

CASE REPORTS

Deep Brain Stimulation of the Subcallosal Cingulate in Severe Anorexia Nervosa: Perioperative Management

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Background

Anorexia nervosa (AN) is a severe psychiatric disorder marked by restrictive eating behaviors, distorted body image, and a significant risk of morbidity and mortality. A subset of AN patients are impervious to conventional therapies, including pharmacological, psychotherapeutic, and nutritional interventions. Deep brain stimulation (DBS) has come to light as a potential therapeutic strategy in treatment-resistant psychiatric conditions.

Case presentation

We report the case of a 26-year-old woman with severe, chronic AN (body mass index [BMI] 13.8 kg/m²) who was unresponsive to multiple therapeutic modalities. After a multidisciplinary evaluation, she underwent stereotactic implantation of bilateral electrodes targeting the subcallosal cingulate cortex.

Outcome

Postoperatively, the patient demonstrated progressive weight restoration, mood stabilization, and improved treatment engagement without surgical or device-related complications.

Conclusion

At 12-month follow-up, her BMI improved to 16.2 kg/m², accompanied by sustained clinical and psychosocial benefits. This case highlights the potential role of DBS in refractory AN, emphasizes the importance of careful patient selection, and contributes to the growing body of evidence supporting neuromodulation as a promising avenue for severe eating disorders.

Introduction

Anorexia nervosa (AN) is a complex psychiatric disorder marked by self-induced weight loss, a distorted body image, and crippling dread of gaining weight. It is associated with significant psychiatric and medical comorbidities and has the highest mortality rate among psychiatric illnesses, estimated to be close to 6% per decade.¹ Despite breakthroughs in treatment, such as psychotherapy, pharmacological approaches, and inpatient rehabilitation, approximately 20–30% of patients fail to respond effectively, resulting in a treatment-resistant subgroup.²

Environmental influences, neurobiological changes, and genetic predisposition interact intricately in the pathophysiology of AN. Cortico-striatal-limbic networks involved in emotional regulation have been shown to exhibit both structural and functional abnormalities in neuroimaging investigations. According to these results, AN might be a condition of abnormal brain connection that can be treated with specific neuromodulation.

Deep brain stimulation (DBS) is a neurosurgical technique that involves the implantation of electrodes to modulate dysfunctional neural circuits. Initially developed for movement disorders such as Parkinson's disease and dystonia,³ its application has since expanded to psychiatric illnesses, including obsessive-compulsive disorder (OCD) and major depressive disorder, where clinical benefit has been demonstrated.⁴ More recently, DBS has been explored as a potential therapy for refractory AN, with promising but limited evidence.⁵

Numerous brain areas such as the subcallosal cingulate (SCC), nucleus accumbens (NAc), and anterior limb of the internal capsule (ALIC), which are linked to compulsivity, mood regulation, and reward processing, have been studied as potential DBS targets in AN.⁶ A neurobiological foundation for intervention is suggested by functional MRI studies that show abnormalities in cortico-striatal-limbic pathways in AN.⁷ We present a young woman with chronic, treatment-resistant AN who underwent SCC DBS with longitudinal follow-up.

Case Presentation

A 26-year-old woman was referred to our neurology and psychiatry departments with a 12-year history of restrictive type anorexia nervosa. She initially developed restrictive eating behaviors during adolescence, manifesting as excessive exercise and persistent fear of weight gain. Various treatment approaches were initiated, including family-based therapy, individual cognitive behavioral therapy, selective serotonin reuptake inhibitors and antipsychotic augmentation. Despite these multiple interventions, her condition remained refractory to treatment. Over the past years, she has required eight hospitalizations for nutritional rehabilitation. However, she relapsed shortly after each discharge.

Upon admission to our center, her height was 153 cm, her weight was 32.5 kg, and her body mass index (BMI) was 13.8 kg/m², indicating that she was severely underweight. She exhibited bradycardia (56 beats/min) and hypotension (90/55 mmHg), and she had experienced amenorrhea for the previous eight years. Laboratory tests revealed several biochemical alterations, including hypokalemia (2.7 mmol/L), hypocalcemia (8.0 mg/dL), and hypoalbuminemia (3.2 g/dL) ([Table 1](#)). A bone mineral density assessment demonstrated a lumbar spine T-score of -3.0 indicating osteoporosis.

Table 1. Clinical and Laboratory Findings Upon Admission of Severe Treatment-Resistant Anorexia Nervosa.

Parameter	Result	Reference Range	Clinical Significance
Vital Signs			
Heart Rate	56 bpm	60–100 bpm	Bradycardia
Blood pressure	90/55 mmHg	100–120 / 60–80 mmHg	Hypotension
BMI	13.8 kg/m ²	18.5–24.9 kg/m ²	Severely underweight
Electrolytes			
Potassium (K ⁺)	2.7 mmol/L	3.5–5.0 mmol/L	Hypokalemia
Calcium (Ca ²⁺)	8.0 mg/dL	8.5–10.5 mg/dL	Hypocalcemia
Proteins			
Albumin	3.2 g/dL	3.5–5.0 g/dL	Hypoalbuminemia
Bone Tests			
Bone mineral density (lumbar spine T-score)	–3.0	> –1.0	Osteoporosis
Psychological Scores			
BDI-II score	35	0–13: minimal 14–19: mild 20–28: moderate 29–63: severe	0–13: minimal 14–19: mild 20–28: moderate 29–63: severe
Y-BOCS score	26	0–7: subclinical 8–15: mild 16–23: moderate 24–31: severe	Severe obsessive-compulsive traits related to food/body image

Y-BOCS = Yale-Brown Obsessive Compulsive Scale; BDI-II = Beck Depression Inventory-II.

Psychometric assessments indicated severe depressive symptoms (Beck Depression Inventory-II [BDI-II] score of 35) and high obsessive-compulsive traits related to food and body image (Yale-Brown Obsessive Compulsive Scale [Y-BOCS] score 26).

Given the patient's long-standing illness, multiple failed treatments and progressive medical complications, she was classified as having treatment-resistant Anorexia Nervosa. The subcallosal cingulate was targeted due to its involvement in regulating mood and intrusive thoughts, which are crucial components of the psychopathology of anorexia nervosa. Prior DBS trials in treatment-resistant depression and obsessive-compulsive disorder (OCD) have shown promise, supporting its experimental use in this case.

Following multidisciplinary evaluation by the departments of psychiatry, neurosurgery and endocrinology, as well as an ethics review, informed consent was obtained from the patient and her family.

Bilateral DBS electrodes were implanted into the subcallosal cingulate gyrus under stereotactic guidance. Postoperative computed tomography (CT) imaging confirmed accurate electrode placement, and the leads were connected to a dual-channel implantable pulse generator ([Figure 1](#)). Initial stimulation parameters were set at 3.0 V, 130 Hz and a pulse width of 90 μ s, with gradual adjustments made according to the patient's tolerance and clinical response.

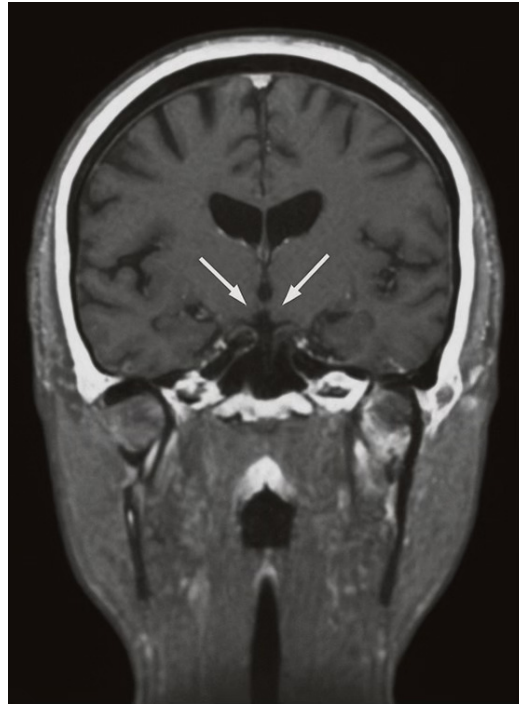


Figure 1. Postoperative CT Confirming Bilateral DBS Electrode Placement in the Subcallosal Cingulate Gyrus

Table 2. Longitudinal Clinical, Behavioral, and Dietary Outcomes Over 12 Months in Severe Treatment-Resistant Anorexia Nervosa

Timepoint	Weight (kg)	BMI (kg/m ²)	Menses	Behavior	Dietary Support
Admission	32.5	13.8	Absent	Depressed, high anxiety	Full support
3 months	34.6	14.6	Absent	Slight improvement	Partial support
6 months	36.5	15.4	Partial	Moderate improvement	Partial support
12 months	38.2	16.2	Resumed	Marked improvement, reduced intrusive thoughts.	Minimal support

Over 12 months of close follow-up, the patient demonstrated modest yet clinically meaningful improvements with weight increasing to 38.2 kg, BMI to 16.2 kg/m², and full return of menses. During the first 3 months, the patient had transitioned from full to partial nutritional support. By 6 months, she had experienced a partial return of menses, and reported improved mood stability, reduction in intrusive food related thoughts, and enhanced social engagement. Although partial nutritional support was still needed as her restrictive eating behavior persisted and ongoing dietary supervision was necessary, by the 12-month mark she had transitioned to requiring minimal nutritional support ([Table 2](#)). No surgical or stimulation-related complications had occurred, therefore supporting the safety of this intervention. While DBS is not a cure, it shows promise in its role as a potential adjunct, yet larger studies are needed to optimize parameters and target selection. The ethical review processes, involving multidisciplinary input and informed consent, underscore the careful consideration given to this experimental approach.

Discussion

Deep brain stimulation (DBS) represents a promising experimental intervention for treatment-resistant anorexia nervosa (AN). Early studies indicate that DBS can modulate dysfunctional cortico-striatal-limbic circuits, improving weight restoration, mood, compulsive behaviors, and overall quality of life.⁸ Phase I trials of subcallosal cingulate (SCC) DBS in chronic AN have demonstrated improvements in affective symptoms and significant weight gain in a subset of patients,⁹ while nucleus accumbens (NAc) DBS has shown reductions in compulsivity and gradual weight restoration.¹⁰ Although patient responses are variable, these findings highlight the therapeutic potential of DBS in targeting neural circuits involved in reward processing, executive control, and emotional regulation.¹¹

Neuroimaging studies reveal hyperactivity and altered connectivity in the SCC, insula, and striatum, regions implicated in reward processing, compulsivity, and affective regulation.¹² By modulating these circuits, DBS may reduce intrusive food-related thoughts, improve mood, and facilitate weight gain. Advances in connectomic-guided targeting and closed-loop DBS may optimize outcomes by individualizing stimulation parameters based on neural signatures.¹³

The present case adds to the literature by providing comprehensive longitudinal follow-up over 12 months, including weight, BMI, menses resumption, behavioral changes, and dietary support, highlighting clinically meaningful improvements. Importantly, no surgical or stimulation-related complications occurred which demonstrates the safety of DBS in a severely malnourished patient. These strengths support the feasibility of DBS as an experimental intervention in carefully selected patients with severe, treatment-resistant AN.

Nevertheless, several gaps remain. The patient's restrictive behaviors persisted, illustrating that DBS alone may not fully normalize eating patterns. Long-term efficacy beyond 12 months remains unknown, and broader studies are needed to determine predictors of response, optimal stimulation targets, and standardized protocols. Furthermore, the single-case nature of this report limits generalizability. Ethical considerations and careful patient selection remain critical given the invasiveness of the procedure and the vulnerability of this population.¹⁴ DBS demonstrates potential as a therapeutic option for severe, treatment resistant AN, with measurable improvements in weight, affective symptoms, and quality of life. While our findings support its safety and feasibility, larger controlled trials are needed to address remaining gaps, define standardized protocols, and establish the durability of response.

Conclusion

Deep brain stimulation constitutes a promising investigational intervention approach for carefully selected patients with severe, treatment-resistant anorexia nervosa. In this case, bilateral stimulation of subcallosal cingulate region was associated with modest weight gain, resumption of menses, and improvements in mood and compulsive symptoms. Although restrictive eating behaviors persisted, the clinical improvements observed suggest a therapeutic effect beyond appetite regulation and underscore the importance of sustained multidisciplinary care. While DBS remains an experimental therapy, its modulatory effects on dysfunctional neural circuits indicates a potential adjunctive treatment in refractory cases. Further studies incorporating refined targeting strategies and biomarker-guided approaches, is necessary to establish long-term efficacy and safety.

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Ethical Considerations

Written informed consent was obtained from the patient for publication of this case report and accompanying images. The procedure and publication were reviewed and approved by the institutional ethics committee.

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