An Overview on Attention Deficit Hyperactivity Disorder (ADHD)

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Abstract:

Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder that is characterized by a lack of inattention, hyperactivity, impulsivity which starts from childhood-onset and continues to adulthood. ADHD was shown to be prevalent in 11.32 percent of primary school pupils in India. When compared to females, males had a greater prevalence (66.7 percent) than females (33.3 percent). The prevalence was determined to be 16.33 percent in the lowest socioeconomic group and 6.84 percent in the middle socioeconomic group.

The diagnostic technique for these diseases is DSM-6, which is substantially updated terminologically. Neuroimaging techniques are also used for diagnostic purposes like FMRI (Functional Magnetic Resonance Imaging), and DTI (Diffusion Tensor Imaging).

Pharmacotherapy includes psychostimulants like methylphenidate which has a good response in treating inattentive symptoms at lower doses whereas at higher doses hyperactivity and impulsivity can be treated.

Nonpharmacological treatment includes behavioral therapy, cognitive therapy, parent behavior training, classroom interventions, and intake of dietary supplements like probiotics, omega fatty acids, and vitamins, etc., the recent advances in the treatment of ADHD is the development of the first non-stimulant Atomoxetine which was approved by FDA and followed by methylphenidate transdermal system, modafinil.

In children, problems paying attention may result in poor school performance. ADHD is associated with other neurodevelopmental and mental disorders as well as some non-psychiatric disorders, which can cause additional impairment, especially in modern society. Although people with ADHD struggle to focus on tasks they are not particularly interested in completing, they are often able to maintain an unusually prolonged and intense level of attention for tasks they do find interesting or rewarding; this is known as hyperfocus.

Adult ADHD is typically marked by inattention and hyperfocus, hyperactivity (often internalized as restlessness), emotional dysregulation, and excessive mind wandering. Specifically, adults with ADHD present with persistent difficulties in following directions, remembering information, concentrating, organizing tasks, completing work within specified time frames and appearing timely in appointments. These difficulties affect several different areas of an ADHD adult's life, causing emotional, social, vocational, marital, legal, financial, and/or academic problems.

Diagnosis follows one or several psychiatric assessment which may include examination of personal history, observational evidence from family members or friends, academic reports, often going back to school years, as well as evaluation to diagnose additional possible conditions which often coexist with ADHD, called comorbidities or comorbid disorders.

Despite the scientifically well-established nature of attention deficit hyperactivity disorder (ADHD), its diagnosis, and its treatment. The controversies involve clinicians, teachers, policymakers, parents, and the media. Positions range from the view that ADHD is within the normal range of behavior to the hypothesis that ADHD is a genetic condition.

Key Words: FMRI (Functional Magnetic Resonance Imaging), DTI (Diffusion Tensor Imaging), prefrontal cortex (PFC), dopamine transporter-1 (DAT-1), (DA) Dopamine, (NE) Norepinephrine. Oppositional Defiant Disorder (ODD), Major Depressive Disorder (MDD).

Introduction:

ADHD is a neurological disorder characterized by inattentive, hyperactive, and/or impulsive symptoms that create difficulty in everyday living [1]. The features of life as it develops and evolves over time. This problem is more 'cognitive' than 'behavioral' due to its length. "Sub syndromic kinds," on the other hand, result in clinical heterogeneity. (Either inattentive, hyperactive/impulsive, or a mix of the two), making identification challenging. In addition, the main etiology. Mechanisms aren't understood fully yet, although they are unquestionably present. With multiple risk variables, especially environmental, prenatal, and perinatal, it's also complex and multidimensional. Toxins, diet, and psychological factors should all be taken into account [2]. ADHD was found to have a significant genetic component in twin research, with an expected heritability of 70–80 percent [3,4].

Synaptic vesicle fusion, neurite outgrowth, and cell adhesion processes, as well as dopaminergic, serotonergic, and glutamatergic neurotransmitter pathways, have all been implicated in genetic studies [5-7].

The term "ADHD" has just recently been added to the list of neurodevelopmental diseases. The Diagnostic and Statistical Manual of Mental Disorders (DSM-V) Fifth Edition (DSM-V) attests to current work which has demonstrated the role of neural network disorders [8,9]. Many brain networks have been related to ADHD in neuroimaging studies, including the fronto-striatal, fronto-parieto-temporal, fronto-cerebellar, and fronto-limbic, yet the clinical entity's existence remains uncertain. Many confounding variables can contribute to over-diagnosis or misdiagnosis, and many confounding factors can lead to over-diagnosis or misdiagnosis of the condition [10].

The French National Health Authority issued guidelines for the primary care management of ADHD in children and adolescents in 2014[11]. Because the condition impacts so many different fields, such as neurology, pediatrics, psychiatry, and neuropsychology, it should be recognized and addressed as a multidisciplinary issue [12].

One difficulty is that ADHD children, either diagnosed or not, grow up to be adults, and multiple studies demonstrate that ADHD symptoms persist long into adulthood. As a result, ADHD is an issue that can last a lifetime [12]. When evaluating cognitive and behavioral difficulties in adults, however, healthcare providers are usually unaware that ADHD symptoms might be present. As a result, greater awareness of adult ADHD may lead to improved management, and treatment, of this population, as this disorder causes functional impairment in daily activities (ADL).

DSM-5 Criteria for Attention-deficit hyperactivity disorder (ADHD

In attention: Six or more of the following symptoms have lasted for at least six months to a developmental level, adversely impacting social, intellectual, and occupational activities.

•Fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities.

•Loses things necessary for tasks or activities (e.g., school materials, books, tools, wallets, keys, eyeglasses, mobiles).

•Often forgetful in daily activities like paying bills, making payments, appointments in work etc.,

•Easily distracted by external stimuli where the person cannot focus on the work.

•Difficulty in organizing or performing works /tasks.

•Engage or dislike to doing the tasks which needs the mental effort.

•Often does not seem to listen when spoken to directly (seems to the absence of mind and thinking somewhere else)

Hyperactivity and Impulsivity: "Hyperactivity/impulsivity" is defined as having six or more symptoms of hyperactivity or impulsivity in children under the age of 16 and five or more in teenagers aged 17 and older. These symptoms are inappropriate for developmental level and have been present for at least six months to a disruptive extent.

•Often blurts out an answer before a question has been completed.

•Often unable to play or engage in leisure activities quietly.

•Often has difficulty waiting his or her turn (e.g., while waiting in line)

•Often interrupts or intrudes on others (e.g., butts into conversations, games etc., may start using other people's things without asking or receiving permission)

Three presentations of ADHD

1)Combined.

2)Predominantly inattentive.

3) Predominantly hyperactive/impulsive.

Epidemiology of ADHD

Incidence of ADHD:

The prevalence of ADHD in children and adolescents ranges from 5–7.1%, and location has little bearing [13-15]. Using structured questionnaires based on DSM-IV criteria, screening for ADHD in the general adult population indicates a prevalence of 1% when all six criteria are satisfied, and 2.5 percent when four out of six are met [16]. The World Health Organization (WHO) conducted a retrospective and structured assessments of ADHD in children and adults in around 20 countries, finding prevalence rates in adults ranging from 4.4 percent in the United States to 3.4 percent over the same period in ten countries, with prevalence rates in children ranging from 1.5 percent (Colombia and South Africa) to 4.4 percent in the United States. Due to methodological inconsistencies in applying previous ADHD criteria and the procedure employed for diagnosing attention deficit hyperactivity disorder (ADHD) in the elderly, adulthood has yielded a broad range of findings, ranging from 4% [18] to 76 percent [19].

Gender proportions:

A male majority of around 3–4:1 has been documented in epidemiological studies of ADHD populations, and even climbs in the clinical population, possibly due to referral bias [20-21],

since women present with lower rates of hyperactive symptoms [22]. Unlike ADHD children's populations, however, the gender ratio in adults tends to level out [23]. This indicates that adult females are diagnosed at a higher rate than males, probably because they are more aware of their own attentional and organizational problems (metacognition).

Etiology of ADHD:

Even though the exact cause of ADHD is unknown, initial hypotheses of decreased brain activity were based on several findings of reduced grey and white matter volume or capabilities in the brain, which led to deficits in cognitive processing, attention, motor planning, speed of processing responses, or other behavioral issues seen in ADHD [24]. The prefrontal cortex (PFC), caudate, and cerebellum have recently been identified as key areas in ADHD impairments. These regions are linked by a neuronal channel, which jointly governs attention, thoughts, emotions, behaviors, and actions [25-26]. Studies have discovered a delayed development of the PFC [27] or a reduction in the volume and activity of the PFC, or cerebellum, in ADHD individuals [25]. The network activity between these areas is "extremely sensitive to the neurochemical environment," [25] and is maintained by neurotransmitters, dopamine, and norepinephrine acting in concert through multiple receptors [25-31], which can be presynaptic or postsynaptic (Figure 1) [32-33].

The brains of ADHD patients are smaller than average [34-36]. The disorder has been connected to polymorphisms in the genes that code for dopamine D4 receptors, D5 receptors, and D6 receptors. The dopaminergic system's function has been disrupted [37] and the condition has been connected to a disturbance of the α2A system.

All of these studies revealed that DA and/or NMDA receptor function is disrupted, resulting in decreased attention, impulsive behavior, and hyperactive [25]. These findings have led to a diminished DA and/or NMDA receptor activity in ADHD, or the NE function hypothesis, that perfectly fits the mechanism of action of the medications used to treat ADHD. For example, methylphenidate, amphetamine, and atomoxetine stimulate DA and NE transport in the PFC, but guanfacine stimulates postsynaptic α2A receptors directly [25]. However, a hyperactive DA or NE system has been related to ADHD in several studies [30].

An A559V gene mutation and an R615C gene mutation in DAT-1 have recently been discovered in some ADHD patients, both of which enhance or decrease dopamine efflux or presynaptic

uptake, reflecting a hyperactive DA response in these people [32,38,39]. Furthermore, inhibiting the enhanced efflux of DA with methylphenidate and amphetamine alleviates ADHD symptoms in the A559V mutant group [32].

Direct postsynaptic receptor activation would help with ADHD symptoms. It is also argued that there is still a fundamental NT-level deficiency in ADHD. As a result, using DA agonists such as piribedil, amantadine [30], or naltrexone, or a dosage of levodopa with carbidopa [40,41], or a dose of levodopa with carbidopa, does not improve attention in ADHD patients.

The hypoactive and hyperactive catecholamine assumptions of ADHD have been combined due to the fact that DA and NE might have had an inverted U-shaped dose-response curve [25] similar to that seen with vitamins, in which either extreme is a problem, and that two different pools of DA [30] and NE [42] exist in the brain. (Figure 1).

The PFC requires an optimum level of DA/NE for proper functioning, and disturbance causes ADHD. Medications for ADHD help to restore the NTs' delicate equilibrium they are covered later in the PFC.

Pathophysiology of ADHD:

Attention deficit hyperactivity disorder is one of the most common neurobehavioral disorders affecting children aged between 6 to 17, with prevalence estimates ranging from 2% to 18% of this age population in the United States [43]. ADHD is a heritable, chronic neurobehavioral condition characterized by hyperactivity, inattention, and impulsivity. [44].

Three subsets of ADHD are today distinguished as predominantly hyperactive-impulsive, predominantly inattentive, and a combined type, defined by a combination of the first 2 subtypes [43,45]. Children and adolescents with ADHD have a difficult time during their formative years.

As a result of impulsive behavior and a slower reaction time, they have slower rates of information processing,[45]do badly on standardized tests, have poorer grades, and are more at risk of dropping out [44]. Impulsivity also raises the likelihood of car accidents and spontaneous sexual encounters, which could explain why these people have more teen pregnancies and are more likely to contract sexually transmitted illnesses [44,46].

Low self-esteem is linked to issues in social connections, a proclivity for substance misuse, and issues with law enforcement [44]. Furthermore, ADHD is frequently associated with one or more comorbidities, such as oppositional defiant disorder (ODD), major depressive disorder (MDD), and anxiety disorders, which add to the difficulty.

Approximately 60 percent to 80 percent of Symptoms of ADHD last throughout adulthood [44]. As a result, ADHD isn't merely a childhood illness that goes away on its own during puberty. In the United States, it is believed that 4% to 4.5 percent of people suffer from ADHD [47,48].

Adults show signs such as poor work performance, poor socioeconomic position, and family issues [49]. As a result, the lower quality of life experienced by people with ADHD required necessary treatment in order to avoid the numerous issues that these people face as children, teenagers, and adults.

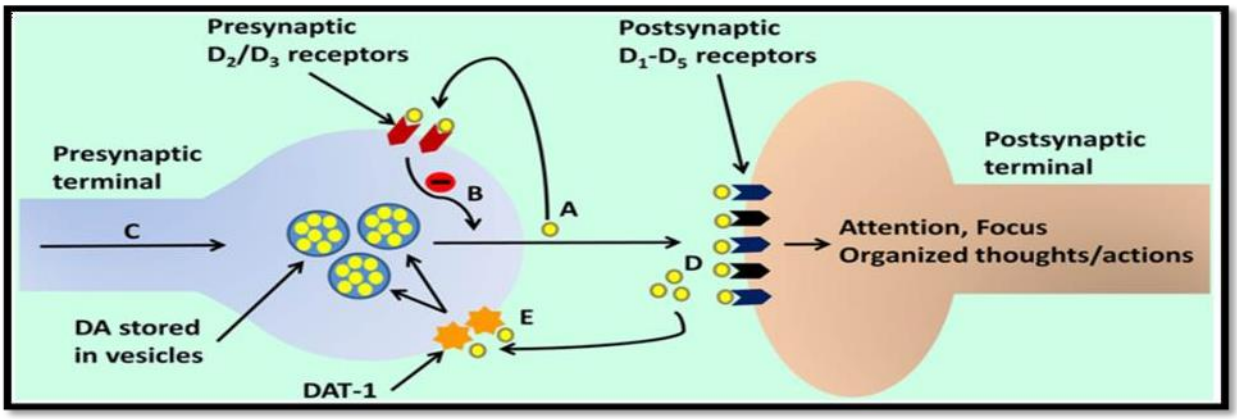

Figure 1: The hypoactive and hyperactivity catecholamine hypotheses of ADHD are combined here.

Dopamine binds to five dopamine receptors (named D1-D5) found on postsynaptic neurons. The D2 and D3 receptors are found on presynaptic neurons. The presynaptic terminal releases a little amount of vesicular DA into the synapse in the absence of an action potential. **(A)**This is the tonic pool, which primarily acts on the D2/D3 presynaptic sites and inhibits the release of DA via feedback inhibition. **(B)** After the presynaptic terminal receives an action potential. **(C)** The phasic pool is formed when a large amount of vesicular DA is released into the synapses. **(D)** The activity of the dopamine transporter-1 (DAT-1) is dependent on the amount of dopamine produced in the phasic pool and tonic pool. Dopamine activity on postsynaptic receptors is halted

when it passes into the presynaptic terminal of the brain. **(E)** Similarly, NE receptors are divided into two types: α1(α1A, α1B, α1D) and α2 (α2A, α2B, α2C) receptors, as well as β1, β2, and β3 receptors. The presynaptic α2 receptors, like the D2/D3 receptors, serve as auto receptors, DAT-1 and DAT-2 reuptakes transported NE into the presynaptic terminal, where it was stored for later release. InADHD, the tonic pool is hypothesized to be reduced, allowing for a higher-than-normal release of dopamine and, as a result, chaotic behavior including inattention and hyperactivity.

Diagnosis of ADHD:

Despite the fact that several researchers have looked at the prospect of establishing a diagnostic marker for ADHD, [50-55] this promise has yet to be realized, owing to the disorder's severity. In the United States, clinicians should diagnose ADHD using the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V) as a guideline [45]. Given that there are three subtypes of ADHD, each with its own set of symptoms, the requirement for children exhibits at least six symptoms for at least six months in order to be labeled with a specific subtype is unnecessary.

There must be evidence of functional impairment in a variety of settings, [43,45,56]. The DSM-V criteria for ADHD are better than the DSM-IV-TR criteria, but they still do not address sex differences in ADHD. Comorbidities such as ODD, MDD, and anxiety disorders make it more difficult to identify ADHD since the symptoms and features of these diseases are similar to those of ADHD [43,57]. Males are three times more likely than girls to have ADHD and to have the mainly hyperactive or mixed type.[44] whilst females are more likely to have the mostly inattentive subtype and to have mental impairment and eating disorders [58]. Males have a higher rate of violence and are more prone to break the law [58]. Clinicians must be aware of these sexual and developmental variations across people to ensure that ADHD is neither over- nor underdiagnosed. As a result, diagnosing ADHD is a complicated and time-consuming procedure that should only be done by qualified specialists after repeated observations and reports from parents, teachers, or caregivers [46,59]. To give objectivity and quantification to the subjective criteria, many rating scales have been developed. These scales are divided into two categories: narrowband and broadband, depending on whether they test for a specific or broad range of behaviors/manifestations [60]. Given the fact that broad-band scales give a superior overall

clinical feature, narrow-band scales are too often used (especially for measuring therapeutic efficacy) due to their reliability and small-time investment. [60].These narrow-band scales may be based on adolescent self-report or those completed by parents, teachers, or caregivers to gauge internalization. As a result, adult-reported scales provide a more accurate evaluation of a treatment's efficacy. The Conners Rating Scales–Revised; Inattention/Overactivity with Aggression (IOWA) Conners Teacher Rating Scale; Swanson, Nolan, and Pelham-IV (SNAP-IV) Questionnaire; Swanson, Kotkin, Agler, M-Flynn, and Pelham (SKAMP) rating scale; ADHD Rating Scale-IV; Vanderbilt ADHD Rating Scale; and ADHD Symptom Rating Scale are the most used narrow-band scales [60]. Though these events are based on DSM-IV-TR criteria for diagnosing ADHD, their limitations [60] as well as their uncertain specificity and sensitivity, would prevent their use as individual diagnostic criteria for ADHD [61]. A lack of specificity despite the high sensitivity of the Conners Teacher and Parent Rating Scales for identifying ADHD has recently been described, resulting in numerous false-positive ADHD diagnoses.

Treatment of ADHD:

Drug therapy, behavioral therapy, or a mixture of these two may be used to treat ADHD. Treatment is suggested for all children, regardless of the strategies utilized, because early and effective treatment of ADHD has been shown to result in a better prognosis and fewer issues in adulthood [44], as well as bring relief to parents and teachers [62]. Many psychologists used to believe that ADHD was caused by bad parenting, which led to a variety of behavioral treatments for the condition [63,64]. These methods, however, were not always beneficial in all cases. After proof of a neurochemical basis for ADHD emerged, pharmacological treatments were considered to be more beneficial than behavioral therapy. Many doctors mixed medicine and behavioral therapy, but the relative efficacy of behavioral therapy vs. pharmacological therapy, the value of combining the two, and the long-term benefit of either strategy was all unknown. These problems were addressed at the same time in a landmark experiment called the Multimodal Treatment Study of Children with ADHD (MTA). The study's findings [65] as well as the follow-up findings [66-68], have been published and thoroughly discussed [56,68-71]. Even though all the treatments studied in the study were found to have long-term benefits, pharmacological therapy was found to be superior to behavioral therapy in managing ADHD symptoms/manifestations. Although behavioral therapy was beneficial, its impact was not as strong as that of

pharmacological treatment. [72]. As a consequence, it may be advantageous for ADHD patients with modest symptoms and minimal impairment, and for parents who prefer it over pharmaceutical treatment. [61]. It can also be used in combination with pharmacological therapy if the FDA-approved drugs only provide a partial response or if comorbid diseases are present [61]. Stimulants like methylphenidate and amphetamines, as well as non-stimulants such as atomoxetine and extended-release α-2 agonists, have been approved by the FDA for treating ADHD. Tricyclic antidepressants Off-label treatments for ADHD include dopamine agonists and bupropion; however, these are only used if the above treatments are ineffective or unavailable. The next sections go through these medications in further depth.

STIMULANTS:

The FDA has approved two stimulant-based medicines for all age groups in the United States: amphetamines and methylphenidate. Table 1 shows the FDA-recommended doses for various medications, although the effective dose required for most adults exceeds the FDA-approved maximum dose per day and is thus deemed off-label. Moreover, there is a scarcity of data on the efficacy and safety of these larger doses [73]. The stimulants bind to and inhibit DAT-1 and the norepinephrine transporter (NET), preventing DA and NE from being reabsorbed.

Both stimulants inhibit the enzyme called monoamine oxidase, which breaks down the catecholamines, although amphetamine is the stronger of the two [30]. As a result, the net impact of either stimulant is to correct the level of NTs in the synapse, such as DA and NE. Because methylphenidate and amphetamine have slightly distinct mechanisms of action, some patients who don't respond to one stimulant respond better to the other.

A) Methylphenidate:

Specific methylphenidate formulations may contain a racemic mixture of the two isomers or only the more active d-isomer (Ritalin, Concerta, Metadate, and Daytrana) (Focalin) [74-78]. Since Daytrana is a transdermal patch, it may be favored by patients who have difficulty ingesting solid dose forms; also, because there is no first-pass impact, it has a higher bioavailability than orally taken dosage forms [61]. Oral dosage forms come in immediate-release and extended-release varieties, as well as a variety of strengths with modest dose increments. Manipulation of the dose makes it simple to achieve the anticipated impact with slight side effects.

B) Amphetamine Salts:

The most often prescribed amphetamine formulations are Adderall XR and Vyvanse, which include amphetamine salts as well as the prodrug lisdexamfetamine. Both the d- and l-optical isomers of amphetamine are active. Methylphenidate has a similar set of side effects [79,80]. Because amphetamines are metabolized by CYP1A2, CYP2C9, CYP2D6, and CYP3A4[79], a wide variety of drug interactions are possible [79,80]. Furthermore, CYP2D6 isoforms create slow and quick metabolizers in the general population, resulting in greater amphetamine concentrations among Latinos and African Americans, as well as around 7% of the population. [69].

NON-STIMULANTS:

Even though stimulants are considered first-line therapy, over 30% of ADHD patients may not benefit from them [81,82]. Non-responsiveness to stimulants or partial ability to respond to stimulants leads to intolerance to their side effects the existence of medical issues such as psychiatric, cardiovascular, or tic disorders, and family dislikes to controlled substances may need the usage of nonstimulant alternative agents that replace the stimulants as adjuncts to

treatADHD. Atomoxetine (Strattera) and extended-release α-2 agonists like clonidine (Kapvay) and guanfacine are the FDA-approved non-stimulants for the treatment of ADHD (Intuniv).

A) Atomoxetine (Strattera):

Atomoxetine (Strattera) has been licensed by the FDA to treat ADHD in children, adolescents, and adults. Atomoxetine, like stimulants, maintains the accessibility of both NE and DA in PFC [83] synapses, helping ADHD patients enhance their PFC function [84,85]. Since it has no actions on the striatum and requires at least 4 to 6 weeks to show full effect, it is less likely to be misused than stimulants [86-88]. As a result, it may be advised for individuals who have a problem with or are concerned about drug abuse [61,69]. The temporal lag linked with atomoxetine, on the other hand, emphasizes the need of encouraging patients and caregivers to be patient with this medication. It may also aid in the treatment of tics and anxiety.

B) α-2 Agonists:

Presynaptic and postsynaptic α-2receptors in neuronal cells are affected by guanfacine and clonidine. Because activating postsynaptic α-2A receptors is likely to be important in treating ADHD symptoms, and guanfacine is more selective at these receptors than clonidine, it may be more effective than clonidine. Guanfacine produces less sedation and dizziness than clonidine due to its specificity for postsynaptic α-2A receptors [69] and longer half-life [90].

C) Bupropion:

Bupropion is an FDA-approved antidepressant and smoking cessation drug [69,91,81] that has also been used off-label to treat ADHD. It inhibits the reuptake of both DA and NE, like stimulants and atomoxetine, but without the risk of abuse and dependence associated with stimulants. this medication should not be used in people having eating disorders and seizures [69].

TRICYCLIC ANTI-DEPRESSANTS:

Imipramine, desipramine, and nortriptyline have all been used, however desipramine seems to be the least recommended due to the risk of mortality [61]. Desipramine doses must also be reduced in situations of hepatic impairment. An ECG must be performed before beginning TCAs and after each dosage increase to ensure cardiac safety [61]. These pharmaceuticals, like every other drug listed above, should be gradually increased in frequency to build tolerance, and if they are to be terminated, a gradual withdrawal should be carried out, similar to that of α-2 agonists [82]. Because TCAs are processed by CYP2D6, slow metabolizers may benefit from a gradual upward dosage [82].

NON-PHARMACOLOGICAL TREATMENT:

AGE	NON-PHARMACOLOGICAL APPROACH
Preschool children	Parent training
Young school children	Parent training Classroom interventions
Older school children	Parent training, Multimodal approach involving parents, teachers
Adolescents	Classroom interventions Social skill training Cognitive behavioral therapy
Adults	Cognitive behavioral therapy Dialectical behavioral therapy

RECENT ADVANCES IN THE TREATMENT OF ADHD:

A) Parent-led behavioral therapy:

Parent-led behavioral treatment aims to address both the fundamental symptoms of ADHD and also the oppositional and non-compliant conduct that comes with it [92,93]. This treatment combines behavior modification techniques with novel therapeutic components based on social and cognitive development ideas [92]. Parents are taught behavioral methods for dealing with parent-child relationships, encouraging positive behavior, and reducing negative behavior [94].

B) Classroom-led behavioral therapy:

Following rules, communicating with peers, avoiding interruption of the teacher and peers, participating in educational activities, and self-organization are all things that children with ADHD may struggle with in the classroom [95]. Classroom-based behavioral therapies are administered in a real-world setting to provide training on desired behavior in the context in which it is needed [95]. These interventions, which combine behavior modification and cognitive behavioral modifying techniques, can be conveyed by teachers and administrators after receiving appropriate training [95,96].

C) Psychoeducation:

Psychoeducation is the process of providing knowledge on ADHD to people with this condition, their families, and others who are close to them [97-100]. Books and other forms of literature, as well as Web resources, can be used to deliver psychoeducation [98]. Although the media can be a useful instrument for disseminating knowledge and attitudes, it can also be a source of misleading assumptions and expectations [98].

D) Neurofeedback:

Neurofeedback [101,102] is a method of behavioral training that use computer-based activities to provide feedback on attention levels. An electroencephalogram is used to assess brain activity whenever a person with ADHD performs a task, which is commonly a computer game. The participant obtains points when their brain activity shows positive improvements [102,103]. Neurofeedback gives real-time feedback on attention levels during an activity [101]. After that, people with ADHD are trained to track and change their brainwave patterns [101].

CONCLUSION:

ADHD is a multifaceted illness that creates a slew of issues and reduces one's quality of life. A chemical imbalance of neurotransmitters like Dopamine and Norepinephrine in the PFC is considered to be to blame. According to studies, ADHD has been diagnosed in 2.4 percent of children aged 2 to 5 years old and 9.6 percent of children aged 6 to 11 years old. The DSM-V scale, as well as numerous neuroimaging methods such as fMRI and DTI, are being used as diagnostic tools for ADHD. The term "pharmacotherapy" refers to any treatment that involves the Drugs that treat this imbalance including stimulants (methylphenidate and amphetamines) and non-stimulants (atomoxetine, -2 agonists, bupropion, and TCAs). As a result, medications should be begun at a low dose and raised gradually, with patients' efficacy and side effects monitored on a frequent basis. Stimulants are the initial line of treatment. This only provides symptom alleviation for a short period of time. In recent years, research in this subject has taken on a much broader scope, and treatment is still a long way off.

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