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Prevalence and Clinical Impact of Attention-Deficit/Hyperactivity Disorder in inpatients with a primary diagnosis of an affective disorder: A Retrospective Study from a Specialized Inpatient Clinic

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Abstract

Background

It is well documented that attention deficit hyperactivity disorder (ADHD) and depression frequently co-occur. The objective of this study was to ascertain the prevalence of ADHD among patients admitted to a ward specializing in affective disorders.

Methods

Upon admission, each inpatient was required to complete an admission examination, which included the Adult ADHD Self-Report Scale (ASRS-v1.1). Those who met cut-off scores underwent a comprehensive ADHD assessment. Diagnostic data were collected encompassing sociodemographic, medical and psychiatric histories, and a psychopathological examination.

Results

58 patients (41.1%) screened positive on the ASRS v1.1 Part A, and 15 (10.6%) met full diagnostic criteria following detailed assessment and sufficient remission of depressive symptoms. Among the 83 ASRS-negative patients, 18 (12.7%) were referred for further evaluation due to anamnestic indicators, resulting in 13 additional clinically confirmed cases (9.2%). The resulting ADHD prevalence was 10.6% based on the structured protocol and 19.9% when clinically judged cases were included.

Limitations

The findings are limited by the small and specialized inpatient sample, potentially restricting generalizability. ADHD diagnoses were established during depressive episodes, so mood-related diagnostic bias cannot be fully excluded.

Conclusions

Considering comorbid ADHD in depression is of particular importance. ADHD screening instruments are valuable for identifying ADHD in depressed patients. Furthermore, the results of our study indicate that a rapid onset of depression, as measured by the ODI, may signal hidden ADHD. Recognizing comorbid ADHD has significant clinical implications, influencing both diagnosis and treatment, which can be a game changer in patient outcomes.

Key words
ADHD, major depression, screening, prevalence, onset of depression

1. Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a prevalent neurodevelopmental disorder beginning in childhood and characterized by persistent inattention, impulsivity and hyperactivity (1). Meta-analytic data show that up to 60 % of affected individuals continue to exhibit clinically relevant symptoms into adulthood (2), with prevalence estimates ranging from 2.8 % (1), to 4.4 % (3) in the general adult population.

Adult ADHD is associated with substantial impairments in quality of life, including poorer social functioning and academic and occupational performance (4). Moreover, ADHD frequently co-occurs with other psychiatric disorders, particularly anxiety, personality and substance use disorders, as well as affective disorders (5).

The considerable symptomatic overlap between ADHD and affective disorders complicates the diagnostic process; depressive symptoms can mask ADHD features and contribute to misdiagnosis or delayed recognition (6, 7).

Epidemiological and clinical studies indicate that up to 57 % of individuals with ADHD experience a comorbid affective disorder during their lifetime (8) and conversely, ADHD is markedly more common among patients with affective disorders than in the general population (9).

ADHD prevalence appears to be particularly high in specialist psychiatric units for affective disorders. A recent study reports that up to 32% of patients in a day clinic for affective and anxiety disorders meet ADHD criteria (10). Similar comorbidity rates have been reported in other clinical samples - ranging from about 19% to 33% (11).

Beyond their clinical overlap, ADHD and affective disorders share neurobiological substrates, including dysfunction of fronto-limbic networks and alterations in dopaminergic and noradrenergic systems, which may underpin the impulsivity, emotional dysregulation and affective instability observed in both disorders (12, 13).

Recognition of ADHD in adults with affective disorders is clinically crucial. Untreated ADHD is associated with more severe and prolonged depressive episodes, poorer treatment response, greater risk of suicidality and higher rates of substance use disorders (14–16). Despite this, diagnosis of ADHD in adults with affective disorders is frequently delayed; one study reported a mean lag of 12.5 years between first psychiatric contact and correct diagnosis (17).

Early identification of ADHD in specialist settings may therefore be essential to optimize treatment planning, including pharmacological strategies, and to improve clinical outcomes (18–22).

The present study sought to determine the prevalence of ADHD among inpatients treated in a specialized affective disorders unit. We hypothesised that the ADHD prevalence would exceed previously reported community-based rates. In exploratory analyses, we further examined sociodemographic and clinical characteristics, comorbid psychiatric disorders, severity and course of depressive symptoms in patients with and without ADHD.

2. Methods

2.1 Sample

This study employed a cross-sectional, monocentric design with data retrospectively extracted from electronic inpatient medical records of a psychiatric ward specialising in affective disorders at the University Hospital Leipzig. All inpatients admitted between January 2023 and December 2023 were routinely invited to complete an initial diagnostic screening procedure at the beginning of treatment.

Inclusion criteria were age ≥18 years, female, male or diverse gender identity, and a primary diagnosis of an affective disorder according to ICD-10 criteria (F31–F33). A total of 119 patients met these criteria and were included in the analyses (mean age = 45,5 ± 18,7years; 63,9 % women). Detailed demographic characteristics of the sample are presented in Table 1. The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the University of Leipzig (040/24-ek).

2.2 Measures

2.2.1 Admission examination

As part of standard clinical routine, experienced clinicians collected diagnostic information, including sociodemographic characteristics and medical and psychiatric histories. In addition, a comprehensive psychopathological examination was conducted.

Depressive symptomatology was assessed using both self-report and clinician-rated instruments. Patients completed the Beck Depression Inventory–II (BDI-II), a 21-item questionnaire covering depressive symptoms over the past two weeks with total scores ranging from 0 to 63 (23). Clinicians rated depressive symptoms with the 17-item Hamilton Depression Rating Scale (HAM-D) (24). Symptom severity was documented at admission, after 14 days, and at discharge. In addition, the Onset of Depression Inventory (ODI) was used to assess the speed of onset of depressive episodes, as rapid onset has been discussed as a potential marker of hidden bipolarity (25,26).

ADHD symptoms were screened with the German version of the Adult ADHD Self-Report Scale (ASRS v1.1, Part A) (27). Individuals who screened positive (≥4 endorsed items) subsequently completed the full 18-item ASRS v1.1, which assesses all DSM-IV ADHD symptoms. The ASRS is a well-established tool with adequate psychometric properties (25, 26).

2.2.2 ADHD diagnosis

All patients completed the ASRS v1.1 Part A at admission. If ≥4 items were endorsed, the full 18-item **ASRS v1.1** was subsequently administered. A cut-off score of ≥9 on the full version was applied to indicate probable ADHD (27). Comprehensive diagnostic assessment was initiated only when depressive symptoms had sufficiently improved to allow valid evaluation, defined as BDI-II < 20.

The Wender Utah Rating Scale (WURS-k) was used as a retrospective self-report instrument for childhood ADHD symptoms. The WURS-k is widely applied in adult ADHD assessment and has demonstrated good reliability and discriminant validity (27). The Conners' Adult ADHD Rating Scale (CAARS) was employed to capture current ADHD symptom domains in adulthood; it is a well-validated measure with high internal consistency and good convergent validity (28). In addition, the Wender-Reimherr Interview (WRI) was conducted as a structured clinical interview to evaluate persistence and functional impact of ADHD symptoms; it has been shown to have high reliability and validity in clinical settings (29).

Where available, elementary school report cards (ages 6–8 years) were systematically reviewed. Comments on attention span, concentration, impulsivity, work and social behavior (e.g., “easily distracted”, “restless behavior”) were extracted and integrated into the diagnostic evaluation. Furthermore, in cases where additional information was required, collateral reports from close relatives or other significant informants were obtained to provide external perspectives on ADHD symptoms across the lifespan.

2.2.4. Psychiatric comorbidities

To establish comorbid psychiatric diagnoses, the Structured Clinical Interview for DSM-5, Clinical Version (SCID-5-CV) was administered by trained clinicians (30). For personality pathology, patients first completed the SCID-5 Screening Personality Questionnaire (SCID-5-SPQ), followed by the Structured Clinical Interview for DSM-5 Personality Disorders (SCID-5-PD) interview when indicated (31). These instruments are internationally established and provide reliable and valid diagnostic assessments.

As a dimensional measure of personality traits, the Personality Style and Disorder Inventory (PSSI) was administered, comprising 140 items across 14 scales that reflect personality styles related to DSM-IV/ICD-10 personality disorders (32).

Substance use was screened using the Alcohol Use Disorders Identification Test (AUDIT) (33) and the Drug Use Disorders Identification Test (DUDIT) (34). Both are brief, internationally validated self-report instruments widely used in clinical settings.

Finally, the Symptom Checklist-90 (SCL-90) was employed to assess current subjective symptom burden across multiple domains of psychopathology (35).

Please insert Fig.1 here

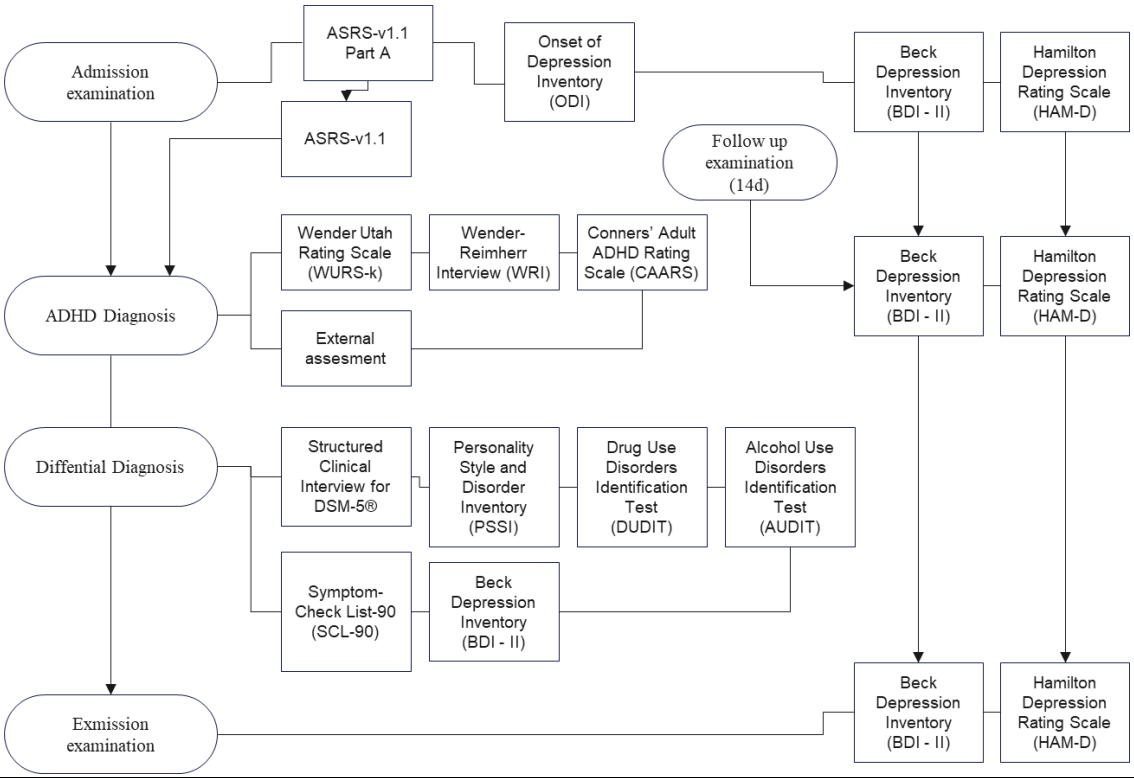


Figure 1. Diagnostic and assessment flow chart: Initial screening included the ASRS-v1.1 Part A, ODI, BDI-II, and HAM-D. Positive ADHD screens were followed by extended assessments (ASRS-v1.1, WURS-k, WRI, CAARS, external ratings).

Differential diagnostics involved the SCID-5, PSSI, SCL-90, DUDIT, and AUDIT. Depressive symptoms (BDI-II, HAM-D) were reassessed after 14 days and at discharge**.**

2.3 Statistic analysis

Analyses were conducted in IBM SPSS Statistics 29.0 (IBM Corp., Armonk, NY, USA). Patients were stratified into two groups, major unipolar depression (MDD) without (MDD-ADHD) and with comorbid ADHD (MDD+ADHD); only unipolar depression cases were included in further analyses to reduce heterogeneity.

Descriptive statistics are reported as means ± Standard deviation (m±SD), medians (Mdn) with interquartile ranges (IQR), or *n* (%). Group comparisons used Pearson's χ^2 tests or Mann–Whitney U tests; Effect sizes are reported as Cramér's V, Cohen's d, or *r*. Two-tailed α =0.05 was used; Bonferroni corrections were applied for multiple testing.

As the groups differed significantly in age and years of education (p=0.031 and p=0.006), both were included as covariates in adjusted models. Binary outcomes (e.g., alcohol, nicotine, drug use) were analysed with logistic regression, ordinal outcomes with ordinal regression, and nominal variables with multinomial logistic regression. Count outcomes (e.g., suicide attempts, depressive episodes) were examined with Poisson or negative binomial regression depending on overdispersion.

Depression severity (BDI-II, HAM-D) at admission, 14 days, and discharge was compared using Mann–Whitney U tests and ANCOVA adjusted for age and education. Longitudinal changes were analysed with repeated-measures ANCOVA (time × group).

Medication classes were analysed using generalized linear mixed models (binomial, logit link, random intercept per subject), adjusted for age and education, with Bonferroni-corrected p-values across classes.

Results are reported as adjusted odds ratios (OR), rate ratios (RR), or unstandardized coefficients (B) with 95% confidence intervals. Models that failed to converge due to sparse data or quasi-complete separation are explicitly noted as non-interpretable.

3. Results

3.1 Prevalence of ADHD in our sample

Figure 2: Flow chart of ADHD assessment

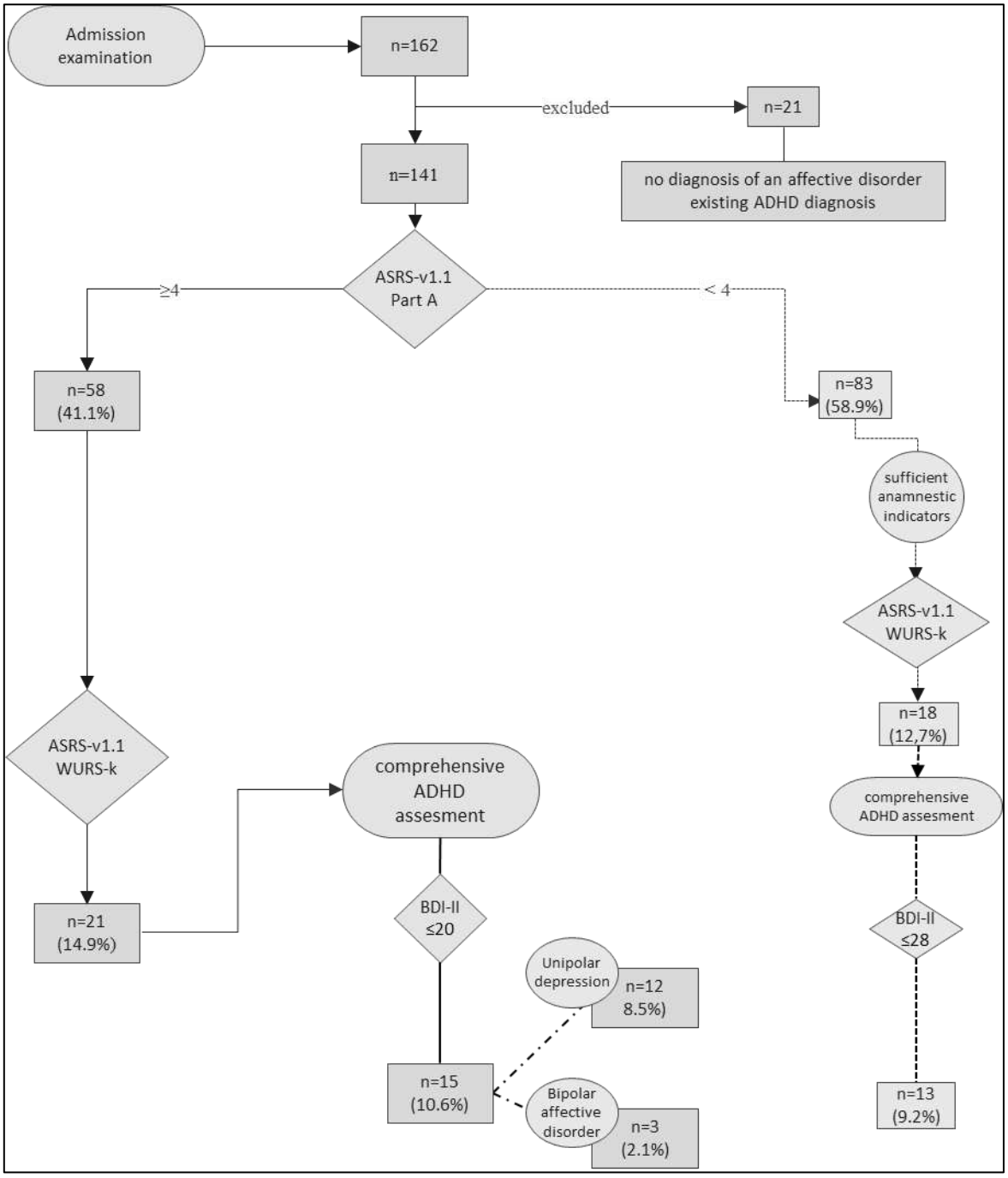


Figure 2: Patients entered either the Criterion-based pathway (ASRS Part A ≥4, ASRS-v1.1 ≥9, WURS-k ≥30, BDI-II ≤20), resulting in 15 confirmed ADHD diagnoses (including 3 with comorbid bipolar disorder), or the anamnestic pathway. 13 patients received an ADHD diagnosis, although cut-off values of screening questionnaires were not consistently exceeded; these cases are therefore reported separately.

Of 162 inpatients screened, 21 were excluded due to either the absence of an affective disorder diagnosis or a pre-existing ADHD diagnosis, leaving 141 patients for analysis.

Criterion-based pathway

At admission, 58 patients (41.1%) screened positive on the ASRS v1.1 Part A (≥4 items). Of these, 21 (14.9%) fulfilled the criteria on both the ASRS v1.1 full version (≥9 items) and the WURS-k (≥30 points), and therefore proceeded to comprehensive ADHD diagnostics. After additionally meeting the requirement of sufficiently reduced depressive symptom severity (BDI-II ≤20), 15 patients (10.6%) received a confirmed ADHD diagnosis, three of whom also presented with comorbid bipolar affective disorder.

Anamnestic pathway

Among the 83 patients who screened negative on the ASRS Part A (58.9%), 18 (12.7%) presented with sufficient anamnestic indicators of ADHD and were referred for further evaluation. In this subgroup, three patients had BDI-II values between 21 and 28, while the remainder showed scores below 20. ADHD diagnoses in this pathway were not based on anamnestic impressions alone but followed a comprehensive assessment including the ASRS full version, the WURS-k, structured clinical interviews, and collateral history. However, in several of these cases the psychometric screening instruments (ASRS-v1.1, WURS-k) did not consistently exceed the established cut-offs. Diagnoses (n = 13, 9.2%) were therefore made on the basis of an integrative clinical judgment combining questionnaire data and anamnestic information. These cases are reported separately to maintain methodological transparency.

Prevalence

Based on the protocol-adherent pathway, the prevalence of ADHD in this inpatient cohort was 10.6%. When the anamnestic pathway is also considered, the prevalence estimate rises to 19.9%. This suggests that the true prevalence in this clinical population may be higher than indicated by the conservative, cut-off–based approach.

3.2 Sociodemographic characteristics

Table 1: Sociodemographic characteristics

	Total sample n=119	MDD+ADHD n=12	MDD-ADHD n=107	*p*-value	effect size
female (n; %)	76; 63.9	6; 50.0	70; 65.4	0.518	
Age in years (m ± SD)	45.5 ± 18.7	33.8 ± 10.8	46.8 ± 19.0	*0.031*	0.70
BMI (m ± SD)	26.3 ± 5.8	25.9 ± 4.8	26.4 ± 5.9	0.744	0.08
Marital status					
single (n; %)	45 37.8	7; 58.3	38 35.5	0.459	0.16
In partnership (n; %)	65; 54.6	5; 41.7	60; 56.1		
Divorced (n; %)	2; 1.7	0	2; 1.9		
Widowed (n; %)	7; 5.9	0	7; 6.5		
Educational level					
None (n; %)	1; 0.8	0; 0.0	1; 0.9	0.294	0.19
Lower secondary (n; %)	50; 42.0	2; 16.7	48; 44.9		
High school diploma (n; %)	65; 54.6	10; 83.3	55; 51.4		
Special needs school (n; %)	1; 0.8	0	1; 0.9		
Other (n; %)	2; 1.7	0	2; 1.9		
Years of education (m ± SD)	15.89 ± 3.18	18.50 ± 3.56	15.58 ± 3.01	*0.005*	0.07
Vocational training					
None (n; %)	19; 16.0	1; 8.3	18; 16.8	0.737	0.12
In training (n; %)	7; 5.9	0; 0.0	7; 6.5		
Completed (n; %)	61; 51.3	7; 58.3	54; 50.5		
University degree (n; %)	31; 26.1	4; 33.3	27; 25.2		
Unknown (n; %)	1; 0.8	0; 0.0	1; 0.9		
Employment status					
Employed (n; %)	63; 52.9	9; 75.0	54; 50.5	0.244	0.19
Unemployed (n; %)	15; 12.6	2; 16.7	13; 12.1		
Retired (n; %)	35; 29.4	1; 8.3	34; 31.8		
Other (n; %)	6; 5.0	0; 0.0	6; 5.6		
Criminal record					
No (n; %)	26; 21.8	1; 8.3	25; 23.4	0.129	0.44
Yes (n; %)	11; 9.2	4; 33.3	7; 6.5		

Unknown (n; %)	82; 68.9	7; 58.3	75; 70.1	

Table 1: Values are presented as mean (m) ± standard deviation (SD) for continuous variables and as n (%) for categorical variables. Group comparisons were conducted between participants with major depressive disorder (MDD) and comorbid attention-deficit/hyperactivity disorder (ADHD) versus those with MDD without ADHD. For continuous variables, differences were tested using the Mann–Whitney *U* test (*p*-values reported); for categorical variables, Pearson's χ^2 test was used (*p*-values reported). Effect sizes are reported as Cohen's *d* for continuous variables, Cramér's *V* for categorical variables, and partial η^2 for analyses of covariance. All tests were two-tailed, and statistical significance was set at $p < 0.05$. Adjusted analyses were performed for years of education (age as a covariate) and for criminal record (age and years of education as covariates).

Table 1 outlines the characteristics of the entire sample, which includes 119 adults with a mean age of 45.5±18.7 years, 63.9% of whom were women. Patients with MDD+ADHD were younger than those without ADHD (33.8±10.8 vs. 46.8±19.0 years; *p*=0.031; *d*=0.70).

Marital status distributions were similar (single: 58.3% vs. 35.5%; in partnership: 41.7% vs. 56.1%; divorced: 0% vs. 1.9%; widowed: 0% vs. 6.5%; overall *p*=0.459; *V*=0.16). Educational level did not differ across categories (none: 0.0% vs. 0.9%; lower secondary: 16.7% vs. 44.9%; high-school diploma: 83.3% vs. 51.4%; special-needs school: 0% vs. 0.9%; other: 0% vs. 1.9%; *p*=0.294; *V*=0.19). By contrast, years of education were higher in the MDD+ADHD group (18.50 ± 3.56 vs. 15.58 ± 3.01; *p*=0.006; *d*=0.95), and the between-group difference remained significant after age adjustment (*p*=0.005; partial $\eta^2 = 0.067$) in the limited sample size of this study.

Distributions for vocational training were comparable (none: 8.3% vs. 16.8%; in training: 0.0% vs. 6.5%; completed training: 58.3% vs. 50.5%; university degree: 33.3% vs. 25.2%; unknown: 0.0% vs. 0.9%; *p*=0.737; *V*=0.12). Employment status also did not differ (*p*=0.244; *V*=0.19).

For criminal record, 33.3% of the MDD+ADHD group and 6.5% of the MDD–ADHD group had a recorded offence; the unadjusted comparison yielded p = 0.021 (V = 0.435). In the adjusted logistic regression including age and years of education, the association was not statistically significant (*p*=0.129).

3.3 Relationship between ADHD and clinical features of depression

Table 2 Clinical Characteristics

	Total sample n=119	MDD-ADHD n=107	MDD+ADHD n=12	*p*-value	effect size
Therapy resistent depression					
No (n;%)	65; 54.6	59; 55.1	6; 50.0	0.641	0.03
Yes (n;%)	54; 45.4	48; 44.9	6; 50.0		
Onset of depression Inventory					
≥ 7 days (n;%)	90; 75.6	87; 90.6	3; 25.0	*<0.001*	1.73
< 7 days (n;%)	16; 13.4	9; 9.4	7;58.3		
Suicidality lifetime					
No (n;%)	89; 74.8	83; 77.6	6; 50.0	*0.017*	0.91
Yes (n;%)	30; 25.2	24; 22.4	6; 50.0		
Number of lifetime suicide Attempts (Mdn; IQR)	0;1-0	0; 0–0	0.5; 0–3	*0.005*	0.74
MDD-Course					
Partially remitted (n;%)	30; 25.6	26; 24.8	4; 33.3	0.473	0.18
Remitted (n;%)	33; 28.2	32; 30.5	1; 8.3	0.736	0.16
Persistent (n;%)	16; 13.7	12; 11.4	4; 33.3	0.031	1.15
Unknown (n;%)	38; 32.5	35; 33.3	3; 25.0		
Atypical symptoms					
None	68; 57.1	64; 59.8	4; 33.3		
Hypersomnia	13; 10.9	12; 11.2	1; 8.3	0.416	0.48

Hyperphagia Hypersomnia + Hyperphagia Unknown	11; 9.2 6; 5.0 21; 17.6	7; 6.5 4; 3.7 20; 18.7	4; 33.3 2; 16.7 1; 8.3	*0.014* *0.029*	1.30 1.59
Length of Inpatient Stay in days (m±SD)	37.9 ± 24.2	39.6 ± 25.8	38.4 ± 27.1	0.466	0.21
Age at Onset of First diagnosed Depressive Episode (m±SD)	36.0 ± 18.7	36.6 ± 19.6	29.9 ± 5.8	*0.011*	0.49
Number of lifetime depressive episodes (Mdn; IQR)	3; 4-2	3; 2–5	2; 1–3	0.313	0.23

Table 2: Values are presented as mean (m) ± standard deviation (SD) for continuous variables), medians (Mdn) with interquartile ranges (IQR) and as n (%) for categorical variables All statistical analyses were adjusted for age and years of education. Logistic regression models were employed for binary categorical variables, multinomial logistic regression for nominal outcomes with more than two categories, Poisson or negative binomial regression for count data, and analysis of covariance (ANCOVA) for continuous variables. Reported *p*-values reflect these adjusted regression models controlling for age and education.
Effect sizes (Cohen's d) were derived from odds ratios using the transformation $d = \ln(OR) \times \sqrt{3} / \pi$. All tests were two-tailed, and statistical significance was set at $p < 0.05$.
Therapy resistent depression: ≥2 antidepressant failures
Partially remitted depression: self-reported clear improvement compared to the acute depressive phase but reported persisting residual symptoms; Remitted depression: reported near or complete absence of depressive symptoms; Persistent depression: self-reported continuous depressive symptoms without sustained remission for at least two years.

The mean duration of inpatient stay did not differ significantly between groups (MDD-ADHD: 39.6±25.8 days; MDD+ADHD: 38.4 ± 27.1 days; *p*=0.466, B = 8.7, 95% CI [−0.79–0.37], d=0.21).

However, the age at onset of the first depressive episode was significantly lower in patients with MDD+ADHD (29.9 ± 5.8 years) compared to those with MDD-ADHD (36.6 ± 19.6 years; *p*=0.011, B = −8.80, 95% CI [−15.54, −2.05], d=0.49). The number of lifetime depressive episodes did not differ significantly between groups (Mdn=3, IQR=2–5 in MDD-ADHD vs. Mdn=2, IQR=1–3 in MDD+ADHD; *p* =0.313, B = 0.41, 95% CI [0.68–3.33], *d*=0.23). There were no significant group differences in partially remitted (*p*=0.473) or remitted (*p* =0.736) courses of the most recent depressive episode. However, a persistent course of the last episode was more frequent among patients with MDD+ADHD (33.3%) compared to those with MDD-ADHD (11.4%; *p*=0.031, OR = 0.13, 95% CI [0.02–0.83], *d*=1.15). A significantly higher proportion of participants with MDD+ADHD reported a rapid depressive onset within less than seven days (58.3%) compared to those without ADHD (9.4%; *p*<0.001, OR=23.31, 95% CI [4.20–129.35], *d*=1.736). Therapy-resistant depression (≥2 antidepressant failures) was present in 54 patients (45.4%). The prevalence did not differ significantly between groups (MDD-ADHD: 44.9%; MDD+ADHD: 50.0%; *p*=0.641, OR = 1.37, 95% CI [0.65–5.14], *d*=0.031). Atypical features were more common in the MDD+ADHD subgroup. Hypersomnia alone was observed in 8.3% (vs. 11.2% in MDD-ADHD; *p* =0.416, OR=2.40, 95% CI [0.23–35.7], *d*=0.48), whereas hyperphagia occurred in 33.3% (vs. 6.5%; *p*=0.014, OR=10.64, 95% CI [1.61–71.43], *d*=1.30). Combined hypersomnia and hyperphagia were present in 16.7% of patients with MDD+ADHD compared to 3.7% in the MDD-ADHD group (*p*=0.029, OR = 17.86, 95% CI [1.35–250.00], *d*=1.59).

Lifetime suicidality was more frequent in the MDD+ADHD group (50.0%) than in the MDD-ADHD group (22.4%; *p*=0.017, OR = 5.26, 95% CI [1.34–20.63], *d*=0.910). The number of lifetime suicide attempts also differed significantly between groups (Mdn=0, IQR=0–0 in MDD-ADHD vs. 0.5, IQR = 0–3 in MDD+ADHD; *p*=0.005, RR = 3.87, 95% CI [1.49–9.10], *d*=0.740).

3.4 Pharmacological treatment trials

Table 3 Prescriptions attempts

	Total sample n=119	MDD-ADHD n=107	MDD+ADHD n=12	*p*-value	effect size

Benzodiazepines (n;%)	15; 4.3	15; 4.8 14.0	0; 0.0	1.000	0.28
Bupropion	10; 2.9	6; 1.9	4; 12.1	*<0.001*	0.35
SSRI (n;%)	62; 17.9	56; 17.8	6; 18.2	0.995	0.53
SNRI (n;%)	39; 11.2	36; 11.5	3; 9.1	0.995	0.55
Esketamine (n;%)	9; 2.6	9; 2.9	0; 0.0	1.000	0.28
Antipsychotics (n;%)	105; 30.3	98; 31.2	7; 21.2	*<0.001*	0.47
Lithium (n;%)	18; 5.2	17; 5.4	1; 3.0	*<0.05*	0.62
Mirtazapine (n;%)	48; 13.8	46; 14.6	2; 6.1	0.576	0.34

Table 3: *n* represents the number of prescriptions attempts within each medication class and diagnostic group. Percentages (%) indicate the proportion of prescriptions within the respective group, not the proportion of individuals receiving a given medication. A total of 347 prescription attempts were recorded across 119 participants. Tricyclic antidepressants and antiepileptic agents were not discussed due to their very low prescription frequency in both groups.

Estimates were adjusted for age and years of education at the individual level (0/1 per class per person) using a generalized linear mixed model (binomial logit link) with a random intercept per subject.

p-values were Bonferroni-adjusted for 12 simultaneous tests. The adjusted significance threshold was $\alpha^* = 0.0042$ (Bonferroni correction).

Effect sizes (Cohen's *d*) were derived from odds ratios using the transformation $d = \ln(OR) \times \sqrt{3} / \pi$, indicating the standardized magnitude of group differences for each medication class.

SSRI = selective serotonin reuptake inhibitor; SNRI = serotonin–noradrenaline reuptake inhibitor.

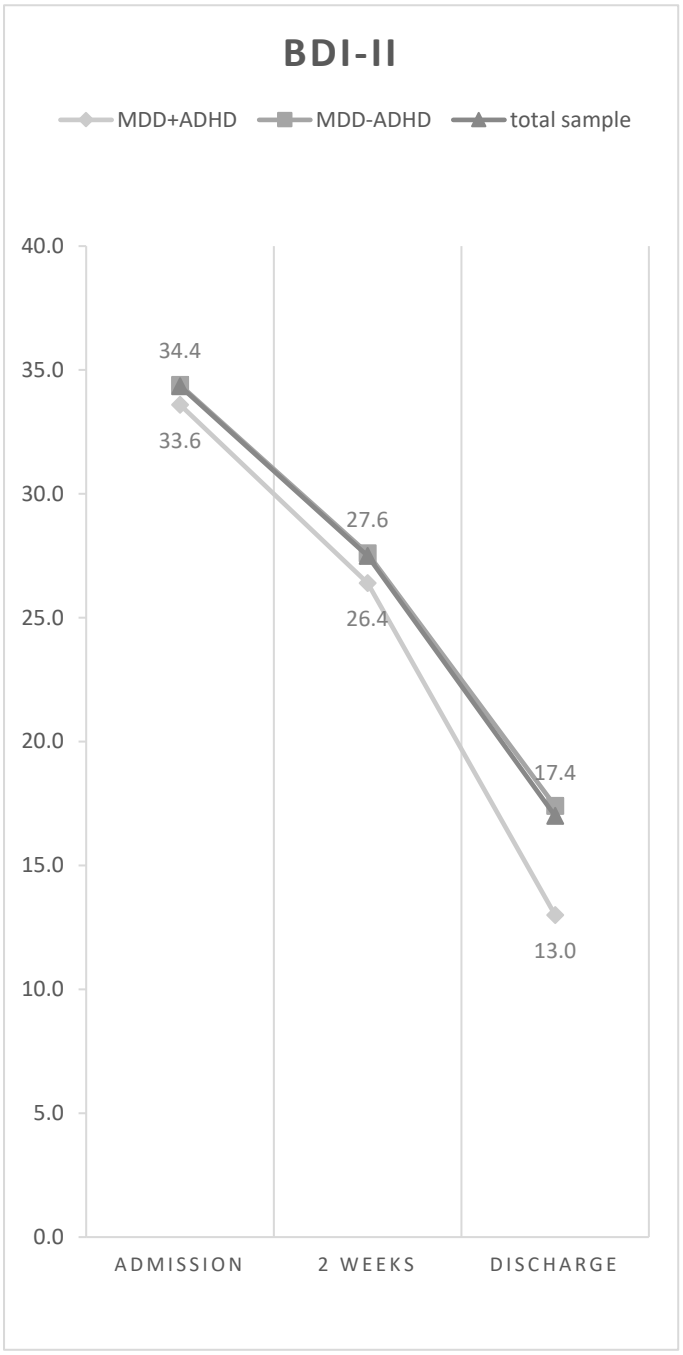
A total of 347 prescription attempts were analyzed in 119 patients. All inpatients with comorbid ADHD received ADHD specific medication, psychostimulants were prescribed in 7 cases (2.0%), Atomoxetine was prescribed in 3 cases (9.1%).

For all other medication classes, prescription frequencies and effect sizes are summarized in Table 3. Bupropion was prescribed more frequently in MDD+ADHD (OR=1.89, 95% CI [1.37–2.61], $p<0.001$, $d=0.35$). Selective serotonin reuptake inhibitors (SSRI) (OR=2.62, 95% CI [1.96–3.49], $p=0.995$, $d=0.53$) and Serotonin–noradrenaline reuptake inhibitors (SNRI) (OR = 2.69, 95% CI [1.89–3.83], $p=0.995$, $d=0.55$) were not significantly associated with the diagnostic subgroup. Antipsychotic prescriptions groups (OR = 2.34, 95% CI [1.69–3.24], $p<0.001$, $d=0.47$) and Lithium (OR = 3.08, 95% CI [1.57–6.03], $p<0.05$, $d=0.62$) differed significantly between the subgroups. Mirtazapine (OR=1.86, 95% CI [1.01–3.44], $p=0.576$, $d=0.34$), Benzodiazepine prescriptions (OR= 1.65, 95% CI [1.05–2.60]; $p=0.360$, d=0.28) or Esketamine (OR = 1.66, 95% CI [0.65–4.27], $p=1.000$, $d=0.28$) did not differ significantly between the groups.

The mean number of pharmacological treatment trials per inpatient was slightly higher in patients with MDD+ADHD (m=3.00, SD=1.04, Mdn=3, IQR=2–4) compared to those with MDD without ADHD (m=2.54, SD=1.04, Mdn =3, IQR=3–4). This difference did not reach statistical significance in the non-parametric group comparison ($\chi^2 = 1.92$, 95% CI [−0.05, 0.29], $p=0.166$). An ordinal logistic regression with the number of treatment trials as the dependent variable showed that comorbid ADHD was associated with a higher likelihood of multiple pharmacological treatment trials ($\beta=-1.50$, SE=0.61, $p=0.014$, 95% CI [−2.69, −0.31]).

3.5 Course of Depressive Symptoms

Table 4 Course of Depressive Symptoms



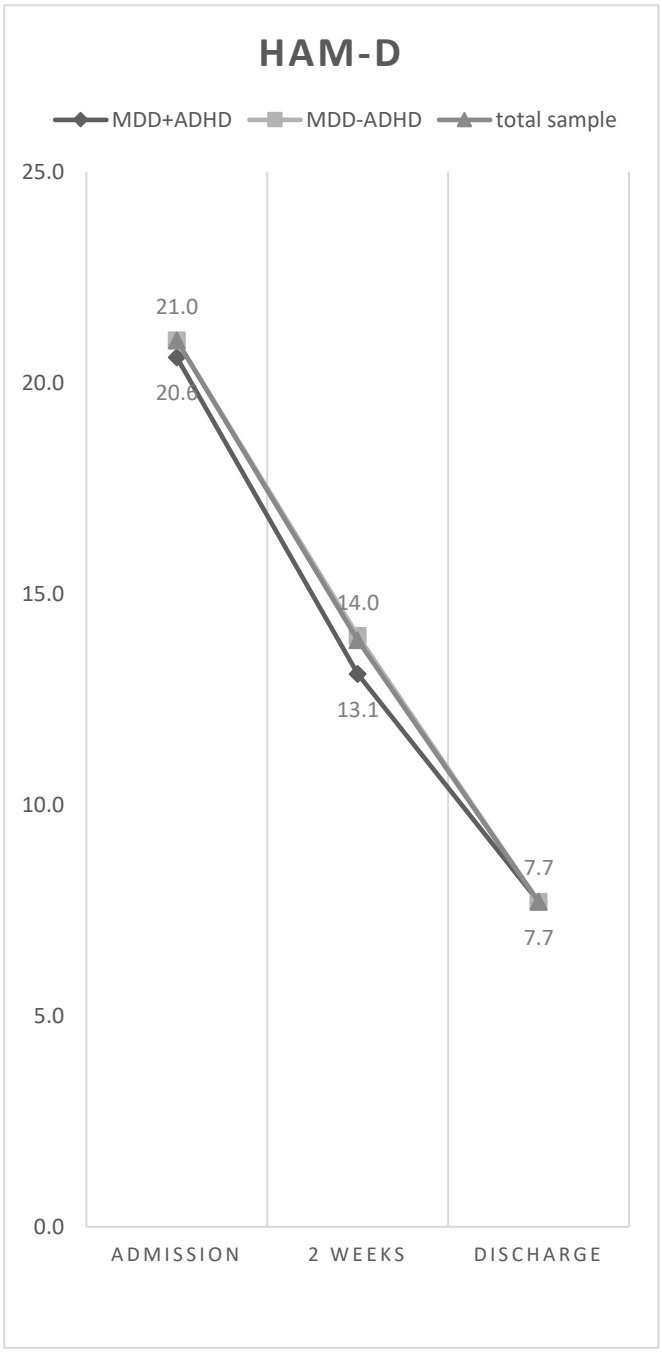


Table 4: Mean BDI-II scores decreased from 34.4 (SD = 10.3) at admission to 27.5 (SD = 11.4) after two weeks and 17.0 (SD = 11.3) at discharge, reflecting marked symptom improvement during inpatient treatment. Patients with and without ADHD showed comparable reductions (MDD–ADHD: 34.4 → 17.4; MDD+ADHD: 33.6 → 13.0).

Mean HAMD-21 scores showed a parallel decline from 20.9 (SD = 5.0) at admission to 13.9 (SD = 5.4) after two weeks and 7.7 (SD = 4.6) at discharge, with no relevant group differences between patients with (20.6 → 7.7) and without ADHD (21.0 → 7.7).

Changes in depressive symptom severity were analyzed using repeated-measures ANCOVAs with time (admission, 2 weeks, discharge) as a within-subject factor, ADHD comorbidity as a between-subject factor, and age and years of education as covariates.

For both measures, no significant between-subject effects were observed for age or years of education (all $p > 0.1$). ADHD comorbidity had no significant main effect on mean depressive symptom levels (BDI-II: $F(1.68) = 1.49$, $p = 0.227$, partial $\eta^2 = 0.021$; HAM-D: $F(1.78) = 0.00$, $p = 0.976$, partial $\eta^2 < 0.001$).

A significant main effect of time was found for both instruments, indicating a reduction in depressive symptom severity over the course of inpatient treatment (BDI: $F(1,68) = 10.21$, $p = 0.002$, partial $\eta^2 = 0.131$; HAMD: $F(1,78) = 8.999$, $p = 0.004$, partial $\eta^2 = 0.103$). The quadratic trends were non-significant for both scales (all $p>0.3$), suggesting a linear decrease in symptom scores.

No significant time interactions were observed with age, years of education, or ADHD comorbidity for either measure (all $p > 0.1$), indicating comparable trajectories of improvement across groups and independence from the covariates.

3.6 Psychiatric comorbidities

The number of newly established comorbid psychiatric diagnoses during inpatient treatment was minimal across all groups. For the total sample, the median number of newly diagnosed comorbid disorders was 0 (IQR = 0–0), with identical values in both subgroups (MDD–ADHD and MDD+ADHD). No relevant group differences were observed.

In addition to the overall number of new diagnoses, specific comorbidities were identified as follows. Substance use disorders were most frequently diagnosed, occurring in 24 patients (20.2% of the total sample), including 21 patients (19.6%) in the MDD–ADHD group and 3 patients (25.0%) in the MDD+ADHD group. Personality disorders were identified in 10 patients (8.4%), all within the MDD–ADHD group. Trauma-related and anxiety disorders were each newly diagnosed in 2 patients (1.7% and 1.9%, respectively), both confined to the MDD–ADHD group. Autism spectrum disorder was newly identified in 3 patients (2.5% of the total sample), including 2 patients (1.9%) in the MDD–ADHD group and 1 patient (8.3%) in the MDD+ADHD group.

3.7 Substance use

Please insert Table 5 here

Table 5 Alcohol Use

	Total sample n=119	MDD-ADHD n=107	MDD+ADHD n=12	*p*-value	Effect size
Alcohol use during the past 12 months					
Yes (n;%)	76; 64.4	65; 61.3	11; 91.7	<0.010	0.92
Frequency of alcohol use in the past 12 months					
1 per month/ less (n;%)	17;22.7	14; 21.9	3;27.3	0.873	0.06
2–4 per month (n;%)	29:38.7	25.39.1	4:36.4		
2-3 per week (n;%)	19; 25.3	15:23.4	4:36.4		
4 – 6 per week (n;%)	7; 9.3	7; 10.9	0; 0.0		
Daily or more (n;%)	3;4.0	3; 4.7	0; 0.0		
Average alcohol consumption					
1–2 drinks (n;%)	36; 50.0	32; 52.5	4; 36.4	0.395	0.33
3-4 drinks (n;%)	25; 34.7	19; 31.1	6; 54.5		
5-6 drinks (n;%)	7; 9.7	6; 9.8	1; 9.1		
7-9 drinks (n;%)	2; 2.8	2; 3.2	0; 0.0		
≥ 10 drinks (n;%)	2; 2.8	2; 3.2	0; 0.0		

Table 5: Values are presented as n (%) for categorical variables All statistical analyses were adjusted for age and years of education. Binary logistic regression was used for alcohol use, and ordinal logistic regressions were applied for frequency and average consumption.
Effect sizes (Cohen's d) were derived from odds ratios using the transformation $d = \ln(OR) \times \sqrt{3} / \pi$.
All tests were two-tailed, and statistical significance was set at $p < 0.05$.

ADHD comorbidity was associated with higher alcohol use (OR = 5.26, 95% CI [0.61, 45.3], p<0.010, d=0.92). No significant associations were found for alcohol use frequency (OR = 1.11, 95% CI [0.32, 3.88], p = 0.873, d=0.06) or average alcohol consumption (OR = 0.55, 95% CI [0.14, 2.16], p=0.395, d=0.33).

Please insert Table 6 here

Table 6 Drug use

	Total sample n=119	MDD-ADHD n=107	MDD+ADHD n=12	*p*-value	Effect size
Drug use during the past 12 months					
Yes (n;%)	30; 25.4	24;22.6	6;50.0	0.132	0.60
Drug type					
Cannabis (n;%)	20; 69.0	15; 65.2	5; 83.3	0.621	0.34
Stimulants (n;%)	5; 17.2	4; 17.4	1; 16.7	0.860	0.12
Drug frequency in past 12 months					
1 per month or less (n;%)	8: 29.6	6; 28.6	2; 33.3	0.241	0.60
2–4 per month (n;%)	8: 29.6	4; 19.0	4; 66.7		
2-3 per week (n;%)	2; 7.4	2; 9.5	0; 0.0		
4 – 6 per week (n;%)	2; 7.4	2; 9.5	0; 0.0		
Daily/more (n;%)	7; 25.9	7; 33.3	0; 0.0		

Table 6: Values are presented as n (%) for categorical variables All statistical analyses were adjusted for age and years of education. Binary logistic regression was used for drug use/type, and ordinal logistic regressions were applied for drug frequency.
Effect sizes (Cohen's d) were derived from odds ratios using the transformation $d = \ln(OR) \times \sqrt{3} / \pi$.
All tests were two-tailed, and statistical significance was set at $p < 0.05$.

No significant group differences were observed in overall drug use during the past 12 months (OR=0.34, 95% CI [0.09, 1.38], p=0.132, d=0.60). Among specific substances, cannabis use was reported by 83.3% of inpatients with comorbid ADHD (OR = 0.54, 95% CI [0.05, 6.41], p=0.621, d=0.34), stimulant use by 16.7% (OR=0.80, 95% CI [0.06, 10.75], p=0.860, d=0.12), and sedative use by 13.0% in the MDD–ADHD group only. Polysubstance use was reported in one case in the MDD-ADHD group.

Regarding the drug frequency use, no significant group difference was found (OR=2.99, 95% CI [0.48, 18.6], p=0.241, d=0.60).

4. Discussion

The present study aimed to identify previously unrecognized ADHD among inpatients with an affective disorder. Using a structured, two-pathway diagnostic approach, 10.6% of patients met criteria for ADHD under a conservative, criterion-based protocol, while inclusion of clinically plausible anamnestic cases increased the estimate to 19.9%, which is notably higher than the reported prevalence in general adult population samples (1, 3).

Several methodological factors probably biased the estimate downward. The reliance on fixed cut-offs on self-report screening instruments (ASRS v1.1, WURS-k) may bias the self-evaluation of ADHD symptoms in acutely depressed patients. The requirement of a BDI-II ≤ 20 before full diagnostic assessment excluded individuals with higher depressive severity, potentially omitting true ADHD cases. This pattern can be observed in the anamnestic pathway, where several patients exhibited clear clinical indicators of lifelong ADHD despite subthreshold psychometric scores. While this complementary pathway helps to compensate for the limitations of cut-off–based screening, it also introduces the risk of overestimating ADHD prevalence through subjective weighting of clinical impressions. These considerations imply that the true prevalence in this setting may be higher than the conservative estimate reported here, precise quantification would require prospective validation.

These findings confirm that ADHD remains underrecognized in adult psychiatric inpatients (36). Existing literature predominantly focuses on investigating depressive episodes in ADHD patients (3, 37), whereas studies examining ADHD in depressive inpatients are comparatively limited (38). While ADHD prevalence exhibits considerable variability depending on the cohort and conducted diagnostic assessment (39), the observed prevalence aligns with recent research findings in clinical populations. A recent meta-analysis reported an average ADHD prevalence of 7% among adult patients with MDD (9). In a more specific population 12.8% of recurrently depressed women exhibited elevated ADHD symptoms, with 3.4% meeting diagnostic criteria for ADHD (40). Our findings are at the higher end of the spectrum reported in recent literature, underscoring the importance of a standardized ADHD assessment in depressive inpatients and suggesting a potentially higher comorbidity rate in specialized inpatient settings.

In contrast to previous studies that have demonstrated lower employment rates, reduced salary levels and increased interpersonal difficulties among adults with ADHD (41–43), our investigation of inpatients did not consistently show significant differences in these domains. However, several distinctions were observed within the ADHD cohort. Patients with comorbid ADHD were significantly younger than those with MDD alone, consistent with the notion that comorbid ADHD may lead to earlier psychiatric referral or hospitalization (44, 45). Patients with MDD+ADHD reported more years of formal education despite similar distributions of educational qualifications. This finding may reflect delayed academic progression, extended training pathways, or the higher educational background of a small number of individuals within the ADHD subgroup. Because years of education and age were moderately correlated, the age-adjusted analysis (partial $\eta^2 = 0.067$) strengthens the robustness of this effect but should still be interpreted cautiously given the unequal group sizes. Sociodemographic distributions in marital status, vocational training, and employment did not differ significantly between groups, suggesting broadly comparable social functioning in this inpatient cohort. The higher prevalence of recorded criminal offences among patients with ADHD (33.3% vs. 6.5%) aligns with epidemiological data linking ADHD to increased impulsivity and risk-taking behavior (46, 47). This association did not remain statistically significant after adjustment for age and education, indicating that these demographic factors likely account for part of the observed difference. The finding should therefore be viewed as exploratory and interpreted in light of the small number of cases.

Consistent with prior findings that depressive symptom traits of adults with ADHD differ in quality, course, and affective tone from those without ADHD (48). The ADHD-linked atypical depression was characterized by an persistent course of the most recent depressive episode, consistent with earlier reports linking ADHD to chronic depression (49). Although in our sample the overall rate of therapy resistance was high in both groups. The number of lifetime depressive episodes and the overall duration of inpatient treatment did not differ between groups, indicating comparable recurrence patterns. Atypical depressive features were more frequent in the ADHD subgroup, particularly hyperphagia and the combination of hyperphagia with hypersomnia. Hypersomnia alone, however, did not reach statistical significance, suggesting that the observed combined effect is likely driven primarily by hyperphagia. This pattern aligns with previous findings that ADHD is often accompanied by atypical mood presentations (50), eating disorders (48) and altered reward sensitivity (51).

Lifetime suicidality and the number of suicide attempts were significantly higher in patients with ADHD, consistent with existing studies linking ADHD to increased suicidal behavior (52, 53).

A key finding of this study was the marked difference in the time of onset of depressive symptoms. A rapid onset of less than 7 days is typically associated with bipolar disorder (26). Bipolar disorder and ADHD share overlapping symptoms such as hyperactivity, distractibility, lack of inhibition, restlessness or racing thoughts (54, 55). In our sample, patients with unipolar depression and comorbid ADHD (58.3%) also showed this rapid onset. A bipolar disorder can be considered unlikely in this context, as all participants underwent structured diagnostic interviews (SCID-I and SCID-II) and cases meeting criteria for bipolar spectrum disorders were explicitly excluded.

ADHD-specific symptoms, such as emotional hyperactivity and affective lability, could play a role in precipitating a rapid-onset of depressive episodes. Emotion regulation has been identified as a mediator of the relationship between ADHD and depressive symptoms in children with ADHD (56). The genetic overlap between ADHD and affective disorders (57, 58) might possibly contribute to further symptom convergence. These findings suggest that the ODI could serve as an additional clinical indicator of underlying ADHD in patients presenting with depression. Subsequent investigations with larger sample sizes should be conducted to verify and potentially expand upon these preliminary findings.

Depressive symptoms decreased substantially over the course of inpatient treatment, as reflected by significant time effects on both self-reported (BDI-II) and clinician-rated (HAM-D) measures. The absence of time × group interactions indicates that patients with and without ADHD benefitted comparably from multimodal inpatient treatment once admitted. This finding suggests that the presence of comorbid ADHD does not necessarily impede the therapeutic response to depression treatment, provided that both disorders are appropriately managed. In this context longitudinal studies have demonstrated that mitigating ADHD symptoms may also prevent later depressive episodes (59). This temporal relationship suggests a causal pathway in which effective management of ADHD symptoms could serve as a prophylactic measure against the development of comorbid depression (59, 60). ADHD could only be diagnosed after sufficient remission of depressive symptoms, as the diagnostic protocol required a BDI-II score of ≤ 20 before comprehensive ADHD assessment. This procedural requirement may also have excluded patients with persistent or severe depression who nonetheless had comorbid ADHD.

Prescription data indicated distinct pharmacological profiles between inpatients with MDD+ADHD and those with MDD alone. All inpatients with ADHD received specific medication, primarily psychostimulants or atomoxetine. In addition, depressive symptom severity showed an adequate reduction over the course of treatment, indicating that the administration of psychostimulants was not associated with a worsening of depressive symptoms Beyond these agents, bupropion was prescribed more frequently in the ADHD subgroup, consistent with clinical practice favoring dopaminergic and noradrenergic agents in individuals with attentional and affective dysregulation (61). Canadian Network for Mood and Anxiety Treatments guidelines for the management of ADHD and MDD recommend bupropion as a first-line treatment option for Depression in ADHD patients (5). The similar use of serotonergic antidepressants (SSRIs and SNRIs) in both groups reflects standard first-line treatment approaches for unipolar depression. The use of antipsychotics and lithium as augmentative strategies was significantly lower in the MDD+ADHD group. This may indicate that depressive symptoms in patients with comorbid ADHD were either more responsive to first-line treatments or alternative favored treatment approaches, such as psychostimulant or bupropion augmentation (62).

Although patients with ADHD tended to receive a greater number of pharmacological treatment trials, this difference was not significant. The ordinal regression nonetheless suggested an increased likelihood of multiple trials among patients with comorbid ADHD, which may reflect greater treatment complexity or suboptimal antidepressant response rather than prescribing bias.

Studies indicate that 80% of adults with ADHD present with at least one additional psychiatric disorder (63–65). In our sample, the number of newly established comorbid diagnoses during inpatient treatment was low across all groups. This likely reflects the study design, which specifically focused on detecting previously unrecognized ADHD after partial remission of depressive symptoms, as well as the high preexisting diagnostic coverage typical of a specialized inpatient settings. Among the few newly identified comorbidities, substance use disorders were most frequent, followed by personality disorders. The prevalence of comorbid personality disorders in clinical samples shows high variability ranging from 21.9% (76) to 65.95% (77) for personality disorders.

In clinical populations, the prevalence of any comorbid substance use disorder ranges from 10.0% (66) to 82.9% (67).

In our study, both groups exhibited elevated rates of substance use. While inpatients with comorbid ADHD reported higher alcohol use than their counterparts, their average consumption per occasion was lower. Cannabis was the most frequently used substance in both groups, aligning with prior findings of increased cannabis use among individuals with depression (68) and ADHD (69). Elevated cannabis use corroborates existing literature on increased cannabis use in ADHD (70, 71) and major depressive disorder (71, 72). However, the overall drug use and specific categories such as cannabis and stimulants did not differ significantly between groups. The numerically higher rate of any drug use among patients with ADHD (50.0 % vs. 22.6 %) is directionally consistent with epidemiological data showing a two- to threefold increase in substance-use risk among adults with ADHD (73–75). Importantly, ADHD diagnoses were only established in patients without acute or severe substance dependence, as these conditions can mimic attentional and executive dysfunction and thereby compromise diagnostic validity. This exclusion criterion likely contributed to the relatively low number of comorbid substance-use cases in the ADHD group.

5. Strengths and Limitations

This study represents an effort to determine the prevalence of ADHD within a well-defined cohort of inpatients treated in a specialized ward for affective disorders. A multi-step screening procedure, augmented by comprehensive diagnostic instruments, was successfully implemented. However, the study was conducted in a highly specialized inpatient population, which may limit the generalizability of the findings. The diagnosis of ADHD was established during a depressive episode, and given the considerable symptom overlap between ADHD and depression, the potential influence of current mood state on diagnostic accuracy cannot be fully excluded. Reassessment of ADHD symptoms during a euthymic phase would therefore be required to confirm diagnostic stability. Moreover, the inclusion criterion of sufficiently reduced depressive symptom severity (BDI-II ≤ 20) before full ADHD assessment may have led to an underestimation of true ADHD prevalence, as patients whose depressive symptoms had not sufficiently improved were not evaluated further and potential ADHD cases could thus have been missedThe relatively small and unbalanced sample sizes, particularly the limited number of participants in the ADHD subgroup, reduce statistical power and may have led to inflated odds ratios and wide confidence intervals, thereby limiting the robustness of between-group comparisons. Consequently, the results should be interpreted with caution, and future studies with larger, more heterogeneous samples and longitudinal follow-up are needed to validate and extend these findings.

6. Conclusion

In cases of depression, and in particular in treatment-resistant depression it is important to consider the possibility of comorbid ADHD. The presence of ADHD can complicate the clinical picture. ADHD screening tools can provide valuable preliminary clinical indications regarding the potential co-occurrence of ADHD in patients who have been primarily diagnosed with depression.

In addition, our study suggests that a rapid onset of depressive episodes might serve as a clinical indicator of underlying ADHD. This finding highlights the necessity of comprehensive assessments that include the evaluation of ADHD symptoms, especially in patients presenting with rapid-onset depression.

The recognition of comorbid ADHD in depressed patients carries substantial clinical implications that extend beyond diagnostic considerations. The presence of ADHD can influence the course and severity of depression, requiring tailored treatment approaches. Effective management of both conditions is essential, as the treatment strategies for depression and ADHD can differ significantly.

Moreover, the identification of ADHD in depressed patients can be a game changer, offering a new perspective on the patient's symptoms and potentially leading to more effective intervention strategies. Addressing comorbid ADHD may not only improve depressive symptoms but also enhance overall functioning and quality of life.

Author Contributions

M.S., N.M., J.H., C.M. and S.W. conceived and designed the study. S.W. drafted the manuscript. J.H. and N.M. advised on statistical methodology and revised the analysis. S.W., J.H., M.S. edited the manuscript. All authors critically revised and approved the final manuscript.

Declaration of competing interest

M.S. has received speaker's fees from Medice, Takeda, Janssen and was an advisory board member for Takeda, Janssen and Medice in the past 3 years.

The other authors do not report any possible conflicts of interest.

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Ethics statement

The studies involving human participants were reviewed and approved by the Ethics Committee of the Medical Faculty, University of Leipzig (approval number: 040/24-ek). The study was conducted in accordance with the Declaration of Helsinki and local legal and institutional requirements. Due to the retrospective design and the use of fully anonymized data, written informed consent was not required in accordance with national legislation and institutional guidelines. Clinical trial number: not applicable.

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