



Case Report

Manual Cranial Treatment in a Young Woman with Anorexia Nervosa: A Case Report

Ulrich Moser*

General Practitioner, Special Pain Therapy, Manual Therapy, Acupuncture, Sports Medicine, Großheubach, Germany

ARTICLE INFO

Article history:

Received: 8 May, 2020

Accepted: 25 May, 2020

Published: 29 May, 2020

Keywords:

Anorexia nervosa

case report

body image

manual cranial treatment

tonus test of deep suboccipital muscles

ABSTRACT

Background: Anorexia nervosa (AN) is a serious mental disorder affecting mainly young women. Despite the severity of the disease, the danger of chronification and premature death, and the social burden, the evidence base for its treatment is weak. In addition to the changed eating behavior, a modified body schema seems to play a major role in AN.

Case Report: Presented is the course of AN in a now 17-year-old female patient in which a definite improvement of her symptoms occurred in temporal connection with manual cranial treatment. The manual approach is based on tonus changes of the deep suboccipital muscles as a monitor to identify relevant dysfunctions, to ensure the optimal therapy setting and to control the therapy outcome.

Conclusion: AN is an example of a neuropsychiatric disorder whose neurobiological correlates could have resulted in the increased circumscribed cranial tension in the case presented. The pleasing course may encourage us to consider manual cranial treatment in similar cases.

© 2020 Ulrich Moser. Hosting by Science Repository.

Background

AN is a serious mental disorder characterized by a disturbance of eating habits, often resulting in a threatening condition of malnutrition, leading to chronic physical and psychosocial suffering and even death of the patients. The focus of this disorder lies in a self-induced low intake of calories, the omission of meals, and occasional self-induced vomiting, leading to extreme weight loss [1]. Often the disease is completely denied. The life of the patient revolves around food intake [2]. Amenorrhea is often present in female patients. If the disorder begins before puberty, the normal pubertal development steps are delayed. After remission, however, there is a retarded but otherwise normal pubertal development, with a delayed entry of the menarche [1].

Cardiovascular complications such as bradycardia, prolonged QT time, and orthostatic states are reported, including loss of subcutaneous adipose tissue, absence of menstrual bleeding, hair loss, and hyperthermia [3]. Due to improved nutrition and the cessation of

abnormal eating habits, most complications are reversible. In a few cases, however, some of the sequelae of AN can be life-threatening, such as electrolyte derangements, severe bradycardia and hypotonia. Some consequences of AN may be irreversible and cause serious health problems, especially osteoporosis, growth retardation, malfunction of the reproductive system, and neurobiological changes of the brain [4, 5]. In addition to the changed eating behavior, AN results in a modified body schema, finding its expression in the patient's erroneous assessment of the spatial dimension of the own body [5-9].

Functional imaging methods of the central nervous system contribute significantly to the understanding of the neuronal correlates of the main features of AN, including the accompanying affective disorders. A network of key regions of affective processing has been identified, working differently in patients with AN than in healthy people [5]. For example, there is reduced basal activity in the bilateral parietal and prefrontal cortex in AN patient [6]. The thalamus region, the insula, the amygdala, the hippocampus and the striatum also appear to be affected

*Correspondence to: Ulrich Moser, M.D., General Practitioner, Special Pain Therapy, Manual Therapy, Acupuncture, Sports Medicine Marktplatz 2, D 63920, Großheubach, Germany; E-mail: u-moser@t-online.de

[7, 8]. Above all, the right dorsal parietal lobe seems to play a decisive role in the disorder of body schema in AN patients [9].

Because AN patients do not accept their condition as pathological, therapeutic interventions are difficult [10]. Nearly one-half of the AN patient are refractory to treatment [11]. Cognitive behavioral therapy after weight stabilization appears to reduce the recurrence risk in adults with AN. However, nothing is known about efficacy in underweight patients [12]. Variants of family therapy show a low level of evidence among adolescents [12]. SSRI-type and tricyclic antidepressants are not effective in reducing the symptoms of AN [12, 13]. There are only limited and contradictory data regarding atypical antipsychotics [14]. Considering the neurobiological correlations of AN and the fact that the availability of evidence-based treatment is limited, new treatment options taking into account the underlying neurobiological mechanisms are of great interest. Invasive methods such as “deep brain -stimulation” or neurosurgery in chronically therapy-resistant cases take this direction [15]. In this context, a manual therapy approach for AN is presented in the following case study.

Case History

The patient is now 17 years old. Her childhood was overall without any abnormalities, except a sudden rejection by her then best girl friend at primary school. This first human disappointment was followed by a long time of grieving. The premature occurrence of the menarche at the age of 10 should be mentioned.

The eating disorder started in May/June 2015 at the age of 12. The body mass index (BMI) was at 23.0 at that time, with a bodyweight of 65 kg at a body size of 168 cm. At the end of the summer holidays, she had lost 10 kg. Her menstruation was suspended. Unusual behavioral patterns occurred. She checked her weight frequently and often burst into tears if the scale indicated 200 g increase, and in addition, she hardly could be reassured. She counted the calories of her food intake and skipped meals pretending having already eaten. At school, she threw away her snack and sometimes only took lunch.

Up to Easter 2016, she had lost 20 kg and weighed 45 kg (BMI 15.9). Psychiatric examinations confirmed the diagnosis of AN but revealed no connection between the her pre-existing pubertas praecox and the present absence of menstruation. An organic cause of weight loss could be excluded. In September 2016, the attending family doctor (the author) arranged an appointment with a youth psychotherapist. After three sessions, the talks were cancelled. The patient and her parents felt “not to be on the right way”.

The results of recent studies on the importance of body image disorders in the etiopathogenesis of AN strengthened the author’s conviction that a disturbance of body schema could play an essential role in the patient’s present disease [9]. If Sutherland’s hypothesis that intracranial processes can be influenced by external stress compensation held true, it should be possible to favorably influence her condition by manual therapy of the cranial region [16]. These considerations encouraged the author to propose to the patient and her parents a series of manual treatments.

Manual Treatment

The first treatment took place in October 2016. For the localization of cranial dysfunctions, the tonus test of the deep neck muscles was used [17]. The matrix inhibition according to Beuckels was applied to prioritize the lesions (to answer the question, which lesion has to be treated first) [18]. The tonus test again served for the therapy setting of a three-dimensional form-tension (balanced tissue tension technique) and for control of therapy success. The tonus test on the right side was clearly positive and actually reflected a possible dysfunction of the rear right parietal lobe. After successful treatment of a temporomandibular joint dysfunction on the right, the parietal lesion was treated successfully in a balanced tissue tension technique. The tonus test then showed a side-by-side normal tension.

Follow-Up

Already the following day there was an amazing positive change in the patient’s eating behavior. She was playing musical instruments for the first time in months and seemed much more balanced.

The following cranial manual treatments were carried out in a similar way as described above, initially at weekly intervals, then, after clinical improvement every 2 weeks and finally every 3-6 weeks. The tension in the right posterior parietal area, over time, could still be felt, but was significantly reduced. The clinical symptoms showed strong fluctuations, many tears still flowed, but overall the situation improved noticeably. In January 2017, the patient returned to normal eating- portions, she ate with pleasure, and she was also interested in the many other things that made her life before the crisis – drawing, making music, reading, and meeting her friends.

The menstrual period came back again in November 2017 in regular four-week intervals. At the last consultation in late April 2020, the patient reported a stable weight condition (62 kg) with a height of 1.69 m (BMI 22, 6); eating portions and eating habits had normalized and she was satisfied with her appearance.

Discussion

The positive development of this young female patient with AN, in whom the application of a manual treatment in the cranial area was proving to be the turning point of her disease, is quite astonishing. The palpable strain in the right posterior parietal area suggests a connection with a possible dysfunction of the posterior parietal lobe, which seems to be decisive for the maintenance of an adequate body schema [9]. The inclusion of the cranium in a manual treatment concept is based on William G. Sutherland’s view [16]. According to Sutherland, the dural system is under continuous tension, with each pull or pressure on one side changing the entire dural unit and producing new tension equilibrium. A “membranous articular strain” can be caused by external, and (as in the AN) by internal processes. The manual treatment accordingly aimed at solving these biases and thus improving the functional disturbances [19].

This hypothesis seems to be plausible and is also supported by current research findings in the neuro- and cellular-biological field: Anatomical

and histological studies show that the fibrous layer of the dura mater (lamina fibrosa externa) facing the cranial bone can be regarded as the "inner periosteum" of the skull. The fibrous tracts of the external lamina fibrosis orientate themselves to the trabeculae of the bone, with which they are tightly connected [20]. In this transitional layer, the fibroblasts of the dura closely link to the arachnoid. All cortical connective tissue layers are interwoven on the cellular plane from the brain surface to the cranial bone. These tissues pass seamlessly into each other. The cerebral connective tissue and the bone structures of the skull form a continuum [21]. Recent studies also show that the cerebral connective tissue does not only have supportive, protective and nutritional functions but is also closely involved in the information system of the brain. In particular, astrocytes are found as integral functional elements of the synapses, reacting to changes in neuronal activity and influencing the regulation of synaptic transmission processes and neuronal plasticity. They play a major role in the formation, maintenance and functionality of synapses in the central nervous system [22, 23].

Conclusion

AN is an example of a neuropsychiatric disorder, the neurobiological correlate of which may have led to the palpated circumscribed tension increase in the cranium. The pleasing course in the patient is encouraging to consider a manual cranial treatment in similar cases. Causality can never be inferred from an uncontrolled observation. An association does not automatically imply a cause-effect relationship. It cannot be excluded that the observation in this case report could also be a mere coincidence. This limitation is shared by all descriptive studies.

Acknowledgement

I would like to thank the patient and her parents who gave their permission for the publication of her history and Dr. Edith Zorn, who helped me to translate my German manuscript into English.

REFERENCES

1. Fitzpatrick KK, Lock J (2009) Anorexia nervosa. *BMJ Clin Evid* 2009: 1011. [[Crossref](#)]
2. Keski Rahkonen A, Hoek HW, Susser ES, Linna MS, Shivola E et al. (2007) Epidemiology and Course of Anorexia Nervosa in the Community. *Am J Psychiatry* 164: 1259-1265. [[Crossref](#)]
3. Katzman DK (2005) Medical complications in adolescents with anorexia nervosa: a review of the literature. *Int J Eat Disord* 37: 52-59. [[Crossref](#)]
4. Treasure J, Claudino AM, Zucker N (2010) Eating Disorders. *Lancet* 375: 583-593. [[Crossref](#)]
5. Kaye WH, Fudge JL, Paulus M (2009) New Insights Into Symptoms and Neurocircuit Function of Anorexia Nervosa. *Nat Rev Neurosci* 10: 573-584. [[Crossref](#)]
6. Lipsman N, Woodside DB, Lozano AM (2014) Neurocircuitry of limbic dysfunction in anorexia nervosa. *Cortex* 62: 109-118. [[Crossref](#)]
7. Suchan B, Busch M, Schulte D, Grönemeyer D, Herpertz S et al. (2010) Reduction of Gray Matter Density in the Extrastriate Body Area in Women With Anorexia Nervosa. *Behav Brain Res* 206: 63-67. [[Crossref](#)]
8. Takano A, Shiga T, Kitagawa N, Koyama T, Katoh C et al. (2001) Abnormal neuronal network in anorexia nervosa studied with I-123-IMP SPECT. *Psychiatry Res* 107: 45-50. [[Crossref](#)]
9. Nico D, Daprati E, Nighoghossian N, Carrier E, Duhamel JR et al. (2010) The Role of the Right Parietal Lobe in Anorexia Nervosa. *Psychol Med* 40: 1531-1539. [[Crossref](#)]
10. Flament MF, Bissada H, Spettigue W (2012) Evidence-based Pharmacotherapy of Eating Disorders. *Int J Neuropsychopharmacol* 15: 189-207. [[Crossref](#)]
11. Arcelus J, Mitchell AJ, Wales J, Nielsen S (2013) Mortality Rates in Patients With Anorexia Nervosa and Other Eating Disorders. A Meta-Analysis of 36 Studies. *Arch Gen Psychiatry* 68: 724-731. [[Crossref](#)]
12. Bulik CM, Berkman ND, Brownley KA, Sedway JA, Lohr KN et al. (2007) Anorexia Nervosa Treatment: A Systematic Review of Randomized Controlled Trials. *Int J Eat Disord* 40: 310-320. [[Crossref](#)]
13. Biederman J, Herzog DB, Rivinus TM, Harper GP, Ferber RA et al. (1985) Amitriptyline in the Treatment of Anorexia Nervosa: A Double-Blind, Placebo-Controlled Study. *J Clin Psychopharmacol* 5: 10-16. [[Crossref](#)]
14. McKnight RF, Park RJ (2010) Atypical Antipsychotics and Anorexia Nervosa: A Review. *Eur Eat Disord Rev* 18: 10-21. [[Crossref](#)]
15. Oudijn MS, Storum JG, Nelis E, Denys D (2013) Is deep brain stimulation a treatment option for anorexia nervosa? *BMC Psychiatry* 13: 277. [[Crossref](#)]
16. Magoun HI (1966) *Osteopathy in the Cranial Field*. 2. Ed. Kirksville, MO: Journal Publishing Company.
17. Moser U (2016) Tonustest der tiefen subokzipitalen Muskeln. Neue Perspektive für die osteopathische Behandlung somatischer Dysfunktionen im kraniozervikalen Bereich? *Manuelle Medizin* 54: 109-114.
18. Beuckels JM (2012) Integrative Ansätze der Osteopathie aus faszialer Sicht. In: *Osteopathische Behandlung von Kindern*, Liehm T (Publisher) Haug Verlag.
19. Sutherland WG (1990) In: Wales AE, Ed: *Teachings in the Science of Osteopathy*. Cambridge, MA, Rudra Press.
20. von Lanz T, Wachsmuth W (1982) *Praktische Anatomie. Ein Lehr- und Hilfsbuch der anatomischen Grundlagen ärztlichen Handelns. Erster Band Erster Teil: Kopf Teil A: Übergeordnete Systeme* Springer, Berlin.
21. Carreiro JE (2004) Die Anatomie der Hirnhäute. In: *Pädiatrie aus osteopathischer Sicht*. Urban&Fischer.
22. Araque A, Navarrete M (2010) Glial cells in neuronal network function. *Philos Trans R Soc Lond B Biol Sci* 365: 2375-2381. [[Crossref](#)]
23. Cragnolini AB, Lampitella G, Virtuoso A, Viscovo I, Panetsos F et al. (2020) Regional Brain Susceptibility to Neurodegeneration: What Is the Role of Glial Cells? *Neural Regen Res* 15: 838-842. [[Crossref](#)]