Stunted Growth and Hypothalamic-Pituitary Dysfunction before and after Eating Disorders in Children and Adolescents

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Abstract. The increasing frequency and the younger onset of eating disorder is becoming a social problem in Japan. The diagnostic criteria of anorexia nervosa according to DSM IV include weight loss and amenorrhea, however, it does not include “no or low weight gain” and “stunted growth” in growing children and adolescents. In this study, we analyzed the growth and endocrine function in 45 patients (39 females and 6 males) with anorexia nervosa. The age at the onset of disease (the onset of weight loss) varied from 10 to 16 yr old. When plotting the changes of height and weight in each individual on the growth chart for Japanese children, 8 out of 45 showed decreased or no weight gain before the apparent onset of weight loss, for more than two years. The stunted growth was associated with decreased weight gain. Endocrine function was tested in 36 patients. Approximately half of the children and adolescents showed exaggerated growth hormone response to provocative tests, while the rest showed low growth hormone levels. Gonadotrophins and thyrotropin responses to LHRH and TSH stimulation were decreased in most of the cases tested. About 80% of the patients recovered from the critical weight loss, but the recovery of height is not always successful. In conclusion, eating disorders with severe weight loss in growing children sometimes result in irreversible growth failure. Plotting the growth chart is helpful for finding the “true” onset of eating disorder.

Key words: anorexia nervosa, eating disorders, growth retardation, growth hormone deficiency, familial discordance

Introduction

Anorexia Nervosa (AN) is defined as a condition of inadequate weight loss with disordered eating behavior followed by sustained emaciation lasting for more than three months. The diagnostic criteria of anorexia nervosa according to DSM IV include weight loss (weight 15% below minimal normal body weight for height and age) and amenorrhea, however, it does not include “no or low weight gain” and “stunted growth” in growing children and adolescents. About a decade ago, AN was a disease known to occur among young adult women. The increasing frequency and the younger onset of eating disorder is becoming a social problem in Japan. A prolonged condition of emaciation with refusal of adequate food intake and paradoxical hyperactivity in growing children may cause profound and
irreversible damage to their body functions.

In this study, we investigated the growth and endocrinological function before and after treatment of AN in children and adolescents.

**Subjects and Methods**

Forty-five children and adolescents with AN were studied. For the data collection and analysis, informed consent was obtained from their parents. The patients consisted of 6 males with the onset age of apparent weight loss ranged between 11–15 yr old and 39 females with the onset age ranged between 9–15 yr old. AN was diagnosed by the definition of DSM-IV criteria.

The growth data were obtained from mother-child health records and health check-up records at school. Height and weight during infancy were measured by local health offices at regular health check-ups of 1, 3, 6-7 mo, and 1.5 and 3 yr of age, and were usually measured every four months at school during childhood and adolescence.

Growth retardation or stunted growth is defined as a pathological decrease of weight and/or height gain for certain periods of time (> one year prior to the apparent and acute weight loss which is recognized as the onset of AN).

Growth retardation or stunted growth was searched for on the growth chart of each patient.

Endocrinological data were obtained before and after treatment for AN.

In patients with severe emaciation, the data was obtained after the general condition had recovered and the body weight exceeded at least −40% of the ideal body weight.

**Results**

Growth disturbances prior to the onset of apparent AN were observed among eight out of 45 subjects (1 male, 7 females). The duration of stunted growth was 49.5 ± 14.23 mo (mean ± SD, range 27–70 mo). A representative case with stunted growth is shown in Fig. 1.

Height SDS at the onset of apparent AN (acute and apparent weight loss) was −1.27 ± 0.07 SDS (range −2.2–2.0 SD). ∆height SDS between target height SDS and present height SDS (after treatment, % overweight >−25%) was −0.65 ± 0.17 SDS.

All girls who had menarche showed amenorrhea (20/20). Only two out of twenty recovered from amenorrhea spontaneously after weight gain. The other girls who did not have menarche showed pubertal delay.

Boys showed small (shrunken testes of 5–8 ml with undetectable serum testosterone levels.

Growth hormone (GH) secretion was determined by the GHRH loading test and/or blood sampling every 20 min for 3 h during sleep. Thirty-six patients were tested; 20 showed low response to GHRH and/or low endogenous GH secretion. Some showed recovery of GH secretion after treatment.

The girls who recovered in weight to above the weight at the beginning of the acute and apparent weight loss showed recovery of growth (Fig. 2).

**Discussion**

The principal features of AN are 1) loss of weight 2) body image distortion, 3) menstrual irregularity, 4) out-of-control eating, 5) Intense fear of gaining weight, 6) preoccupation with weight, and 7) denial of current low weight and its adverse impact on health. Those at risk for anorexia are known to have the following risk factors: family history of eating disorder or obesity; a first-degree relative with an affective illness or alcoholism; and participation in ballet, gymnastics, modeling, or other “visual sports”.

The major screening rule for AN is to use simple screening questions when symptoms of disordered eating are recognized and a careful diagnostic interview to establish the presence or absence of formal criteria for eating disorders including the principal features mentioned above and menstrual irregularity (1, 2). Recently, the
onset age of AN is becoming younger and the patients of preadolescent and early adolescent ages are increasing. Our study revealed that the true onset of AN was earlier than is usually noticed, or a pre-AN state proceeds for years before the onset of AN in this age group of patients. The simple analysis of growth charts seems to be the best way to find these pre-AN state patients.

The background of each patient with a pre-AN state varied, however, most of the patients had discordance in character between family members either including or not including the patients themselves. The degree of discordance ranged from relatively mild in which cases the individuals are too sensitive to accept it, to extreme, which was not be able to repair by any procedure. Early intervention is necessary, because eating disorders are serious and complex. Early intervention may lead to prevention and treatment of AN, and the methods of intervention should be individualized according to the patient’s background.

Cultural context is also a major factor promoting the onset and severity of AN, with known features such as obsession with appearance, shame about one’s body, and low self-esteem. In young age groups, low self-esteem is especially important with various background risk factors.

Recovery of growth was observed only in some patients with childhood/adolescent onset AN. The recovery of hormonal disruption takes time, and the age-dependent gain of peak bone mass and maturation and reconstruction of the neural network of the brain would be missed in those patients with delayed recovery (3, 4).

In conclusion, eating disorders with severe weight loss in growing children sometimes result
in irreversible growth failure. Plotting the growth chart is helpful for finding the true onset of eating disorder.

References