Negative correlation between PANSS negative subscale score and obesity in a chronic group of patients with schizophrenia, an observational study

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ABSTRACT

Background. Schizophrenia, as a chronic mental disorder with lifelong evolution is associated with low functionality and a high degree of disability. Obesity is an overlapping risk factor that is present in almost half of the patients worldwide and is responsible for increasing the overall mortality by itself or by predisposing to extensive morbidity. Its pathophysiology describes many mechanisms, the central position being occupied by the appetite regulation throughout the lipids and glucose metabolism. It is important to analyze whether the trends of correlation pairs of obesity in schizophrenia in Romanian patients are similar to those described in literature and if they are depicted in smaller groups of patients.

Methods. This study paper is an observational cross-sectional study taking into account 110 inpatients with schizophrenia. The study included 110 inpatients diagnosed with schizophrenia admitted in “Prof. Dr. Alexandru Obregia” Psychiatry Hospital. They were divided into two groups by applying the criterion of BMI with a cut-off point set at 25 and were applied PANSS and GAFs altogether with a structured basic anamnestic interview.

Results. The mean age of the patients was 39.15 years in the study group (SG) and 40.95 years in the control group (CG), the sex ratio was similar between the two groups. Regarding previous treatment, 59.3% normal weight patients were priorly medicated and 38.1% of those with BMI>25 received prior treatment. Both groups had a similar mean value of the positive and the general psychopathology subscales, the SG registered a slightly higher mean score on the negative subscale as well as on the total PANSS score. The mean value of Global Assessment of Functioning score was higher for the normal weight group.

Discussion. Our findings point out that only PANSS negative subscale score has a negative statistical correlation (p=.005) with obesity in the chronic schizophrenic group we selected. No other statistically significant correlations between sex, age, place of living, education, employment and professional statuses, previous treatment, current antipsychotic medication and obesity were made possible due to reasons such as study design choice or the large number of obese patients related to the entire sample.

Conclusion. There is an important number of schizophrenia patients that are overweight or obese. We found no major differences between the two groups on the topic of demographics or clinical variables, resulting in the conclusion that schizophrenia carries an important morbidity load, so that obesity can indiscriminately impact these patients.

Keywords: schizophrenia, obesity, BMI, psychosis, inpatients, PANSS negative subscale

INTRODUCTION

Schizophrenia is reportedly a neurodevelopmental disorder [1], with symptoms typically emerging in late adolescence or early adulthood. As a chronic affliction within the psychotic spectrum, it constitutes one of the most significant global burdens of all mental illnesses. At a global scale, one study suggests that in 2017 versus previous data from 1990 the disability-adjusted life years (DALYs) of schizophrenia increased to 12.66 million while age-standardized incidence (ASIR) decreased [2]. Obesity and overweight
are common contributing factors to overall disability mainly as a metabolic risk factor for cardiovascular disease. The onset of obesity is set early in the natural course of schizophrenia, some of the patients are obese prior to the first episode and their BMI continues to rise as treatment begins. In the first year of treatment, approximately 66% of the patients experience clinically significant weight gain [3]. A systematic review from 2021 shows that female patients with schizophrenia are 1.44 times more likely than males to live with obesity [4]. The prevalence of obesity is higher in individuals with schizophrenia than in the general population, 50% of them have been reported to have obesity and 40% as having a metabolic syndrome [5].

There is a keen connection between obesity rates in schizophrenia and increased DALYs, for this lifestyle, shared genetics, comorbidities, risk factors and medication play an important role. A Nature study from 2022 shows that both obesity and schizophrenia negatively impact brain structure, imaging studies showing alterations in frontal and mesial temporal regions of the brain as well as the ventricles. Participants diagnosed with schizophrenia exhibited a statistically significant reduction in cortical thickness, compared to the control group, in all regions except for the entorhinal cortex. Furthermore, a positive correlation was observed between higher body mass index (BMI) and cortical thinning in numerous regions, similar to those found in individuals with schizophrenia. Notably, thinner entorhinal cortex was uniquely associated with higher BMI [6].

For what concerns the complex pathophysiology of weight gain in schizophrenia, it is not fully understood. One described mechanism is that of elevated levels of several inflammatory markers such as tumor necrosis factor alpha, interleukin-6, interleukin-1-beta, encountered both in acute and chronic psychosis. Antipsychotic medication, especially second-generation molecules carry an important contribution. The main mechanism is based on their interference in the glucose and lipids metabolism and appetite regulation through antagonism of histamine-1, serotonin and dopamine-2 receptors of the hypothalamus; moreover, they also alter the monoaminergic systems, increasing the appetite additionally [7,8]. The first meta-analysis on this subject showed that, versus the placebo on which patients lost weight, most antipsychotic treatments led to weight gain, reportedly varying from 2.10 to 4.45 kg. This causal relation has later been confirmed by following meta-analyses. It has been shown that olanzapine and clozapine are the ones to contribute the most to the rise of BMI, risperidone and sertindole were responsible for intermediate amounts. Aripiprazole and amisulpride induced intermediate to low levels of weight gain and ziprasidone was occupying the lowest place [8-10]. Other studies suggest that antipsychotic medication diminishes energy consumption and intervene among gut microbiota, leading to weight gain [11].

Among people with schizophrenia, a proactive, health-oriented behavior is diminished, this enhancing even more the predisposition to obesity and the apparition of its risk factors [12]. Obesity enhances the risk for comorbidities such as cardiovascular disease, type II diabetes mellitus, coronary disease, arterial hypertension, cerebrovascular events, sleep apnea, rising the overall mortality [13]. Literature describes that a decrease in quality of life, negative symptoms and severe insomnia are associated with obesity [14].

METHODS

The aim of the study was to evaluate whether certain demographics, lifestyle, or treatment correlate with obesity in persons with schizophrenia from a cohort of inpatients. The current paper is an observational cross-sectional study regarding 110 inpatients with schizophrenia admitted in “Prof. Dr. Alexandru Obregia” Psychiatry Hospital. Body Mass Index (BMI) with a cut-off point set at 25 criterion was applied and two groups were formed. Data was gathered and analyzed in SPSS and we used the Positive And Negative Syndrome scale (PANSS) and Global Assessment of Functioning (GAF) scale to get a better understanding of the weight impact in this pathology. We hypothesized that patients suffering from schizophrenia that have a BMI over 25 thus being overweight or obese have a worse outcome of the main disorder by scoring lower to the assessment tests. We wanted to assess whether there is any correlation between demographics, lifestyle, treatments with overweight and obesity and for that we used logistic regression and t test.

RESULTS

Patients were divided into two groups, one control group (CG) of 46 patients (41.8%) with BMI below the cut-off of 25 and a study group (SG) of 64 patients (58.2%) with BMI above that value. Furthermore, the latter group was composed of 47 (42.73%) overweight subjects and 17 (15.5%) with obesity. The mean age of the participants was 39.15 years in the CG and 40.95 in the SG. Regarding demographics, sex distribution ratio of females to males was similar between the two groups, with a higher value in the SG. The normal weight group was represented by 25 (54.3%) women and 21 (45.7%) men, whilst the overweight and obese group registered a higher proportion of women with BMI >25 (62.5%) compared to men (37.5%). The data showed that environment was not responsible for favouring weight gaining, in both groups 89.1% of individuals were living in urban areas and 10.9% came from rural regions (Chart I). 13% of the CG had a bachelor’s degree, versus 10.9% of the SG.
Within the CG, 73.9% of the patients were unemployed because of the illness versus 65.6% from the other group. In the CG 54.3% had a high level of education, 32.6% a medium one and 13% a low level, distribution similar to the one in the SG. Regarding previous treatment, 59.3% normal weight patients were previously medicated and only 38.1% of those with BMI>25. On the topic of current medication, 88.9% of the CG patients were administered 2nd generation antipsychotics (SGAs) versus 84.4% in the study one (Chart 2).

We used Positive and Negative Syndrome Scale (PANSS) to assess the symptoms, as seen in Table I. Both groups had almost the same mean value on the positive and the general psychopathology subscales, the SG registered a slightly higher mean score on the negative subscale as well as on the total PANSS score. The mean value of Global Assessment of Functioning score was higher in the normal weight group.

DISCUSSIONS
Despite literature that attributes some of the characteristics we analyzed to obesity in schizophrenia, our study does not point out statistic significant correlations between those risk factors and obesity in
TABLE 1. Mean values of main clinical scales

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Weight</th>
<th>Height</th>
<th>PANSS positive subscale score</th>
<th>PANSS negative subscale score</th>
<th>PANSS psychopathology subscale score</th>
<th>PANSS total score</th>
<th>GAFs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal weight group</td>
<td>39.15 y</td>
<td>62.54 kg</td>
<td>167 cm</td>
<td>21.72</td>
<td>23.52</td>
<td>46.80</td>
<td>92.04</td>
<td>75.22</td>
</tr>
<tr>
<td>Overweight and obese group</td>
<td>40.59 y</td>
<td>78.00 kg</td>
<td>166 cm</td>
<td>21.27</td>
<td>24.61</td>
<td>46.89</td>
<td>92.77</td>
<td>75.08</td>
</tr>
</tbody>
</table>

TABLE 2. Independent Samples Test

<table>
<thead>
<tr>
<th></th>
<th>p value</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.172</td>
<td>-.897</td>
</tr>
<tr>
<td>Longest period of active working</td>
<td>.508</td>
<td>-.146</td>
</tr>
<tr>
<td>Height</td>
<td>.797</td>
<td>.509</td>
</tr>
<tr>
<td>Age of disorder onset</td>
<td>.220</td>
<td>.776</td>
</tr>
<tr>
<td>Time spent with disorder</td>
<td>.689</td>
<td>-1.340</td>
</tr>
<tr>
<td>Number of hospital admissions</td>
<td>.415</td>
<td>-.479</td>
</tr>
<tr>
<td>PANSS positive subscale score</td>
<td>.691</td>
<td>.622</td>
</tr>
<tr>
<td>PANSS negative subscale score</td>
<td>.005</td>
<td>-1.265</td>
</tr>
<tr>
<td>PANSS psychopathology subscale</td>
<td>.235</td>
<td>-.064</td>
</tr>
<tr>
<td>PANSS total score</td>
<td>0.768</td>
<td>-.306</td>
</tr>
<tr>
<td>GAFs score</td>
<td>.189</td>
<td>.078</td>
</tr>
</tbody>
</table>

the SG, except for the PANSS negative subscale score, which negatively correlates with obesity (Table 2). A reason for that would be the study design we chose. The cross-sectional approach imposes certain limitations, the main one being the short time span of the observation. For example, if we were to make an historical inquiry of the treatment these patients were under since the beginning of their disorder, they would probably respect the literature model i.e. those who were prescribed 2nd generation antipsychotics have higher risk of weight gain, similar to our group (Chart 2). Another reason for this occurrence might be that there is an important number of overweight and obese patients relative to the entire group, so that even though there were some correlations, those were not significant.

We found little data searching online literature about correlations between the negative subscale and BMI. Among current studies, one reporting, similar to our result, negative correlation between PANSS negative subscale score and obesity in patients with schizophrenia [15], while another stating that negative symptoms associates, among other metabolic parameters, with a higher BMI [16].

CONCLUSION

There is an important number of patients suffering from schizophrenia that are overweight or obese. We found no major differences between the two groups on the topic of demographics or clinical variables, resulting in the conclusion that schizophrenia carries an important morbidity load, so that obesity can indiscriminately impact these patients. There is certainly room for more research on how obesity impacts the social life of our patients. As obesity is often present from early phases of psychotic disorders, early interventions are mandatory. Also, we believe that there is an acute need for more feasible and applied solutions to interrupt this negative feedback continuum between obesity and the chronic negative symptoms of schizophrenia. Regarding little and sometimes contradictory data on clinical findings, more research is necessary to shed light upon this intensely heterogenous disorder.

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REFERENCES


