ISA Journal of Medical Sciences (ISAJMS)

Homepage: https://isapublisher.com/isajms/

Volume 3, Issue 2, March-April, 2026 ISSN: 3049-1746

Evaluation of Renal Function of Schizophrenic Patients Treated With Haloperidole and Olanzapine in Benin City, Edo State, Nigeria

Charity Obasuyi[1,2]; Augustina Chika Odili Isabu[2]; John Osamede Obasuyi[3]; Ambrose Lamani[4] & Nosakhare Lawrence Idemudia[3]

[1]*Department of Nursing Services, Federal Neuropsychiatric Hospital, Uselu, Benin City.*
[2]*Departent of Mental health and Psychiatric Nursing, Faculty of Nursing Science, Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria.*
[3]*Department of Medical Laboratory Services, University of Benin Teaching Hospital, Benin City, Nigeria.*
[4]*Department of Clinical Services, Federal Neuropsychiatric Hospital, Uselu, Benin City, Nigeria*

Received: 15.02.2026 | **Accepted:** 23.02.2026 | **Published:** 02.03.2026

***Corresponding Author:** John Osamede Obasuyi

DOI: *10.5281/zenodo.18834060*

Abstract **Original Research Article**

There is growing evidence that long use of haloperidol and Olanzapine, a first and second generation antipsychotic drugs respectively have some variation effects on the renal function. Available reports seem to be conflicting, and also coupled with fact that these claims have not been sufficiently evaluated in our clime. This study was conducted to evaluate the effects of these drugs on renal function of schizophrenic patients being treated in Benin City, Nigeria. A total of 104 patients were recruited for the study. The 104 patients at the baseline comprises of 51(49.04%) males and 53(50.96%) females from whom baseline blood samples were collected as control samples, after which patients were placed on standard doses of Olanzapine or Haloperidol respectively and blood samples were collected at subsequent study visits being 1st, 2nd and 3rd months of drugs treatment for renal function test which comprises of urea, creatinine and electrolytes (sodium, potassium, bicarbonate and chloride) using a standard laboratory techniques. There were attritions along the line of study as some of the patients missed appointments while, others declined further participation. Of the 104 participants, 92 and 12 were placed on Olanzapine and Haloperidol respectively. The attrition rates for Olanzapine were 20.7%, 42.5% and 14.3% for visits 1st, visits 2nd and visits 3rd respectively while that for Haloperidol were 25%, 33.3% and 0% on the other hand. The ages of the participants ranges from 18 to 77 years, the mean age 37, with age group 28-37 are the most frequent (43.27%), while age groups 56-67, and 68-77 with 1.92% each were less frequent. Patients treated with Olanzapine were found to have sodium levels significantly raised from first month through the third months of treatment ($p<0.0001$), while bicarbonate levels were significantly decreased from second month to third month on treatment ($p<0.05$). Blood urea, creatinine, potassium and chloride levels were unaffected for both patients treated with Olanzapine and Haloperidol.

Keywords: Haloperidol, Olanzapine, Renal function, Schizophrenia.

INTRODUCTION

Schizophrenia is a severe and life-long neurodevelopment sickness that impairs how an individual behaves, reasons or feels. Persons suffering schizophrenic disorder may experience hallucinations, disoriented or incoherent speech and behaviour, delusions and imperfect cognitive ability. In addition, they may observe things that are not there or hear voices that do not exist. They may think another person or individuals around them are reading their thoughts, or trying to manipulate their minds or even planning to hurt them. These observable behaviours can be frightening and disturbing to the people suffering this disorder and it makes them withdrawn or exceptionally agitated, National Institute of Mental Health (1).

The World Health Organisation (WHO) states that Schizophrenia ranked the 10th most common non-fatal ailment globally (2). Though, the annual incidence is low, because of the chronic pattern of the illness the life-long prevalence is about 1%, (3). The signs of the disorder normally appear about the ages of 16 and 30. In few cases, children can develop Schizophrenia too (1).

Nigeria is regarded as the most populous African country with an estimated population of over 200 million people, (4). In Nigeria, an estimated report revealed that about 1.86 million people are living with Schizophrenic disorder, (5). In an hospital based study carried out by Chukwujekwu on "the prevalence, patterns and correlation of Schizophrenia among out-patient clinic attendees" revealed a prevalence rate of 21.9% (6). The report of Oluyomi *et al.*, (7) shows 7.7% prevalence of Schizophrenia in developing nations, Nigeria inclusive. From the study, the onset was reported to occur during childhood, while prevalence was low among older ones that are above the ages of 45years.

The key method of treating Schizophrenia is the use of antipsychotic medication, which is used as a therapy to maintain relapse prevention. Globally, the second generation antipsychotics (SGAS) are most commonly used in managing schizophrenia (8) and (9), while in some countries, the first generation of antipsychotics (FGAS) are drugs of choice most frequently (10). Amongst the SGAS, Olanzapine is the most commonly used due to its broad spectrum and efficacy in Schizophrenia management and other types of psychotic disorder. Compared with first generation antipsychotics, the lower extrapyramidal and tardive dyskinesia side effect is one of its advantages. However, the metabolic dyslipidaemia and the corresponding gain in weight increases the probability of cardiovascular disease (11).

The exact mode of action of Olanzapine is not cleared or known, but it is said to have a wide array of receptor preferences that explain the clinical and adverse effects. Olanzapine act as an antagonist that binds with high affinity to serotonin, dopamine, histamine, and adrenergic (a1) receptors. It is an antagonist with mild affinity binding for serotonin (5-HT$_3$) and muscarinic M1-5 receptors. The pharmacological effects Olanzapine are probably mediated via antagonism at dopamine and 5-HT2a receptors with antagonism at other receptors resulting in side effects, especially the muscarine M3 receptor that has been linked to increased risk of diabetes (12).

Haloperidol is among the most regularly used antipsychotic medications globally. It is a first-generation antipsychotic (FGAS) drug. It is very potent in the management of Schizophrenia. Haloperidol interferes with the activities of neurotransmitters in brain. It blocks receptors serotonin type 2 and dopamine, so the nerves are not activated by these neurotransmitters (13). Haloperidol has been reported to impair liver function. Report also indicates that Haloperidol causes short-lived leukopenia and neutropenia, less reduction in red blood cells count, anaemia or a tendency toward lymphomonocytosis (14).

In Nigeria, haloperidol, which belong to the first generation antipsychotic drugs and olanzapine a second generation antipsychotic drug are commonly and frequently used in schizophrenia management (15). These drugs have been reported to have effects on the renal functions of patient on a long term therapy [(15); (16)]. These claims have not been sufficiently evaluated in our clime as there are no

studies to substantiate the consequences of the prolong use of these antipsychotic agents in Nigeria neuropsychiatric hospitals in particular the study location (Benin City).

Materials and methods

This research is a longitudinal experimental study comprises of pre-test or before drug intervention, post-test one month, two months and three months drug intervention. Patients diagnosed of Schizophrenic mental condition placed on haloperidol or olanzapine were selected for this work. A base line (pre-test drug administration) blood sample was collected as controls before commencement of treatment, followed by a repeat of collection after one, two and three months of treatment with these drugs. A defaulter or a non-drug compliant patient was excluded from the work. All patients were duly informed of the processes and the commitment required from them at the very onset of their enrolment. Patients with known renal, liver, diabetes mellitus, on drugs that are known to impaired renal function were also excluded from the study.

Sample Size determination

Sample size was determined by the formula of Taykaran and Tamogha (17)

$$n = \frac{z^n p_q}{d^2}$$

Where

n = sample size

z = critical value at 95% confidence level,

p = prevalence

q = 1-p

d = precision of 5% (0.05).

Assuming a precision of 7.7% prevalence of schizophrenia in developing nations, Nigeria inclusive, (7).

$$n = \frac{(1.96)^2 \times 0.077 \times (1-0.077)}{(0.05)^2}$$

$$n = \frac{3.8416 \times 0.077 \times 0.923}{0.0025}$$

$$n = \frac{0.2730263536}{0.025}$$

n= 109

A total of 109 schizophrenic patients were recruited for this study, those that are not in any medication before or pre hospital visit. In the process, 5 patients who later decline from participation after the initial consent were dropped from the study. The work now began with 104 participants. An initial sample (baseline) were collected to serve as controls before the patients commenced 10mg/day Haloperidol or 20mg/day Olanzapine and a follow up samples collection at one, two and three month interval respectively. A structured and validated questionnaire was used to collect data on socio-demography.

Inclusion Criteria: All clinically diagnosed schizophrenic patients to be treated with Olanzapine or Haloperidol were recruited from 18 years and above. In addition to the above, patients who willingly consented to participate were recruited.

Exclusion Criteria: Participants with other known infectious disease, pregnant women, those less than 18 years of age, patients with other psychotic disorder, patients having history of epilepsy, mental retardation, substances abuse disorders or any other organic brain disease as well as patients with any other medical illness contraindicating the use of haloperidol and Olanzapine were excluded. Patients receiving medications that can impact their renal function or biochemical parameters and those that refuse to give informed consent either the patients or patient's guardian were not recruited.

Specimen collection and processing

From each participating patient, 2mls of venous blood was carefully collected and dispense into a well labelled Lithium heparin sample container and gently mixed. All samples were transported to the laboratory in a cold chain via Jablo box packed with ice pack. Upon arrival at the laboratory, the samples were centrifuged at 4000rpm for 5 minutes and the plasma obtained was separated into a well labelled

cryovials ready for analysis. Ion Selective Electrode (ISE) 4000 machine manufactured by (SFRI, France) was used for electrolyte. Selectra ProS machine (Elitech, Vital scientific, Germany,) was used to analysed urea and creatinine following the manufacturer instructions.

Ethical consideration

Ethical approval (refPHA.864/Vol. XV/115, dated 2th May, 2020) was obtained from ethical committee of the Federal Neuropsychiatric Hospital, Uselu, Benin City. Written or verbal informed consent of willingness to participate in the study was obtained from participants.

Data analysis: Results obtained were subjected to statistical analysis using statistical package for social sciences (SPSS) version 16.0. Determination the significant difference was done by two ways ANOVA without replication in the test groups.

RESULTS

A total of 104 schizophrenic patients were recruited comprising of 51 males (49.04%) and 53 females (50.96%). These patients were monitored from zero (baseline) through 1st, 2nd and 3rd months of drugs administration. However, number of participants (n) varied along the line as some patients missed appointment while some others declined further participation. Baseline (n=104), 1st month on treatment (n=73), 2nd month on treatment (n=40) and 3rd month on treatment, (n=54).

The ages of recruited participants ranges from 18 to 77 years. This was further classified into six groups, ages 18-27 (17.31%), ages 28-37 (43.27%), ages 38-47 (25%), ages 48-57 (10.58%), ages 58-67 (1.92%) and ages 68-77 (1.92%) respectively. Ages 28-37 years has the highest number of participants with 43.27%, followed by ages 18-27 years with 17.31%.

The ethnicity pattern of participants shows that the Benins were highest with 37 (35.58%). This is not unconnected to the fact that Federal neuropsychiatric hospital is located at the heart of Benin City where the Benins are the dominant ethnic group. Esan ethnic group followed with 13(12.5%), Urhobos 11(10.6%), Igbo 10(9.62%), , Etsako and Ika 7(6.7%) respectively, Isoko 5(4.8%), Okwanle 5(4.8%), Ezon 3(2.89%), Efik 2(1.9%), while Idomah, Itshekiri, Ogoni and Yoruba are 1(0.96%) respectively.

Educational records of the participants show that 2(1.92%) had no formal education, 19(18.27%) had primary education, 52(50%) secondary education while 29(29.81%) attained tertiary education levels. Of the 104 participants, 1(0.96%) practice traditional religion, 98(94.23%) were Christians, 4(3.85%) practice Islam, while 1(0.96%) had no formal religion.

Marital status of participants shows that 4(3.85%) were devoiced, 7(6.73%) were separated, 3(2.88%) were widows, 22(21.15%) were married, while 68(65.38%) were singles (Table 1).

The results also shows that paranoid schizophrenia were more among the schizophrenic patients that participated in the research with 83(79.41%), followed by undifferentiated schizophrenia 19(18.63%) while catatonia schizophrenia were the least with 2(1.96%).

Table 1: Socio-demographic characteristics and schizophrenia variant of the studied population.

Characteristics	Number	Percentage (%)
Gender		
Male	51	49
Female	53	51
Age (years)		
18 – 27	18	17.3

28 – 37	45	43.3
38 – 47	26	25
48 – 57	11	10.6
58 – 67	2	1.9
68 – 77	2	1.9
Ethnicity		
Benin	37	35.6
Efik	2	1.9
Esan	13	12.5
Etsako	7	6.7
Ezon	3	2.9
Idomah	1	0.96
Igbo	10	9.6
Ika	7	6.7
Isoko	5	4.8
Itshekiri	1	0.96
Ogoni	1	0.96
Okwuani	5	4.6
Urhobo	11	10.6
Yoruba	1	0.96
Educational status		
No formal	2	1.9
Primary	19	18.3
Secondary	52	50
Tertiary	31	29.8
Religion		
No formal	1	0.96
Christianity	98	94.2
Islam	4	3.88
Traditionalist	1	0.96
Marital status		
Single	68	65.4
Married	22	21.2
Divorced	4	3.82
Separated	7	6.7
Widow	3	2.88
Schizophrenia variants		
Catatonic	2	1.92
Paranoid	83	79.81
Undifferentiated	19	18.27

Table 2: Effects of <<Olanzapine and Haloperidol medication on Renal Function parameters

Parameters	**Baseline** $\bar{X}$ **± SEM** ***n = 104***	**Olanzapine** $\bar{X}$ **± SEM** ***n = 92***	**Haloperidol** $\bar{X}$ **± SEM** ***n = 12***	**F-value**	**P-value**
Urea (mg/dl)	25.24 ± 1.34	24.89 ± 0.92	23.89 ± 1.38	0.108	0.897
Cr.(mg/dl)	0.73 ± 0.02	0.77 ± 0.01	0.78 ± 0.05	1.054	0.350

Na^+ (mMol/L)	$138.66^{AB} \pm 0.43$	$140.93^{A} \pm 0.28$	$139.39^{B} \pm 0.74$	11.098	<0.0001
K^+(mMol/L)	4.21 ± 0.07	4.50 ±0.37	4.04 ± 0.13	0.300	0.741
HCO_3^- (mMol/L)	22.88 ± 0.36	21.79 ± 0.35	21.67 ± 0.80	2.282	0.104
Cl^- (mMol/L)	103.52 ± 1.14	105.22 ± 0.77	103.17 ± 0.92	1.035	0.357

Legend: Data from Table 2 above shows that Olanzapine had increased significant effect on Sodium p= 0.000; baseline (138.66±0.43), Olanzapine (140.93±0.28), while K^+, HCO_3^- and Cl^-, including urea and creatinine were not statistically significant. Haloperidol shows no significant effect on electrolytes, nor creatinine and urea.

Keys: Cr =Creatinine**,** Na^+ = Sodium, K^+ =Potassium, HCO_3^- = Bicarbonate, Cl^- = Chloride

SEM = Standard Error of Mean

Post hoc: A=P <0.0001; B=P> 0.05

Established Reference Ranges

Urea 10 – 50mg/dL

Cr 0.6 – 1.3mg/dL

Na^+ 135 -145mMol/L

K^+ 3.5 - 5.5mMol/L

HCO_3^- 20 - 30mMol/L

Cl^- 97 – 108mMol/L

Table 3: Effects of duration of treatment with Olanzapine on Renal function parameters

Parameters	**Baseline (Control)** $\bar{X}$ **± SEM** **n= 86**	***First Month*** $\bar{X}$ **± SEM** **n= 73**	***Second Month*** $\bar{X}$ **± SEM** **n= 42**	**Third Month** $\bar{X}$ **± SEM** **n= 48**	**F-value**	**P-value**
Urea (mg/dl)	25.24 ± 1.34	25.72 ± 1.70	25.24 ± 1.39	23.49 ± 1.30	0.355	0.785
Cr. (mg/dl)	0.73 ± 0.02	0.74 ± 0.02	0.81 ± 0.03	0.78 ± 0.03	1.579	0.195
Na^+(mMol/L)	$138.66^{B} \pm 0.43$	$140.39^{A} \pm 0.43$	$140.71^{A} \pm 0.56$	$141.83^{A} \pm 0.48$	8.605	<0.0001
K^+(mMol/L)	4.21 ± 0.07	4.63 ± 0.56	4.17 ± 1.15	3.85 ± 0.08	0.330	0.803
HCO_3^- (mMol/L)	$22.88^{AB} \pm 0.36$	22.59 ± 0.48	$21.88^{A} \pm 0.62$	$20.60^{B} \pm 0.73$	3.741	0.012
Cl^-(mMol/L)	103.52 ± 1.14	104.79 ± 0.57	103.18 ± 0.72	107.30 ± 2.21	1.746	0.158

Legend: Table 3 above shows the effect of duration of treatment with Olanzapine on the urea, creatinine and electrolytes. From the table, it is obvious that the plasma sodium level was significantly increased as the duration of treatment increases (p=0.000) when compared with baseline. On the other hand, bicarbonate shows decreased significant difference from the second month of treatment with Olanzapine (baseline versus second month, baseline versus third month, p=0.012). Potassium and chloride shows no significance as well as urea and creatinine.

Key: SEM = Standard Error of Mean, Cr =Creatinine**,** Na^+ = Sodium, K^+ =Potassium, HCO_3^- = Bicarbonate, Cl^- = Chloride
A & B = P < 0.05
A Vs B = P < 0.01;

Table 4: Effects of duration of treatment with Haloperidol on Renal function parameters

Parameters	Baseline (Control) $\bar{X}$ ± SEM n= 18	*First Month* $\bar{X}$ ± SEM n=12	*Second Month* $\bar{X}$ ± SEM n=9	Third Month $\bar{X}$ ± SEM n=6	F-value	P-value
Urea (mg/dl)	25.24 ± 1.34	23.00 ± 1.75	20.60 ± 2.04	27.67 ± 2.73	0.402	0.752
Cr. (mg/dl)	0.73 ± 0.02	0.80 ± 0.09	0.66 ± 0.09	0.85 ± 0.04	0.946	0.421
Na^+(mMol/L	138.66 ± 0.43	139.43 ± 0.72	137.00 ± 0.77	140.17 ± 1.78	1.022	0.386
K^+(mMol/L)	4.21 ± 0.07	4.03 ± 0.26	3.98 ± 0.27	4.10 ± 0.18	0.331	0.803
HCO_3^- (mMol/L)	22.88 ± 0.36	21.14 ± 1.16	20.80 ± 1.74	23.00 ± 1.41	1.072	0.364
Cl^-(mMol/L)	103.52 ± 1.14	103.00 ± 1.36	103.00 ± 1.00	103.50 ± 2.32	0.009	0.999

Legend: Table 4 above shows that Haloperidol treatment of schizophrenia has no significant effect on the patient electrolytes, urea and creatinine.

Keys: SEM = Standard Error of Mean, Cr =Creatinine**,** Na^+ = Sodium, K^+ =Potassium, HCO_3^- = Bicarbonate, Cl^- = Chloride

DISCUSSION

This study was carried out to evaluate the effects of Haloperidol and Olanzapine antipsychotic drugs commonly used for the treatment of schizophrenic disorder on renal function. A total of 104 schizophrenic patients were used comprising of 51 males (49.04%) and 53 females (50.96%). These patients were monitored from zero (baseline) through 1st, 2nd and 3rd months of drugs administration. However, number of participants (n) varied along the line as some patients missed appointment while some others declined further participation. Baseline (n=104), 1st month on treatment (n=73), 2nd month on treatment (n=40) and 3rd month on treatment, (n=54).

The ages of recruited participants ranges from 18 to 77 years. This was further classified into six groups, ages 18-27 (17.31%), ages 28-37 (43.27%), ages 38-47 (25%), ages 48-57 (10.58%), ages 58-67 (1.92%) and ages 68-77 (1.92%) respectively. Ages 28-37 years has the highest number of participants with 43.27%, followed by ages 18-27 years with 17.31%.

The ethnicity pattern of participants shows that the Benins were highest with 37 (35.58%). This is not unconnected to the fact that Federal neuropsychiatric hospital is located at the heart of Benin City where the Benins are the dominant ethnic group. Esan ethnic group followed with 13(12.5%), Urhobos 11(10.6%), Igbo 10(9.62%), , Etsako and Ika 7(6.7%) respectively, Isoko 5(4.8%), Okwanle 5(4.8%), Ezon 3(2.89%), Efik 2(1.9%), while Idomah, Itshekiri, Ogoni and Yoruba are 1(0.96%) respectively.

Educational records of the participants show that 2(1.92%) had no formal education, 19(18.27%) had primary education, 52(50%) secondary education while 29(29.81%) attained tertiary education levels. Of the 104 participants, 1(0.96%) practice traditional religion, 98(94.23%) were Christians, 4(3.85%) practice Islam, while 1(0.96%) had no formal religion.

Marital status of shows that 4(3.85%) were devoiced, 7(6.73%) were separated, 3(2.88%) were widows, 22(21.15%) were married, while 68(65.38%) were singles (Table 1).

The results also shows that paranoid schizophrenia were more among the schizophrenic patients that participated in the research with 83(79.41%), followed by undifferentiated schizophrenia 19(18.63%) while catatonia schizophrenia were the least with 2(1.96%).

The results from the study as shown in table 2 and 3 indicated that Olanzapine has effect on the electrolyte of the treated patients. There was a strong observable significant increase in the sodium concentration down the treatment duration

($p<0.001$). This significant observation was obvious from first month of treatment through the third month as compared to the baseline. Though the sodium levels were still within the acceptable locally established normal range (135 – 145mMol/L), but there was a systematic significant increased from first month of treatment through to the third month. The mean sodium blood level of patients at baseline (before commencement of treatment) was 138.66 ± 0.43 mMol/L, which indicates that the patient had normal blood level of sodium before treatment started. At the first month of treatment, the mean plasma sodium significantly rise to 140.39 ±0.43, at second month, the mean plasma sodium level rises marginally to 140.71±0.56 mMol/L and at the third month, the mean value rises to 141.83 ± 0.48 mMol/L at $p=0.000$ level of significance. There was no such observation in patients treated with haloperidol.

The commonly reported derangement in electrolytes especially with sodium is hyponatreamia (18), though, not entirely consistent across all studies (19). There was also report of Olanzapine association with increase blood sodium level (20). Michelle *et al*., (21), reported that, if sodium levels are elevated, it is usually due to dehydration or other factors, in certain patient populations, not directly the side effect of the drug itself. This conflicting report prompted this current study. Further studies may be needed to arrive at a convincing true implication of Olanzapine on the blood sodium concentration of patients treated with it.

The blood bicarbonate levels of patient treatment with Olanzapine also showed a significant decrease from second and the third months of treatment compared to the baseline, though, still within the established normal range (20 –30mMol/L). There was no significant difference between the baseline and the first month of treatment in bicarbonate concentration. As for patients treated with Haloperidol, there was no such observable significant difference. This tendency toward acidosis might be attributed to Olanzapine association with significant increase in dyslipidemia, body mass index (BMI) and weight gain, which are precursors to metabolic disturbances that leads to acidosis. With this observation, dose review and regulation including regular monitoring of patient blood electrolyte levels to identify those who are susceptible to developing complications and may need timely and effective intervention and management especially those treated with Olanzapine is advocated.

The study showed that Olanzapine and Haloperidol have no effect on blood levels of chloride and potassium of patients all through the duration of study. From the study also, Olanzapine and Haloperidol showed no significant effect on urea and creatinine in the studied subjects. Neither the medications itself nor the duration of medication had any direct significant effect on the renal function of the patients. However, it is suggested that renal function be check probably due to metabolic disturbances associated with the use of Olanzapine in particular and generally antipsychotic drugs which may lead to renal damage.

Conclusion: Haloperidol, as shown in this study has no effects on renal function either directly or indirectly on patients treated with it, but Olanzapine may exacerbate some metabolic disturbances that may indirectly results in renal impairment if not mitigated by early identification and intervention.

Authors' contribution: The research work was conducted and approved in collaboration between all authors. A.C.O.I. and A.L. designed the study, C.O. and J.O.O. sourced for funding; C.O, J.O.O. and A.L wrote the protocol. C.O., L.N.I and J.O.O. did the experiments; L.N.I and J.O.O. did the statistical analysis. C.O and J.O.O. drafted the manuscript; A.C.O.I. and A.L. supervised the study. C.O. and J.O.O. wrote the final manuscript. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Data Availability Statement: Data from MSc project conducted at the Faculty of Nursing Science, department of Mental health and Psychiatry, Niger Delta University, Wilberforce Island, Amasoma, Bayelsa State, Nigeria.

Conflict Interest: The authors declare no conflict of interest.

REFERENCES

1. National Institute of Mental Health (NIH) 2019; Schizophrenia.US Department of Health and Human Services.19-MH-8052.
2. World Health Organization (2000). Mental Health Burden of Schizophrenia in the year 2000.
3. Michele, S. (2011). Schizophrenia: Clinical Features and Diagnosis. *Clinical Pharmacist.* Volume 3.
4. Abel, K.M., Drake, R., and Goldstein, J.M., (2020). Sex Differences in Schizophrenia. *International Review of Psychiatric*. 22:417
5. Ayuso-Mateos, J.L. (2001). Global Burden of Schizophrenia in the Year 2000.*Version 1 Estimates. California: WHO*. 1:11.
6. Chukwujekwu, C.D. (2019). Prevalence, Patterns and Correlates of Schizophrenia among Out-Patient Attendees at Madonna University Teaching Hospital, Elele; *A 3-Year Review. Clinical Psychiatry 5:12.*
7. Oluyomi, B. E., Akin, O., and Oye, G. (2012). Epidemiology of Schizophrenia. An update with a focus on developing Countries. *International Review of Psychiatric.* 24(5):387-392
8. Benerjee, I., Roy, B., Sathian, B., Benerjee, I., Chakraborty, P.K., and Saha, A., (2013). Sociodemographic Profile and Utilization Pattern of Antipsychotic Drugs Among Schizophrenic Patients. A Cross Sectional Study from Western Region of Nepal. *Biomedcentral. Psychiatric.*13: 96.
9. Park, S.C., Lee, M.S., Kang, S.G., and Lee, S.H. (2014). Patterns of antipsychotics prescription to patients with schizophrenia in Korea: Results from the health insurance review and assessment service – national patient sample. *Journal of Korean Medical Science. 2 :719-28.*
10. Igbinomwanhia, N.G., Olotu, S.O., and James, B.O. (2017). Prevalence and correlates of antipsychotic polypharmancy among out-patients with schizophrenia attending a tertiary psychiatric facility in Nigeria.*Therapeutic Advances Psychopharmacology*. 3-10.
11. Komossa, K., Rummel-Kluge, C., Hunger, H.C., Schmid, F., Scwarz, S., Duggan, L., Kissling, W. and Leucht, S. (2010). Olanzapine Versus other atypical antipsychotics for schizophrenia. *Cochroine Data base systematic review (Vol. 3)*.
12. Rummel-Kluge, C.I., Komossa, K., Schwarz, Hunger, H., Schmid, F., Schwarz, S., Duggan, L, Kissling, W., Davis, J.M. and Leucht, S. (2010). Head-head comparisons of metabolic side effects of second generation antipsychotics in the treatment of schizophrenia: A systematic review and meta-analysis. *Schizophrenia Research,* 123 (2-3); 225-33).
13. Dold, M., Samara, M.T., and Tardy, M.L. (2015). Haloperidol Verses First-generation antipsychotics for treatment of Schizophrenia and other psychotic disorders. *Cochrane Database of Systematic Review, Issue I.* CD009831. https:// doi: org/10:1002/14651858. CD009831. pus 2.
14. Ortho-McNeil-Janssen (2010); Haldol.Ortho-McNeil-Janssen Pharmaceuticals, Inc. https//www.prenewswire.com/news-rel.
15. Dominguez-Jimenez, J.L., Puentes-Gutierrez, J.J., Pelado-Garcia, E.M., Cuesta-Cubillas, D., and Garcia-Moreno, A.M, (2012). Liver toxicity due to Olanzapine. *Review of Spanish Association of Digestive. 104:617-8.*
16. Perez-Iglesias, R., Mata, 1., Pelayo-teran, J.M., Amado, J.A., Garcia-UnzuetaM.T., Berja A., Martinez-Garcia O., Vazquez-BarqueroJ.L. and Crespo-Facorro, B. (2009). Glucose and Lipid disturbances after 1 year of Antipsychotic treatment in a drug-naïve population. *Schizophrenia Research*. 107(2) 115-121.
17. Taykaran C and Tamogha B. (2013). How to calculate sample size for different study

Designs in medical research? *Indian Journal of psychological medicine. 10. (4) 210-230*

18. Sachdeva, A. and Choudhary M. (2017). Hyponatremia with olanzapine – A suspected Association. *Shanghai Archive of psychiatry*. 29(3): 177-179.
19. Jessani M., Montgomery J., Fedde T.D., Josiassen R.C. (2006). Lack of association between antipsychotics and hyponatreamia in chronic schizophrenia. *Schizophrenic Research*. 83:307-9.
20. Recep, T., Huseyin G., and Hakan, B. (2012). Olanzapine-induced neuroleptic malignant syndrome with hypernatremia. *Bulletin of clinical psychopharmacology*. 22(2): 184-186.
21. Michelle A., McNally, Mehmet Camkurt, Lokesh R.Shahani., Rania E. (2020). Olanzapine – induced hyponatremia presenting with seizure requiring intensive care unit admission. *Cureus Journal of Medical science.* 12(5):e82112.