International Journal of Healthcare Sciences ISSN 2348-5728 (Online)
Vol. 8, Issue 1, pp: (158-161), Month: April 2020 - September 2020, Available at: www.researchpublish.com

Attention-deficit and Hyperactivity Disorder (ADHD) and Binge-Eating Disorder (BED) Comorbidity Mechanism: A Literature Review

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Abstract: **ADHD and BED comorbid nature is reviewed considering BED has a very strong relationship with obesity, a risk factor for cardiovascular diseases which is globally known as the leading cause of death. It is suggested that the comorbidity manifests as specific neurobiologic disorder in which ADHD and BED symptoms are overlapped. A review on the comorbidity of ADHD and BED is important as an effort to find out the pathological mechanism in both disorders and clinically, providing insights for effective case management strategies when both disorders occur simultaneously, as well as preventing BED behavior in ADHD cases through early detection of BED symptoms. The goal of this literature review is to further explore the currently proposed comorbidity mechanisms of both ADHD and BED pathophysiology.**

Keywords: **ADHD, Binge-eating Disorder, Comorbidity.**

I. INTRODUCTION

Attention Deficit and Hyperactivity Disorder (ADHD) is a pattern of persistent behavior in the form of lack of attention and / or impulsive behavior and hyperactivity that persists for at least 6 (six) months[1]. The prevalence of ADHD in school-age children around the world is reported to be around 3-7% and in America the prevalence of ADHD is reported around 2-26%. In other countries, it varies between 2-20% for example in Ukraine, the prevalence of ADHD in school-age children is reported to be 20%. The prevalence of ADHD in Indonesia is not known with certainty. Research in Jakarta reported a prevalence of ADHD of 4.2%, most commonly found in elementary school children and in boys[2]. In a study conducted by Saputro in 2009, the prevalence of ADHD in elementary school children in DKI Jakarta was 26.2% in the age range of 6-13 years[3]. ADHD can be categorized based on its dominant characteristics and severity. The characteristics of ADHD are divided into three types; inattention dominant, hyperactive-impulsive dominant, and combination of those two. The severity of ADHD is divided into three degrees; mild, moderate, and severe ADHD[4].

The characteristics of ADHD are manifestations of neurobiological disorders on structures in the brain that are involved in a reward pathway (RP) mechanism[5]. RP is a pathway in the brain that processes natural rewards—for example food and sex—into the release of dopamine (DA) in the dopaminergic system, which manifests in the form of feelings of pounding, precarious and desire for something. This pathway is influenced by a number of neurotransmitters in the brain, especially DA, as the primary neurotransmitter in the natural reward mechanism, obtained from activities such as eating and exercise[6]. In ADHD, the mechanism of inhibition of DA release in pre-synaptic neurons and / or DA receptors in post-synaptic neurons is dysfunctional so that DA levels in dopaminergic system structures are low[7]. These structures for example are the ventral tegmentum area, accumbens nucleus, olfactory tuberculus, ventral striatum, and most importantly, the frontal cortex. Furthermore, DA can also underlie other unnatural reward-seeking behaviors such as cigarette addiction, alcohol, and binge-eating behavior[5].

Binge-eating Disorder (BED) is an abnormality in eating patterns in the form of repeated binge-eating episodes in the form of eating in very large quantities and in a short time to cause discomfort, in however, the sufferers feel unable to control themselves during binge-eating episodes take place. More than 18 million women in the United States are

overweight and around 10-20% of them have BED and 60-75% of BED patients are women. This eating disorder usually starts when reaching adulthood. Three out of ten people who are on a diet have binge-eating disorder[1]. The global prevalence of BED is 0.9%). Although women (1.4%, 1.1-1.7%) have a greater prevalence than men (0.4%, 0.3-0.6%), there is no significant difference in prevalence in high-income countries (0.9%, 0.8-1.1%) compared to lower middle income countries (0.7%, 0.3-1.1%)[8]. The diagnosis of BED is based on the diagnostic criteria stated in DSM-5[1].

Further review on the comorbidity of BED with ADHD is important as an effort to gather out the pathological mechanism in both disorders and clinically, providing insights for effective case management strategies when both disorders occur simultaneously, as well as preventing BED behavior in ADHD children through early detection of BED symptoms. Knowledge about BED and ADHD comorbidity is important given the strong relationship between BED and obesity, which is a risk factor for cardiovascular diseases as the number one killer in the world[9]. The binge-eating behavior of ADHD children is only one of the other reward-seeking behaviors, which can create other problems in the community and one of which is drug abuse[6].

Based on the description above, it is estimated that ADHD affects the incidence of BED through RP or mesolimbic pathways and DA receptors in the dopaminergic system. In other words, comorbid ADHD and BED can be manifestations of specific neurobiological disorders in the brain with accompanying symptoms.

II. ATTENTION-DEFICIT AND HYPERACTIVITY DISORDER (ADHD)

Attention-deficit and Hyperactivity Disorder (ADHD) are disorders of the brain characterized by patterns of attention and / or hyperactivity-impulsivity that can interfere with one's development and life in general. Deficit of attention means deviating from the work or task done, lack of perseverance, difficulty in maintaining focus, and not organized. Hyperactivity means often moving constantly, including in situations that are less appropriate or making small movements, especially on the hands and feet (fidgeting), tapping fingers or feet (taps) or talking with excessive intensity. In adults, it can also manifests as extreme anxiety. Impulsivity means reckless, risky decision making without prior consideration. Impulsivity can also be interpreted as a desire to get rewarded as soon as possible or an inability to delay gratification (National Institute of Mental Health, 2017).

The prevalence of ADHD in the world in children under 18 years is 5.29% -7.1%. In the United States, the prevalence of ADHD in the 6-17 year age range is 2% to 18%. However, in a study conducted by Saputro in 2009 the prevalence of ADHD in elementary school age children in DKI Jakarta was 26.2% in the age range of 6-13 years. This figure is very much higher compared to the prevalence of ADHD in the world[3].

ADHD can be identified by excessive motor activity, impulsive nature, easily distracted so that it cannot maintain focus. The dominant symptom characteristic divides ADHD into three subtypes namely combination type, inatentive dominant type, and hyperactive-impulsive dominant type. Diagnosis of ADHD is based on DSM-5 or PPDGJ III adopted from DSM IV-TR [4].

The etiology of ADHD is still unknown, however, there are hypotheses regarding changes in volume or functionality of gray matter and white matter in the brain resulting in deficits in the processes of cognition, attention, motor planning, processing speed and other behavioral problems, as well as polymorphisms in DA receptors especially D2 receptors and D4. These changes are mainly focused on the structures traversed by the mesocortical and mesolimbic pathways (especially the prefrontal cortex (PFC), caudate and cerebellum) as part of the dopaminergic system in the brain associated with the acquisition aspect of a reward. In ADHD children, PFC develops more slowly, is smaller or more inactive than non-ADHD children[5].

Mesolimbic activity is influenced by neurotransmitters in the brain, especially DA, as the primary neurotransmitter in the reward mechanism. The mechanism of regulation of DA release by neurons in the mesocortical and mesolimbic pathways is divided into two, namely the tonic phase and the wicked phase. The tonic phase functions as a mechanism of inhibition of the wicked phase and is carried out continuously however, with a smaller quantity of DA than the wicked phase. In addition to disruption to the mechanism of DA inhibition and deficits in general, the etiology of ADHD also mentions the involvement of DA receptor polymorphisms that are responsible for impulse continuity so that abnormalities in these receptors can cause difficulty maintaining concentration, impulsivity, and hyperactivity[7].

III. BINGE-EATING DISORDER (BED)

Binge-eating disorder (BED) is an eating disorder characterized by episodes of binge-eating, which is eating in large quantities, with a short amount of time that causes discomfort. When the binge-eating episode progresses, sufferers feel unable to control themselves. Other characteristics include feelings of shame, depression and guilt after binge-eating episodes but, unlike bulimia nervosa, BED sufferers are not involved in compensatory actions in the form of forced vomiting. Before being categorized separately, BED is included in the EDNOS subtype on DSM IV-TR (or OSFED on DSM-5)[1].

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IV. MECHANISMS OF COMORBIDITY

The complex pathophysiology of GPPH can be traced from the typical clinical findings in ADHD, namely: 1) decrease in functionality of brain and 2) decrease in dopamine receptor density along with central amines, greatly influencing the development of brain.

4.1. Reduced Brain Function

Eating behavior is regulated by three kinds of pathways in the brain. Noting that eating behaviour setting consists of the importance of stimulus, reward system, dan control of consumption. The insula area regulates the stimulus for eating, while the sensation of reward and control of food are regulated by the ventral striatum, nucleus accumbens, putamen, caudate and dorsal caudate, dorsal accessories, prefrontal cortex, and parietal cortex area[10]. Primarily, the prefrontal cortex, caudate, and cerebellum has shown deficits in ADHD individuals. Deficits include slower maturation, smaller volume, and reduced activity. These areas are heavily dependent on neurotransmitter level throughout the brain[7].Meanwhile, overeating behaviour in BED is attributed to an elevated experience of reward, a reduced ability to control the drive to eat, or some combination of the two which refers to the activities of two out of three brain pathways mentioned. The function of PFC as a center of cognition, attention and planning is lacking in children with ADHD. The very same deficit appears in clinical symptoms as inattentiveness and impulsivity which could trigger an eating disorder in the form of overeating because of lack of memory and planning capability makes it difficult to have a normal eating patterns. In addition, impulsivity that results from inhibitory control deficit appears clinically as a delayed aversion or the need for a reward as soon as possible. As a characteristic of RDS, delayed aversion triggers BED behavior in children with GPPH through ignorance on daily calorie intake, which is sought as soon as possible where in normal children is achieved within a span of one day.

4.2. Brain Receptors Density

Dopamine D2 receptors (DR2) are G-protein coupled receptors located pre- and post-synaptically, which function as inhibitor to calcium ion entrance via voltage-gated channel. Dopaminergic and dopaminoceptive neurons are regulated by these receptors. Changes or alterations in these receptors' activity has shown an effect on feeding behaviors, in this case, binge-eating behavior[10]. The decrease in DA receptors density is caused by gene polymorphisms that regulate D4 and D5 receptors, as well as DA transporter 1 (DT-1). This causes a decrease in the function of the dopaminergic system. In α_{2A} receptors aiding norepinephrine's (NE) function, disruption was also found, causing impaired attention and impulsive behavior. The DT-1 gene polymorphism causes hyperactivity in the DA and NE systems thereby increasing DA efflux or decreasing DA reuptake in presynaptic neurons[7]. In binge eating disorder it is likely that the dopamine neurons stop responding to the primary reinforcer and then start responding to the conditioned stimulus[12]. All signs of decreased DA function are characteristic of Reward-deficiency Syndrome (RDS), which commpensate in the form of reward-seeking behaviors both natural and unnatural. Eating activity as a reward-seeking behavior that is natural, but is carried out abnormally, is the behavior chosen in children with ADHD and/or BED.

All these findings are also in-tune with pharmacodynamics of drugs used to treat ADHD such as methylphenidate. Methylphenidate works by increasing DA and NE transmission in PFC. Moreover, in proving whether DA uptake inhibition did influence appetite, a study comparing genotyped BED and non-BED individuals showed that reduced appetite was found in groups with methylphenidate[11]. This finding strengthens the comorbid relationship between ADHD and BED, or overeating behaviour at the very least.

V. DISCUSSION

Given that there are overlapped clinical symptoms in ADHD and BED, it is possible that they are the consequences of the very same brain structure involved in both conditions' pathology. Knowledge about BED and ADHD comorbidity is quite important given the strong relationship between overeating, BED, and obesity, which is a risk factor for cardiovascular diseases as the number one killer in the world[9]. The binge-eating behavior of ADHD children is only one of the other reward-seeking behaviors, which can create other problems in the community and one of which is drug abuse[6]. Further confirms their connections, a cross-sectional study in two mental health clinics shows significant association between ADHD and BED[9]. Although the results have came out clear, there will always be other possibilities explaining how ADHD medications could also suppress overeating behaviour, in which the children aren't diagnosed with BED yet.

However, the limitation is that this paper do recommend to always refer to provided diagnostic tools, and not to instantly diagnose these two conditions based on only comorbidity. Instead, should the results lead us to pay more attention to early screenings of both conditions.

VI. CONCLUSION

The characteristics of ADHD are manifestations of neurobiological disorders on structures in the brain that are involved in a reward pathway (RP) mechanism. The pathway is influenced by a number of neurotransmitters in the brain, especially DA, as the primary neurotransmitter in the natural reward mechanism, obtained from activities such as eating and exercise. In ADHD, the mechanism of inhibition of DA release in pre-synaptic neurons and / or DA receptors in post-synaptic neurons is dysfunctional so that DA levels in dopaminergic system structures are low. These structures for example are the ventral tegmentum area, accumbens nucleus, olfactory tuberculus, ventral striatum, and importantly, the frontal cortex. It is estimated that ADHD affects the incidence of BED through RP or mesolimbic pathways and DA receptors in the dopaminergic system. In other words, the comorbidity of ADHD and BED can be manifestations of specific neurobiological disorders in the brain with accompanying symptoms.

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