Addison's disease presenting as anorexia nervosa in a young man

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Introduction

Psychological changes are common in Addison's disease and frank psychosis is well recognized. Anorexia nervosa, predominantly a disease of young women, has never been reported in association with Addison's disease. We report a case of anorexia nervosa, apparently precipitated by acute adrenal insufficiency, in a young man with a long history of minor psychological disturbances and its complete remission following corticosteroid replacement for Addison's disease.

Case report

A 20 year old vegan was admitted to hospital following a visit from his general practitioner at the request of his parents. They were concerned about their son's recent weight loss, his depressed and apathetic mood, loss of appetite and intermittent vomiting. He had lost approximately 18 kg in weight in the previous 6 months and had refused to seek medical help. He had strictly adhered to a vegetarian diet for the past two years and had numerous food fads since the age of 10 years. He was seen by a psychologist at that time because of a refusal to take food, at which stage he was showing 'minor psychological disturbances including abnormal obsessional traits'.

He admitted to recent depression with insomnia and lack of concentration which he attributed to social isolation because of chronic unemployment. Sexual interest and potency had declined and he insisted that he was taking adequate nutrition, denying that he was underweight.

On examination, he was malnourished and dehydrated, weighing 40 kg (body mass index 14.3). He was not pigmented and blood pressure was 90/60 mmHg. Serum sodium was 126 mmol/l, potassium 3.8 mmol/l, urea 6.4 mmol/l and glucose 4.7 mmol/l.

While in hospital he refused all food, only taking water. He had episodic vomiting which was frequently witnessed as self-induced and the vomit was sometimes concealed. On formal psychiatric assessment he was considered to have classical anorexia nervosa with a typical premorbid personality. He refused further investigation and treatment and took his own discharge against medical advice. One month later, he was readmitted severely dehydrated and cachectic and now weighing only 38.5 kg. Serum sodium had fallen to 114 mmol/l, potassium was 5.0 mmol/l, urea 31.0 mmol/l and glucose 5.4 mmol/l. Other investigations were as follows: 09.00 h cortisol 124 nmol/l (normal 130–690 nmol/l) with no response to 1 mg of tetracosactrin, ACTH 1687 ng/l (normal 10–80); adrenal antibodies, Mantoux test 1:1000, sputum, urine and bone marrow aspirate for acid-fast bacilli all negative, chest and abdominal X-rays normal.

He was commenced on hydrocortisone 20 mg in the morning and 10 mg in the afternoon with

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fludrocortisone 100 μg and he made a spectacular recovery, eating normally and gaining 12 kg in weight in the following two months. His mental state, likewise, returned to normal, showing interest in his treatment and securing employment within a short period of time.

Three years later, he remains entirely well. His obsessive personality persists but his eating behaviour is normal. He accepts his present weight of approximately 64 kg as his ideal body weight.

Discussion

Although in his original description of the disease in 1855 Addison referred to the frequency of psychological abnormalities in his 11 patients, it was not until 1942 that the occurrence of abnormal mental states was first clearly described. In 1953, the first definitive comparative psychiatric appraisal of the mental state of patients with Addison's disease was published in which three categories of psychiatric disturbance were described. The first was an acute organic reaction characterized by delirium and recognized as a feature of acute or terminal adrenal insufficiency. The second and third, chronic mood abnormalities and psychoses, were found only in chronic adrenal insufficiency. It is now known that psychological changes are found in almost all patients with severe Addison's disease and frank psychosis is well recognized. Many cases of acute psychotic reactions, usually of a paranoid schizophrenic nature, or severe conversion disorders, usually responding to replacement corticosteroid therapy, have been reported. More recently, chronic self-mutilation in a 14 year old girl that regressed completely following treatment for previously unsuspected Addison's disease has been described.

In man, raised circulating β-endorphins occur in association with raised ACTH levels suggesting a possible explanation for the observed psychological changes since β-endorphin administration is known to produce a neuroleptic-like syndrome in rats.

Almost invariably anorexia nervosa is associated with hypercortisolism due to reduced metabolic clearance of cortisol, and basal plasma ACTH is normal. Nevertheless we cannot entirely discount the possibility that adrenal failure was a consequence of chronic, severe malnutrition in our patient. However, the primacy of Addison's disease as the basis for anorexia nervosa is suggested by the rapid improvement in mental state following corticosteroid administration. Although spontaneous remission in anorexia nervosa is sometimes prompted by a near-death experience, we feel that this sequence of events is most unlikely in our patient because of his sustained well-being 3 years later. It is likely that the onset of Addison's disease precipitated the psychological and somatic manifestations of anorexia nervosa. Although vomiting and weight loss frequently occur in untreated chronic adrenal insufficiency, and indeed our patient's cachectic state may have been partly attributable to this, nevertheless, he displayed the classical features of anorexia nervosa at presentation, fulfilling the DSM-III diagnostic criteria for this condition.

Anorexia nervosa is relatively rare in males, accounting for approximately 10% of new cases. Its occurrence may indicate underlying Addison's disease which should be excluded in all cases.

References


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