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A COMPARATIVE STUDY ON SAFETY, EFFICACY AND PHARMACOTHERAPEUTIC ADHERENCE OF DESVENLAFAXINE VERSUS ESCITALOPRAM IN DEPRESSION AT A TERTIARY CARE TEACHING HOSPITAL

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ABSTRACT

INTRODUCTION

Major Depressive Disorder (MDD) is a prevalent mental health condition, with pharmacological treatments like Escitalopram (SSRI) and Desvenlafaxine (SNRI) being widely used. This study compared their efficacy, safety, adherence and metabolic effects in MDD patients.

OBJECTIVES

The primary objective was to evaluate depressive symptom reduction using the Hamilton Depression Rating Scale (HAM-D) over 16 weeks. Secondary objectives included assessing lipid profiles, platelet counts, adverse effects and adherence.

METHODS

This prospective observational study included 203 MDD patients (Escitalopram: n=105; Desvenlafaxine: n=98). HAM-D scores, lipid profiles, and platelet counts were evaluated at baseline and week 16. Adverse effects were monitored throughout the study.

RESULTS

Both treatments significantly reduced HAM-D scores ($p < 0.001$), with Escitalopram showing greater improvement at all time points ($p < 0.001$). Escitalopram improved lipid profiles, reducing TC ($p < 0.001$), TG ($p < 0.001$), and VLDL ($p < 0.001$), while increasing HDL ($p < 0.001$). LDL remained unchanged ($p = 0.842$). Desvenlafaxine increased TC ($p < 0.001$) and VLDL ($p < 0.001$) but also raised HDL ($p < 0.001$), with no significant changes in TG ($p = 0.954$) or LDL ($p = 0.839$). Platelet counts decreased slightly with Escitalopram ($p = 0.012$) but remained stable with Desvenlafaxine ($p = 0.806$). Adverse effects were more frequent with Desvenlafaxine (9.2% at 4 weeks) than Escitalopram (6.7%).

CONCLUSION

Escitalopram demonstrated superior efficacy in reducing depressive symptoms and a more favourable impact on lipid profiles compared to Desvenlafaxine, which had a higher incidence of adverse effects. Escitalopram is preferable for MDD patients with metabolic concerns, while Desvenlafaxine remains a viable alternative with careful monitoring. Individualized treatment strategies are essential for optimal MDD management.

Keywords: Major Depressive Disorder, Escitalopram, Desvenlafaxine, HAM-D, SSRI, SNRI

INTRODUCTION

Major depressive disorder (MDD), commonly known as depression, is one of the most prevalent mental health disorders in the world. Approximately 254 million people across all age groups are estimated to be affected by depression globally. In India alone, nearly 56 million people suffer from depression, according to the 2017 WHO report [1]. The most common symptoms of depression include persistent sadness, loss of interest or pleasure in activities, feelings of guilt, low self-worth, sleep disturbances, loss of appetite, poor concentration, and low energy. Depression is not merely a fluctuation in emotions but a serious condition that significantly impairs an individual's ability to function, leading to adverse personal, social, and financial consequences [2].

Historically, the term "depression" originates from the Latin word *deprimere*, meaning "to press down." This term replaced the older concept of *melancholia*, which was used to describe depressive conditions during the 19th century [3]. The treatment of depression has undergone significant evolution over time. Early therapeutic approaches included extreme measures such as immersion in water, electroshock therapy, enemas, and induced vomiting. Later, invasive procedures like lobotomies were introduced, which often resulted in severe side effects, including personality changes and even death [4,5]. By the mid-20th century, a breakthrough occurred when isoniazid, originally developed for tuberculosis, was found to have mood-elevating effects in depressed patients [6]. This discovery paved the way for the development of tricyclic antidepressants (TCAs), with imipramine becoming a widely used treatment. By the late 20th century, selective serotonin reuptake inhibitors (SSRIs) were introduced, revolutionizing depression therapy due to their improved safety and efficacy compared to earlier treatments [7].

Today, the management of depression primarily relies on second-generation antidepressants, including SSRIs and serotonin-norepinephrine reuptake inhibitors (SNRIs). These medications work through distinct mechanisms compared to older drugs, offering fewer side effects and better therapeutic outcomes [8]. Among SSRIs, escitalopram is one of the most commonly prescribed agents. Approved by the FDA in 2002, it selectively inhibits serotonin reuptake and is favored for its well-established efficacy and tolerability [9,10]. On the other hand, desvenlafaxine, an SNRI, inhibits the reuptake of both serotonin and norepinephrine, making it particularly beneficial for patients experiencing fatigue or low energy. It received FDA approval in 2008 and was later approved by CDSCO in 2009 [11].

Despite the availability of numerous treatment options, achieving remission in depression remains challenging due to its chronic nature, the need for long-term therapy, and the critical role of patient adherence. Poor adherence to treatment can lead to disease progression, increased economic burden, and diminished quality of life [12]. In India, additional barriers such as limited access to mental healthcare, socioeconomic disparities, and societal stigma further complicate treatment adherence [13]. Moreover, individual variability in drug response necessitates a personalized approach to selecting the most suitable antidepressant for each patient.

Escitalopram and desvenlafaxine are among the most frequently prescribed antidepressants over the past two decades. However, they differ in their mechanisms of action—escitalopram targets serotonin alone, while desvenlafaxine affects both serotonin and norepinephrine [14]. This difference may influence their effectiveness in alleviating specific symptoms such as emotional numbness, fatigue, and cognitive impairment, as well as their overall tolerability. Despite their widespread use, comparative data between these two drugs—especially in the Indian population—remain limited. Factors such as cultural influences, socioeconomic conditions, stigma, and genetic variations may significantly impact treatment outcomes in this context [15]. The lack of robust comparative evidence often leaves clinicians uncertain about the optimal choice of antidepressant for individual patients.

To address this gap, the present study aims to provide a detailed comparison of escitalopram and desvenlafaxine in terms of safety, efficacy, and treatment adherence. By generating reliable data, this

research seeks to assist clinicians in making informed decisions, ultimately improving therapeutic outcomes for patients with depression.

MATERIALS AND METHODS

This prospective, observational study was conducted over an 18-month period (August 2023 to January 2025) as a collaborative effort between the Department of Pharmacology and Department of Psychiatry at G.S.V.M. Medical College, Kanpur. The research was carried out in accordance with ethical guidelines after obtaining proper institutional approvals and written informed consent from all participants.

Patients diagnosed with major depressive disorder (MDD) according to the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) criteria were systematically recruited from the psychiatry outpatient clinics. The study employed a comparative design with participants who received one of two treatment groups: those receiving escitalopram (10 mg/day) or desvenlafaxine (50 mg/day). The sample size was calculated using Cochran's formula to ensure sufficient statistical power for detecting clinically meaningful differences between the treatment groups. The efficacy of treatment was evaluated using changes in Hamilton Depression Rating Scale (HAM-D) scores from baseline to study completion. HAM-D is a validated, observer-administered scale consisting of 17 items that quantitatively assess depression severity. The first 9 items are scored on a 0-4 scale (0 = absent, 1 = questionable, 2 = mild, 3 = moderate, 4 = severe), while the remaining 8 items are scored 0-2 (0 = absent, 1 = doubtful, 2 = clearly present). This scoring system allows for comprehensive evaluation of both psychological and somatic symptoms of depression, with higher total scores indicating greater depression severity. Serial HAM-D assessments were conducted at each follow-up visit (weeks 4, 8, 12, and 16) to monitor therapeutic response, with the magnitude of score reduction serving as the primary efficacy outcome measure. Scores were interpreted as: ≤7 = normal, 8-13 = mild depression, 14-18 = moderate depression, 19-22 = severe depression, ≥23 = very severe depression

Inclusion criteria:

- Patients aged between 18 and 60 years.
- Patients diagnosed with depression as per DSM-5 criteria (Annexure-II).
- Patients of either sex.
- Patients who were willing to provide written informed consent for participation in the study.

Exclusion criteria:

- Patients suffering from depression secondary to any general medical or neurological conditions.
- Patients already on another antidepressant drug therapy.
- Patients unwilling to provide consent for participation in the study.
- Pregnant or lactating women.
- Patients with a known hypersensitivity to Desvenlafaxine or Escitalopram.

At baseline, comprehensive evaluations were conducted including:

- Detailed demographic profiling (age, gender, occupation, education level)
- Complete clinical history and physical examination
- Assessment of depression severity using the Hamilton Depression Rating Scale (HAM-D)
- Laboratory investigations including complete lipid profile (measuring total cholesterol, triglycerides, HDL, LDL, and VLDL levels) and platelet count

Patients were followed up at regular intervals (4, 8, 12, and 16 weeks) with standardized assessments including:

- Serial HAM-D scoring to monitor therapeutic response
- Repeat lipid profile and platelet count at 16 weeks to evaluate metabolic effects
- Documentation of any adverse drug reactions using validated assessment tools
- Evaluation of treatment adherence through standardized questionnaires

STATISTICAL ANALYSIS

Data was recorded using a structured proforma and after its proper validation, checked for error; coding & data compilation, and segregation were done in MS Excel. Statistical analysis was performed using IBM SPSS Statistics V.30. HAM-D scores and Lipid profile changes were analysed using Paired t-test (for within-group comparisons) and independent t-test (for between-group comparisons). Chi square test was used to determine the association among categorical variables. Continuous variables were presented as mean ± standard deviation (SD) and mean difference. Categorical variables were presented as frequency (n) and percentage (%). A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 203 participants with depression who received either of the two medications were enrolled, with 105 patients in the Escitalopram group and 98 patients in the Desvenlafaxine group. The demographic and clinical characteristics were well-balanced between the two groups, as detailed below.

Age Distribution

The majority of participants were aged 18–30 years (46.3%, n=94), followed by 31–40 years (26.6%, n=54), 41–50 years (11.8%, n=24), and 51–65 years (15.3%, n=31). Both treatment groups had comparable age distributions, though the Escitalopram group had a slightly higher proportion of participants aged 51–65 years (19.0%, n=20) compared to the Desvenlafaxine group (11.2%, n=11) (fig 1)

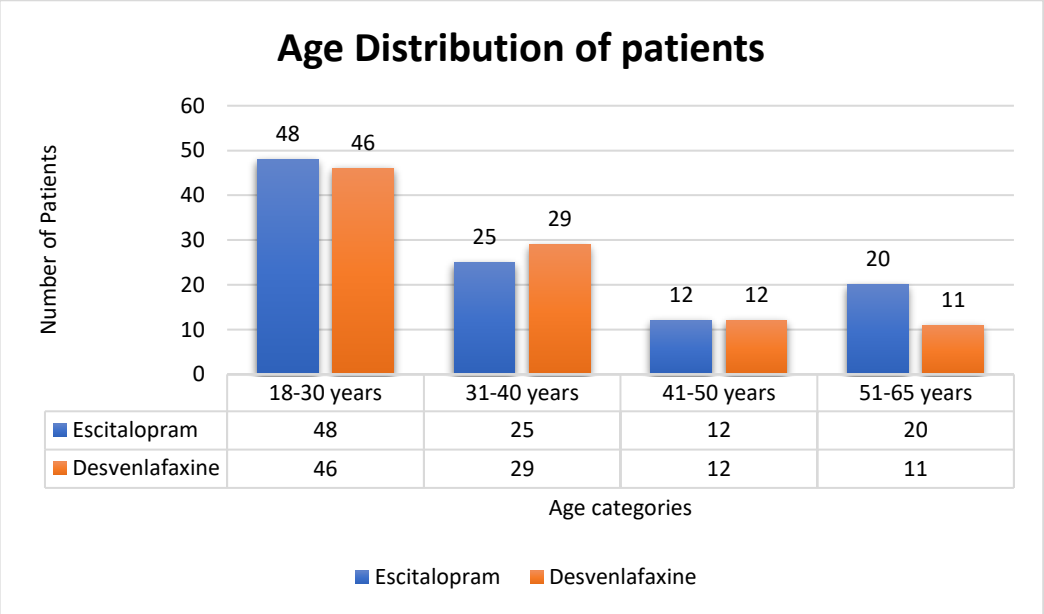

Fig 1: Age distribution of patients

Gender Distribution

The study population comprised 46.3% females (n=94) and 53.7% males (n=109). In the Escitalopram group, 52.1% (n=49) were female, while in the Desvenlafaxine group, 47.9% (n=45) were female, indicating a balanced gender distribution across groups (fig 2).

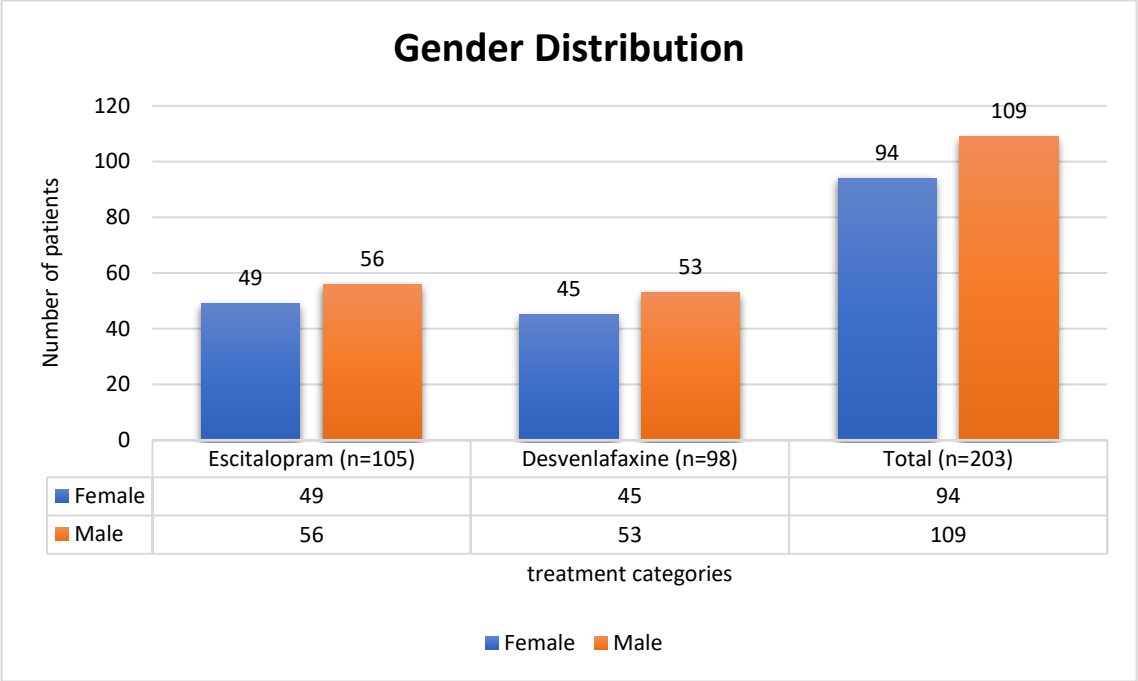

Fig 2: Gender distribution of patients

Duration of Illness

The duration of illness prior to treatment was predominantly 5–8 weeks (36.5%, n=74), followed by 1–4 weeks (19.7%, n=40), 9–12 weeks (19.2%, n=39), and 13–16 weeks (12.8%, n=26). Longer durations included 17–20 weeks (4.9%, n=10), 21–24 weeks (4.4%, n=9), and 25–48 weeks (2.5%, n=5). The Desvenlafaxine group had a higher proportion of participants with illness duration of 13–16 weeks (17.3%, n=17) compared to the Escitalopram group (8.6%, n=9) (Fig 3).

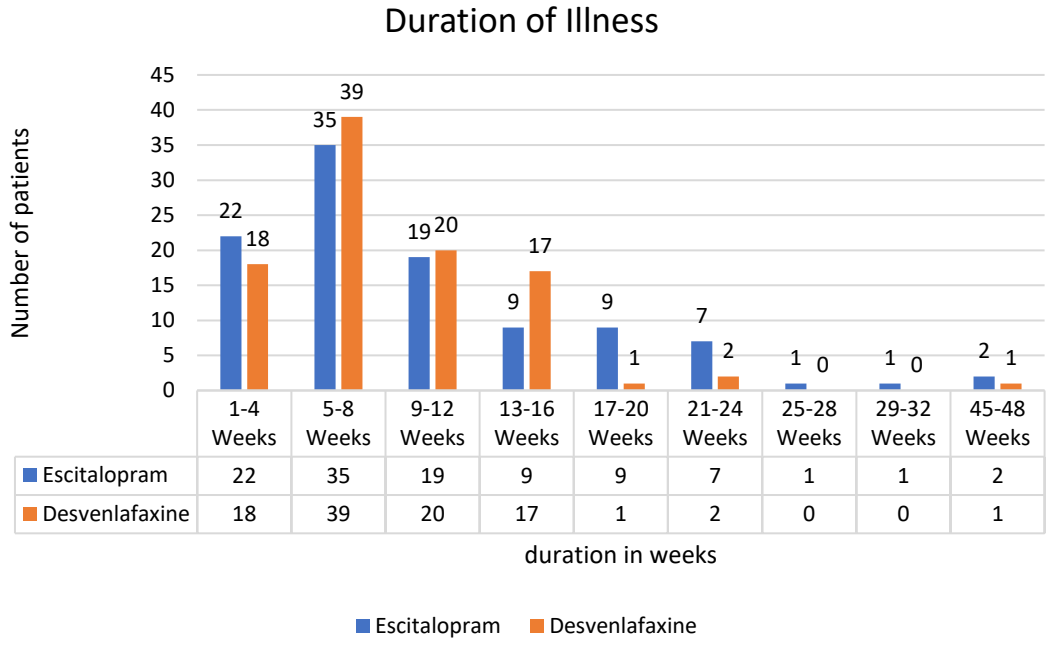

Fig 3: Duration of illness of patients

Severity of Depression

Depression severity, assessed using the Hamilton Depression Rating Scale (HAM-D), was predominantly moderate (82.3%, n=167), followed by severe (15.8%, n=32) and mild (2.0%, n=4). The Desvenlafaxine group had a higher proportion of severe depression cases (19.4%, n=19) compared to the Escitalopram group (12.4%, n=13) (Fig 4).

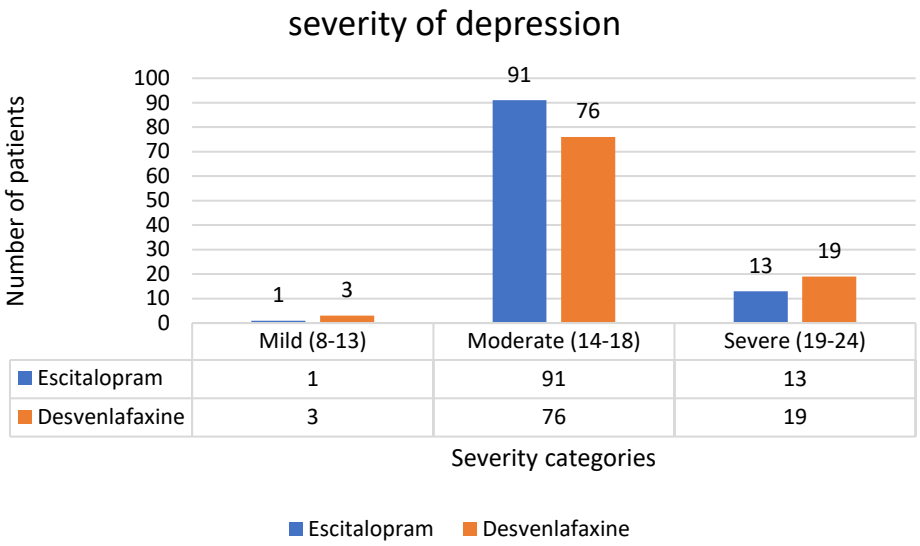

Fig 4: Severity of depression of patients

Changes in HAM-D Scores

Both treatments significantly reduced HAM-D scores over 16 weeks ($p<0.001$ for all time intervals within groups). In the Escitalopram group, mean HAM-D scores decreased from 17.10 ± 1.34 at baseline to 13.71 ± 1.73 at 4 weeks, 10.59 ± 1.64 at 8 weeks, 7.73 ± 1.64 at 12 weeks, and 4.81 ± 1.76 at 16 weeks. In the Desvenlafaxine group, scores decreased from 17.39 ± 1.91 at baseline to 14.72 ± 1.67 at 4 weeks, 12.01 ± 1.92 at 8 weeks, 9.39 ± 2.02 at 12 weeks, and 6.77 ± 2.10 at 16 weeks. (table 1)

Table 1: Within the group changes in HAM-D scores

Treatment Group	Time Interval	Mean HAM-D Scores (Mean ± SD)	p-value
Escitalopram	Baseline → 4 weeks	17.10 ± 1.34 → 13.71 ± 1.73	<0.001
	4 weeks → 8 weeks	13.71 ± 1.73 → 10.59 ± 1.64	<0.001
	8 weeks → 12 weeks	10.59 ± 1.64 → 7.73 ± 1.64	<0.001
	12 weeks → 16 weeks	7.73 ± 1.64 → 4.81 ± 1.76	<0.001
Desvenlafaxine	Baseline → 4 weeks	17.39 ± 1.91 → 14.72 ± 1.67	<0.001
	4 weeks → 8 weeks	14.72 ± 1.67 → 12.01 ± 1.92	<0.001
	8 weeks → 12 weeks	12.01 ± 1.92 → 9.39 ± 2.02	<0.001
	12 weeks → 16 weeks	9.39 ± 2.02 → 6.77 ± 2.10	<0.001

Table 2: Between Group comparison of Changes in HAM-D Scores

Time Point	Escitalopram (Mean ± SD) (N=105)	Desvenlafaxine (Mean ± SD) (N=98)	p-value
HAM-D-0 (Baseline)	17.10 ± 1.34	17.39 ± 1.50	0.156
HAM-D-4 (4 weeks)	13.71 ± 1.73	14.72 ± 1.67	<0.001

HAM-D-8 (8 weeks)	10.59 ± 1.64	12.01 ± 1.96	<0.001
HAM-D-12 (12 weeks)	7.73 ± 1.64	9.39 ± 2.02	<0.001
HAM-D-16 (16 weeks)	4.81 ± 1.76	6.77 ± 2.10	<0.001

At baseline, HAM-D scores were similar between groups (p=0.156). However, at weeks 4, 8, 12, and 16, the Escitalopram group exhibited significantly lower HAM-D scores compared to the Desvenlafaxine group (p<0.001 at each time point), indicating greater improvement in depressive symptoms with Escitalopram (table 2).

Lipid Profile Changes

Baseline Lipid Profile

At baseline, no significant differences were observed between groups for total cholesterol (TC), high-density lipoprotein (HDL), low-density lipoprotein (LDL), or very low-density lipoprotein (VLDL) (p>0.05). However, triglycerides (TG) were significantly higher in the Desvenlafaxine group (123.88 ± 40.44 mg/dl) compared to the Escitalopram group (111.50 ± 42.39 mg/dl, p=0.035) (table 3).

Lipid Profile at 16 Weeks

At week 16, TG remained significantly lower in the Escitalopram group (108.42 ± 41.56 mg/dl) compared to the Desvenlafaxine group (123.99 ± 43.60 mg/dl, p=0.010). No significant differences were observed for TC, HDL, LDL, or VLDL (p>0.05) (table 4).

Table 3: Baseline lipid profile values

Variable	Escitalopram (Mean ± SD) mg/dl	Desvenlafaxine (Mean ± SD) mg/dl	p-value (Baseline)
TC-0	165.54 ± 21.87	163.11 ± 18.68	0.397
TG-0	111.50 ± 42.39	123.88 ± 40.44	0.035
HDL-0	50.46 ± 4.22	51.02 ± 5.20	0.396
LDL-0	88.22 ± 27.58	91.64 ± 22.57	0.336
VLDL-0	20.12 ± 6.32	19.56 ± 8.08	0.58

Table 4: Between group comparison of Lipid profile values at 16 weeks

Variable	Escitalopram (Mean ± SD) mg/dl	Desvenlafaxine (Mean ± SD) mg/dl	p-value (Week 16)
TC-16 weeks	163.12 ± 22.27	166.38 ± 18.81	0.264
TG-16 weeks	108.42 ± 41.56	123.99 ± 43.60	**0.010**
HDL-16 weeks	53.75 ± 4.06	53.21 ± 5.38	0.42
LDL-16 weeks	87.97 ± 26.60	92.00 ± 23.77	0.258
VLDL-16 weeks	19.11 ± 5.79	20.08 ± 7.88	0.318

Within-Group Lipid Profile Changes

In the Escitalopram group, significant reductions were observed from baseline to week 16 in TC (165.54 ± 21.87 to 163.12 ± 22.27 mg/dl, p<0.001), TG (111.50 ± 42.39 to 108.42 ± 41.56 mg/dl, p<0.001), and VLDL (20.12 ± 6.32 to 19.11 ± 5.79 mg/dl, p<0.001), with a significant increase in HDL (50.46 ± 4.22 to 53.75 ± 4.06 mg/dl, p<0.001). LDL levels remained unchanged (p=0.842).

In the Desvenlafaxine group, TC increased significantly (163.11 ± 18.68 to 166.38 ± 18.81 mg/dl, p<0.001), as did VLDL (19.56 ± 8.08 to 20.08 ± 7.88 mg/dl, p<0.001). At the same time HDL was also increased significantly (51.02 ± 5.20 to 53.21 ± 5.38 mg/dl, p<0.001). TG levels remained stable (p=0.954), and LDL showed no significant change (p=0.839) (table 5).

Table 5: Within group changes if Lipid profile at 16 weeks

Treatment Group	Lipid Parameter	Baseline (Mean ± SD) (mg/dl)	Week 16 (Mean ± SD) (mg/dl)	p-value
Escitalopram	TC	165.54 ± 21.87	163.12 ± 22.27	<0.001
	TG	111.50 ± 42.39	108.42 ± 41.56	<0.001
	HDL	50.46 ± 4.22	53.75 ± 4.06	<0.001
	LDL	88.22 ± 27.58	87.97 ± 26.60	0.842
	VLDL	20.12 ± 6.32	19.11 ± 5.79	<0.001
Desvenlafaxine	TC	163.11 ± 18.68	166.38 ± 18.81	<0.001
	TG	123.88 ± 40.44	123.99 ± 43.60	0.954
	HDL	51.02 ± 5.20	53.21 ± 5.38	<0.001
	LDL	91.64 ± 22.57	92.00 ± 23.77	0.839
	VLDL	19.56 ± 8.08	20.08 ± 7.88	<0.001

Platelet Count Changes

In the Escitalopram group, platelet count decreased slightly from 2.4510 ± 0.4448 lakhs at baseline to 2.4329 ± 0.4433 lakhs at week 16 ($p<0.05$). In the Desvenlafaxine group, platelet count remained stable (2.3268 ± 0.3644 to 2.3273 ± 0.3631 lakhs, $p=0.806$) (table 6).

Table 6: within group changes in platelet count from baseline to 16 weeks

Treatment Group	Time Point	Platelet Count (Mean ± SD) (in lakhs)	p-value
Escitalopram	Baseline	2.4510 ± 0.4448	< 0.05
	Week 16	2.4329 ± 0.4433	
Desvenlafaxine	Baseline	2.3268 ± 0.3644	0.8
	Week 16	2.3273 ± 0.3631	

Incidence of Adverse Effects

Escitalopram Group

Adverse effects were reported by 6.7% (n=7) of patients at week 4, 5.7% (n=6) at week 8, and 3.8% (n=4) at week 12. The most common adverse effects were anorexia (5.7%, n=6), nausea (3.8%, n=4), and insomnia (2.9%, n=3), followed by headache and lethargy (1.9% each, n=2) (table 7).

Table 7: Frequency of Adverse effects in Escitalopram group

Adverse Effect	Week 4 (n, %)	Week 8 (n, %)	Week 12 (n, %)	Total (n, %)
Anorexia	2 (1.9%)	3 (2.9%)	1 (1.0%)	**6 (5.7%)**
Headache	1 (1.0%)	-	1 (1.0%)	**2 (1.9%)**
Insomnia	-	2 (1.9%)	1 (1.0%)	**3 (2.9%)**
Lethargy	2 (1.9%)	-	-	**2 (1.9%)**
Nausea	2 (1.9%)	1 (1.0%)	1 (1.0%)	**4 (3.8%)**

Desvenlafaxine Group

Adverse effects were reported by 9.2% (n=9) of patients at week 4, 5.1% (n=5) at week 8, and 3.1% (n=3) at week 12. The most frequent adverse effects included anorexia (4.1%, n=4), followed by dizziness, dry mouth, fatigue, and lethargy (2.0% each, n=2), and nausea (3.1%, n=3), headache, and

insomnia (1.0% each, n=1). Dry mouth and dizziness were exclusive to the Desvenlafaxine group (table 8).

Table 8: Frequency of Adverse effects in Desvenlafaxine group

Adverse Effect	Week 4 (n, %)	Week 8 (n, %)	Week 12 (n, %)	Total (n, %)
Anorexia	1 (1.0%)	2 (2.0%)	1 (1.0%)	**4 (4.1%)**
Dizziness	1 (1.0%)	-	1 (1.0%)	**2 (2.0%)**
Dry Mouth	2 (2.0%)	-	-	**2 (2.0%)**
Fatigue	2 (2.0%)	-	-	**2 (2.0%)**
Headache	1 (1.0%)	-	-	**1 (1.0%)**
Insomnia	-	-	1 (1.0%)	**1 (1.0%)**
Lethargy	1 (1.0%)	1 (1.0%)	-	**2 (2.0%)**
Nausea	1 (1.0%)	1 (1.0%)	1 (1.0%)	**3 (3.1%)**

Severity of Adverse Drug Reactions (ADRs)

All ADRs were non-serious, with no withdrawals required. In the Escitalopram group, ADRs were classified as probable (24%), possible (73%), and doubtful (3%). In the Desvenlafaxine group, ADRs were probable (28%), possible (69%), and doubtful (3%). Most ADRs were mild to moderate, with no severe reactions reported.

Treatment Adherence

Escitalopram group demonstrated higher adherence (92.4%) compared to the Desvenlafaxine group (89.8%), which may have contributed to its superior outcomes in reducing depressive symptoms

DISCUSSION

From the results of this study, it is evident that both Escitalopram and Desvenlafaxine are effective in reducing depressive symptoms during a 16-week treatment period, as indicated by the significant reductions in Hamilton Depression Rating Scale (HAM-D) scores in both groups. The escitalopram group showed a more significant and consistent decreased in HAM-D scores at all time points when compared to Desvenlafaxine which indicates its higher efficacy in achieving remission or minimal depressive symptoms.

Regarding the distribution of gender, escitalopram group had 51.4% male participants whereas desvenlafaxine group had 48.67% males. In the study done by Maity et al. [16] similar to our study, 64.1% of participants in the escitalopram group were male, compared to 60.5% in the desvenlafaxine group. The initial HAM-D scores were 17.10 ± 1.34 (escitalopram) and 17.39 ± 1.50 (desvenlafaxine, p=0.156), indicating no significant baseline difference.

SH Lee et al. (2022) [17] did a study comparing the efficacy and safety of Escitalopram, Desvenlafaxine, and Vortioxetine in treating depression with cognitive complaints. The findings indicated that Escitalopram resulted in a significantly greater reduction in HAM-D scores compared to Desvenlafaxine at 6th week, with an overall p-value of 0.025. Our study indicated that Escitalopram resulted in a more significant reduction in depressive symptoms, evidenced by a mean HAM-D score of 4.81 ± 1.76 at week 16, when compared to 6.77 ± 2.10 observed in the Desvenlafaxine group. Research conducted by Gupta et al. (2016) [18] and Maity et al. (2014) indicated a greater reduction in HAM-D scores in the escitalopram group when compared to desvenlafaxine group; however, these findings were not statistically significant. The present study indicates that at the end of the 4-week treatment period, the mean HAM-D score in the desvenlafaxine group decreased from a baseline of 17.39 to 14.72, while in the escitalopram group, it decreased from a baseline of 17.10 to 13.71, demonstrating a greater reduction in the escitalopram group. This contrasts with the findings of Bandaru et al. (2024) [19], which indicate a mean reduction in HAMD scores from baseline values of

22.80 to 12.40 in the desvenlafaxine group and from 22.14 to 12.62 in the escitalopram group at 4th week. However, the same study indicated a more significant reduction in HAM-D scores in the escitalopram group (6.10) compared to the desvenlafaxine group (6.16) at the 8-week mark, aligning with the findings of this study.

At baseline, no significant differences were observed between the two groups regarding Total Cholesterol (TC), High-Density Lipoprotein (HDL), Low-Density Lipoprotein (LDL), Very Low-Density Lipoprotein (VLDL), or platelet count. Triglycerides (TG) levels were significantly elevated in the Desvenlafaxine group (123.88 ± 40.44) relative to the Escitalopram group (111.50 ± 42.39) ($p = 0.035$). Over a 16-week period, the Escitalopram group demonstrated a significant increase in HDL ($p < 0.001$) and reductions in TC, TG, and VLDL, with LDL remaining unchanged ($p = 0.842$). A study conducted by Ashique et al. (2017) [20], to explore the effect of escitalopram treatment on lipid profile in depression, demonstrated a significant reduction in total cholesterol (TC), low-density lipoprotein (LDL), and triglycerides (TG) at 6 weeks, which is similar to the findings of this study. However, it also demonstrated a decrease in HDL levels, which contrasts with the findings of this study. On the other hand, a study by Amin Unis et al. (2014) [21], reported a significant increase in HDL levels in the escitalopram group. In the current study, the Desvenlafaxine group exhibited increase in total cholesterol (TC), low-density lipoprotein (LDL) but also increased in high-density lipoprotein (HDL), ($p > 0.05$), although the increases in triglycerides (TG) and LDL were not statistically significant. A study conducted by Karen A. Tourian et al. (2011) [22], demonstrated comparable results, indicating increase in all lipid parameters following treatment with desvenlafaxine.

The present study compares the Escitalopram and Desvenlafaxine groups at week 16, indicating that the sole statistically significant difference was observed in triglyceride (TG) levels ($p = 0.010$). The Escitalopram group exhibited significantly lower triglyceride levels (108.42 ± 41.56 mg/dL) in comparison to the Desvenlafaxine group (123.99 ± 43.60 mg/dL). This indicates that Escitalopram may positively influence triglyceride levels, which is significant due to the link between elevated triglycerides and cardiovascular risk. No significant differences were observed between the two groups regarding total cholesterol (TC), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) at week 16 ($p > 0.05$).

This study observed a significant reduction in platelet count within the escitalopram group, aligning with findings from H.R. Song et al. (2012) [23] which also reported a notable decrease in platelet count following escitalopram treatment by the end of first month. In contrast, no significant reduction in platelet count was noted with other antidepressants, including venlafaxine and bupropion. In the current study, desvenlafaxine did not demonstrate any significant reduction in platelet count.

The Escitalopram group exhibited adverse effects at 4 weeks (6.7%), 8 weeks (5.7%), and 12 weeks (3.8%) during the analysis of these effects over time. The most frequently observed adverse effects in this cohort included anorexia (5.7%), nausea (3.8%), and insomnia (2.9%). In the Desvenlafaxine cohort, adverse effects were documented at 4 weeks (9.2%), 8 weeks (5.1%), and 12 weeks (3.1%). The most commonly reported adverse effects in this cohort included anorexia (4.1%), lethargy (2.0%), dizziness (2.0%), dry mouth (2.0%), and fatigue (2.0%).

A comparison of adverse effects between the two groups revealed that the overall incidence was marginally higher with Desvenlafaxine (9.2%) compared to Escitalopram (6.7%) at the 4-week mark. Anorexia and nausea occurred frequently in both groups, whereas dry mouth and dizziness were exclusively reported in the Desvenlafaxine group.

The findings align with prior research comparing the safety profiles of Escitalopram and Desvenlafaxine. A randomized, open-label study by Vishal R. Tandon et al. (2016) [65], demonstrated that both Escitalopram and Desvenlafaxine significantly decreased depression and anxiety scores from baseline. Notably, Escitalopram showed greater tolerance and a reduced incidence of side effects compared to Desvenlafaxine.

In conclusion, both Escitalopram and Desvenlafaxine are effective for treating depression and anxiety; however, Escitalopram appears to have a more favorable adverse effect profile, especially regarding the occurrence of anorexia, nausea, dry mouth, and dizziness.

CONCLUSION

This 16-week hospital-based observational study compared escitalopram and desvenlafaxine in treating Major Depressive Disorder. Both drugs significantly reduced HAM-D scores, with escitalopram showing superior efficacy, particularly within the first 8 weeks. Escitalopram also improved lipid profiles and slightly decreased platelet counts, while desvenlafaxine increased certain lipid parameters. Adverse effects were more frequent with desvenlafaxine, including dry mouth and dizziness. Most side effects were mild to moderate. Overall, escitalopram was more effective and better tolerated, though desvenlafaxine remains a valid alternative. The study highlights the importance of individualized therapy in optimizing outcomes for patients with depression.

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CONFLICT OF INTEREST

No conflict of interest.

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