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Research Article

Antihypertensive/Glycemic Effects of Escitalopram: A Selective Serotonin Reuptake Inhibitor Opens a New Window for Researchers

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Abstract

Background: Hypertension is a global cause of numerous cardiovascular illnesses affecting eyes, brain, heart, vessels and kidneys. Various treatment agents have been produced by the researchers but the story still revolves around the compliance worldwide from the 3rd world nations to UK and USA. We just tried to monitor the additional blood pressure effects of a famous antidepressant Escitalopram in depressed patients under treatment.

Methodology: Consultants of Sir C.J Institute of Psychiatry provided us Patients of depression on escitalopram treatment. Blood pressure measurements were done by digital and mercury apparatus on day one under signature for willing participation for study. Patients were counseled for diet and medicine, exercise and Follow up. Blood pressure was measured on different follow up visits and finally after 6wks.

Statistical Analysis: Student's t-test (Pair Sample), SPSS Version 21, Calculated and compared the pre and post mean values of the data including minimum and maximum.

Results: We were surprised to note the reduced systolic and diastolic blood pressure having pretreatment mean of 125 + 8.7 mmHg and 78.04 + 6.3 mmHg to 111.4 + 8.2 mmHg and 65.93 + 6.2 mmHg systolic and diastolic respectively. P-value was 0.001 for both parameters.

Conclusion: Escitalopram possess antihypertensive properties that need further clarification regarding the mechanism responsible for such effects may be the Serotonin receptors, sympathetic out flow inhibition, some interference with serum electrolytes like sodium or RAAS.

Keywords: *RAAS; Serotonin; Escitalopram; Antihypertensive*

Introduction

Hypertension has long history behind it and still remains a mystery to be discovered for 95% of the patients regarding its etiology. Advancements in science research and technology has provided the hypertensive population a large variety of drugs interfering with a variety of targets. Pharmacological groups of antihypertensive agents include 1. Beta adrenergic blockers (Selective and nonselective), 2. Alpha adrenergic blockers (Reversible and nonreversible), 3. Calcium Channel blockers (Vessel specific and cardio specific), 4. ACE Inhibitors (Angiotensin Converting Enzyme Inhibitors), 5. ARBs (Angiotensin Receptor Blockers), 6. Renin Blockers, 7. Centrally acting agents

(Clonidine, methyldopa), 8. Nerve blocking agents, 9. Vasodilators, 10. Diuretics, 11. Tyrosine Hydroxylase Inhibitor (Metyrosine) [1,2]. Despite of this long list, the blood pressure control is poor due to non-compliance which varies from region to region with china, Russia, Germany, Italy, France, UK, USA and other countries of the world. Expected factors consist, genetic, environmental, lifestyle and stress and its coping strategies [3]. Secondary HTN has well defined etiology like tumor of the adrenal gland, renal artery stenosis, thyroid over activity (Hyperthyroidism), steroid misuse etc. Complication of the systemic HTN are organ specific in eye it causes hypertensive retinopathy in heart it leads to ischemic heart disease (47%) in brain may result in stroke (54%) [4]. Hypertrophy of the left ventricle (Cardiomyopathy), Hypertensive nephropathy is damage to the kidney parenchyma due to persistent elevated pressure and premature death may result from HTN. Who expects 1.56 billion hypertensive adults by 2025 [5]. JNC 8 guideline are the best for the treatment of hypertension specially its recommendations about the selection of a particular group and combination [6] is the expertise of the physician according to goals. Stress and depression is among the causes of raised blood pressure. High sodium intake is one of the contributors to the hypertension any agent that reduces the blood Na concentration possess the antihypertensive potential. Sympathetic over stimulation if persists for longer duration of time makes the patient hypertensive. Drugs that affect many targets responsible for elevation in blood pressure may prove superior to those that are directed to a single target. So, we tried to observe some beneficial effects of a well reputed and well used antidepressant, escitalopram having broad spectrum for uses [7] on blood pressure and blood glucose in depressive subjects using escitalopram for their depression.

Methods

Study consisted large number of parameters to be evaluated in depressive patient under the approval of PHRC (Pakistan Health Research Council) following ethical committee approval. Patient selection was done by the Psychiatrist from Sir C.J Institute of Psychiatry under informed written type of the consent. Recording of blood pressure according to international guidelines after ruling out the previous sickness and drug usage was assured. Tab escitalopram 10 mg/day/oral were prescribed initially raising to 20 mg/day subsequently according to response in depressive symptoms. Follow up was strictly observed for compliance and diet and exercise. Hypertension was excluded on startup screening similarly the diabetes mellitus. Final Fasting blood glucose and blood pressure were revaluated at an interval of 6 weeks of therapy. Further Detailed methodology was published previously [8].

Results

Surprising results were recoded for antihypertensive and glucose reducing properties of escitalopram. Pre and post medication status of patients for systolic blood pressure was 125 + 8.7 mmHg and 111.4 + 8.2 mmHg respectively with p-vale 0.001 while for diastolic pressure they were 78.04 + 6.3 mmHg and 65.93 + 6.2 mmHg respectively (p = 0.001). Mean of FBS were 78.46 ± 12.32 mg/dl and 68.92 ± 12.40 mg/dl pre and post therapy at highly significant P-value 0. 0001 The minimum recoding was 60 mg/dl pretreatment while the same was noted to be 40 mg/dl after 6 weeks advising treatment. The maximum was found 108 mg/dl and 109 mg/dl respectively for pre and post treatment FBS.

Observations	Reading at day 01 N=64	Reading after 6wks N=64	P-Value
Mean SBP	125 mmHg	111.4 mmHg	0.001
S.D	8.7 mmHg	8.2 mmHg	
Mean DBP	78.04 mmHg	65.93 mmHg	0.001
S.D	6.3 mmHg	6.2 mmHg	
Mean FBS	78.46 mg/dl	68.92 mg/dl	0.0001
S. D	12.32 mg/dl	12.40 mg/dl	

Table 1: *Difference between pre and post treatment blood pressure with escitalopram.*

SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; S.D: Standard Deviation

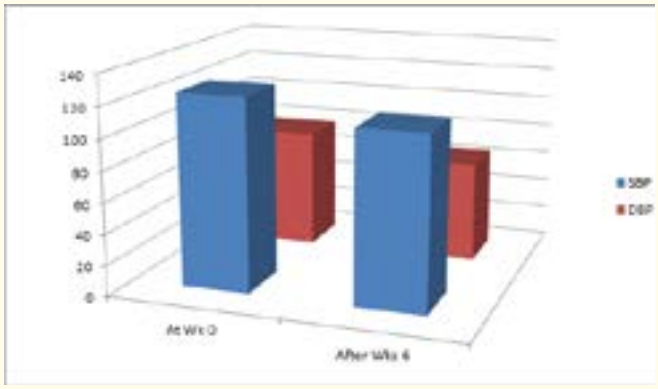

Figure 1: *Bar Charts of blood pressure changes during the study.*

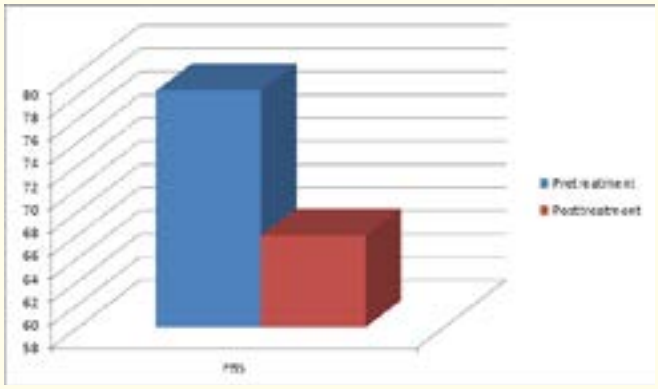

Figure 2: *Bar chart of changes in Fasting blood sugar during study period.*

Discussion

Changes is systolic and diastolic blood

M Beyazyuz., *et al.* (2013) also reported resembling outcomes in his work regarding the blood pressure effects of escitalopram [9]. There was marked reduction from pre to post value of both the systolic and diastolic blood pressure in our study. This Reduction of pressure may attribute to reduction of sympathetic flow that is usually increased in depression as well as a cause of HTN. Other possible explanation to this decrease in blood pressure is the SSRIs effects on serum sodium concentration as this group of antidepressants is believed to cause Hyponatremia [10]. However, the reason remains still unexplained either some potential diuretic effects or some effects on aldosterone. We left this gape for other to dig into what is the extent of electrolytes changes brought by the under observation drug.

Changes in Glucose levels (FBS)

A very recent study by Jana Radojkovic., *et al.* (2016) emphasized on reduction in HbA1C along term representative of good or bad glycemic control in his diabetic subjects. He concluded a significant decrease in HbA1c in patients taking escitalopram. Our results are consistent with the quoted study with exception of study population he studied the diabetics while we studied the depressive ones. Study showed a significant decrease in fasting blood glucose levels which is in agreement with the results of who concluded a marked reduction in HbA1C levels after treatment with Escitalopram [11]. His study population was diabetic patients so he preferred HbA1C while our subjects were non-diabetic so we preferred fasting blood sugar. Resembling findings were declared by Gehlawat., *et al*. (2013) and

Dhavale (2013) making our results supportive and stronger [12,13]. M Beyazyuz., *et al*. (2013) showed non -significant results for glucose control that was in contrast to what we declared [9]. The most probable cause could be that his subjects were having poorly controlled diabetes mellitus. Mehnaz G., *et al*. (2013) in her animal model suggested the SSRIs of affect the glycemic status of Rats [14]. The probable justification to glycemic effects of escitalopram may be its effects on insulin secretion or on enhancement of sensitivity at insulin receptors enhancement levels seems to be due to insulin secretion or increasing its sensitivity at receptor level. It may also be serotonin mediated effect still inviting the researchers to probe into some other new aspects of this drug.

Conclusion

Escitalopram possess antihypertensive and antiglycemic effect in addition to its antidepressant actions.

Recommendations

1. Further research is recommended to measure the electrolyte changes brought about by escitalopram and serum aldosterone levels to explain the mechanism behind the additional effects.
2. Insulin levels need to be evaluated in animal models in search of hypoglycemic mechanism.
3. Its use in depressive hypertensive and diabetic patients is recommended on superiority bases when compared to other antidepressants.

Acknowledgement

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Conflict of Interest

No such issue lies between the authors.

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