Disturbances of sex hormones in anorexia nervosa in the male

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Summary

Sex hormone levels were measured in a male patient with anorexia nervosa throughout the course of his illness and recovery. Gonadotrophin levels returned to normal with weight gain but his testosterone remained low. Possible explanations for these findings are discussed.

Introduction

It is accepted that anorexia nervosa occurs in males (Beaumont, Beardwood and Russell, 1972; Crisp and Toms, 1972). Three diagnostic criteria have been suggested for this disorder (Beaumont *et al.*, 1972): (i) behaviour to induce weight loss, including dieting, self-induced vomiting and purgation, and strenuous exercise, all often carried out secretly; (ii) marked fear of gaining weight, pre-occupation with body size, and disturbance of body image; (iii) an endocrine dysfunction analogous to that found in females with this condition.

There have been few reports of the nature of the endocrine dysfunction in males with anorexia nervosa, and of the changes that occur with weight gain. Low testosterone and gonadotrophin levels appear to be a universal feature during the acute phase of the disorder (Bell *et al.*, 1966; Beaumont *et al.*, 1972; Garfinkel *et al.*, 1975; Davidson, 1976; Hasan and Tibbetts, 1977) and serial estimations during recovery have suggested that these abnormalities do not return to normal (Bell *et al.*, 1966; Beaumont *et al.*, 1972; Garfinkel *et al.*, 1975). However, the hormonal investigations in such studies have not been continued for more than a few weeks after recovery.

In the patient described here, sex hormone levels were recorded during the course of his illness and for several months after recovery. The findings have implications for our understanding of the nature of the hormonal disturbance in this condition.

Case history

A deaf, 27-year-old, married, illustrator presented with a 2-year history of weight loss, from 76 to 48 kg

(height 180 cm). Over the previous year he had black-outs which appear to have been caused by hypokalaemia (serum $K^+ = 2.28$ mmol/l on admission). He had otherwise been well, and denied any psychiatric symptoms. Initially he was admitted to a medical ward for investigation of his electrolyte disturbance, which proved to be due to secret vomiting following meals.

He had always been a fussy eater. His dieting had started 2 years previously after his father had myocardial infarction and was advised to lose weight. Within one year the patient's weight fell from 76 to 64 kg. However, he then continued to lose weight despite eating normal meals. It seems his vomiting had started at this time.

On psychiatric admission, when weighing 48 kg, he was started on a behavioural treatment programme, similar to that used with females with anorexia nervosa (Eckert *et al.*, 1979), in which weight gain was rewarded by an increase in activities and privileges. His progress was very rapid and after 3 weeks he weighed 67 kg. He maintained this weight over the follow-up period of 9 months.

His serum testosterone and gonadotrophin levels during and after his illness are shown in Fig. 1. Initially, his serum gonadotrophins were low, but increased to normal with weight gain. However, his serum testosterone, which initially was towards the lower end of the normal range, did not increase (apart from an initial rise) in response to the increase in gonadotrophins. Nine months after recovery his testosterone level remained abnormally low. During the disorder he denied loss of sexual interest, but his wife refused intercourse because of his emaciated appearance. However, he showed no evidence of sexual dysfunction when the sexual relationship resumed following stabilization of his weight gain and his wife soon became pregnant.

Discussion

This patient's low gonadotrophin and testosterone levels during the acute phase of the illness, and the return of his gonadotrophin levels to normal in

FIG. 1. Testosterone and gonadotrophin levels during course of treatment.

parallel with weight gain, are in keeping with previous reports (Bell *et al.*, 1966; Beaumont *et al.*, 1972; Garfinkel *et al.*, 1975). These findings suggest that the disturbances in sex hormones are, in part at least, due to malnutrition (Ismail and Harkness, 1966), rather than being a primary disturbance, as in females with anorexia nervosa, where the hormonal disturbance is often not reversed for months or even years after weight gain (Crisp *et al.*, 1973).

However, the patient's serum testosterone levels failed to return to normal with weight gain and remained low several months after his weight had stabilized. There are several possible explanations for this. First, primary gonadal dysfunction might be part of the disorder. This could be due to long-standing hypogonadism or result from the illness. Long-term serial sex hormone estimations in males who have recovered from anorexia nervosa would clarify whether or not this is a permanent dysfunction.

Secondly, the persisting low testosterone levels could be explained by changes in sex hormone-binding due to the illness such that overall serum testosterone levels, as measured in this subject, remained low despite an increase in free serum testosterone concentration. This would be in keeping with the apparent return of normal sexual function.

Finally, since the patient's weight did not return to the pre-morbid level of 76 kg but stabilized at 67 kg he could have remained relatively undernourished which may have continued to affect his gonadal function. This possibility could be explored further by hormonal investigation of subjects whose weights have returned to premorbid levels.

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