INTRODUCTION

Anorexia nervosa is a rare condition and epidemiological studies show that it is more prevalent in women (female to male ratio is 10:1) (Sadock, 2003). Factors accounting for its female dominance are: 1. Female physical development starts before emotional development due to the early onset of puberty which leads to disruption in body image; 2. Women are more affected than men by the socio-cultural pressure stressing the importance of being thin and beautiful; 3. Male anorexics hide their illness as they see the condition as a "female disorder" or preferring sports instead of dieting (Muise et al., 2003).

The comorbidity of anorexia nervosa with other psychiatric disorders, especially with depression and anxiety disorders is quite high (Wildman et al., 2003; Kaye et al., 2004). Syndrome metamorphosis which is defined by some authors as the display of various psychopathological symptomatologies during the course of a disorder (Böning and Kachel, 1990), is frequent in anorexia nervosa. However, comorbidity of anorexia nervosa and schizophrenia is relatively rare. Studies show that the prevalence of comorbid schizophrenia in anorexic patients is between 0% and 12% (Hsu et al., 1981; Herzog et al., 1988; Ferguson and Damluji, 1988). There are various theories about the comorbidity of anorexia nervosa-schizophrenia:

1. Eating disorders and psychoses interact with each other, but are separate disorders (Hsu et al., 1981; Ferguson and Damluji, 1988; Deckelman et al., 1996).

2. Transient psychosis can develop based on the feeling better after gaining some weight (Grounds, 1982; Hugo and Lacey, 1997).

3. Over involvement with food and eating can be a
defense against psychosis (David et al., 1986). In fact, Hugo and Lacey (1997) proposed that there is a reverse relationship between the two disorders after observing that remission in eating disorders triggers or exacerbates psychotic symptoms. Additional support for this explanation came from Ferguson and Damluji (1988) who concluded that eating disorders provide a feeling of identity to schizophrenia patients whose ego borders have vanished and helps them to organize their lives.

4. These two disorders are in fact different phenotypes of the same illness process and distorted thoughts about to eating and body image can be related to the cognitive disorder that is the result of schizophrenia (Small et al., 1981; Yamashita et al., 1999). For example, intense mental preoccupations about eating may be considered as overvalued ideas which are important components of psychotic process. (Ferguson and Damluji, 1988).

5. Anorexia nervosa and schizophrenia exist on a continuum. Schizophrenia is at one end of this continuum and neuroses or personality disorders are at the opposite end (Lyon and Silber, 1989).

In this paper a young male patient with comorbid anorexia nervosa and schizophrenia is presented. In addition, anorexia in males and comorbidity of anorexia and schizophrenia will also be discussed.

Case

HA is an 18-year-old senior student in a high school in which students are selected by an entrance examination. Nearly one month ago his teachers observed some changes in his speech and behavior, and warned his family. After a short while, HA's family also observed these changes. For example, he kept the curtains closed during the day in order to prevent anyone from seeing the inside of the house, and from time to time he told his mother that he was suspicious of a hidden camera in the house. He frequently started talking about absurd scientific projects, but as his family had considered him a genius since childhood they did not pay attention to his new projects. He could not sleep well and one night he waited on the balcony until morning to answer a neighbor who he thought was sending him messages by opening and closing curtains. He believed that the conversations on television were directed to him and he replied to these conversations. He was hostile and impatient towards his parents, especially his mother, whose behaviors he thought were sexually driven, and he did not want her to come close or to touch him. He rejected the notion of having mental problems and was brought to our clinic by his parents. In the assessment, his speech was poor in content although the amount has increased. He frequently gave irrelevant answers to questions and had perseverations. His thought content included delusions of reference and persecution vulnerability. Although he denied having delusions, his mother reported seeing him occasionally talking and laughing to himself.

HA said that he had never used any drugs, including nicotine. His family also did not have any suspicions about drugs use. His laboratory findings, EEG, and cranial MRI screening, which were conducted in order to detect an underlying physical illness, were normal. He was diagnosed with schizophrenia and began taking olanzapine 20mg/day. Approximately 10 days later, his psychotic symptoms disappeared. However, 2 weeks after this dramatic recovery his symptoms reappeared, despite the continued use of medication, and he was hospitalized. In the hospital, 800-mg/day amisulpride treatment was initiated and his symptoms were controlled.

HA did not want to use his medication, but this was not only due to his denial of his condition; olanzapine increased his appetite and he had gained 7 kilos in one month. As he had had a fear of getting fat since he was 14 years old, this situation was unacceptable to him. He had been an overweight child until he was 14. Due to this, his family criticized him and his friends bullied him. At the age of 14, he fell in love and decided to loose weight. In order to reach his target he restricted his food intake. In addition, he rode his bike for many hours everyday and started intense exercising, such as bodybuilding. With these efforts, he lost 10 kilos in 2 months. He had prepared himself a very detailed diet list by using calorie values of foods he obtained from the Internet. For example, in the morning he would have a 1200-calorie breakfast with the necessary vitamins and ate nothing else during the rest of the day. When he ate even a very small amount he felt very guilty. He decided that his body mass index (BMI) should be 19%-19.5%, which is normal. He carried on his diet and exercise regime for 4 years until his admission to our clinic. Two months before his admission, he gained weight as he got taller and his preoccupation with his weight and body increased and he started to follow a very restricted diet. He believed that his thighs were very thick and one day he tried to understand whether this thickness was due to muscle tissue or fat tissue by pricking himself with a needle. He wanted to see whether it would bleed or not as he thought that there were fewer veins in fat tissue than in muscle and that there would be less bleeding if his thick thighs were because of fat. He lost 11 kilos in...
2 months, but he regained much of it due to olanzapine treatment. He felt bloated and wanted to get rid of what he considered the ugly mass around his legs and waist. He was very disturbed by the increase of skin in the joints of his fingers. His BMI was in the normal range at the time of hospitalization.

Both HA's mother and father were university graduates with respectable careers, which provided them with a good income. His brother, who is 6 years older, went abroad to continue his education after successfully completing university. Among the family, his brother was always presented as a role model and his parents frequently compared them. HA believed that his parents would only love him when he became the most successful child. In fact, his school performance had always been top level, although he did not study very much. Approximately 1 year ago, his teachers warned his family about his social withdrawal. In fact, his family was also aware that he spent most of his time in front of the computer or doing sports; however, they observed this change with appreciation of his self-discipline. His family history includes an uncle with schizophrenia who is still under olanzapine treatment.

During his discharge and first follow-up, positive symptoms of schizophrenia were not evident; however, negative symptoms, such as anhedonia and social withdrawal, were still evident. As he could not tolerate the extrapyramidal side effects of amisulpride, quatiapine 750 mg/day was initiated and this treatment continues, as of the time of this writing, with success. Because he was in a psychotic state at the time of his hospitalization, no treatments regarding his eating disorder were initiated.

After the recovery of his psychosis, the treatment team decided to follow him without administering any interventions, as he was not an appropriate candidate for psychotherapy and his weight was close to normal. After his discharge from the hospital, he went back to school; however, there was a marked decline in his school performance, which improved within 2 months. Although he could not study as much as he wanted to due to his concentration difficulties, he achieved a very high score on his university exams and gained entrance to the school of his choice. At university, HA did not have the same academic success he did in high school. He had virtually no relationships with other people, including his family. He spent most of his time with computers and did not go to class. His family was aware that he had absurd and childish relationships that were not appropriate for a person with his cognitive ability. As soon as his positive psychotic symptoms disappeared he went back on his diet and exercise regime. In conclusion, he had lost all the weight he gained due to olanzapine treatment. He still continues his diet and exercise regime, which keeps his BMI below normal.

DISCUSSION

To the best of our knowledge, there is only one previous case study of a comorbid anorexia-schizophrenia diagnosis (Cheung and Wilder-Smith, 1995). Therefore, our case is the second of its kind in the literature; however, we think that in order to evaluate this case as a comorbid anorexia-schizophrenia case, there are certain points, which need to be reflected on that are related to the validity of both diagnoses.

The first question is whether the patient was anorexic or not. HA's eating behavior during the 4 years before his hospitalization was very similar to anorexic patients. In fact, this is not a surprising similarity. Research shows that male and female anorexics are similar in terms of onset of illness, premorbid personality characteristics, and illness characteristics (Eliot and Baker, 2001). HA's eating disorder, in the form of restricting food intake, skipping meals, and preferring low calorie food, resulted in marked weight loss. In addition to changes in his eating behavior, HA also exercised intensely. This weight loss method is very common among anorexics. In addition to these, the intense mental effort expended on body image and food that is observed in anorexics was evident in our patient's behavior, as evidenced by the needle experiment he did and his use of a detailed calorie scale. Although we have insufficient information about his BMI during the initial period of his food restriction, we know that he had reached his target of having an ideal thin body. Yet, his weight loss alone was not sufficient enough to diagnose anorexia nervosa at the time of his admission to our clinic. However, his involvement with his body and weight were serious and intense, at least as much as an anorexic, and his past history and family dynamics very much resembled the style seen in anorexic patients. According to the DSM-IV classification, this case is diagnosed as, eating disorder, not otherwise specified, with anorexic characteristics (American Psychiatric Association, 1994). In other words, this case does not fully satisfy the diagnostic criteria for anorexia nervosa; however, the following question remains: Loosing a few kilos more he can easily go beyond that threshold, but is this supposed to add more to his psychopathology? The answer is probably no, since subthreshold cases are reported to be as serious as full-blown syndromes in many
ways (Crow et al., 2002; Watson and Andersen, 2003). Based on this, we concluded that HA showed an anorexic course in the four-year period before the development of psychosis. Although his over involvement related to eating decreased a little during and right after the psychosis, it never disappeared and eventually returned to the same pattern. As Deckelman (1996) also stated, although the negative symptoms and insufficient target-related behaviors decreased the severity and duration of eating behavior for a period of time, these behaviors were still evident in the presented case example.

The second question is whether this was a schizophrenia case or not. First of all, as it is the first time HA exhibited psychosis, an organic etiology was assessed in his differential diagnosis. In addition to the physical and neurological findings of the patient being normal, the normal laboratory and screening assessments excluded a possible general medical condition that might lead to psychosis. Another factor that might lead to psychosis, substance abuse, was also excluded based on his history; however, no additional laboratory assessments related to substance use were performed, as the family’s statements were reassuring. This may be a limitation. In addition, although there are some speculative explanations, there is no information about the nature of the comorbidity of these 2 disorders. In conclusion, further studies are needed in order to understand the complex interaction between psychosis and eating disorders.

REFERENCES


