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COMPARATIVE STUDY ON SAFETY & EFFICACY OF ESCITALOPRAM VS VORTIOXETINE IN DEPRESSION DISORDER

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ABSTRACT

Aims: The aim of the study is comparing the safety and efficacy of SSRI'S (Escitalopram) and serotonin receptor modulator (vortioxetine) in depressive patients. **Objectives:** To assess the relative efficacy and safety of Escitalopram. Safety and efficacy of Vortioxetine in depressive patients. To analyse the age, gender and socio demographic data. **Methodology:** The study will be conducted at Maurya Hospital, Kurnool, over six months as a prospective observational study. It will involve 50-100 patients, with data collected through patient forms and interviews. Participants must be aged 18-60, diagnosed with depression, and prescribed Escitalopram or Vortioxetine, with voluntary consent. Excluded are those unwilling to participate, on psychotropic medications, or diagnosed with schizophrenia or bipolar disorder. **Discussion:** A study on 60 depression patients compared Escitalopram (Group A) and Vortioxetine (Group B). Both groups had similar age and gender distributions. Group A showed higher depression severity and burnout, while Group B had slightly higher compassion scores. Both treatments significantly improved depression scores, with Group B showing greater reductions in burnout and secondary traumatic stress. The findings indicate effectiveness for both medications, with differences in psychological outcomes**. Conclusion:** The study compared 60 patients on Escitalopram (Group A) and Vortioxetine (Group B). Group A had higher initial depression and burnout, while Group B showed more compassion. Both groups improved in HAM-D scores, with Group A reducing burnout and traumatic stress, and Group B experiencing declines in overall quality of life scores. Both treatments were effective.

KEYWORDS: Escitalopram, Vortioxetine, Escitalopram, Depression.

DEFINITION

Depression, or major depressive disorder, is a widespread and serious mental health condition that affects mood, behaviour, and cognition. It is marked by persistent sadness or loss of interest in activities, lasting most of the day for at least two weeks. Symptoms include sleep and appetite disturbances, fatigue, poor concentration, feelings of worthlessness, and suicidal thoughts. Unlike normal emotional fluctuations, depressive episodes significantly impair daily functioning. The condition stems from a complex interplay of biological, psychological, and social factors, with increased risk among individuals exposed to trauma, abuse, or significant loss.

Depression can affect anyone and often coexists with other medical conditions. It is associated with changes in brain chemistry, particularly deficiencies in neurotransmitters like serotonin, dopamine, and noradrenaline. Hormonal imbalances, substance abuse, and chronic illnesses such as thyroid disorders, diabetes, and neurological conditions may also contribute. Understanding these multifactorial causes is essential for effective diagnosis and treatment, which often involves a combination of medication, psychotherapy, and lifestyle interventions.

Types of Depression (Pointwise)

1. Major Depressive Disorder

- Persistent low mood or loss of interest.
- May involve recurrent or prolonged episodes.

2. Atypical Depression

- Mood reactivity, increased appetite, hypersomnia.
- Sensitivity to interpersonal rejection.

3. Postpartum Depression

- Occurs after childbirth.
- Linked to hormonal and emotional changes.

4. Catatonic Depression

- Characterized by motor immobility or excessive motor activity.

- May include mutism or stupor.

5. Seasonal Affective Disorder (SAD)

- Depression triggered by seasonal changes.
- Most common in winter months.

6. Melancholic Depression

- Severe anhedonia, early morning awakening.
- Psychomotor retardation or agitation.

7. Bipolar Disorder (Manic Depression)

- Alternating mood episodes:
  - Depressive episode
  - Manic episode
  - Hypomanic episode
  - Mixed mood states

8. Dysthymia (Persistent Depressive Disorder)

- Chronic low-grade depression lasting for years.
- Symptoms are less severe but long-lasting.

9. Situational Depression

- Triggered by specific life events or stressors.
- Often short-term and reactive.

10. Psychotic Depression

- Depression accompanied by delusions or hallucinations.
- Requires urgent psychiatric intervention.

11. Endogenous Depression

- Originates from internal biological or genetic factors.
- Not linked to external stressors.

EPIDEMIOLOGY

Depression is a leading global health concern, affecting over 350 million individuals

across all age groups, as reported by the World Health Organization. In the United States, major depressive disorder ranks as the primary cause of disability among individuals aged 15 to 44, underscoring its profound impact on productivity and quality of life during peak working years. Epidemiological data reveal significant gender disparities, with women nearly twice as likely to experience major depression compared to men. The severity of depressive symptoms tends to increase with age, contributing to greater functional impairment in older populations. Alarmingly, approximately 30% of individuals diagnosed with depressive disorders attempt suicide, highlighting the urgent need for early detection, comprehensive treatment, and sustained mental health support.

ETIOLOGY

1. Genetic Factors

- Family history of depression increases susceptibility.
- Hereditary transmission plays a significant role.

2. Environmental Factors

- Exposure to trauma, abuse, neglect, or significant loss.
- Chronic stress and adverse life events.

3. Biochemical Factors

- Deficiency of neurotransmitters: **serotonin**, **dopamine**, and **noradrenaline**.
- Reduced **dopaminergic activity** in depression; increased in manic states.

4. Endocrine Factors

- Hormonal imbalances such as:
  - **Hypothyroidism**
  - **Cushing's syndrome**
  - Other endocrine disorders

5. Substance Abuse

- Abuse of alcohol and recreational drugs can trigger or worsen depression.
- Withdrawal from substances like **benzodiazepines** may contribute.

6. Hormonal Changes

- Fluctuations in hormone levels due to puberty, pregnancy, menopause, or medical

conditions.

7. Physical Illnesses

- Chronic or severe medical conditions linked to depression:
  - **Viral infections**
  - **Carcinomas**
  - **Neurological disorders**
  - **Thyroid disease**
  - **Multiple sclerosis**
  - **Pernicious anemia**
  - **Diabetes**
  - **Systemic lupus erythematosus**
  - **Addison's disease**

8. Medication-Induced Depression

- Certain drugs may contribute to depressive symptoms:
  - **Analgesics**
  - **Antidepressants**
  - **Antihypertensives**
  - **Anticonvulsants**
  - **Antipsychotics**

Clinical Manifestations

1. **Mood and Thought Disturbances**
   - Pessimistic thinking
   - Suicidal ideation in severe cases.

2. **Psychotic Features** (in severe depression)
   - Hallucinations
   - Delusions

3. **Sleep and Appetite Changes**
   - Insomnia or hypersomnia
   - Loss of appetite
   - Weight loss

- Reduced libido.

4. **Cognitive Impairments**
- Poor concentration
- Slowed thinking
- Impaired memory, especially for recent events.

Pathophysiology of Depression

Depression is associated with multiple neurobiological mechanisms that attempt to explain its onset and progression

- **Biogenic Amine Hypothesis**: Suggests depression results from a deficiency of monoamines, particularly serotonin and noradrenaline. However, it does not account for the delayed therapeutic response to antidepressants.
- **Receptor Sensitivity Hypothesis**: Proposes that depression stems from pathological changes in receptor sensitivity, including up-regulation and super sensitivity. Antidepressants may induce receptor desensitization and down-regulation over time.
- **Serotonin-only Hypothesis**: Focuses solely on serotonin dysfunction, minimizing the role of noradrenaline. This model is limited by its inability to explain delayed symptom relief and the involvement of other neurotransmitters.
- **Permissive Hypothesis**: Emphasizes the balance between serotonin and noradrenaline in regulating mood. Low levels of both are linked to depression, while low serotonin with elevated noradrenaline may lead to mania.
- **Electrolyte Membrane Hypothesis**: Suggests that electrolyte imbalances, such as hypocalcemia and hypercalcemia, may influence mood states—mania and depression, respectively.
- **Neuroendocrine Hypothesis**: Attributes mood disturbances to altered endocrine function, particularly dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis.

DIAGNOSIS OF DEPRESSION

1. Clinical Evaluation

- **History Collection**: Includes medical, psychiatric, and personal background.
- **Mental Status Examination**: Assesses mood, cognition, behavior, and thought process.

2. ICD-10 Criteria (WHO)

- **Usual Symptoms**: Depressed mood, loss of interest, reduced energy
- **Common Symptoms**: Poor concentration, low self-esteem, guilt, pessimism, suicidal thoughts, sleep/appetite changes
- **Severity**
  - Mild: ≥2 usual + ≥2 common symptoms
  - Moderate: 2–3 usual + ≥3 common symptoms
  - Severe: All 3 usual + ≥4 common symptoms (some severe)

3. DSM-IV Criteria

- Depressed mood or loss of interest + ≥4 symptoms (e.g., appetite/sleep changes, fatigue, psychomotor changes) for ≥2 weeks

4. Differential Diagnosis

- Rule out other psychiatric disorders (e.g., bipolar, anxiety, schizophrenia)
- Exclude medical causes: neurological, endocrine, infectious, nutritional, and medication/substance-related conditions

Treatment of Depression

1. Pharmacological Treatments

Antidepressants

- **MAO Inhibitors**
  - Irreversible: Isocarboxazid, Iproniazid, Phenelzine, Tranylcypromine
  - Reversible: Moclobemide, Cordyline
- **Tricyclic Antidepressants (TCAs)**
  - NA & 5-HT reuptake inhibitors: Imipramine, Amitriptyline, Doxepin, Dothiepin, Clomipramine
  - NA reuptake inhibitors: Desipramine, Nortriptyline, Amoxapine
- **Selective Serotonin Reuptake Inhibitors (SSRIs)**
  - Fluoxetine, Fluvoxamine, Sertraline, Citalopram
- **Atypical Antidepressants**
  - Trazodone, Mianserin, Mirtazapine, Venlafaxine, Duloxetine, Bupropion, Tianeptine
- **Adjunct Medications**
  - Mood stabilizers and antipsychotics may be added for treatment-resistant cases

2. Physical Treatments

- **Electroconvulsive Therapy (ECT)**
- **Sleep Deprivation Therapy**
- **Light Therapy**
- **Transcranial Magnetic Stimulation (TMS)**
  - FDA-approved for treatment-resistant depression
- **Vagus Nerve Stimulation (VNS)**
  - FDA-approved for long-term use in patients unresponsive to ≥4 medications
- **Ketamine Nasal Spray**
  - Used alongside oral antidepressants for treatment-resistant depression

3. Psychological Therapies

- **Cognitive Behavioural Therapy (CBT)**
- **Problem-Solving Therapy**
- **Interpersonal Therapy (IPT)**
- **Non-directive Counselling**
- **Bereavement Counselling**

DRUG MONOGRAPHY

1) ESCITALOPRAM (Lexapro)

Class: Selective Serotonin Reuptake Inhibitor (SSRI)

Indications: Escitalopram is indicated for the acute and maintenance treatment of Major Depressive Disorder (MDD) and for Generalized Anxiety Disorder (GAD) in adults. It helps restore the balance of serotonin in the brain, improving mood, sleep, and energy levels while reducing anxiety and nervousness.

Mechanism of Action

Escitalopram is the S-enantiomer of citalopram and acts as a highly selective serotonin reuptake inhibitor (SSRI). It increases serotonin levels in the synaptic cleft by blocking its reuptake into presynaptic neurons, thereby enhancing serotonergic neurotransmission. It has negligible effects on norepinephrine (NE) or dopamine (DA) systems, contributing to a favourable side-effect profile.

Dosage and Administration

Escitalopram is administered orally once daily, with or without food, either in the

morning or evening. Adults (MDD, GAD): 10 mg once daily; may increase to 20 mg after at least one week. Some patients may not gain additional benefit beyond 10 mg. Adolescents (12–17 years, MDD): 10 mg once daily; may increase to 20 mg after at least three weeks.

Elderly or hepatic impairment: 10 mg once daily (due to increased drug exposure). Renal impairment: No adjustment in mild–moderate cases; caution in severe impairment (limited data). Avoid concurrent use with MAO inhibitors or within 14 days of stopping one due to the risk of serotonin syndrome. When discontinuing therapy, taper gradually to prevent withdrawal symptoms such as dizziness, agitation, and sensory disturbances.

Contraindications

Concurrent or recent (within 14 days) MAOI use.

Concomitant pimozide administration (risk of QT prolongation).

Known hypersensitivity to escitalopram or citalopram.

Warnings and Precautions

Suicidality: Increased risk in children, adolescents, and young adults during early treatment; monitor closely. Withdrawal symptoms: Taper slowly to avoid dizziness, irritability, and “electric shock” sensations.

Seizures: Use cautiously in patients with seizure disorders.

Mania/Hypomania: May precipitate manic episodes in bipolar patients. Hyponatremia/SIADH: Particularly in elderly or diuretic-treated patients.

Bleeding risk: Caution when used with NSAIDs, aspirin, or anticoagulants.

Serotonin Syndrome or NMS-like reactions: May occur with other serotonergic or antidopaminergic agents. Psychomotor impairment: May cause drowsiness or affect coordination—avoid hazardous activities until effects are known.

Drug Interactions

Metabolized mainly by CYP2C19 and CYP3A4.

Caution with CYP2D6 substrates and CYP inhibitors/inducers that may alter plasma concentrations. Serotonergic drugs (e.g., triptans, tramadol, SSRIs) increase serotonin syndrome risk. NSAIDs, aspirin, warfarin: Increase bleeding risk.

Pharmacokinetics

Absorption: Well absorbed; peak levels in ~5 hours; food has no effect.

Distribution: 56% protein bound; crosses placenta; enters breast milk.

Metabolism: Hepatic via CYP3A4 and CYP2C19.

Elimination: Renal; half-life 27–32 hours.

Elderly: 50% higher exposure; hepatic impairment doubles half-life.

Storage

Store tablets at 25°C (77°F), with allowable excursions between 15–30°C.

Patient Counselling

Take once daily at the same time each day, with or without food.

Improvement may take 1–4 weeks; continue as prescribed.

Do not stop abruptly—consult your provider for tapering.

Report worsening mood, suicidal thoughts, or unusual changes in behaviour.

Recognize signs of serotonin syndrome (agitation, fever, muscle stiffness, confusion).

Avoid alcohol and caution with driving until effects are known.

If pregnant, breastfeeding, or taking other medications.

2) VORTIOXETINE (Trintellix)

Drug class: Serotonin modulator and stimulator (SSRI-like)

Indication: Vortioxetine is indicated for the treatment of Major Depressive Disorder (MDD) in adults.

Mechanism of Action:

Vortioxetine enhances serotonergic activity through inhibition of serotonin reuptake and modulation of multiple 5-HT receptor subtypes. It acts as an antagonist at 5-HT_3, 5-HT_1D, and 5-HT_7 receptors, a partial agonist at 5-HT_1B, and a full agonist at 5-HT_1A receptors. This multimodal activity is thought to improve mood, cognition, and overall antidepressant efficacy. Vortioxetine has no clinically significant effect on norepinephrine or dopamine transporters.

Dosage and Administration

Administer orally once daily, with or without food, at the same time each day.

Adults: Start 10 mg daily; may increase to 20 mg after at least one week if tolerated. Reduce to 5 mg if not tolerated. CYP2D6 poor metabolizers: Maximum 10 mg daily.

Strong CYP2D6 inhibitors (e.g., fluoxetine, paroxetine): Reduce vortioxetine dose by 50%. Strong CYP inducers (e.g., rifampin, carbamazepine): Dose may need to be increased up to 3-fold. Hepatic impairment: Mild–moderate, no change; severe, not recommended.

Renal impairment, elderly, gender, or ethnicity: No dosage adjustment.

When discontinuing, taper from 15–20 mg to 10 mg daily for one week to reduce withdrawal effects such as headache, dizziness, and irritability.

Contraindications

Hypersensitivity to vortioxetine (including angioedema).

Concomitant use with or within 14 days of MAOIs; allow 21 days after stopping vortioxetine before starting an MAOI.

Concurrent linezolid or intravenous methylene blue.

Warnings and Precautions

Monitor closely for suicidal thoughts or behaviour, especially during initiation and dose changes. Risk of serotonin syndrome increases with other serotonergic agents (SSRIs, SNRIs, triptans, tramadol). Use caution with NSAIDs, aspirin, or warfarin due to increased bleeding risk. May cause mania/hypomania in bipolar disorder, angle-closure glaucoma due to pupillary dilation, and hyponatremia/SIADH, particularly in elderly or volume-depleted patients. Avoid confusion with Brilinta (ticagrelor).

Drug Interactions

Metabolized primarily by CYP2D6; minor pathways include CYP3A4/5 and CYP2C19. Strong CYP2D6 inhibitors reduce clearance; dose adjustment is required. Strong CYP inducers may necessitate a higher dose. Concomitant use with serotonergic or anticoagulant drugs increases risk of serotonin syndrome or bleeding. Highly protein-bound drugs may alter free plasma levels.

Pharmacokinetics

Bioavailability: 75%; peak plasma time 7–11 hrs; half-life ~66 hrs (steady state in 2 weeks). Extensively metabolized hepatically to inactive metabolites. Excreted mainly in urine (59%) and feces (26%). Protein binding 98%. Food has no significant effect on absorption.

Adverse Effects

Most common: nausea (dose-related, transient). Others include headache, dizziness, constipation, dry mouth, and mild sexual dysfunction (less frequent than SSRIs). Abrupt discontinuation of doses ≥15 mg may cause headache, dizziness, irritability, or muscle tension.

Storage

Store tablets at 25°C (range 15–30°C).

Patient Counselling

Take once daily at the same time, with or without food. Do not stop abruptly without medical advice. Report any new or worsening depression or suicidal thoughts immediately. Recognize symptoms of serotonin syndrome (fever, agitation, muscle stiffness, confusion). Avoid alcohol and other serotonergic drugs unless approved. Use caution when driving until effects are known. Inform healthcare providers if pregnant, breastfeeding, or taking NSAIDs or anticoagulants.

MATERIALS AND METHODS

Study Design: Prospective observational study.

Study Site: Maurya Hospital, Kurnool.

Study Duration: Six months.

Sample Size: 60 patients.

Inclusion Criteria: Patients aged 18–60 years diagnosed with major depressive disorder as per DSM-5 criteria, receiving Escitalopram or Vortioxetine, and providing informed consent. Exclusion Criteria: Patients unwilling to participate, on psychotropic medications other than study drugs, or diagnosed with schizophrenia or bipolar disorder. Data Collection: Patient data were collected using structured documentation forms and clinical interviews. The Hamilton Depression Rating Scale (HAM-D) and Professional Quality of Life (ProQOL) were used to assess efficacy and quality of life. Safety was assessed based on adverse effects and tolerability.

RESULTS

Age wise distribution

- A total of 60 patients were included in the study and subjects aged between 30-40 years (41%) are more prominent and subjects aged between 10-20 years (4%) are less prominent.

Table No. 01: Distribution of patients based on Age.

Sl.no	Age	Group A	Group B	TOTAL
1	11-20	2	-	2 (4%)
2	20-30	4	6	10(17%)
3	30-40	11	14	25(41%)
4	40-50	9	7	16(26%)
5	50-60	4	3	7 (12%)
6	TOTAL	30	30	100%

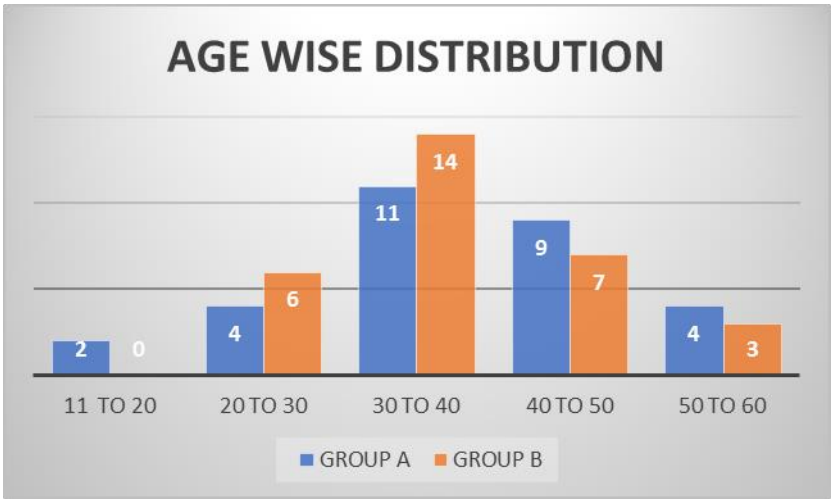

Figure 01: Age wise distribution.

The image shows a bar graph comparing two groups, Group A and Group B, across different age range

Distribution of patients based on gender:

- A total of 60 patients were included in the study out of them 23(49.4 %) females & 37 (50.6%) males

Table No. 02: distribution of patients based on Gender.

SL NO	GENDER	Group A	Group B	TOTAL
1	MALES	18	19	50.6%
2	FEMALES	12	11	49.4%
3	TOTAL	30	30	100%

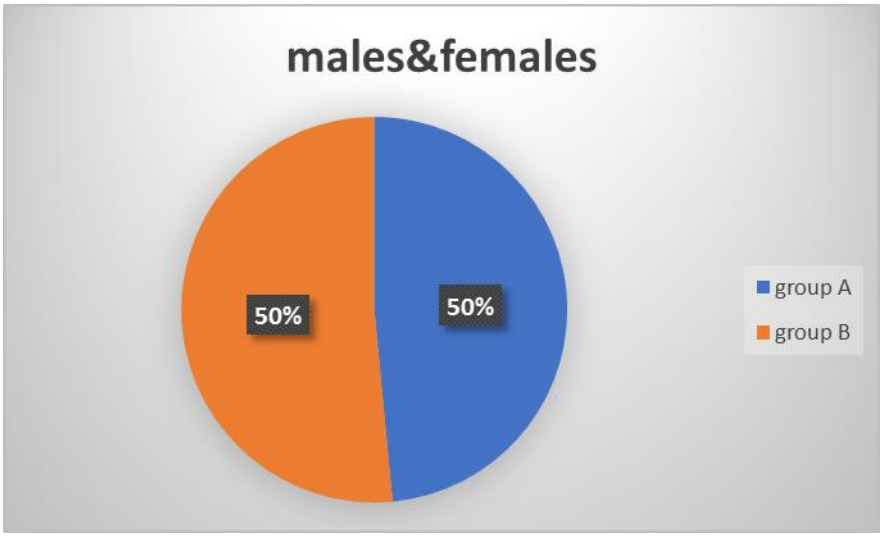

Figure 02: distribution of patients based on Gender.

Base line of HAM-D &Professional quality of life scale score:

The following Table no: 03 shows different scales parameters

Sl.no	Scales	Group A	Group B
1	HAM-D	47.59±16.96	42.95±12.78
2	Compassion scale	24.96 **Error! Filename not specified.** 5.850	26.81±5.540
3	Burnot scale	31.86±6.064	28.28±5.853
4	Secondary traumatic stress scale	29.46±4.978	30.25 **Error! Filename not specified.** 4.582

- The table displaying baseline scores of the Hamilton Depression Rating Scale (HAM-D) and Professional Quality of Life Scale (ProQOL) for two groups, labelled Group A and Group B. The scales assessed include HAM-D, Compassion scale, Burnout scale, and Secondary traumatic stress scale. Numerical values, presented as mean ± standard deviation, indicate the scores for each group on the respective scales.

Effects of treatment on HAM-D score

The following Table No: 04 indicates effects of HAM- D Score Initial & final visit of both groups.

SL.NO	VISITS	Initial visit	Final visit	P*VALUE
1	Group A	47.59±16.96	38.8±0.8	< 0.0245
2	Group B	42.95±12.78	30.4±6.9	< 0.002

Figure: 04: shows initial & final visit of Groups A &Groups B.

- The image shows a line graph comparing two groups (Group A and Group B) at two different time points (Initial Visit and Final Visit). The graph illustrates changes in the measured variable over time for each group.
- The vertical axis (y-axis) represents the measured variable, with values ranging from 0 to 50.
- The horizontal axis (x-axis) represents the groups, with two categories: Group A and Group B.

PROFFESIONAL QUALITY OF LIFE SCALE:

Table No. 5: Representing variables quality of life in the initial visit and final visit of group A.

S.NO	Variables quality of life	Group A Initial visit	Group A Final visit	Status level	P*value
1.	Compassion satisfaction scale	24.96±5.850	24.19±3.913	Moderate	<0.7630
2	Burnot scale	31.86±6.064	29.48±5.072	High	< 0.007
3	Secondary traumatic stress scale	29.46±4.978	28.19±6.082	High	< 0.022

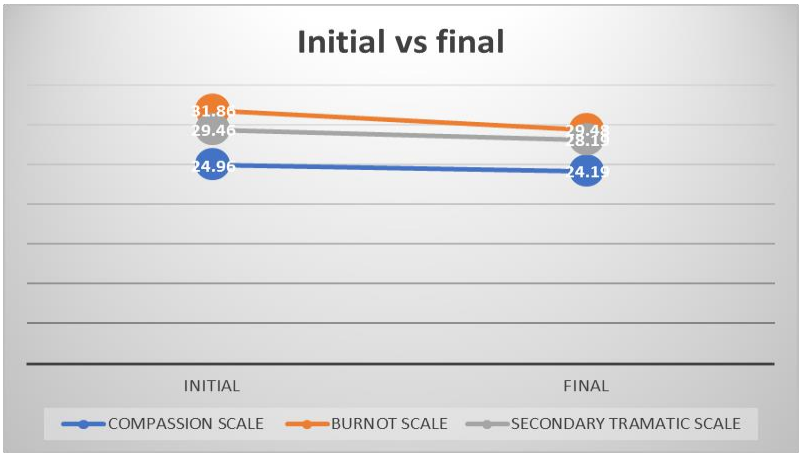

Figure 05: initial visit & final visit of Group A.

The graph shows a line chart comparing three scales (Compassion Scale, Burnout Scale, and Secondary Traumatic Scale) between two groups, Group A and Group B, Initial visit The chart indicates the scores of each group on these scales.

Group B

Table No. 5: Representing variables quality of life in the initial visit and final visit of group B.

S.NO	Variables quality of life	Group B Initial visit	Group B Final visit	Status level	P*value
1.	Compassion satisfaction scale	26.81±5.540	25.4±5.010	Severe	<0.039
2	Burnot scale	28.28±5.853	27.9±4.85	Severe	<0.395
3	Secondary traumatic stress scale	30.25±4.582	29.46±4.978	Severe	<0.140

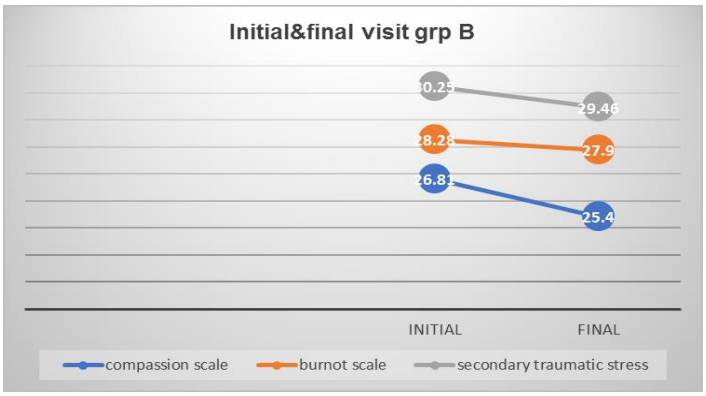

Figure 06: show Initial & Final visit of group B.

The graph shows a line graph comparing three scales across two groups, Final visit labelled A and B. The scales are Compassion Scale, Burnout Scale, and Secondary Traumatic Scale.

DISCUSSION

In this study, patients were randomly divided into two treatment groups—Group A, which received *Escitalopram*, and Group B, which received *Vortioxetine*. The primary objective was to compare the safety and efficacy of these two antidepressants in patients diagnosed with depressive disorders.

Table 1 presents the age-wise distribution of patients across both groups. Each group consisted of 30 individuals, making a total sample size of 60. The majority of participants belonged to the 30–40-year age group, with 11 individuals in Group A and 14 in Group B (41% of the total population). This indicates that depressive disorders were most prevalent among middle-aged adults in this sample, consistent with existing epidemiological data suggesting higher rates of depression in this demographic.

Table 2 shows the gender distribution of participants. There were 18 males in Group A and 19 males in Group B, comprising 37 males (50.6%) of the total population, while females accounted for 23 participants (49.4%). The nearly equal gender representation supports the general understanding that depression affects both males and females, although previous literature indicates a slightly higher prevalence among females.

Table 3 compares mean scores of four psychological scales between the two groups—HAM-D (Hamilton Depression Rating Scale), Compassion Scale, Burnout Scale, and Secondary Traumatic Stress Scale. Group A showed higher average HAM-D (47.59) and Burnout (31.86) scores than Group B (42.95 and 28.28, respectively), indicating that Escitalopram-treated patients initially exhibited greater depressive severity and burnout. Conversely, Group B demonstrated slightly higher Compassion scores (26.81 vs 24.96), suggesting improved emotional responsiveness among Vortioxetine-treated patients. The similarity in Secondary Traumatic Stress scores (29.46 vs 30.25) suggests comparable levels of emotional strain across both groups at baseline.

Table 4 highlights the change in HAM-D scores between initial and final visits for both groups. A statistically significant reduction in depressive symptoms was observed in both. For Group A, the HAM-D score decreased from 47.59 ± 16.96 to 38.8 ± 0.8 ($p < 0.0245$), while Group B showed a reduction from 42.95 ± 12.78 to 30.4 ± 6.9 ($p < 0.002$). This demonstrates that both Escitalopram and Vortioxetine effectively reduced depressive symptoms, though Vortioxetine exhibited slightly greater improvement.

Table 5 evaluates quality-of-life variables in Group A. A moderate decrease in Compassion Satisfaction and Burnout scores was observed between initial and final visits, with a significant reduction in burnout levels ($p < 0.007$). Secondary Traumatic Stress also decreased significantly ($p < 0.022$), reflecting improved coping ability and reduced emotional strain following treatment.

Table 6 analyses the same parameters for Group B. Compassion Satisfaction decreased from 26.81 ± 5.54 to 25.4 ± 5.01, Burnout from 28.28 ± 5.85 to 27.9 ± 4.85, and Secondary Traumatic Stress showed a significant decline ($p < 0.001$). Although a slight reduction in Compassion Satisfaction was noted, the overall decline in burnout and stress scores suggests enhanced emotional stability and resilience with Vortioxetine therapy. Overall, both medications were well tolerated, with no severe adverse effects reported. Mild symptoms such as nausea and headache were transient and self-limiting. The observed findings are consistent with previous studies reporting comparable antidepressant efficacy between Escitalopram and Vortioxetine. However, Vortioxetine demonstrated added benefits in improving cognitive and psychosocial functioning, aligning with its multimodal serotonergic mechanism of action. In summary, both Escitalopram and Vortioxetine significantly improved depressive symptoms and quality of life. Vortioxetine exhibited slightly superior outcomes in psychological well-being, supporting its role as an effective alternative to conventional SSRIs in managing major depressive disorder.

CONCLUSION

This study compared the safety and efficacy of Vortioxetine and Escitalopram in patients with major depressive disorder (MDD). Depression was observed across all age groups, with a higher number of males receiving Vortioxetine and more females treated with Escitalopram. Both medications were found to be effective and well tolerated. However, Vortioxetine demonstrated slightly greater efficacy, particularly in improving cognitive and emotional parameters, as reflected by lower HAM-D scores and better ProQOL outcomes. Reported adverse effects of Vortioxetine included mild tremors (8%), fatigue (15%), and increased sweating (10%), which were manageable and non-serious. Overall, while both Escitalopram and Vortioxetine are effective options for managing MDD, Vortioxetine showed marginal superiority in symptom reduction, cognitive improvement, and quality-of-life enhancement, making it a promising alternative to

traditional SSRIs.

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