Heliyon 5 (2019) e01862

Contents lists available at ScienceDirect

Heliyon

journal homepage: www.heliyon.com

Height and weight changes after deep brain stimulation in patients with Parkinson disease: role of clinical subtypes

Hesham Abboud [a,b,*], Dennys Reyes [c], Gencer Genc [a], Anwar Ahmed [a], Michal Gostkowski [a], Hubert H. Fernandez [a]

[a] Center for Neurological Restoration, Cleveland Clinic, 9500 Euclid Avenue, Desk U2, Cleveland, OH, 44195, USA
[b] Parkinson's and Movement Disorders Center, University Hospitals of Cleveland, Case Western Reserve University School of Medicine, 11100 Euclid Avenue, Cleveland, OH, 44106, USA
[c] Neurology Department, Cleveland Clinic Florida, 2950 Cleveland Clinic Blvd Fl 3, Weston, FL, 33331, USA

ARTICLE INFO

Keywords:
Neurology
Parkinson's disease
Deep brain stimulation
Body mass index
Height
Weight

ABSTRACT

Increased body mass index (BMI) after deep brain stimulation (DBS) in Parkinson's disease (PD) has been repeatedly reported in literature. However, little is known about the effect of PD clinical subtypes on weight and height changes after DBS. We aimed to study the differential effect of tremor-predominant versus hypokinetic-rigid disease on weight and height changes after DBS. Methodology: we chart-reviewed PD patients who underwent DBS at our center from 2006 to 2011. Weight and height data were obtained at the pre-surgical period, at 1-year post-surgery, and at the latest available follow-up (LAF). Results: There were 130 patients in the dataset (70% male, mean age 63 + /-9.1). Eighty-eight patients had available data at 1-year post-DBS or longer. Mean LAF was 4.36 + /-1.64 years. A BMI increment by 1 Kg/m2 or more was noticed in 35% after 1-year. Increased height (1cm-or-more) was seen in 24% of patients at 1-year. At 1-year post-DBS, 41.8% of patients with hypokinetic-rigid subtype increased in height compared to only 14.2% in the tremor-predominant group (OR 4.3, 95 % CI 1.3167-14.1246, P=0.015). There was no correlation between PD subtype and weight change after DBS. Conclusion: This study confirms BMI increase after DBS in PD patients and reports a novel finding of increased height after DBS in patients with hypokinetic-rigid PD. This might be secondary to improved axial rigidity following DBS. Resolution of tremor is probably unrelated to the increase in body weight after surgery since weight gain did not differ between patients with tremor-predominant and those with hypokinetic-rigid subtype.

1. Introduction

Deep Brain Stimulation (DBS) has been established as a superior therapeutic option for advanced Parkinson's disease (PD) [1]. Increased body mass index (BMI) after DBS has been repeatedly reported in literature and several theories have been proposed to explain this interesting finding [2]. One possible explanation suggests that patients gain weight after DBS secondary to a reduction in their metabolic rate after resolution of tremor and/or dyskinesia [3, 4]. Others suggested that DBS might have a direct stimulation effect on appetite centers [5]. However, the differential effect of PD clinical subtypes on BMI changes after DBS is not clear. Do patients with tremor-predominant PD gain more weight after DBS compared to those with hypokinetic-rigid disease as a factor of tremor resolution? Can DBS affect patients' standing height as a factor of improved axial rigidity and posture? Is the effect on height different in hypokinetic-rigid patients compared to those with tremor-predominant disease? How does the effect on height influence BMI changes after DBS? We sought to answer those questions and explore their clinical implications on PD patients undergoing DBS.

2. Methods

We chart-reviewed PD patients who underwent DBS at the Cleveland Clinic between 2006 and 2011 with complete data. Weight and height data were obtained at the latest preoperative evaluation, at 1-year post surgery, and at the latest available follow-up (LAF). Weight and height for all patients were measured by the intake nurse at our movement disorders center using the same scales. Patients were weighed in the on-state while fully dressed and were asked to stand as erect as possible during height measurement per height measuring standards. We classified patients into tremor-predominant PD versus hypokinetic-rigid PD based on the predominant symptoms and the tremor versus

* Corresponding author.
E-mail address: Hesham.abboud@uhhospitals.org (H. Abboud).

https://doi.org/10.1016/j.heliyon.2019.e01862
Received 20 January 2019; Received in revised form 27 February 2019; Accepted 28 May 2019

Table 1
Baseline demographics and clinical data of the tremor-predominant versus hypokinetic-rigid groups.

	Tremor-predominant PD	Hypokinetic-rigid
Male %	85%	69%
Average age	64.03 years	62.5 years
Average disease duration at surgery	8.5 years	11.2 years
Average preoperative ON UPDRSIII	23.98	18.09
Average preoperative LEDD	925.6 mg	1151.2 mg
Dyskinesia presence %	17.8%	54.5%
Average Preoperative weight	91.7 kg	79.6 kg
Average Preoperative height	176.6 cm	171.1 cm
Average Preoperative BMI	29.3 kg/cm^2	27 kg/cm^2

bradykinesia/rigidity subscores on the unified Parkinson disease rating scale motor sub-scale (UPDRSIII/MDS-UPDRSIII). We defined tremor predominant PD as a score of 2 or more on the tremor subscore with a score of 1 or less in the bradykinesia and rigidity subscores. We defined hypokinetic rigid PD as a score of 2 or more on the rigidity and bradykinesia subscores with a score of 1 or less on the tremor subscore. All other patients were considered mixed. We compared patients who had weight or height gain after DBS in the two groups and tested significance with the chi-square test. A P value less than 0.05 was considered significant. Patients with mixed phenotype were not included in the comparative analysis.

3. Results

There were 130 patients in the dataset (70% male, mean age 63+/-9.1). Most patients were implanted in the subthalamic nucleus (STN) = 124 (95.3%). Eighty-eight patients had available weight and height data at 1-year post-DBS or longer. Mean LAF was 4.36+/-1.64 years post-DBS at the time of data analysis. The average preoperative weight was 83.3 kg (SD: 18.4), height was 172.8 cm (SD: 10.4), and BMI was 27.8 kg/M^2 (SD: 5.5). At one year post surgery, BMI increased in 55% of the patients and the increment was higher than 1 kg/M^2 in 35% and higher than 2 kg/M^2 in 17%. An increase in standing height occurred in 33% of the patients at 1 year post-surgery and the increment was higher than 1 cm in 24% and higher than 2 cm in 15%. The average postoperative weight at 1-year was 84.8 kg (SD: 18.6) higher than the preoperative average by 1.5 kg. The average postoperative standing height at 1-year was 173.09 cm (SD: 9.6) higher than the preoperative average by 0.29 centimeter. The average postoperative BMI at 1-year was 28.2 kg/M^2 (SD: 5.3) higher than the preoperative average by 0.4 kg/M^2.

At LAF, 54% of the patients continued to have a BMI that was higher than their preoperative value but 45% of the patients experienced a decrease in their BMI as the disease progressed abolishing the overall difference in the average BMI between the preoperative and the postoperative periods of the cohort as a whole (average postoperative weight and BMI at LAF of 83.4 kg and 27.8 kg/M^2 respectively). Twenty three patients (48%) out of the original 48 with postoperative increased weight maintained their weight gain at the LAF while the remaining patients lost weight with time. Sixteen patients (40%) out of the original 40 with postoperative stable or decreased weight at 1-year post surgery lost more weight at the LAF. Increased standing height compared to preoperative value was still seen in 23% of patients at LAF and the increment was higher than 1 cm in 19% and higher than 2 cm in 14%. However, 28% of the patients experienced reduction in their standing height at LAF bringing down the average of the entire cohort at LAF to the same value of the preoperative average (172.8 cm, SD: 9.4).

Based on clinical symptoms and UPDRSIII/MDS-UPDRSIII sub-scores, 28 patients were classified as tremor-predominant PD and 55 were classified as hpokinetic-rigid PD, while 5 patients could not be classified into either group. Table 1 summarizes the demographic and clinical data of both groups. At 1-year post-DBS, 41.8% of patients with hypokinetic-rigid subtype increased in standing height compared to only 14.2% of the tremor-predominant patients (OR 4.3, 95 % CI 1.3167 to 14.1246, P = 0.015). This difference was not maintained at LAF. There was no correlation between PD clinical subtype and weight change at 1-year post DBS or at the LAF.

4. Discussion

Our results suggest that the effect of weight gain after DBS on patients' BMI may be mitigated in some patients by a concomitant increase in standing height although the effect on weight remains more pronounced. Fourteen patients had an increase in both weight and height at one year post-surgery accounting for 29% of all patients who had increased BMI. This means that in a subset of PD patients postoperative increase in weight is partially offset by concomitant increase in height mitigating the net effect on BMI. There was no correlation between the rate of weight gain and the increase in height. The increase in height seems to occur more frequently in patients with hypokinetic-rigid parkinsonism which suggests that DBS may improve axial rigidity. Although several studies have shown that DBS can improve axial dystonia in camptocormic patients based on angular improvement [6, 7], our study strongly suggests that DBS might result in actual improvement in axial rigidity as represented by the increase in standing height 1-year post-surgery even in the absence of camptocormia (i.e: in patients with average stooping due to axial rigidity in absence of full-blown truncal dystonia). This observation is of particular importance as it suggests that the limited effect of DBS on axial symptoms [8, 9] might not be absolute. In addition, our study suggests that tremor resolution with DBS is unlikely a major factor in the observed weight gain after surgery since there was no difference in weight gain between tremor-predominant and hypokinetic-rigid patients. Other factors may be implicated in post-DBS weight gain like decreased dyskinesia or appetite simulation effect. These factors will require further studies in the future. BMI changes after DBS is an important topic since preoperative BMI may influence DBS outcomes as reported previously by us and other authors [10, 11]. As expected, the effect of DBS on weight and height was most pronounced at 1-year post surgery then it became less pronounced at the LAF due to disease progression although a subset of patients maintained their weight and height gain several years postoperatively. Weight loss in advanced PD has been frequently described and is often multifactorial with proposed contributions from dysphagia, anorexia, hypomotility of the gastrointestinal tract, depression, and increased energy expenditure secondary to tremor and dyskinesia [12]. One important limitation to our study is that the height and weight changes after DBS were not compared to a non-DBS control group. Also since height measuring was done as part of routine intake rather than a structured research protocol, there is a chance that some of the smaller variations in height were technical in nature.

5. Conclusion

In summary, our findings confirm previous reports of weight gain after DBS and highlight a novel finding of increased height in patients with hypokinetic-rigid PD which might mitigate DBS effect on BMI by virtue of increased denominator. These effects become less prominent as the disease progresses leading to weight loss and worsened posture. In addition, tremor resolution is probably not a major factor in postoperative weight gain. The effect of perioperative weight and height changes on different DBS outcomes is a potential area for future research.

Declarations

Author contribution statement

Hesham Abboud: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Contributed

reagents, materials, analysis tools or data; Wrote the paper.

Dennys Reyes: Conceived and designed the experiments; Analyzed and interpreted the data.

Gencer Genc, Anwar Ahmed, Michael Gostkowki, Hubert Fernandez: Performed the experiments; Contributed reagents, materials, analysis tools or data.

Funding statement

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Competing interest statement

The authors declare the following conflict of interests:

Hesham Abboud; Dr. Abboud is a member of the speaker bureau of Biogen and Genentech.

Hubert H. Fernandez; Dr. Fernandez Has received honoraria from Advanced Health Media, Cleveland Clinic CME, Medical Communications Media, Movement Disorders Society, Vindico MedicalEducation, as a speaker in CME events. He has also received honoraria from Ipsen, Merz Pharmaceuticals, Pfizer, Teva Neuroscience, Zambon Pharmaceuticals, as a speaker and/or consultant.

Dr. Fernandez has received personal compensation for serving as Co-Medical Editor of the Movement Disorders Society Website.

Dr. Fernandez Has received royalty payments from Demos Publishing and Manson LTD for serving as a book author/editor.

Dr. Fernandez has received research support from Abbott, Acadia, Biotie Therapeutics, EMD-Serono, Huntington Study Group, Merck, Michael J. Fox Foundation, Movement Disorders Society, National, Parkinson Foundation, NIH/NINDS, Novartis, Parkinson Study Group, Synosia, Teva, but has no owner interest in any pharmaceutical company

Additional information

No additional information is available for this paper.

References

[1] J.M. Bronstein, M. Tagliati, R.A. Alterman, A.M. Lozano, J. Volkmann, A. Stefani, F.B. Horak, M.S. Okun, K.D. Foote, P. Krack, R. Pahwa, J.M. Henderson, M.I. Hariz, R.A. Bakay, A. Rezai, W.J. Marks Jr., E. Moro, J.L. Vitek, F.M. Weaver, R.E. Gross, M.R. DeLong, Deep brain stimulation for Parkinson disease an expert consensus and review of key issues, Arch. Neurol. 68 (2011) 165–171.

[2] K.A. Mills, R. Scherzer, P.A. Starr, J.L. Ostrem, Weight change after globus pallidus internus or subthalamic nucleus deep brain stimulation in Parkinson's disease and dystonia, Stereotact. Funct. Neurosurg. 90 (6) (2012) 386–393. Epub 2012 Aug 23. PMID:22922491.

[3] P. Sauleau, E. Leray, T. Rouaud, S. Drapier, D. Drapier, S. Blanchard, G. Drillet, J. Péron, M. Vérin, Comparison of weight gain and energy intake after subthalamic versus pallidal stimulation in Parkinson's disease, Mov. Disord. 24 (14) (2009 Oct 30) 2149–2155.

[4] M. Barichella, A.M. Marczewska, C. Mariani, A. Landi, A. Vairo, G. Pezzoli, Body weight gain rate in patients with Parkinson's disease and deep brain stimulation, Mov. Disord. 18 (11) (2003) 1337–1340.

[5] E. Markaki, J. Ellul, Z. Kefalopoulou, E. Trachani, A. Theodoropoulou, V. Kyriazopoulou, C. Constantoyannis, The role of ghrelin, neuropeptide Y and leptin peptides in weight gain after deep brain stimulation for Parkinson's disease, Stereotact. Funct. Neurosurg. 90 (2) (2012) 104–112.

[6] W.J. Schulz-Schaeffer, N.G. Margraf, S. Munser, A. Wrede, C. Buhmann, G. Deuschl, C. Oehlwein, Effect of neurostimulation on camptocormia in Parkinson's disease depends on symptom duration, Mov. Disord. 30 (3) (2015 Mar) 368–372. Epub 2015 Feb 12.

[7] A. Umemura, Y. Oka, K. Ohkita, T. Yamawaki, K. Yamada, Effect of subthalamic deep brain stimulation on postural abnormality in Parkinson disease, J. Neurosurg. 112 (6) (2010 Jun) 1283–1288.

[8] J.E. Visser, J.H. Allum, M.G. Carpenter, R.A. Esselink, P. Limousin-Dowsey, F. Honegger, G.F. Borm, B.R. Bloem, Effect of subthalamic nucleus deep brain stimulation on axial motor control and protective arm responses in Parkinson's disease, Neuroscience 157 (4) (2008 Dec 10) 798–812.

[9] R.J. St George, J.G. Nutt, K.J. Burchiel, F.B. Horak, A meta-regression of the long-term effects of deep brain stimulation on balance and gait in PD, Neurology 75 (14) (2010 Oct 5) 1292–1299.

[10] H. Abboud, G. Genc, N.R. Thompson, et al., "Predictors of functional and quality of life outcomes following deep brain stimulation surgery in Parkinson's disease patients: disease, patient, and surgical factors, Parkinson's Dis. 2017 (2017). Article ID 5609163, 8 pages.

[11] A. Rouillé, S. Derrey, R. Lefaucheur, A. Borden, D. Fetter, M. Jan, D. Maltête, Pre-operative obesity may influence subthalamic stimulation outcome in Parkinson's disease, J. Neurol. Sci. 359 (1-2) (2015 Dec 15) 260–265.

[12] K. Kashihara, Weight loss in Parkinson's disease, J. Neurol. 253 (Suppl 7) (2006 Dec) VII38–41.