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Etiopathogenesis in Neuropsychological Aspects of Mood Disorders and Self-Harming Behavior in Adolescents: A Review

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Abstract: The average prevalence of self-harm in school-age adolescents in the world is 17.20% with bipolar spectrum disorders (37%) apparently higher than among adolescents with depressive disorders (13%). Based on etiopathogenesis, neuropsychological factors are associated with the emergence of mood disorders and self-harm behavior. The method used is the preparation of this article review was carried out through the process of searching, collecting data, and analyzing several articles from Science Direct, Proquest, Pubmed, and Google Scholar. The extracted data were analyzed to summarize the findings related to the etiopathogenesis of mood disorders and self-harm behavior in adolescents reviewed from a neurophysiological aspect. Discussion: Based on the results of the analysis of several articles, the etiopathogenesis of mood disorders and self-harm behavior in adolescents reviewed from a neurophysiological aspect is related to the influence of neurotransmitters and changes in the functional structure of the brain. The related neurotransmitters are serotonin, dopamine, GABA, glutamate, norepinephrine, and histamine. In adolescents who are not accompanied by psychological maturity, it can cause conflicts that result in emotional stress and distress as well as the emergence of negative emotions related to changes in brain function and structure related to brain neuroplasticity and disruption of the reward and punishment system in the limbic system.

Keyword: Adolescents; Mood disorders; Neuropsychology; Self-injurious behavior

Introduction

Mood disorders are a psychological illness that can be characterized by a loss of control over a person's emotions, energy, and motivation (Rakofsky & Rapaport, 2018). According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), mood disorders are categorized as depressive disorders (unipolar) and mood swing disorders (bipolar) (Corponi et al., 2020). Unipolar disorders are characterized by depressed mood, irritability, loss of motivation and interest in activities. Bipolar disorders are further divided into bipolar and cyclothymia which are characterized by episodes of mania and hypomania which are characterized by an increase in mood, abnormal increase in activity and energy that may or may not interfere with social functioning and endanger oneself or the environment, may or may not be accompanied by psychotic symptoms (delusional disorder), are not the effects of a substance or medical condition and. Mood disorders in bipolar can change at any time, initially experiencing an increase, then experiencing a rapid and alternating decrease in mood (Bobo, 2017).

According to the World Health Organization, the prevalence of mood disorders in the world in the age range of 10-19 years covers 16% of the global burden of disease and injury (Chen et al., 2024; Saikia et al., 2023). Based on the results of the Indonesian National Adolescent Mental Health Survey (I-NAMHS), the

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incidence of mental disorders including mood disorders in adolescents aged 10-17 years shows that 1 in 3 or around 15.50 million Indonesian adolescents have mood disorders, while one in twenty or 2.45 million Indonesian adolescents have mood disorders in the last 12 months (Agustin Syakarofath et al., 2024). Based on these data, adolescents are vulnerable to experiencing mood disorders because at this age they will experience changes in their body, thought patterns, behavior, social roles, and experience more fluctuating emotional turmoil (Paramita et al., 2021; Povey et al., 2022; Gajalakshmi & Meenakshi, 2023). Mood disorders occur in adolescents who have not achieved psychological stability due to immature thought patterns and unstable emotional control (Xiao et al., 2022).

Mood disorders can cause a person to experience prolonged negative emotions, increasing the occurrence of tension that makes the inability to control emotions. This inability can lead to self-harming behavior to cope with emotional pain in order to help oneself feel better (Ryoo & Kim, 2022; Edmondson et al., 2016; Gray et al., 2022). Injurious behavior is often found in adolescents as a form of channeling negative emotions as an effort to overcome or regulate painful emotional responses caused by mood disorders (Nurany et al., 2022). Self-harm behavior is an act of intentionally hurting or injuring oneself to relieve stress or difficulties felt in various ways such as cutting, cutting, burning, pulling hair, and others (Tofthagen et al., 2022). Self-harm behavior is not actually a mental disorder, but is a person's failure to cope with stress with the aim of overcoming emotional pain (Kusumadewi et al., 2020). The average prevalence of self-harm in school-age adolescents in the world is 17.20% (Yazici & Hocaoglu, 2023). Self-harm behavior peaks in adolescence between the ages of 13-17, with population rates ranging from 5 – 39%. In adults, the prevalence is around 6% in the general population, and around 20% in psychiatric patient samples.

While the rate of self-harm in adolescents with bipolar spectrum disorders (37%) appears to be higher than in adolescents with depressive disorders (13%) (Fang et al., 2022; Morgan et al., 2017). Meanwhile, the case in Indonesia is that 4 out of 10 Indonesians have experienced self-harm and suicidal ideation during the COVID-19 pandemic, especially people in the 18-24 age range, unemployed retirees and freelancers, women, members of minority and marginalized communities, and experiences of loneliness during the pandemic (Liem et al., 2022). Based on the background, this literature review will discuss the role of neuropsychological factors in the etiopathogenesis of mood disorders and their relationship to self-injurious behavior in adolescents. This literature review aims to analyze and further study the causes of neuropsychological aspects and current developments in mood disorders and self-injurious behavior that are rampant among adolescents so as to increase awareness and social support for cases of self-injurious behavior.

Method

This literature review uses various types of sources, namely articles in scientific journals, books, and government guidelines and related agencies. The preparation of this article review was carried out through a process of searching, collecting data, and analyzing several articles from Science Direct, Proquest, Pubmed, and Google Scholar. The extracted data was analyzed to summarize findings related to the role of neuropsychology in the etiopathogenesis of mood disorders and self-harm behavior in adolescents. The keywords used are "Etiopathogenesis", "Neurophysicology", "Mood Disorde", "Depression", "Bipolar", Self Injury, "Adolescents". The inclusion criteria used were scientific articles published in 2014-2024, scientific articles in Indonesian and English, and studies in the form of literature reviews, systematic reviews, and case studies. The exclusion criteria for this article search were non-scientific articles, editorials, or opinions. The extracted data was analyzed to summarize related findings.

Result and Discussion

Adolescence is a time full of changes in the body, behavioral patterns, and social roles. During this period, the center of the world of teenagers begins to shift from being at home and centered on the family, to being centered on peers and the environment. During adolescence, there is also an introduction to a number of new, complex emotions and more fluctuating emotional turmoil, so that an emotional regulation strategy is needed that is able to filter and hone the mindset of teenagers to be more mature (Ahmed et al., 2015; Mestre et al., 2017). Changes that occur during adolescence that are not accompanied by psychological maturity can cause conflict that results in emotional stress and distress as well as the emergence of negative emotions related to changes in brain function and structure (Antonovich & Sergeevich, 2022; Leo et al., 2023).

Structures in the brain and nerves have individual and collective functions that are influenced by their connections with each other, for example connections with the neuroendocrine system, especially the hypothalamic pituitary adrenal axis (HPA) which regulates the response to stress modulated by hormonal processes in the control of the autonomic nervous system. Other changes such as in the heart related to the parasympathetic and sympathetic also modify the input

to the brain (Rusch et al., 2023). The hippocampus plays a role in emotional processing of input and provides output to other networks in the brain. In the process, stem cells appear that are influenced by brain-derived neurotrophic factor in the brain (BDNF). The formation of these new cells comes from proinflammatory cytokines that can reduce neurogenesis, neural integrity and dendritic growth, thereby reducing the functional capacity of the hippocampus.

This is due to neuroplasticity that can be caused by genetics, stress-induced cell damage that will be accelerated by psychosocial stress (Miranda et al., 2019; Mattova et al., 2023). This stress will cause an increase in glucocorticoids that cause neuron atrophy by facilitating glutamatergic which plays a role in remodeling hippocampal dendrites by inhibiting glucose transport, thus disrupting neurogenesis and causing neuron damage. and changes that occur in the brain driven by stress are thought to support the emergence of mood disorders, such as depression and bipolar disorder (Malhi et al., 2021). Repeated and prolonged stressors will damage the neurotransmitter and intraneuronal working systems, including neuron loss and excessive reduction in synaptic contacts. Traumatic events cause disruption of HPA axis function, serotonin transmission, neuroplasticity, immunity, calcium signaling, and circadian rhythms experiencing changes in affect regulation, impulse control, reduced need for sleep, and decreased cognitive function and can increase the risk of self-harm.

Prolonged stressors also affect brain-derived neurotrophic factor in the brain (BDNF) and cause a decrease in serotonin, norepinephrine, and dopamine and atrophy of the hippocampus area. Mood disorders are caused by disorders of neurotransmitters such as serotonin, dopamine, GABA, glutamate, norepinephrine, and histamine. Serotonin is a neurotransmitter produced in the raphe nuclei and projected to the limbic system, basal ganglia, thalamus, cortex, and cerebellum. Increased serotonin reuptake in the presynaptic gap of the dorsal raphe nuclei is associated with symptoms of mood disorders such as insomnia, anxiety, and suicidal behavior. Dopamine is produced in the substantia nigra and is projected to the dorsal part of the striatum which plays a role in regulating motor function. The dopamine that is released plays a role in the experience of feeling pleasure. In people with depression, it is characterized by a decrease in dopamine concentration in the mesolimbic system (regulating emotional regulation and the reward system) with symptoms such as loss of interest and motivation, sadness, slow motor responses. In manic conditions with hyperactive behavior, it is also related to disruption of dopamine concentration (Ashok et al., 2017).

Norepinephrine is produced in the ventrolateral tegmental area and locus ceroleus which will be projected to the hypothalamus, reticular formation, prefrontal cortex, and thalamus. Its decrease is characterized by cognitive deficits, dysphoria, apathy, and fatigue which are usually found in depressive disorders, while an increase in norepinephrine levels is associated with bipolar disorder. GABA is an inhibitory amino acid neurotransmitter. Decreased synthesis and release of GABA as a response to acute and chronic stress. While glutamate as an excitatory neurotransmitter whose increase is associated with depressive disorders. Histamine plays a role in regulating memory, learning processes, endocrine balance, the need to eat, drink, and sleep. Increased histamine levels are associated with depressive disorders with decreased histamine H1 receptors associated with symptom severity (Cui et al., 2024). When someone with a mood disorder experiences negative emotions such as sadness, anger, despair, anxiety and frustration, it will increase the occurrence of tension which makes it impossible to control emotions, thus allowing dissociation to overcome this tension in the form of deliberate actions to harm oneself (Alifiando et al., 2022).

These findings are supported by the theory of the mechanism of depression that is directly involved in the process of self-harm behavior, which is the generation and release of opioid peptides (beta-endorphins), acting as pain relievers and mood enhancers through reducing negative affect. Therefore, it seems that depressive symptoms and self-harm behavior produce a neurobiochemical foundation. Self-harm behavior uses this common mechanism to control affect and this regulatory function can reinforce self-harm behavior (Shafti et al., 2021). Based on research by Radziwiłłowicz et al. (2017), It is stated that the severity of depression is correlated with self-harming behavior, this is also supported by research by Zhu et al. (2023) that the greater the symptoms of depression, the greater the tendency to self-harm. Research by (Sutin et al., 2018), stated that there is a significant positive correlation between depression and self-harming behavior, as reported in a study by Russell et al. (2020). In comparison, there is also a correlation between depression and self-harm in the CASE study in European countries namely Belgium, England, Hungary, Ireland, the Netherlands, and Norway and Australia in adolescents aged 14-17 years, where it was stated that increasing severity of self-harm was associated with higher levels of depression and also self-harm can act as a trigger for depression.

The goal for those who engage in self-harm behavior is to avoid negative emotions, which seems similar to a coping mechanism to overcome depression

or reduce frustration that comes from biological dysregulation (Kupferberg & Hasler, 2023). Depression is a mood disorder identified by evidence of depressed mood, anhedonia, and anergia associated with poorer mental well-being and subsequently increased risk of self-harming thoughts and behaviors (Lavretsky et al., 2010). The severity of depressive symptoms, such as self-deprecation and biological dysregulation, correlated with the importance of self-harm behavioral functions, including affect regulation, interpersonal boundary building, self-punishment, and dissociative escape. In contrast, depression was significantly correlated only with impaired self-reward and regulation when it manifested itself in social problems (Ait Oumeziane et al., 2019). When someone injures or hurts themselves, the body will respond with a positive effect, namely producing endorphins and released as pain relievers and negative feelings that provide temporary relief or a feeling of peace in a short time.

After the positive effect occurs, a negative effect will occur, namely someone will feel ashamed and guilty for the actions they have done (Avezahra et al., 2023). However, self-harm behavior has an addictive nature so that this feeling can make it difficult for the individual to stop the behavior so that if not treated immediately, the cycle will continue to repeat. Teenagers who self-harm still feel pain but some teenagers say that the pain caused is easier to heal than the emotional or mental pain that causes someone to do self-harm behavior (Parker & Davies, 2020). In someone who experiences recurrent mood disorders that can increase vulnerability to self-harm, for example in affective dysregulation and also impulsive behavior. People with bipolar tend to be more impulsive than mentally healthy people. Individuals with recurrent mood disorders experience neuroticism, namely a disposition to experience psychological distress and negative affect in various situations. In nonclinical and clinical populations, impulsivity and neuroticism can increase the likelihood of thoughts of self-harm (Carballo et al., 2020).

When the human brain senses a stressful stimulus, it will respond by activating parts of the brain to send physiological stress signals (increased heart rate, adrenaline surge, etc.). If the stressful stimulus passes quickly or can be reduced, cognitive coping mechanisms help to downregulate, or reduce the heightened emotional state, and the person feels better. However, in people with effective dysregulation, the biological pathways and coping mechanisms do not function properly and the mind remains stuck in a state of stress. Psychological theory and neurological research suggest that self-injurious behavior is an attempt to escape from prolonged and highly emotional states in people with deficits in emotional regulation (Allen et al., 2020). History of trauma or abuse affects cortical and subcortical development that directly affects the top-down process in the anterior cingulate cortex and bottom-up in the striatum involved in self-injurious behavior. This can be exacerbated by environmental factors, genetics, stress, mental disorders affect neuromaturation and potentially increase vulnerability. These factors will cause changes in brain structure and function. Self-injurious behavior involves structural and functional changes in the striatum, amygdala, and AC.

In self-injurious behavior with suicidal ideation, it involves connections between the posterior cingulate cortex network and the anterior insula network as well as between the ventrolateral prefrontal cortex and the amygdala. Meanwhile, self-injurious behavior without suicidal ideation shows changes in the relationship between the anterior cingulate cortex and the amygdala (Huang et al., 2020; Ho et al., 2021). Acute stress can directly impact brain development, and simultaneously, can disrupt top-down cortical processes associated with rumination, self-referential processing, and future-oriented thinking, which can lead to suicidal and non-suicidal ideation. Recurrent or chronic stress can overload the limbic system that modulates arousal and approach behavior, disrupting bottom-up and top-down connections, and potentially, facilitating the transition from ideation to behavior.

More broadly, stress also elicits a range of negative emotions (e.g., sadness, anger), and in the absence of effective emotion regulation strategies, these can then lead to self-injurious behavior (Navas-Casado et al., 2023). The brain's reward system consists of a frontal-striatal network between the limbic system, which produces hedonic experiences, and structures in the frontal cortex responsible for evaluating those experiences. When a stimulus is pleasurable, it produces a hedonic experience in the limbic (emotional) system and the evaluative frontal cortex forms a judgment about the experience (e.g., continuing to do it feels good or stopping because it doesn't feel good or has other bad consequences). Disrupted connectivity in brain structures in the striatal, frontal and limbic systems results in altered inhibitory control of emotions and pain processing in individuals with self-injury behavior. In addition, the POC structures involved in pain perception in humans, and the central opercular cortex covering the upper part of the insular lobe and thought to play a role in thermosensory and nociceptive processing.

Thus, disrupted connectivity in these regions may result in altered pain processing such that people who engage in self-injury are unable to perceive pain (Case et al., 2021). Individuals with a history of self-injury exhibit amygdala hyperactivity that is associated with increased distress and emotional dysregulation. In addition, increased activity in the hippocampus, anterior

cingulate cortex (ACC), and middle and inferior orbital frontal cortex (OFC) is associated with difficulty processing emotions. Given the role of the hippocampus in memory retrieval, it is possible that its hyperactivation signals the automatic retrieval of emotional memories in response to the perception of emotional images, a process that would also increase distress and self-injury. Prolonged self-injury behavior would lead to atypical processing of information about the self due to altered brain processing patterns that would characterize individuals to self-injure repeatedly (Timberlake et al., 2020; Colle et al., 2020; Masi et al., 2023).

Conclusion

The prevalence of mood disorders develops in adolescence because adolescence is a period full of changes in the body, behavioral patterns, and social roles. In adolescence, people begin to experience transitions and experience introduction to a number of new, complex emotions and experience more fluctuating emotional turmoil so that emotional regulation strategies are needed. Adolescents who have not experienced psychological maturity will find it difficult to regulate their emotions, which can cause mood disorders. Mood disorders can cause someone to experience prolonged negative emotions, increasing tension that makes them unable to control their emotions. This inability can lead to self-harm behavior to overcome emotional pain in order to help themselves feel better. In terms of etiopathogenesis in terms of neuropsychological aspects, mood disorders and self-harm behavior are influenced by neurotransmitter factors and changes in brain structure and function and are correlated with genetic and psychosocial factors.

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Author Contributions
Conceptualization; E. A.; methodology.; F. D.; validation; E. A.; formal analysis; F. D.; investigation.; E. A; resources; F. D.; data curation: E. A.; writing—original draft preparation. F. D.; writing—review and editing: E. A.; visualization: F. D. All authors have read and agreed to the published version of the manuscript.

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The authors declare no conflict of interest.

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