were classified as having either mild (GCS score: 13–15 points; 6 cases), moderate (GCS score: 9–12; 2 cases), or severe (GCS score: 3–8; 2 cases) TBI. Eight patients had injuries limited to the cranial facial region, whereas only 2 patients had injuries in multiple body systems (multiple fractures in the left tibia in 1 patient and a collapsed lung in the other). Eight patients had closed TBI injuries, and 2 had cerebrospinal fluid fistulas with rhinorrhea and intracranial air or otorrhea. All patients were injured in motor vehicle accidents, and no patient had suffered multiple head injuries. Follow-up evaluations were carried out in all patients for at least 1 year after the initial assessment, and response to different medication trials was also assessed.

Controls

Neuropsychological performance of the OCD group was compared with that of a group comprising 10 normal control (NC) individuals recruited from the hospital staff according to recent proposed criteria for normal volunteers (20). The OCD and NC groups were matched on the basis of age, sex, and education. None of the NC subjects exhibited psychopathologic findings or major systemic illnesses that could affect cognitive performance. Neuroimaging studies were not performed in the NC group.

Neurologic Evaluation

All patients received a complete neurologic evaluation carried out by a board-certified neurologist.

Psychiatric Evaluation

Information about family history of psychiatric disorders was obtained on all patients. Moreover, the existence of a personal history of psychiatric symptoms (including anxiety and affective disorders), use and/or abuse of alcohol or other substances, tics, and Tourette syndrome (14,21) was specifically tested by interviewing the patients themselves and at least one close relative.

OCD

Psychiatric diagnoses of the patients were based on the Structured Clinical Interview for DSM-III-R: Patient Version (22). According to DSM-III-R (23) and DSM-IV (24) criteria, OCD was defined by the presence of obsessions and compulsions that caused marked distress or interfered with the patient's normal routine (25). The clinical characteristics and severity of OC symptoms were further investigated using the following psychiatric rating scales.

Yale-Brown Obsessive Compulsive Scale. This is a clinician-rated scale that contains two components: a symptom checklist designed to elicit past and current obsessions and compulsions organized in 15 categories

according to their thematic content (e.g., aggressive obsessions, counting compulsions) and a severity scale composed of five items for obsessions and five items for compulsions (25,26). Each item is rated from 0 (no symptoms) to 4 (extreme symptoms).

In the present study, the severity of OC symptoms was measured with this scale. A total score ≥ 16 indicates clinically significant OC symptoms. Associated behaviors (i.e., avoidance, slowness, indecisiveness, overvalued sense of responsibility, pathologic doubting) were also investigated.

Leyton Obsessional Inventory. This is a self-rated scale that quantifies the range of obsessional thoughts and compulsive behaviors (27). It contains 69 “yes/no” questions dealing with the subjective assessment of obsessional symptoms (questions 1–46) and traits (questions 47–69) as well as the degree of resistance and interference with the patient’s life. Because the resistance and interference scales are time-consuming, these measurements were not administered, and the severity of OC symptoms was only assessed with the previous scale.

Slowness Questionnaire. This is a self-rated scale to assess pervasive slowness in activities of daily living (i.e., dressing) (28). Patients were asked to rate the speed of their own performance on each of these activities in comparison with healthy persons. The maximum possible score is 60, and scores ≥ 30 indicate a clinically significant slowness.

Compulsive Exercising. Compulsive exercising was diagnosed when the degree and frequency of exercising were well beyond what would be the normal range for persons of similar age (29). As part of the psychiatric evaluation, patients and family members were asked if physical activity was judged to be “out of control.”

Comorbid Psychopathology

The occurrence of comorbid psychiatric disorders usually associated with both OCD (i.e., non-OCD anxiety, affective disorders) and TBI (i.e., loss of impulse control) were also examined using the Structured Clinical Interview for DSM-III-R: Patient Version (22). In addition, the characteristics and severity of comorbid psychopathologic symptoms were assessed with the following scales.

Hamilton Depression Rating Scale. This is a 17-item scale that rates psychological and physiologic symptoms of depression in a range from 0 (no depressive features) to 52 (maximum score), with a score ≥ 15 indicating major depression (30).

Tyrer Brief Scale for Anxiety. This is a 10-item scale that measures psychic complaints and somatic symptoms and signs of anxiety in a range from 0 (no anxiety) to 60 (maximum score) (31).

Impact of Event Scale. This is a self-report questionnaire that examines intrusive and avoidant symptoms as-

sociated with posttraumatic stress disorder (32). The intrusion subset contains seven questions (range subscale score: 0–35), whereas the avoidance subset contains eight questions (range subscale score: 0–40) (33). The patients indicated their experiences during the past 7 days but not during the entire period since the TBI.

Neuropsychological Assessment

Cognitive performance was assessed by using a battery of neuropsychological tests thought to be sensitive to the variety of deficits associated with both TBI (34) and OCD (35), which assessed intellectual, memory, language, and executive functions. This battery included the Wechsler Adult Intelligence Scale (36), the Wechsler Memory Scale (WMS) (37), the Boston Naming Test (BNT) (38), the Controlled Oral Word Association Task (COWAT) (39), the Wisconsin Card Sorting Test (WCST) (40), the Road Map Test (RMT) (41), and the Trail-Making Test (TMT) (parts A and B) (42).

Neuroimaging

After giving informed consent, magnetic resonance imaging (MRI) was performed for all patients. The same MRI unit (1.5-T Signa scanner; General Electric Medical Systems, Milwaukee, WI) was used to scan all patients. All MRI scans were performed using the same protocol (17), and images were evaluated by one investigator (who was unaware of the clinical assessment) for the presence of structural brain abnormalities usually associated with TBI (i.e., focal contusions, hemorrhages, cerebral atrophy).

Statistical Analysis

Statistical data are expressed as means and SDs. Categorical data were analyzed by χ^2 test. Between-group comparisons (OCD vs. NC) of scores on neuropsychological scales were tested with nonparametric Mann-Whitney *U* tests. Within-group comparisons (i.e., patients with obsessional slowness vs. patients without this behavior) were compared using planned *t* tests. Probability values reported are two-tailed, with significance set at $p < 0.05$. Because of the exploratory nature of this study, we decided not to correct for type I errors.

RESULTS

Demographic Findings

Demographic characteristics and scores of psychiatric rating scales are shown in Table 1. Nine of the 10 patients had a negative family history of OCD, tics, or Tourette syndrome. The maternal aunt of 1 patient had a diagnosis of acquired OCD due to a cavernous angioma of the left temporal lobe (17). Four patients had a positive personal history of psychiatric symptoms before TBI. Two patients

TABLE 1. *Demographic information and scores on psychiatric rating scales for obsessive-compulsive disorder and normal control groups*

	Obsessive-compulsive disorder group (N = 10)	Control group (N = 10)
Sex (M/F) (% male)	4/6 (40)	4/6 (40)
Age (years) (mean \pm SD)	30 \pm 9.3	29.6 \pm 11.1
Education (years) (mean \pm SD)	10.6 \pm 2.5	11.8 \pm 2.8
Psychiatric history (% positive)	40	0
Duration of OCD (months) (mean \pm SD)	51.1 \pm 63.9	
Interval between OCD onset and testing (months) (mean \pm SD)	54.4 \pm 68.9	
Yale-Brown Obsessive Compulsive Scale (mean \pm SD)		
Obsessions	12.5 \pm 3.2	
Compulsions	11.7 \pm 3.1	
Leyton Obsessional Inventory (mean \pm SD)		
Symptoms	27.6 \pm 6.4	
Traits	13.3 \pm 2.9	
Slowness Questionnaire (mean \pm SD)	15.2 \pm 17.7	
Hamilton Depression Rating Scale (mean \pm SD)	15.6 \pm 6.2	
Tyrer Brief Scale for Anxiety (mean \pm SD)	23.0 \pm 5.5	
Impact of Event Scale (mean \pm SD)		
Intrusion	25.6 \pm 4.3	
Avoidance	20.6 \pm 6.6	

OCD, obsessive-compulsive disorder.

had lifelong indecisiveness and perfectionism but not OC symptoms, and another 2 patients had suffered posttraumatic stress disorder during adolescence related to sexual abuse but were free of stress symptoms, at least during the 3 years before TBI (18). No patient suffered from use and/or abuse of alcohol or other substances or was intoxicated at the time of TBI. At the time of entrance in the study, 5 patients were receiving disability insurance, 3 patients were housewives, and 2 patients were employed. No patient was pursuing claims for compensation.

Nine patients developed OC symptoms and comorbid psychopathology, particularly generalized anxiety and symptoms of posttraumatic stress disorder, within the first month after the TBI. The remaining patient had posttraumatic stress disorder in the acute period but developed typical OC symptoms 8 months after the TBI. At the time of referral to our unit, all but 2 patients (cases 5 and 6) had OC symptoms of long duration (see Table 1), and 3 of them had received previous evaluations and psychopharmacologic treatments (75 mg per day of clomipramine in 2 patients; 40 mg per day of fluoxetine in 1 patient) with partial benefit.

Neurologic Findings and Neuroimaging Correlates

Five of the six patients with mild TBI had normal neurologic examinations, although one patient in this group showed transient anosmia. All six patients had normal MRI scans, and in two patients who underwent single photon emission computed tomography, there were decreased perfusion rates in the left frontotemporal region (case 5) or inferior parietal cortices bilaterally (case 4).

One of the two patients (case 7) with moderate TBI had a normal neurologic examination. The MRI scan showed laminar bifrontal hygromas and atrophic changes in the head of the left caudate nucleus. The other patient (case 8) with moderate TBI in the acute period had otorrhea and palsies of the seventh and eighth cranial nerves on the right side due to a transversal temporal bone fracture and later developed stereotyped right hand movements. The MRI scan revealed multiple contusions affecting the left orbitofrontal and mesial temporal cortices as well as the white matter anterolateral to the head of the caudate nucleus. One of the two patients (case 9) with severe TBI had impaired alternating movements with the left hand, and an MRI scan (performed several years after TBI) showed a small contusive focus in the left anterior cingulate cortex and marked atrophic changes in the frontotemporal cortices and left caudate nucleus. The remaining patient (case 10) with severe TBI had left visual loss and anosmia associated with small cortical contusions involving the orbitofrontal regions bilaterally. In the acute period, this patient had suffered fractures in the ethmoidal and left orbital walls, causing a cerebrospinal fluid rhinorrhea with intracranial air and transient mutism and double incontinence. Electroencephalograms were normal in nine patients, and one patient (case 9) had bilateral slowing over the frontotemporal cortices. No patient experienced seizures.

OC Symptom Severity and Phenomenology

All patients had total scores on the Yale-Brown Obsessive Compulsive Scale that reached the cutoff score of 16

points or higher for clinically significant OCD (25,43), with similar severity scores for obsessions and compulsions. All patients reported multiple obsessions and compulsions unrelated to the direct consequences of TBI (6 patients also described OC symptoms thematically related to the circumstances of TBI). The mean overall number of current obsessions was 3.7 (SD = 1.5), and the mean overall number of current compulsions was 3.6 (SD = 1.1). The most common obsessions were aggression and contamination, followed by symmetry/exactness, somatic, and sexual obsessions, whereas checking, cleaning/washing, and repeating were the most frequently observed compulsions (Table 2). Associated behaviors (i.e., avoidance, pathologic doubting) were also common.

Obsessional Slowness

Three patients (cases 4, 9, and 10) displayed obsessional slowness. In case 4 (a 31-year-old man with minor TBI), obsessional slowness was related to indecisiveness and mental rituals. His score on the Slowness Questionnaire was 27 points. In case 9 (a 46-year-old man with severe TBI), obsessional slowness was related to rumination, excessive preoccupation with tidiness, and newly acquired OC personality traits (perfectionism, orderliness). His score on the Slowness Questionnaire was 28 points. In case 10 (a 35-year-old woman with severe TBI), obsessional slowness was the most incapacitating OC symptom, and it was apparently due to the use of mental strategies of avoidance related to contamination and repetitive wash-

ing/cleaning rituals. Her washing compulsions were so prominent that she developed eczematoid dermatitis in her hands and bilateral carpal tunnel syndrome. Her score on the Slowness Questionnaire was 55 points. Although two of these three patients (cases 4 and 9) scored just below the cutoff score of 30 points in the Slowness Questionnaire established by Hymas et al (28) required to be classified as having clinically significant obsessional slowness, all three patients had prominent slowness in activities of daily living, and they were designated as "outliers" on the Slowness Questionnaire because they scored 2 or more SDs above the mean obtained by the remaining seven OCD patients without obsessional slowness. Therefore, they were grouped together, and their scores on psychiatric rating scales and neuropsychological tests were compared with those obtained by the remaining OCD patients without obsessional slowness. Patients with obsessional slowness obtained significantly higher mean scores on the compulsion scale of the Yale-Brown Obsessive Compulsive Scales (14.6 ± 3.0) than patients without obsessional slowness (10.7 ± 2.2) ($t = -2.33$; $df = 8$; $p = 0.047$). On the Leyton Obsessional Inventory, there was a tendency for patients with obsessional slowness to have higher mean scores on the symptom scale (32.3 ± 6.5) than patients without obsessional slowness (25 ± 5.0) ($t = 1.95$; $df = 8$, $p = 0.086$).

Compulsive Exercising

Three patients (cases 7–9) had compulsive exercising, and the strenuous physical activity coexisted with obsessional slowness in one of them (case 9). Patient 7, a 21-year-old man with moderate TBI, indulged for a brief period in strengthening exercise activities (about 400 sit-ups daily in spite of having bone fractures in his left leg) against medical advice while in the hospital. Patient 8 was a 46-year-old man with moderate TBI who ran about 15 miles daily (in spite of having psoriatic arthritis affecting his feet). Patient 9 was a 46-year-old man with severe TBI who, although he had never taken part in exercising routines before the TBI, became obsessed with exercising and began to run 6 km daily for 6 years after the onset of OCD. In these three patients, compulsive exercising was unrelated to abnormal eating habits. These three patients with compulsive exercising were grouped together, and their scores on psychiatric rating scales and neuropsychological tests were compared with those obtained by OCD patients without compulsive exercising ($n = 7$). Between-group comparisons of the scores on psychiatric rating scales did not show statistically significant differences.

Comorbid Psychopathology

Associated diagnoses seen most frequently in the present sample included non-OCD anxiety (100%), depression

TABLE 2. *Symptom categories in patients with obsessive-compulsive disorder due to traumatic brain injury*

Symptom categories*	Number	%
Obsessions		
Aggressive (i.e., harm to self or others)	10	100
Contamination (i.e., dirt, germs, illness)	8	80
Sexual	4	40
Hoarding/saving	2	20
Religious (scrupulosity)	1	10
Symmetry/exactness	6	60
Somatic	4	40
Compulsions		
Washing/cleaning	9	90
Checking	10	100
Repeating	8	80
Counting	5	50
Ordering/arranging/straightening	4	40
Hoarding/saving/collecting	4	40
Associated behaviors		
Avoidance	6	60
Slowness	3	30
Indecisiveness	6	60
Overvalued sense of responsibility	6	60
Pathologic doubting	7	70
Compulsive exercising	3	30

*Indicates that symptom categories were derived from the Yale-Brown Obsessive Compulsive Scale, except for compulsive exercising.

(90%), posttraumatic stress disorder (70%), and personality change (aggressive type) (30%) (clinically similar to intermittent explosive disorder). The six patients with minor TBI had posttraumatic stress disorder, generalized anxiety disorder (associated with panic attacks in 4 patients), and depression (major depression in cases 1–5 and depressive disorder not otherwise specified in case 6), and one patient (case 4) additionally had aggressive personality change due to TBI and trichotillomania. Of the two patients with moderate TBI, one (case 7) had posttraumatic stress disorder and mild anxiety, whereas the other (case 8) had generalized anxiety disorder, depression, and aggressive personality change alternating with apathy. The two patients (cases 9 and 10) with severe TBI had generalized anxiety disorder and dysthymic disorder; patient 9 additionally had aggressive personality change alternating with apathy, whereas patient 10 with longstanding OC symptomatology had a period of compulsive alcohol use for 4 years before examination. No patients had motor or vocal tics. All patients with posttraumatic stress disorder reported certain OC symptoms in the symptom checklist of the Yale-Brown Obsessive Compulsive Scale that were related to the circumstances of TBI (intrusive images and hyperreactivity to noises).

Neuropsychological Findings

The performance of OCD patients in neuropsychological tests was significantly poorer than that of NC subjects in measures of general intelligence (Wechsler Adult Intelligence Scale), memory (memory quotient of the WMS,

mental control, logical memory, digit-span, and visual reproduction subtests), language (COWAT and BNT), and executive functions (WCST, RMT, and TMT) (Table 3). It should be noted, however, that the average level of intellectual functioning in the NC group was probably inflated relative to the group of TBI patients because of the inclusion in the former of some subjects with greatly discrepant mean values. Therefore, we also analyzed individual test scores in the TBI group, and performances were considered abnormal when an individual score fell below the normal range established in published normal values (44,45). Major deficits were found in executive, memory, and language domains. Nine patients (90%) had impaired performance on the TMT-part B, 8 patients (80%) on the TMT-part A, 6 patients (60%) on the WCST, and 4 patients (40%) on the RMT. Patients' performance on the WMS was compromised in nearly all subtests. In fact, all 10 patients (100%) had impaired performance on logical memory, 7 (70%) on the memory quotient, 4 (40%) on digit span, 4 (40%) on visual reproduction, 3 (30%) on paired associated learning, and 1 (10%) on mental control. Seven patients (70%) had reduced word retrieval on the BNT and on the word list generation portion of the COWAT. Finally, 2 patients (20%) had decreased verbal IQ scores, and 1 patient (10%) had a decreased performance IQ score.

Comparisons between OCD patients with obsessional slowness ($n = 3$) and those without this abnormal behavior ($n = 7$) showed that the former subgroup was more impaired on tests of memory, language, and executive

TABLE 3. Neuropsychologic testing

	Obsessive-compulsive disorder group (N = 10) (mean \pm SD)	Control group (N = 10) (mean \pm SD)
Intelligence		
WAIS verbal IQ	96.5 \pm 10.5*	119.1 \pm 15.5
WAIS performance IQ	102.2 \pm 10.6*	130.6 \pm 21.1
Memory: Wechsler Memory Scale		
Memory quotient	91.9 \pm 15.7*	112.7 \pm 15.0
Mental control	7.5 \pm 1.2*	9.3 \pm 0.9
Logical memory	6.5 \pm 2.9*	12.0 \pm 3.2
Paired associated learning	15.5 \pm 4.0	16.8 \pm 4.0
Digit span	8.8 \pm 1.9*	11.0 \pm 1.3
Visual reproduction	7.9 \pm 2.9*	12.0 \pm 2.8
Language		
Boston Naming Test (total words)	43.9 \pm 8.5*	54.3 \pm 4.1
Letter fluency (total words)	24.0 \pm 9.3*	46.6 \pm 17.8
Executive Function		
Wisconsin Card Sorting Test (categories)	2.8 \pm 2.6*	4.9 \pm 1.3
Road Map Test (errors)	9.2 \pm 6.2*	1.4 \pm 2.2
Trail-Making Test (part A) (seconds)	75.5 \pm 43.3*	31.0 \pm 13.1
Trail-Making Test (part B) (seconds)	171.0 \pm 94.9*	80.0 \pm 78.8

WAIS, Wechsler Adult Intelligence Scale.

* $p < 0.05$ (Mann-Whitney U Test).

functions. Statistically significant differences were found on memory quotient ($t = 3.7$; $df = 8$; $p = 0.005$), logical memory ($t = 3.9$; $df = 8$; $p = 0.004$), paired associated learning ($t = 5.56$; $df = 8$; $p = 0.0005$), and visual reproduction ($t = 3.15$; $df = 8$; $p = 0.01$) as well as on the BNT ($t = 2.91$; $df = 8$; $p = 0.01$), COWAT ($t = 2.4$; $df = 8$; $p = 0.042$) and TMT (part B) ($t = -2.39$; $df = 8$; $p = 0.043$). Comparison of performance on neuropsychological tests between patients with compulsive exercising ($n = 3$) and those without this behavior ($n = 7$) disclosed that the former subgroup had statistically significant poorer performance on the WCST ($t = 2.44$; $df = 8$; $p = 0.04$) but not in other cognitive tests.

DISCUSSION

This study examined the behavioral, cognitive, and neuroimaging correlates of OCD after TBI. There were several findings. First, OCD is an important sequela of TBI regardless of its severity. Second, the phenomenology of OCD in this group showed that the content of OC symptoms and comorbid psychopathology was remarkably similar to that reported in previous patients with posttraumatic OCD, although some unusual symptoms such as slowness and vigorous exercising were relatively frequent in a subgroup of our patients. Third, the pattern of cognitive deficits is consistent with previous results of OCD after TBI and suggests dysfunction of the frontal-subcortical circuits (46).

In our sample, there was a negative family history for OCD, and only four patients had a positive personal history for OCD and related psychopathologic disorders. Two patients had premorbid obsessional personality traits, and another two had suffered posttraumatic stress disorder several years before TBI (18). Similar abnormal personality traits and anxiety disorders have also been reported in case studies or small clinical series of patients with posttraumatic OCD (11,13). Hence, studies on larger samples are required to further examine whether or not these anxiety disorders are overrepresented among individuals who develop OCD after TBI.

In the present study, 6 of the 10 patients developed OCD after suffering mild TBI. Although a number of studies have suggested that enduring psychiatric symptoms and cognitive deficits after mild TBI are the exception rather than the rule, this might not be exactly the case for OCD, because previous reports indicate that most patients developed severe OC symptoms after suffering mild, often indolent, TBI with no evidence of structural brain changes on computed tomography and MRI scans (15,16). Our findings are consistent with those of an earlier study of psychiatric disorders in 50 outpatients with TBI (47). Fann and co-workers (47), using the Hopkins Symptom Checklist 90—Revised (a self-report inventory

for psychiatric symptoms), found that patients with mild TBI developed depression, anxiety, and OC symptoms together with distress and somatization more frequently than patients with moderate or severe injuries. In addition, one epidemiologic study of OCD in adolescents found that a past history of TBI was the only significant medical event in this population (48), thus raising the possibility that TBI, at least in certain populations, might be a risk factor for the development of OCD.

It has been noted that free-floating anxiety and heightened emotional arousal (stress reactions, posttraumatic stress disorder) after the TBI episode can precede OC symptoms (49). Our findings are also consistent with these observations, because all six of our patients with mild TBI had generalized anxiety and posttraumatic stress disorder before the onset of OCD. Although some symptoms of posttraumatic stress disorder overlapped with those of postconcussive syndrome (i.e., dizziness, insomnia, decreased concentration), other complaints (i.e., hyperarousal; intrusive memories and images; avoidance of thoughts, feelings, and activities reminding the subject of the original traumatic event) have exclusively been described in posttraumatic stress disorder (24). Even though MRI scans were negative in this subgroup, the coexistence of different anxiety disorders (OCD, posttraumatic stress disorder, panic, and phobias) might have resulted from disruption of a common neural substrate that encompasses the paralimbic cortical regions, inferior frontal cortex, and subcortical gray nuclei (46,50). Thus, it could be speculated that the physical effects of mild TBI induced a symptomatic overlap of OCD with related anxiety disorders, presumably due to a dysregulation of multiple neural systems, such that anxious symptoms were improperly heightened in response to environmental and interoceptive signals, thereby motivating the use of neutralizing responses to cope with stressors.

One notable aspect of the present study was that the clinical phenomenology of OC symptoms associated with TBI was examined using psychiatric rating scales in a consecutive series of patients for the first time. All our patients showed multiple OC symptoms: aggressive, contamination, symmetry/exactness and somatic obsessions were the most commonly reported obsessions and checking, cleaning/washing, and repeating rituals were the most commonly accompanying compulsions. We found a remarkably similar constellation of OC symptoms on reviewing data gathered in clinical studies or using the Yale-Brown Obsessive Compulsive Scale symptom checklist in single cases and small series of patients (total of 12 patients) with OCD secondary to TBI (10–13,15,16).

Instances of obsessions without compulsions or the reverse pattern have been reported in two patients with severe TBI involving the frontal lobes bilaterally (11,12), but this symptom dissociation was not observed in our

sample. Interestingly, our patients as well as those from the literature (total of 22 cases) showed the same pattern of co-occurrence of obsessions and compulsions (i.e., aggressive obsessions with checking compulsions) that has been identified among patients with idiopathic OCD (51). Given the small number of patients, however, these symptom clusters require replication with larger sample sizes.

Obsessional slowness in activities of daily living and vigorous exercising were common in our OCD patients but not in previous cases (10–16). Slowness in OCD refers to the excessive time spent by patients in initiating and completing a range of daily activities usually because of the implementation of avoidant strategies, mental rituals, repetitive actions, indecisiveness, and perfectionism (28, 52, 53). In fact, our three OCD patients with slowness showed more pervasive OC symptoms than the nonslow group, because the former patient group scored significantly higher in the Leyton Obsessional Inventory (symptoms scale) and Yale-Brown Obsessive Compulsive Scale (compulsion scale) than the latter patient group, which is a pattern of results that replicates previous findings in OCD patients with no evidence of structural brain damage (28). Slowness in OCD is also associated with prominent deficits in frontal lobe tests and abnormally high metabolic rates in orbital frontal cortex, premotor cortex, and midfrontal cortex bilaterally (54). Our three OCD patients with slowness were found to be more impaired than the nonslow subgroup for memory, naming, verbal fluency, and executive functions, and two of them had orbitofrontal damage probably reflecting more severe dysfunction in frontal-subcortical circuits (46).

Compulsive exercising was present in three patients in our sample; one of them also displayed obsessional slowness, which is an unusual symptom cluster that has occasionally been reported in cases of OCD (24, 55), eating disorders (24, 55–57), or catatonia (58, 59). High levels of physical activity and drive to exercise are key features of anorexia nervosa and, less specifically, of OCD (56, 57).

Although there is some phenomenologic overlap between OCD and anorexia nervosa (56, 57), compulsive exercise was unrelated to abnormal eating habits in our three patients as well as in a previous case of posttraumatic OCD (11). Indeed, our patients had OC symptoms unrelated to food, body shape, or weight. Rather, increased physical activity was explained by the patients themselves [including the case cited by Jenike and Brandom (11)] as an effort to recover their physical effectiveness and to neutralize distressing obsessions and related anxious reactions but not in pursuit of an ideal body thinness as occurs in anorexia nervosa. In contrast to individuals with anorexia nervosa, who view their self-imposed vigorous exercising as a sign of extraordinary self-discipline (24), two of our three OCD patients considered their engagement in exercise programs unreasonable and out of their own con-

trol. From a phenomenologic standpoint, exaggerated motor activity in our three OCD patients may be viewed not only as another compulsive symptom (designed to reduce distress), but it may also be attributed to disturbances in drive regulation (designed to obtain gratification) (60). Although the biological basis of physical activity is still poorly understood, the three patients had lesions involving mainly the frontal cortex and caudate nucleus, thus suggesting that dysfunction of frontal-subcortical circuits may have caused increased spontaneous motor activity at the expense of lack of inhibition of internal motor drives (60).

In conclusion, OCD is an important sequela of TBI regardless of the severity of injury. Posttraumatic OCD has a relative specific pattern of symptoms even in patients with mild TBI and is associated with a variety of other psychiatric disorders, particularly non-OCD anxiety. Cognitive deficits affecting mainly memory and executive functions coupled with the topographic distribution of MRI abnormalities in patients with moderate and severe TBI suggest dysfunction of frontal-subcortical circuits.

REFERENCES

1. McAllister TE. Neuropsychiatric sequelae of head injuries. *Psychiatr Clin North Am* 1992;15:395–413.
2. Fedoroff JP, Starkstein SE, Forrester AW, et al. Depression in patients with acute traumatic brain injury. *Am J Psychiatry* 1992;149:918–23.
3. Starkstein SE, Mayberg HS, Berthier ML, et al. Secondary mania: neuroradiological and metabolic findings. *Ann Neurol* 1990;27:652–9.
4. Jorge RE, Robinson RG, Starkstein SE, et al. Secondary mania following traumatic brain injury. *Am J Psychiatry* 1993;150:916–21.
5. Buckley P, Stack JP, Madigan C, et al. Magnetic resonance imaging of schizophrenia-like psychoses associated with cerebral trauma: clinicopathological correlates. *Am J Psychiatry* 1995;150:146–8.
6. Fujii DEM, Ahmed I. Psychosis secondary to traumatic brain injury. *Neuropsychiatry Neuropsychol Behav Neurol* 1996;9:133–8.
7. Oder W, Goldenberg G, Spatt J, et al. Behavioural and psychological sequelae of severe closed head injury and regional cerebral blood flow: a SPECT study. *J Neurol Neurosurg Psychiatry* 1992;55:475–80.
8. Epstein RS, Ursano RJ. Anxiety disorders. In: JM Silver, Yudofsky SC, Hales RE, eds. *Neuropsychiatry of traumatic brain injury*. Washington, DC: American Psychiatric Press;1994:285–311.
9. Bryant RA, Harvey AG. Relationship between acute stress disorder and posttraumatic stress disorder following mild traumatic brain injury. *Am J Psychiatry* 1998;155:625–9.
10. Drummond LM, Gravestock S. Delayed emergence of obsessive-compulsive neurosis following head injury. *Br J Psychiatry* 1988;153:839–42.
11. Jenike MA, Brandom AD. Obsessive-compulsive disorder and head trauma: a rare association. *J Anxiety Disord* 1988;2:353–9.
12. Max JF, Smith WL, Lindgren SD. Case study: obsessive-compulsive disorder after severe traumatic brain injury in an adolescent. *J Am Acad Child Adolesc Psychiatry* 1995;34:45–9.
13. Donovan NJ, Barry JJ. Compulsive symptoms associated with frontal lobe injury. *Am J Psychiatry* 1994;151:618 (letter).
14. Kraus JK, Jankovic J. Tics secondary to craniocerebral trauma. *Mov Disord* 1997;12:776–82.
15. McKeon J, McGuffin P, Robinson P. Obsessive-compulsive neurosis following head injury—a report of four cases. *Br J Psychiatry* 1984;144:190–2.

16. Kant R, Smith-Seemiller L, Duffy JD. Obsessive-compulsive disorder after closed head injury: review of literature and report of four cases. *Brain Inj* 1996;10:55–63.
17. Berthier ML, Kulisevsky J, Gironell A, et al. Obsessive-compulsive disorder associated with brain lesions. Clinical phenomenology, cognitive function, and anatomical correlates. *Neurology* 1996;47:353–61.
18. Berthier ML, Kulisevsky J, Fernández-Benitez JA, et al. Reactivation of posttraumatic stress disorder after minor head injury. *Depress Anxiety* 1998;8:43–7.
19. Teasdale GM, Jennett WB. Assessment of coma and impaired consciousness. *Lancet* 1976; ii:1031–34.
20. Shtasel DL, Gur RE, Mozley D, et al. Volunteers for biomedical research: recruitment and screening of normal controls. *Arch Gen Psychiatry* 1991;48:1022–5.
21. Fahn S. A case of post-traumatic tic syndrome. In: Friedhoff AJ, Chase TH, eds. *Gilles de la Tourette syndrome*. New York: Raven;1982:349–50.
22. Spitzer RL, Williams JBW. *Structured clinical interview for DSM-III-R: patient version (SCID-P)*. New York: New York State Psychiatric Institute, Biometrics Research, 1985.
23. American Psychiatric Association: *Diagnostic and statistical manual of mental disorders*, 3rd ed, revised. Washington, DC: American Psychiatric Association, 1987.
24. American Psychiatric Association, Committee on Nomenclature and Statistics: *Diagnostic and statistical manual of mental disorders*, 4th ed. Washington DC: American Psychiatric Association, 1994.
25. Goodman WK, Price LH, Rasmussen SA, et al. The Yale-Brown Obsessive Compulsive Scale. I. Development, use and reliability. *Arch Gen Psychiatry* 1989;46:1006–11.
26. Goodman WK, Price LH, Rasmussen SA, et al. The Yale-Brown Obsessive Compulsive Scale: Validity. *Arch Gen Psychiatry* 1989; 46:1012–6.
27. Cooper JE. The Leyton Obsessional Inventory. *Psychol Med* 1970; 1:48–64.
28. Hymas N, Lees A, Bolton D, et al. The neurology of obsessional slowness. *Brain* 1991;114:2203–33.
29. Davis C, Kennedy SH, Ravelski E, et al. The role of physical activity in the development and maintenance of eating disorders. *Psychol Med* 1994;24:957–67.
30. Hamilton MA. A rating scale for depression. *J Neurol Neurosurg Psychiatry* 1960;23:56–62.
31. Tyrer P, Owen RT, Cichetti DV. The brief scale for anxiety: a subdivision of the comprehensive psychopathological rating scale. *J Neurol Neurosurg Psychiatry* 1984;47:970–5.
32. Horowitz M, Wilner N, Alvarez W. Impact of the event scale: a measure of subjective stress. *Psychosom Med* 1979;41:209–18.
33. Zilberg NJ, Weiss DS, Horowitz MJ. Impact of Event Scale: a cross validation study and some empirical evidence supporting a conceptual model of stress response syndromes. *J Consul Clin Psychol* 1982;50:407–14.
34. Leininger BE, Gramling SE, Farrell AD, et al. Neuropsychological deficits in symptomatic minor head injured patients after concussion and mild concussion. *J Neurol Neurosurg Psychiatry* 1990;53: 293–6.
35. Otto MW. Normal and abnormal information processing. A neuropsychological perspective on obsessive-compulsive disorder. *Psychiatr Clin North Am* 1992;15:825–48.
36. Wechsler DA. *Wechsler adult intelligence scale*. New York: Psychological Corporation, 1955.
37. Wechsler DA. A standardized memory scale for clinical use. *J Psychol* 1945;19:87–95.
38. Kaplan E, Goodglass H, Weintraub S. *The Boston naming test*. Philadelphia: Lea & Febiger, 1983.
39. Borkowsky JG, Benton AL, Spreen O. Word fluency and brain damage. *Neuropsychologia* 1967;5:135–40.
40. Berg EA. A simple objective test for measuring flexibility in thinking. *J Gen Psychol* 1948;39:15–22.
41. Money J. *A standardized road map test of direction sense*. San Rafael, CA: Academic Therapy Publications, 1976.
42. Boll TJ. The Halstead-Reitan Neuropsychology Battery. In: Filskov SB, Boll TJ, eds. *Handbook of clinical neuropsychology*. New York: Wiley-Intersciences, 1981.
43. Goodman WK, Price LH. Assessment of severity change in obsessive-compulsive disorder. *Psychiatr Clin North Am* 1992;15:861–9.
44. Spreen O, Strauss EA. *Compendium of neuropsychological tests*. New York: Oxford University Press, 1991.
45. Lezak MD. *Neuropsychological assessment*, 3rd ed. New York: Oxford University Press, 1995.
46. Cummings JL. Frontal-subcortical circuits and human behavior. *Arch Neurol* 1993;50:873–80.
47. Fann JR, Katon WJ, Uomoto JM, et al. Psychiatric disorders and functional disability in outpatients with traumatic brain injuries. *Am J Psychiatry* 1995;152:1493–9.
48. Flament MF, Whitaker A, Rapoport JL, et al. Obsessive-compulsive disorder in adolescence: an epidemiological study. *J Am Acad Child Adolesc Psychiatry* 1988;27:764–71.
49. Davidoff DA, Kessler HR, Laibstein DF, et al. Neurobehavioral sequelae of minor head injury: a consideration of post-concussive syndrome versus post-traumatic stress disorder. *Cogn Rehabil* 1993;March/April:8–13.
50. Lucey JV, Costa DC, Adshear G, et al. Brain blood flow in anxiety disorders. OCD, panic disorder with agoraphobia, and post-traumatic stress disorder on 99m TcHMPAO single photon emission tomography (SPET). *Br J Psychiatry* 1997;171:346–50.
51. Leckman JF, Grice DE, Boardman J, et al. Symptoms of obsessive-compulsive disorder. *Am J Psychiatry* 1997;154:911–7.
52. Ratnasuriya RH, Marks IM, Forshaw DM, et al. Obsessional slowness revisited. *Br J Psychiatry* 1991;159:273–4.
53. Veale D. Classification and treatment of obsessional slowness. *Br J Psychiatry* 1993;162:198–203.
54. Sawle G, Hymas N, Lees AJ, et al. Obsessional slowness: functional studies with positron emission tomography. *Brain* 1991;114: 2191–202.
55. Lopez OL, Berthier ML, Becker JT, et al. Creutzfeldt-Jakob disease with features of obsessive-compulsive disorder and anorexia nervosa: the role of cortical-subcortical systems. *Neuropsychiatry Neuropsychol Behav Neurol* 1997;10:120–4.
56. Rothenberg A. Adolescence and eating disorder: the obsessive-compulsive syndrome. *Psychiatr Clin North Am* 1990;13:469–88.
57. Holden N. Is anorexia nervosa an obsessive-compulsive disorder?. *Br J Psychiatry* 1990;157:1–5.
58. Fisher CM. "Catatonia" due to disulfiram toxicity. *Arch Neurol* 1989;46:798–804.
59. Rogers D. Catatonia: a contemporary approach. *J Neuropsychiatry Clin Neurosci* 1991;3:334–40.
60. Depue RA, Collins PF. Neurobiology of the structure of personality: dopamine, facilitation of incentive motivation, and extraversion. *Behav Brain Sci* 1999;22:491–569.