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Research Article

Attention-Deficit/Hyperactivity Disorder in Children and Adolescents: Contemporary Advances in Diagnosis and Management-A Clinical Update

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Abstract

Background: Attention-deficit/hyperactivity disorder (ADHD) is one of the most common neurodevelopmental disorders of childhood and is associated with substantial academic, behavioral, social, emotional, and health-related burdens. Contemporary care extends beyond symptom identification to include developmentally informed assessment, recognition of comorbidities, shared decision-making, and measurement-based multimodal treatment. *Objective:* To provide an updated, clinically oriented synthesis of current evidence on the epidemiology, neurobiology, diagnosis, differential diagnosis, comorbidities, treatment, monitoring, and long-term management of ADHD in children and adolescents. *Methods:* This narrative clinical review used a focused PubMed/MEDLINE search covering January 1, 2018, through August 22, 2026, supplemented by foundational publications and authoritative guidance from the American Academy of Pediatrics, National Institute for Health and Care Excellence, US Food and Drug Administration, Centers for Disease Control and Prevention, and National Institute of Mental Health. The search identified 183 records for initial consideration. Evidence was selected purposively according to pediatric relevance, methodological rigor, clinical applicability, and regulatory status. *Results:* ADHD is a heterogeneous disorder arising from complex genetic, neurobiological, and environmental influences. Diagnosis remains clinical and requires developmentally inappropriate symptoms, functional impairment, childhood onset, and manifestations in more than one setting, while excluding alternative explanations. Rating scales support but do not replace comprehensive clinical assessment. Comorbid learning, anxiety, depressive, oppositional, autism spectrum, tic, sleep, and substance-use disorders can substantially influence presentation and management. Behavioral parent training is recommended as first-line treatment for preschool-aged children. For most school-aged children and adolescents with clinically significant impairment, stimulants provide the strongest evidence for reducing core symptoms, whereas atomoxetine, extended-release guanfacine, extended-release clonidine, and extended-release viloxazine are appropriate alternatives for selected patients. Effective management integrates medication when indicated with psychoeducation, behavioral strategies, school-based interventions, and treatment of comorbidities. Monitoring should address symptoms, functioning, adherence, appetite, growth, sleep, blood pressure, heart rate, mood, and medication misuse or diversion. Digital interventions may offer adjunctive benefits, but current evidence does not support replacing established treatments. *Conclusions:* Pediatric ADHD should be managed as a chronic, developmentally evolving condition. Accurate diagnosis, individualized multimodal treatment, systematic monitoring, and planned transition to adult services can improve symptoms and functioning. Objective biomarkers, pharmacogenomics, neurostimulation, microbiome-directed therapies, and most digital interventions remain adjunctive or investigational.

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1. Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by persistent and developmentally inappropriate inattention and/or hyperactivity-impulsivity that interferes with functioning or development [1-4]. Its clinical importance extends beyond classroom behavior. ADHD is associated with academic underachievement, family conflict, peer difficulties, accidental injury, reduced quality of life, emotional dysregulation, risky behavior, and increased use of health and educational services. Symptoms frequently persist into adolescence and adulthood, although their expression changes with development [1-5].

Recognition and treatment have improved, but important challenges remain. Symptoms overlap with normal developmental variation and with manifestations of anxiety, depression, trauma, sleep deprivation, learning disorders, autism, substance exposure, and environmental adversity. Girls and children with predominantly inattentive symptoms may be recognized late, while children from minoritized or socioeconomically disadvantaged groups may experience both underdiagnosis and barriers to evidence-based care. Conversely, diagnosis based only on brief reports or a single context can result in misclassification [3, 4, 6].

Contemporary ADHD care therefore requires more than confirmation of diagnostic criteria. Clinicians must determine whether symptoms are pervasive, developmentally inappropriate, impairing, and better explained by another condition; identify comorbidities and family priorities; establish measurable functional goals; select interventions according to age, severity, risk, and preference; and reassess outcomes over time. This review summarizes current evidence and translates it into a practical pediatric framework.

2. Methods

This clinical update was structured as a narrative review. A focused search of PubMed/MEDLINE was conducted for literature published from January 1, 2018, through August 22, 2026. The search combined the terms “attention-deficit/hyperactivity disorder” or “ADHD” with “children,” “adolescents,” or “pediatric,” and with “diagnosis,” “treatment,” “stimulants,” “behavioral therapy,” “nonpharmacological intervention,” or “digital intervention.” The core search prioritized systematic reviews, meta-analyses, clinical guidelines, and practice guidelines and identified 183 records for initial consideration. Targeted supplementary searches were used to locate pivotal randomized trials, major observational studies, and foundational publications issued before 2018 when they continued to define current practice.

Eligible sources addressed human pediatric populations, diagnosis or differential diagnosis, rating instruments, pharmacological or nonpharmacological treatment, adverse effects, monitoring, comorbidity, functional outcomes, or continuity of care. English-language peer-reviewed studies, major professional guidelines, and current authoritative regulatory or prescribing-information sources were considered. Adult-only studies, animal or in-vitro studies without direct clinical relevance, non-peer-reviewed opinion pieces, and reports lacking sufficient information to support a clinical statement were excluded. Final selection was purposive rather than exhaustive and was based on pediatric relevance, methodological rigor, clinical applicability, and contribution to the scope of this update. The resulting synthesis cited 54 sources.

Authoritative recommendations and safety information were obtained from the American Academy of Pediatrics (AAP), National Institute for Health and Care Excellence (NICE), US Food and Drug Administration (FDA), Centers for Disease Control and Prevention (CDC), National Institute of Mental Health (NIMH), and current DailyMed prescribing information. All regulatory and prescribing-information citations from 2025-2026 were rechecked against their authoritative sources on September 3, 2026. Because this was a narrative clinical update rather than a systematic review, a PRISMA flow diagram, formal risk-of-bias assessment, and meta-analysis were not undertaken. Ethical approval was not required because the review used only previously published, nonidentifiable information and generated no participant-level dataset.

3. Results of the Narrative Evidence Synthesis

Current evidence characterizes ADHD as a common, heterogeneous, and developmentally dynamic neurodevelopmental disorder. Diagnosis remains clinical and treatment should be multimodal, age appropriate, and measurement based. The evidence is synthesized below around clinical burden, pathophysiology, diagnosis, comorbidity, intervention, monitoring, and continuity of care [2, 3, 5, 10].

3.1. Epidemiology and Clinical Burden

International estimates generally place the prevalence of ADHD in children and adolescents near 5%, although reported rates vary according to diagnostic criteria, informant, age range, ascertainment method, and healthcare context [5, 7]. Administrative and parent-reported diagnosis rates may be higher than research estimates based on structured diagnostic assessment. Differences among countries do not necessarily indicate large biological variation; they often reflect awareness, access to care, educational expectations, diagnostic practices, and service organization.

ADHD is diagnosed more often in boys during childhood, particularly when hyperactive, impulsive, or disruptive behavior is prominent. The sex ratio narrows with age because girls more frequently present with inattentive symptoms, internalizing comorbidity, compensatory strategies, and less overt classroom disruption. Delayed recognition can lead to academic failure, anxiety, low self-esteem, or depression before ADHD is identified.

The burden is strongly influenced by comorbidity and social context. ADHD accompanied by conduct problems, severe emotional dysregulation, learning disability, autism, substance use, family instability, or limited educational support is associated with greater impairment than uncomplicated ADHD. Clinical outcomes should therefore be judged not only by symptom reduction but also by attendance, assignment completion, family interactions, peer functioning, safe driving, self-management, and quality of life. A recent meta-analysis indicates that stimulant and nonstimulant medications can improve quality of life, although these effects are generally smaller than their effects on core ADHD symptoms [8].

3.2. Etiology and Contemporary Pathophysiology

ADHD reflects interactions among genetic susceptibility, neurodevelopment, and environmental modifiers rather than a single neurotransmitter deficit. Common and rare variants implicate neuronal development, synaptic organization, and brain maturation [9-11]. Group-level studies identify differences in attention, default-mode, salience, reward, corticostriatal, and cerebellar networks, with contributions from catecholaminergic, glutamatergic, and GABAergic signaling [10-13]. Prematurity, low birth weight, prenatal exposures, lead, disrupted sleep, and severe adversity may modify risk or symptom expression [5, 10].

Epigenetic, inflammatory, metabolic, and microbiota-gut-brain pathways remain investigational [14, 15]. No genetic, neuroimaging, electrophysiological, inflammatory, metabolic, or microbial biomarker has sufficient sensitivity and specificity for individual diagnosis or medication selection. Current evidence therefore supports a multilevel developmental model and individualized clinical assessment [5, 10].

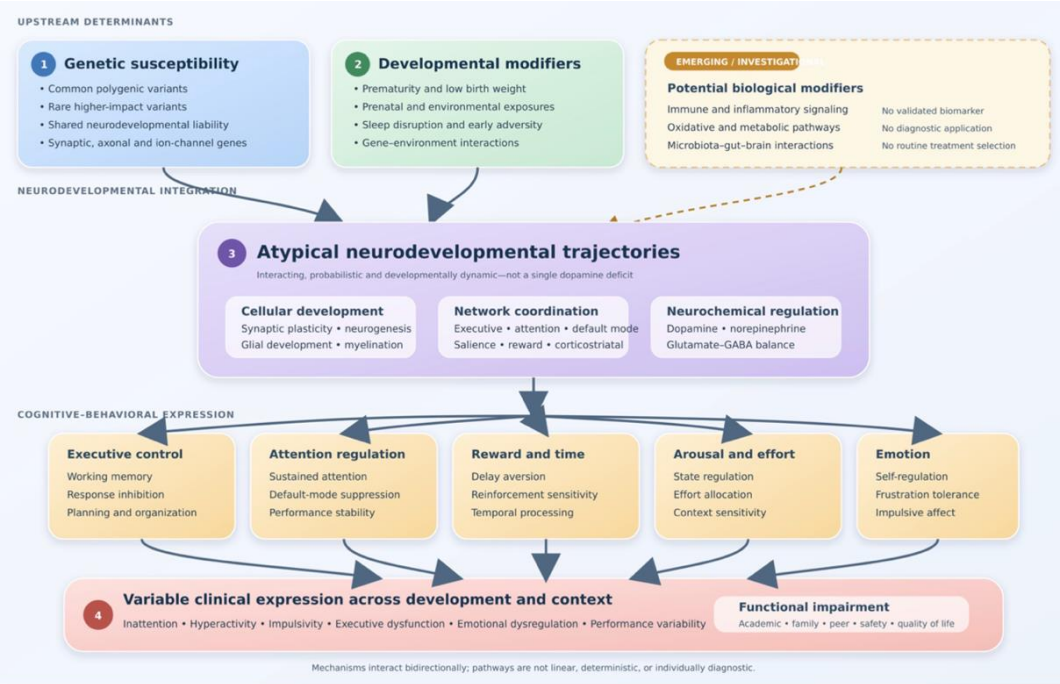

Figure 1. *Contemporary multilevel model of the pathophysiology of attention-deficit/hyperactivity disorder in children and adolescents.*

Figure legend: Interacting genetic, developmental, neural-network, neurotransmitter, and environmental influences affect attention, executive control, reward, arousal, and emotional regulation. Emerging immune, metabolic, and microbiota pathways are not clinically validated.
Source: Author's own elaboration based on references [5, 9-15].

3.3. Clinical Presentation Across Development

Preschool presentations commonly involve excessive activity, impulsivity, unsafe behavior, and difficulty following routines; normal developmental variability must be distinguished from persistent cross-setting impairment. In school-aged children, incomplete work, careless errors, lost materials, classroom disruption, and peer conflict become prominent as demands increase. Academic difficulty may also indicate learning, language, or intellectual disorders [1-5].

In adolescence, overt hyperactivity often becomes internal restlessness while planning, time management, emotional regulation, sleep, driving, adherence, and risk appraisal become more important. Adolescents should be interviewed privately regarding mood, self-harm, substance use, medication adherence, and stimulant diversion [2-5].

3.4. Diagnostic Assessment

3.4.1. Core Diagnostic Principles

Diagnosis is clinical. DSM-based criteria require a persistent pattern of inattention and/or hyperactivity-impulsivity that is inconsistent with developmental level, begins during childhood, occurs in more than one setting, causes clinically meaningful impairment, and is not better explained by another mental disorder or clinical condition [1-4]. For children up to 16 years, at least six symptoms in a domain are required; for individuals aged 17 years or older, the threshold is five. Symptoms must have persisted for at least six months, several must have been present before age 12, and impairment must be evident rather than inferred from symptom counts alone [1].

The AAP and CDC recommend evaluation of children and adolescents aged 4-18 years who present with academic or behavioral problems and symptoms of inattention, hyperactivity, or impulsivity [2, 16]. Information should be obtained from parents or caregivers and from teachers or other adults who observe the child in different settings. In adolescents, obtaining school-based observations may be more difficult because multiple teachers have limited contact; input from several teachers, coaches, counselors, and the adolescent becomes valuable.

3.4.2. Components of a Comprehensive Evaluation

1) the presenting concern and family priorities;
2) onset, duration, developmental appropriateness, and setting-specific expression of symptoms;
3) functional impairment at home, school, with peers, and in extracurricular activities;
4) developmental, medical, neurological, psychiatric, educational, family, and social history;
5) pregnancy and birth history when relevant;
6) sleep duration, sleep quality, snoring, restless sleep, and circadian pattern;
7) hearing and vision status;
8) medication, caffeine, nicotine, cannabis, alcohol, and other substance exposure;
9) trauma, bullying, housing instability, family conflict, and other psychosocial stressors;
10) review of school reports, grades, attendance, disciplinary events, psychoeducational testing, and individualized educational plans;
11) physical examination, including growth, blood pressure, heart rate, neurological observations, and signs suggesting an alternative diagnosis; and
12) systematic assessment for coexisting conditions.

3.4.3. Rating Scales and Ancillary Testing

Standardized rating scales strengthen diagnostic consistency by documenting symptom frequency, impairment, and change over time across settings. Common instruments include the Vanderbilt scales, Conners 4, ADHD Rating Scale-5, and Strengths and Difficulties Questionnaire [17-20]. Results should be interpreted using age- and sex-appropriate norms and integrated with the interview, developmental history, school information, and direct assessment. Disagreement between informants is common and should prompt examination of setting-specific demands, observation opportunities, classroom structure, family stress, and possible comorbidity rather than automatic rejection of either report.

Cross-cultural validity cannot be assumed merely because a rating scale has been translated. Symptom thresholds, informant expectations, classroom norms, language, and reference-group effects can influence scores. Whenever possible, clinicians should use a linguistically validated version with norms appropriate to the child's population and should document whether measurement invariance has been demonstrated. Even well-validated scales must be interpreted alongside developmental history, impairment, and reports from more than one setting rather than by applying a universal cutoff in isolation [51].

Computerized continuous-performance tests can quantify omissions, commissions, reaction time, and response variability, but their diagnostic accuracy is insufficient for stand-alone use [21]. Activity-augmented systems such as QbTest may provide supplementary information in selected cases; regulatory clearance indicates that a device may aid assessment, not that it independently establishes ADHD [22-24]. Neuropsychological testing is appropriate when intellectual disability, language disorder, learning disability, memory problems, or complex executive dysfunction is suspected, but normal performance does not exclude ADHD.

Routine electroencephalography, neuroimaging, genetic panels, inflammatory markers, microbiome testing, and pharmacogenomic panels are not recommended for diagnosis. EEG is reserved for clinical indications such as suspected seizures; imaging for focal neurologic findings or another suspected intracranial disorder; and laboratory testing for a plausible medical differential diagnosis. Objective and digital

measures may complement-not replace-a comprehensive clinical assessment [2, 3, 5, 21-25].

3.4.4. Differential Diagnosis

Many conditions can resemble or intensify ADHD. Normal developmental variability is distinguished by the absence of persistent, cross-situational impairment. Sleep restriction, obstructive sleep apnea, restless legs, circadian delay, and inconsistent routines can cause inattention, irritability, and hyperactivity; sleep history should therefore be routine. Anxiety often produces attention difficulty during worry-provoking situations, while depression is suggested by episodic decline, anhedonia, hopelessness, or neurovegetative change. Trauma-related symptoms may include hyperarousal, avoidance, intrusive memories, and context-linked deterioration.

Academic inattention confined to reading, writing, or mathematics suggests a specific learning disorder. Language disorder, hearing or vision impairment, intellectual disability, autism spectrum disorder, tic disorders, and developmental coordination disorder require targeted assessment when indicated. Oppositional behavior may reflect frustration or impulsivity, but persistent angry, defiant, or vindictive behavior beyond attention-demanding tasks suggests a coexisting disruptive-behavior disorder. Medication effects, thyroid disease, anemia, seizures, substance use, lead exposure, and other medical causes should be investigated when supported by the history or examination rather than through indiscriminate testing.

Diagnostic reasoning should establish chronology, pervasiveness, developmental appropriateness, and functional consequences. ADHD and another disorder may coexist; the presence of anxiety, autism, tics, learning disability, or depression does not preclude ADHD when full criteria are independently met [1-5].

3.5. Comorbidity

Learning disorders, anxiety, depression, oppositional or conduct disorders, autism spectrum disorder, tics, sleep disorders, and substance-use risk are common and materially affect impairment and treatment [2, 3, 5]. Assessment should distinguish symptoms attributable to ADHD from those requiring a separate intervention. Acute suicidality, psychosis, mania, severe depression, or dangerous aggression takes priority over routine ADHD titration.

3.6. Management Principles and Nonpharmacological Treatment

ADHD should be managed as a chronic condition using a medical-home or coordinated-care model [2, 16]. Treatment begins with psychoeducation that is accurate, non-stigmatizing, and developmentally appropriate. Families should understand that ADHD is neither a moral failure nor simply a lack of discipline; at the same time, diagnosis does not eliminate the need for expectations, skill development, and accountability.

The treatment plan should specify measurable targets-for example, completing and submitting assignments, reducing classroom removals, improving morning routines, decreasing unsafe impulsive behavior, or sustaining attention during homework. Symptom scores are useful, but functional goals determine clinical significance. Shared decision-making should address expected benefits, adverse effects, treatment burden, family preferences, access, cost, school schedule, stigma, and the child's capacity to participate.

Psychoeducation should explain ADHD as a neurodevelopmental condition, establish functional goals, and support shared decisions. Behavioral parent training is first-line for preschool-aged children and remains useful for disruptive behavior, routines, reinforcement, and consistent consequences. Classroom interventions include clear instructions, reduced distraction, frequent feedback, organizational support, daily report cards, and legally appropriate accommodations [2, 3, 26-28].

Adolescents may benefit from organizational-skills training and cognitive-behavioral strategies targeting procrastination, planning, and emotional regulation. Sleep, physical activity, and balanced nutrition should be addressed, but restrictive diets, supplements, neurofeedback, and digital interventions should not replace established care because evidence is inconsistent or limited [26-28].

The evidence base is strongest for behavioral parent training, contingency-management approaches, and structured classroom interventions. Organizational-skills training may improve planning, assignment management, and homework performance, particularly in school-aged children and adolescents, although effects on blinded ratings of core symptoms are less consistent. Cognitive-behavioral strategies can be useful for residual executive-function and emotional-regulation difficulties, but they should be matched to developmental level and should not be presented as substitutes for effective medication when medication is indicated [26-28].

Digital interventions include computerized cognitive training, serious games, prescription digital therapeutics, telehealth-delivered parent training, and digital tools for reminders or measurement-based follow-up. Trials and meta-analyses report small or selective improvements in attention, executive function, or symptom ratings, but findings are heterogeneous and the overall certainty of evidence remains limited; safety and adverse effects are also inconsistently reported [52, 54]. The FDA has granted De Novo classification to a product-specific digital therapeutic for pediatric ADHD, but that authorization should not be generalized to unrelated applications or interpreted as a replacement for comprehensive clinical assessment, behavioral care, school support, or indicated medication [53]. Digital tools should therefore be used only as adjuncts with explicit targets, attention to privacy and access, and periodic assessment of functional benefit.

3.7. Pharmacological Treatment

Medication is appropriate when symptoms cause clinically meaningful impairment and expected benefits outweigh risks. Stimulants have the strongest short-term evidence for reducing core symptoms in school-aged children and adolescents [28-30]. Methylphenidate or amphetamine formulations should be selected according to age, required duration, swallowing ability, adverse effects, misuse risk, availability, and family preference. Titrate from a low dose to meaningful functional improvement rather than to a predetermined weight-based target. If one stimulant class is ineffective or poorly tolerated, a trial of the other class is reasonable [2, 3, 28-31].

Atomoxetine, viloxazine extended release, guanfacine extended release, and clonidine extended release are alternatives when stimulants are ineffective, poorly tolerated, contraindicated, or associated with misuse concern. Atomoxetine and viloxazine have a gradual onset and require monitoring for suicidal thinking. Alpha-2 agonists may help hyperactivity, sleep difficulty, or tics but can cause sedation, hypotension, and bradycardia and must be tapered gradually [32-35, 49, 50]. The principal classes are summarized in Table 1.

Table 1. *Principal Medication Classes Used for Pediatric ADHD*

Class	Typical pediatric use	Approximate coverage	Principal considerations
Methylphenidate/dexmethylphenidate	Usually first stimulant trial in school-aged children; immediate- and extended-release oral or transdermal formulations	3-12 hours, product dependent	Titrate to function and tolerability; monitor appetite, growth, sleep, pulse, blood pressure, mood, and misuse [28-31, 36-39].
Amphetamine derivatives, including lisdexamfetamine	Alternative first-line stimulant; several immediate- and extended-release formulations	4-16 hours, product dependent	Often potent; products are not milligram-for-milligram interchangeable. Apply controlled-substance safeguards [28-31, 40-46].
Centanafadine extended release	An FDA approval letter was issued on July 24, 2026, for patients aged 6 years or older who weigh at least 20 kg; marketing was contingent on completion of controlled-substance scheduling, so current availability and labeling must be verified.	Once daily	Triple monoamine reuptake inhibitor and central nervous system stimulant. Labeling addresses suicidal ideation or behavior and abuse, misuse, and addiction; verify current FDA labeling and scheduling status before clinical use [47, 48].
Atomoxetine	Nonstimulant from age 6 years; useful with stimulant intolerance, anxiety, tics, or misuse concern	All-day after gradual onset	Dose by weight; monitor mood, blood pressure, pulse, growth, rare hepatic symptoms, and CYP2D6 interactions [33].
Viloxazine extended release	Nonstimulant from age 6 years	Once daily	Monitor suicidal thoughts/behavior, activation, blood pressure, pulse, sleep, and CYP1A2 interactions [35].
Guanfacine or clonidine extended release	Monotherapy or stimulant adjunct from age 6 years; may help hyperactivity, tics, sleep, or evening symptoms	Once daily or divided, product dependent	Sedation, hypotension, bradycardia, and rebound hypertension; taper gradually [34, 49, 50].

Durations and indications vary by jurisdiction and formulation. Doses should be individualized and verified against current product-specific prescribing information; similarly named immediate- and extended-release products may not be interchangeable.
Source: Author's own elaboration based on current clinical guidelines, systematic reviews, and product-specific prescribing information.

3.8. Monitoring and Developmentally Adapted Long-Term Care

Before medication, document target symptoms and functional goals; current medicines; height, weight, pulse, and blood pressure; sleep and appetite; psychiatric history; tics; seizures; substance use; and personal and family cardiac history. Routine electrocardiography is not required in an asymptomatic child without risk factors, but cardiology review is appropriate for exertional syncope or chest pain, known heart disease, pathologic murmur, unexplained seizures, or a family

history of sudden early death [2, 3].

Start at a low dose and titrate systematically using parent, teacher, and patient reports. The best dose produces meaningful functional benefit with acceptable adverse effects; it is not determined solely by body weight. If a stimulant is ineffective after an adequate trial, confirm adherence and diagnosis, then consider another formulation or stimulant class before a nonstimulant. Combination therapy may be appropriate in selected partial responders but requires explicit goals and monitoring.

At each review assess functioning, duration of benefit, rebound symptoms, adherence, appetite, weight, growth, sleep, pulse, blood pressure, mood, psychosis or mania, tics, and misuse or diversion. Small average increases in pulse and blood pressure can occur with stimulants and atomoxetine. Persistent abnormalities require reassessment. Appetite loss can be addressed by dosing after breakfast, nutrient-dense meals when medication effect wanes, or changing dose/formulation. New psychotic or manic symptoms warrant prompt discontinuation and assessment. Alpha-2 agonists must be tapered to avoid rebound hypertension; atomoxetine, viloxazine, and centanafadine require monitoring for suicidal thinking according to labeling [31-35, 47-50].

For preschool-aged children, behavioral parent training is first-line. Medication is considered when impairment remains moderate to severe after an adequate behavioral intervention or when such treatment is unavailable; methylphenidate has the strongest evidence but requires conservative titration and close growth monitoring [2, 3]. For school-aged children, medication plus parent and classroom interventions is usually most effective. Written goals should address academic productivity, behavior, peer relationships, and family functioning.

Adolescents should participate directly in shared decisions. Review adherence, mood, sleep, substance use, driving safety, sexual health when relevant, and stimulant diversion. Longer-acting formulations may reduce school dosing and diversion opportunities, but selection should match daily coverage needs. Transition planning should identify an adult clinician, transfer diagnostic and treatment records, teach medication self-management, and reassess ongoing impairment [2-5].

Treatment priorities depend on risk and impairment. Stabilize acute suicidality, mania, psychosis, severe depression, or dangerous aggression before routine ADHD titration. With anxiety, stimulants may still be effective, while atomoxetine or combined cognitive-behavioral therapy may be useful. With tics, methylphenidate, atomoxetine, or alpha-2 agonists are reasonable; monitor individual response. Learning disorders require explicit educational remediation because ADHD medication does not correct a skill deficit. Autism spectrum disorder may require slower titration because adverse effects can be more frequent. Adolescents with substance-use risk benefit from integrated treatment, secure storage, limited quantities, and consideration of long-acting or nonstimulant options [2, 3, 5, 26-30].

ADHD is often chronic, but its expression changes as demands increase. Periodic reassessment should determine whether criteria and impairment persist, whether the formulation still covers the relevant day, and whether treatment goals have changed. Planned, supervised trials without medication may occasionally clarify continuing need when functioning is stable; abrupt unsupervised discontinuation should be discouraged. Long-term care should emphasize meaningful outcomes-learning, relationships, safety, autonomy, and quality of life-rather than symptom scores alone.

3.9. Equity, Access, and Emerging Approaches

Diagnostic and treatment inequities arise from language barriers, referral patterns, cost, school resources, stigma, and cultural expectations. Clinicians should obtain information from multiple settings, use validated translations when available, distinguish impairment from culturally mediated interpretations of behavior, and connect families with educational and community supports. Shared decisions should acknowledge preferences, feasibility, and medication availability without lowering diagnostic standards [3, 5, 6].

Digital therapeutics, telehealth platforms, computerized monitoring, neurostimulation, pharmacogenomic panels, microbiome-directed interventions, and artificial intelligence are active areas of research. Product-specific regulatory authorization or promising group-level findings do not validate these approaches as general diagnostic biomarkers or first-line replacements for established treatment. Pharmacogenomic testing has not demonstrated sufficient utility to select an ADHD medication routinely; neurostimulation and microbiome-directed approaches remain investigational; and artificial-intelligence outputs require clinician oversight and external validation. These approaches should be described as adjunctive or investigational unless supported by indication-specific regulatory authorization and adequate comparative evidence [5, 10, 24, 52-54].

3.10. Practical Clinical Algorithm

Confirm developmental inappropriateness, childhood onset, impairment, and symptoms in more than one setting; evaluate alternative explanations and comorbidity; establish measurable goals; provide psychoeducation and school support; initiate age-appropriate behavioral treatment; add medication when indicated; titrate systematically; and reassess function, adverse effects, adherence, growth, cardiovascular parameters, mood, sleep, and misuse risk. Revise the diagnosis or plan when meaningful improvement does not occur [2, 3].

4. Discussion

ADHD care has shifted from symptom counting toward a developmental, functional, and longitudinal model. The diagnosis remains clinical because available objective tests lack

adequate stand-alone accuracy. Multi-informant assessment is essential, although discrepancies between home and school reports should be interpreted as contextual information rather than simple error [1-5, 17-25].

Treatment evidence supports behavioral parent training for preschool-aged children and stimulant medication for most school-aged children and adolescents with substantial impairment. Nonstimulants expand options when response, tolerability, comorbidity, cardiovascular factors, or misuse risk limits stimulants [2, 3, 26-35]. Medication improves core symptoms more reliably than it corrects learning deficits, family conflict, or social disadvantage; educational and psychosocial interventions therefore remain necessary.

Digital interventions may extend access and offer adjunctive improvement in selected outcomes, but the evidence is heterogeneous and generally less robust than that supporting established behavioral and pharmacological treatments. Similarly, current data do not support routine use of pharmacogenomics, neurostimulation, microbiome-directed therapy, or objective biomarkers to diagnose ADHD or select treatment [5, 10, 24, 52-54].

The practical priorities are individualized goals, systematic titration, monitoring of growth and cardiovascular and psychiatric safety, management of comorbidity, and planned transition to adult services. Access, language, stigma, medication availability, and school resources can influence outcomes and should be incorporated into shared decisions [3, 5, 6].

This narrative review is limited by the absence of systematic study selection, formal risk-of-bias assessment, and meta-analysis. Product authorizations and safety information may change. Clinicians should consult current local guidelines and prescribing information. Future research should emphasize long-term functional outcomes, comparative effectiveness, complex comorbidity, and implementation in diverse and underserved populations.

5. Conclusions

Pediatric ADHD is a heterogeneous and often persistent neurodevelopmental disorder. Accurate diagnosis requires developmental history, impairment, evidence from more than one setting, and exclusion of better explanations. Treatment should combine psychoeducation, behavioral and educational support, medication when indicated, and management of comorbidities. Systematic monitoring and developmentally planned continuity of care can improve symptoms, functioning, safety, and quality of life. Emerging biomarkers and digital technologies remain adjunctive or investigational.

Abbreviations

AAP	American Academy of Pediatrics
ADHD	Attention-deficit/Hyperactivity Disorder
CDC	Centers for Disease Control and Prevention
CYP1A2	Cytochrome P450 1A2
CYP2D6	Cytochrome P450 2D6
EEG	Electroencephalography
FDA	US Food and Drug Administration
GABA	Gamma-aminobutyric Acid
NICE	National Institute for Health and Care Excellence
NIMH	National Institute of Mental Health

Author Contributions

Vicente Manuel Martinez Cardenas: Conceptualization, Data curation, Investigation, Methodology, Resources, Visualization, Writing – original draft, Writing – review & editing

Data Availability Statement

No new dataset was generated or analyzed for this narrative review.

Conflicts of Interest

The author declares no conflicts of interest.

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