www.alcrut.com

International Journal of Clinical Endocrinology and Disorders

Research article | International Journal of Clinical Endocrinology and Disorders | Open Access

Hyperprolactinemia and Body Weight: Endocrine Disorders among Women of Reproductive Age

Saritha Garrepalli

Department of Pharmaceutical Sciences, India

***Corresponding Author**: Saritha Garrepalli, Department of Pharmaceutical Sciences, India

Received Date: January 02, 2022; **Accepted Date:** January 12, 2022; **Published Date:** January 14, 2022

Citation: Saritha Garrepalli, Hyperprolactinemia and Body Weight: Endocrine Disorders among Women of Reproductive Age, Int. Journal of Endocrinology and Disorders

Abstract:
Prolactin (Prl) is a single-chain polypeptide involved in several actions, such as lactation, luteal function, reproduction, appetite, suppression of fertility, homeostasis, osmotic balance, immunity, and coagulation.Prl receptor (Prl-R) gene expression has already been described in adipose tissue, and an increase in this expression during lactation has been documented in rats and humans. Prl-R-deficient mice have shown reduced abdominal fat and leptin concentration compared to controls.

Keywords: Body weight; hyperprolactinemia; obesity; overweight; prolactinoma

Introduction

Prolactin(Prl) is a single- chain polypeptide involved in several conduct, similar as lactation, luteal function, reduplication, appetite, repression of fertility, homeostasis, bibulous balance, impunity, and coagulation(,2).

Prl receptor(Prl- R) gene expression has formerly been described in adipose towel, and an increase in this expression during lactation has been proved in rats and humans(,4). Prl- R-deficient mice have shown reduced abdominal fat and leptin attention compared to controls(5). In hamsters, inhibition of Prl stashing by bromocriptine has led to a reduction in fat deposit, without reducing food input or body weight(2). In mortal adipose towel, Prl suppresses lipid storehouse and adipokine release and also has a paracrine/ autocrine function in relation to adiponectin by binding to its receptors(6- 8). Prl increases the expression of adiponectin receptors, a hormone that's buried by adipose towel, adding insulin perceptivity(9). In humans, outside gestation, Prl stashing is altered by adding body weight in grown-ups and children. still, no molecular base has been set up which links Prl with adding body fat, weight and appetite, although some data suggest the involvement of Prl with leptin(10).

In recent times, some studies have shown an increased frequence of rotundity in cases with prolactinomas(,12), while others have reported weight loss(12- 15) or weight gain after treatment of hyperprolactinemia(,17). still, there are no studies with internal health diseases thannon-obese children(6).

These data

probing variations in body weight associated with different degrees of hyperprolactinemia or their relation to the cause of hormone hypersecretion.

This study aimed to estimate the frequence of rotundity and fat in cases with hyperprolactinemia, whether or not associated with prolactinomas, and to relate Prl situations with body mass indicator(BMI) in hyperprolactinemia of colorful causes.

Accoutrements and styles

The medical records of successive treatment-naive cases with hyperprolactinemia attending the Center for Neuroendocrinology at Universidade Federal de Ciências da Saúde de Porto Alegre(UFCSPA), Brazil, between 2000 and 2010 were retrospectively reviewed. The study was approved by the original Ethics Committee(Institutional Review Board-original) and was conducted in agreement with the vittles of the protestation of Helsinki.

Grounded on medical record review, 115 cases with prolactinoma and 82 cases with idiopathic or medicine- convinced hyperprolactinemia, both with negative imaging findings, had sufficient data for addition in the analysis.

Cases with prolactinoma were defined as those with significant hyperprolactinemia and pituitary lesions less than 1 cm(microprolactinomas) or lesser than or equal to 1 cm(macroprolactinomas) who responded to dopamine agonists(normalization of Prl situations and/ or radiographic substantiation of significant drop in lesion size). At the moment of the evaluation, no case was under dopamine agonist remedy. Hyperprolactinemia was considered as serum Prl lesser than the reference value for the system according to coitus. Cases with a history of use of medicines that beget hyperprolactinemia and negative imaging findings, were considered as having hyperprolactinemia secondary to medicines, the cases without history of use of medicines causing hyperprolactinemia and negative imaging findings, were considered as cases with idiopathic hyperprolactinemia. Cases were classified according to BMI, as follows normal weight(BMI19.0-24.9); fat(BMI 25-29.9); class I rotundity(BMI 30-34.9); class II rotundity(BMI 35-39.9); and class III rotundity(BMI> 40).

Rejection criteria were primary hypothyroidism, polycystic ovary pattern, habitual renal failure, cirrhosis, adrenal insufficiency, irritative lesions of the casket wall or spinal cord, gestation, breastfeeding, nipple manipulation, and physical or emotional stress. Cases entering medical treatment for fat or rotundity, cases using dopamine agonists, and cases witnessing surgery or radiotherapy for treatment of prolactinoma were also barred from analysis. Data on coitus, age, weight(kg), height(m), BMI(kg/ m2), serum Prl at opinion(birth Prl) and maximum Prl position achieved during the case's clinical course(outside Prl) were estimated.

complaint etiology was divided into microprolactinoma, macroprolactinoma, and other causes of hyperprolactinemia for analysis.

Statistical analysis

Quantitative data were expressed as mean and standard divagation, and in the presence of asymmetry, as median and interquartile range. Correlations were estimated using analysis of friction(ANOVA) and the Spearman rank correlation. The position of significance was set at 5. Data were anatomized using the Statistical Package for the Social lores(SPSS), interpretation17.0.

Results

Of 197 medical records originally reviewed, 138 were eligible and anatomized for this report. Of these, 101(73.2) were women and 37(26.8) were men, and mean age was36.2 ±13.6 times. Regarding the etiology of hyperprolactinemia, 73(52.9) cases had prolactinoma – 46(63) with macroprolactinoma(31 women, 15 men) and 27(37) with microprolactinoma(24 women, 3 men) – and 65(47.1) had other causes of hyperprolactinemia(46 women, 19 men). Data on age, BMI, and Prl situations according to the etiology of hyperprolactinemi. No differences were set up in the data anatomized between women and men, and thus they were anatomized together as a group. The cases diagnosed as having hyperprolactinemia secondary to medicines, the inviting maturity(82) was related to neuroleptics and antidepressants.

Regarding body weight,65.2 of cases were fat or fat. The number of fat and/ or fat cases, according to the etiology of hyperprolactinemia, is shown in Table 1.

	Total (n=138)	**Macroprolactinoma (n=46)**	**Microprolactinoma (n=27)**	**Other causes (n=65)**
Normal weight	48 (34.8%)	15 (30%)	11 (40%)	19 (30%)
Overweight	42 (30.4%)	9 (20%)	8 (30%)	25 (37.9%)
Obesity – class I	24 (17.4%)	9 (20%)	5 (20%)	13 (20%)
Obesity – class II	16 (11.6%)	7 (16%)	2 (6.7%)	7 (10.6%)
Obesity – classIII	8 (5.8%)	6 (14%)	1 (3.3%)	1 (1.5%)
Overweight + obesity	65.2%	70%	60%	70%

Table 1 Number of normal weight, fat and fat cases, according to the etiology of hyperprolactinemia.

As for the etiology of hyperprolactinemia, there was a positive correlation between birth and maximum Prl situations and pituitary excrescence volume, macroprolactinomas showing birth and maximum Prl situations significantly advanced than those of microprolactinomas and other causes of hyperprolactinemia(p<0.001). In relation to lipid and glycemic profile, no differences were set up between the different groups according to the etiology of hyperprolactinemia and the correlations with prolactin situations. There was a significant but weak correlation of BMI with birth Prl and maximum Prl(rs = 0.3), demonstrating that birth Prl(p = 0.014, rs = 0.21) and maximum Prl(p = 0.013, rs = 0.21) values increased as BMI increased. When analaccording to the cause of hyperprolactinemia, only the microprolactinoma group showed a significant but moderate correlation between BMI and birth Prl(p = 0.02, rs = 0.45).

Discussion

Hyperprolactinemia is one of the most common hypothalamicpituitary endocrine diseases among women of reproductive age. It frequently occurs associated with prolactinomas and secondary to the use of centrally acting medicines. Women were more current in our sample(73.2), with mean age of36.27 times. further than half of the cases had prolactinoma as a cause of hyperprolactinemia. In cases with prolactinoma, serum Prl situations are generally commensurable to the size of the prolactinoma, with macroprolactinomas flaunting situations above 250 ng/ mL(18). Regarding medicine convinced hyperprolactinemia, Prl values vary greatly and are likely to reach situations analogous to or advanced than those reported for macroprolactinomas. Idiopathic hyperprolactinemia generally shows a moderate increase in Prl situations(19). The overall etiologic distribution and associated Prl situations set up in our study are veritably analogous to data formerly reported in the literature(,18).

The number of fat people in Brazil continues to increase every time, getting a serious public health problem. Data from the Brazilian Institute of Geography and Statistics(IBGE) show that, in 2006,42.7 of the population was fat, adding to48.1 in 2010. In Brazil, rotundity affected11.4 of grown-ups in 2006 and 15 in 2010(20). In the present study,34.8 of hyperprolactinemic cases were fat, a 132 advanced rate than that described for the general population of our country(20).

In a study describing the association of Prl and weight in 47 cases with prolactinoma, before treatment, over 50 of cases reported weight gain after the complaint(11.8 ±2.0 kg), with mean weight of 83 ±2.4 kg and BMI of27.3 ±0.6 kg/ m2(11). In another study, in which 42 cases with prolactinoma were estimated and 36 individualities with clinicallynon-functioning adenomas(NFA) were used as controls, a high frequence of rotundity was also described in prolactinomas(12). Recent weight gain(8 to 22 kg) was set up in13/42 cases with prolactinoma against only one in the control group. Mean weight was 93 ±3.4 kg in cases with prolactinoma and 78 ±2.7 kg in NFA cases. In that study, weight gain and elevated body weight were constantly associated with prolactinomas(12). In a study involving 22 cases with prolactinoma, with median Prl of 144 ng/ mL and BMI of29.5 kg/ m2, frequence of rotundity and fat was 45 and 27, independently(14). In a study assessing 37 cases with prolactinoma, cases with microprolactinoma had significantly lower original body weight than those with macroprolactinoma(p = 0.006)(17).

In this study, 50 of cases with macroadenomas were fat and 20 were fat. In cases with microadenomas, 30 were fat and 30 were fat, and in cases of other causes of hyperprolactinemia,32.1 of cases were fat and37.9 were fat. There was no difference in the prevalence or duration of hypogonadism among the groups(microprolactinoma, macroprolactinoma, and other cases of hyperprolactinemia). There was a frequence of rotundity in cases with macroprolactinoma, compared to microprolactinoma and other causes of hyperprolactinemia. In a study of cases with antipsychotic- convinced hyperprolactinemia, Prl situations weren't identified with BMI(21). Likewise, in cases with prolactinoma, no correlation was set up between Prl situations and BMI(14). In a study comparing cases with prolactinoma and NFA, weight gain and elevated body weight were frequently associated with prolactinomas(12). In the present study, a significant but weak correlation was set up between Prl and BMI in all causes of hyperprolactinemia estimated.

When examining the possible effect of rotundity on the pathogenesis of hyperprolactinemia, which should be considered in cases of idiopathic hyperprolactinemia, robotic Prl release has been shown to be significantly elevated in fat women in direct proportion to the size of the visceral fat mass(22). Because Prl is inhibited by activation of the dopamine D2 receptor(D2R), increased Prl stashing may do due to reduced D2R vacuity in the brain, which makes

these individualities more likely to have elevated Prl stashing(6). In a study considering hyperprolactinemia as a result of fat, elevated Prl stashing in fat women was reported to be significantly reduced after loss of 50 of fat(22). In that study, the authors suggested that enhancement of deficiency D2R- intermediated neurotransmission and/ or dropped circulating leptin/ estrogen situations might be involved in this miracle. Weight reduction, with accompanying drop in insulin situations, has been shown to lead to a normalization of Prl response in utmost, but not all, circumstances(10). In a study with fat subjects, no significant association was set up between birth Prl situations and the degree of rotundity in these cases(23). One time after gastric bypass surgery, and consequent massive weight loss, no significant changes in the attention of the hormone were set up, suggesting that Prl alone plays no part in the pathophysiology of rotundity. therefore, although the magnitude of the effect of weight gain on Prl situations is still controversial, varying degrees of weight gain are generally observed in cases with prolactinoma and, as reported in

the present study, also in cases with other causes of hyperprolactinemia.

The association between Prl, weight gain and rotundity suggests that Prl may play a part in the modulation of body weight and composition. still, it remains unclear whether weight gain is associated with hyperprolactinemia due to stimulation of lipogenesis or due to dislocation of central nervous system(CNS) dopaminergic tone(24).

In this study, the frequence of rotundity was significantly high, anyhow of the degree of rotundity, in hyperprolactinemia of colorful causes. We detected a advanced frequence of rotundity in cases with macroprolactinomas, compared to those with microprolactinomas and other causes of hyperprolactinemia. It's thus important to cover BMI in hyperprolactinemic cases in order to estimate the influence of treatment of hyperprolactinemia on these variables and to introduce remedial intervention in rotundity.

References

1. Ben-Jonathan N, Mershon JL, Allen DL and Steinmetz RW. **Extrapituitary prolactin: distribution, regulation, functions, and clinical aspects.** *Endocr Rev.* 1996; **17**:639-69.
2. Freeman ME, Kanyicska B, Lerant A and Nagy G. **Prolactin: structure, function, and regulation of secretion.** *Physiol Rev.* 2000; **80**:1523-631.
3. Ling C, Hellgren G, Gebre-Medhin M, Dillner K, Wennbo H, Carlsson B and Billig H. **Prolactin (PRL) receptor gene expression in mouse adipose tissue: increases during lactation and in PRL-transgenic mice.** *Endocrinology.* 2000; **141**:3564-72.
4. Ling C and Billig H. **PRL receptor-mediated effects in female mouse adipocytes: PRL induces suppressors of cytokine signaling expression and suppresses insulin-induced leptin production in adipocytes in vitro.** *Endocrinology.* 2001; **142**:4880-90.
5. Freemark M, Fleenor D, Driscoll P, Binart N and Kelly P. **Body weight and fat deposition in prolactin receptor- de ficient mice.** *Endocrinology.* 2001; **142**:532-7.Kok P, Roelfsema F, Frolich M, Meinders AE and Pijl
6. H. **Prolactin release is enhanced in proportion to excess visceral fat in obese women.** *J Clin Endocrinol Metab.* 2004; **89**:4445-9.
7. Nilsson L, Binart N, Bohlooly YM, Bramnert M, Egecioglu E, Kindblom J, Kelly PA, Kopchick JJ, Ormandy CJ, Ling C and Billig H. **Prolactin and growth hormone regulate adiponectin secretion and receptor expression in adipose tissue.** *Biochem Biophys Res Commun.* 2005; **331**:1120-6.
8. Ben-Jonathan N, Hugo ER, Brandebourg TD and LaPensee CR. **Focus on prolactin as a metabolic hormone.** *Trends Endocrinol Metab.* 2006; **17**:110-6.
9. Weyer C, Funahashi T, Tanaka S, Hotta K, Matsuzawa Y, Pratley RE and Tataranni PA. **Hypoadiponectinemia in obesity and type 2 diabetes: close association with insulin resistance and hyperinsulinemia.** *J Clin Endocrinol Metab.* 2001; **86**:1930-5.
10. Kopelman PG. **Physiopathology of prolactin secretion in obesity.** *Int J Obes Relat Metab Disord.* 2000; **24 Suppl 2**:S104-8.
11. Creemers LB, Zelissen PM, van 't Verlaat JW and Koppeschaar HP. **Prolactinoma and body weight: a retrospective study.** *Acta Endocrinol (Copenh).* 1991; **125**:392-6.
12. Greenman Y, Tordjman K and Stern N. **Increased body weight associated with prolactin secreting pituitary adenomas: weight loss with normalization of prolactin levels.** *Clin Endocrinol (Oxf).* 1998; **48**:547-53.
13. Doknic M, Pekic S, Zarkovic M, Medic-Stojanoska M, Dieguez C, Casanueva F and Popovic V. **Dopaminergic tone and obesity: an insight from prolactinomas treated with bromocriptine.** *Eur J Endocrinol.* 2002; **147**:77-84.
14. dos Santos Silva CM, Barbosa FR, Lima GA, Warszawski L, Fontes R, Domingues RC and Gadelha MR. **BMI and metabolic profile in patients with prolactinoma before and after treatment with dopamine agonists.** *Obesity (Silver Spring).* 2011; **19**:800-5.
15. Berinder K, Nystrom T, Hoybye C, Hall K and Hulting AL. **Insulin sensitivity and lipid profile in prolactinoma patients before and after normalization of prolactin by dopamine agonist therapy.** *Pituitary.* 2011; **14**:199-207.
16. Delgrange E, Donckier J and Maiter, D. **Hyper prolactinaemia as a reversible cause of weight gain in male patients?** *Clin Endocrinol (Oxf).* 1999; **50**:271.
17. Soran H, Wilding J and MacFarlane I. **Body weight and prolactinoma: a retrospective study.** *Int J Obes Relat Metab Disord.* 2004; **28**:183.
18. Klibanski A. **Clinical practice. Prolactinomas.** *N Engl J Med.* 2010; **362**:1219-26.
19. Bernichtein S, Touraine P and Goffin V. **New concepts in prolactin biology.** *J Endocrinol.* 2010; **206**:1-11.
20. Brazilian Ministry of Health. **Brazilian Institute of Geography and Statistics (IBGE)**. Coordenação de Índices de Preços, Pesquisa de Orçamentos Familiares 2008/09 [Consumer Price Index, Household Budget Survey].
21. Melkersson K, Berinder K and Hulting AL. **Effect of antipsychotic-induced hyperprolactinemia on anthropometric measures, insulin sensitivity and lipid profile in patients with schizophrenia or related psychoses.** *Neuro Endocrinol Lett.* 2011; **32**:428-36.
22. Kok P, Roelfsema F, Langendonk JG, de Wit CC, Frolich M, Burggraaf J, Meinders AE and Pijl H. **Increased circadian prolactin release is blunted after body weight loss in obese premenopausal women.** *Am J Physiol Endocrinol Metab.* 2006; **290**:E218-24.
23. Ernst B, Thurnheer M and Schultes B. **Basal serum prolactin levels in obesity--unrelated to parameters of the metabolic syndrome and unchanged after massive weight loss**. *Obes Surg.* 2009; **19**:1159