Neuromodulation in the treatment of anorexia nervosa – a literature review

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Abstract

Introduction: Anorexia nervosa (AN) is a disorder in which an unnaturally low body weight is deliberately maintained, with the coexistence of a distorted body image. The effectiveness of AN treatment so far is about 40%. For this reason, new methods of treatment are sought, which include brain neuromodulation techniques.

Purpose: The aim of this article was to determine the effectiveness of neuromodulation techniques in the treatment of AN.

Materials and methods: The literature on the use of neuromodulation techniques in patients with AN was reviewed via electronic databases: PubMed/MEDLINE, Google Scholar, ClinicalTrials.gov. The scope of the search was limited to 01/01/2008-03/01/2023. Articles in English, such as randomized controlled trials, non-randomised controlled trials, case reports, were included. In order to verify the quality of the article, the six-point SANRA scale (Scale for the Assessment of Narrative Review Articles) was used.

Results: The results of the studies conducted so far are not homogeneous. The limitation of conclusions is primarily due to the small number and type of clinical trials conducted, involving small groups of subjects. The literature on the subject lacks systematic reviews and meta-analyses, which makes it impossible to reliably determine the effectiveness of therapy.

Conclusions: Based on Evidence-Based Medicine and the level of credibility of the available data, there is insufficient evidence to recommend neuromodulation techniques for the standard treatment of AN. Further randomized, double-blind clinical trials are needed to assess the effectiveness of this form of treatment. However, the existing data is promising.

anorexia nervosa; electroconvulsive therapy; neuromodulation; repeated transcranial magnetic stimulation; transcranial direct current stimulation

INTRODUCTION

Anorexia nervosa (AN) – a type of eating disorder, classically defined as the deliberate maintenance of an unnaturally low body weight, associated with a strong fear of gaining weight and a distorted image [1].
According to the existing ICD-10 criteria, the diagnosis of AN (F50.0) was based on a BMI of 17.5 or less or an actual body weight at least 15% below expected body weight [2]. The ICD-11 classification introduces some diagnostic changes to the existing ICD-10 classification. According to the new criteria, the diagnosis of AN is based on a BMI of less than 18.5 kg/m² in adults and a body weight below the 5th percentile for age in children and adolescents. Rapid weight loss (e.g., more than 20% of total body weight in 6 months) may replace BMI as long as other diagnostic requirements are met. In children and adolescents, the criterion of weight loss may be replaced by the criterion of failure to gain weight as expected for age [3].

It is estimated that approximately 1.4% of women and 0.2% of men will develop anorexia nervosa during their lifetime [4]. Martinez-Gonzalez et al. conducted a systematic review of 31 studies published between 1980 and 2019 on the prevalence of anorexia nervosa in women of different age groups, mainly from Western countries. The cumulative incidence rate from studies based on outpatient services was 8.8 per 100,000, higher than the cumulative hospital admission rate of 5.0 per 100,000. Compared to these rates for all age groups, the cumulative incidence rates were higher for women aged 10–29 [5]. The epidemiology of AN was also influenced by the SARS-CoV-2 pandemic, during which many researchers observed an increase in the incidence of AN. According to Agostino et al., the incidence of the disease increased from 24.5 to 40.6 cases per month, and the number of hospitalizations among these patients rose from 7.5 to 20.0 per month. During the first wave of the pandemic, the disease onset was more rapid and more severe than before the pandemic [6]. On the other hand, researchers from Australia, since the beginning of the COVID-19 pandemic, have observed a 104% increase in the need to hospitalize children with AN compared to the three previous years [7].

Despite advances in medicine and the development of various therapeutic methods, the effectiveness of AN treatment so far is only about 40% [8], and approximately 60% of patients affected by the disease still meet the diagnostic criteria of AN two decades after the onset of the disease [9]. It is worth noting that AN is characterised by a very high mortality rate and is associated with many medical complications, such as electrolyte disturbances, arrhythmias or endocrine complications [10]. According to a cohort study reported by Tseng et al., the mortality rate for AN was 5.42 deaths per 1000 person-years. Subjects from the AN group had a significantly higher risk of mortality from both natural and unnatural causes compared to healthy individuals. Suicide was the most common cause of death [11].

Due to the numerous somatic complications, high mortality risk and often lack of efficacy of existing forms of AN treatment, it is necessary to search for new methods, such as neuromodulation therapies. Interventions targeting the underlying neurobiological dysfunction are, to date, rarely used in the treatment of AN. One of the reasons for this may be that the exact etiology of AN is still not fully understood. However, advances in brain imaging techniques have identified areas of the brain involved in the pathogenesis of AN, allowing the use of therapies that potentially target the biological basis of AN [12].

The pathogenesis of AN is thought to be related to abnormalities in the reward system. In their recent study, researchers Bronleigh et al. used fMRI to compare patterns of brain activity associated with food stimuli in a group of AN patients with a healthy control group. In patients with AN, hypoactivation was found in brain areas related to the reward system – the amygdala and lentiform nucleus, and in the area responsible for interoceptive processing – the insula. Patients with AN also showed hyperactivation in the prefrontal cortex and anterior cingulate cortex, which are involved in the control of cognitive functions [13].

The key areas of the reward system are the striatum, particularly the nucleus accumbens, and the amygdala, and dopamine is the main neurotransmitter. One of the causes of AN is thought to be a dysfunction of the dopamine-striatal system, which may explain the lack of pleasure associated with eating [1]. Interoceptive processing refers to functional devices such as satiety, hunger, and taste [14]. The area responsible for interoception is the anterior insula, through which the peripheral nervous system communicates with limbic and cortical homeostatic control centers [15]. Patients with AN...
have abnormal hunger perception; impaired interoceptive processing is thought to contribute to the persistence of eating disorders [16].

Cognitive control regions, primarily the prefrontal cortex, are connected to both the reward system and areas involved in interoception. Researchers agree that these areas are responsible for regulating eating behaviour. The prefrontal cortex integrates goals, interoceptive states, and sensory input to guide control functions such as behavioural inhibition and decision making [17].

The hypothalamus has also been implicated in the pathogenesis of AN. The hypothalamus plays a key role in behavioural and emotional responses. Patients with AN do not adapt their eating behaviour to the energy needs of the body, leading to a dysfunction of the orexigenic and anorexigenic neuropeptides responsible for appetite regulation in the hypothalamus. The lateral area of the hypothalamus contains orexigenic neurons and sends projections to the brain’s reward circuits. It has been suggested that dysfunction of hypothalamic orexigenic neuropeptides may alter the reward system in AN [1].

The above reports suggest the possibility of using neuromodulation techniques as a therapy targeting the biological causes of AN. For this reason, we decided to collect information on the effectiveness of neuromodulatory therapies such as repetitive transcranial magnetic stimulation (rTMS), transcranial direct current stimulation (tDCS) and electroconvulsive therapy (ECT) in the treatment of anorexia nervosa.

**Purpose**

The aim of this narrative review was to determine the effectiveness of neuromodulatory therapies such as repetitive transcranial magnetic stimulation (rTMS), transcranial direct current stimulation (tDCS) and electroconvulsive therapy (ECT) in the treatment of anorexia nervosa.

**MATERIALS AND METHODS**

The literature on the use of neuromodulation techniques in patients with AN was reviewed via electronic databases: PubMed/MEDLINE, Google Scholar, ClinicalTrials.gov. The search period was limited to 01/01/2008-03/01/2023. The search included articles in English, such as: randomized controlled trials, non-randomized controlled trials, case reports. The searches included “anorexia nervosa” in combination with “neuromodulation”, “electroconvulsive therapy”, “repeated transcranial magnetic stimulation”, transcranial direct current stimulation”. In order to verify the quality of the article, the six-point SANRA scale (Scale for the Assessment of Narrative Review Articles) and the seven-point checklist were used. The use of a 7-item checklist made it possible to identify key questions about the work, the literature search process and its quality and also allowed the article to be assessed as a narrative review and its implications for further research and clinical practice [18]. Thanks to the use of the SANRA scale, it was possible to maintain the correct structure of the article by taking into account its components: presentation of the meaning and purpose of the review, description of the literature search, references to key issues, scientific reasoning and presentation of relevant data regarding the final points of the article.

**RESULTS**

For the sake of clarity, the article has been divided into the following subsections: 1. Repetitive Transcranial Magnetic Stimulation (rTMS), 2. Transcranial direct current stimulation (tDCS), 3. Electroconvulsive Therapy (ECT).

Repetitive Transcranial Magnetic Stimulation (rTMS)

Repetitive transcranial magnetic stimulation is a non-invasive brain stimulation technique that uses a pulsed magnetic field applied to the surface of the scalp to produce focal electrical stimulation of the cortical surface [19]. In rTMS, there is an increase or decrease in cortical excitability in the target areas of the brain, depending on the frequency of the electrical current applied. Frequencies of up to 1 Hz are thought to have an inhibitory effect on neurons, similar to the LTD effect, while at higher rTMS frequencies excitatory neuronal effects are observed, similar to the effect of long-term neuronal potentiation (LTP) [20].

Based on clinical observations to date, it is believed that rTMS is well tolerated and safe [21]. rTMS is currently being investigated for use in
the treatment of various mental disorders. So far, the strongest reports on its effectiveness concern the use of rTMS in the treatment of depression [22].

rTMS in the treatment of AN

In the TIARA project, a double-blind randomized controlled trial of rTMS in AN, high-frequency rTMS was applied to the dorsolateral prefrontal cortex (DLPFC) in 34 patients diagnosed with severe and persistent AN. After 4 months from the intervention, medium and large intergroup differences were observed in terms of mood and quality of life, and small intergroup differences for BMI. All of these results favored the rTMS-treated groups [23]. At the 18-month follow-up of the TIARA study, mood improvement remained stable in the rTMS group. Regarding the BMI parameter, a faster rate of weight gain was observed after 18 months in the rTMS group compared to the control group (BMI above 18.5 kg/m2: 46% vs. 9%). Based on these results, it can be concluded that the effect of rTMS on mood in AN precedes the effect on BMI [24]. In addition, a decrease in food restriction was observed in patients after rTMS treatment. The reasons for this phenomenon are seen in functional changes in the DLPFC and related neural circuits [25].

The results of the TIARA RCT study show that rTMS may be a potential treatment option for patients with AN, but the results are inconsistent. The diversity of results may result from neuroanatomical, neurofunctional and sociodemographic factors [26].

Jessica McClelland et al. presented the cases of 5 women diagnosed with AN and a BMI ranging from 14-18.5 kg/m2, treated with 20 series of high-frequency repetitive transcranial magnetic stimulation directed at the left DLPFC. After treatment, there was an improvement in stress levels, anxiety and the desire to reduce food intake. Qualitative feedback from participants and tutors was encouraging. However, BMI did not change significantly in comparison to the period before the rTMS procedures. Moreover, after 12 months of follow-up, BMI decreased again in the range of 0.46 to 1.39 kg/m2, which corresponded to a weight loss of 1.2–3.9 kg [27].

Transcranial direct current stimulation (tDCS)

Transcranial DC stimulation is a non-invasive technique for inducing long-term functional changes in the human brain. This method increases (anodic tDCS) or decreases (cathodic tDCS) cortical excitability by applying a direct current flow through electrodes placed on the scalp [28].

It is one of the techniques that is easy and inexpensive to use, as well as safe – most of its side effects are mild and disappear soon after stimulation [29].

tDCS in anorexia nervosa

A recent randomized clinical trial investigated the effect of 10 sessions of anodal tDCS on DLPFC in 43 patients with AN. The effects were assessed using the Eating Disorders Research Questionnaire (EDE-Q) and the Zung Depression Self-Rating Scale (ZUNG), as well as changes in BMI. After 4 weeks of observation, it was concluded that tDCS had no significant effect on weight gain in the cases studied. On the other hand, tDCS reduced the need to follow specific dietary rules and improved patients’ body image ratings [30].

The literature on the subject also contains anecdotal reports of the use of this technique with positive results. In a single-blind, clinically controlled study, 23 adolescents with AN received 18 sessions of tDCS. After 6 weeks of treatment, BMI increased in the study group and this effect was maintained for 1 month of follow-up. Improvements in AN symptoms, mood and anxiety symptoms were observed [31].

A similar improvement in mood and eating behavior was observed after 20 anodal tDCS procedures in a series of 10 cases of patients hospitalized with AN diagnosis. Unfortunately, no changes in BMI were observed in these patients as a result of the therapy [32].

It is worth mentioning the currently ongoing randomized pilot clinical trial in Australia [33] aimed at determining the possibility of using high-resolution tDCS in the lower part of the left parietal lobe (IPL) among patients with AN. The IPL is a new potential target for neuromodulation in AN due to studies indicating reduced connectivity in this region [34] and its role in eye movement, multisensory integration and body...
image. Possible discoveries may become the basis for future tDCS trials in AN.

Moreover, in 2022 a randomized double-blind clinical trial was launched in Lublin, the aim of which is to determine the impact of tDCS on the mental state and progress in nutritional rehabilitation of patients with AN, with particular emphasis on the safety of this therapy. A low-intensity direct current is used – up to 2000uA-2mA. Possible results confirming the effectiveness of this therapy could be an argument for introducing it to treatment in psychiatric wards or at home [35].

| Table 1. rTMS-Summary of key findings from articles included in the review. |
| Author | Population | Outcome measurement | Results |
| McClelland et al., 2016 (27) | 5 patients | BMI; Eating Disorders Examination Questionnaire (EDE-Q); Depression, Anxiety and Stress Scale (DASS-21) | No significant effect on BMI; after 12 months of follow-up, BMI decreased again, ranging from 0.46 to 1.39 kg/m²; improvement in levels of stress, anxiety and willingness to restrict eating (EDE-Q, DASS-21 scales) |
| Dalton et al., 2020 (TIARA) (23) | 34 patients | BMI; Eating Disorder Examination Questionnaire version 6.0 the Fear of Food Measure; the Self-Starvation Scale; the Eating Disorder Recovery Self-Efficacy Questionnaire; Depression, Anxiety and Stress Scale (DASS-21); the Positive and Negative Affect Schedule; the Profile of Mood States; the revised Obsessive-Compulsive Inventory; EuroQol Quality of Life Scale (5-level EQ-5D) | Medium and large between-group differences in mood and quality of life and small between-group differences in BMI in the rTMS groups; faster rate of weight gain in the rTMS group compared with the control group at 18 months (BMI over 18.5 kg/m²: 46% vs. 9%). |

| Table 2. tDCS-Summary of key findings from articles included in the review. |
| Author | Population | Outcome measurement | Results |
| Costanzo et al., 2018 (31) | 23 teenagers | EDI-3, Bulimia (B), Global mental maladjustment (GPM), Interpersonal problems, BMI | Increased BMI in the study group; this effect persisted throughout 1 month of follow-up |
| Strumila et al., 2019 (32) | 10 patients | Eating Disorders Inventory (EDI), Eating Disorders Examination Questionnaire (EDE-Q), Body Shape Questionnaire (BSQ-34), Beck Depression Inventory (BDI), BMI | No change in BMI; decrease in depression symptoms; EDI decline; decrease in scores on the EDE-Q questionnaire |
| Baumann et al., 2021 (30) | 43 patients | Eating Disorders Examination Questionnaire; Zung Depression Self-Rating Scale (ZUNG); BMI | No significant effect on weight gain; reducing the need to follow specific dietary rules, improving body image assessment |
| Rząd, Szewczyk et al., 2022 (35) | 1 patient (case report) | Fasting venous blood: electrolyte levels, red blood cell, hemoglobin, hematocrit, mean corpuscular volume, mean corpuscular hemoglobin, ferritin, white blood cell, lymphocytes, monocytes, FT3, FT4, TSH; Eating Attitudes Test, Rosenberg Self-Esteem Scale, BDI, EDE-Q, Body Feeling Scale, Perceived Stress Scale. | Improving of anthropometrics measurements; reduction of depression symptoms and stress; improvement in body image |
Research in progress:

<table>
<thead>
<tr>
<th>Author</th>
<th>Population</th>
<th>Outcome measurement</th>
<th>Results</th>
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<tr>
<td>Phillipou, Kirkovski, et al., 2019 (34)</td>
<td>20 patients</td>
<td>Physical measurements (Tanita body composition scale), BMI, AN symptom scales, Eating Disorders Examination Questionnaire (EDE-Q), Stunkard Figure Rating Scale (FRS), Dysmorphic Concern Questionnaire (DCQ; modified for one week), Depression Anxiety Stress Scale (DASS-42), State Trait Anxiety Inventory (STAI)</td>
<td>ongoing</td>
</tr>
<tr>
<td>Rząd, Szewczyk et al., 2022</td>
<td>40 patients</td>
<td>Fasting venous blood: electrolyte levels, red blood cell, hemoglobin, hematocrit, mean corpuscular volume, mean corpuscular hemoglobin, ferritin, white blood cell, lymphocytes, monocytes, FT3, FT4, TSH; Eating Attitudes Test, Rosenberg Self-Esteem Scale, BDI, EDE-Q, Body Feeling Scale, Perceived Stress Scale.</td>
<td>ClinicalTrials.gov ID NCT05814458</td>
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Table 3. ECT-Summary of key findings from articles included in the review.

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<th>Author</th>
<th>Population</th>
<th>Outcome measurement</th>
<th>Results</th>
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<tr>
<td>Poutanen et al., 2009 (44)</td>
<td>1 patient (case report)</td>
<td>BMI</td>
<td>BMI increase from 15.0 to 15.3</td>
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<td>Andersen et al., 2017 (45)</td>
<td>1 patient (case report)</td>
<td>Body weight; Children’s Depression Rating Scale (CDRS-R)</td>
<td>Weight increase from 54 kg to 63.7 kg; stable BMI after completion of therapy (21.78 kg/m²); Children’s Depression Rating Scale (CDRS-R) scores decreased from 43 to 17.</td>
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<tr>
<td>Naguy et al., 2019 (46)</td>
<td>1 patient (case report)</td>
<td>Body weight</td>
<td>4kg increase in body weight</td>
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<tr>
<td>Duriez et al., 2020 (47)</td>
<td>1 patient (case report)</td>
<td>Eating Disorder Inventory 2; Hospital Anxiety and Depression Scale (HADS); BMI</td>
<td>Minor changes in the Eating Disorder Inventory (182 vs. 141), HADS for Anxiety (11 vs. 14) and HADS for Depression (10 vs. 9). The baseline BMI was 19.6 kg/m²; after 2 months, the BMI decreased to 13.6 kg/m².</td>
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<tr>
<td>Smallenburg et al., 2022 (43)</td>
<td>1 patient (case report)</td>
<td>BMI; Hamilton Depression Index</td>
<td>Increase in BMI, decrease in Hamilton Depression Index (from 27 to 18 points); recurrence of AN symptoms after several months</td>
</tr>
<tr>
<td>Shilton et al., 2020 (42)</td>
<td>30 patients</td>
<td>BMI; Clinical Global Impression Severity Scale (CGI-S)</td>
<td>Statistically significant reduction in CGI-S score; increase in BMI (pre-ECT BMI 19.43 ± 1.06 vs post-ECT BMI 20.14 ± 1.22)</td>
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Electroconvulsive Therapy (ECT)

Electroconvulsive therapy is a non-pharmacological biological treatment that consists of inducing a seizure by applying an electrical stimulus to the brain through electrodes, usually placed on either side of the scalp. The procedure is performed under general anaesthesia and muscle paralysis [36]. The electrical impulse stimulates neurons in the brain by changing their internal electrical environment and ion concentration. Simultaneous depolarization of groups of neurons causes convulsions that have a therapeutic effect on various neuropsychiatric disorders [37]. ECT modulates the process of neurotransmission in the brain. It regulates the levels of transcription factors, neurotransmitters, neurotrophic factors and hormones such as sero-
tonin, dopamine, acetylcholine, endogenous opioids, epinephrine and norepinephrine. It acts at many levels of neurotransmission in the brain – affecting the synthesis, release, binding to receptors and reuptake of neurotransmitters [38]. It is suggested, among others, that ECT modulates dopaminergic receptors, transiently increases dopamine receptor binding and enhances dopaminergic neurotransmission in the brain [39].

The use of ECT in AN therapy

At the outset, it should be noted that there have been no randomised clinical trials of the efficacy and safety of ECT in AN. The first available descriptions of the use of ECT among AN patients date back to 1954 [40]. Electroconvulsive therapy was rarely used as the primary method of AN treatment, however, it was recommended in severe cases that did not respond to standard forms of therapy [41]. In 1993, Ferguson suggested that indications for ECT in patients with AN may include persistent vomiting, refusal to eat, coexisting depressive episode or psychotic symptoms. Since then, no specific guidelines for the use of ECT in patients with AN have been developed. The optimal ECT protocol for these patients also remains unclear. However, there are reports in the literature on the effectiveness of ECT in patients with AN and depression, which are presented below.

A broader analysis of the subject was made by Shilton et al. Their study described 30 patients who were hospitalised with a diagnosis of anorexia nervosa with coexisting symptoms of major depression and suicidal ideation. The BMI of the patients ranged from 11.8 to 17.1 kg/m2. The severity of eating disorders and depressive symptoms was assessed retrospectively using the Clinical General Impressions Scale. All patients showed resistance to the current antidepressant pharmacotherapy. After an average of 17 bilateral ECT sessions, both AN and depression symptoms were reduced. No serious side effects were reported. Unfortunately, approximately 53% of patients were rehospitalized within the first year after ECT due to increasing symptoms of depression and suicide attempts. Over the next several years after hospitalization, 46.6% of patients did not show a recurrence of depression, suicidal thoughts or symptoms of eating disorders, while 23% had only symptoms of AN [42].

The study of the literature on the subject also includes descriptions of individual cases of AN patients treated with ECT. One of these, described by L.C.S. Smallenburg et al., concerns a 21-year-old Dutch woman with AN, depression and suicidal tendencies. Attempts of both cognitive-behavioral psychotherapy and pharmacotherapy with various antidepressants (fluoxetine 40 mg, clomipramine 150 mg, venlafaxine 225 mg and nortriptyline with lithium) were unsuccessful. Due to the patient’s deteriorating mental state (27 points on the Hamilton Depression Scale), a history of serious suicide attempts and malnutrition, ECT was decided upon. Twelve bilateral ECT sessions were performed, two per week. The mean duration of an epileptic seizure on the EEG was 50 seconds. The patient’s well-being improved during treatment. A daily intake of at least 1800–1900 kcal/day was maintained without coercion, which was not the case before ECT, achieving a BMI of 15 in the middle of the therapy. The Hamilton Depression Index dropped to 18 points. After being discharged home, the patient reported that she had maintained a normal BMI. Unfortunately, after a few months, the patient began to restrict her food intake and lose weight again [43].

On the other hand, Outi Poutanen et al. in their study presented the case of a patient diagnosed with AN and a coexisting major depressive episode. The dominant symptoms were decreased energy, anhedonia, loss of appetite, feelings of worthlessness, and delusions about one’s own body and weight. The patient refused food and water, provoked vomiting, and exercised intensively. Initially, 10 sessions of ECT were performed, resulting in a decrease in the severity of depression symptoms. There was also an increase in BMI from 15.0 to 15.3. Electrotherapy was supplemented with another 12 ECT sessions, and then 23 maintenance electroconvulsive therapy (mECT) sessions were recommended. Pharmacological treatment was maintained in the months without ECT. The applied therapeutic interventions turned out to be effective in alleviating the symptoms of depression, but had little effect on the patient’s weight gain [44].

Andersen et al. presented the case of a 14-year-old girl with AN requiring nasogastric feeding
and coexisting major depressive disorder with suicidal tendencies who was successfully treated with electroconvulsive therapy. A total of 22 ECT sessions were administered over 15 weeks – 13 sessions during hospitalisation and 12 outpatient sessions with a gradually decreasing frequency. Weight increased from 54 kg on admission to 63.7 kg after full ECT therapy, with a stable body mass index of 21.78 kg/m². The Children’s Depression Rating Scale (CDRS-R) score decreased from 43 to 17, indicating remission of the depressive disorder [45].

In the literature there are also case reports of the use of ECT in AN in the absence of comorbidities. One of them concerned a 16-year-old patient with a baseline BMI of 16 kg/m², who gradually gained weight after ECT by a total of 4 kg. After the end of ECT therapy, continuation of nutritional treatment and weekly CBT sessions were recommended. After 12 weeks of observation, the patient maintained her body weight and adhered to treatment recommendations [46].

On the other hand, researchers Duriez et al. presented a description of a patient with a chronic and severe form of AN without coexisting mental illness, in whom ECT was used, achieving only a short-term therapeutic effect. The patient received a total of 10 ECT treatments, after which no clinically significant improvement was observed, which was objectified by the Eating Disorders Inventory 2 and the Hospital Anxiety and Depression Scale (HADS). There was a slight change in the Eating Disorders Inventory score (down 22.5%), in the HADS for anxiety (down 21.4%) and in the HADS for depression (down 10%), reflecting the lack of impact of ECT on both mood and eating behavior. The patient was discharged with a normal BMI of 19.6 kg/m². Unfortunately, despite the restoration of normal body weight after hospitalization, 2 months after the end of treatment, the patient required hospitalization again due to rapid weight loss, reaching a BMI of 13.6 kg/m² with hypoglycemia, Bradycardia and hypokalaemia [47].

CONCLUSIONS

Based on Evidence-Based Medicine and the level of credibility of the available data (levels 1B, 4, no evidence of the highest value – 1A), there is currently insufficient evidence to recommend neuromodulation techniques as standard treatment for AN. The results of the studies conducted so far are not homogeneous. The limitations of the conclusions are mainly due to the small number and type of clinical trials conducted, involving small groups of subjects. There are reports of the efficacy of neuromodulation techniques in the treatment of AN, but there is a lack of systematic reviews and meta-analyses in the literature, which does not allow a reliable determination of the effectiveness of therapy. Further randomised, double-blind clinical trials are needed to assess the effectiveness of this form of treatment. However, the existing data supporting the use of rTMS, tDCS and ECT in the treatment of eating disorders are promising.

REFERENCES

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