

tention to the possible benefits of anti-convulsants and dopamine-antagonists.

Carol A. Coutts, M.D.

Ondria C. Gleason, M.D.

Dept. of Psychiatry, Univ. of
Oklahoma College of Medicine,
Tulsa, OK

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***Achalasia May Mimic
Anorexia Nervosa,
Compulsive Eating Disorder,
and Obesity Problems***

TO THE EDITOR: In the past, physicians did exhaustive medical evaluation in the pursuit of organic pathology for patients with eating disorders.¹ Judging from the literature, the incidence of anorexia nervosa increased over the past century until the 1970s,² and now, physicians have an increased awareness of it and find it easier to diagnose. The consequence is the increasing failure to notice organic pathology in patients who have a history of eating disorders.³ We report the case of a young man referred for evaluation of anorexia nervosa, who, after investigation, turned out to be suffering from achalasia.

Case Report

Mr. A, a 24-year-old Caucasian patient, had a history of vomiting and a 60-kg weight loss over the preceding 7 months (Body Mass Index [BMI] at admission: 17.6). He had suffered from asthma since his childhood. When he

was 18, his weight was 96 kg, and he described, from the age of 18 to age 23, compulsive eating behavior with bingeing, but not purging, and use of laxatives or diuretics. Six months before the beginning of symptoms mimicking anorexia nervosa, his best friend had died in an automobile accident. At this time, he was 120 kg (BMI: 37). He is still very affected by this accident.

When he was admitted, his clinical evaluation was normal except for a low potassium level (2.7 mmol/liter) and frequent complaints about “a lump in the throat.” His parents believed that he practiced self-induced vomiting, although he denied this. Frequently, the parents would force him to eat and wait with him during some time after meals to make sure that he did not vomit, because the boy had uncontrollable vomiting after every meal. The early symptoms were heartburn and dysphagia. The patient stated that he often had chest pain after food or liquid intake. Body-image distortions were absent, but the intention to lose weight was present at the early stage. He denied self-induced vomiting, but did note that vomiting improved his symptoms and that he was preoccupied by food, without any rituals. The patient did not use laxatives or diuretics.

Family history revealed that the mother had been diagnosed as having morbid obesity and had had bariatric surgery. She was currently receiving psychotherapy for depression.

For his general practitioner, the conflict about food and autonomy between Mr. A and his parents was thought to have contributed to the illness.

An upper gastrointestinal radiographic series revealed a grossly dilated esophagus and a tight esophageal sphincter compatible with the diagnosis of achalasia. Pneumatic dilatation of the lower esophageal sphincter was

successful, and the patient has gained weight since that time. He is still under psychotherapeutic treatment for his family and behavioral problems.

Discussion

Dysphagia is the initial and main clinical feature of achalasia. Often, several years elapse before the disease is diagnosed, and, during this time, other symptoms, such as vomiting and weight loss, are common.⁴ During this period, achalasia can be mistaken for anorexia nervosa. Moreover, previous obesity of the patient is of interest, since an association between morbid obesity and achalasia has been described.⁵ This, along with episodes of asthma,⁶ leads us to believe that the patient probably had his achalasia before his symptoms of dysphagia.

Nevertheless, differential diagnosis between achalasia and anorexia nervosa is not always obvious. First, it has been reported that esophageal motor disorders are common in patients with a diagnosis of primary anorexia nervosa.⁷ For example, patients with eating disorders frequently have gastric emptying abnormalities causing bloating, postprandial fullness, and vomiting. These symptoms usually improve with refeeding, but sometimes pro-motility agents may be necessary.⁸ Second, willful avoidance of food and spontaneous or self-induced vomiting have been reported in patients with achalasia.^{9–11} Thus, gastrointestinal disorders are common in eating-disorder patients, and many gastrointestinal diseases sometimes present like eating disorders. But, for Rosenzweig and Traube,¹² errors in diagnosis are related to delay in obtaining appropriate investigations or misinterpretation of their results. Abell and Werkman¹³ suggest that a careful clinical history can localize gastrointestinal motility disorders,

and they suggest appropriate diagnostic tests.¹³ They differentiate between two groups of symptoms: first, dysphagia, odynophagia, heartburn, and reflux have esophageal origins and occur in achalasia. The appropriate diagnostic tests in this case are barium-swallow endoscopy¹⁰ and esophageal motility studies (esophageal manometry or scintigraphy). The second group of symptoms includes nausea, vomiting, anorexia, bloating, and abdominal pain, which are symptoms of motor disorders of the stomach and small intestine.

In summary, the exclusion of organic disease must be a priority, even if a psychotherapeutic intervention may be needed in the global care of this group of patients.

Martin Desseilles

Sonia Fuchs

Marc Ansseau

Dept. of Psychiatry, Univ. of Liège,
Belgium

Sandra Lopez

Elena Vinckenbosh

Antonio Andreoli

Dept. of Psychiatry, Univ. of Geneva,
Switzerland

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Postconcussional Symptoms Not a Syndrome

TO THE EDITOR: Taber's Cyclopedic Medical Dictionary defines syndrome as "a group of symptoms, signs, laboratory findings, and physiological disturbances that are linked by a common anatomical, biochemical, or pathological history." It is my view that symptoms typically attributed to post-concussion are so nonspecific and are associated with such a wide variety of other conditions that they do not meet the definition of a syndrome. Iverson and McCracken¹ showed that postconcussive-like symptoms are not unique to the sequelae of mild traumatic brain injury and can also be seen in conditions of chronic pain. Gouvier et al.² compared undergraduate students and their families with a group of head-injury patients. They concluded that there were "no significant differences found between the brain-damaged individuals and normals on items assessing self-reported memory problems, problems

becoming interested in things, frequent loss of temper, irritability, fatigue, or impatience."²

Lees-Haley et al.³ compared 50 control subjects against 170 personal-injury claimants. The injury claimants had no history of brain injury or toxic exposure. In spite of this, they reported very high rates of complaints generally associated with the so-called "post-concussion syndrome."

Chan⁴ studied base rates of symptoms in patients who had not suffered a head injury. The study showed that a high proportion of participants reported symptoms similar to those with so-called post-concussion syndromes.

Rees⁵ opined that "published observational work on the nature and etiology of "persistent post-concussive syndrome" and, more particularly, its cognitive sequelae, have been characterized by an unfortunate lack of data, errors in sampling, and insecure methodology."⁵

McAllister and Arciniegas⁶ pointed out that the term "post-concussive syndrome" is used inconsistently in the literature, that the symptoms have high base rates in the general population, and that they are nonspecific in nature.

In summary, the so-called symptoms of post-concussional syndrome are notable in that: 1) they are present in a significant number of the normal population, and 2) they are present in very significant numbers of patients who have suffered trauma not involving concussion or brain injury.

Therefore, I conclude there is inadequate evidence that these symptoms meet the definition of a "syndrome."

It is unfortunate that Dr. Hall and colleagues have not referenced these controversies in their otherwise excellent review article.⁷

Derryck H. Smith, M.D., FRCP(C)
Clinical Professor, University of
British Columbia, Medical