Attention-deficit/hyperactivity disorder management: A biopsychosocial model

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ABSTRACT

Attention-deficit/hyperactivity disorder (ADHD) is the most common neurobehavioral disorder of childhood. It represents a complex problem because it appears at an early age, affects the daily lives of children from all areas of development, and is likely to persist throughout the life. The main management interventions in ADHD include pharmacotherapy, behavioral management, and family therapy which vary in their precedence according to age of the child and degree of impairment in the child. The biopsychosocial model tries to elucidate the interactions between biological, psychological, and social factors which determine the cause, manifestation, and outcome of any disease. In this article, we discuss how the biopsychosocial model of disease can be applied to ADHD to explain the symptoms as well as how it can help in planning of management at individual level.

Key words: Attention-deficit/hyperactivity disorder, Behavior therapy, Biopsychosocial model, Classroom management, Management

ttention-deficit/hyperactivity disorder (ADHD) is the most common neurobehavioral disorder of childhood; one of the most prevalent chronic health conditions affecting school-aged children and also the most extensively studied mental disorder of childhood [1]. It represents a complex problem because it appears at an early age, affects the daily lives of children from all areas of development, and is likely to persist throughout life [2]. Although the symptoms of ADHD begin in childhood, it can continue through adolescence and adulthood. Even though hyperactivity tends to improve as the child becomes an adolescent, problem with inattention, disorganization, and poor impulse control often continues through the adolescent years and into adulthood. Current understanding is that ADHD may be caused by interactions between genes and environmental factors. The non-genetic factors that may contribute to ADHD are (i) cigarette smoking, alcohol use, or drug use during pregnancy, (ii) exposure to environmental toxins, such as high levels of lead, at a young age, (iii) low birth weight, and (iv) brain injuries [3].

Although there is no global consensus, meta-regression analyses have estimated the worldwide ADHD prevalence to be between 5.3% and 7.1% in children and adolescents and at 3.4% in adults [4]. Evidence suggests that the prevalence of ADHD is greater in males than females. Gender ratios varied by country ranging from 1:3 to 1:16 in females:males [5]. The American Psychiatric Association identifies three core symptoms that characterize ADHD: Inattention, hyperactivity, and impulsivity. According to DSM V, six or more of the symptoms of inattention or hyperactivity/impulsivity should have persisted for at least 6 months to a degree that is maladaptive and inconsistent with development level to fulfill the diagnosis of ADHD. For children aged more than 17 years, only five criteria are necessary. Other criteria that have to be fulfilled include onset of symptoms before 12 years of age, presence in two or more settings, and clear evidence of significant impairment in social, academic, or occupational functioning. The symptoms should not occur exclusively during the course of a pervasive developmental disorder, schizophrenia, or other psychotic disorder and are not better accounted for by another mental disorder [6].

BIOPSYCHOSOCIAL APPROACH

The biopsychosocial model of health and illness is a framework developed by Dr. George L. Engel in 1977 that states that interactions between biological, psychological, and social factors determine the cause, manifestation, and outcome of wellness and disease [7]. The biopsychosocial model argues that any one factor is not sufficient and that it is the interplay between people's genetic makeup (biology), mental health and behavior (psychology), and cultural (social) context that determines the course of their healthrelated outcomes. It emphasizes the importance of understanding human health in a holistic context [8]. This approach can be used in explaining the symptoms and also targeting these symptoms in the management. It has been primarily used within clinical settings to address various psychological disorders including depression and anxiety as well as medical ailments, including cancer and pain [9]. The International Classification of Functioning, Disability, and Health: Children and Youth Version, endorsed by the World Health Organization (2007), is based on the biopsychosocial model that emphasizes how the child/adolescent

functions as a complex interaction between the individual factors and contextual factors of the environment [10].

ADHD, as a clinical and behavioral entity, may be considered likely to be approached from the biopsychosocial approach, since it is recognized that the basic neurobiological disorder is equally determined and influenced by other multiple external and environmental factors that can lead to important implications in daily, functional, and social activities and exacerbate the clinical manifestations and comorbidities in different contexts [11].

BIOLOGICAL ASPECT

ADHD is a neurobiological disorder of the prefrontal cortex and its connections. Many symptoms in ADHD-afflicted persons are comparable with symptoms from patients suffering from frontal lobe damage, which highlights the importance of the frontal cortical networks [12]. Prefrontal cortex determines executive functions of the brain which include the ability to (i) inhibit inappropriate behaviors and thoughts, (ii) regulate our attention, (iii) monitor our actions, and (iv) plan and organize for the future. Circuits within the prefrontal cortex relevant to ADHD include dorsal front-striatal, orbito-fronto-striatal, and frontocerebellar circuits [13]. Optimal levels of dopamine acting at D1 receptors and norepinephrine acting at postsynaptic alpha 2A-adrenoceptors are essential to prefrontal cortex function. Low levels of these neurotransmitters and abnormal activation of the fronto-striato-cerebellar circuits are seen in children with ADHD. Genetic factors such as mutations in DAT1 gene and DRD4 gene have been implicated for abnormalities in the function of neurotransmitter receptors [14].

Pharmacotherapy in ADHD is directed to the correction of the above defects in neurotransmitter regulation and circuitry. Stimulants such as methylphenidate and amphetamine group drugs target the dopamine transporter (DAT) and the noradrenaline transporter in presynaptic neuron, thereby inhibiting dopamine and noradrenaline reuptake into presynaptic neuron. Hence, there is increased dopamine and noradrenaline levels in the synaptic cleft for action in the prefrontal cortex.

Stimulants also cause direct stimulation of alpha2 and D1 receptors in postsynaptic neuron. In addition, amphetamine promotes the release of catecholamines from vesicles into the cytosol and from the cytosol into the synaptic cleft, thereby directly increasing their concentration in the synaptic cleft. Methylphenidate is the most widely used and recommended drug among the stimulants. Start with low dose and go slow in increasing the dose are the rules in stimulants. Start at 5-10mg/day and slowly increases every 5-7 days until optimal results are achieved or side effects appear [15]. Delayed release preparations have the advantage of less frequent dosing, more compliance, and less abuse potential but are more costly. Adverse effects include reduced appetite, abdominal discomfort, headache, irritability, sleep problems, and tachycardia. Small reduction in height velocity is an uncommon but significant concern, and hence, height and weight must be monitored at least semi-annually. If the height drops across two major centiles, stop the drug and treatment with a non-stimulant is to be instituted. Amphetamine group of drugs is presently not available in India for the treatment of ADHD because of abuse potential.

Another commonly used medication is atomoxetine - a selective noradrenaline reuptake inhibitor. It targets the norepinephrine transporter and increases extracellular levels of dopamine and noradrenaline in the prefrontal cortex because norepinephrine transporter in prefrontal cortex also causes reuptake of dopamine. However, in contrast to MPH, it does not increase dopamine levels in striatum or nucleus accumbens, so there are no drug abuse liabilities [16]. In comparative studies, the efficacy of atomoxetine is found to be non-inferior to immediaterelease methylphenidate but significantly less effective than the extended-release preparation. It is started at a dose of 0.5 mg/kg per day and later increased up to 1.2 mg/kg. The advantage of atomoxetine over stimulants includes lack of abuse risk and usefulness in comorbid anxiety or tic disorders. Adverse effects include somnolence, gastrointestinal upset/nausea, and reduced appetite and weight loss [17].

Alpha 2 agonists are third-line drugs used in the treatment of ADHD. They cause direct stimulation of α 2-adrenergic receptors leading to improve the efficiency of synaptic transmission in prefrontal cortex through the reduction of camp production. They are indicated, if the patient fails to respond to trials of two different stimulants, has intolerable side effects, or fails to respond to a trial of atomoxetine. They can also be used as an add-on to stimulants in patients with tic disorders. The main drugs in this group include clonidine and guanfacine. Guanfacine is a selective alpha-2a agonist and has the advantage of less sedation [18]. Other drugs that may be used in ADHD included antidepressants such as bupropion, venlafaxine, tricyclic antidepressants, and modafinil.

PSYCHOLOGICAL ASPECT

Psychological component of the biopsychosocial model seeks to find a psychological foundation for a particular array of symptoms and target them in treatment. Various neuropsychological theories have highlighted the behavior mechanisms reinforcing the symptoms of ADHD [19]. The dysfunctioning dopaminergic prefrontal connections lead to executive deficits and altered reinforcement and extinction processes, characteristic of ADHD. These include (i) problems in working memory, (ii) defective response inhibition (i.e., suppression of actions that are inappropriate in a given context and that interfere with goaldriven behavior), (iii) delay aversion (i.e., the tendency to choose a smaller immediate reward rather than wait for a large delayed reward), and (iv) deficient operant extinction. These deficits are targeted in behavior therapy. Most behavioral therapy programs for children with ADHD consider immediate positive reinforcement for good behavior (in the form of praise or social rewards) as an important component. In addition, caregivers of ADHD children should prevent the development of unwanted behavior, because the extinction deficit makes it difficult to reverse such behavior, once established. Hence, the second key element is to make consequences for bad behavior (negative reinforcement) clear, understandable, and consistent. Furthermore, minor attention-seeking inappropriate behaviors have to be ignored [20].

In adolescents with ADHD, cognitive behavior therapy can be applied for helping them develop a more planned and reflective way of thinking and behaving. It helps them to systematically work on their deficit areas and to adopt a more, systematic, and goal-directed approach to tasks and problem-solving. For example, children are encouraged to adopt a four-point scheme when faced with a problem or task: (i) What is the problem? (ii) What is my plan? (iii) Do I use my plan? And (iv) How did I do? It helps them to evaluate and monitor their own progress [21].

SOCIAL ASPECT

Social implications in ADHD are multidimensional. Disruptive social environments can predispose to the development of ADHD, and at the same time, ADHD itself can have manifold effects on the individual child and family. Parental ADHD is associated with disruptive family environment, suboptimal parenting practices, and negative emotional family environments, which predispose unfavorable behavioral development in an ADHD child and increase the risk of later oppositional defiant disorder and conduct disorder [22], particularly in boys. Other factors which increase risk include parental substance abuse or criminality, poor monitoring and supervision of children, low parental involvement, authoritarian child-rearing attitudes, harsh, lax or inconsistent discipline, and history of violent victimization. Research indicates that the presence of ADHD in a child is associated with disturbances in family and marital functioning, disrupted parent-child relationships, reduced parenting selfefficacy, and increased levels of parental stress [23]. Hence, parent education, family education, and family therapy play an important role in the management of ADHD.

ADHD in a child is associated with greater risks for low academic achievement, poor school performance, retention in grade, school suspensions, and expulsions. Schools often force children to conform to a narrow, predefined standard of child development and behavior. As children with ADHD, due to their inherent deficits in executive functions, find it difficult to conform to a socially constructed norm, they are considered maladaptive and suffer social rejection by peers which further lowers their self-esteem.

Children with ADHD may require changes in their educational program, including (a) provision of tutoring or resource room support (either in a one-on-one setting or within the classroom), (b) classroom modifications, (c) accommodations, and (d) behavioral interventions. The problem is that optimal contingencies only exist during the training session or under certain circumstances, and outside these, inconsistent and unpredictable contingencies are the rule. The school may, however, help an ADHD child to adjust to the school requirements by creating an optimal learning environment [24]. Classroom behavior management is an important aspect of ADHD management in school-going children. It can be given by mainstream teachers with appropriate training. Various strategies that can be used include (i) verbal reinforcement of good behavior, (ii) selectively ignore minor inappropriate attention-seeking behavior, (iii) remove nuisance/ distraction items, (iv) allow for "escape valve" outlets, (v) hurdle helping shadow teacher, (vi) peer mediation - study buddy, and (vii) proximity control - seat near the teacher. Academic difficulties can be helped by making assignments clear and not long and repetitive. Teachers have to be sensitive to self-esteem issues in the student [25,26].

Approximately 50% of ADHD children have significant problems in social relationships with other children because of impulsivity, poor listening skills, and aggressiveness [27]. Options include (i) social skill classes with role play which model different solutions to common social problems, (ii) problemsolving sessions with discussions with a small group of students where the conflict arise, and (iii) talking to each other in a supervised setting to resolve their problems.

CONCLUSION

It is important to understand that biological, psychological, and social factors play equally important roles in the pathogenesis and the manifestations of ADHD. Therefore, the management of child with ADHD should include components addressing all these areas, as appropriate for the age. Pharmacotherapy by a clinician will help in reducing the symptoms of ADHD, but behavior impairments and academic difficulties require behavior and family therapy and educational assistance from teachers. Similarly, vice versa, behavior therapy alone is inferior to pharmacotherapy combined with behavior therapy. Hence, the various modalities of treatment should not be considered mutually exclusive, but a right combination of them will help in ultimately improving the quality of life of the child and the family.

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