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Nota Clínica

Can pediatricians recognize eating disorders? A case study of early-onset anorexia nervosa in a male child

¿Puede un pediatra sospechar un trastorno de la conducta alimentaria? Un caso de comienzo precoz de anorexia nerviosa en un varón

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Abstract

Introduction: this article examines a boy who was diagnosed with anorexia nervosa at eight years old. Pediatricians and family physicians encountered difficulties during the diagnosis and treatment stages due to the fact that early onset of the disease is rare.

Case report: a boy aged eight years and four months presented with bradicardia, malnutrition and dehydration as far away from the preliminary diagnosis of anorexia nervosa investigated for possible organic pathologies at the pediatric ward. Finally, he was diagnosed with early-onset anorexia nervosa and anxiety disorder based on the Diagnostic and Statistical Manual of Mental Disorders 5.

Anorexia nervosa. Child. Eating disorder.

Key words:

Discussion: the case highlights two important points. First, this case report demonstrates a limited awareness of anorexia nervosa and of a psychosomatic or psychiatric origin of weight loss in children among physicians. Second, disrupted eating behaviors and mood changes may be predicting factors for the development of full-blown anorexia nervosa.

Resumen

Introducción: se presenta el caso clínico de un niño diagnosticado de anorexia nerviosa a los 8 años. Tanto los pediatras como los médicos de familia tuvieron problemas durante el diagnóstico y en el tratamiento debido a que su aparición a tan corta edad es poco frecuente.

Caso clínico: un varón de ocho años y cuatro meses ingresó por bradicardia, desnutrición y deshidratación en un hospital pediátrico. Tras el despistaje de un origen orgánico, se diagnosticó de anorexia nerviosa y trastorno de ansiedad según DSM-5.

Palabras clave: Anorexia nerviosa.

Niños. Trastorno de la conducta alimentaria.

Discusión: el caso pone de relieve dos aspectos. El primero, demuestra la atención limitada que prestan los médicos a un origen psicosomático o psiquiátrico, incluyendo la anorexia nerviosa, en una pérdida de peso en un niño. El segundo, que una alteración en la conducta alimentaria acompañada de cambios de carácter pueden ser factores predictores del desarrollo de un cuadro de anorexia nerviosa florido.

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INTRODUCTION

Anorexia nervosa (AN) is an eating disorder (ED) that is associated with distorted body image and is characterized by excessive weight loss and an intent to lose weight by not eating enough (1). The main problem for AN is that even though patients have a body weight below the normal values, they refuse to eat because of an intense fear of being fat (2). AN has a 5% mortality rate and typically begins during adolescence and early adulthood (10-29 years). It primarily affects young girls; only one in ten of AN cases are men (3,4). The reported prevalence of AN is 0.1-1% in girls aged 15-19 years and 0.02% in boys aged 15-19 years (5).

This article will discuss the case study of a prepubertal boy who was diagnosed with early-onset AN. Physicians encountered problems during diagnosis and treatment due to the early onset.

CASE REPORT

A boy aged eight years and four months was admitted to the Pediatric Gastroenterology Polyclinic due to a lack of appetite and fatigue. He was directed to the Emergency Department because he was extremely exhausted and cachectic and required intravenous fluid. He did not have any systemic or chronic diseases. It was discovered that he had refused to eat solid food for four months and that he had neither eaten nor drunk anything for almost 15 days. During this period, his weight dropped from 20 kg to 16 kg, and he had become withdrawn from his favorite activities and refused to eat even his favorite foods. The symptoms could not be diagnosed by family physicians or pediatricians. Birth and developmental history were normal. There was no ongoing physical or psychological illness in his family.

At admission to the Emergency Department, he was exhausted, sluggish and cachectic enough not to stand up. He did not communicate verbally, did not answer questions, and avoided eye contact (Fig. 1). On physical examination, heart rate was 55 beats/min (bradycardic), respiratory rate was 16/min (normal), blood pressure was 85/65 mmHg (normal), oxygen saturation was 99% (on room air), Glasgow Coma Scale (GCS) score was 15 (normal), and capillary refilling time was below two seconds (normal). In addition, mucous membranes were dry and skin turgor was reduced. Systemic physical examination and neurological examination findings were normal. No physical examination finding suggested child abuse.

In laboratory tests, complete blood count, serum electrolytes, and liver and kidney function tests were normal. Serum uric acid level was 10 mg/dl (high), blood glucose level was 44 mg/dl, and complete urinalysis was normal except for 2+ ketones. In peripheral blood smear, there were 48% polymorphonuclear neutrophils, 46% lymphocytes, and 6% monocytes. Platelets and erythrocytes were normal. There were a few atypical lymphocytes, no abnormal findings on the ECG other than sinus bradycardia were found, and chest X-ray was normal.



Figure 1.

At admission to the Emergency Department, the patient was exhausted, sluggish and cachectic enough not to stand up. He did not communicate verbally, did not answer questions, and avoided eye contact.

CLINICAL APPROACH AND DIAGNOSTIC PHASE IN THE EMERGENCY DEPARTMENT

Blood glucose level was found to be 40 mg/dl. This was interpreted as ketotic hypoglycemia due to prolonged hunger. The patient was given 5 cc/kg of 10% dextrose intravenously. Maintenance and replacement fluid therapy was initiated to provide a glucose infusion rate at 8 mg/kg/min. Oxygen therapy was started with a mask at a flow rate of 6 l/min. Follow-up blood glucose level was 80 mg/dl. Because of personality and behavioral changes, loss of weight, and bradycardia, the primary differential diagnosis was an intracranial tumor. Cranial magnetic resonance imaging was normal. No medical treatment for bradycardia was given because there was no evidence of increased intracranial pressure and the patient was hemodynamically stable. Lymphoma was considered in the second rank in the differential diagnosis; however, there were no organomegaly and night sweats. He had no family history that might support a tuberculosis diagnosis, and the chest X-ray was normal. Because atypical lymphocytes were seen on peripheral blood smear, he was referred to the Children's Ward for further examination and treatment with the preliminary diagnoses of leukemia and lymphoma.

CLINICAL APPROACH AND DIAGNOSTIC PHASE IN THE CHILDREN'S WARD

At admission to the Children's Ward, he was exhausted and agitated, but could stand up. Body weight was 16.8 kg (< 3 p; SDS: -4.01). Height was 122 cm (10 p). His weight-to-height ratio was 74%. His body mass index (BMI) was 11.2 (SDS: -4.75). When the vital findings were examined, heart rate was 60 beats/min, respiratory rate was 20/min, blood pressure was 90/65 mmHg, and body temperature was 37 $^{\circ}$ C.

Physical examination revealed mild dehydration. Neurological examination was not performed due to patient incompatibility. Intravenous fluid/electrolyte therapy was continued. Bone marrow aspiration was performed at the recommendation of the Department of Hematology/Oncology and was found to be normal. Thyroid function measurements were normal. Coeliac disease-related antibodies were negative. Thoracic and abdominal computed tomography was requested to exclude other possible oncologic pathologies; however, his family rejected this suggestion. The patient reacted violently to nasogastric tube feeding and had angry outbursts before every feeding attempt.

During follow-up treatment, he exhibited agitation and anger in refusal of a variety of nutrients and fluids to be administered either orally or nasogastrically. On the third day of hospitalization, he was evaluated by the Child and Adolescent Psychiatry Clinic. The psychiatric assessment indicated that he was depressed, that it was impossible to communicate with him, and that he did not provide clear answers to questions. In addition, it was reported that the patient's perception of his body was distorted, and 10 mg of fluoxetine and 2.5 mg of olanzapine per day were recommended for the preliminary diagnoses of AN and psychotic depression. However, on the fifth day of hospitalization, despite the insistence of the medical staff, the patient and his family refused the treatment and indicated that they would continue with it only after getting another physician's opinion. A week later, the father contacted the hospital to indicate that the boy's general condition was getting worse, and that they would administer the medication at home and would cooperate with the Child and Adolescent Psychiatry Clinic only if this condition was agreed to. In the second week of treatment, the patient was again evaluated by the Child and Adolescent Psychiatry Clinic.

A SECOND PSYCHIATRIC ASSESSMENT PERFORMED BY THE CHILD AND ADOLESCENT PSYCHIATRY CLINIC

According to the assessment, the patient had an elevated anxiety level, did not want to leave his mother, gave short answers to questions, and did not speak without being prompted. The mother was depressed and afraid of losing her son as the result of AN. She was observed to be over-controlling. The patient continued to struggle with his body image, did not eat in the evenings to avoid weight gain, and still felt fat. His Children's Depression Inventory (CDI) score was 22, and his State-Trait Anxiety Inventory for Children (STAIC) score was 43. His weight had dropped from 20 kg to 15 kg over the previous four months, and his perception of his body had become distorted.

Based on the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), the patient was diagnosed with anorexia nervosa, childhood depression and separation anxiety disorder. It was recommended that he continue to receive 10 mg of fluoxetine and 2.5 mg of olanzapine per day. Individual and parental interviews were scheduled; however, the patient and his family did not come to the appointment. Information about whether the medicines were given regularly and his eating habits was obtained via telephone. It was recommended that he continued to receive 10 mg of fluoxetine and 2.5 mg of olanzapine per day. Individual and parental interviews were scheduled. These updates indicated that he took both medicines regularly and started consuming liquid foods again. At the end of the first month of regular eating and medication, he was admitted to the Pediatric Emergency Department, and his body weight was recorded as 18 kg. He showed a better general condition and was active and interested in his surroundings and gave clear answers to guestions (Fig. 2). It was reported that he had started attending school and playing the games he had previously enjoyed.

DISCUSSION

The rate of children with EDs who are identified by Primary Health Care physicians is very low (6). Since AN typically occurs after normal-weight or overweight adolescents make the decision to start a diet, early-onset AN can be easily overlooked by family physicians and pediatricians.



Figure 2.

At the end of the first month, body weight was 18 kg. He also had a better general condition, was active and interested in his surroundings and gave clear answers to the questions. Written informed consent from the parents was obtained.

It has been reported that EDs which do not meet the full diagnostic criteria are seen in 5-8% of boys and in 9-14% of girls among primary school children (7). It was clear that the patient had a disruption in eating behaviors before he met the full diagnostic criteria, but it could not be recognized by family physicians and pediatricians because there was no physical deterioration. AN can also present as a failure to gain weight rather than a loss of weight. For this reason, it can be misleading to evaluate patients on the basis of their BMI alone. Lask et al. reported that thoughts of patients about weight and body shape can be a good predictor for the development of AN in those admitted to the family physician for EDs (5). It is important that family physicians and pediatricians are able to recognize that disrupted eating behaviors, including food refusal, may reflect a patient's mental status. The authors of this report conclude that selective eating behaviors (one of the predictors of AN) accompanied by a reduced awareness of surroundings and mood changes could be factors that signal AN to physicians. For our patient, bradycardia, behavioral changes, and loss of weight pointed to central nervous system malignancies in the differential diagnosis. However, bradycardia can also occur due to the activation of the parasympathetic nervous system in patients with AN. Further examinations were performed in accordance with other differential diagnoses, including lymphoma and leukemia. AN was not initially considered.

A psychiatric consultation was requested for the patient because he exhibited agitation and aggressive behavior toward orogastric tube feeding. The detection of his distorted body perception during the first meeting suggested the final diagnosis. The Diagnostic and Statistical Manual of Mental Disorders (DSM-5) lists the following criteria for the diagnosis of AN: body weight of less than 85% of the normal range, disruption of judgment regarding weight or body image, and pathological fear of weight gain despite a body weight below the normal values (2). In our patient, the diagnosis of AN was based on his refusal to eat due to intense fear of gaining weight and his self-perception as fat, despite being too weak to walk and having a body weight of less than 85% of normal values. Between 25-75% of patients with AN have also experienced childhood-onset anxiety disorders (8). In our patient, AN was accompanied with separation anxiety disorder and depression.

The delay in diagnosis damaged the family's trust in the doctors and led to a lack of cooperation with their approaches to treatment and follow-up.

This case reveals the importance of pediatricians' consideration of AN as a potential diagnosis in prepubertal children. Disrupted eating behaviors and mood changes may be predictive factors for the development of AN. An increased awareness of AN in family physicians and pediatricians is important for facilitating early diagnosis and treatment of the disease and preventing unnecessary deaths.

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