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Research Article

Psychophysiological Differences in Patients With/Without Schizophrenia: A Comparative Study

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Article Info

Received: March 23, 2021 Accepted: March 29, 2021 Published: March 31, 2021

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Citation: Antonis T, Konstantinos F, "Psychophysiological Differences in Patients With/Without Schizophrenia: A Comparative Study.". Neurosurgery and Neurology Research, 2(3); DOI: http://doi.org/03.2021/1.1013.

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Abstract

Background: Schizophrenia is a chronic and severe mental disorder affecting 20 million people worldwide. Characterized by distortions in thinking, perception, emotions, language, sense of self and behaviour.

Objective: Differences in body mass index, weight and height, in patients with schizophrenia, mood disorders vs. normal controls.

Method: The study sample included 76 patients with unipolar depression, 16 patients with schizoaffective disorder, 122 patients with schizophrenia, and 78 patients with other mental disorders, and 788 subjects normals as a control group **Results:** Significant differences in terms of height between unipolar depressive males and normal females (p<0.05), normal males (p<0.05), and males with schizophrenia (p<0.05), with unipolar depressive patients being shorter in comparison to the other groups. In terms of weight no significant differences were among groups.

Conclusions: This analysis provides evidence that female patients with schizophrenia are significantly heavier than the general population.

Key Words: schizophrenia; body mass index

Introduction

Increase in body weight was observed in more than 50% of schizophrenic patients who receive antipsychotic drugs in general. Body mass index (BMI) is significantly higher in schizophrenic patients compared to psychiatric patients with other diagnosis and to the general population (Allison et al. 2001). Additionally, increased body weight is also associated with reduced self-esteem, treatment dropout, (Gama, Souza, Lobato & Abreu, 2004; Bernstein, 1987) and increased risk of comorbid conditions The evidence report, 1998). Data from the Brazilian Ministry of Health show a prevalence of 32% for overweight and 8% for obesity in the general population (Baptista, 1999).

What is more, Schizophrenia has long been known to be highly genetic; it often runs in families. A large genome-wide association study of people with schizophrenia, published in 2014, linked the disorder to small DNA variations at more than 100 distinct locations on the human genome, which is the complete set of DNAs for humans (Geschwind, 2014).

Based on the extensive evidence of weight gain associated with antipsychotic drug use and on the lack of specific studies in schizophrenic patient, a study was carried out for the assessment of differences in weight and obesity among patients with schizophrenia, mood disorders versus normal controls.

The main aim of this study is to observe and compare the BMI differences among patients with schizophrenia, mood disorders versus normal controls.

Material and methods

A. Study sample

The study sample included 1116 subjects, of which 788 were normal control subjects, 76 patients with unipolar depression, 16 patients with schizoaffective disorder, 122 patients with schizophrenia and 78 patients with other mental

J Neurosurgery and Neurology

6

a mixture of severe forms of OCD, psychotic and mood disorders other than the before mentioned as well as severe personality disorders. There were no alcohol or substance abuse cases. The gender and age composition of the sample is shown in table 1. All patients were inpatients or outpatients of a private mental hospital. All control subjects and patients gave informed consent and the protocol received approval by the University's Ethics Committee.

	Normal N=788		Unipola r depressi on N=76		Bipolar N=36		Schizoaf fective N=16		Schizop hrenia N=122		Other mental disorder N=78		
	fe m al es	m al es	fe m al es	m al es	fe m al es	m al es	fe m al es	m al es	fe m al es	m al es	fe m al es	m al es	All Gro ups
N	46 1	32 7	54	22	19	17	14	2	49	73	39	39	1116
Age													
M e a n	39 .3 3	42 .0 4	52 .1 1	57 .2 7	49 .5 8	40 .7 6	45 .4 3	34 .0 0	36 .0 2	32 .7 3	45 .3 1	44 .1 3	41.1 6
S tr d D e v	11 .1 8	12 .2 8	12 .7 0	12 .5 8	12 .7 0	12 .3 5	15 .1 9	8. 49	11 .4 7	8. 31	17 .6 6	16 .5 8	12.8 4
Hei	Height												
M e a n	16 4. 98	17 6. 94	16 1. 59	17 0. 55	16 4. 00	17 3. 65	15 9. 93	17 8. 00	16 5. 39	17 7. 90	16 5. 05	17 5. 51	169. 74
S tr d D e v	5. 61	6. 67	6. 35	5. 64	4. 86	7. 28	5. 80	0. 00	6. 17	7. 81	8. 57	7. 38	8.78
We	ight												
M e a n	65 .4 9	83 .1 9	72 .8 0	77 .4 1	75 .2 1	83 .8 2	70 .1 4	81 .5 0	69 .3 3	81 .9 6	68 .6 9	81 .5 1	73.7 1
S tr d D e v	12 .3 2	12 .5 4	13 .1 1	13 .8 6	17 .4 7	12 .5 7	12 .3 7	2. 12	15 .4 9	14 .9 5	18 .4 4	14 .6 1	15.4 2
BM	BMI												
M e a n	24 .0 9	26 .5 4	27 .9 8	26 .5 6	27 .8 4	27 .8 0	27 .4 3	25 .7 3	25 .3 6	25 .9 1	25 .1 0	26 .3 6	25.5 0
S tr d D e v	4. 50	3. 55	5. 29	4. 13	5. 72	4. 02	4. 60	0. 67	5. 51	4. 61	6. 06	3. 87	4.57

	Wilk		Effect	Error	
	S	F	df	df	р
		73,9			>0,00
age	0,832	4	3	1101	1
diagnosis	0,976	1,82	15	3040	0.027
sex	0,997	1,09	3	1101	0.351
diagnosis*se					
X	0,972	2,1	15	3040	0.008

B. Clinical diagnosis

The diagnosis was put according to DSM-IV-TR criteria on the basis of a semi structured interview based on the Schedules for Clinical Assessment in Neuropsychiatry version 2.0 (SCAN v 2.0) ($\underline{1}$).

C. Somatometric measurement

The height and weight of all control subjects and patients was measured. The subjects' weight was measured with standardized weighting machines so as to have a reliable measurement and comparable across machines. BMI was calculated as the ratio of weight in kilograms to the square of height in meters. The means and standard deviations of the subgroups of the study sample are shown in table 1.

D. Statistical analysis.

The first step in the analysis was to transform the data (shown as means and standard deviations in table 1) to percentile scores. The Rank and Percentile method was used.

The statistical analysis of percentiles included Multiple Analysis of Covariance (MANCOVA) with diagnosis and gender as grouping variables, age as covariate and height, weight and BMI as dependent variables. The Scheffe was used as post-hoc test analysis ($\underline{2}$)

Results

The MANOVA results suggested an effect of age (p<0.001) and diagnosis (p<0.05) but not of sex (p>0.1) but also of the interaction between diagnosis and sex (p<0.01). The detailed results are shown in table 2.

The scheffe test revealed significant differences in terms of height between unipolar depressive (UD) males and normal females (p<0.05), normal males (p<0.05), and males with schizophrenia (p<0.05), with unipolar depressive patients being shorter in comparison to the other groups. In terms of weight there were no significant differences among groups. In terms of BMI, UD females had significantly higher BMI in comparison to normal females (p<0.001) and males with schizophrenia (p<0.05). The detailed descriptive statistics of the study sample characteristics are shown in table 2.

Discussion - Conclusions

The main findings of this study were: unipolar depressive patients being shorter in comparison to the other groups. In terms of weight there were no significant differences among groups. In

J Neurosurgery and Neurology

terms of BMI, UD females had significantly higher BMI in comparison to normal females and males with schizophrenia Sample results show that the overweight and obesity problem 1. affects both patients with schizophrenia and mood disorders. This aspect is apparently in opposition to several studies showing increased weight gain with the use of second-generation antipsychotics, compared to first-generation antipsychotics. Most evidence reported in the literature is based on case-control studies, pharmacovigilance and database reviews. Many of them present disadvantages, such as their retrospective nature, heterogeneous methodology, presence of systematic assessment errors and lack of adequate or well-characterized controls (American Diabetes Association, 2004).

Antipsychotics, both typical and atypical, produce weight gain, 4. although it is difficult to differentiate weight gain patterns between these drugs. Although weight gain represents a collateral effect commonly reported for antipsychotic drugs, it seems to be more common in patients taking atypical antipsychotics (Bobes et 5. al. 2003).

Meta-analyses, literature reviews, data from clinical trials and clinical experience show that some patients present a significant weight gain while taking antipsychotics. An extensive metaanalysis, including more than 80 studies and more than 30,000 measurements, has associated clozapine, as well as olanzapine, 7. with more weight gain compared with other antipsychotics (typical and atypical) (Wirshing et al. 2014),

Patients with schizophrenia are aware of their actual weight. It has been suggested in the literature that overweight and obesity have the same impact on their self-esteem and well-being as the in general population (Strassnig et al., 2005: Awad and Voruganti, 2004). Strassnig et al. (2005) and Weiden et al. (2004) suggest that patients with schizophrenia are less capable to manage their weight gain through exercise and dietary changes and thus can be 9. more prone to be noncompliance with medication that induces weight gain versus the control group.

Due to the study design, there are limitations associated with cross-sectional studies collecting data from chronic patients with long lasting illnesses using different drugs throughout the 11. III Diretrizes brasileiras sobre as dislipidemias e diretriz de treatment and with poor medical records about weight. Additionally, drug type, dose and duration of use were not specified, neither there was any control of cultural, social, genetic and psychological variables associated with eating behavior, 12. Brasil, Ministério da Saúde. Inquérito Domiciliar sobre consumption and energy expenditure (Wilson & Fairburn, 1993). Despite the limitations, it is important to stress that the findings in this study allow us to state that schizophrenic patients, under continued use of antipsychotics are at higher risk for obesity and deserve clinical, nutritional, psychiatric and psychological attention, since obesity is a risk factor for several health problems 14. that increase morbidity and mortality rates.

In this context, the authors warn about the necessity of more detailed studies, including higher number of subjects and with better information about previous treatment, in order to disentangle the process and magnitude of weight gain between different neuroleptics, with the identification of interactions with other drugs, especially anticonvulsants and antidepressants, and assessment of the residual effect of a drug over the subsequent one.

References

- Gama CS, Souza CM, Lobato MI, Abreu PSB. Clozapine use report in 56 patients seen by Clerkship of Health and Environment of the State of Rio Grande do Sul's Program of Attention to the Refractory Schizophrenia. Rev Psiquiatr RS. 2004;26(1):21-28.
- Henderson DC, Cagliero E, Gray C, Nasrallah RA, Hayden 2. DL, Schoenfeld DA, et al. (2000). Clozapine: diabetes mellitus, weight gain, and lipid abnormalities: a five-year naturalistic study. Am J Psychiatry. 157(6):975-981.
- Allison DB, Casey DE. Antipsychotic-induced weight gain: 3. a review of the literature. J Clin Psychiatry. 2001;62 Suppl 7:22-31.
- Allison DB, Mentore JL, Heo M, Chandler LP, Cappelleri JC, Infante MC, et al. (1999). Antipsychotic-induced weight gain: a comprehensive research synthesis. Am J Psychiatry. 156(11):1686-1696.
- Blackburn GL. (2000). Weight and antipsychotic medication. J Clin Psychiatry. 61 Supll 8:36-41.
- Bobes J, Rejas J, Garcia-Garcia M, Rico-Villademoros F, 6. García-Portilla MP, Fernández I, et al. (2003). Weight gain in patients with schizophrenia treated with risperidone, olanzapine, quetiapine, or haloperidol: results of the EIRE study. Schizophr Res. 62(1-2):77-88.
- American Diabetes Association, American Psychiatric Association. American Association of Clinical Endocrinologists. North American Association for the Study of Obesity. Consensus development conference on antipsychotic drugs and obesity and diabetes. Diabetes Care. 2004:27(2):596-601.
- 8. Baptista T. (1999). Body weight gain induced by antipsychotic drug: mechanisms and management. Acta Psychiatr Scand. 100(1):3-16.
- Bernstein JG. (1987). Induction of obesity by psychotropic drugs. Ann NY Acad Sci. 499:203-215.
- Clinical guidelines on the identification, evaluation, and 10. treatment of overweight and obesity in adults: the evidence report. National Institute of Health. Obes Res. 1998;6 Suppl 2:51S-209S.
- prevenção da aterosclerose do departamento de aterosclerose da Sociedade Brasileira de Cardiologia. Arq Bras Endocrinol Metab. 2002;44 Suppl 1:58-535.
- comportamentos de risco e morbidade referida de doenças e agravos não transmissíveis: Brasil, 15 capitais e Distrito Federal 2002-2003. Rio de Janeiro: INCA. 2004.
- 13. World Health Organization. Obesity epidemic puts millions at risk. Disponível em.
- Newman SC, Bland RC. (1991). Mortality in a cohort of patients with schizophrenia: a record linkage study. Can J Psychiatry. 36(4):239-245.
- Harris EC, Barraclough B. (1998). Excess mortality of mental 15. disorder. Br J Psychiatry. 173:11-53.
- Ganguli R. (1999). Weight gain associated with antipsychotic 16 drugs. J Clin Psychiatry. 60 Suppl 21:20-24.
- Wirshing DA, Wirshing WC, Kysar L, Berisford MA, 17. Goldstein D, Pashdag J, et al. (1999). Novel antipsychotics: comparison of weight gain liabilities. J Clin Psychiatry. 60(6):358-363.

- 18. McIntyre RS, Mancini DA, Basile VS. (2001). Mechanisms 35. Azevedo MHP, Soares MJ, Coelho I, Dourado A, Valente J, of antipsychotic-induced weight gain. J Clin Psychiatry. 62 Suppl 23:23-29.
- 19. Nasrallah HA, Korn ML. (2004). Metabolic disorders in 36. Martins CDN. Sintomas de humor em esquizofrenia: schizophrenia: relationship to atypical antipsychotic treatment. Medscape Psychiatry Ment Health. 9(2):130-137.
- 20. Thakore JH, Mann JN, Vlahos I, Martin A, Reznek R. Increased visceral fat distribution in drug-naïve and drug-free 37. Clinical guidelines on the identification, evaluation, and patients with schizophrenia. Int J Obes Relat Metab Disord. 2002;26(1):137-141.
- 21. Ryan MC, Collins P, Thakore JH. Impaired fasting glucose tolerance in first-episode, drug-naïve patients with schizophrenia. Am J Psychiatry. 2003;160(2):284-289.
- 22. Ryan MC, Thakore JH. (2002). Physical consequences of schizophrenia and its treatment: The metabolic syndrome. Life Sci. 71(3):239-257.
- 23. Muldoon MF, Sved AF, Flory JD, Perel JM, Matthews KA, Manuck SB. (1998). Inverse relationship between fenfluramine-induced prolactin release and blood pressure in humans. Hypertension. 32:972-975.
- 24. Fahy U et al. (1999). The lipoprotein profile of women with hyperprolactinaemic amenorrhoea. Human Reproduction. 14(2):285-287.
- 25. Yarali H, Yildirir A, Aybar F, Kabakci G, Bukulmez O, Akgul E, et al. (2001). Diastolic dysfunction and increased serum homocysteine concentrations may contribute to increased cardiovascular risk in patients with polycystic ovary syndrome. Fertil Steril. 76(3):511-516.
- 26. Muldoon MF, Mackey RH, Williams KV, Korytkowski MT, Flory JD, Manuck SB. (2004). Low central nervous system serotonergic responsivity is associated with the metabolic syndrome and physical inactivity. J Clin Endocrinol Metab. 89(1):266-271.
- 27. Halbreich U, Kahn LS. (2003). Hormonal aspects of schizophrenias: an overview. Psychoneuroendocrinology. 28 Suppl 2:1-16.
- 28. Compton MT, Miller AH. (2002). Antipsychotic-induced hyperprolactinemia dysfunction. and sexual Psychopharmacol Bull. 36(1):143-164.
- 29. Toalson P, Ahmed S, Hardy T, Kabinoff G. (2004). The metabolic syndrome in patients with severe mental illnesses. Prim Care Companion J Clin Psychiatry. 6(4):152-158.
- 30. Aronne LJ. (2001). Epidemiology, morbidity, and treatment of overweight and obesity. J Clin Psychiatry. 62 Suppl 23:13-22.
- 31. Stanton JM. (1995). Weight gain associated with neuroleptic medication: a review. Schizophr Bull. 21(3):463-472.
- 32. Kapckzinski F, Abreu MGB, Restelato R, Ziegler DR, Abreu P. (2001). Evolução de parâmetros antropométricos em crianças e adolescentes com exposição a medicamentos psicoativos: um estudo controlado em abrigados da Secretaria do Trabalho, Cidadania e Assistência Social -STCAS - RS. Rev Psiquiatr RS. 23(2)91-98.
- 33. Rio Grande do Sul, Secretaria da Saúde, Guia de Serviços de Saúde Mental. Cuidar, sim, Excluir, não. Rio Grande do sul: Secretaria da Saúde; 2002.
- 34. McGuffin P, Farmer A, Harvey I. (1991). A polydiagnostic application of operational criteria in studies of psychotic illness. Development and reliability of the OPCRIT system. Arch Gen Psychiatry. 48(8):764-70.

- Macedo A, et al. (1993). Versão portuguesa da "entrevista diagnóstica para estudos genéticos". Psiquiatr Clin. 4:213-17.
- associação com história familiar e personalidade pré-mórbida [dissertação]. Porto Alegre: Universidade Federal do Rio Grande do Sul; 2004.
- treatment of overweight and obesity in adults: executive summary. Expert Panel on the Identification, Evaluation, and Treatment of Overweight in Adults. Am J Clin Nutr. 1998:68(4):899-917.
- 38. Grundy SM. (1998). Multifactorial causation of obesity: implications for prevention. Am J Clin Nutr. 67(3 Suppl):563S-72S.
- 39. Covell NH, Weissman EM, Essock SM. (2004). Weight gain with clozapine compared to first generation antipsychotic medications. Schizophr Bull. 30(2):229-40. Wilson GT, Fairburn CG. (1993). Cognitive treatments of eating disorders. J Consult Clin Psychol. 61(2):261-9.