

“Changes in Food Selectivity” : Evolution towards Self-Induced Vomiting in a Boy with Autism Spectrum Disorder

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Abstract

Introduction: Food selectivity (FS) is a common problem in Autism Spectrum Disorders (ASD) for its negative impact on nutrient adequacy, mealtime behavior problems, spousal stress, and food choices for all family. The majority of research on FS in ASD is focused on early childhood. However, the symptoms may persist into adulthood.

Methods: Here, we describe the history of 17 years old boy with ASD, early onset of FS, gastrointestinal dysfunction and recent onset of self-induced vomiting. We report the clinical, instrumental and therapeutic approach in a boy with ASD. We reviewed the literature; the keywords were “food selectivity”, “eating disorders”, “feeding problems” in ASD. Inclusion criteria were studies written in English and in which children were the subjects of the study.

Results: The patient showed a peculiar evolution of FS, towards an eating disorder characterized by self-induced vomiting. During the clinical course presented weight loss, cachexia, dehydration and electrolytes alterations. A rehydration was necessary. Pharmacologic treatment with aripiprazole and intensive behavioural intervention helped the resolution of self-induced vomiting, but not the complete resolution of the FS.

Conclusion: The case of our patient suggests further scenarios of FS evolution, up to strong complex and difficult to treat food behaviour: the self-induced vomiting.

Keywords: Autism spectrum disorder; Food selectivity; Feeding problems; Gastrointestinal dysfunction; Self-induced vomiting

Abbreviations ASD: Autism Spectrum Disorder; FS: Food Selectivity; GI: Gastrointestinal; DSM-IV: Diagnostic and Statistical Manual of Mental Disorders-IV; ADOS-2: Autism Diagnostic Observation Schedule-Second Edition; ADI-R: Autism Diagnostic Interview-Revised; VABS-II: Vineland Adaptive Behavior Scales-Second Edition

Introduction

The feeding problems (as unusual eating patterns, problematic mealtime behaviors, rituals and selective eating) are a common problem for children with Autism Spectrum Disorder (ASD), estimated in 46-89% of children [1]. The food selectivity (FS), defined as accepting only a limited variety of foods and refusing many foods [2,3], is reported as a principal cause of the feeding problems in ASD. The etiology is still not clear, considering that “eating” is a complex behavior for children with ASD: the eating variety requires the change and managing novelty, exposure to novel food, processing the similarities and differences (i.e., texture, color, shape, foods mixed together, foods touching each other). The difficulties with sensory processing differences, behavioral disorders, oral motor impairments, fine motor impairments and gastrointestinal problems common in

individuals ASD may also contribute to feeding p [3-5]. The majority of research on FS in ASD is focused on early childhood and primary school age children, proving a prevalence of FS increased from age 2 to 5 and then levelled off with increasing age. It is interesting to note that in many studies reported in the literature, eating disorders precede the ASD diagnosis with early onset [6], as if it were an intrinsic trait of the ASD. However, the FS in ASD does not necessarily resolve in these early years, although few studies have included adolescent and adult participants [7,8], suggesting that FS continues to be an important issue for adolescents/young adults with ASD. Even fewer studies have examined FS longitudinally to understand atypical or deviant developmental trajectory for FS in ASD, concluding that in young adults with autism there is a reduction of food refusal, but not an increase of food repertoire (as number of accepted foods). Here, we describe the history of 17 years old boy with ASD, early onset of food refusal and FS, gastrointestinal (GI) dysfunction and recent onset of self-induced vomiting.

Case study

A 17-year-old male was followed in the Child Neuropsychiatry of University Federico II of Naples as urgent admission due to self-induced vomiting and weight loss. He was born at term after an uncomplicated pregnancy and delivery, and the birth parameters and psychomotor development were normal. He had been normally breastfed for 10 months, presenting the first problems during weaning,

with refusal of the teaspoon and solid foods, then spontaneously attenuated. At 19 months of age, his parents noted, for the first time, behavioural anomalies and at the age of 3 years and 8 months, the diagnosis of ASD, according to the DSM-IV criteria, was performed. His medical history was otherwise unremarkable, with the exception of a FS characterized by a narrow food repertoire and low variety: only white food (as pasta, fish or yogurt) and colored drinks; the child was also selective by brand, texture, temperature and utensil requirement. The parents described the meal time as complicated since 24 months of life. Nevertheless, its growth was normal. All the haematological and instrumental tests were negative.

At age of 15 yrs, his eating behaviours deteriorated with the appearance of a further restrictive pattern (associated with chronic constipation), hydrophobia, self-induced regurgitation or vomiting during or within minutes after meals and consequent gradual weight loss.

At age of 16 yrs, he was hospitalized for a severe weight loss (14 kg in 6 months) and worsening of eating restriction (only plain boiled pasta and soft drinks, i.e. cola) with an absolute refusal of water and modifications of his meal patterns. During meal observations the boy presented anomalies in the speed of food intake (extremely rapid) and put in place a series of rituals, such as abnormal swallowing, movements of the head, suction of air, belching, which encouraged rumination and self-induced vomiting.

On admission he weighed 32.6 kg (<5%) and measured 167 cm tall (between 10–25%), he presented poor clinical conditions, malnutrition [Body Mass Index: 11.7 kg/m² (<<5%)] and severe dehydration (blood pressure 90/50 mmHg; capillary refill 3–4").

Several exams were performed to exclude organic diseases, like blood tests (celiac disease, thyroid function, autoimmunity, tumor markers), stool exams (calprotectin, parasitological and bacteriological culture), and instrumental investigations (abdominal ultrasound and X-rays, esophageal manometry, impedance and pH monitoring, barium contrast radiography), which resulted normal. An esophagogastroduodenoscopy showed a grade II esophagitis. A cranial MRI scan revealed a small arachnoid cyst (9 mm) located in proximity of the cerebellar flocculus, free of mass effect and without a pathological significance.

Laboratory exams showed a normal total blood count, a severe hypernatremia, a minimal renal dysfunction and a moderate hypertransaminasemia. Electrocardiography and ecocardiography revealed non-specific repolarizations anomalies and a left ventricular hypokinesia with interventricular septal thinning and paradoxical septal motion, probably related to advanced cachexia, dehydration and electrolytes alterations.

Correction of sodium was done slowly in 72 hrs via fluid therapy using an isotonic solution (0.9% saline), to avoid risk of cerebral edema. At the same time, due to continuous vomiting and insufficient oral intake, we started a high calorie nasogastric tube feeding: starting continuously for 24 hrs, then adding during the night extra energy to what consumed orally during the day.

After fluid replacement, sodium levels, renal function tests, transaminases and electrocardiographic findings normalized.

A psychodiagnostic evaluation was carried out; a multidisciplinary workups and standardized tools: Autism Diagnostic Observation Schedule, Second Edition (ADOS-2), Autism Diagnostic Interview-

Revised (ADI-R), the Vineland Adaptive Behavior Scales, Second Edition (VABS-II), and cognitive evaluation were performed.

The results of ADOS-2 module 3 (for children and adolescents with fluent speech) confirmed to the diagnosis of autism with a moderate level of symptomatology. The results of the ADI-R showed scores greater than the cut-off for autism in the three domains, and confirmed developmental anomalies noted before 36 months. Adaptation (evaluated with VABS-II) was low in all domains: socialization, daily life, and communication. The cognitive profile was not evaluable with the progressive matrices and Leiter test for the poor understanding and collaboration.

After an ineffective treatment with olanzapine (15 mg/day) for 6 weeks, aripiprazole (10 mg/day) was prescribed with gradual reduction of symptoms. During hospitalization, intensive behavioural intervention program was started, with parent education focused on self-induced vomiting. Gradually he discontinued nasogastric tube feeding, reintroducing the daily three meals and a variety of foods previously refused, gaining weight subsequently (+4 kg in three weeks). Vomiting and rumination disappeared but ritual feeding patterns and the intake of water remained unchanged.

Afterwards, the patient received a referral for occupational therapy (OT), to address feeding concerns, 30 minutes every day at the time of the meal, flanked by her mother and the therapist. Observations and interventions during this time helped the patient access reinforcement while improving his acceptance of novel food. After 6 months of treatment, the boy's food repertoire increased from three-four food to more than 10. The parents reported a significant improvement of mealtime behaviours at home. However, water consumption remains limited, and it is only possible in a therapeutic setting.

Discussion

Several authors have investigated FS in children with ASD [9,10]. Food preference was affected by food texture and food presentation or by sensory modulation problems as tactile and oral hypersensitivities [11,12]. The refusal to consume particular foods coupled with the inability to tolerate, digest, and absorb them can compromise an individual's overall nutrition status [7].

Despite FS is common in early childhood and primary school age children, the symptoms may persist into adulthood [8,9,10], but far less is known about FS during adolescence and adulthood. Few are the reports in the literature concerning how FS in children with ASD changes with age, usually leading to an improvement, perhaps due also to global behavioral improvement, such as inflexibility or social skills. Both Bandini's [9] and Kushner's studies [3] suggested an improvement of food refusal, but not an increase of food repertoire (as number of accepted foods), concluding that the FS continues to be an important issue for adolescents/young adults with ASD, overall characterized by a food neophobia.

The case of our patient suggests further and new scenarios of FS evolution, up to strong food refusal towards a complex and difficult to treat food behaviour: the self-induced vomiting. Should be this classified as an eating disorder? or considered as an evolution of the previous food difficulty? In this second hypothesis, the etiology could be multifactorial, speculating on sensory processing difficulties, intrinsic GI and behavioral disorders of ASD (as rituals, stereotypical behaviour). It can be assumed that early feeding problems, alteration of taste sensitivity [12], restricted diet, together with the GI disorders

have triggered a vicious circle. Another aspect to consider was the comorbidity and/or related GI disorders as the grade II esophagitis, which may have favored the chronicity of symptoms. Furthermore, in our case, autistic severity and IQ performance were other aggravating factors, as suggested in other study [13]. These data are in line with study [14,15], where the subjects with GI problems had significantly higher levels of affective problems. The transitioning to adulthood presents challenges for all adolescents and their families. It is known that in adolescents and adults with autism there is a significant improvement social cognition, social communication and social motivation. Simultaneously, depression, anxiety, affective problems and other internalizing or externalizing problem can increase [14]. The early identification of eating problem or GI disturbances would be important in order to design specific intervention for these symptoms frequently associated to challenging behaviours in ASD.

Another concern is in regard to the treatment of this disorder. Hypothetically an early attention at the feeding behavior in children with ASD could prevent various problems in adolescence or adults. To date, some studies have reported a correlation between nutritional intervention and behavioral outcome(s). Most of the scientific studies on feeding therapy for children with ASD consist of behavioral intervention, including momentum, response cost, simultaneous presentation, sequential presentation, positive reinforcement for eating, planned consequences for inappropriate meal time behavior or differential reinforcement of alternative behavior [16,17]. It should be noted that these approaches have not been studied empirically.

In our case it has been set a short-term intensive behavioral program, focalized on self-induced vomiting. This approach led to great progress in eating behavioral: both vomiting and rumination disappeared, yet the patient experienced no changes in his ritual feeding patterns, and the intake of water remained insufficient. In our case the therapeutic intervention was divided into an acute phase, aimed at the recovery of body weight and the reduction of vomiting, followed by the setting of a chronic treatment with the purpose of expanding the food repertoire. Drug therapy with aripiprazole has contributed to the reduction of levels of irritability and anxiety during feeding, amplifying the effects of behavioral methodologies. An overlapping experience is reported in the literature in a single clinical case [18]. An attempt to propose guidelines comes from Berry et al. that suggest a multidisciplinary approach for a gastrointestinal evaluation (including a history of reflux, diarrhea, constipation, and presence of blood in the stool), a workup to determine food allergies and sensitivities, an oral motor control to investigate mastication and swallowing functions, and a nutritional analysis to determine potential deficiencies.

Conclusion

Our case suggests further scenarios of FS evolution in ASD subjects, up to strong complex and difficult to treat food behavior: the self-induced vomiting. Further studies are needed to fully understand eating disorders in individuals with ASD, and we think that the reporting of cases like ours can help in the clinical management of patients.

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