SUMMARY

Background. Impairments of neurocognitive performance in female patients with anorexia nervosa have been reported in many studies. To our knowledge neuropsychological studies of male eating disorder patients remain rare. The goals of our study were to present the profile of neurocognitive functioning in a male adolescent anorexia nervosa patient and, given that the clinical manifestation of both eating disorders and obsessive-compulsive disorder presents strong psychopathological similarities, to determine how neurocognitive performance could be associated with the patient's obsessive-compulsive disorder symptoms.

Material and methods. A male adolescent anorexia nervosa patient who satisfied DSM-IV diagnostic criteria participated in the study. A battery of neuropsychological assessment methods was used, including the Wechsler Adult Intelligence Scale-Revised, Auditory Verbal Learning Test, subtest for long-term memory of the Choynowski Memory Scale, the Rey-Osterrieth Complex Figure Test, the Benton Visual Retention Test, the Diagnosis of Brain Damage battery, the Trail Making Test, parts A and B. The Yale-Brown Obsessive-Compulsive Scale was also used. Longitudinal neurocognitive data are presented from an inpatient neuropsychological assessment conducted at ages 13 and 18.
Results. Several neuropsychological deficits were observed in our patient in both verbal and nonverbal domains.

Conclusions. The results of this study indicate that obsessive-compulsive disorder symptomatology was very closely related to the patient's neurocognitive performance. The patient's rigid, inflexible and perfectionist behavior patterns interfered with his neuropsychological functioning.

INTRODUCTION

Anorexia nervosa (AN), which is regarded as the most chronic and life-threatening psychiatric disorder, with an increasing incidence and prevalence among the adolescent population, occurs in males in approximately 5 to 10% of the cases seen in eating disorders units (Sharp et al. 1994, Striegel-Moore et al. 1997). The characteristic behaviors associated with both restricting and binge eating/purging AN subtypes are not indifferent to the patient’s biological, psychological and social condition and functioning.

Impairments of neurocognitive performance in adolescent patients with AN have been reported in many studies. Relative to healthy unaffected controls, individuals with AN present several deficits in various neuropsychological domains, including verbal and nonverbal memory, verbal and nonverbal learning, attention, visuospatial ability, psychomotor speed, and executive functioning (Thompson 1993, Mathias & Kent 1998, Tchanturia et al. 2001, Koba et al. 2002, Moser et al. 2003, Murphy et al. 2004, Ohrrmann et al. 2004, Steinglass et al. 2006). To date, preliminary evidence indicates that there may be some degree of improvement in cognition with weight recovery (Katzman et al. 2001, Kerem & Katzman 2003, Moser et al. 2003). Contrary to this statement, other recent evidence has shown that not all of these neuropsychological deficits show improvement with weight restoration (Tchanturia et al. 2002, Tchanturia et al. 2004). Impairments of neurocognitive performance in female patients with AN have been reported in many studies (Tchanturia et al. 2005). Nevertheless, neuropsychological case studies of eating disorders (ED) in male patients remain rare.

The present study describes the case of an 18-year-old adolescent male (referred to hereinafter by the initial P), with childhood-onset AN (restricting type) and obsessive-compulsive disorder (OCD), who was referred to the Child and Adolescent Unit of the Department of Developmental Psychiatry of the Medical University of Gdańsk at the age of 13, and consecutively at the age of 18 for treatment due to longstanding psychopathological symptomatology. His personal background is as follows: natural delivery, normal psychomotor development, acute bacterial food-borne intoxication at the age of 2. Despite high academic achievement, inadequate social adaptation was observed. P was perceived as an anxious, inhibited, child, restrained in emotional expression and initiative, harm avoidant, overcontrolled and compliant. At the same time, he displayed high levels of school-related anxiety, including refusal to go to school.
At age 11, P presented with severe obsessive-compulsive symptomatology (excessive and ritualized bathing, cleaning household, need for exactness and order) and selective mutism. Subsequent to unsuccessful outpatient treatment at the age of 12, P was hospitalized, and his psychopathological symptoms diminished progressively after 3 months of inpatient treatment (both psychotherapy and pharmacotherapy were indicated). However, perfectionism and interpersonal difficulties persisted.

At the same time, P severely restricted his daily caloric intake, eliminated fatty food from his diet and concentrated on healthy food. He also began to participate in excessive physical exercise, but did not exhibit purgative behavior. At the age of 13, his weight was 24.900 kg, his height was 1.42m (body mass index [BMI] = 12.34). After unsuccessful outpatient treatment, he was hospitalized for 2 weeks at the Pediatric Unit in order to stabilize his somatic condition, and afterwards transferred to the Child and Adolescent Unit of the Department of Developmental Psychiatry. He was then diagnosed, according to DSM-IV, as having AN (restricting type). Moreover, during that time obsessive-compulsive symptomatology worsened, and the diagnosis of OCD was made, according to DSM-IV guidelines. Additionally, the patient showed symptoms of major depression.

The psychopathological tests included the Children's Yale-Brown Obsessive-Compulsive Scale (CY-BOCS; obsession score was 12 and compulsion score was 12; severe), the Children's Depression Rating Scale-Revised (CDRS-R score was 44; severe) and the Hamilton Depression Rating Scale (HAMD score was 29; severe). The formation of abnormal personality from Cluster C (according to DSM-IV) was also observed. Computerized tomography (CT) showed no significant abnormalities; however, electroencephalography results (EEG) gave indications for pathological patterns of the cerebral cortical activity (theta rhythms in the temporal lobes). After 5 months of psychotherapy and pharmacologic treatment the patient reached the weight of 35.800 kg (BMI = 17.75). Unpredictably, his mother terminated treatment contrary to medical indications.

5 years later, at the age of 18, P was once more referred to our clinic for treatment due to a chronic, life-threatening psychosomatic condition. At the time of referral his weight was 24.500 kg, his height was 1.48 m (BMI = 11.18). Additionally, at his age the minimal ideal height should reach 1.75m and weight 63.000kg (BMI = 20.57kg/m2). Tanner's Scale indicated the first stage of male sexual development (prepubertal stage). Premorbid anancastic personality traits, OCD traits and maturity fears were still present. The Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) scores were 12 and 9 for obsession and compulsion, respectively (moderate), the Beck Depression Inventory (BDI) score was 19 (mild) and HAMD score was 24 (moderate). EEG results showed no significant abnormalities. In the course of 3 months of inpatient treatment (individual and family therapy, pharmacotherapy were indicated) progressive reduction of both ED and psychiatric comorbidity.
symptomatology was observed. The patient's mother terminated treatment unexpectedly, in contradiction of the medical indications. His weight was then 37.500 kg (BMI = 17.12).

Nine months after discharge the patient's death was reported. His sudden decease was attributed to urinary system infection and sepsis.

**NEUROPSYCHOLOGICAL PROFILE**

A battery of formal neuropsychological assessment methods was used (including the Wechsler Adult Intelligence Scale-Revised, the Auditory Verbal Learning Test, the subtest for long-term memory of the Choynowski Memory Scale, the Rey-Osterrieth Complex Figure Test, the Benton Visual Retention Test, the Diagnosis of Brain Damage, the Trail-Making Test, parts A and B) in order to evaluate patient's neurocognitive functioning. Furthermore, taking into consideration that clinical manifestation of both ED and OCD present strong psychopathological similarities, the authors attempted to determine how neurocognitive performance could be associated with the individual's obsessive-compulsive disorder symptoms. Longitudinal neurocognitive data are presented from inpatient neuropsychological assessment conducted at ages 13 and 18.

P scored 144 and 131 on the Verbal Scale; 89 and 77 on the Nonverbal Scale; 119 and 108 on the Full Scale of the Revised Wechsler Intelligence Scale for Children (WISC-R) and the Revised Wechsler Adult Intelligence Scale (WAIS-R), respectively. Progressive deterioration in the IQ scores was observed during the 6-year follow-up. Furthermore, an abnormal pattern of performance on both the WISC-R and the WAIS-R was detected. The patient's psychomotor speed was considerably reduced during the initial and follow-up examination. Throughout each test administration it took 10 hours to evaluate the patient's IQ. Notably, on the subtests with time restriction (Nonverbal Scale) P managed to adjust to the test instructions and time limits, which tended to be consistent with his endurance and good organization. Nevertheless, on the subtests without time restriction (Verbal Scale), P responded with sentences to each of the inquiries, scrupulously selecting expressions and meticulously detailing each answer. Moreover, he returned to the beginning of each sentence, verifying its formal structure and content, which was generally consistent with his personality characteristic of perfectionism, fear of anticipated failure or negative outcome, and general tendency to overachievement.

P displayed several neuropsychological deficits in both verbal and nonverbal domains. It is essential to indicate that specific dysfunctions in the patient's cognitive performance were observed. He displayed high error rates, particularly confabulation errors (Auditory Verbal Learning Test, AVLT; Choynowski's Memory Scale) and perseveration errors (AVLT), as well as rotation errors (Diagnosis of Brain Damages, DBD; Benton Visual Retention
Test, BVRT) and distortion errors (BVRT). Moreover, our results suggest that complex visuospatial construction and organization abilities measured by the Rey-Osterrieth Copy Figure Test (CFT) were impaired. The Gestalt approach, which tends to be the most mature and perceptually well-organized method of strategic performance on the CFT, characterized only the patient's initial performance. At follow-up the level of organizational strategy had deteriorated, and the spatial piecemeal approach, an immature strategy characteristic of younger children, was detected. Additionally, the patient's complex visuospatial ability of working memory, as measured by the Trail Making Test, part B, was also impaired. Impaired psychomotor functioning was likewise observed on the CFT. It took him 30 min and 24 min during the initial examination, 25 min and 30.5 min during follow-up to perform on the copy trial and delayed recall trial, respectively.

The results of this neuropsychological follow-up indicate that OCD symptomatology was strictly related to the patient's neurocognitive performance. The patient's rigid, inflexible and perfectionistic nature, his fear of not saying things right, his need to know and remember, and exaggerated exactness considerably interfered with his neuropsychological functioning.

**DISCUSSION**

A holistic and longitudinal perspective on male AN cases is still rare. In the case described here, the coexistence of AN and OCD is of special interest. The sudden onset of OCD symptoms took place before the clinical manifestation of ED. When AN begun, OCD started again and an increase in the severity of symptoms was observed. Our case report supports a number of previous studies that have reported the co-occurrence of both pathologies and proposed the hypothesis that ED could be a phenomenological variant of OCD (Bellodi et al. 2001). It is also consistent with the assumptions made by some studies (Milos et al. 2002) that the coexistence of both pathologies accounts for the severity of clinical manifestation and its longer duration. Moreover, our data confirm, consistent with previous findings (Cavedini et al. 2004, Murphy et al. 2002, Tchanturia et al. 2004), that there is significant linkage between neurocognition and OCD traits. Our patient's neuropsychological functioning was strictly related to the OCD symptomatology. The impairments described, in terms of flexibility vs rigidity, perseverativeness, preoccupation with details, high levels of perfectionism and obsesssionality, were found to play a significant role as both vulnerable and precipitating factors and led to fatal outcome in the course of AN treatment.

The conclusions that could be drawn from the present study provide further evidence for a link between AN and OCD, in both the psychological and neurocognitive domains. Our preliminary findings show that OCD predated the onset of AN, indicating that OCD could be a risk factor for the development and the maintenance of AN, and could also account for the poorer therapeutic
outcome. Therefore, in the authors' opinion, it would be important to continue research in both the male and female child and adolescent population, in order to identify the underlying mechanisms in the etiology and the maintenance of both ED and OCD. Further findings would definitely contribute to the development of effective preventive and treatment interventions.

REFERENCES


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