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Imipramine versus escitalopram in generalized anxiety disorder

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Abstract:
Generalized anxiety disorder (GAD) is a common, chronic condition, and real-world comparative data on imipramine and escitalopram are limited. Therefore, it is of interest to compare their effectiveness and safety in outpatients with newly diagnosed GAD over 24 weeks. In this prospective observational study at Gandhi Medical College and Hamidia Hospital, Bhopal, 100 patients received imipramine (n=50) or escitalopram (n=50) at the treating physician's discretion. Anxiety was assessed with the Hamilton Anxiety Rating Scale (HAM-A) at baseline and at weeks 4, 12 and 24, and adverse drug reactions were recorded. Mean HAM-A score fell from 35.05 to 9.42 (73.1%) with imipramine and from 36.08 to 8.95 (75.2%) with escitalopram. Drowsiness was significantly more frequent with imipramine (36% vs 2%; $P<0.001$). Thus, both drugs produced comparable, substantial reductions in anxiety, with better tolerability for escitalopram; randomized trials are warranted.

Keywords: Generalized anxiety disorder (GAD), imipramine, escitalopram, hamilton anxiety rating scale, tricyclic antidepressant, selective serotonin reuptake inhibitor

Background:
Mental health is an essential component of overall health, encompassing emotional, psychological and social well-being **[1].** Anxiety disorders are among the most prevalent psychiatric conditions worldwide and impose a substantial burden on individuals and healthcare systems. Generalized anxiety disorder (GAD) is characterized by excessive, persistent and difficult to control worry accompanied by psychological and somatic symptoms. Its reported one-year prevalence is approximately 3.8%, with community based estimates ranging from 1.6% to 5.0% and higher rates reported in clinical settings **[2, 3].** GAD is frequently associated with depression and other psychiatric comorbidities, which may contribute to greater symptom severity, functional impairment and a more persistent clinical course. Chronic anxiety has also been associated with adverse physical health outcomes, further emphasizing the importance of effective and sustained treatment **[4-6].** Guidelines recommend selective serotonin reuptake inhibitors (SSRIs) and serotonin–noradrenaline reuptake inhibitors as first-line pharmacological treatment for GAD **[7].** Imipramine, a tricyclic antidepressant (TCA) and escitalopram, an SSRI, differ in tolerability: TCAs are associated with more anticholinergic adverse effects **[8]**, whereas escitalopram is generally well tolerated **[9]**. Therefore, it is of interest to compare the effectiveness (change in Hamilton Anxiety Rating Scale (HAM-A) score) and safety (adverse drug reactions) of imipramine and escitalopram in outpatients with GAD over 24 weeks.

Methods:
Study design and setting:
This prospective observational study was conducted in the Department of Psychiatry, Gandhi Medical College and Hamidia Hospital, Bhopal, Madhya Pradesh, India. Patients with newly diagnosed GAD were followed for 24 weeks. Treatment was prescribed at the treating physician's discretion and patients receiving imipramine or escitalopram were followed to assess effectiveness and safety in routine practice. The Institutional Ethics Committee of Gandhi Medical College, Bhopal, approved the study and all participants provided written informed consent before enrollment.

Study participants:
One hundred men and women aged 18–60 years who met the diagnostic criteria for newly diagnosed GAD were included (imipramine, n=50; escitalopram, n=50). Exclusion criteria were lifetime psychotic or bipolar disorder, dementia, elevated suicide risk, medical instability, ongoing psychotherapy or antidepressant and anxiolytic treatment, use of other psychoactive medications, drug abuse or dependence, pregnancy or lactation and unwillingness to participate.

Baseline assessment and study procedure:
At enrollment, age, sex, body mass index (BMI), associated medical conditions, personal habits; family history, previous drug history and duration of anxiety and depressive symptoms were recorded. Patients were followed at baseline and at weeks 1, 4, 12 and 24. Imipramine was initiated at 75 mg once daily (maximum 150 mg/day) and escitalopram at 10 mg once daily (maximum 20 mg/day), both orally, with dose escalation according to clinical response. Treatment exposure, dose changes and adverse events were documented at each visit.

Assessment of anxiety severity:
Anxiety severity was assessed using the HAM-A at baseline and at weeks 4, 12 and 24. The 14-item scale scores each item from 0 to 4 (total 0–56); higher scores indicate greater severity **[10]**. The

primary outcome was the change in HAM-A score from baseline over 24 weeks. Percentage reduction in mean HAM-A score at each visit was calculated as: Percentage reduction = ((Baseline mean HAM-A − Follow-up mean HAM-A) / Baseline mean HAM-A) × 100. Safety was assessed. By the occurrence and frequency of adverse drug reactions (ADRs) reported during follow-up.

Statistical analysis:

Continuous variables were summarized as mean±standard deviation (SD) and categorical variables as frequencies and percentages. We compared baseline characteristics using Welch's t-test (continuous variables) and the chi-square test (categorical variables). Changes in mean HAM-A scores were summarized descriptively as absolute and percentage reductions from baseline. ADRs were compared using Fisher's exact test. Analyses were performed in IBM SPSS Statistics version 22.0 (IBM Corp., Armonk, NY, USA); a two-sided $P<0.05$ was considered statistically significant.

Results:

A total of 100 patients with GAD were enrolled and completed baseline assessment: 50 received imipramine and 50 received escitalopram. Mean age was 32.80±6.85 years with imipramine and 32.22±3.43 years with escitalopram; males constituted 64% and 70%, respectively. Mean BMI was 28.20±7.11 and 27.15±5.93 kg/m², respectively. The groups did not differ significantly in age, sex, or BMI (all $P>0.05$; **Table 1**). Mean duration of anxiety symptoms was 7.11±2.19 months with imipramine and 7.27±2.16 months with escitalopram; most patients (62% and 66%, respectively) had symptoms for 6–9 months. Mean duration of depressive symptoms was 7.96±2.13 and 7.11±2.24 months, respectively (difference not significant, $P=0.055$; **Table 1**). Imipramine was started at 75 mg/day and escitalopram at 10 mg/day; all patients remained on starting doses at weeks 1 and 4. At weeks 12 and 24, 43 patients (86%) on imipramine received 75 mg/day and 7 (14%) received 150 mg/day, while 31 (62%) on escitalopram received 10 mg/day and 19 (38%) received 20 mg/day **(Table 2).** Mean HAM-A scores decreased progressively in both groups **(Table 3, Figure 1).** With imipramine, scores fell from 35.05 at baseline to 24.10, 14.59 and 9.42 at weeks 4, 12 and 24, respectively; with escitalopram, they fell from 36.08 to 25.26, 14.15 and 8.95. Reductions from baseline with imipramine were 10.95 points (31.2%) at week 4, 20.46 points (58.4%) at week 12 and 25.63 points (73.1%) at week 24; with escitalopram, they were 10.82 (30.0%), 21.93 (60.8%) and 27.13 points (75.2%). Week-24 scores differed by only 0.47 points (8.95 vs 9.42), with a numerically greater percentage reduction with escitalopram (75.2% vs 73.1%). ADRs occurred in both groups. With imipramine, the most frequent were weight gain (21 patients, 42%), drowsiness (18, 36%) and dry mouth (12, 24%); with escitalopram, nausea/vomiting and weight gain (12 each, 24%) and dry mouth (8, 16%). Drowsiness was significantly more frequent with imipramine (36% vs 2%; $P<0.001$). Weight gain was numerically more frequent with imipramine (42% vs 24%; $P=0.088$). Other ADRs did not differ significantly **(Table 4).** One imipramine-treated patient (2%) reported delusion; no hallucinations or convulsions occurred in either group.

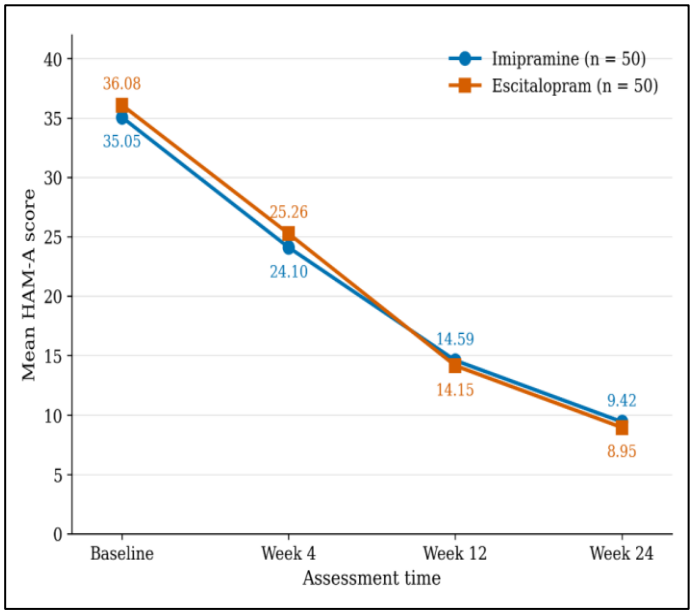


Figure 1: Change in mean HAM-A scores over 24 weeks according to treatment group

Table 1: Baseline demographic and clinical characteristics of the study participants

Characteristic	Imipramine (n=50)	Escitalopram (n=50)	P-value
Age, years, mean±SD	32.80±6.85	32.22±3.43	0.594
Age 20 – <25, n (%)	11 (22.0)	9 (18.0)	
Age 25 – <30, n (%)	25 (50.0)	24 (48.0)	0.885*
Age 30 – <35, n (%)	9 (18.0)	12 (24.0)	
Age ≥35, n (%)	5 (10.0)	5 (10.0)	
Male, n (%)	32 (64.0)	35 (70.0)	0.523
Female, n (%)	18 (36.0)	15 (30.0)	
BMI, kg/m², mean±SD	28.20±7.11	27.15±5.93	0.425
BMI <18.5, n (%)	6 (12.0)	5 (10.0)	
BMI 18.5–24.9, n (%)	13 (26.0)	13 (26.0)	0.971*
BMI 25.0–29.0, n (%)	22 (44.0)	24 (48.0)	
BMI 29.0-30.0, n (%)	9 (18.0)	8 (16.0)	
Duration of anxiety, months, mean±SD	7.11±2.19	7.27±2.16	0.714
Duration of anxiety <6 mo, n (%)	11 (22.0)	7 (14.0)	
Duration of anxiety 6–9 mo, n (%)	31 (62.0)	33 (66.0)	0.556*

Duration of anxiety >9 mo, n (%)	8 (16.0)	10 (20.0)	
Duration of depression, months, mean±SD	7.96±2.13	7.11±2.24	0.055

**Overall chi-square p-value for the categorical distribution across all listed subgroups.*

Table 2: Treatment exposure, follow-up visits and dose distribution

Assessment	Imipramine, n (%)	Imipramine dose (mg/day)	Escitalopram, n (%)	Escitalopram dose (mg/day)
Baseline	50 (100)	0	50 (100)	0
Week 1	50 (100)	75	50 (100)	10
Week 4	50 (100)	75	50 (100)	10
Week 12	43 (86) / 7 (14)	75 / 150	31 (62) / 19 (38)	10/20 (mg/day)
Week 24	43 (86) / 7 (14)	75 / 150	31 (62) / 19 (38)	10/20 (mg/day)

Table 3: Mean HAM-A scores and percentage reduction from baseline over 24 weeks

Treatment group	Baseline	Week 4	Week 12	Week 24
Imipramine (n=50)	35.05	24.10 (31.2%)	14.59 (58.4%)	9.42 (73.1%)
Escitalopram (n=50)	36.08	25.26 (30.0%)	14.15 (60.8%)	8.95 (75.2%)

Values in parentheses are percentage reductions from baseline.

Table 4: Adverse drug reactions reported during the 24-week follow-up

Adverse drug reaction	Imipramine, n (%)	Escitalopram, n (%)	P-value†
Nausea and vomiting	8 (16.0)	12 (24.0)	0.454
Constipation	6 (12.0)	4 (8.0)	0.741
Dry mouth	12 (24.0)	8 (16.0)	0.454
Drowsiness	18 (36.0)	1 (2.0)	<0.001
Diarrhea	2 (4.0)	1 (2.0)	1
Abdominal cramps	7 (14.0)	5 (10.0)	0.76
Weight gain	21 (42.0)	12 (24.0)	0.088
Hallucination	0 (0)	0 (0)	1
Delusion	1 (2.0)	0 (0)	1
Convulsion	0 (0)	0 (0)	1

†Fisher's exact test.

Discussion:

In this prospective study of outpatients with GAD, imipramine and escitalopram produced comparable, substantial reductions in HAM-A scores over 24 weeks (73.1% vs 75.2%), with a week-24 difference of only 0.47 points. Drowsiness was significantly more frequent with imipramine (36% vs 2%; P<0.001). The similar effectiveness is consistent with earlier evidence. In GAD, escitalopram was superior to placebo (response 68% vs 41% at week 8) **[11]** and comparable to agomelatine in severe GAD (64.8% vs 60.9%) **[12]** and its efficacy across anxiety disorders has been reviewed **[13]**. Sertraline and imipramine produced similar responses in depressed patients with prominent anxiety (60% vs 58%) **[14]**. Indirect evidence from depression points the same way: SSRIs showed modest advantages over TCAs in young patients **[15]**, escitalopram was at least as effective as milnacipran **[16],** and comparative studies in older patients are broadly consistent **[17, 18]**. These comparisons should be interpreted cautiously because populations, designs, durations and outcome definitions differ. The ADR profile matched the pharmacology of the two drugs. Weight gain (42%) and drowsiness (36%) were most frequent with imipramine and nausea or vomiting and weight gain (24% each) were most frequent with escitalopram. Earlier studies likewise reported more dry mouth, constipation and urinary difficulty with imipramine than with an SSRI-class comparator **[8]** and more dry mouth and somnolence with amitriptyline than with citalopram **[19, 20]**. Escitalopram had a low adverse-event rate in long-term treatment **[21]** and pooled data from more than 4,000 patients identified nausea as the main excess event over placebo **[9]**. Because treatment allocation followed clinical practice rather than randomization, these findings describe comparative effectiveness and do not establish superiority or causality. Genetic variation in CYP2D6, CYP2C19 and CYP2B6 can alter antidepressant exposure, response and ADRs, and CPIC guidelines provide genotype-informed recommendations for SSRIs and TCAs, including imipramine **[22, 23]**. Pharmacogenetically guided therapy may improve outcomes and reduce adverse events **[24, 25]** and actionable CYP2D6 and CYP2C19 variants are highly prevalent among Central Indian patients with common mental disorders **[26]**. An umbrella review of randomized trials likewise reported higher remission and response rates with pharmacogenomic-guided antidepressant prescribing **[27]**. Incorporating pharmacogenomic data may therefore help individualize antidepressant selection in future GAD studies. More broadly, antidepressants outperform placebo in GAD **[28]** and the rising global burden of anxiety disorders **[29]** underscores the need for real-world comparative evidence. A 2025 meta-analysis in major depression, although a different disorder, likewise reported higher response or remission with escitalopram than with other antidepressants **[30]**. GAD is among the most common mental disorders and is frequently managed in primary care **[31, 32]**, and recent US data show a rising one-year prevalence (2.1% in 2012 to 7.4% in 2022), with pre-existing depression the strongest risk factor **[33]**. Reviews consistently support SSRIs and SNRIs, alongside cognitive behavioral therapy, as first-line treatment, and advise against benzodiazepines as first-line or long-term therapy **[32, 34]**. Imipramine is regarded as efficacious in GAD but is generally reserved for patients unresponsive to multiple agents because of its tolerability profile and cardiotoxicity in overdose

[35], which is consistent with the higher rate of drowsiness with imipramine observed here. Imipramine has also been shown to be efficacious in GAD without comorbid depression or panic disorder, and it is suggested as an option when depressed mood is present, typically after other agents have failed **[35]**; this is relevant because our patients had depressive symptoms for a mean of about 7 to 8 months. Up To Date rates imipramine higher than escitalopram for drowsiness, anticholinergic effects and weight gain **[35]**, in line with our ADR findings. The doses used here (imipramine 75 to 150 mg/day; escitalopram 10 to 20 mg/day) lie within the usual ranges for GAD (75 to 200 and 10 to 20 mg/day, respectively) **[35]**. This study has limitations. It was a single-centre, open-label, non-randomized study, so selection bias, confounding and observer bias cannot be excluded. The absence of a placebo or untreated group precludes distinguishing drug effects from spontaneous recovery or regression to the mean. The sample (50 per group) limits precision, particularly for infrequent ADRs. The study used only HAM-A as an effectiveness measure; it did not assess patient-reported, functional, or quality-of-life outcomes. Individual-level HAM-A scores and SDs were not available, so the authors described the primary outcome but did not compare it statistically. Finally, the 24-week follow-up does not establish longer-term comparative effectiveness or safety.

Conclusion:
Imipramine and escitalopram were both associated with substantial, sustained improvement in anxiety over 24 weeks (HAM-A reduction 73.1% vs. 75.2%), indicating broadly comparable effectiveness. Escitalopram caused significantly less drowsiness (2% vs. 36%) and had a more favourable overall tolerability profile. Larger randomized controlled studies are needed to confirm these findings.

Advancement to knowledge:
Imipramine and escitalopram gave comparable 24-week anxiety reduction in routine GAD care, but escitalopram was better tolerated, with significantly less drowsiness.

Declarations:
Ethics approval and consent to participate:
The study followed the Declaration of Helsinki and was approved by the Institutional Ethics Committee of Gandhi Medical College and Hamidia Hospital, Bhopal, Madhya Pradesh, India (approval no. 627/MC/IEC/2020, dated 04/01/2020). Written informed consent was obtained from all participants before enrolment.

Consent for publication:
Not applicable. No individual-level identifiable information or images are presented in this manuscript.

Availability of data and materials:
The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request, subject to applicable ethical and institutional restrictions.

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Authors' contributions:
YJ, JPA: Study conception and design, data collection, data analysis, interpretation of results and manuscript preparation.
AM: Data collection, interpretation of results and critical revision of the manuscript.
PKM: Data collection, data interpretation and critical revision of the manuscript.
HV: Data collection, interpretation of results and critical revision of the manuscript.
SC: Data analysis, interpretation of results, literature review and manuscript preparation and revision.
JPA: Clinical supervision, study oversight, interpretation of clinical findings and critical revision of the manuscript.
All authors read and approved the final manuscript and agreed to be accountable for all aspects of the work.

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References:
[1] Wittchen HU *et al. Arch Gen Psychiatry.* 1994 **51**:355 [PMID: 8179459]
[2] Tyrer P *et al. Psychol Med.* 2004 **34**:1385 [PMID: 15724870]
[3] Munk-Jørgensen P *et al. Psychiatr Serv.* 2006 **57**:1738 [PMID: 17158488]
[4] Hasin DS *et al. Arch Gen Psychiatry.* 2005 **62**:1097 [PMID: 16203955]
[5] Tully PJ *et al. Psychol Health Med.* 2013 **18**:627 [PMID: 23324073]
[6] Martens EJ *et al. Arch Gen Psychiatry.* 2010 **67**:750 [PMID: 20603456]
[7] Bandelow B *et al. World J Biol Psychiatry.* 2008 **9**:248 [PMID: 18949648]
[8] Suominen J *et al. Pharmacopsychiatry.* 1988 **21**:238 [PMID: 3067246]
[9] Baldwin DS *et al. Ann Pharmacother.* 2007 **41**:1583 [PMID: 17848424]

[10] Hamilton M. *Br J Med Psychol.* 1959 **32**:50 [PMID: 13638508]
[11] Davidson JRT *et al. Depress Anxiety.* 2004 **19**:234 [PMID: 15274172]
[12] Stein DJ *et al. Eur Neuropsychopharmacol.* 2018 **28**:970 [PMID: 30135032]
[13] Pelissolo A. *Encephale.* 2008 **34**:400 [PMID: 18922243]
[14] Russell JM *et al. Depress Anxiety.* 2001 **13**:18 [PMID: 11233456]
[15] Qin B *et al. Clin Ther.* 2014 **36**:1087 [PMID: 24998011]
[16] https://www.ijbcp.com/index.php/ijbcp/article/view/25
[17] Krause M *et al. Eur Neuropsychopharmacol.* 2019 **29**:1003 [PMID: 31327506]
[18] Forlenza OV *et al. Sao Paulo Med J.* 2000 **118**:99 [PMID: 10887385]
[19] Kyle CJ *et al. Depress Anxiety.* 1998 **8**:147 [PMID: 9871816]
[20] Mathur A *et al. Indian J Psychiatry.* 2005 **47**:89 [PMID: 20711288]
[21] Jiang K *et al. Neuropsychiatr Dis Treat.* 2017 **13**:515 [PMID: 28255239]
[22] Bousman CA *et al. Clin Pharmacol Ther.* 2023 **114**:51 [PMID: 37032427]
[23] Hicks JK *et al. Clin Pharmacol Ther.* 2017 **102**:37 [PMID: 27997040]
[24] Chenchula S *et al. J Affect Disord.* 2024 **367**:832 [PMID: 39265864]
[25] Chenchula S *et al. Pharmacogenomics J.* 2024 **24**:9 [PMID: 38490995]
[26] Chenchula S *et al. Front Pharmacol.* 2025 **16**:1697866 [PMID: 41378208]
[27] Tesfamicael KG *et al. Front Psychiatry.* 2024 **15**:1276410 [PMID: 39086729]
[28] Kopcalic K *et al. Cochrane Database Syst Rev.* 2025 **1**:CD012942 [PMID: 39880377]
[29] Zhou J *et al. Depress Anxiety.* 2025 **2025**:4178541 [PMID: 40688542]
[30] Das P *et al. Int J Pharm Pharm Sci.* 2025 **17**:47 [DOI: 10.22159/ijpps.2025v17i8.55347]
[31] https://pubmed.ncbi.nlm.nih.gov/28722900/
[32] DeGeorge KC *et al. Am Fam Physician.* 2022 **106**:157 [PMID: 35977134]
[33] Druet-Cabanac A *et al. Curr Med Res Opin.* 2025 **41**:1053 [PMID: 40611531]
[34] Byrne GJ. *Curr Opin Psychiatry.* 2023 **36**:134 [PMID: 36705012]
[35] https://www.ncbi.nlm.nih.gov/books/NBK552064/

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