Successful treatment of anorexia nervosa and alleviation of chronic Guillain Barré syndrome

Anneke Aden, Thomas Stegemann, Andreas Richterich

University Medical Center Hamburg-Eppendorf Dep. of Child and Adolescent Psychiatry, Hamburg, Germany

Abstract

Eating disorders do not typically occur in conjunction with specific neurological disorders. Only very few cases of Guillain-Barré-Syndrome (GBS) associated with eating disorders have been reported. The objective of this paper is to describe and discuss a case of anorexia nervosa and concomitant chronic GBS. We report on a course of medical management for a 15 year old female patient, who presented with acute neurological syndrome (GBS) which was followed by the onset of a severe eating disorder. The patient was diagnosed to have two different entities, with the association between the two remaining unclear. The mainstay of management was focused on the eating disorder. Using an integrative psychiatric therapy a significant improvement of the eating disorder was achieved. The patient's body weight was stabilised and the locomotor deficits improved. Though a significant somatic disorder was evident, it proved to be advantageous to primarily focus on the eating disorder, until it was under control. The possible correlations between the two distinct disorders are discussed.

Introduction

We report on the case of a 15 year old female adolescent presenting with severe neurological symptoms, which fulfilled the criteria for Guillain-Barré-Syndrome (GBS). The patient also came down with a severe eating disorder.

GBS is comprised of a group of heterogeneous disorders. Since the eradication of poliomyelitis it has become the major cause of acute neuromuscular paralysis in the Western world.¹ The incidence is lower in children than in adults with numbers between 0.4-0.6 and 1.1-1.3/100000.²

The main symptoms of GBS are progressive loss of sensory function, dysaesthesia, flaccid quadriplegia, hypo- or areflexia, which can be accompanied by worsening bulbar and oropharyngeal dysphagia. Diagnostic criteria by defi-

nition are progressive paralysis in more than one limb (comparatively symmetric) and areflexia, at a minimum affecting the distal reflexes, while the sensory-related symptoms are relatively moderate.³

Patients with anorexia nervosa (AN) or other eating disorders (ED) can show symptoms of neurological disorders.^{4,5} The most common form is peroneal nerve palsy.⁶ All these changes might be a consequence of weight loss or malnutrition. They normally subside with weight gain.⁷ To date there is known no relationship between ED and GBS.

Case Report

Initially, the patient was admitted to the department of Paediatrics with numbness in the left corner of her mouth and tongue. Then paraesthesia and numbness of the left hand and forearm occurred. One week before admission the patient had undergone antibiotic treatment for pharyngeal infection, which was no more evident at the time of admission.

A cerebrospinal fluid (CSF) examination revealed an elevated liquor protein concentration without pleocytosis. Cranial and spinal magnetic resonance imaging were normal and, therefore, the patient was discharged from hospital. After an initial clinical improvement, the patient presented with a reactivation of paraesthesia, yet again accompanied by hyperthermia. Within three weeks the patient additionally reported headache, loss of appetite and a weight-loss of 4.5 kg. This corresponded to 10% of her actual body weight. Therefore she was readmitted to the paediatric ward again.

The second CSF examination revealed a severe disturbance of the blood-brain barrier with an elevation of the CSF protein concentration up to 1775 mg/L. No pleocytosis, oligoclonal bands, intrathecal synthesis of immunoglobulins were found. The serum/CSF albumin ratio was 20.4×10^{-3} . Cranial and spinal magnetic resonance imaging, analysis of blood including serological analysis, urinary and stool measurements excluded infections or autoimmune processes. Electroencephalographic and ophthalmological examination were normal.

Due to the continuous weight loss (weight 37.4 kg, height 160 cm, BMI 14.6 kg/m²), the patient was transferred to the Department of Paediatric Neurology. The third CSF examination showed minimal improvement (protein concentration 1320 mg/L); neurophysiological tests revealed a predominantly left-sided axonal sensory neuropathy with evidence of demyelination of the left sural nerve. We found no evidence of endocrine, lysosomal and peroxisomal disorders. A vitamin deficiency syn-

Correspondence: Anneke Aden, University Medical Center Hamburg-Eppendorf, Dep. of Child and Adolescent Psychiatry, Martinistr. 52 D-20246 Hamburg, Germany.

E-mail: aaden@uke.uni-hamburg.de

Key words: Guillain-Barré-syndrome, anorexia nervosa, eating disorder, child and adolescent psychiatry, interdisciplinary treatment.

Contributions: all authors mentioned meet the criteria for authorship/contributorship.

Conflict of interest: the authors report no conflicts of interest.

Received for publication: 22 December 2010.

Revision received: 19 January 2011.

Accepted for publication: 24 January 2011.

This work is licensed under a Creative Commons Attribution 3.0 License (by-nc 3.0).

©Copyright A. Aden et al., 2011

Licensee PAGEPress, Italy

Mental Illness 2011; 3:e1

doi:10.4081/mi.2011.e1

drome, homocysteinuria, granulomatosis or involvement of the autonomic nervous system was not detected. Microbiology tests were normal. Charcot-Marie-Tooth-Syndrome was excluded by investigations of the PMP-22- and MPZ-gene. A slightly elevated titer of antinucleosid- and antigangliosid-antibodies was related with a postinfectious inflammatory polyneuropathy.

The diagnosis of an unspecified eating disorder combined with symptoms of depression was made. Therefore she was treated with fluoxetine 20 mg o.i.d.

No other psychiatric, neurological or chronic somatic diseases were found in the patient's family.

Loss of appetite, nausea, vomiting and rejection of adequate food-intake demanded for the insertion of a nasogastric tube. In the meantime the patient had reached the lowest weight during her illness with a BMI of 13.9 kg/m². Then the patient completely refused food intake. Unaided walking became impossible because of weakness, sensory disturbances and coordination problems. A behavioural therapy regimen, applied for the treatment of eating disorders, was implemented resulting in weight gain. Signs and symptoms of depression improved substantially, but agitation, uncontrollable restlessness and delusion-like fears asked for neuroleptic medication (olanzapine). A modification of the step-by-step plan with more focus on autonomy and individual responsibility, during week 10 of treatment, led to a reduction of stubbornness, a better cooperation and improved oral food intake (Figure 1).

Figure 1. Development of body weight.

An improvement was noted regarding her eating disorder through multimodal treatment (structured feeding plan, one-to-one nursing, individual academic advancement, movement and music therapy, supportive individual psychotherapy and family therapy) in the future course. She gained weight continuously and developed more autonomy. Her neurological symptoms also improved.

Dismissal was arranged with a weight of 45.4 kg (BMI 17.96 kg/m²) and further improving neurologically after 32 weeks. Fourth examination of cerebrospinal fluid showed no abnormalities. The patient was followed up on an outpatient basis by the Child and Adolescent Mental Health team and continued physiotherapy.

Post discharge control examinations were conducted over a period of eight months. The

weight remained constant. Neurological examinations showed improvement with regard to balance, motor system and power. Beside a slight paresis of the left facial nerve, there was reduced sensation of both plantars, while her gait had improved. The patient was attending school regularly, did not show symptoms of depression and her eating behavior was described as being normal.

Conclusions

Although there is no evidence of any aetiological association between GBS and anorexia nervosa, the interaction of both diseases had an important influence on the course of both. Presenting with several risk factors for mental illness the patient developed a severe psychiatric disorder under the distress of a life-threatening neurological disease.

The specific treatment of the psychiatric symptoms included primarily a behavioral approach to the eating disorder combined with neuroleptic medication. An interdisciplinary diagnostic and therapeutic approach focused on the neurological symptoms and confirmed the diagnosis of GBS.

The interventions primarily led to an improvement of the eating disorder and were secondarily followed by an alleviation of the neurological symptoms.

As a result of these experiences we recommend the specific and early treatment of a psy-

chiatric syndrome even if the neurological symptoms are complex and aetiological and diagnostic approaches have not definitely been completed and confirmed.

References

1. Pritchard J. What's new in Guillain-Barré syndrome? *Pract Neurol* 2006;6:208-217.
2. Korinthenberg R, Schessl J, Kirschner, J. Clinical presentation and course of childhood Guillain-Barré syndrome: a prospective multicenter study; *neuropsychiatrics* 2007;38:10-17.
3. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. *Ann neurol* 1990;27:S21-4.
4. Chowdury U, Lask B. Neurological Correlates of Eating Disorders. *Eur Eat Dis Rev* 2000;8:126-33.
5. Herzog W, Munz D, Kächele H. Essstörungen. *Therapieführer und psychodynamische Behandlungskonzepte*. 2. Auflage, Stuttgart, Schattauer, 2004.
6. Kershenbaum A, Jaffa T, Zeman A, Boniface S. Bilateral Foot Drop in a Patient with Anorexia Nervosa. *Int J of Eat Disord* 1997;22:335-7.
7. Herpertz-Dahlmann B, Resch F, Schulte-Markwort M, Warnke A. *Entwicklungspsychiatrie*, 2. Auflage, Stuttgart, Schattauer, 2008.