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Dyslipidemia Prevalence of Severe Mentally Ill Patients Who are under Pharmacotherapy Scheme

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Abstract

There is an increased mortality rate associated with severe mental illness due to preventable causes; such as, metabolic disorders, cardiovascular diseases, diabetes mellitus and prevalence of obesity associated to the use of pharmacotherapy. Patients prescribed with antipsychotics generally have metabolic abnormalities that lead to lipid imbalance, which increases their morbidity and mortality. Little research has been found in relation to this problem, suggestion further research in this area, focusing on the type of disease and the types of prescribed drugs. The aim of this study was to determine the prevalence of dyslipidemia in a trial with patients with severe mental illness under a drug scheme. Sample for study involved 21 patients, with psychiatric disorders diagnosed; from Supersition Mountain Mental Health Center (SMMHC)", Arizona, USA. In addition, these patients were receiving psychopharmacological treatment, aged 18, who agreed to participate voluntarily in the study. Evaluations were conducted on samples of lipid profile and glucose, in addition to clinical and anthropometric assessment of analyzed nursing records, plus medical examination of the last annual checks, according to SMMHC protocols. Altered serum cholesterol, HDL, LDL and VLDL to 42% of values were found on the studied population. It is assumed that the consumption of antidepressant drugs induces dyslipidemia, and management recommended a multidisciplinary team including a nutritionist to approve the use of those medications. However, it is recommended that a continuous study involving a larger group of patients is completed in order to ensure validity and reliability of the results.

Keywords: Antipsychotics; Antidepressants; Cholesterol; Dyslipidemia; Schizophrenia; Metabolic Disorders

Summary

There has been an increase in the mortality rate associated with drug therapy in patients with severe mental illness due to preventable causes; such as, metabolic disorders, cardiovascular diseases, diabetes mellitus and obesity prevalence. Patients prescribed antipsychotics generally have metabolic abnormalities that lead to an imbalance of lipids, increasing morbidity and mortality. Few studies have been reported in connection with this problem, and research in this area focuses more on the type of disease and prescribed medications. The aim of this study was to determine the prevalence of dyslipidemia in a population of patients with severe mental illness undergo a psychopharmacological drug regimen. The study included 21 patients assigned to the Mental Health Center Superstition Mountain Mental Health Center (SMMHC) ", Arizona, USA, who agreed to participate voluntarily in the study diagnosed psychiatric disorders. The evaluations were performed on samples profile lipids and blood glucose of patients, in addition to the clinical evaluation and anthropometric that were taken from nursing records, plus medical examination of recent annual checks, according to the protocols SMMHC. It was found that in the study population 42% of the values of serum cholesterol, HDL, LDL and VLDL were altered. It is assumed that the use of antidepressant drugs induced dyslipidemia in patients and a multidisciplinary team that includes a nutritionist in the protocol approval to recommend the use of these drugs. However, it is recommended that an ongoing study involving a larger group of patients, in order to ensure the validity and reliability of results.

Introduction

There is consensus that individuals with unhealthy living habits; such as increased consumption of snuff, diets high in saturated fat and sugars, high energy intake and decreased physical activity, among others, are prone to diseases such as; obesity, diabetes, dyslipidemia and hypertension[1-8]. In patients with acute mental illness, the problem becomes more conspicuous, as different studies have reported that these additional having less healthy lifestyle (increased consumption of snuff, diets high in saturated fat and sugars, high energy intake and individuals less physical activity, etc.), there is a negative effect of anti-psychotic drugs that have been prescribed [10-13]. Brown, et al. [14] noted high levels of mortality in patients with schizophrenia. It has also been reported that people with serious mental illness, largely treated with anti-psychotic, present a substantial risk of death from cardiovascular disease (CVD) [14-16] and presents a probability overweight, diabetes, hypertension and dislipidemia [17]. It was found that the mortality rate of such patients is 2-3 times higher than the general population.

Dyslipidemia is characterized by high cholesterol and triglycerides concentrations in plasma. It is considered as the 90th percentile of a clinically normal population. Although there are several classifications, there is no classification that includes phenotypic and etiopathogenic classification as one. The usefulness of phenotypic classification is based on formation of a general criterion. Etiopathogenic classification facilitates diagnosis based on classifying basic etiopathogenic dyslipidemia in primary (the cause is a genetic lipoprotein disorder) and secondary (the lipoprotein alteration is result of an underlying disease). Dyslipidemia is a major risk factor for coronary heart disease, the leading cause of death in the United Statesand Cardiovascular disease (CVD) due to atherosclerosis of the arterial vessel wall and to thrombosis is the foremost cause of premature mortality and of disability-adjusted life years (DALYs) in Europe, and is also increasingly common in developing countries. In the European Union, the economic cost of CVD represents annually E192 billion1 in direct and indirect healthcare costs [18].

Dyslipidemia, as a risk factor of CVD, is manifested by elevation or attenuation of plasma concentration of lipoproteins. Several methods used to classify this include the lipoproteins in respect to their density, physical, and chemical properties. Based on These classifications, different types of lipoproteins, including chylomicrones, IDL1, VLDL2, LDL3, and HDL4, and apolipoproteins (Apo), including Apo A, Apo B, Apo C, and Apo E, have been introduced. Generally, the dyslipidemia is defined as the total cholesterol, LDL, triglycerides, apo B or LP (a) levels above the 90th percentile or HDL and apo a level below the 10th percentile of the overall population [18].

According to Ceruelo, et al. [19], the most widely accepted classification of antipsychotics is typical antipsychotics (AT) and atypical antipsychotics (AA). The AT is the oldest, with antidopaminergic action and mainly characterized by their effectiveness in controlling positive psychotic symptoms (delusions, hallucinations) and ineffective on negative psychotic symptoms (depression, social isolation). Its use is often associated with extrapyramidal symptoms (EPS) and hyperprolactinemia.

The AA are characterized by simultaneously blocking dopamine receptors and serotonergic and be effective in both positive and negative symptoms [20]. However, any

drug has some potential risks, and among these risks, antipsychotics may contribute to dyslipidemia [21].

Antipsychotic medication may induce weight gain and increase the risk of metabolic adverse effects, resulting in a higher incidence of cardiovascular disease [21,22]. The effects produced in the weight can be derived, on the one hand, the anticholinergic action facilitates constipation and urinary retention and, on the other hand, action of an antihistamine (H1 receptor blocking) resulting in increased appetite and subsequent weight gain in patients undergoing prolonged treatment.

Finally, they can produce other adverse effects such as boosting the intake of carbohydrates, also favoring weight gain, which is increased with use of tricyclic tertiary [23]. Particularly in patients with bipolar disorder, a history of depression and / or taking antidepressants, it is also starting to see an increase of factors modifiable cardiovascular risk [24-26]. However, to date, the database regarding the risks of the drugs used to treat unipolar depression or bi polar, such as antidepressants or mood stabilizers, is not as extensive as antipsychotics [27] drugs. Despite the high risk of these patients, their access to general health care is limited and its prevention opportunities are lower than expected for the population affected with mental health problems [28,29]. Also, studies related to metabolic disorders suffered by patients with mental illness; for example, schizophrenia [30] is little referenced. The lack of consensus as to who should take responsibility for the needs of attention overall mental health in these patients, has faced a continuing failure to provide appropriate services. The aim of this study was to determine the prevalence of dyslipidemia in patients with severe mental illness in drug scheme when assessing lipid and blood glucose levels in patients taking antipsychotic drugs [31].

Methods

We started dating the report of the 50 registered at the Center for Mental Health "supersition Mountain Mental Health Center (SMMHC)" Arizona, United States patients. Of the 50 patients 21 were selected, which had altered values in the lipid profile and greater autonomy, and therefore reasonably likely to outpatient care. Patients were excluded for elderly, elevated impairment for suffering severe mental retardation, or because the expectations of rehabilitation are not feasible in the short to medium term. The selected patients were medicated with at least one of the drugs listed in (Table 1).

Active Ingredient		Brand Name	Pharmacological Action	Pharmacodynamics and receptors	Absorption and Excretion
1	Acid Valproic	Depakote	Anticonvulsant	GABA (Antagonist)	Hepatic Absorption
2	Aripiprazole	Abilify	Antipsychotic	D_2 (dopamine agonist)	Urinary (1%) and faeces (18%)
3	Benzatropine	Cogentin	Antipsychotic (negative symptoms)	ACh (anticholinergic)	Duodenal Absorption
4	Bupropion	Wellbutrin, Budeprion	Antidepressant	ISRS (Serotonin reuptake inhibitor)	Hepatic Absorption
5	Buspirone	Buspar	Anxiolytic	$5HT_{1A}$ (serotonin agonist)	Hepatic Absorption
6	Citalopram	Celexa	Antidepressant	ISRS (Serotonin reuptake inhibitor)	Hepatic Absorption
7	Clonazepam	Klonopin	Anxiolytic - sedative	GABA-A BZD_{w1} y BZD_{w2} (Agonist)	Hepatic Absorption
8	Chlordiazepoxide	Librium	Anxiolytic - sedative	GABA-A BZD_{w1} and BZD_{w2} (Agonist)	Hepatic Absorption
9	Clozapine	Leponex	Antipsychotic	D_4 (dopamine antagonist)	Urinary (50%) and faeces (30%)
10	Diphenhydramine	Benadryl	Sedative - hypnotic	H_1 (Histamine nonselective Antagonist)	Hepatic Absorption
11	Duloxetine	Cymbalta	Antidepressant	IRSN (Serotonin reuptake inhibitor-noradrenaline)	Duodenal Absorption
12	Eszoplicona	Lunesta	Sedative - hypnotic	H_1 (Histamine nonselective Antagonist)	Hepatic Absorption
13	Fluoxetine	Prozac	Antidepressant	ISRS (Serotonin reuptake inhibitor)	Hepatic Absorption
14	Fluvoxamine	Luvox	Antidepressant	ISRS (Serotonin reuptake inhibitor)	Hepatic Absorption

15	Haloperidol	Haldol	Antipsychotic	ISRD (Dopamine reuptake inhibitor)	Urinary (40%) and faeces (60%)
16	Hidroxicine	Vistaril	Sedative	H_1 (Histamine nonselective Antagonist)	Hepatic Absorption
17	Lamotrigine	Lacmital	Antiepileptic	$5HT_3$ (serotonin antagonist)	Hepatic Absorption
18	Lorazepam	Ativan	Anxiolytic	GABA-A BZD_{w1} and BZD_{w2} (Agonist)	Hepatic Absorption
19	Mirtazapine	Remerón	Antidepressant	$5HT_{2A}$ (Serotonin Agonist)	Urinary (75%) and faeces (25%)
20	Olanzapine	Zyprexa	Antipsychotic	$5HT_{2A}$ (Serotonin Agonist)	Urinary (57%) and faeces (30%)
21	Paroxetine	Paxil	Antidepressant	ISRS (Serotonin reuptake inhibitor)	Hepatic Absorption
22	Quetiapine	Seroquel	Antipsychotic	D_2 y $5HT_{2A}$ (Antagonist)	Urinary and faeces
23	Risperidone	Risperdal	Antipsychotic	D_2 y $5HT_{2A}$ (Antagonist)	Urinary 35% to 45%
24	Temazepam	Restoril	Anxiolytic - sedative	GABA-A BZD_{w1} and BZD_{w2} (Agonist)	Hepatic Absorption
25	Trazodone	Trittico	Antidepressant	Inhibitor no selective of serotonin recaptation	Urinary
26	Venlafaxine	Effexor	Antidepressant	IRSN (Serotonin reuptake inhibitor-noradrenaline)	Duodenal Absorption
27	Ziprasidone	Geodon	Antipsychotic	D_2 and $5HT_{2A}$ (Antagonist)	Urinary (1%) and faeces (4%)
28	Zolpidem	Ambien	Sedative	BZD_{w1}	Urinary

Table 1: Drugs usually prescribed for patients with severe mental illness.
Source: [31] Salazar, Peralta y Pastor (2006).

Data determinations of triglycerides, HDL-cholesterol, LDL-cholesterol, VLDL and glucose in blood samples from patients were analyzed, in addition to clinical and anthropometric assessment of nursing records and recent medical examination of controls annually, according to the protocol of the Center for Mental Health "superstition Mountain Mental Health Center (SMMHC)." The sample (21 patients) of both genders, aged 18 years or more with psychiatric disorders such as schizophrenia, bipolar disorder and depression, diagnosed according to criteria of the Diagnostic and Statistical Manual of Mental Disorders DSM IV-TR; psychopharmacological treatment with antidepressant action, antipsychotic and sedative-hypnotic.

Results and Discussion

Patients used drugs and their mechanisms of action. Psychotic disorder patients were medicated as the pharmacodynamics of the drug, as specific dopamine agonists Aripiprazole D2 receptor, D4 receptor antagonists such as clozapine, inhibitors of the reuptake of dopamine (ISRD) as Haloperidol; antagonists serotonergic (5HT2A) and olanzapine; and antagonists of D2 and 5HT2A combined action as Quetiapine, Ziprasidone and Risperidone. Dopamine is a major neurotransmitter involved in the pathogenesis of schizophrenia. The current approach to dopaminergic functioning is the presence of a hypodopaminergic to the prefrontal cortex and hyperdopaminergic state, mainly in the basal ganglia [32].

Dopamine D2 receptor antagonists exert a lock on these inhibiting dopamine release; since they interact with the G protein G a type i, for which inhibits adenylyl cyclase [33] and therefore inhibits the formation of cAMP, and the opening of Ca2 + channels. The reduction in cAMP formation that decreases the activity of PKA phosphorylates the synapsis I and II, the addition of a phosphate group to weaken the bonding synapsins synaptic vesicles cytoskeleton easy transport to the active region; so in this case the vesicles tend to be attached to the cytoskeleton. Most importantly, inhibition of $Ca^{2\pm}$ activated voltage reduces the entry of the cation that occurs in response to action potentials reaching synaptic terminal decreasing the probability of vesicle fusion [34,35]. Apparently stimulation of D3 and D4 receptors goes in the same direction as the D2, since they are a subtype; so that in the mechanism described includes dopamine D4 receptor agonists. Particularly aripiprazole besides being a partial agonist at D2 receptors is able to antagonize or enhance dopaminergic activity in the CNS inversely related to the dominant dopaminergic tone [36].

Clozapine deserves special attention to be employed in drug-resistant schizophrenia because they have the ability to bind to numerous receptors: dopamine, serotonin, alpha-adrenergic, muscarinic and histamine (H1) at which antagonizes with predominantly dopamine binding limbic level and mesocortical D1 and D4 receptors [37]. Serotonergic neurons originating in the raphe nuclei exert an inhibitory tone on dopaminergic transmission in the nigrostriatal and mesocortical areas limiting its synthesis and release. In schizophrenia this inhibition of dopamine by serotonergic control is exaggerated, which partly explains the nigrostriatal and mesocortical dopaminergic hypoactivity. This inhibition can be lifted by the serotonergic antagonist molecules [37]. Antagonists of D2 and 5HT2A combined action, have a higher affinity for the 5-HT2A receptor than for D2 receptors. This lower affinity for D2 receptors results in a lower blocking the actions of dopamine in the nigroestrial pathway and consequently a drastic reduction in motor side effects [38]. It is believed that the negative and cognitive symptoms of schizophrenia improved with partial agonist activity at the 5HT1A quetiapine. The high affinity for 5HT1A quetiapine relative D2 receptor occupancy indicates substantial 5HT1A receptor sites in therapeutic doses [39]. Certain classical neuroleptics exert a modest 5-HT2 antagonist activity, but much lower than that observed with risperidone or clozapine. In the latter case, the occupation 5-HT 2 is predominant with respect to the D2 antagonistic effect [37].

The mechanism of weight gain of these antipsychotics is related to the anticholinergic, serotonergic and histaminergic locks, which are related to appetite stimulation [40]. The facilitation of serotonergic neurotransmission reduces food intake; antagonism of central 5-HT2C receptors by antipsychotic induced food intake in spite of satiety, leading to long term weight gain [41]. All that drugs that block D2 receptors cause weight gain [42].

Moreover patients who had depressive disorder were medicated, according to the pharmacodynamics of the drug, reuptake inhibitors (SSRIs) such as Bupropion, Citalopram, Fluoxetine, Fluvoxamine and Paroxetine; inhibitors, serotonin-norepinephrine as Wenlafaxina and nonselective inhibitor of serotonin reuptake Trazodone. Currently little is known about the biological basis of depression and the mechanisms of action of antidepressants; monoaminergic hypothesis that deficits in neurotransmission by biogenic amines, whose levels decreased would be found to be the main reason for the depression is accepted; the results of multiple studies demonstrate the important role of serotonin and norepinephrine [43]. It has been shown that the levels of tryptophan (essential amino acid precursor of serotonin synthesis) in cerebrospinal fluid are smaller in depressed patients compared with control subject levels. On the other hand, it was observed that, when tryptophan experimentally is depleted by the administration of a special diet supplemented with high doses of neutral amino acids, patients depressed in remission relapsed rapidly, although they maintained their antidepressant medication; however, when they were given a supplement of tryptophan patients quickly recovered euthymic state [44].

Based on the above, it facilitates understanding of the mechanisms of action of antidepressant drugs. For reuptake inhibitors (SSRIs) pharmacodynamics is based on (but not exclusively) highly selective blockade of serotonin reuptake into presynaptic neurons [45]. Pump towards serotonin presynaptic neuron is readily inhibited by administering an SSRI. This causes an immediate increase in the somatodendritic serotonin available area and not at the terminal axon area where the therapeutic action of the postsynaptic neuron to be exercised. If the SSRI is administered in prolonged, increasing serotonin in the somatodendritic area of the presynaptic neuron triggers desensitizing 5-HT1A auto receptor, leading to a higher driving pulse, releasing more serotonin in the axon, stimulating postsynaptic receptor. The disinhibition of different serotonergic pathways and consequent stimulation of the 5-HT2puede explain the broad spectrum of therapeutic actions of SSRIs. The disinhibition of serotonergic neurotransmission in the pathway from the midbrain raphe to the prefrontal cortex would be related to its antidepressant action [46]. In the pharmacological treatment of depression reuptake inhibitors of serotoninnoradrenaline acting on these two neurotransmitters and less effect on dopamine was also recognized, but without blocking a 1-adrenergic receptors, cholinergic or histamine, what becomes “cleaner” than other dual drug as some tricyclic [47].

Antidepressants to have the opposite effect to antipsychotics as to serotonergic antagonism, prolonging the effect of postsynaptic serotonin and adrenaline level would not produce a significant change in weight gain weight patients under the effect of these drugs; although the use of these drugs can be combined with antipsychotics and sedative-hypnotics. The use of psychoactive drugs as widespread in the world, is also known for the diverse range of potential adverse or side effects they generate; iatrogenic such alterations involve various areas of the organism either concomitantly or separately, as for example metabolic, endocrine, extrapyramidal, cardiovascular, reproductive,

gastrointestinal, and / or hematologic. Among the likely consequences of some of the drugs, which are very few can be used under close medical supervision in cases of nursing, can generate orthostatic hypotension, hypersalivation in night hours, increases the risk of non-insulin dependent diabetes mellitus, as well that is quite common weight gain much of psychoactive drugs, elevations in liver transaminases, hypertriglyceridemia, hyperglycemia, hypercholesterolemia, decreased T4 levels, extrapyramidal effects, clumsiness, weakness or fatigue, ataxia, incoordination, dry mouth, sexual dysfunction, blurred vision, headache, nervousness, dizziness, anterograde amnesia, arrhythmia, psychomotor agitation, among others.

From the previously reviewed, is especially relevant adjuvant psychotherapeutic support in line with pharmacotherapy, this with the purpose of consolidating adherence in its different variants (medical, pharmacological, psychological, social) so that patients can get higher chances of recovery within a shorter period [48]. Biochemical evaluation in patients selected for the study. According to (Table 2), triglycerides, total cholesterol, HDL, LDL and VLDL to 42% of the study population values were altered. A 67% of patients had overweight or obese 1, which relates to 42% of mixed dyslipidemia and hypertriglyceridemia 23 percent and 14% of cholesterol and therefore an increased risk in these patients.

Parameters	Patients	Normal Values
Cholesterol (mg/dL)	195.53 ± 47.09	< 200
Triglycerides (mg/dL)	158.63 ± 112.21	< 150
HDL cholesterol (mg/dL)	46.84 ± 14.68	> 39
LDL cholesterol (mg/dL)	112.63 ± 37.90	< 130
VLDL (mg/dL)	29.83 ± 19.53	0 - 29
Glycemia (mg/dL)	96.25 ± 11.61	< 100
IMC Kg/mts^2	28.44 ± 6.06	18.50-24.99
Age	43.00 ± 16.40	----------

Table 2: Evaluation of dyslipidemia in patients using psychotropic drugs.
Results are expressed as mean ± standard deviation.

Because patients who are under the effect of antipsychotic drugs experience a marked increase in food intake described above. The increased appetite, however small, should not be underestimated, as it has been calculated that an increased intake of 125 Kcal could result in accumulation of approximately 6 kg of adipose tissue is a year [41], this brings resulting metabolic alterations that affect the health status of patients.

Short-term alterations in lipid metabolism as seen in the results of the serum of these patients are evident. Triglycerides are transported in the circulation as lipoproteins, as these triglycerides are generated from excess carbohydrates and dietary protein lipoproteins containing and in the liver are called lipoproteins, very low density (VLDL), which also contain cholesterol. By action of lipoprotein lipase, an enzyme present in the vascular epithelium, the present triglycerides in VLDL degrade fatty acids and glycerol which will deposited in adipose tissue as triglycerides. Now with a reduced VLDL triglyceride content is called VLDL remnants, after further removing the remaining triglycerides are called VLDL to intermediate density lipoprotein (IDL). With the additional elimination of triglycerides from IDL by the action in the hepatic sinusoids of hepatic triglyceride lipase, the IDL are degraded to form low-density lipoprotein (LDL) [48], presenting a high-cholesterol and lower triglycerides. Based on these mechanisms is a correlation between the concentrations of VLDL compared to triglycerides and LDL cholesterol relative to the set.

Moreover, high concentrations of VLDL remnant and LDL as well as age, are risk factors for developing atherosclerotic disease, since it is a precursor to the development of foam cells in the subintimal space and macrophages bind and internalize acids existing fatty modified LDL oxidation, accumulation of foam cells deform the overlying endothelium, exposing the blood to foam cells and the underlying extracellular matrix, these airlines serve the airport exposure to platelet adhesion to release cytokines process that perpetuate and increase the potential for thrombus formation; this process, over time, resulting in the occurrence of an ischemic event such as acute myocardial infarction or acute stroke [49].

Moreover, a decrease in high density lipoprotein (HDL) results also important metabolic implications since the HDL promotes reverse cholesterol transport, cholesterol molecule is exposed on the outside of the endothelial membrane protein action of the ATP binding cassette (ABC1) where the HDL does accept and carry it to the core through an enzymatic modification of cholesterol to cholesterol ester catalyzed by lecithin-cholesterol acyltransferase, collaborating in this way to control risk of an atherosclerotic process. HDL function is not limited only to this point but also promotes the transport of cholesterol to the liver by the action of the protein of cholesteryl ester transfer (CETP), from the interaction between HDL and VLDL This protein promotes transfer of cholesterol from HDL to the VLDL, while the latter transfers triglyceride to HDL. This is evident as the HDL promotes adequate metabolic control serum cholesterol.

The long-term increase of adipose tissue, particularly visceral level, leads to an increased insulin resistance due to the release of adiponectin secreted by adipocytes that opposes the action of insulin, resistin. [50] As part of the compensatory mechanisms of the bodies' insulin production rises, however this mechanism results in an inability of the beta cells of the pancreas to produce insulin which triggers chronic hyperglycemia results in bringing the suffering of diabetes mellitus. The sum of these metabolic complications along with the appearance of hypertension is the criteria for diagnosis of metabolic syndrome.

Conclusion

Considering the values of serum lipid metabolism indicators, it can be concluded that patients have dyslipidemia, conditioning this elevation of risk factors for myocardial infarction, stroke and long-term metabolic syndrome. High on the lipid profile of patients presumed values is indirectly related to the use of drugs antipsychotics, because of its relation to increased appetite. For all this, the nutritional management of patients is recommended by a multidisciplinary team that also involves treating physicians, a nutritionist from nutritional strategies to improve the eating habits of these patients, a psychologist for psychoeducational training directed to inform patients about the operation of antipsychotics and their possible adverse effects and the benefits of a healthy lifestyle. All this would contribute to a better control of the side effects of these drugs on patients. However for a more conclusive statement requires an ongoing assessment of patients, and additional data.

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References

1. Isomaa B (2003) A major health hazard: The metabolic syndrome. Life Sciences 73(19): 2395-2411.

2. Bonnet F, Irving K, Terra JL, Nony P, Berthezene F, et al. (2005) Anxiety and depression are associated with unhealthy lifestyle in patients at risk of cardiovascular disease. Atherosclerosis 178(2): 339-344.

3. Jimenez GR, Hernandez BV, Jimenez TI, Carrasco GP, Lopez de A, et al. (2009) Trends in cardiovascular risk factors and lifestyle behaviors among Spanish adults with diabetes (1993–2003). J Diabetes Complications. 23(6): 394-400.

4. Pietraszek A, Gregersen S, Hermansen K (2010) Alcohol and type 2 diabetes. A review. Nutr Metab Cardiovasc Dis 20(5): 366-375.

5. Reimers CD, Reimers AK, Knapp G (2013) Erhohung prevention and physical activity life expectancy. Prevention and treatment of neurological diseases by and sport psychological: 3-20.

6. Anton SD, Karabetian C, Naugle K, Buford TW (2013) Obesity and diabetes as accelerators of functional decline: Can lifestyle interventions maintain functional status in high risk older adults? Experimental Gerontology 48(9): 888-897.

7. Carlsson AC, Wändell PE, Gigante B, Leander K, Hellenius ML, et al. (2013) Seven modifiable lifestyle factors predict reduced risk for ischemic cardiovascular disease and all-cause mortality regardless of body mass index: A cohort study. Int J Cardiol 168(2): 946-952.

8. Ahmed HM, Blaha MJ, Blumenthal RS (2014) Modifiable lifestyle risk cardiovascular disease, and all-cause mortality. Int J Cardiol 172 (1): 199-200.

9. Annuzzi G, Rivellese AA, Bozzetto l, Riccardi G (2014) the results of Look AHEAD do not row against the implementation of lifestyle changes in patients with type 2 diabetes. 24(1): 4-9.

10. Schroeder M, Perez E, Lares M, Brito S, Cira Lenin (2010) A review: Association of Preventable Medical Conditions, Healthy Nutrition, the Ingestion of Psychotropic Medication and People Who Suffer from Severe Mental Illnesses. Venezuelan journal Archives of Pharmacology and Therapeutics 29(2): 20-25.

11. Oliver K, Suzana U, Miro J (2010) Metabolic Syndrome in patients with psychotic disorders: diagnostic issues, co morbidity and side effects of antipsychotics. Psychiatr Danub 22(1): 76-86.

12. Fernando RA, Enrique B, Enrique E, Juan Francisco MT, Susana M, et al. (2006) Measurement and control of metabolic and cardiovascular risk in patients with schizophrenia. Med Clin (Barc) 127: 542-548.

13. Denayer, DeHert M, Scheen A, L VanGaal, Peuskens J (2007) Metabolic disorders associated with atypical antipsychotics: Belgian consensus on what to do. The Brain 33(2): 197-202.

14. Brown S, Inskip H, Barraclough B (2000) Causes of the excess mortality of schizophrenia. Br J Psychiatry 177: 212-217.

15. Capasso RM, Lineberry TW, Bostwick JM, Decker PA, St Sauver J (2008) Mortality in schizophrenia and schizoaffective disorder: An Olmsted County, Minnesota cohort: (1950-2005). Schizophr Res. 98(1-3): 287-294.

16. Casey DE, Haupt DW, Newcomer JW, Henderson DC, Sernyak MJ, et al. (2004) Antipsychotic-induced weight gain and metabolic abnormalities: implications for increased mortality in patients with schizophrenia. J Clin Psychiatry 65(S7): 4-18.

17. Citrome (2007) L Risk benefits analysis of available treatments for schizophrenia. Psychiatric Times 1: 27-30.

18. Hernández María José (2011) Protocolo de dyslipidemia, México.

19. Ceruelo Bermejo J, García Rodicio S (2007) Antipsicóticos típicos. Antipsicóticos atípicos. FMC, 14(10): 637-647.

20. Ortiz Lobo A, De la Mata Ruiz I (2001) New antipsychotics. National Health System, 25(1): 1-8.

21. Kim SH, Kim K, Kwak MH, Kim HJ, Kim HS, et al. (2010) The contribution of abdominal obesity and dyslipidemia to metabolic syndrome in psychiatric patients. Korean J Intern Med 25(2): 168-173.

22. Boehm G, Racoosin JA, Laughren TP, Katz R (2004) American Diabetes Association. Consensus Development Conference on Antipsychotic Drugs and Obesity and Diabetes. Diabetes Care 27: 596-601.

23. Bolaños P, Rocio Cabrera R (2008) Influence of psychoactive drugs in body weight. Eating disorder 8: 813-832.

24. Angst F, Stassen HH, Clayton PJ, Angst J (2002) Mortality of patients with mood disorders: follow-up over 34-38 years. J Affect Disord 68(2-3): 167-181.

25. Berken GH, Weinstein DO, Stern WC (1984) Weight gain. A side effect of tricyclic antidepressants. J Affect Disord 7: 133-138.

26. Carnethon MR, Biggs ML, Barzilay JI, Smith NL, Vaccarino V, et al. (2007) Longitudinal Association Between Depressive Symptoms and Incident Type 2 Diabetes Mellitus in Older Adults. The Cardiovascular Health Study. Arch Intern Med 167: 801-808.

27. Zimmermann U, Kraus T, Himmerich H, Schuld A, Pollmächer T (2003) Epidemiology, implications and mechanisms underlying drug-induced weight gain in psychiatric patients. J Psychiatr Res 37: 193-220.

28. Druss BG (2007) Improving medical care for persons with serious mental illness: challenges and solutions. J Clin Psych 68(4): 40-44

29. Fleischhacker WW, Cetkovich BM, De Hert M, Hennekens C, Lambert M, et al. (2008) Cormorbid somatic illnesses in patients with severe mental disorders: clinical, policy and research challenges. J Clin Psychiatry 69: 514-549.

30. Nasrallah HA, Meyer JM, Goff DC, McEvoy JP, Davis, et al. (2006) Low rates of treatment for hypertension, dyslipidemia and diabetes in schizophrenia: data from the CATIE schizophrenia trial sample at baseline. Schizophr Res 86: 15-22.

31. Salazar M, Peralta C, Pastor J (2006) Handbook of Psychopharmacology. Panamericana Editorial. Madrid Spain.

32. Graff GA, Apiquian R, Fresán A, García Anaya M (2001) Neurobiological Perspective of schizophrenia. Mental Health magazine 24(6): 36-42.

33. Mata, Esquiviaz M (2010) Evaluation of the levels of dopamine and dopamine D2 receptors in responses to stimuli motivational reward and punishment in rats. Thesis Mexico D.F.

34. Bahena Trujillo, R Flores, JA G Arias Montano (2000) Dopamine synthesis, release and receptors in the central nervous system. Rev Biomed 11: 39-60.

35. Gomez, R.A (2005) Elements for a Psychobiology. 2nd Edition. Editorial Brujas. Córdoba.

36. Salazar M, Peralta C, Pastor FJ (2009) Psychopharmacology Treaty bases and clinical application. 2nd edition. Panamericana medical publishing. Spain.

37. Santos O, Bourin M (2005) Mechanism of action compared the different antipsychotics. Rev Hosp. Havana Psychiatric 2: 2.

38. Puig MV, Celada P, Atigas F (2004) serotnoninérgico Control of the prefrontal cortex. Rev Neurol. 39(6): 539-547.

39. Derito N, Martinez G, Fernandez L, D Bordalejo (2005) atypical antipsychotics Monchablon A (Part2) Magazine Argentina Neuropsychiatric Clinic 12: 1.

40. Bolaños P, Cabrera R (2008) Influence of psychoactive drugs in body weight. Eating disorder 8: 813-832.

41. G Jufe (2008) Metabolic syndrome induced by antipsychotics, the problem of obesity. Argentina Rev Psychiatry 19: 338-347.

42. Suarez M (2001) Obesity associated with the use of medication antipsychotic. Rev clinical Fondopsi 24: 3-14.

43. Lorenzo P, Moreno A, Lizasoain I, Leza JC, Moro MA, et al. (2008) Pharmacology Basic and Clinical. 18th. Edition. Panamericana medical publishing. Madrid.

44. Moreno J, Campos G, Lara C, Tornet C (2005) The serotonergic system in the depressed patient. First part. Mental Health 28(6): 20-26.

45. Benlloch Ortiz L, Alvarez Lopez P (2003) Antidepressants. Rev Medicine 8(105): 5665-5674.

46. Retamal P (2001) psychopharmacotherapy antidepressant. Rev Méd Chile 129(7): 813-818.

47. González PD, Martin Goméz MC, Franco MP, Sánchez IS (2007) Pharmacological treatment of depression. Medicine 9(85): 5469-5477.

48. Salazar M, Peralta C, Pastor J (2006) Psychopharmacology Manual. Madrid, Spain: Editorial Médica Panamericana.

49. Smith C, Marks A, Lieberman M (2006) Marks Basic Biochemistry 2nd Edn clinical approach. Editorial Mc Graw Hill. Madrid.

50. Mahan LK, Stump S, Krause Dietotherapy Escott 12th Edition. Editorial Elsevier. Spain.