METABOLIC SYNDROME AND ANTIPSYCHOTIC DRUGS — A REVIEW

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ABSTRACT

Metabolic syndrome is characterised by a constellation of obesity, dyslipidemia, insulin insensitivity, deranged glucose metabolism and hypertension. The interface between psychiatry and metabolic syndrome is of importance today due to the increasing recognition of this syndrome among patients receiving psychotropic medications. Antipsychotics and possibly other psychotropic drugs like antidepressants can induce weight gain or worsen other metabolic parameters, particularly increasing the cardiovascular risk factors. Mounting evidence has suggested decreased life expectancy in psychiatric patients by one to three decades. Changes associated with metabolic syndrome appear to play a significant role in them. This article reviews metabolic syndrome in association with psychiatric illnesses, antipsychotic treatment and recent guidelines for its detection and management.

Key words: Dyslipidemia, Glucose intolerance, Antipsychotics, Metabolic syndrome.

INTRODUCTION

Though the earliest use of the term "Metabolic syndrome" (MS) was probably by German researchers in late 1970s in association with atherosclerosis, it was described by Gerald Reaven in 1988 and named as Syndrome X[1]. Metabolic Syndrome also known as insulin resistance syndrome and CHAOS (a mnemonic for Coronary artery disease, Hypertension, Adult onset diabetes, Obesity and Stroke) is essentially a constellation of obesity, dyslipidemia, insulin insensitivity, deranged glucose metabolism and hypertension.

Over the last decade, a number of definitions highlighting specific criteria for diagnosis of MS have been offered. In 2001, the National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) proposed a definition that was revised in 2005 by the American Heart Association/National Heart, Lung, and Blood Institute (updated ATP III)[2]. In the same year the International Diabetes Federation (IDF) proposed another definition[3]. These definitions (Table 1) lay emphasis on abdominal obesity as measured by abdominal circumference of >102 cm for men and >88 cm for women, with the corresponding values being 90 and 80 cm respectively in people of Asian origin. The other criteria being triglyceride levels being elevated beyond 150 mg/dl, high density lipoproteins (HDL) below 40 mg/dl and 50 mg/ dl for men and women respectively, elevated blood pressure >130/85 mmHg and elevated fasting plasma glucose levels >110 mg/dl.

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A major difference between the ATP and IDF definitions is the necessity of central obesity for making a diagnosis. While the updated ATP III definition requires any three of the five criteria for a diagnosis, the IDF definition needs central obesity plus any other two abnormalities.

According to the new guidelines released by the Union Health Ministry of India, a person with a body mass index (BMI) of 23 kg/m^2 is considered overweight, as against earlier 25 kg/m^2. Now, those with BMI of 25 kg/m^2 are clinically termed obese. Changes were made keeping in mind the different body, genetic and metabolic composition of the Indian population[4].

METHODOLOGY FOR THE REVIEW PROCESS

For identifying articles that focused on metabolic syndrome and atypical antipsychotics, the terms 'metabolic syndrome and atypical antipsychotics', 'atypical antipsychotic side effects', 'atypical antipsychotics and diabetes', 'atypical antipsychotics and hyperlipidemia' or 'elevated lipids' were used. These search strategy results were searched in the following data bases with the time frame being specified from 1999 through 2012. The databases used were Medline, Pubmed and the Cochrane Database on Systematic Reviews. In total, 212 articles were identified which included reviews, mini reviews, meta analyses, safety analyses and randomized controlled trials in populations on atypical antipsychotic treatment. The randomized controlled trials reviewed were centered on those addressing metabolic syndrome with antipsychotics.

We included trials and quantitative studies with sample sizes of more than 50 participants and that reported either mean scores or percentages with appropriate statistical analysis. All authors reviewed all of the

Table 1
Diagnostic criteria for metabolic syndrome

Criteria	ATP III (Any three Of five criteria)	IDF (Central) obesity+ Any other two abnormality
Waist Circumference		
Men	>=102	>= 94 (>= 90 for Asian men)
Women	>= 88	>= 80 (Same for Asian women)
Blood pressure (mm Hg)	>=130/85	>=130/85
HDL (mmol/L)		
Men	< 1.03	<1.03
Women	<1.30	<1.30
Fasting blood triglycerides	>= 1.7	>= 1.7
Levels (mmol/ lt)	(>= 150 mg %)	(>= 150 mg %)
Fasting Blood Glucose levels	>= 6.1	>=5.6
		(>= 100 mg%)
	>110 mg%	Or previously
	Or drug	diagnosed
	treatment for	type II
	elevated glucose	Diabetes

articles and the most relevant ones were chosen for this review. The papers reviewed in this article include articles, trials and research papers on atypical antipsychotics and metabolic syndrome. This is supplemented with the personal clinical experience of all the authors in this field who work regularly with this group of patients and have further insight into the problems faced by them. All the authors are psychiatrists working in a tertiary hospital and medical college where there is a regular atypical antipsychotic use and we see on an average 50-70 patients with schizophrenia daily in our out patient department.

METABOLIC SYNDROME AND PSYCHIATRIC ILLNESSES

People with severe mental illnesses, such as schizophrenia, depression or bipolar disorder have worse physical health and reduced life expectancy compared to the general population, losing approximately 25 to 30 years of potential life primarily due to premature cardiovascular mortality. There is a possibility that this mortality gap associated with mental illness has widened in the recent decades compared to the general population[5]. The excess cardiovascular mortality associated with severe mental illnesses is attributed in part to an already increased risk of the modifiable coronary heart disease risk factors i.e. obesity, smoking, diabetes, hypertension and dyslipidaemia in these patients[6-9]. In addition, over the recent years it has become apparent that antipsychotic agents can have a negative impact on some of these modifiable risk factors. Part of this negative impact can be explained by the liability of some antipsychotics to induce significant weight gain and increased risk for adverse changes in glucose and lipid metabolism. A recent study indicates that these metabolic changes are dose independent[10].

Authors have studied the risk of coronary heart disease (CHD) and stroke in addition to lifestyle factors in 102 patients with schizophrenia and reported either obesity or overweight in 70% of male patients and 86% of female patients. The mean 10-year risk of coronary heart disease was increased (9.6%) compared to the general population (6.4%), as was the risk of stroke (4.1%)[11]. The cause of increased cardiometabolic risk in this population can include non disease-related factors such as poverty and reduced access to medical care. Patients may have less opportunity for cardiovascular risk screening and prevention than would be expected in a non-psychiatric population. Factors such as life style, poor diet, lack of exercise, high rates of substance abuse and smoking may be contributory too[12-14]. Research suggests an over-activity of the hypothalamic-pituitary-adrenal axis leading to hypercortisolaemia can also result in excessive visceral fat accumulation[15].

Due to the increasing concern surrounding metabolic derangements in patients with severe mental illnesses, the role of psychiatrists in the physical healthcare of their patients has been the focus of recent attention. A study found inadequate recording of physical health pa-

rameters and risk factors in 63 patients under the care of a rehabilitation and recovery team and discusses the need to routinely monitor physical and lifestyle factors[16]. Some authors have also discussed the challenge of including improvement in physical health, alongside that in mental health, in treatment plans by taking into account both medication-related and lifestyle factors[17].

METABOLIC SYNDROME AND ANTIPSYCHOTICS

There has been a gradually increasing concern about the contribution of antipsychotic medication to the prevalence of the metabolic syndrome and its components, especially since the introduction of atypical antipsychotics.

Studies report an increased prevalence of metabolic syndrome and cardiovascular risk in 90 people treated with antipsychotics, compared to age and gender matched controls[18]. BMI, disorders of lipid and glucose metabolism and risk for cardiovascular disorder were increased in the mentally ill. In a study of 367 adults treated with second generation antipsychotics, it was reported that metabolic syndrome was present in 137 (37.3%) patients and was significantly associated with the 10-year risk of Coronary Heart disease events[19]. As per another study, using ATP III criteria, metabolic syndrome was diagnosed in 13 (37%) out of 35 patients with schizophrenia treated with antipsychotic medication[20]. A few studies also demonstrate that there was no significant difference in glycemic parameters with antipsychotic drugs[21, 22].

Derangement of glucose metabolism

A number of studies have demonstrated changes in insulin and fasting glucose levels with antipsychotic therapy less with typical and more so with atypical antipsychotics. Following the introduction of phenothiazines in 1952, there have been reports associating phenothiazine treatment with abnormal glucose tolerance and an increased predilection to develop diabetes. In one such study[23], it was demonstrated that treatment with antipsychotics was associated with significant increases in fasting glucose (mean change from baseline 6.6 mg/dl with olanzepine, 4.3 mg/dl with risperidone, 6.9 mg/dl with haloperidol., $P<0.01$ for combined treatment group vs control) and glucose post-OGTT (mean change from baseline 21.6 mg/dl with olanzepine; 21 mg/dl with risperidone; 6.8 mg/dl with haloperidol, $P<0.001$ for combined treatment group vs control).

Another study reported that 10.1% of patients developed diabetes mellitus after only six weeks of antipsychotic therapy ($P=0.016$). Given the higher glucose levels post-OGTT at baseline, this study demonstrates that patients with schizophrenia may have an inherent tendency toward glycemic abnormalities which can develop into frank diabetes with antipsychotic therapy[24]. It has been found that olanzapine was associated with a significant risk of diabetes compared to risperidone and conventional antipsychotics in a number of studies[25]. Authors have reported a case of olanzapine-induced diabetic ketoacidosis (DKA), which resolved following discontinuation of olanzapine treatment[26]. Authors have also used a national database to study the prevalence of diabetes mellitus in patients receiving atypical and typical antipsychotics over a 4 month period and found that the odds of being diabetic was increased for clozapine, olanzapine and quetiapine but not for risperidone, when all age groups were considered[27].

Analyzed data from a randomized, double-blind trial of clozapine, olanzapine, risperidone and haloperidol in 108 patients with schizophrenia or schizoaffective disorder showed that blood glucose levels were significantly increased in patients treated with clozapine and haloperidol after 8 weeks and in patients treated with olanzapine after 14 weeks treatment. However the increases remained within clinically normal ranges and did not correlate with the significant weight gains seen[28].

It has been reported that two patients treated with clozapine subsequently developed diabetes mellitus on routine blood testing. Blood sugar level returned to within the normal range after discontinuation of clozapine in one of the patients, but not in the other. Clozapine may contribute to insulin resistance by different mechanisms: by decreasing uptake of glucose in brain and peripheral tissue as well as by impaired â cell function. They stress the need for baseline measurements prior to and following initiation of treatment with clozapine[29].

It is worth noting that there are relatively few reports describing an association between diabetes mellitus, quetiapine, risperidone and ziprasidone. The limited reporting for quetiapine and ziprasidone may be related to the relatively limited use of these agents currently[30, 31]. Even though risperidone has received extensive use, it has triggered relatively few reports of an association with diabetes mellitus including one report of hyperglycemia and 1 report of diabetic ketoacidosis in an HIV infected man. Hence, as reported, the collective data consistently implicate olanzepine and clozapine as being associated with increased insulin resistance, whereas amisulpiride and ziprasidone have been demonstrated to have improved glycemic parameters[24]. Risperidone & haloperidol have a neutral effect at best but negatively affect glucose insulin homeostasis. Ziprasidone has been demonstrated to improve HbA1c levels[32, 33].

DERANGEMENTS IN LIPID METABOLISM

Dyslipidemia is an important component of the metabolic syndrome, occuring along with glucose dysregulation and weight gain in patients being treated with antipsychotics; the risk being more with atypical antipsychotics than with typicals. The mechanism of dyslipidemia with atypical agents is poorly understood, but atypical antipsychotics have been shown to increase lipogenesis, reduce lipolysis and enhance the antilipolytic

effects of insulin in adipocytes; leading to a net effect of lipid accumulation in adipocytes.

Authors suggest that the pathogenesis of hyperlipidemia is related to weight gain, accumulation of abdominal fat, and increase release of free fatty acids in the liver and accelerating hepatic triglyceride synthesis as well as very low density lipoprotein (VLDL) release. They further suggest that increased lipids impair glucose metabolism, leading to hyperglycemia and Type 2 DM[34].

It has been reported in randomized trials using placebo, and a number of active comparators that olanzapine is associated with elevated lipid levels. Active control agents in these studies have included haloperidol, risperidone and clozapine, although prior antipsychotic use was not excluded, which can be an important confounding factor[35, 36].

Researchers[37] re-examined the lipid profile of 9 patients with schizophrenia, after initiating treatment with olanzapine. Though they did not observe a change in cholesterol or lipoprotein levels, the level of triglyceride increased from a mean of 170 mg/dl to 240 mg/dl. However in another study[38] fasting cholesterol was raised in 26% of patients, along with elevated fasting triglycerides in 55% of antipsychotic treated patients.

In a discussion on the large Clinical Antipsychotics Trial of Intervention Effectiveness (CATIE) study it was indicated that olanzapine treatment was associated with the greatest adverse effect on metabolic endpoints like body weight and plasma triglyceride, and increased prevalence of a metabolic syndrome diagnosis at 3 and 9 months in contrast to other tested agents such as perphenazine and ziprasidone which produced limited adverse metabolic effects in the sample studied or even improvements in some metabolic parameters[39]. This observation is consistent with a growing number of experimental and larger-scale pharmacoepidemiologic studies[40, 41].

A research study compared 185 patients with schizophrenia who had been switched from olanzapine, risperidone or conventional antipsychotics to ziprasidone for 1 year. Within 6 weeks of switching to ziprasidone, there were significant improvements in total cholesterol and triglycerides reported in these patients[42]. Various studies[43-45] mention that olanzapine is associated with unfavourable lipid arrangements when compared with aripiprazole. Studies have also shown that risperidone has favourable effects on total cholesterol compared with olanzapine and placebo[46]. Amongst the typical antipsychotic group, certain studies mention that though some agents (eg, haloperidol) have no effect on lipids; phenothiazines (eg, chlorpromazine) tend to raise triglyceride levels and reduce levels of high-density lipoproteins[47].

To conclude, just like with glucose abnormalities, olanzapine has the greatest propensity for causing proatherogenic hyperlipidemia. Trial evidence for other antipsychotics and dyslipidemia is less consistent. Haloperidol and quetiapine may elevate lipid levels, whereas risperidone may increase or decrease lipids. Ziprasidone, however, is likely to improve lipid parameters[24].

WEIGHT GAIN

Weight gain in metabolic syndrome primarily refers to the visceral adiposity and is measured by waist circumference. Atypical antipsychotics are known to cause changes in weight in psychiatric patients in the course of their treatment. Some researchers performed a comprehensive review of research literature to estimate and compare the effects of both conventional and atypical antipsychotics on weight gain[48]. This was followed by meta-analysis, with estimated mean weight change calculated using both fixed and random effects models. Weight gain associated with five atypical antipsychotics was examined in the study – ziprasidone (0.04 kg), risperidone (2.10 kg), sertindole (2.92 kg), olanzapine (4.15 kg) and clozapine (4.45 kg). Though the two conventional antipsychotics molindone and pimozide were associated with weight loss, the effects were not significant at 10 weeks. The study indicated that patients may gain more than 5% of initial body weight, with the weight gain becoming more pronounced with time, increasing thereby the risk of related physical comorbidities.

As per another study, switching from one antipsychotic medication to another can have significant effects on body weight and metabolic parameters[49]. In this context, we report a study of overweight or obese (BMI > 26 kg/m^2) olanzapine-treated patients[50]. They were switched to risperidone and followed for 20 weeks. 32% reductions in the rate of the metabolic syndrome at study endpoint, as well as significant reductions in body weight, BMI, waist circumference, and blood pressure were reported. Unlike extrapyramidal side effects, antipsychotic weight gain does not appear to be heavily influenced by dosage within the usual therapeutic range. However, another study[51] with clozapine showed different results. The study combined low-dose clozapine with fluvoxamine, which inhibits clozapine metabolism and raises clozapine plasma levels. In a 12-week RCT, inpatients with treatment-resistant schizophrenia were assigned to high-dose clozapine (600 mg daily) or low-dose clozapine (250 mg daily) plus adjunctive fluvoxamine (50 mg). At study endpoint, only the high-dose clozapine group had elevations relative to baseline body weight, fasting glucose, and triglycerides.

Aripiprazole is an atypical reported to cause little or no weight gain. Researchers have compared aripiprazole and olanzapine in a 26 week multi-center randomized controlled trial, especially in terms of significant weight gain. By the end of the trial, 37% of olanzapine-treated patients reported significant weight gain ($p<0.001$) compared to a mere 14% of patients treated with aripiprazole[52]. Treatment with olanzapine was associated once again, with a worsening of the lipid profile.

There are certain issues in interpreting research literature on weight gain. These include the duration of trials, differing outcome measures used, failure to consider baseline BMI and limited information on confounding variables. It is important to consider all the available information (efficacy, tolerability and patient's previous response if known) before deciding on which antipsychotic to prescribe[53].

HYPERTENSION

There are very few studies reporting the effect of antipsychotics on blood pressure[54]. Nevertheless, hypertension is one component of the metabolic syndrome which is not as commonly associated with treatment with atypical antipsychotics. Studies reported a prevalence of 29% for hypertension in 208 patients treated with antipsychotics[55].

MANAGEMENT OF METABOLIC SYNDROME: PSYCHIATRIC PERSPECTIVE

There is now a consensus that antipsychotic medications are associated with various degrees of weight gain and metabolic disturbance[12]. Modifying lifestyle factors and treating individual components of the metabolic syndrome is suggested for those affected. Psychiatrists have an important role to play in managing various metabolic complications associated with antipsychotic use. To understand the management of metabolic syndrome, we can categorize it under the following three headings

- ***Screening:*** Do a baseline screening of blood parameters, physical examination, & maintain regular follow up for patients on antipsychotics.
- ***Prevention***: Weight management and lifestyle advice should be offered to all patients to prevent metabolic abnormalities from developing.
- ***Intervention***: Treatment of individual components of metabolic syndrome using a combination of pharmacological and non pharmacological interventions.

SCREENING

Screening simply refers to a strategy used in a population to detect a disease in individuals without signs or symptoms of that disease. The association between mental illness, psychotropic medications and increased cardiometabolic risk, suggests a role of psychiatrists in monitoring risk in relation to prescribing antipsychotic medications. Modifiable risk factors that should be evaluated at or near baseline and serially after prescription of antipsychotics include

- Weight
- BMI
- Waist circumference
- Fasting plasma lipids (total, LDL, HDL, and triglycerides)
- Fasting and/or postprandial plasma glucose
- Blood pressure

In addition, a personal and family history of obesity, diabetes, dyslipidemia, hypertension or cardiovascular disease should be taken account of.

Laboratory parameters should be monitored periodically (table 2), paying particular attention to changes in any value after initiation of a new antipsychotic agent[4]. The American Diabetes Association (ADA) (2004) recommends weight monitoring at 4, 8, and 12 weeks after initiating a patient on antipsychotic therapy, and quarterly thereafter. A weight gain of >5% of baseline weight may signal the need to switch to a different atypical antipsychotic agent. In addition to monitoring by physicians, patients should be encouraged to track their own weight and waist circumference[56].

The Mount Sinai guidelines suggest that fasting glucose or glycated hemoglobin (A1C) could be used for glucose monitoring. However, the ADA recommendations for screening in the general population advise against

Table 2: Monitoring patients on antipsychotic drugs (APDs) for metabolic syndrome.*

Parameters	Suggested frequency	Action to be taken if levels are deranged
Blood lipids including triglycerides, & cholesterol.	Baseline, 3months then yearly	Non-pharmacological measures, such as exercise, dietary changes. Consider change of APD &/or lipid lowering drugs.
Plasma glucose then yearly	Baseline, 6 monthly,	Non-pharmacological measures, such as exercise, dietary changes. Consider change of APD &/or lipid lowering drugs.
Blood pressure	Baseline, every fortnightly during initial follow-ups, then intermittently	Non-pharmacological interventions. Manage hypertension with antihypertensives if the APD cannot be changed. Slow rate of titration of APD. Educate patient about measures to be taken if hypotension.
Body weight	Baseline, fortnightly for three months, then yearly	Non-pharmacological measures, such as exercise, dietary changes Consider change of APD or weight reducing drugs (see text for details).

the use of A1C because of its relative insensitivity as a screening measure. The introduction of regular routine monitoring should allow for the early detection of changes in these risk factors, and allow for improvement in the overall long-term health of patients with schizophrenia and other mental illnesses.

Hence, screening enables us to detect derangements in metabolic parameters at an early stage in our patients before symptoms present. Consequently, the treatment is more effective and in the best of cases, lives are saved.

PREVENTION

'Healthy lifestyle promotion' is recommended as the primary management for metabolic syndrome by the IDF, comprising of calorie restriction, increased physical activity, abstinence from smoking and dietary modification[57]. Because insulin resistance is a feature associated with schizophrenia independent of any specific drug treatment, lifestyle advice should be given to all patients with a diagnosis of schizophrenia. This should start immediately as part of the package of care at the first onset of illness. It is also hypothesized that a diet low in saturated fat and glycemic load, but high in polyunsaturated fatty acids might be beneficial in alleviating the symptoms of schizophrenia.

Unfortunately however, there isn't sufficient evidence still to prove the best way to achieve lifestyle changes in a patient with schizophrenia. A study that reviewed all the studies assessing the behavioral management of antipsychotic-induced weight gain, including both dietary and exercise interventions found that current studies were methodologically flawed, with none meeting the criteria for a randomized controlled trial[58]. Approaches aimed at the management of obesity in the general population[59] are not necessarily transferable to people with schizophrenia. More research in this area is warranted.

INTERVENTION

Treatment of individual components of the metabolic syndrome using medication and behavioral approaches is advised for people not responding to lifestyle interventions.

Pharmacological strategies

Of all the pharmacologic strategies, choice of psychotropic medication may have the greatest influence on weight gain and associated metabolic disturbance. It has been suggested that one must use antipsychotic agents with little or no propensity to cause weight gain, diabetes or dyslipidaemia, to use the lowest effective dose, routinely asking for symptoms of diabetes and monitoring for diabetic symptoms and being aware of factors such as certain ethnic populations[60]. Even measuring serum antipsychotic levels is recommended to optimize dose. Switching from one antipsychotic medication to another can have significant effects on body weight and metabolic parameters. Data from the recently reported CATIE trials provide evidence of beneficial weight change after switching to an antipsychotic medication having a more neutral metabolic profile.

Authors have reported that weight and metabolic benefit depended on which drug patients were switched from. Those switched from olanzapine experienced the greatest metabolic benefit, with significant reductions in body weight (1.76 kg), non fasting total cholesterol, and triglycerides. Those switched from risperidone experienced less weight loss (0.86 kg) but similar reductions in non fasting total cholesterol and triglycerides. However, those switched from conventional antipsychotics had no change in weight or lipid measures[61].

Other researchers highlight that discontinuing or switching antipsychotics may prove to be critical, especially when the development of diabetes or diabetic ketoacidosis can be clearly linked to a new antipsychotic prescription. Discontinuation or switching the antipsychotic can lead to resolution of diabetes in this situation[62]. Others mention that in the absence of treatment directed towards metabolic syndrome as a whole, treatment of individual conditions is the preferred option. Both the consensus statements (IDF and AHA/ NHLBI) provide guidance of optimal treatments for hypertension, diabetes and dyslipidaemia as well as advice on lifestyle risk factors such as obesity, physical inactivity and atherogenic diet. The AHA/NHLBI statement goes further in advising low-dose aspirin therapy/prophylaxis for the prothrombotic state, which has been recognized as part of the syndrome[54].

In a comprehensive review, eight different medications to treat obesity were studied. These include orlistat, sibutramine, fluoxetine, topiramate, amantadine, nizatidine and cimetidine, and metformin, of which the first two are licensed for treatment of obesity in the UK. The underlying receptor mechanisms include antagonism of serotonergic, dopaminergic, histaminergic and glutaminergic receptors. Other mechanisms involve neuropeptide Y, cholecystokinin and leptin. Both orlistat and sibutramine were effective in double-blind RCTs in which subjects in the treatment arm lost 7 – 10 kg in weight, compared to 3 – 6 kg in the controls. Treatment with fluoxetine or topiramate has been associated with dose-related weight loss, though evidence from RCTs is not available[63]. Other drugs such as the noradrenaline reuptake inhibitor reboxetine have been studied as well. In a 6 week double-blind, placebo-controlled, randomized study of two groups of 10 patients each (treated with olanzapine or placebo), patients in both groups gained weight during the trial, but those receiving olanzapine and reboxetine gained significantly less weight (mean=2.5 Kg) compared to patients treated with olanzapine and placebo (mean weight gain of 5.5 Kg).

The beneficial effects of small amounts of weight loss (5% of body weight in obese subjects) or even 1% weight loss in overweight individuals and the resultant reduction in hypertension have been noted[64].

Behavioural interventions and their role

Authors have studied the role of behavioral interventions in management of weight gain and found that education, weight monitoring, dietary changes, cognitive strategies and community based education programmes were found to be effective[58]. Longer-term studies show persisting benefits of behavioral interventions. A study has shown that over a three-year period, a behavioral intervention comprising weekly weight monitoring along with nutrition discussion and education sessions was found to be associated with a 5 kg weight loss and 2 point reduction in BMI[65].

Authors have discussed use of the technique of Transcendental meditation (TM) as a behavioral intervention in metabolic syndrome[66]. It decreased insulin resistance, systolic blood pressure and mean arterial pressure. This technique allows the mind to settle inward to the most silent and peaceful level of consciousness, which is associated with significantly increased EEG coherence and physiological rest. TM was shown to increase the Heart rate variability, indicating an improvement in cardiac autonomic nervous system tone, and decreasing the overall cardiovascular morbidity and mortality.

CONCLUSION

Metabolic side effects of atypical antipsychotic drugs are a common place today in routine psychiatric clinical practice. Management of and recognition of these problems are a must for the busy clinician. This review has analyzed metabolic syndrome from various perspectives and serves to add to the existing knowledge on this subject from a psychiatric point of view.

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