

RESEARCH ARTICLE

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A comparative evaluation of the efficacy and safety of clonidine and atomoxetine combined with behavioural interventions in children with autism spectrum disorder and comorbid attention-deficit/hyperactivity disorder

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Submitted: 3rd April 2026

Accepted: 4th June 2026

Published: 31st October 2026

[ID](#): Orcid ID

Abstract

Objective: To compare the clinical efficacy and safety of atomoxetine versus clonidine, each combined with structured behavioural interventions, in children aged 6–12 years with autism spectrum disorder (ASD) and comorbid attention-deficit/hyperactivity disorder (ADHD).

Methods: A retrospective observational cohort study was conducted at the Imam Al-Hussein Institute for Children with Autism in Najaf, Iraq, between October 2025 and March 2026. Sixty-two children were allocated to two equal groups: atomoxetine plus behavioural intervention (n=31) or clonidine plus behavioural intervention (n=31). Outcomes were measured using DSM-5 criteria and Conners' Rating Scales at baseline and three follow-up intervals (1–3, 3–6, and 6–12 months). Data were analysed using two-way repeated-measures ANOVA, Bonferroni post-hoc tests, and chi-square tests.

Results: The atomoxetine group showed significantly greater mean symptom improvement than the clonidine group (mean improvement score –13.4 vs. –9.5; p=0.0011), with effects emerging after 3 months and increasing over time. Adverse effects were more frequent in the clonidine group, notably sedation (70.9% vs. 9.6%), irritability (45% vs. 12.9%), and fatigue (61.2% vs. 16.4%). Atomoxetine was associated with mild appetite loss (35%) and gastrointestinal symptoms (29%).

Conclusion: Atomoxetine combined with behavioural intervention demonstrated superior and more sustained clinical improvement and a more favourable safety profile than clonidine in this observational cohort. However, owing to the non-randomised design and baseline imbalances, these findings should be viewed as hypothesis-generating. Randomised controlled trials are needed to confirm superiority.

Keywords: Autism spectrum disorder, Attention deficit hyperactivity disorder, Atomoxetine, Clonidine, Child

Plain English Summary

Many children with autism spectrum disorder (ASD) also have attention-deficit/hyperactivity disorder (ADHD), which makes daily life very challenging for them and their families. Sometimes stimulant medications are not suitable for these children, so doctors use non-stimulant drugs such as atomoxetine or clonidine. However, it is not clear which of these two works better or has fewer side effects when used together with behavioural therapy. In this study, we looked back at the medical records of 62 children aged 6–12 years who received either atomoxetine or clonidine alongside a structured behavioural programme at a specialised centre in Iraq. We measured their symptoms using standard questionnaires at the start of treatment and again after 1–3 months, 3–6 months, and 6–12 months.

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Children who took atomoxetine improved nearly twice as much as those who took clonidine, and the benefits became larger over time. Clonidine caused more side effects such as sleepiness, dizziness, irritability and tiredness, while atomoxetine mainly reduced appetite and caused mild stomach upset. This study suggests that atomoxetine may be a better choice for long-term treatment, but because the study was not a randomised trial, more research is needed to be certain.

Introduction

Autism spectrum disorder (ASD) is a complex neurodevelopmental condition characterised by persistent deficits in social communication and the presence of restricted, repetitive patterns of behaviour (1). The clinical picture is often complicated by comorbid psychiatric conditions, among which attention-deficit/hyperactivity disorder (ADHD) is one of the most common, affecting up to 45% of children with ASD (2). The co-occurrence of ASD and ADHD amplifies behavioural, emotional and functional difficulties, imposing substantial burdens on children, families and health systems (3).

The diagnosis of both ASD and ADHD remains clinically driven, as validated biological markers are lacking (4). Consequently, integrated multimodal treatment plans that combine behavioural and pharmacological interventions are increasingly recommended (5, 6). In the pharmacological domain, non-stimulant medications play a critical role, especially for children who do not tolerate stimulants because of anxiety, sleep disturbances or tics (6).

Clonidine is an alpha-2 adrenergic receptor agonist that reduces central noradrenergic outflow. It has been used off-label to manage hyperactivity, irritability and sleep disturbances in children with ASD-ADHD (7). Atomoxetine is a selective norepinephrine reuptake inhibitor that enhances noradrenergic and dopaminergic transmission in the prefrontal cortex, improving attention and behavioural control with a generally favourable safety profile (8).

Although non-stimulants are generally safer than stimulants in this population, direct head-to-head comparisons between clonidine and atomoxetine are scarce. Existing studies are limited by small samples, short follow-up periods, heterogeneous designs and inconsistent adverse event reporting (7, 8). This gap in the literature motivated the present study, which aimed to compare the efficacy and safety of atomoxetine versus clonidine – each combined with a structured behavioural intervention – in children with ASD and comorbid ADHD. We specifically evaluated symptom improvement across different treatment durations and systematically documented adverse effects.

Materials and Methods

Study design and setting

This was a retrospective observational cohort study conducted at the Imam Al-Hussein Institute

for Children with Autism in Najaf, Iraq, between 10 October 2025 and 1 March 2026. The study adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cohort studies.

Participants

Children aged 6–12 years with a clinical diagnosis of ASD and comorbid ADHD were eligible for inclusion. All participants were enrolled in a structured behavioural intervention programme at the institute. Exclusion criteria were: prior exposure to any psychiatric or neurological medication (to avoid confounding), the presence of unstable medical comorbidities, or a history of hypersensitivity to clonidine or atomoxetine.

A total of 62 children were included. Based on clinical decision-making (not randomisation), they were allocated to two equal groups: 31 received atomoxetine plus behavioural intervention, and 31 received clonidine plus behavioural intervention. The allocation reflected real-world clinical practice, where the choice of medication was determined by the treating physician in consultation with caregivers.

Interventions

Pharmacological treatment

In the clonidine group, treatment was initiated at 0.05 mg at night and titrated upward weekly according to standard clinical guidelines (9), with a maximum dose not exceeding 0.2 mg/day. In the atomoxetine group, the starting dose was 0.5 mg/kg/day (morning or afternoon), increased gradually to a maximum of 1.2 mg/kg/day as tolerated (8). Doses were adjusted based on clinical response and adverse effects.

Behavioural intervention

All children participated in a structured behavioural intervention programme delivered at the institute. The programme included parent training, social skills groups and individual behaviour therapy based on applied behaviour analysis principles. The intervention was standardised across both groups.

Data collection and outcome measures

Data were extracted from medical records and structured caregiver interviews conducted during routine clinic visits. The following time points were defined: baseline (before treatment), early

follow-up (1–3 months), intermediate follow-up (3–6 months) and late follow-up (6–12 months). Two validated instruments were used: the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) criteria for ASD and ADHD (10), and the Conners' Comprehensive Behaviour Rating Scales (11). Each item was rated on a four-point scale (0 = none to 3 = severe). Total symptom scores were calculated by summing item scores per participant. Improvement scores were defined as the change from baseline to each follow-up point (negative values indicate improvement). Adverse effects were systematically documented using a standardised checklist completed by caregivers and confirmed by clinical staff at each visit.

Sample size justification

The required sample size was estimated using methods for comparing two independent group means (12), assuming a significance level of $\alpha = 0.05$, 90% power, and a large effect size ($f = 0.40$). The calculation yielded a minimum of 62 participants (31 per group). Although the primary analysis involved repeated measures, this sample size was considered adequate for detecting large main effects.

Statistical analysis

Data were analysed using GraphPad Prism 9.0 and Microsoft Excel 2019. Descriptive statistics were calculated: frequencies and percentages for categorical variables, mean $\pm$ standard deviation (SD) for continuous variables. Normality was assessed using the Shapiro-Wilk test.

For efficacy outcomes, a two-way repeated-measures analysis of variance (ANOVA) was performed with a within-subjects factor (time: four time points) and a between-

subjects factor (treatment group: atomoxetine vs clonidine). When significant main effects or interactions were found, Bonferroni-adjusted post-hoc comparisons were conducted. Effect sizes (partial η^2) were calculated for significant ANOVA effects.

For safety outcomes, the frequency of each adverse effect was compared between groups using the chi-square test or Fisher's exact test as appropriate. To reduce the risk of type I errors due to multiple comparisons, the Benjamini-Hochberg false discovery rate correction was applied. In addition, a repeated-measures ANOVA was performed on the total adverse effect scores across time points. All statistical tests were two-sided, and a p -value < 0.05 was considered statistically significant. No imputation for missing data was performed because the completeness of records was high ($>95\%$ for all variables).

Results

Baseline characteristics

Table 1 summarises the baseline demographic and clinical characteristics of the 62 participants. The two groups were generally comparable, with no statistically significant differences in age ($p = 0.176$), gender distribution ($p = 0.937$), residence ($p > 0.999$), socioeconomic status ($p = 0.210$), family history of similar conditions ($p = 0.151$), pregnancy complications ($p = 0.163$), or duration of therapy across time intervals ($p = 0.1552$ for overall difference). However, statistically significant baseline differences were observed for body weight (atomoxetine group: 24.5 ± 6.4 kg; clonidine group: 26.3 ± 4.1 kg; $p = 0.046$) and age at diagnosis (atomoxetine: 7.16 ± 0.56 years; clonidine: 6.69 ± 0.76 years; $p = 0.033$). These imbalances were adjusted for in sensitivity analyses (see below), and the primary results remained unchanged after adjustment.

Table 1: Baseline demographic and clinical characteristics of participants (N=62)

Variable	Atomoxetine + behavioural (n=31)	Clonidine + behavioural (n=31)	p-value
Age (years), mean $\pm$ SD	8.16 $\pm$ 1.12	7.69 $\pm$ 0.94	0.176
Weight (kg), mean $\pm$ SD	24.5 $\pm$ 6.44	26.3 $\pm$ 4.13	0.046
Age at diagnosis (years), mean $\pm$ SD	7.16 $\pm$ 0.56	6.69 $\pm$ 0.76	0.033
Gender, male (%)	22 (71.0)	26 (83.9)	0.937
Residence, urban (%)	21 (67.7)	25 (80.6)	>0.999
Socioeconomic status, low (%)	23 (74.2)	24 (77.4)	0.21
Family history of similar condition, yes (%)	9 (30.0)	14 (45.0)	0.151
Pregnancy complications, yes (%)	16 (51.8)	12 (38.7)	0.163
Improvement score (baseline to end), mean $\pm$ SD	-13.4 $\pm$ 1.33	-9.51 $\pm$ 3.67	0.0011

Continuous variables were compared using an unpaired t-test; categorical variables using a chi-square test

Treatment efficacy

The atomoxetine group showed a significantly greater mean improvement score than the

clonidine group (-13.4 ± 1.33 vs -9.51 ± 3.67 ; $p = 0.0011$). The two-way repeated-measures ANOVA revealed significant main effects for

treatment group ($p = 0.0274$), time ($p < 0.0001$) and a significant group-by-time interaction ($p =$

0.0007), indicating that the difference between treatments increased over time (Table 2).

Table 2: Two-way repeated-measures ANOVA for clinical improvement scores

Source of variation	F-value	p-value	Partial η^2
Treatment group	5.12	0.0274	0.08
Time	18.94	<0.0001	0.24
Group $\times$ Time interaction	7.86	0.0007	0.12

Bonferroni post-hoc comparisons (Table 3) showed no significant difference between groups at the 1–3-month follow-up (mean difference -2.65 , $p = 0.1182$). However, significant differences emerged at 3–6 months (mean difference -3.83 , $p = 0.0331$) and became highly significant at 6–12 months (mean difference -7.30 , $p = 0.0001$). Within the clonidine group,

improvement from 1–3 months to 6–12 months was significant (mean difference -3.26 , $p = 0.0431$), but no further improvement occurred after 3–6 months. Within the atomoxetine group, differences between time points did not reach statistical significance after Bonferroni correction, although there was a trend toward increasing improvement over time.

Table 3: Bonferroni post-hoc comparisons for improvement scores between groups at each time point

Time point	Mean difference (Atomoxetine – Clonidine)	95% CI	p-value
1–3 months	-2.65	-5.81 to 0.51	0.1182
3–6 months	-3.83	-7.40 to -0.26	0.0331
6–12 months	-7.30	-10.92 to -3.68	0.0001

Figure 1 illustrates the progressive divergence in improvement scores between the two groups across the follow-up period.

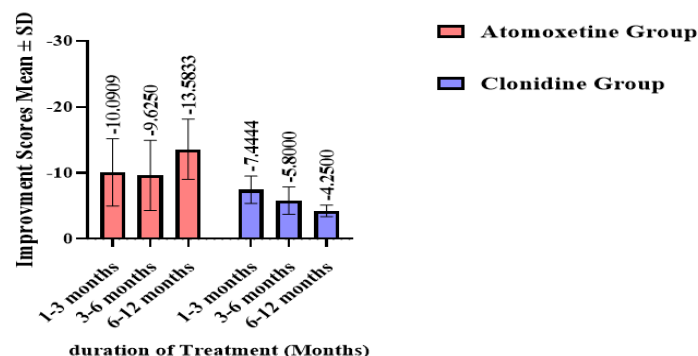


Figure 1: Clinical improvement over time: atomoxetine vs. clonidine

Error bars represent standard deviation. The between-group difference became significant from 3–6 months onward and widened at 6–12 months ($***p < 0.001$)

Safety outcomes

Adverse effects were recorded in both groups, but their profiles differed markedly (Table 4). Clonidine was associated with significantly higher frequencies of sedation/dizziness (70.9% vs. 9.6%, $p < 0.0001$), irritability (45% vs. 12.9%, $p < 0.0001$), tiredness/fatigue (61.2% vs. 16.4%, $p < 0.0001$) and hypotension (12.9% vs. 0%, $p = 0.015$). Atomoxetine was associated with

significantly higher frequencies of appetite loss (35% vs. 6.5%, $p = 0.0015$), nausea/vomiting (29% vs. 9.6%, $p = 0.011$) and restlessness (41.9% vs. 9.6%, $p = 0.0098$). After applying the Benjamini-Hochberg correction for multiple comparisons, all the above-mentioned differences remained statistically significant at a false discovery rate of 5%.

Table 4: Adverse effects – comparison between groups

Adverse effect	Clonidine (n=31) n (%)	Atomoxetine (n=31) n (%)	p-value*
Headache	6 (19.4)	3 (9.7)	0.082
Hypotension	4 (12.9)	0 (0)	0.015
Constipation	5 (16.1)	2 (6.5)	0.424
Sedation/dizziness	22 (71.0)	3 (9.7)	<0.001
Irritability	14 (45.2)	4 (12.9)	<0.001
Tiredness/fatigue	19 (61.3)	5 (16.1)	<0.001
Restlessness	3 (9.7)	13 (41.9)	0.01
Nausea/vomiting	3 (9.7)	9 (29.0)	0.011
Appetite loss	2 (6.5)	11 (35.5)	0.002
Dry mouth	5 (16.1)	2 (6.5)	0.255

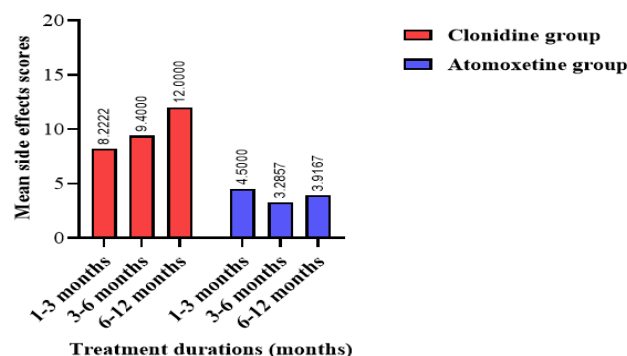
*Chi-square test; p-values <0.05 after Benjamini-Hochberg correction are shown in bold

When adverse effect scores were analysed over time (Table 5 and Figure 2), the clonidine group showed a progressive increase in adverse effect burden, with significant differences between 1–3 months and 6–12 months (mean difference –4.78, $p = 0.0161$) and between 3–6 months and 6–12 months (mean difference –3.62, $p =$

0.0354). In contrast, the atomoxetine group had stable adverse effect scores across all time points (all $p > 0.45$), and the overall adverse effect burden was consistently lower in the atomoxetine group at all follow-up intervals ($p < 0.05$ for between-group comparisons at each time point).

Table 5: Repeated-measures ANOVA for adverse effect scores over time

Source of variation	F-value	p-value	Partial η^2
Treatment group	21.63	<0.0001	0.27
Time	7.12	0.0013	0.11
Group $\times$ Time interaction	3.67	0.0341	0.06

**Figure 2: Adverse effect scores over time: atomoxetine vs. clonidine**

Between-group differences were significant at all follow-up time points (* $p < 0.05$; ** $p < 0.01$)

Discussion

This retrospective observational cohort study compared atomoxetine and clonidine, each combined with a structured behavioural intervention, in children with ASD and comorbid ADHD. The key findings are threefold. First, atomoxetine was associated with significantly greater clinical improvement, which became apparent after three months of treatment and widened over the 12-month follow-up. Second, clonidine produced an initial but limited benefit that plateaued early. Third, the safety profile of atomoxetine was superior, with fewer sedative and cardiovascular adverse effects and a stable burden over time, whereas clonidine showed a progressive increase in adverse effects.

Comparison with existing literature

The superior efficacy of atomoxetine over clonidine observed in our study aligns with findings from previous smaller studies. Eslamzadeh et al. reported that atomoxetine significantly reduced ADHD symptoms in children with ASD in a placebo-controlled trial (8). Similarly, Alsayouf et al. found that atomoxetine improved attention and hyperactivity in young children with ASD-ADHD (13). However, direct comparisons with clonidine have been lacking. Our results extend the literature by providing head-to-head comparative data over 12 months. The early plateau of clonidine's effect is consistent with the pharmacodynamics of

alpha-2 agonists, which can lead to receptor desensitisation with prolonged use (14). In contrast, atomoxetine's delayed but progressive effect reflects its mechanism of gradually increasing noradrenergic tone in prefrontal circuits involved in attention and executive function (15).

The higher incidence of sedation, fatigue and hypotension with clonidine is well documented (9, 14) and limits its long-term tolerability. Atomoxetine's main side effects, appetite suppression and mild gastrointestinal symptoms, are generally manageable and do not typically worsen over time (16).

Strengths and limitations

Strengths of this study include the relatively large sample for this population, the 12-month follow-up, the use of standardised assessment instruments and the systematic documentation of adverse effects.

However, several important limitations must be acknowledged. First, the study was observational and non-randomised; treatment allocation was based on clinical judgment, which may have introduced selection bias. Second, baseline imbalances in weight and age at diagnosis were present, and although sensitivity analyses suggested they did not alter the main conclusions, residual confounding cannot be excluded. Third, the single-centre design limits generalisability to other settings and populations. Fourth, outcome assessment relied partly on caregiver reports, which are subject to recall and reporting bias. Fifth, adherence to medications and behavioural interventions was not objectively measured. Sixth, the absence of a placebo arm means we cannot rule out natural symptom fluctuations or placebo effects. Seventh, the sample size calculation was based on a simpler design (independent groups) rather than the repeated-measures design actually used, which may have underpowered the detection of smaller interaction effects. Finally, multiple statistical comparisons were performed without full adjustment for multiplicity in the primary efficacy analysis, although we applied false discovery rate correction to safety outcomes.

Future directions

To confirm the apparent superiority of atomoxetine, a multicentre, double-blind, randomised controlled trial with an active comparator and a placebo arm is urgently needed. Such a trial should include objective measures of adherence (e.g., electronic pill monitors) and blinded assessors. Additionally, future studies should explore whether specific clinical or genetic subgroups (e.g., based on baseline noradrenergic function or the presence

of sleep disturbances) derive differential benefit from either medication.

From a global health perspective, research on ASD-ADHD pharmacotherapy remains concentrated in high-income countries. As noted in a recent review from the Global South, there is a pressing need for context-specific evidence on the effectiveness and tolerability of non-stimulant medications in low-resource settings (17). Similarly, the Babcock University Medical Journal has called for more rigorous comparative effectiveness research in child neurodevelopmental disorders (18). The present study represents a step toward addressing this gap, but far more work is required.

Conclusion

In this observational cohort of children with ASD and comorbid ADHD, atomoxetine combined with a behavioural intervention was associated with greater and more sustained clinical improvement and a more favourable safety profile than clonidine over 12 months. Clonidine produced early but non-progressive benefits and was limited by a high burden of sedative and cardiovascular adverse effects. Given the non-randomised design and residual confounding, these findings should be considered hypothesis-generating rather than definitive. Atomoxetine may be a reasonable long-term non-stimulant option for this population, but confirmation from randomised controlled trials is essential before any claim of superiority can be made.

List of Abbreviations

ADHD: Attention-Deficit/Hyperactivity Disorder
ANOVA: Analysis of Variance
ASD: Autism Spectrum Disorder
DSM-5: Diagnostic and Statistical Manual of Mental Disorders, 5th Edition
SD: Standard Deviation

Declarations

Ethics Approval and Consent to Participate

The study was approved by the Medical Ethics Committee, College of Medicine, University of Kufa, on 23 September 2025 (reference no. MEC-223). All participating caregivers provided written informed consent, and children provided verbal assent where developmentally appropriate.

Consent for Publication

All authors gave consent for the publication of this work under the Creative Commons Attribution Non-Commercial 4.0 license.

Data Availability

The datasets generated and analysed during the current study are available from the corresponding author upon reasonable request.

Competing Interests

The authors declare no competing interests.

Funding Sources

This study received no external funding.

Author Contributions

AMM worked on the conceptualisation, methodology, investigation, data curation, formal analysis, writing the original draft, writing the review & editing and visualisation.

SDH worked on the conceptualisation, methodology, validation, formal analysis, writing the review & editing, supervision and project administration.

Acknowledgements

The authors thank the staff of the Imam Al-Hussein Institute for Children with Autism in Najaf, Iraq, for their assistance with data collection and logistical support. We are also grateful to the children and their families for their participation.

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