

Psychopathology and hormonal disturbances in eating disorders

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Abstract

Background: Our aim was to study the relationship between hormonal disturbances and psychopathology in Eating Disorders (ED).

Methods: Forty-nine women diagnosed as Eating Disorders according to DSM-IV were subjected to control plasma levels of TSH, FT3, FT4, LH, FSH, 17beta-estradiol, prolactin, cortisol, DHEAS, GH and IGF-1. They were also administered by SCL-90R, BAT, DES II questionnaires.

We applied multivariate regression models.

Results: Our results highlight a statistically significant relation between LH, FSH and prolactin decreased levels, mood and thought disturbances (subscales 3, 5, 7, 8 and 9 of SCL-9or) which are associated to Body Attitude (BAT total scale) and Dissociative Experiences (DES II total scale).

Conclusions: Decreased sexual hormones levels could have a role in ED psychological disturbances, not inquired yet.

Key words: eating disorders, hormonal disturbances, neuropsychological functions

Introduction

Neuroendocrine alterations have been studied in Eating Disorders, both in Anorexia Nervosa (AN) [1] and in Bulimia Nervosa (BN) [2] patients. According to Krassas [1] Gonadotrophins (LH, FSH) and Estrogens levels are decreased in AN patients. Decreased gonadotrophins levels have also been found in BN, mostly in women with the lowest weight [3]. It has also been observed that Growth Hormone (GH) is elevated and that Insulin Growth Factor-1 (IGF-1) is significantly decreased in AN patients (1), while normal GH levels [4] and relatively decreased IGF-1 levels [5] have been observed in BN patients. With regard to thyroid hormone levels, Krassas [1] has found that Thyrotropin Stimulating Hormone (TSH) and Thyroxine (T4) levels are usually normal, while Triiodothyronine (T3) is lower. Relating to Hypothalamic-Pituitary-Adrenal Axis, higher Cortisol levels and normal Corticotropin (ACTH) levels have been found in AN patients [1]. Levels of Cortisol and ACTH have, instead, been found to be normal in BN patients (6). Galderisi et al. [7] have observed that Dehydroepiandrosterone and its Sulfate Metabolite (DHEA and DHEAS) levels were increased in ED patients. An alteration of Prolactin was also found in ED patients although data are conflicting: a prolactin increase has been reported by Mecklenburg et al. $[8\mu]$, while a prolactin decrease was observed by Rolla et al. [9)

Some neuropsychological studies have examined cognitive functions in ED patients [10]. Particularly, Attention has been investigated [11] and deficits in the areas of Vigilance and Selective Attention have been observed [12]. Memory has been studied by Bradley et al. (1997) and a selective Memory bias for words related to eating, body shape and weight has been found [13]. Tchanturia et al. [14] studied thinking styles such as cognitive rigidity/flexibility measured as setshifting and in AN patients and they showed setshifting deficits [15]. There are only a few researchers who are trying to correlate cognitive disturbances with hormonal alterations [7]. In particular, Galderisi et al. [7] have observed that DHEA and DHEAS were increased and positively correlated with accuracy on the executive task, while cortisol was positively correlated with speed of noneffortful learning. Other works have



studied the role of vasopressin, that improves memory [16] and that is increased in AN patients [17] and the role of oxytocin, that reduces memory consolidation and limits its recall [7].

Our study aims to relate hormonal disturbances and Eating disorders psychopathology, which implicate not only cognitive functions but also emotional processes. So, we have explored Attitude towards the Body (Body Attitude Test, BAT), frequency of Dissociative Experiences (Dissociative Experiences Scale II, DES II), mood alterations, such as depression and anxiety, and thought styles, such as paranoid ideation and psychoticism (subscales of Symptom Checklist-90 Revised, SCL-90-R) and then we have correlated them with hormonal alterations.

Methods

Participants

Our clinical sample was composed of voluntary patients admitted to the treatment at the University Hospital "A. Gemelli", Rome, Italy, from May 2005 to May 2006. The sample was made of forty-nine women, predominantly Caucasian. These 49 patients had a mean age of 24.5 ± 5.3 years (mean \pm SD) (range, 16-35 years) and a mean BMI of 18.3 ± 3.1 kg/m² (range, 11.7-25.3 kg/m²). All patients met the Diagnostic and Statistical Manual-IV (DSM-IV) criteria for AN (19 patients) and for BN (30 patients). Age of onset in this sample was 18.1 ± 4.3 years, and mean duration of illness before admission to our Day Hospital was 2.7 ± 0.9 years.

Procedure

Patients underwent clinical examination, dietist evaluation and psychological assessment. The following hormones were measured: TSH, FT3, FT4, LH, FSH, 17-beta-estradiol, prolactin, cortisol, DHEAS, GH and IGF-1. Blood samples were taken in all patients in the morning, the seventh day of menstrual cycle or the day supposed equivalent in women in amenorrea.

Mood alterations and thinking styles were measured using the Symptom Checklist-Revised (SCL-90-R). This instrument has nine subscales: Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychoticism.

The Body Attitude Test (BAT) was used to evaluate own body attitude. This instrument has three subscales: "negative appreciation of body size", "lack of familiarity with one's own body", "general body dissatisfaction".

The Dissociative Experiences Scale II (DES II)

was used to measure the frequency of dissociative experiences. It has three subscales: amnestic dissociation, absorption and imaginative involvement, depersonalization and derealization.

Statistical Analysis

Frequencies and means with Standard Deviation (m \pm SD) were used to describe the sample.

Multivariate regression models were realized to evaluate outcomes of GSI scale and its nine subscales (SCL1, SCL2, SCL3, SCL4, SCL5, SCL6, SCL7, SCL8, SCL9). In every model hormones levels, BMI, age (years), results of total scales of DES II and BAT were used as predictive variables (covariates). Regression models were realized using Stepwise method with backward elimination. The significance level of every analysis was set equal to p<0.05. Data were analysed using SPSS 12.00 software for Windows.

Results

These 49 patients had a mean age of 24.5 ± 5.3 years (mean \pm SD) (range, 16-35 years) and a mean BMI of 18.3 ± 3.1 kg/m² (range, 11.7-25.3 kg/m²).

Table 1 presents results of the Mann Whitney test's application to the AN patients group and to BN plus AN binge-purging patients group. We observe that the differences between mean values of the investigated variables referring to the two groups are not significant, with the exception of the BMI (AN = 16.87; SD = 2.68; BN = 20.60; SD = 2.36) and the ESTR 17 (AN = 47.81; SD = 54.90; BN = 116.27; SD = 98.45.36) being p<0.001 and p=0.007, respectively.

Table 2 shows that the BMI groups are significant different respect to FT3 (p=0.001), LH (p=0.005), 17-beta-estradiol (p=0.010), IGF-1 (p=0.020) and cortisol levels (p=0.013). A statistically significant difference between the three subgroups is also observed by considering the mean values of the SCL2 subscales (p=0.020).

Multivariate linear regression analysis referring to outcome "GSI" highlights that total scales of the BAT (β =0.442, p=0.01) and of the DES II (β =0.349, p=0.047) are significant, among predictive variables. The R² for this model is 0.61.

Significant variables referring to outcome SCL1 were not observed.

The Obsession scale (SCL2) is associated in a significant way with the age (β =0.448, p=0.013) and the total scale of DES II (β =0.347, p=0.047) (R^2 for the model 0.638).

The Interpersonal Sensitivity scale (SCL3) is associated in a significant way with FT3 (β =0.793, p = 0.003), with the total scale of DES II (β =0.560,



p = 0.002), with FT4 (β = -0.605, p = 0.011) and with FSH (β = -0.340, p = 0.045) ($R^2 = 0.706$).

The Depression scale (SCL4) is associated with the age (β =0.464, p= 0.009) and with the total scale of BAT (β =0.482, p= 0.007) (R² = 0.55).

The Anxiety scale (SCL5) is associated with the age (β =0.738, p= 0.000), with the total scale of BAT (β =0.422, p= 0.003) and with PRL (Prolactin) (β = -0.423, p= 0.005) (R² = 0.739).

The Hostility scale (SCL6) is associated with the total scales of BAT (β =0.598, p=< 0.001) and of DES II (β =0.330, p= 0.028) (R^2 = 0.715).

The Phobic Anxiety scale (SCL7) is associated with the age (β =0.471, p= 0.016), with FT3 (β =0.485, p= 0.025), with TSH (β =-0.382, p= 0.032) and with PRL (β =-0.712, p= 0.003) (R^2 = 0.604).

The Paranoid Ideation scale (SCL8) is associated with the total scale of DES II (β =0.498, p= 0.010)

and with LH (β = -0.439, p= 0.021) (R^2 = 0.536).

The Psychoticism scale (SCL9) is associated with FSH (β =0.324, p= 0.025), with the total scale of BAT (β =0.480, p= 0.001), with the total scale of DES II (β =0.344, p=0.021), with LH (β =-0.342, p= 0.015) and with PRL (β =-0.308, p= 0.028). The correlation coefficient for the model, R², is 0.814.

Discussion

Our data highlights a relationship between hormones, particurarly LH, FSH and Prolactin, and psychopathology in ED. In substance, all the scales of SCL-90-R are involved, including mood and its alterations, such as depression and anxiety, and also thinking styles, such as paranoid ideation, as well as the global functioning of personality. Typical psychopathological aspects of ED, such as Body Attitude and Dissociative Experiences (BAT and DES II) are also involved. In short, decreased LH levels are correlated with the increased paranoid ideation (SCL8), which is correlated to the total scale of DES II (dissociative experiences). Decreased LH levels are also correlated with increased psychoticism (SCL9), which is positively correlated to the total scales of DES II and BAT (body image disturbance).

Decreased FSH levels are correlated with increased interpersonal sensivity (SCL3), which is

Table 1. Mann Whitney test results referring to BMI, hormones, and SCL90-R scales for both the AN and the BN group.

Variables	Mann-Whitney U	p
BMI (Kg/m²)	69,000	<,001*
TSH (µUĬ/MĹ)	241,500	0,540
FT3 (PG/ML)	220,000	0,209
FT4 (PG/ML	213,000	0,220
LH (MUI /ML)	128,500	0,112
FSH (MUI /ML)	187,500	0,901
17 ESTR (PG/ML)	74,000	0,007*
GH (NG/ML)	144,500	0,933
IGF-1 (NG/ML)	104,000	0,454
PRL (NG/ML)	185,000	0,481
CORT (NG/ML)	177,000	0,274
DEA-S (NG/ML)	123,000	0,239
SCL 1	274,000	0,745
SCL 2	269,000	0,669
SCL 3	262,000	0,439
SCL 4	206,500	0,089
SCL 5	270,500	0,691
SCL 6	244,000	0,349
SCL 7	288,500	0,976
SCL 8	243,500	0,344
SCL 9	270,500	0,691
GSI	264,500	0,604

Table 2. Kruskal Wallis test results referring to hormones and SCL90-R scales for the three BMI groups.

Variables	Chi-Square	p
TSH (µUI/ML) FT3 (PG/ML) FT4 (PG/ML) FT4 (PG/ML LH (MUI /ML) FSH (MUI /ML) 17_ESTR (PG/ML) GH (NG/ML) IGF-1 (NG/ML) PRL (NG/ML) CORT (NG/ML)	0,367 13,434 5,434 10,495 3,527 9,242 0,030 7,784 0,332 8,755	0,833 0,001* 0,066 0,005* 0,171 0,010* 0,985 0,020* 0,847 0,013*
DEA-S (NG/ML)	2,709	0,354

correlated to the total scale of DES II.

Decreased prolactin levels are correlated with increased anxiety (SCL5) and phobic anxiety (SCL7).

Although the test sample was not very large, it is believed that the results are statistically significant. Our hypothesis is that these hormones have a protective role on the equilibrium of some mental functions, suggesting that their critical reduction could cause psychopathological developments in ED patients.

References

- 1) Krassas GE. Endocrine abnormalities in Anorexia Nervosa. Pediatr Endocrinol Rev 2003;1(1):46-54.
- 2) Levine RL. Endocrine aspects of eating disorders in adolescents. Adolesc Med 2002:13(1):129-43, vii.
- 3) Weltzin TE, Cameron J, Berga S, Kaye WH. Prediction of reproductive status in women with bulimia nervosa by past high weight. Am. J. Psychiatry 1994;151:136-8.
- 4) Coiro V, Capretti L, Volpi R, D'Amato L, Marchesi C, De Ferri A, Rossi G, Bianconi L, Marcato
- 5) A, Chiodera P. Abnormal growth hormone and cortisol, but non thyroid-stimulating hormone, responses to an intravenous glucose tolerance test in normal weight, bulimic women. Neuropsychobiology 199;23
- 6) Levy BA, Malarkey WB. Growth hormone and somatomedin-C in bulimia. Psychoneuroendocrinology 1988;13:359-362.
- 7) Coiro V, Volpi R, Marchesi C, Capretti L, Speroni G, Rossi G, Caffari G, De Ferri A, Marcato A, Chiodera P. Abnormal growth hormone and cortisol, but non thyroid-stimulating hormone, responses to an intravenous glucose tolerance test in normal



weight, bulimic women. Psychoneuroendocrinology 1992;7:639-45.

- 8) 6. Galderisi S, Mucci A, Monteleone P, Sorrentino D, Piegari G, May M. Neurocognitive functioning in subjects with eating disorders: the influence of neuroactive steroids. Biol Psychiatry 2003;53(10):921-7.
- 9) Mecklenburg RS, Loriaux DL, Thompson RH, Andersen AE, Lipsett MB. Hypothalamic dysfunction in patients with anorexia nervosa. Medicine 1974;53:147-59.
- 10) Rolla M,Andreoni A, Belliti D, De Vescovi S, Mariani G. Effects of dopamine antagonists (domperidone, metoclopramide) on the release of the adenohypophyseal hormones in patients at different staged of anorexia nervosa. In: Venturoli S, Givens R, Flamigni C. (Eds.) Adolescence in Females. Chicago: Year Book Medical Publishers Inc., 1985:475-479.
- 11) Duchesne,M. Mattos P, Fontanelle L.F, Veiga H, Rizo L, Appolinario JC. Neuropsychology of eating disorders: a systematic review of the literature. Revista Brasileira de Psiquiatria 2004;26:2.
- 12) Laessle RG, Fischer M, Fichter MM, Pirke KM, Krieg JC. Cortisol levels and vigilance in eating disorder patients. Psychoneuroendocrinology 1992;17:475-84.

- 13) Rieger E, Scotte DE, Touyz SW, Beumont PJ, Griffiths R, Russell J. Attentional biases in eating disorders: a visual probe detection procedure. Int J Eat Disord 1998;123:199-205.
- 14) Sebastian SB, Williamson DA, Blouin DC. Memory bias for fatness stimuli in the eating disorders. Cogn Ther and Res 1996;20:275-86.
- 15) Tchanturia K, Iain CC, Robin M, Treasure J. Neuropsychological studies in Anorexia Nervosa. Int J Eat Disord 2005;37:S72-6.
- 16) Steinglass JE, Walsh BT, Stern Y. Set shifting deficit in anorexia nervosa. J Int Neuropsychol Soc 2006;12(3):431-5.
- 17) Heinrichs M, Meinlschmidt G, Wippich W, Ehlert U, Hellhammer DH. Selective amnesic effects of oxytocin on human memory. Physiology & Behavior 2004;83:31-8.
- 18) Frank GK, Kaye WH, Altemus M, Greeno CG. CSF oxytocin and vasopressin levels after recovery from bulimia nervosa and anorexia nervosa, bulimic subtype.
- Biol Psych 2000;48(4):315-8.